DOE/OR/07-1846/V4&D2 PRIMARY DOCUMENT

I - 04410 - 0012

Remedial Investigation Report for Waste Area Grouping 28 at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky

Volume 4 of 4 Baseline Risk Assessment



August 2000



DOE/OR/07-1846/V4&D2

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Volume 4 of 4. Baseline Risk Assessment

Date Issued—August 2000

Prepared for the U.S. Department of Energy Office of Environmental Management

by

BECHTEL JACOBS COMPANY LLC managing the Environmental Management Activities at the Paducah Gaseous Diffusion Plant under contract DE-AC05-98OR22700 for the U.S. DEPARTMENT OF ENERGY

PREFACE

This Remedial Investigation for Waste Area Grouping (WAG) 28 at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky (Vol. 4. Baseline Risk Assessment) (DOE/OR/07-1846/V4&D2) was prepared in accordance with the requirements under the Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) of 1980 and the Resource Conservation and Recovery Act (RCRA) and utilizes and references information found in Vols. I, II, and III of this report. This document provides information on the baseline risks posed to human health and the environment from contamination at WAG 28 that will be used to evaluate the need for remedial action in WAG 28 and to assist in the selection of the remedial alternatives. This report was prepared under Work Breakdown Structure 1.4.12.07.1.27.03 (Activity Data Sheet OR45301).

In accordance with Section IV of the draft *Federal Facilities Agreement for the Paducah Gaseous Diffusion Plant*, this integrated technical document was developed to satisfy both CERCLA and RCRA corrective action requirements. It is noted that the phases of the investigation process are referenced by CERCLA terminology within this document to reduce the potential for confusion.

ACKNOWLEDGMENTS

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This baseline risk assessment (BRA) was written and compiled by the WAG 28 BRA Report Team. This interdisciplinary team included L. D. Bloom, G. Holdsworth, N. Keene, A. Obery, B. Shaw, and C. J. E. Welsh. The T N & Associates, Inc., risk team would like to thank the technical oversight support provided by the Environmental Applications Group of the Center for Information Studies at the University of Tennessee. In addition, the T N & Associates, Inc., risk team would like to thank M. Cummins for technical editing, G. Stroupe for word processing, and D. Cardwell, S. Caudle, and their staff for document production.

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ACRONYMS

ABS	dermal absorption factor
ALAD	aminolevulinic acid dehydrase
ALS-PD	Amyotrophic Lateral Sclerosis and Parkinsonism-Dementia
AOC	area of concern
ARAR	Applicable or Relevant and Appropriate Requirement
ATSDR	Agency for Toxic Substances and Disease Registry
BAF	bioaccumulation factor
BaP	benzo(a)pyrene
BCWMA	Ballard County Wildlife Management Area
BERA	baseline ecological risk assessment
bgs	below ground surface
BHC	benzene hexachloride
BHHRA	baseline human health risk assessment
BRA	baseline risk assessment
BRHS	British Regional Heart Study
CAS	Chemical Abstracts Service
CDI	chronic daily intake
CERCLA	Comprehensive Environmental Response, Compensation, and Liability Act of 1980
CNS	central nervous system
COC	chemical of concern
COPC	chemical of potential concern
COPEC	chemical of potential ecological concern
DAF	dilution-attenuation factor
DNA	deoxyribonucleic acid
DNT	dinitrotoluene
DOE	U.S. Department of Energy
ELCR	excess lifetime cancer risk
EPA	U.S. Environmental Protection Agency
HEAST	Health Effects Assessment Summary Tables
HI	hazard index
HQ	hazard quotient
HSDB	Hazardous Substances Database
IAEA	International Atomic Energy Agency
ICRP	International Commission on Radiological Protection
IEUBK	Integrated Exposure Uptake Biokinetic (Model)
IQ I	intelligence quotient
IRIS	Integrated Risk Information System
KDEP	Kentucky Department of Environmental Protection
KDFWR	Kentucky Department of Fish and Wildlife Resources
KPDES	Kentucky Pollutant Discharge Elimination System
Kow	octanol-water partitioning coefficient
KSNPC	Kentucky State Nature Preserves Commission
LD _{so}	median lethal dose
LOAEL	lowest-observed-adverse-effects level
MCL	maximum contaminant level
MEPAS	Multimedia Environmental Pollutant Assessment System
MOC	medium of concern
NCI	National Cancer Institute

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NCDD	National Council on Radiation Protection and Measurement
NCRP NRC	National Research Council
NRCC	National Research Council of Canada
· · · ·	
NTP	National Toxicology Program
NOAEL	no-observed-adverse-effects level
ORNL	Oak Ridge National Laboratory
OSWER	Office of Solid Waste and Emergency Response
OU	operable unit
PAH	polycyclic aromatic hydrocarbon
PCB	polychlorinated biphenyl
PGDP	Paducah Gaseous Diffusion Plant
PHEA	public health and ecological assessment
POC	pathway of concern
PVC	polyvinylidine chloride
RAGS	Risk Assessment Guidance for Superfund
RAIS	Risk Assessment Information System
RBC	risk-based concentration
RCRA	Resource Conservation and Recovery Act
RDA	recommended dietary allowance
RfC	reference concentration
RfD	reference dose
RGA	Regional Gravel Aquifer
RGO	remedial goal option
RI	remedial investigation
RME	reasonable maximum exposure
SAS	Statistical Analysis System
SQL	sample quantitation limit
SSL	soil screening level
SWMU	solid waste management units
T&E	threatened and endangered (species)
TARA	Toxicology and Risk Analysis
TCE	trichloroethene
TEF	toxicity equivalency factor
TVA	Tennessee Valley Authority
TWA	time-weighted average
UCL	upper confidence limit
UCRS	Upper Continental Recharge System
UST	underground storage tank
UV	ultraviolet
VOC	volatile organic compound
WAG	waste area grouping
WKWMA	Western Kentucky Wildlife Management Area
44.12 AA 16147	Western Remucky Whome Wanagement Area

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EXECUTIVE SUMMARY

In 1999, the U.S. Department of Energy (DOE) conducted a Remedial Investigation (RI)/Resource Conservation and Recovery Act Facility Investigation for Waste Area Grouping (WAG) 28. WAG 28 includes Solid Waste Management Units (SWMUs) 99, 193, 194, and Area of Concern (AOC) 204 at the Paducah Gaseous Diffusion Plant (PGDP) in Paducah, Kentucky. SWMUs 99 and 193 were further subdivided into units based upon area and historical use (99a, 99b, 193a, 193b, and 193c.) The overall purpose of this investigation was to determine the presence, nature, and extent of contamination at SWMUs 99a, 99b, 193a, 193b, 193c, 194 and AOC 204. The primary focus of the RI was to collect sufficient information about surface soil, subsurface soil, and the shallow groundwater of the Upper Continental Recharge System (UCRS) contamination to support an assessment of risks to human health and the environment and the selection of remedial actions to reduce these risks. In addition, contamination in the Regional Gravel Aquifer (RGA) and McNairy Formation groundwater was characterized to determine if contamination in the sites acted as a secondary source of contamination to groundwater.

This baseline risk assessment utilizes information collected during the recently completed RI of WAG 28 and the results of previous risk assessments for sites in WAG 28 to characterize the baseline risks posed to human health and the environment from contact with contaminants in soil and groundwater. In addition, this baseline risk assessment uses results of fate and transport modeling to estimate the baseline risks posed to human health through contact with media impacted by contaminants migrating off site from the various sources in WAG 28. The ecological assessment focuses on exposure to contaminants in surface soil. Evaluation of off-site streams is deferred to the surface water operable unit. Baseline risks are those that may be present now or in the future in the absence of corrective or remedial actions. Methods used for fate and transport modeling are presented in Sect. 5 of Vol. 1 and Appendix B of Vol. 4.

Consistent with regulatory guidance and agreements contained in the approved human health risk assessment methods document (DOE 1996a), the baseline human health risk assessment (BHHRA) evaluates scenarios that encompass current use and several hypothetical future uses of the WAG 28 sites and the areas to which contaminants may migrate. The following scenarios are assessed:

- Current industrial—direct contact with surface soil (0–1 ft below ground surface)
- Future industrial—direct contact with surface soil and use of groundwater drawn from aquifers below WAG 28
- Future excavation scenario—direct contact with surface and subsurface soil (0–15 ft below ground surface)
- Future recreational user—ingestion of game exposed to contaminated surface soil
- Future on-site rural resident—direct contact with surface soil, use of groundwater drawn from aquifers below WAG 28, and ingestion of vegetables grown in this area
- Off-site rural resident—use of groundwater drawn from aquifers at the PGDP fence boundary

Also consistent with regulatory guidance and the strategy for the ecological risk assessment of source units (DOE 1993, EPA 1998c), the baseline ecological risk assessment (BERA) evaluates risks

under both current and potential future conditions to several nonhuman receptors that may come into --- contact with contaminated media at or migrating from sources in WAG 28. As with the BHHRA, information collected during the recently completed RI and from the fate and transport information in Sect. 5 of Vol. 1 and in the baseline risk assessment in Appendix B of Vol. 4 was used in the BERA.

Not every medium was present for the assessment of every land use for each of the sites assessed for risks to human health. The land uses and media assessed for risks to human health for each site in WAG 28 are presented in Table ES.1. Table ES.2 indicates the scenarios for which human health risk exceeds de minimis levels. Tables ES.3–ES.9 summarize the risk characterization results for each site.

Information collected during the WAG 28 RI will also be used in the plant-wide BHHRA and BERA for PGDP. These assessments will be completed at a future date as discussed in *Site Management Plan*, *Paducah Gaseous Diffusion Plant*, *Paducah*, *Kentucky* (DOE/OR/07-1207&D2) (DOE 1996b).

	Location									
Land use scenario	SWMU 99a	SWMU 99b	SWMU 193a	SWMU 193b	SWMU 193c	SWMU 194	AOC 204			
Current industrial worker		•								
Surface soil	X	· .	x	X	x					
Current terrestrial biota			x							
Future industrial worker										
Surface soil	х		х	х	х					
RGA groundwater	х	х	х	х	х		х			
McNairy groundwater	x	л	х	х	• X					
Future excavation worker	•									
Surface and subsurface soil	x	х	X	X	Х	Х	х			
Future recreational user							-			
Soil (game)	x		х	Χ.	Х					
Future on-site rural resident						· .				
Surface soil	X	· ,	X	х	Х					
RGA groundwater	X	Х	X	х	х					
McNairy groundwater	x	•	X .	X	X		Х			
Off-site rural resident				·						
Groundwater	X	X	Х	Х	X		X			
Future terrestrial biota	• X		х	X	х					

Table ES.1. Land uses and media assessed for WAG 28 sites

Notes: Scenarios that were assessed in this baseline risk assessment are marked with an "X."

Major conclusions and observations of the BHHRA and BERA are presented below.

General

For all sites, the cumulative human health excess lifetime cancer risk (ELCR) and systemic toxicity exceed the accepted standards of the Kentucky Department of Environmental Protection (KDEP) and the U.S. Environmental Protection Agency (EPA) for one or more scenarios when assessed using default exposure parameters. The scenarios for which risk exceeds de minimis levels [i.e., a cumulative ELCR of 1E-6 or a cumulative hazard index (HI) of 1] are summarized in Table ES.2. This information is taken from the risk summary tables (Tables ES.3–ES.9), which present the cumulative risk values for each scenario, the chemicals of concern (COCs), and the pathways of concern (POCs).

	Site									
Scenario	SWMU	SWMU		SWMU	SWMU	SWMU	AOC			
	99a	99b	193a	193b	193c	194	204			
Systemic toxicity ^a		<u> </u>								
Current industrial worker				a .rh	376					
Exposure to soil	· · - ·	NA		Xb	Xc	NA	NA			
Future industrial worker			1.12	h						
Exposure to soil		NA	-	Xb	X°	NA	NA			
Exposure to RGA groundwater	Xď	Xb	Xb	Xb	Xb	NA	Хp			
Exposure to McNairy groundwater	Xb	NA	Xp	-	\mathbf{X}^{d}	NA	NA			
Future on-site rural resident ^a										
Exposure to soil	Xb	NA	Xb	Xb	Xď	NA	NA			
Exposure to RGA groundwater	\mathbf{X}^{d}	Xb	Xp	Xp	Xb	NA	Xp			
Exposure to McNairy groundwater	X٥	NA	X۴	Xb	\mathbf{X}^{d}	NA	NA			
Off-site rural resident										
Exposure to groundwater ^e	Xe		Xe		Xe	Xe	Xe			
			Л	-	Λ	Λ	л			
Future recreational user ^a		27.4			370	27.4	27.4			
Exposure to soil	-	NA	_		Xc	NA	NA			
Future excavation worker	d			·b	d	6				
Exposure to soil	Xď	-	· · -	Xb	Xď	X°				
Excess lifetime cancer risk					. •					
Current industrial worker										
Exposure to soil	Х	NA	Х	Х		NA	NA			
Future industrial worker	Þ									
Exposure to soil	Χ.	NA	х	X	_	NA	NA			
Exposure to RGA groundwater	х	X	X	X	х	NA	x			
Exposure to McNairy groundwater	x	NA	х	·	X -	NA	⁻ NA			
Future on-site rural resident ^f	•	· · · ·	· ·			• .				
Exposure to soil	\mathbf{X}^{*}	NA	x	х	-	NA	NA			
Exposure to RGA groundwater	x	X	X	x	х	NA	X			
Exposure to McNairy groundwater	x	NA	x	x	x	NA	NA			
<u> </u>	А	1177	л	Λ	1	1121	лл			
Off-site rural resident										
Exposure to groundwater ^e		-	-	-	_	_	Xe			
Future recreational user ^f										
Exposure to soil	Х	NA	X	-	·	NA	NA			
Future excavation worker										
Exposure to soil	Х	X	X	х	х	Х	X -			

Table ES.2. Scenarios for which human health risk exceeds de minimis levels

Notes: Scenarios where risk exceeded benchmark levels (HI of 1/ELCR of 1E-6) are marked with an "X." Scenarios where risk did not exceed a benchmark level are marked with a "-."

"NA" indicates that the scenario/land use combination is not appropriate.

^a For the future recreational user and the future on-site rural resident, the results for a child are presented.

^b These scenarios are of concern even though lead was not detected.

^c If contribution from lead is not considered, the total HI falls below I, and the scenario is not of concern.

^d Lead is present, and the scenario is of concern whether or not the element is included in the assessment.

^e Based on the results of contaminant transport modeling, "X" indicates that the location contains a source of unacceptable off-site contamination.

^f For excess lifetime cancer risk regarding the future recreational user and the future on-site rural resident, the valves are for lifetime exposure.

	1	=	1		1	· ·	· .	;	1	
Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total HI	COCs	% Total HI	POCs	% Total HI
Current industrial worker at current concentrations (soil only)	3.1E-4	Beryllium Benzo(a)pyrene Benzo(b)anthracene Benzo(b)fluoranthene Cesium-137 Dibenz(a,h)anthracene Indeno(1,2,3-cd)pyrene Neptunium-237 Uranium-238	70 6 1 3 3 1 9 5	Incidental ingestion Dermal contact External exposure	2 81 17	H] < 1	-	-		-
Future industrial worker at current concentrations (soil only)	3.1E-4	Beryllium Benzo(a)pyrene Benzo(b)anthracene Benzo(b)fluoranthene Cesiuim-137 Dibenz(a,h)anthracene Neptunium-237 Uranium-238	70 6 1 3 3 9 5	Incidental ingestion Dermal contact External exposure	2 81 17	HI < 1		_		_
Future industrial worker at current concentrations (RGA groundwater only)	5.6E-4	1,1-Dichloroethene Arsenic Beryllium Trichloroethene Radon-222	14 5 38 8 35	Incidental ingestion Dermal contact Inhalation of vapors/particles	44 12 43	5.11	Aluminum Arsenic Chromium Iron Manganese Trichloroethene Vanadium	2 4 10 15 8 42 14	Ingestion Dermal contact Inhalation of vapors/particles	73 15 12
Future industrial worker at current concentrations (McNairy groundwater only)	7.6E-5	1,1-Dichloroethene Carbon tetrachloride Trichloroethene	61 2 37	Ingestion Dermal contact Inhalation of vapors/particles	52 10 38	1.64	cis-1,2- Dichloroethene Trichloroethene Carbon tetrachloride	11 84 4	Ingestion Dermal contact Inhalation of vapors/particles	53 17 29
Future child rural resident at current concentrations (soil only)	NA	NA	NA	NA	NA	17.2	Barium Beryllium Chromium PCB-1016 PCB-1254 Pyrene Zinc	19 4 28 18 26 < 1 4	Ingestion Dermal contact Ingestion of vegetables	1 18 81

Table ES.3. Summary of human health risk characterization for SWMU 99a without lead as a COPC





Table ES.3. (Continued)

Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total HI	COCs	% Total HI	POCs	% Total HI
Future child rural resident	NA	NA	NA	NA	NA	97.3	1,1-Dichloroethene	1	Ingestion	26
at current concentrations	· ·						Aluminum	1	Dermal contact	2
(RGA groundwater only)						1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 - 1997 -	Arsenic	2	Inhalation of	51
(non ground and only)	- · ·						Barium	<1	vapors/particles	
- 1		· · ·					Bervillium	<1	Ingestion of vegetables	21
· · · · ·							Chromium	5	ingestion of regeneties	
							<i>cis</i> -1,2-	<1	· .	
÷							Dichloroethene	~ 1		
							Cobalt	< 1		
							Copper	<1		
							Iron	9	1	
· · ·							Lithium	<1		
								3		
· · · · ·		· · · ·					Manganese	<1	·	
							Mercury	-		
. • I							Nickel	<1		
							Trichloroethene	68	· · · · ·	· .
· · · ·							Vanadium	6		
							Zinc	< 1		
Future child rural resident at current concentrations (McNairy groundwater only)	NA	NA	NA	NA	NA	53.1	1,1-Dichloroethene Carbon tetrachloride cis-1,2- Dichloroethene Trichloroethene	1 5 13 80	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	11 1 73 15
Future adult rural resident	>1E-2*	Beryllium	< 1	Ingestion	<1	5.05	Barium	19	Dermal contact	12
at current concentrations	- 12-2	Benz(a)anthracene	<1	Dermal Contact	<1	5.05	Beryllium	3	Ingestion of vegetables	88
(soil only)		Benzo(a)pyrene	<1	External Exposure	<1		Chromium	25	ingestion of vegetables	
(son only)		Benzo(a)fluoranthene	<1	Ingestion of vegetables	99		PCB-1016	20	· .	
		Cesium	<1	ingestion of vegetables	,,,,		PCB-1254	28		
		Cesium	_				Zinc	4		1
		Change								
	1.1	Chrysene	<1				Zinc		· .	
		Dibenz(a,h)anthracene	<1				Zinc		n at t	
		Dibenz(a,h)anthracene Indeno(1,2,3-cd)pyrene	<1 <1				Line			
. I		Dibenz(a,h)anthracene Indeno(1,2,3-cd)pyrene Neptunium-237	<1 <1 <1							
		Dibenz(a,h)anthracene Indeno(1,2,3-cd)pyrene Neptunium-237 PCB-1016	<1 <1 <1 <1							
		Dibenz(a,h)anthracene Indeno(1,2,3-cd)pyrene Neptunium-237 PCB-1016 PCB-1254	<1 <1 <1 <1 <1 <1							
		Dibenz(a,h)anthracene Indeno(1,2,3-cd)pyrene Neptunium-237 PCB-1016 PCB-1254 PCB-1260	<1 <1 <1 <1 <1 <1 <1							
		Dibenz(a,h)anthracene Indeno(1,2,3-cd)pyrene Neptunium-237 PCB-1016 PCB-1254 PCB-1260 Technetium-99	<1 <1 <1 <1 <1 <1 <1 96							
		Dibenz(a,h)anthracene Indeno(1,2,3-cd)pyrene Neptunium-237 PCB-1016 PCB-1254 PCB-1260 Technetium-99 Thorium-234	<1 <1 <1 <1 <1 <1 <1 96 <1							
		Dibenz(a,h)anthracene Indeno(1,2,3-cd)pyrene Neptunium-237 PCB-1016 PCB-1254 PCB-1260 Technetium-99	<1 <1 <1 <1 <1 <1 <1 96							

Table ES.3. (Continued)

Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total HI	COCs	% Total HI	POCs	% Total HI
Future adult rural resident at current concentrations (RGA groundwater only)	5.6E-3	1,1-Dichloroethene Arsenic Beryllium bis(2-Ethylhexyl)pthalate Trichloroethene Radon-222 Technetium-99	40 4 26 < 1 11 9 9	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	24 3 45 28	28.3	1,I-Dichloroethene Aluminum Arsenic Barium Beryllium Chromium <i>cis</i> -1,2- Dichloroethene Iron Lithium Manganese Nickel Tricholoroethene Vanadium	1 2 3 <1 <1 7 <1 11 <1 4 1 59 9	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	37 4 36 23
Future adult rural resident at current concentrations (McNairy groundwater only)	1.7E-3	1,1-Dichloroethene Carbon tetrachloride Trichloroethene	75 1 24	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	12 1 70 17	13.3	1,1-Dichloroethene cis-1,2- Dichloroethene Carbon tetrachloride Trichloroethene	1 13 5 80	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	18 3 60 19
Future child recreational user at current concentrations (soil only)	NA	NA	NA	NA	NA	HI < 1	-	-	·	-
Future teen recreational user at current concentrations (soil only)	NA	ŇĂ	NA	NA	NA	HI < 1	. .	-		-
Future adult recreational user at current concentrations (soil only)	2.7E-6	Dibenz(a,h)anthracene	38	Ingestion of venison Ingestion of rabbit Ingestion of quail	11 72 11	HI < 1		-	-	-



Table ES.3. (Continued)

Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total HI	COCs	% Total HI	POCs	% Total HI
Future excavation worker at current concentrations	2.1E-4	Aldrin Arsenic Benz(a)anthracene Benzo(a)pyrene	<1 5 <1 11	Ingestion Dermal contact Inhalation of vapors/particles	21 63 < 1	1.46	2-Nitroaniline Antimony Chromium Manganese	8 35 16 14	Ingestion Dermal contact Inhalation of vapors/particles	13 83 3
. · ·		Benzo(b)fluoranthene Beryllium bis(2-chloroethyl)ether	1 35 < 1	External exposure	16		Aluminum Arsenic Barium	6 5 3		
	· .	Cesium-137 Dibenz (a,h) anthracene	37				Beryllium Cadmium	2		
		Dieldrin Hexachlorobenzene Indeno (1,2,3-cd) pyrene	<1 1 <1	· · · · · ·						
		Neptunium-237 N-nitroso-di-n-propylamine	11 8						a de la companya de	
		Thorium-234 Toxaphene	<1 <1							
	•	Uranium-234 Uranium-238	< 1 7							

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Notes: NA = ECLR not applicable to child and teen cohorts. Values for adult include exposure as child and teen. none = ELCR or HI is above the benchmark, but no COCs or POCs fulfill the selection criteria. ND = No Data (no samples were taken from the medium under consideration).

- = There are no COCs or POCs.

*= The ELCR is approximate because the linearized multistage model returns imprecise values at risks > 1E-2.

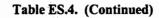
Table ES.4. Summary of human health risk characterization for SWMU 99b without lead as a COPC

•						r —					
Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total Hi	COCs	% Total HI	POCs	% Total H1	
Current industrial worker at current concentrations (soil only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Future industrial worker at current concentrations (soil only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Future industrial worker at current concentrations (RGA groundwater only)	2.6E-4	Radon-222 Trichloroethene	47 53	Ingestion Dermal contact Inhalation of vapors/particles	31 12 57	7.00	Chromium Trichloroethene	3 94	Ingestion Dermal contact Inhalation of vapors/particles	54 19 26	
Future industrial worker at current concentrations (McNairy groundwater only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Future child rural resident at current concentrations (soil only)	NA/ND	NA/ND	NA/ND	NA/ND	NA/ND	ND	ND	ND	ND	ND	
Future child rural resident at current concentrations (RGA groundwater only)	`NA	NA	NA	NA	NA	208	Barium Chromium Iron Manganese Trichloroethene	<.1 1 <1 <1 98	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	12 2 71 15	
Future child rural resident at current concentrations (McNairy groundwater only)	NA/ND	NA/ND	NA/ND	NA/ND	NA/ND	ND	ND	ND	ND	ND	
Future adult rural resident at current concentrations (soil only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
Future adult rural resident at current concentrations (RGA groundwater only)	2.3E-3	Radon-222 Trichloroethene	13 87	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	19 4 58 20	52.9	Barium Chromium Iron Manganese Trichloroethene	<1 2 <1 <1 97	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	20 4 58 18	

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Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total HI	COCs	% Totai HI	POCs	% Total HI		
Future adult rural resident at current concentrations (McNairy groundwater only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND		
Future child recreational user at current concentrations (soil only)	NA/ND	NA/ND	NA/ND	NA/ND	NAND	ND	ND	ND	ND	ND		
Future teen recreational user at current concentrations (soil only)	NA/ND	NA/ND	NA/ND	NA/ND	NA/ND	ND	ND	ND	ND	ND		
Future adult recreational user at current concentrations (soil only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND		
Future excavation worker at current concentrations	2.1E-4	Arsenic Beryllium	7 93	Ingestion Dermal contact Inhalation of vapors/particles	5 95 < 1	HI < I	_	-	-	· . –		

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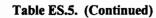
Notes: NA = ECLR not applicable to child and teen cohorts. Values for adult include exposure as child and teen. none = ELCR or HI is above the benchmark, but no COCs or POCs fulfill the selection criteria. ND = No Data (no samples were taken from the medium under consideration).

- = There are no COCs or POCs.

Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total HI	COCs	% Total HI	POCs	% Total HI
Current industrial worker at current concentrations (soil only)	1.5E-5	Benzo(a)pyrene Dibenz(a,h)anthracene	60 31	Dermal contact Ingestion	97 4	HI < 1	-	-	-	-
Future industrial worker at current concentrations (soil only)	1.5E-5	Benzo(a)pyrene Dibenz(a,h)anthracene	60 <u>3</u> 1	Dermal contact Ingestion	97 4	HI < 1	-	-		-
Future industrial worker at current concentrations (RGA groundwater only)	2.6E-5	Pentachlorophenol Technetium-99 Trichloroethene 1,1-Dichloroethene bis(2-Ethylhexyl)phthalate	45 6 42 3 3	Ingestion Dermal contact Inhalation	48 42 9	1.64	Iron Trichloroethene Fluroide	62 33 4	Ingestion Dermal contact Inhalation of vapors/particles	82 9 9
Future industrial worker at current concentrations (McNairy groundwater only)	1.1E-6	none	· -	None	-	4.69	Iron cis-1,2- Dichloroethene	94 6	Ingestion Dermal contact Inhalation of vapors/particles	96 2 2
Future child rural resident at current concentrations (soil only)	NA	NA	NA	NA	NA	6.25	Chromium	99	Dermal contact Ingestion of vegetables	40 59
Future child rural resident at current concentrations (RGA groundwater only)	NA	NA	NA	NA	NA	28.6	Fluoride Iron Trichloroethene <i>cis</i> -1,2- Trichloroethene	2 39 58 < 1	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	32 1 43 24
Future child rural resident at current concentrations (McNairy groundwater only)	NA	NA	NA	NA	· NA	59.9	<i>cis</i> -1,2- Dichloroethene Iron Trichloroethene	17 82 < 1	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	51 < 1 13 36
Future adult rural resident at current concentrations (soil only)	7.1Ė-4	Benz(a)anthracene Benzo(a)pyrene Benzo(b)fluoranthene Dibenz(a,h)anthracene Indeno(1,2,3-cd)pyrene	4 60 1 31 4	Ingestion Dermal contact Ingestion of vegetables	< 1 6 93	1.66	Chromium	99	Dermal contact Ingestion of vegetables	29 70

Table ES.5. Summary of human health risk characterization for SWMU 193a without lead as a COPC





Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total HI	COCs	% Total HI	POCs	% Tota HI
Future adult rural resident at current concentrations (RGA groundwater only)	2.4E-3	1,1-Dichloroethene bis-(2-Ethylhexyl) phthalate Pentachlorophenol Technetium-99 Trichloroethene	1 < 1 2 90 7	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	3 1 4 92	8.69	Fluoride Iron Trichloroethene	2 49 48	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	43 2 29 25
Future adult rural resident at current concentrations (McNairy groundwater only)	4.1E-4	Technetium-99 Tricholorethene Uranium-238	98 < 1 < 1	Ingestion Inhalation of vapors/particles Ingestion of vegetables	1 < 1 99	21.2	<i>cis</i> -1,2- Dichloroethene Iron	12 87	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	59 < 1 7 33
Future child recreational user at current concentrations (soil only)	NA	NA	NA	NA	NA	HI < 1		-		, -
Future teen recreational user at current concentrations (soil only)	NA	NA	NA	NA	NA	HI < 1		-		-
Future adult recreational user at current concentrations (soil only)	3.6E-6	Benzo(a)pyrene Dibenz(a,h)anthracene	35 59	Ingestion of venison Ingestion of rabbit Ingestion of quail	31 57 12	HI < 1		-		-
Future excavation worker at current concentrations	1.7E-4	Beryllium Benzo(a)pyrene Dibenz(a,h)anthracene	91 5 3	Ingestion Dermal contact	4 96	.HI < 1	-	-		-

Notes: NA = ECLR not applicable to child and teen cohorts. Values for adult include exposure as child and teen. none = ELCR or HI is above the benchmark, but no COCs or POCs fulfill the selection criteria.

ND = No Data (no samples were taken from the medium under consideration).

- = There are no COCs or POCs.

Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total HI	COCs	% Total HI	POCs	% Tota Hl		
Current industrial worker at current concentrations (soil only)	5.1E-4	Beryllium	100	Ingestion Dermal contact	< 1 100	5.25	Beryllium Chromium Vanadium	3 60 37	Dermal contact	100		
Future industrial worker at current concentrations (soil only)	5.1E-4	Beryllium	100	Ingestion Dermal contact	< 1 100	5.25	Beryllium Chromium Vanadium	3 60 37	Dermal contact	100		
Future industrial worker at current concentrations (RGA groundwater only)	4.4E-5	1,1-Dichloroethene Carbon tetrachloride Trichloroethene	16 8 74	Ingestion Dermal contact Inhalation of vapors/particles	59 18 23	1.74	Carbon tetrachloride Trichloroethene	8 90	Ingestion Dermal contact Inhalation of vapors/particles	52 19 29		
Future industrial worker at current concentrations (McNairy groundwater only)	< 1E-6	-		-	-	HI < 1	-	-		-		
Future child rural resident at current concentrations (soil only)	NA	. NA	NA	NA	NA	66.7	Beryllium Chromium Vanadium	3 68 30	Ingestion Dermal contact Ingestion of vegetables	< 1 46 53		
Future child rural resident at current concentrations (RGA groundwater only)	NA	NA	NA	NA	NA	55.5	1,1-Dichloroethene Acetone Carbon tetrachloride <i>cis</i> -1,2- Dichloroethene Trichloroethene	<1 1 9 <1 88	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	11 2 73 15		
Future child rural resident at current concentrations (McNairy groundwater only)	NA	NA	NA	NA	NA	2.69	cis-1,2- Dichloroethene Trichloroethene	53 47	Ingestion Inhalation of vapors/particles Ingestion of vegetables	11 71 17		
Future adult rural resident at current concentrations (soil only)	3.0E-3	Beryllium	100	Ingestion Dermal contact Ingestion of vegetables	1 50 49	17.3	Beryllium Chromium Vanadium	2 69 28	Dermal contact Ingestion of vegetables	34 65		
Future adult rural resident at current concentrations (RGA groundwater only)	1.0E-3	1,1-Dichloroethene Carbon tetrachloride bis(2-Ethylhexyl)phthalate Technetium-99 Trichloroethene	20 5 < 1 29 46	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	14 2 40 44	13.9	Acetone Carbon tetrachloride <i>cis</i> -1,2- Dichloroethene Trichloroethene	1 9 <1 88	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	18 3 60 18		

Table ES.6. Summary of human health risk characterization for SWMU 193b without lead as a COPC

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Table ES.6. (Continued)

Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total HI	COCs	% Total HI	POCs	% Totai HI
Future adult rural resident at current concentrations (McNairy groundwater only)	1.2E-5	Trichloroethene	. 100	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	22 4 50 23	HI < 1	_	-	-*	
Future child recreational user at current concentrations (soil only)	NA	NA	NA	NA	NA	H1 < 1	-	-		-
Future teen recreational user at current concentrations (soil only)	NA	NA	NA	NA	NA	H1 < 1	-	-	-	
Future adult recreational user at current concentrations (soil only)	< 1E-6	_	-		-	H1 < 1	_	-	-	-
Future excavation worker at current concentrations	1.7E-4	Beryllium	100	Ingestion Dermal contact	2 98	1.75	Chromium Vanadium	59 37	Dermal contact Ingestion	97 3

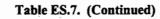
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Notes: NA = ECLR not applicable to child and teen cohorts. Values for adult include exposure as child and teen. none = ELCR or HI is above the benchmark, but no COCs or POCs fulfill the selection criteria. ND = No Data (no samples were taken from the medium under consideration).
 - = There are no COCs or POCs.

				· · ·			•		·	
Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total HI	COCs	% Total HI	POCs	% Tota HI
Current industrial worker at current concentrations (soil only)	< 1E-6	-	-	_	_	H1 < 1	-		-	-
uny,										
Future industrial worker at current concentrations (soil only)	< 1E-6	-		· · -	-	HI < 1	-	-	-	
				. · ·				. ·		
Future industrial worker at current concentrations (RGA groundwater only)	1.0E-5	Trichloroethene	100	Ingestion Dermal contact Inhalation of vapors/particles	59 23 18	1.46	1,2-Dichloroethene Trichloroethene	65 35	Ingestion Dermal contact Inhalation of vapors/particles	60 7 33
Future industrial worker at current concentrations (McNairy groundwater only)	4.2E-4	1,1-Dichloroethene 1,2-Dichloroethane Arsenic Beryllium Carbon tetrachloride Tetrachloroethene Radon-222 Vinyl chloride	3 <1 15 54 <1 <1 11 16	Ingestion Dermal contact Inhalation of vapors/particles	71 15 14	9.92	Aluminum Antimony Arsenic Cadmium Chromium Iron Manganese Vanadium	4 33 4 10 6 20 3 16	Ingestion Dermal contact	86 14
Future child rural resident at current concentrations (soil only)	NA	NA	NA	NA	NA	3.04	Chromium Zinc	91 9	Dermal contact Ingestion of vegetables	37 62
Future child rural resident at current concentrations (RGA groundwater only)	NA .	NA	NA	NA	NA	80.7	1,2-Dichloroethene Trichloroethene	80 20	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	7 <1 48 45

Table ES.7. Summary of human health risk characterization for SWMU 193c without lead as a COPC





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Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total HI	COCs	% Total HI	POCs	% Total HI
Future child rural resident at current concentrations (McNairy groundwater only)	NA	NA	NA	NA	NA	103	1,1,2-Trichloroethane 1,1-Dichloroethene 1,2-Dichloroethane Aluminum Antimony	< 1 < 1 < 1 4 33	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of	56 4 4 37
· · · ·	• .						Anumony Arsenic Barium Benzene Beryllium	33 4 <1 <1 <1	vegetables	
	•						Cadmium Carbon tetrachloride Chromium Chloroform	7 2 5 < 1		
							cis-1,2-Dichloroethene Cobalt Iron Manganese	< 1 < 1 21 2		
	·						Molybdenum Nickel trans-1,2- Dichloroethene	1 <1 <1		
					· .		Trichloroethene Silver Uranium Vanadium	< 1 < 1 < 1 14		
Future adult rural resident at current concentrations (soil only)	<1E-6	_	-	_ ·		HI < 1	-	-	-	-
Future adult rural resident at current concentrations (RGA groundwater only)	1.5E-4	Trichloroethene	100	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	22 4 50 23	22	1,2-Dichloroethene Trichloroethene	82 18	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of	11 < 1 36 52
									vegetables	

Table ES.7. (Continued)

Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total HI	COCs	% Total Hi	POCs	% Total HI
Future adult rural resident at current concentrations (McNairy groundwater only)	4.0E-3	1,1,2-Trichloroethane 1,1-Dichloroethane 1,2-Dichloroethane Arsenic Benzene Beryllium Bromodichloromethane Carbon tetrachloride Chloroform Polychlorinated biphenyl Radon-222 Tetrachloroethene Trichloroethene Vinyl chloride	<pre><1 <1</pre>	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	41 4 16 39	38.5	Aluminum Antimony Arsenic Barium Benzene Beryllium Cadmium Carbon Tetrachloride Chromium Iron Manganese Molybdenum Nickel Silver Vanadium	4 33 4 < 1 < 1 < 1 8 1 5 21 3 1 < 1 1 1 4	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	62 5 2 31
Future child recreational user at current concentrations (soil only)	NA	NA	NA	NA	NA	H1 < 1		-		-
Future teen recreational user at current concentrations (soil only)	NA	NA	NA	NA	NA	HI < 1	-	-	-	-
Future adult recreational user at current concentrations (soil only)	< IE-6	-	-	_	_	HI < I	_	-	-	-
Future excavation worker at current concentrations (soil only).	1.7E-4	Beryllium	100	Ingestion Dermal contact	2 98	2.09	Chromium Iron Manganese Vanadium	28 31 17 14	Ingestion Dermal contact	12 88

Notes: NA = ECLR not applicable to child and teen cohorts. Values for adult include exposure as child and teen. none = ELCR or HI is above the benchmark, but no COCs or POCs fulfill the selection criteria.

ND = No Data (no samples were taken from the medium under consideration).

- = There are no COCs or POCs.



Table ES.8. Summary of human health risk characterization for SWMU 194 without lead as a COPC

· · · · · ·	· · · ·											
Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total HI	COCs	% Total HI	POCs	% Total HI		
Current industrial worker at current concentrations (soil only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND		
Future industrial worker at current concentrations (soil only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND		
Future industrial worker at current concentrations (RGA groundwater only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND		
Future industrial worker at current concentrations (McNairy groundwater only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND		
Future child rural resident at current concentrations (soil only)	NA/ND	NA/ND	NA/ND	NA/ND	NA/ND	ND	ND	ND	ND	ND		
Future child rural resident at current concentrations (RGA groundwater only)	NA/ND	NA/ND	NA/ND	NA/ND	NA/ND	ND	ND	ND	ND	ND		
Future child rural resident at current concentrations (McNairy groundwater only)	NA/ND	NA/ND	NA/ND	NA/ND	NA/ND	ND	ND	ND	ND	ND		
Future adult rural resident at current concentrations (soil only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND		
Future adult rural resident at current concentrations (RGA groundwater only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	. ND		
Future adult rural resident at current concentrations (McNairy groundwater only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND		

Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total Hi	COCs	% Total HI	POCs	% Total HI
Future child recreational user at current concentrations (soil only)	NA/ND	NA/ND	NA/ND	NA/ND	NA/ND	ND	ND	ND	ND	ND
Future teen recreational user at current concentrations (soil only)	NA/ND	NA/ND	NA/ND	NA/ND	NA/ND	ND	ND	ND	ND	ND
Future adult recreational user at current concentrations (soil only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Future excavation worker at current concentrations	3.1E-4	Beryllium	100%	Ingestion Dermal contact	2 98	HI < 1		- - -		-

Table ES.8. (Continued)

Notes: NA = ECLR not applicable to child and teen cohorts. Values for adult include exposure as child and teen. none = ELCR or HI is above the benchmark, but no COCs or POCs fulfill the selection riteria.

ND = No Data (no samples were taken from the medium under consideration).

- = There are no COCs or POCs.





Table ES.9. Summary of human health risk characterization for AOC 204 without lead as a COPC

Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total HI	COCs	% Total HI	POCs	% Totai HI
Current industrial worker at current concentrations (soil only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Future industrial worker at current concentrations (soil only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Future industrial worker at current concentrations (RGA groundwater only)	1.3E-3	1,1-Dichloroethene PCB-1254 PCB-1260 Polychlorinated biphenyls Tetrachloroethene Trichloroethene	13 6 14 43 21 3	Ingestion Dermal contact Inhalation of vapors/particles	40 53 8	33.3	1,2-Dichloroethane PCB-1254 Tetrachloroethene Trichloroethene	2 88 4 5	Ingestion Dermal contact Inhalation of vapors/particles	43 55 2
Future industrial worker at current concentrations (McNairy groundwater only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Future child rural resident at current concentrations (soil only)	NA/ND	NA/ND	NA/ND	NA/ND	NA/ND	ND	ND	ND	ND	ND
Future child rural resident at current concentrations (RGA groundwater only)	NA	NA	NA	NA	NA	279	1,1-Dichloroethane 1,1-Dichloroethene cis-1,2- Dichloroethene PCB-1254 Tetrachloroethene Trichloroethene	9 1 < 1 66 5 19	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	35 18 21 27
Future child rural resident at current concentrations (McNairy groundwater only)	NA/ND	NA/ND	NA/ND	NA/ND	NA/ND	ND	ND .	ND	ND	ND
Future adult rural resident at current concentrations (soil)	ND	ND	ND	ND	ND -	ND	ND	ND	ND	ND

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Table ES.9. (Continued)

Receptor	Total ELCR	COCs	% Total ELCR	POCs	% Total ELCR	Total HI	COCs	% Total HI	POCs	% Total HI
Future adult rural resident at current concentrations (RGA groundwater only)	> 1E-2*	1,1-Dichloroethene PCB-1254 PCB-1260 Polychlorinated biphenyls Tetrachloroethene Trichloroethene Vinyl chloride	33 6 7 38 11 3 <1	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	19 12 28 41	102	1,1-Dichloroethane 1,1-Dichloroethene PCB-1254 Tetrachloroethene Trichloroethene	6 I 74 5 13	Ingestion Dermal contact Inhalation of vapors/particles Ingestion of vegetables	39 25 11 24
Future adult rural resident at current concentrations (McNairy groundwater only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Future child recreational user at current concentrations (soil only)	NA/ND	NA/ND	NA/ND	NA/ND	NA/ND	ND	ND	ND	ND	ND
Future teen recreational user at current concentrations (soil only)	NA/ND	NA/ND	NA/ND	NA/ND	NA/ND	ND	ND	ND	ND	ND
Future adult recreational user at current concentrations (soil only)	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
Future excavation worker at current concentrations	1.1E-6	none	· –	none	- .	HI < 1	-	- .	-	-

Notes: NA = ECLR not applicable to child and teen cohorts. Values for adult include exposure as child and teen. none = ELCR or HI is above the benchmark, but no COCs or POCs fulfill the selection criteria.

ND = No Data (no samples were taken from the medium under consideration).

- = There are no COCs or POCs.

* = The ELCR is approximate because the linearized multistage model returns imprecise values at risks > 1E-2.

For the BERA, the conceptual model defined in the approved WAG 28 work plan (DOE 1998a) -defined the potential sources of contamination in SWMU 194 and AOC 204 as being contained within subsurface soil (i.e., drainfields and buried debris pile). Consequently surface soils are not impacted and do not require an ecological evaluation to be performed.

Lack of quality habitat in the industrial setting of WAG 28 sites within the fence boundaries limits exposure of ecological receptors at most sites under current conditions (with the exception of SWMU 193a). However, an assessment of potential risks in the future, assuming conditions change so that suitable habitat becomes available for ecological receptors, was conducted. Several contaminants in surface soils were found to be at concentrations greater than levels that are protective of future nonhuman receptors.

BHHRA—Specific

As a measure of the threat of systemic toxicological effects arising through contact with contaminated media at WAG 28, values for HIs were, for the most part, greater than 1000 when lead was retained as a chemical of potential concern (COPC). Such high values are related to the use of a provisional reference dose (RfD) provided by KDEP, an approach that may overemphasize the potential threat of this contaminant. Accordingly, in this assessment, HIs for all receptor/land use combinations were routinely calculated with lead both included and excluded from the determinations, thereby permitting an evaluation of the overall threat to human health of other contaminants at WAG 28 for sites where lead is present.

In an effort to reduce the uncertainty surrounding the assessment of systemic toxicity at sites at WAG 28 where lead is present, two further analytical approaches are included in this risk assessment. Risks to exposed children were estimated using EPA's Integrated Exposure Uptake Biokinetic (IEUBK) model, and the reasonable maximum exposure (RME) concentrations of lead in soil and groundwater samples were compared to KDEP and EPA screening values.

Applying the biokinetic model for lead indicates that the concentration of the element in McNairy Formation groundwater at SWMU 193c (250 μ g/L) and in RGA groundwater at SWMU 99a (81.3 μ g/L) results in a greater than 5 percent probability of a child having blood lead levels greater than 10 μ g/dL (84 percent probability for SWMU 193c McNairy and 38 percent for SWMU 99a RGA). These findings are consistent with the respective lead-driven HIs of 278,000 and 90,600, as calculated for an on-site resident child exposed to contaminants in these aquifers.

The RME lead concentrations in SWMU 193c McNairy and SWMU 99a RGA are also greater than the KDEP and EPA screening level concentrations for this element (4 and 15 μ g/L, respectively); therefore, when these findings are considered together, there is qualitative agreement on the potential hazards of prevailing lead concentrations in the groundwater at these sites.

Where the element was detected in surface or subsurface soil, lead-driven HIs of greater than 1000 contrast with very low probabilities (< 0.02 percent) of children having blood levels greater than 10 μ g/dL, as determined by the IEUBK model. Furthermore, lead concentrations in subsurface soil at SWMUs 99a, 193c, and 194 do not exceed the soil screening values specified by either agency; however, as illustrated in Exhibit 1.39, the concentration of lead in surface soil at SWMU 193c exceeds the KDEP benchmark but not that of EPA (20 < 24.9 < 400 mg/kg).

Because the risks calculated using the provisional lead RfD are so uncertain, all observations presented in Tables ES.3-ES.9 exclude the quantitative contribution from lead.

Exposure Routes

When the major contributions of each applicable exposure route to the overall risk or hazard posed by all sites in WAG 28 are considered, dermal contact appears to contribute the largest proportion of the threat from contact with soil. This observation holds good irrespective of toxicological endpoint (i.e., systemic toxicity or carcinogenicity) and is also irrespective of the applicable land use scenario; however, a significant portion of the threat to the future on-site rural resident from soil constituents comes also from ingestion of vegetables grown in contaminated soil.

By contrast, a plurality of the hazard or cancer risk arising from contact with contaminants in groundwater comes from ingestion, again irrespective of toxicological endpoint; however, inhalation of vapors (while showering and/or during household use) and ingestion of vegetables irrigated with contaminated groundwater make significant contributions to the overall hazard or risk to the future on-site rural resident.

Contaminants

A measure of the relative importance of different media contaminants to the overall hazard and risk that arises from contact with soil and groundwater may be obtained by ranking by occurrence the priority COCs across WAG 28 as a whole. When all sites are considered, the priority COCs contributing 10 percent or more to the total HI or ELCR at one or more of the sites can be ranked according to the number of sites at which the contaminant is a priority COC, as follows:

Soil samples—

Beryllium	(6/7)	Iron	(1/7)
Chromium	(4/7)	Technetium-99	(1/7)
Polycyclic Aromatic Hydrocarbons	(2/7)	Neptunium-234	(1/7)
Polychlorinated Biphenyls (PCBs)	(2/7)	Barium	(1/7)
Vanadium	(2/7)	1,1-Dichloroethene	(1/7)
Manganese	(2/7)	Tetrachloroethene	(1/7)
Antimony	(1/7)	Trichloroethene	(1/7)

Groundwater samples—

Trichloroethene	(6/6)	PCBs	(1/6)
cis-1,2-Dichloroethene	(3/6)	Tetrachloroethene	(1/6)
1,1-Dichloroethene	(3/6)	PCB-1254	(1/6)
Radon-222	(3/6)	Antimony	(1/6)
Iron	(3/6)	Cadmium	(1/6)
Technetium-99	(2/6)	Chromium	(1/6)
Beryllium	(2/6)	Pentachlorophenol	(1/6)
Vanadium	(2/6)	Arsenic	(1/6)
1,2-Dichloroethene	(1/6)	Uranium-238	(1/6)
Vinyl chloride	(1/6)		

Scenario Hazards/Risks

The overall extent of the threats of systemic toxicity or induction of carcinogenicity presented by sites in WAG 28 is indicated by the following ranges of SWMU-specific HIs and ELCRs that were

calculated for each primary on-site receptor. Also listed are the number of sites for each receptor/land use ---- combination in which the overall ELCR is greater than 1E-4 or where the HI is greater than 1.

HI

· · · · · · · · · · · · · · · · · · ·			
Receptor	Medium	Range of values	Sites > 1
Future on-site rural resident	Groundwater	278,000 - 2.69	10/10
	Soil	247,000 - 6.25	4/4
Future industrial worker	Groundwater	25,100 - < 0.1	9/10
	Soil	3620 - 0.432	2/4
Future excavation worker	Subsurface soil	250 - < 0.1	4/7
ELCR			

Receptor	Medium	Range of values	Sites > 1E-4
Future on-site rural resident	Groundwater	> 1E-2 [•] - 4.1E-4	10/10
	Soil	> 1E-2 [•] - 1.1E-9	3/4
Future industrial worker	Groundwater	1.3E-3 – 8.4E-7	9/10
	Soil	5.1E-4 – 1.7E-10	2/4
Future excavation worker	Subsurface soil	3.1E-4 - 1.1E-6	6/7

"(The ELCR is approximate because the linearized multistage model returns imprecise values at risks > 1E-2.)

These data clearly demonstrate that for both the default "worst-case" scenario (future on-site rural resident) and the most likely future receptor (industrial worker), most sites had HIs and ELCRs greater than the EPA's range of concern.

When considered in detail, the most plausible future use scenario, future industrial worker, has total HIs and ELCRs exceeding de minimis levels at all sites except SWMU 194, for which this scenario/land use combination did not apply. As discussed in the BHHRA, the future industrial land use scenario is identical to the current industrial land use scenario except that the future industrial land use scenario also evaluates use of RGA and McNairy groundwater. Addition of groundwater as a medium of exposure adds significantly to the risk for this scenario. If groundwater contribution is removed from the risk totals, the primary pathways are identical to the current industrial use scenario.

The driving contaminants contributing to more than 10 percent of total HIs for the future industrial worker at SWMU 99a (excluding lead) in RGA groundwater are trichloroethene, chromium, iron, and vanadium, with ingestion as the primary pathway. The driving contaminant contributing to more than 10 percent of total HIs at SWMU 99b is trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at SWMU 199b is trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at SWMU 193b is trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at SWMU 193b is trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at SWMU 193b is trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at SWMU 193c are 1,2-dichloroethene and trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at SWMU 193c are 1,2-dichloroethene and trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at SWMU 193c are 1,2-dichloroethene and trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at AOC 204 are PCBs with dermal contact and ingestion as the primary pathways.

The driving contaminants contributing to more than 10 percent of total ELCR for the future industrial worker exposed to RGA groundwater at SWMU 99a are 1,1-dichloroethene, beryllium, and radon-222, with incidental ingestion and inhalation of vapors and particulates as the primary pathways. The driving contaminants contributing to more than 10 percent of total ELCR at SWMU 99b are trichloroethene and radon-222, with inhalation as the primary pathway. The driving contaminants contributing to more than 10 percent of total ELCR at SWMU 193a are pentachlorophenol and trichloroethene, with ingestion and dermal contact as the primary pathways. The driving contaminants contributing to more than 10 percent of total ELCR at SWMU 193b are trichloroethene and 1,1-dichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total ELCR at SWMU 193b are trichloroethene and 1,1-dichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total ELCR at SWMU 193b are trichloroethene and 1,1-dichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total ELCR at SWMU 193c is trichloroethene, with ingestion as the driving pathway. The driving contaminants contributing to more than 10 percent of total ELCR at AOC 204 are PCBs, trichloroethene, and 1,1-dichloroethene, with dermal contact as the primary pathway.

The COCs for analytes migrating from sources in WAG 28 soil and groundwater as determined by risk estimates for off-site residential groundwater users are chromium, lithium, manganese, strontium, technetium-99, and trichloroethene.

BERA—Specific

The conceptual model defined in the approved WAG 28 work plan (DOE 1998a) defined the potential sources of contamination in SWMU 194 and ACO 204 as being contained within subsurface soil (i.e., drainfields and buried debris pile). Consequently surface soils are not impacted and do not require an ecological evaluation to be performed.

Lack of quality habitat in the industrial setting of WAG 28 sites within the fence boundaries limits exposure of ecological receptors at most sites under current conditions (with the exception of SWMU 193a); however, an assessment of potential risks in the future, assuming conditions change so that suitable habitat becomes available for ecological receptors, was conducted. Several contaminants in surface soil were found to be at concentrations greater than levels protective of future nonhuman receptors.

Chemical and radionuclide contaminants were evaluated for surface soil from SWMUs 99a, 193a, 193b, and 193c. Detectable concentrations that exceeded background were evaluated for the potential of inducing adverse ecological effects to a representative set of receptor species that potentially could inhabit the WAG 28 area. Table ES.10 summarizes chemicals of potential ecological concern (COPECs) that were identified based on the results of screening contaminant concentrations against ecological benchmarks. Risks for ecological receptors were not evaluated at SWMUs 99b, 194, or ACO 204 because it was previously determined that surface soil was not a medium of concern at these sites.

Six nonradionuclide COPECs, all inorganics, exceed background and benchmarks for at least one receptor group (Table ES.10). The inorganics are boron, barium, chromium, lead, vanadium, and zinc; however, chromium and lead are near background levels (maximum of 1.05 and 1.53× background, respectively). Confidence in the benchmarks for boron and chromium is low. Potential risks from chromium are largely based on chromium being present as the more toxic Cr(VI) rather than the more likely Cr(III); however, chromium exceeds benchmarks for plants and soil invertebrates at all four sites with the highest concentrations occurring at SWMUs 99a and 193b. Barium is only a potential concern for plants at SWMU 99a, and the concern is driven by a maximum detected concentration more than an order of magnitude higher than other detects in that SWMU. Lead is only a concern for plants in SWMU 193c, but the lead concentration is near background levels. Zinc is a potential concern for plants at SWMU 193c and plants and soil invertebrates at SWMU 99a but, as with lead, concentrations are near background levels. Vanadium is a potential concern for plants and wildlife at SWMU 193b. The potential for adverse effects to ecological receptors exposed to chemicals in surface soil from WAG 28 sites is low.

Table ES.10. Summary of chemicals with maximum detected or reasonable maximum exposure concentrations resulting in ecological hazard quotients greater than 1 for one or more nonhuman receptor groups

	*	SWM	U ^a	
Receptor group	99a	193a	193b	193c
Plants ^b	barium, chromium,	chromium	chromium,	boron, chromium,
	zinc, technetium-99 ^c		vanadium	lead, zinc
Soil invertebrates ^b	chromium, zinc,	chromium	chromium	chromium
	technetium-99 ^c			
Terrestrial wildlife ^d	none	none	vanadium	none

Surface soil was not a medium of concern at SWMUs 99b, 194, or AOC 204; therefore, ecological risks were not evaluated at these sites.

^b Plant and soil invertebrate results are based on maximum detected concentrations or activities.

^c See text for discussion of situation resulting in unusually high activity for technetium-99.

^d Terrestrial wildlife results are based on reasonable maximum exposure concentrations or activities.

Estimated doses from exposure to radionuclides in soil are below recommended dose rate limits for all receptors in all sites except for plants and soil invertebrates at SWMU 99a, in which technetium-99 is the radionuclide of concern.

The following paragraphs and Table ES.10 summarize analytes of potential concern and receptors potentially at risk should future exposures occur.

SWMU 99a. While chromium and zinc exceed benchmarks for plants and soil invertebrates and barium exceeds benchmarks for plants, potential risks to plant and soil invertebrate communities from future exposure to surface soil at this site appear low. The barium risk is due to a location (station 099-014) where the concentration is more than an order of magnitude higher than at other stations. Zinc is near background levels and results in low exceedances of benchmarks. There is considerable uncertainty in the benchmarks for chromium, which is based on the more toxic Cr(VI) rather than the more likely Cr(III).

Estimated doses from exposure to radionuclides in soil are below recommended dose rate limits for wildlife, but dose rates for plants and soil invertebrates are higher than the recommended dose rate limit of 1 rad/day. Technetium-99 is the radionuclide of concern based on its occurrence in a single sample.

SWMU 193a. Risks to terrestrial receptors are not expected from current or future exposures at this site. No radionuclides were detected, and only chromium, for which toxicological benchmarks are likely highly conservative, exceeds levels of potential concern for plants and soil invertebrates.

SWMU 193b. Potential future risks from exposure of plants, soil invertebrates, and wildlife to chromium or vanadium were identified. While there is considerable uncertainty associated with the benchmarks available for chromium, concentrations of both chromium and vanadium are elevated relative to other areas in WAG 28, indicating a greater potential to cause adverse effects.

SWMU 193c. Potential future risks from exposure of plants to boron, chromium, lead, and zinc and exposure of soil invertebrates to chromium were identified, but there is considerable uncertainty associated with the benchmarks available for boron and chromium. Lead and zinc are near background levels, and chromium concentrations are lower at this site than in other areas in WAG 28. Lower chromium concentrations relative to other areas in WAG 28 do not necessarily equate with no risk, but potential risks from chromium are lower.



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The purpose of this assessment was to evaluate the likelihood that adverse ecological effects may -occur or are occurring as a result of exposures at WAG 28. For sites within the fence boundaries, under current conditions, complete exposure pathways are not expected for terrestrial biota, except at SWMU 193a, and even this area is within the industrialized portion of the plant. Thus, this evaluation focused on hypothetical future exposures assuming loss of industrial controls and buildings and development of a larger area of suitable habitat. Analytes that were retained as COPECs may require further study to determine whether adverse ecological effects are likely if decisions for remedial actions will be based on ecological concerns. Uncertainty concerning the future condition, the bioavailability or form of various metals (e.g., boron, ba rium, chromium, lead, vanadium, and zinc), and use of only one line of evidence (comparison of exposures to single chemical toxicity values) may have lead to an overestimate of potential future ecological risks.

1. BASELINE HUMAN HEALTH RISK ASSESSMENT

This baseline human health risk assessment (BHHRA) utilizes information collected during the recently completed remedial investigation (RI) of Waste Area Grouping (WAG) 28 to characterize the baseline risks posed to human health from contact with contaminants in soil and water at Solid Waste Management Units (SWMUs) 99, 193, 194, and Area of Concern (AOC) 204 in WAG 28 at the Paducah Gaseous Diffusion Plant (PGDP) in Paducah, Kentucky. SWMUs 99 and 193 were further subdivided into units based upon area and historical use (99a, 99b, 193a, 193b, and 193c.) The overall purpose of the RI was to determine the presence, nature, and extent of contamination at each of the SWMUs and AOC 204. The primary focus of the RI was to collect sufficient information about surface soil, subsurface soil, and groundwater of the Upper Continental Recharge System (UCRS) contamination to support an assessment of risks to human health and the environment and the selection of actions to reduce those risks. In addition, contamination in the Regional Gravel Aquifer (RGA) and McNairy Formation groundwater was characterized to determine if contamination in the RGA acted as a secondary source of contamination to groundwater.

The methods and presentations used in this BHHRA are consistent with those presented in the U.S. Department of Energy's (DOE) *Methods for Conducting Human Health Risk Assessments and Risk Evaluations at the Paducah Gaseous Diffusion Plant* (DOE 1996a), as modified by regulatory comments, which is henceforth referred to as the "Methods Document." This document integrates human health risk assessment guidance from the U.S. Environmental Protection Agency (EPA) and the Kentucky Department of Environmental Protection (KDEP) and incorporates instructions contained in regulatory agency comments on earlier risk assessments performed at PGDP. The Methods Document received final approval from the Commonwealth of Kentucky for use in environmental investigations and restoration activities at PGDP in February 1998 (KDEP 1998).

Consistent with the Methods Document, this BHHRA is presented in eight sections. Section 1.1 reviews the results of previous risk studies that are useful in understanding the risks posed to human health from contaminants at, or migrating from, the WAG 28 area. This section also presents sources of information that were used to complete the exposure assessment contained in the BHHRA. Section 1.2 describes the evaluation of data collected during the WAG 28 field investigation and under other programs and identifies chemicals of potential concern (COPCs) for WAG 28. Section 1.3 documents the exposure assessment for WAG 28, including the characterization of the exposure setting, identification of exposure pathways, consideration of land use, determination of potential receptors, delineation of exposure points and routes (including development of the conceptual site model), and calculation of chronic daily intakes (CDIs). Section 1.4 presents the toxicity assessment, including information on the noncarcinogenic and carcinogenic effects of the COPCs and the uncertainties regarding toxicity information. Section 1.5 reports the results of the risk characterization for current and various future land uses and identifies contaminants, pathways, and land use scenarios of concern. Section 1.6 contains qualitative and quantitative analyses of the uncertainties affecting the results of the BHHRA. Section 1.7 summarizes the methods used in the BHHRA and presents conclusions and observations. Section 1.8 uses the results of the BHHRA to develop site-specific, risk-based remedial goal options (RGOs). Chapter 3 contains references.

Because of their length, all tables cited within the BHHRA are presented in Appendix A of this volume (Vol. 4). The BHHRA also includes exhibits within the text that summarize much of the material presented in the tables. All figures cited are presented in the text.

1.1 RESULTS OF PREVIOUS STUDIES

The following three reports contain risk assessment results that are useful in understanding the risks to human health posed by exposure to contaminants present at, or migrating from, the WAG 28 area:

- Results of the Site Investigation, Phase I, at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky (CH2M Hill 1991a)
- Results of the Public Health and Ecological Assessment, Phase II, at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky (CH2M Hill 1991b), Vol. 6 in Results of the Site Investigation, Phase II, Paducah Gaseous Diffusion Plant, Paducah, Kentucky (CH2M Hill 1992)
- Comparison of Trichloroethene (TCE) Concentrations at Area of Concern (AOC) 204 to Human Health Risk-Based Concentrations, Paducah Gaseous Diffusion Plant, Paducah, Kentucky (ORNL 1996)

In addition, the following studies containing historical data pertaining to WAG 28 were used in the preparation of this BHHRA. These studies are not summarized in detail in this report:

- Results of the Site Investigation, Phase II, Paducah Gaseous Diffusion Plant, Paducah, Kentucky (CH2M Hill 1992)
- Report of the Paducah Gaseous Diffusion Plant Groundwater Investigation, Phase III, Paducah, Kentucky (Clausen et al. 1992)
- Groundwater Phase IV Investigation as presented in the Northeast Plume Preliminary Characterization Summary Report, Paducah Gaseous Diffusion Plant, Paducah, Kentucky, Vols. 1 and 2 (DOE 1995a)
- Sampling and Analysis Plan for a Site Evaluation at the Outfall 011 and 012 Areas, Paducah Gaseous Diffusion Plant, Paducah, Kentucky (Carter et al. 1995)
- Final Site Evaluation Report for the Outfall 010, 011 and 012 Areas, Paducah Gaseous Diffusion Plant, Paducah, Kentucky (DOE 1995b)

Finally, the following studies containing information regarding the environmental conditions around WAG 28 were used in the preparation of this BHHRA. These studies were primarily used to complete the exposure assessment step of the BHHRA and are not summarized in detail here:

- Integrated Remedial Investigation/Feasibility Study Work Plan for Waste Area Grouping 27 at Paducah Gaseous Diffusion Plant, Paducah, Kentucky (DOE 1998b)
- Remedial Investigation Report for Waste Area Grouping 27 at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky, Vol. 4 of 4, Baseline Risk Assessment (DOE 1999)
- Environmental Investigations at the Paducah Gaseous Diffusion Plant and Surrounding Area, McCracken County, Kentucky (COE 1994a)
- Work Plan for Waste Area Grouping 28 Remedial Investigation/Feasibility Study and Waste Area Grouping 8 Preliminary Assessment/Site Investigation at the Portsmouth Gaseous Diffusion Plant, Paducah, Kentucky (DOE 1998a)

1.1.1 Results of the Site Investigation, Phase I, at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky (CH2M Hill 1991a)

The Phase I investigation evaluated the nature and extent of off-site contamination originating at PGDP and determined risk presented by this contamination to off-site receptors. In the investigation, risks from chemicals and radionuclides found off site were characterized using methods described in EPA's Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual (Part A, Baseline Risk Assessment) (RAGS) (EPA 1989a). Although this guidance document is primary among those used in preparation of the Methods Document, the methods used in the Phase I assessment are not consistent with those in the Methods Document. The primary reason for this is that the methods used in the assessment in the Phase I investigation do not incorporate guidance developed later by the regulatory community [e.g., Supplemental Guidance to RAGS: Region 4 Bulletins, Human Health Risk Assessment, Interim (EPA 1995a) and Risk Assessment Guidance (KDEP 1995)].

The results of the risk assessment of groundwater usage are discussed in Sect. 6.5, "Risk Characterization," of the Phase I report and tabulated in Appendix 6C and 6D of that report. Risk results of the Phase I report are summarized in Exhibits 1.1 and 1.2.

Well category and exposure	Excess lifetime cancer risk			Hazard index		
assumptions ^a	Ingestion	Inhalation ^b	Total	Ingestion	Inhalation ^b	Total
Average exposure assumptions						
Residential	2E-5	2E-5	4E-5	0.6	0.3	0.9
Monitoring	1E-5	6E-6	2E-5	1.1	< 0.1	1.1
TVA	5E-5	7E-7	6E-5	0.5	< 0.1	0.5
Maximum exposure assumptions						
Residential	3E-4	4E-4	7E-4	2.0	0.7	[~] 2.7
Monitoring	1E-4	9E-5	2E-4	3.8	0.1	3.9
TVA	7E-4	2E-5	7E-4	1.7	< 0.1	1.7

Exhibit 1.1. Excess lifetime cancer risk and hazard indices from chemicals in groundwater—residential use scenario (CH2M Hill 1991a)

^a See Chap. 4 in CH2M Hill (1991a) for a description of well categories. The residential well category may include wells not completed in the RGA. See Table 6-29 and the discussion in Sect. 6.4.5.1 in CH2M Hill (1991a) for descriptions of exposure assumptions and dose calculations.

^b The dose from inhalation was estimated using dose from ingestion. See Sect. 6.4.5.1 in CH2M Hill (1991a).

Exhibit 1.2.	Excess total cancer incidence from radionuclides in groundwater-
	residential use scenario (CH2M Hill 1991a)

Well category ^a	Average exposure assumptions ^b	Maximum exposure assumptions
Residential	4E-6	5E-5
Monitoring	3E-6	5E-5
TVA	1E-5	3E-4

^a See Chap. 4 in CH2M Hill (1991a) for a description of well categories. The residential well category may include wells not completed in the RGA.

^b See Table 6-51 and the discussion in Sect. 6.5.2.2 in CH2M Hill (1991a) for descriptions of exposure assumptions and dose calculations.

As shown in Exhibit 1.1, total excess lifetime cancer risk (ELCR) from residential use of off-site groundwater exceeds the de minimis level defined in the Methods Document (i.e., 1E-6) for all well

categories under average and maximum exposure assumptions. Hazard indices (HIs) for all wellcategories exceed the de minimis level defined in the Methods Document (i.e., 1) for all well categories under maximum exposure assumptions, but only for the monitoring well category under average exposure assumptions.

The contaminants in groundwater contributing most significantly to ELCRs and HIs are relatively consistent among well categories [i.e., residential, monitoring, and Tennessee Valley Authority (TVA) wells]. For ELCR, the primary contaminants for all well categories are trichloroethene, arsenic, and bis(2-ethylhexyl)phthalate. For HIs, the primary contaminants for all well categories are various metals, carbon tetrachloride, and bis(2-ethylhexyl)phthalate.

As shown in Exhibit 1.2, total cancer incidence from ingestion of radionuclides in groundwater during residential use exceeds de minimis levels for all well categories under both average and upper bound exposure assumptions. The primary contaminants in groundwater for all well categories are uranium-234, uranium-238, and technetium-99.

Uncertainties in the Phase I risk assessment and the effects of uncertainty on the risk characterization are presented in Table 6-66 of the Phase I report. Most uncertainties discussed are common to all risk assessments (e.g., uncertainties related to cancer potency factors, toxicity values, effect of absorption, magnitude of exposure factors, and assumption of additive effects); however, the following four specific uncertainties make the results of the Phase I assessment differ significantly from those presented later in this BHHRA.

- The exposure assessment in the Phase I assessment did not consider all possible pathways and routes of exposure. For example, dermal contact with groundwater was not assessed quantitatively. Although this pathway often contributes little to cumulative risk in most risk assessments, its absence reduced the estimate of cumulative risk. Similarly, the exposure assessment did not consider ingestion of foods raised using contaminated groundwater.
- Current concentrations were used to determine potential future risk. Because the source of contamination was not determined before the Phase I assessment was performed, it was not possible to determine how much contaminant concentrations may increase or decrease in the future; therefore, there is considerable uncertainty in the estimate of future risk.
- Because measured concentrations were used to develop the representative concentrations of contaminants in groundwater, all sources of contamination at PGDP were integrated in the risk estimates (i.e., the results are not specific to contamination originating at WAG 28).
- The groundwater sampling methods used during the Phase I investigation did not incorporate the use of low-flow technologies. Groundwater was collected during the WAG 28 RI using low-flow technologies that greatly reduced the turbidity of the samples collected. With low turbidity, the metals concentrations are markedly lower.

1.1.2 Results of the Public Health and Ecological Assessment, Phase II, at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky, Vol. 6 (CH2M Hill 1991b) in Results of the Site Investigation, Phase II, Paducah Gaseous Diffusion Plant (CH2M Hill 1992)

The Phase II investigation, Public Health and Ecological Assessment (PHEA) (CH2M Hill 1991b) further evaluated the nature and extent of off-site contamination originating from PGDP and characterized on-site units by identifying contaminant migration routes that may contribute to off-site contamination. The Phase II investigation used this information to develop a baseline risk assessment (BRA) as part of

1-5

the PHEA. As with the other assessments that contain information relevant to the WAG 28 investigation, risk from chemicals and radionuclides were characterized using methods described in EPA's *Risk* Assessment Guidance for Superrfund (RAGS); however, as with the assessment reviewed in Sect. 1.1.1, the methods used are not consistent with those in the Methods Document. Again, the methods used in this assessment do not incorporate guidance developed later by the regulatory community [e.g., Supplemental Guidance to RAGS: Region 4 Bulletins, Human Health Risk Assessment, Interim (EPA 1995a) and Risk Assessment Guidance (KDEP 1995)].

The results of the risk assessment of off-site groundwater usage are discussed in Sect. 3.3, "Risk Characterization," of the Phase II report and tabulated in Appendix H of that report. Risk results of the Phase II report are summarized in Exhibits 1.3–1.5.

	Excess lifetime cancer risk			Hazard index		
Weli category ^a	Ingestion	Inhalation ^b	Total	Ingestion	Inhaiation ^b	Total
Maximum exposure assumptions ^c						
Residential	4E-4	3E-4	7E-4	2.4	0.9	3.3
Off-site monitoring	4E-4	4E-5	5E-4	2.5	0.08	2.6
TVA	3E-3	3E-7	3E-3	8.8	0.04	8.8

Exhibit 1.3. Excess lifetime cancer risk and hazard indices from chemicals in groundwater—residential use scenario (CH2M Hill 1991b)

^a See Sect. 3.2.3.2 in CH2M Hill (1991b) for a description of well categories. The residential well category may include wells not completed in the RGA.

^b The dose from inhalation was estimated using dose from ingestion. See Sect. 3.2.3.3 in CH2M Hill (1991b).

^c See Table 3-10 and the discussion in Sect. 3.2.3 in CH2M Hill (1991b) for descriptions of exposure assumptions and dose calculations.

	Maximum exposure
Well category ^a	assumptions ^b
Residential	2E-5
Off-site monitoring	2E-5
TVA	6E-5

Exhibit 1.4. Excess total cancer incidence from radionuclides in groundwater—residential use scenario (CH2M Hill 1991b)

^a See Sect. 3.2.3.2 in CH2M Hill (1991b) for a description of well categories. The residential well category may include wells not completed in the RGA.

^b See Table 3-10 and the discussion in Sect. 3.2.3 in CH2M Hill (1991b) for descriptions of exposure assumptions and dose calculations.

Exhibit 1.5.	Excess lifetime cancer risk and hazard indices from chemicals and radionuclides	
	in off-site soil—residential use ⁴ (CH2M Hill 1991b)	

Maximum exposure	Excess lifeth	ne cancer risks	Hazard index		
assumptions ^b	Chemical	Radiological	Adult	Child	
Ingestion	3E-5	5E-7	0,04	0.39	
Dermal absorption	1 E-4	NC	0.03	NC	
Sum of risks	2E-4	5E-7	0.07	NC	

^a Not SWMU-specific information.

^b See Table 3-10 and the discussion in Sect. 3.2.3 in CH2M Hill (1991b) for descriptions of exposure assumptions and dose calculations.

^c Not calculated.

As shown in Exhibit 1.3, total ELCRs from residential use of groundwater exceed the de minimislevel for all well categories under maximum exposure assumptions. HIs for all well categories exceed the de minimis level for all well categories under maximum exposure assumptions.

The contaminants in groundwater contributing most significantly to ELCRs and HIs are relatively consistent among well categories. For ELCR, the primary contaminants for all well categories are trichloroethene, arsenic, and beryllium. For HIs, the primary contaminants for all well categories are various metals and carbon tetrachloride.

As shown in Exhibit 1.4, total cancer incidence from ingestion of radionuclides in groundwater during residential use exceeds de minimis levels for all well categories under maximum exposure conditions. The primary contaminants in groundwater over all well categories are uranium-234, uranium-238, neptunium-237, plutonium-239 and technetium-99.

Off-site chemical and radiological nisks associated with surface soil are discussed in Sect. 3.3.4, "Risk Characterization," of the Phase II report and tabulated in Appendix H of that report. Risk results are summarized in Exhibit 1.5.

As shown in Exhibit 1.5, total ELCRs associated with surface soil exceed the de minimis level for chemical exposures under maximum exposure assumptions. Total ELCRs for radiological exposures do not exceed de minimis levels. HIs associated with surface soil do not exceed the de minimis level under maximum exposure assumptions.

The primary contributors to carcinogenic risks are various polycyclic aromatic hydrocarbon (PAH) constituents, arsenic, and beryllium. The primary contributors to noncarcinogenic hazards are arsenic and manganese.

Uncertainties in this assessment and the effects of uncertainty on the risk characterization are summarized in Sect. 3.4 and Table 3-33 of the PHEA. The uncertainties important to the final risk estimates in this assessment are similar to those discussed for other assessments.

1.1.3 Comparison of Trichloroethene (TCE) Concentrations at Area of Concern (AOC) 204 to Human Health Risk-Based Concentrations (ORNL 1996)

The purpose of this investigation was to determine if current concentrations of trichloroethene at AOC 204 exceeded human health risk-based concentrations (RBCs) and if future trichloroethene concentrations at their associated compliance points may exceed human health RBCs. The results were to be used as part of the documentation to determine if further action was required for AOC 204. The authors concluded the following:

- For the industrial worker, current exposure to trichloroethene in soil does not exceed the 1E-6 RBC; current exposure to trichloroethene in groundwater exceeds the 1E-4 RBC; future exposure to trichloroethene in groundwater does not exceed the 1E-6 RBC.
- For the industrial surveillance worker, current exposure to trichloroethene in sediment does not exceed the 1E-6 RBC; current exposure to trichloroethene in surface water does not exceed the 1E-4 RBC (use of personal protective equipment would eliminate dermal exposure and reduce risk by an order of magnitude).
- For the recreational visitor, current exposure to trichloroethene in sediment does not exceed the 1E-6 RBC; current exposure to trichloroethene in surface water exceeds the 1E-4 RBC.

1.1.4 Preliminary COPCs Identified in the Work Plan for WAG 28

As part of the production of the WAG 28 work plan (DOE 1998a), maximum detected concentrations of analytes in samples collected during previous investigations were screened against a series of values to develop a preliminary list of COPCs. Sources of screening values are presented in Exhibit 1.6. The preliminary COPCs identified as the result of the screening are presented by unit in Exhibit 1.7. Note that the preliminary COPC list is based on sampling conducted before the recently completed field investigation. Because the earlier sampling was limited for some areas, the list in Exhibit 1.7 differs from the list of COPCs presented later in the BHHRA.

Screening values for soil			
Soli	Source		
Site-specific risk-based concentration residential use	Methods Document (DOE 1996a)		
Commonwealth of Kentucky soil screening value	Risk Assessment Guidance (KDEP 1995)		
Background value	Background Levels of Selected Radionuclides and Metals in Soil and Geologic Media at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky (DOE 1997)		
Water			
Site-specific risk-based concentration residential use	Methods Document (DOE 1996a)		
Commonwealth of Kentucky water screening value	Risk Assessment Guidance (KDEP 1995)		
Background value	Methods Document (DOE 1996a) RGA only		
Maximum contaminant level (MCL)	Chemical-specific Applicable or Relevant and Appropriate Requirements (ARARs): Federal/Kentucky (Energy Systems 1996)		

Exhibit 1.6	Screening va	lues used in the	WAG 28 work	plan to identif	y preliminar	v COPCs

SWMU 99ª (grou	ndwater)
Organics:	trichloroethene
Inorganics:	aluminum, barium, iron, and manganese
Radionuclides:	technetium-99
SWMU 99 ^a (soil)	
Organics:	trichloroethene
Inorganics:	antimony, barium, beryllium, cadmium, chromium, iron, lead, manganese, and
	vanadium
Radionuclides:	technetium-99
SWMU 193ª (gro	undwater)
Organics:	1,2-dichloroethane, cis-1,2-dichloroethene, and trichloroethene
Inorganics:	none
Radionuclides:	none
SWMU 193 ^a (soil)
Organics:	trichloroethene
Inorganics:	cadmium, chromium, and lead
Radionuclides:	none
SWMU 194 (soil (only)
Organics:	none
Inorganics:	cadmium, chromium, and lead
Radionuclides:	none
AOC 204 (ground	iwater)
Organics:	trichloroethene
Inorganics:	not analyzed
Radionuclides:	not analyzed
AOC 204 (soil)	
Organics:	trichloroethene
Inorganics:	not analyzed
Radionuclides:	not analyzed

Exhibit 1.7. Preliminary COPCs identified in the WAG 28 work plan

* SWMUs 99 and 193 were not subdivided for the WAG 28 work plan.

1.2 IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN

This section describes the processes used to determine the COPCs in both the BHHRA and the baseline ecological risk assessment (BERA) (Chap. 2). Specifically, the sources of data, the procedures used to screen the data, and the methods used to derive representative concentrations in environmental media and biota under both current and future conditions are described. Additionally, this section describes the site characterization data used in the exposure assessment performed in Sect. 1.3.

1.2.1 Sources of Data

Data used in the BHHRA and BERA describing current contaminant concentrations in soil and groundwater are from the recently completed field investigation at WAG 28 and previous investigations. These data and the manner in which they were generated are described in Chaps. 4 and 5 of Vol. 1 of this report. The data sets presented in the RI and the risk assessments may differ in minor details due to different assessment methodology (e.g., spatial versus statistical). Data from the Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA) Phases I and II Site Investigations and Phases III and IV Groundwater Investigations (Clausen et al. 1992, DOE 1995a) are used in Chap. 4 of Vol. 1 to describe the nature and extent of contaminants at WAG 28 and in Chap. 5 of Vol. 1 to determine the environmental fate and transport of contaminants at WAG 28. The analytical results of the environmental fate and transport modeling are used in the BHHRA to determine future contaminant concentrations in groundwater at points of exposure to which contaminants may migrate. Additionally, the current contaminant concentration data are used in this BHHRA to model contaminant concentrations in animals and vegetables. The methods and models used to determine contaminant concentrations in biota are described in Sect. 1.3 of the BHHRA.

SWMUs 99, 193, and 194 and AOC 204 were investigated as specific areas of contamination within WAG 28. SWMUs 99 and 193 were further subdivided into units based upon area and historical use (99a, 99b, 193a, 193b, and 193c).

A list of sampling stations (by site) used in this BHHRA and BERA is presented in Table 1.1. Because these sites are not contiguous, neither the BHHRA nor the BERA include evaluations of WAG 28 soil and groundwater data as a whole. Groundwater data from the RGA are evaluated separately. from groundwater data from the underlying McNairy Formation.

1.2.2 General Data Evaluation Considerations

Data were evaluated to ensure appropriateness for use in the BRAs. A general description of this evaluation is provided in this section. A graphical presentation of this evaluation is shown in Fig. 1.1.

Data evaluation was performed in eight steps:

- (1) **Evaluation of sampling**—Data were examined to ensure that sampling methods were adequate for determining the nature and extent of contamination.
- (2) Evaluation of analytical methods—Methods used to analyze samples were evaluated to determine if they were approved by EPA.
- (3) Evaluation of sample quantitation limits (SQLs)—The SQLs for each analyte and sample were examined to determine if these limits were below the concentration or activity at which the analyte may pose a risk or hazard to human health or the environment. If the maximum SQL for an analyte

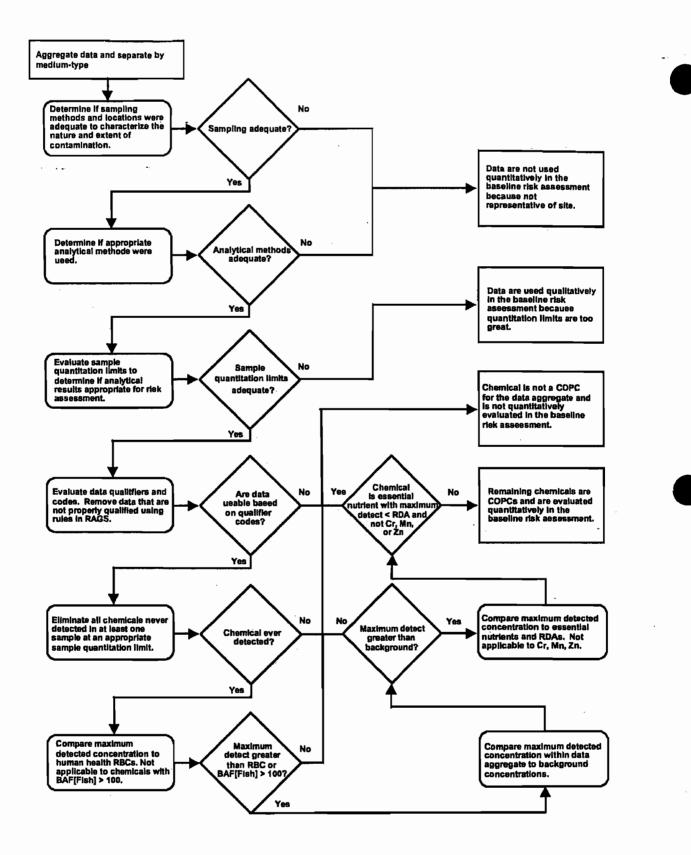


Fig. 1.1. Data evaluation steps

over all samples within a medium was greater than the concentration or activity that may pose a... threat to human health or the environment, and that analyte was not detected in any sample, the data for that analyte were deemed of insufficient quality, and only a qualitative assessment for that analyte is presented in this assessment. The maximum SQL for the chemical was used in developing the qualitative assessment for such analytes if historical or process knowledge indicated that the chemical could potentially be present. If historical or process knowledge indicated that the chemical was not expected to be present, one-half of the SQL was used in the qualitative assessment.

- (4) **Evaluation of data qualifiers and codes**—The data used in the risk assessment were tagged with various qualifiers and codes that are defined in Sect. 2.8.2 of Vol. 1. Tagged data were evaluated following rules in Exhibits 5-4 and 5-5 of RAGS (EPA 1998b). Radionuclides with negative activity values were retained.
- (5) Elimination of chemicals not detected—For each sample, any analyte not detected in at least one sample using an appropriate SQL was eliminated from the data set.
- (6) Examination of toxicity of detected analytes—A comparison of the analyte's maximum detected concentration to that analyte's residential use human health RBC was performed for the data set created for the BHHRA. The human health RBCs used in this comparison are derived according to equations in the Methods Document (see Appendix A, Tables 1.12–1.38 in this volume) using the most recent toxicity values available [http://risk/lsd.ornl.gov/tox/rap_hp.shtml (DOE 1998c)]. To ensure that the human health risk-based screening criteria used in this step were conservative, the exposure routes used to develop the criteria for chemicals were (1) ingestion of potentially contaminated media, (2) dermal contact with potentially contaminated media, and (3) inhalation of vapors and particulates emitted by potentially contaminated media. Direct contact exposure routes used to develop screening criteria for radionuclides were (1) ingestion of potentially contaminated media, (2) inhalation of vapors and particulates emitted by potentially contaminated media. Direct contact eminated media, (2) inhalation of vapors and particulates emitted by potentially contaminated media, (3) external exposure to ionizing radiation emitted by potentially contaminated media.

The target ELCRs and target HIs used in calculating the criteria for chemicals were established by regulatory agreement in the Methods Document as 1E-7 and 0.1, respectively. The target cancer risk used in calculating the criteria for radionuclides is 1E-6. In this screen, the lower of the human health risk-based screening criteria calculated for cancer effects from lifetime exposure and for systemic toxicity in children was used. In addition, per regulatory agreement in the Methods Document, this screen was not applied to those analytes known to accumulate significantly in biota [i.e., not used for analytes with a bioaccumulation factor (BAF) for fish greater than 100].

(7) Comparison of maximum analyte concentrations and activities detected in site samples to analyte concentrations and activities detected in background samples—Background concentrations for soil were taken from Background Levels of Selected Radionuclides and Metals in Soils and Geologic Media at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky (DOE 1997) and were compared to the maximum detected analyte concentration over all site samples. The background concentrations for soil are presented in Table 1.2.

Background data sets for RGA and McNairy Formation groundwater were not available for this assessment because these values are undergoing revision; therefore, a comparison between maximum detected concentrations in groundwater and background concentrations was not performed. Since the publication of the D0 version of this document, provisional (i.e., draft) groundwater background values have become available and are presented in Table 1.2 (Bonczek 1999).

(8) Comparison of analyte maximum concentrations that are essential human nutrients to Recommended Dietary Allowances (RDAs) for children—For the data set developed for the BHHRA, the maximum detected concentration of analytes known to be essential nutrients were compared to their respective RDAs for children to determine if it would be appropriate to remove any essential nutrients from the data set. Generally, analytes whose potential intakes based on the maximum detected concentrations were less than one-fifth of the RDA for children were removed from the data set, as agreed upon by the Commonwealth of Kentucky and EPA in the Methods Document. Seven analytes known to be essential nutrients and known to be toxic only at extremely high concentrations can be removed from the data set on the basis of regulatory guidance (EPA 1995a). These analytes are calcium, chloride, iodine, magnesium, potassium, sodium, and phosphorous. Three essential nutrients, chromium, manganese, and zinc, are not screened using this process because of toxic effects seen from exposure to these chemicals at low concentrations.

1.2.3 Risk Assessment-Specific Data Evaluation

The specific processes used to evaluate data and calculate exposure concentrations under both current and future conditions are described in this section. Section 1.2.3.1 summarizes the evaluation performed to determine representative concentrations of COPCs under current conditions. Section 1.2.3.2 summarizes the evaluation performed to determine modeled representative concentrations of COPCs under future conditions.

1.2.3.1 Current conditions

The specific processes used to evaluate data and calculate exposure concentrations under current conditions are described in this section. The Statistical Analysis System (SAS[®]) (SAS 1990) was used to input and evaluate the data. The following material summarizes the actions performed by various programs during the evaluation. The complete programs are presented in Appendix C of this volume.

First SAS[®] Program (Data Consolidation). The first program read the data set developed from sampling during the recently completed field investigation into SAS[®]. This program read the data into fields to produce a data set with a uniform format to facilitate further data handling. The following are the specific functions performed by this program:

- Eliminated all groundwater data except that from the RGA and McNairy Formation— Groundwater data from samples collected from the UCRS were eliminated because this groundwater is not available for use because of poor yields from wells completed in the UCRS (see Chap. 4 of Vol. 1). While the UCRS was not evaluated as a drinking water source in this assessment, contamination in the UCRS was evaluated as a source of contamination for groundwater drawn from the RGA and McNairy Formation.
- Segregated soil samples into surface [collected 0-1 ft below ground surface (bgs)], subsurface (0-15 ft bgs), and deep (greater than 15 ft bgs) classes—These soil sample depth classes were developed because they were the classes used for the selected exposed populations discussed in Sect. 1.3. In previous risk assessments at PGDP, the subsurface class only contained samples collected from 0 to 10 ft bgs. The ending depth was increased to 15 ft in this assessment because many of the utilities in the WAG 28 area are at or below 10 ft bgs.
- Assigned each sampling station to a site—See Table 1.1.

- Checked spelling of all analytes and their association with chemical abstracts service (CAS)... registry numbers—This screen allows the SAS[®] program to accurately merge contaminant and toxicity information later in the assessment.
- Converted units of measure to those units used in the calculation of CDIs—All chemical concentrations were converted to units of mg/kg or mg/L, and all radionuclide activities were converted to units of pCi/g or pCi/L. This conversion places all chemical information upon a common basis and allows SAS[®] to accurately calculate the representative exposure concentrations used in the derivation of contaminant doses. In addition, the units of measure to which chemicals are converted are the same as those in the toxicity value database; therefore, this conversion allows SAS[®] to merge the contaminant and toxicity information correctly during risk characterization.
- Distinguished between and coded observations as detects and nondetects—Because specific rules must be followed when investigating nondetects, this program performed two filters. The first filter converted the nondetected concentration for analytes not believed to be site-related contaminants to one-half the SQL, and the nondetected concentration for analytes believed to be site-related contaminants to the SQL. Site-related analytes are trichloroethene and its degradation products (1,2-cis-dichloroethane, 1,2-trans-dichloroethane, 1,2-cis-dichloroethene, 1,2-trans-dichloroethene, and vinyl chloride), polychlorinated biphenyls (PCBs), uranium isotopes, metallic uranium, technetium-99, and fluoride. The second filter eliminated those observations that had nondetected concentrations exceeding an analyte's maximum SQL.

Second SAS[®] Program (Precursor Program). This program organized all the subroutines that were run in the third SAS[®] program.

Third SAS[®] Program (Summary Statistics Preparation). This program calculated summary statistics for the data set prepared by the first SAS[®] program. The following are included in the summary (see Tables 1.3, "Data summary for all analytes", and 1.4, "Data summary for detected analytes" in Appendix A): analyte name, frequency of detection, range of detected values, range of nondetected values (i.e., the range of the SQLs used in samples in which the analyte was not detected), form of the distribution of the data, arithmetic means of the detected concentrations, and units of measure for the analyte. In addition, this program created a permanent SAS[®] data set.

Fourth SAS[®] Program (Residential Use Human Health RBC). This program compared the maximum detected concentration of each analyte in each medium to the analyte's medium-specific residential use human health RBC (see Table 1.5). Even though land use at WAG 28 is currently industrial, the residential use human health risk-based screening criteria were used to comply with previous agreements with the regulatory agencies specified in the Methods Document. The data set used in the BERA was not treated in this manner, because human health RBCs are not applicable to nonhuman receptors. The exposure routes included in the calculations of the RBCs were (1) ingestion of a potentially contaminated medium, (2) inhalation of emissions from a potentially contaminated medium, and (3) dermal contact with a potentially contaminated medium. The exposure routes included in the calculations of a potentially contaminated medium, (2) inhalation of emissions from a potentially contaminated medium, (2) inhalation of emissions from a potentially contaminated medium, (2) inhalation of emissions from a potentially contaminated medium, (2) inhalation of emissions from a potentially contaminated medium, (2) inhalation of emissions from a potentially contaminated medium, and (3) external exposure to ionizing radiation emitted from a potentially contaminated medium.

As discussed in the Methods Document, the target HI and ELCR used in the calculation of RBCs for chemicals were 0.1 and 1E-7, respectively, and the target ELCR used in the calculation of RBCs for radionuclides was 1E-6. Also, per regulatory agreement, the lesser (i.e., more conservative) of an analyte's hazard and cancer risk-based screening criteria was used when performing the comparisons.

Analytes known to bioaccumulate or bioconcentrate significantly were not removed from the data set based upon this comparison. The benchmark used to determine if an analyte bioaccumulates significantly was the BAF for fish. The factor was used per regulatory agreement (Methods Document) because of the known propensity of fish to bioaccumulate contaminants and because data on chemical bioaccumulation in fish are readily available. Specifically, if an analyte's BAF for fish exceeded 100, that analyte was not eligible for removal from the data set based on the toxicity screen. The results of the BAF screen are not reported individually in Table 1.5.

Fifth SAS[®] Program (Background and RDA Screen). This program compared the maximum detected concentration of each analyte in soil against its respective background concentration and compared the maximum detected concentration of essential nutrients in soil and groundwater to one-fifth of that nutrient's RDA for children. The background values used in this comparison were taken from *Background Levels of Selected Radionuclides and Metals in Soils and Geologic Media at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky* (DOE 1997) and are presented in Table 1.2. The results of the comparison are shown in Table 1.6. The RDAs are used in this comparison are shown in Table 1.7, and the results are shown in Table 1.8. The data set developed for the ecological risk assessment was not compared against RDAs because the RDAs are not relevant for exposure of nonhuman receptors.

As discussed in the Methods Document, before comparing an analyte's maximum detected concentration to one-fifth of the analyte's RDA, the analyte's concentration was converted to a daily intake for a child. For soil, this conversion was performed by multiplying the analyte's maximum detected concentration in soil by an intake of 200 mg/day and then converting this result to a g/day dose. For water, this conversion was performed by multiplying the maximum detected concentration by an intake of 1 L/day and then converting this result to a g/day dose.

Per regulatory agreement (Methods Document), seven analytes known to be essential nutrients and known to be toxic only at extremely high concentrations can be removed from the data set on the basis of regulatory guidance (EPA 1995a). These analytes are calcium, chloride, iodine, magnesium, potassium, sodium, and phosphorous. Three essential nutrients, chromium, manganese, and zinc, are not screened using this process because of toxic effects from exposure to these chemicals at low concentrations.

Sixth SAS[®] Program (Toxicity Values). This program determined the COPCs based on the previous screening steps, then merged the chemical-specific information [e.g., toxicity values (see Sect. 1.4 and http://risk.lsd.ornl.gov/tox/rap_hp.shtml) such as RfDs and slope factors, Henry's Law constant, toxicity equivalency factor, soil uptake factor, etc.] necessary to run the forward risk equations.

Seventh SAS[®] Program (Output Production). This program compiled the results of the previous programs and produced the following tables:

Table 1.3	Data summary for all analytes by location and medium
Table 1.4	Data summary for detected analytes by location and medium
Table 1.5	Comparison of maximum detected concentrations and activities to human health risk- based screening criteria by location and medium
Table 1.6	Comparison of maximum detected concentrations and activities to background concentrations by location and medium
Table 1.7	RDAs of essential human nutrients

- Comparison of maximum detected concentrations of essential nutrients to RDAs for Table 1.8 children Table 1.9
- Chemicals of potential concern
- Table 1.10 Summary of data evaluation

Table 1.10 is a complete summary of the data evaluation process and lists all detected analytes by location and medium. This table also contains the analyte's frequency of detection, range of nondetected values, range of detected values, arithmetic mean of detected values, background value, human health systemic toxicity and ELCR-based concentrations, RDA (one-fifth value shown), and units of measure. The last column of this table indicates whether the analyte is a human health COPC and, if the analyte is selected as a COPC, the basis for its selection. Codes used to indicate the basis are "P," "B," "E," "Oual," and "Bio." In some cases, an analyte's basis of selection may include more than one code. The following are definitions of these codes:

- **P**—Analyte is a COPC because the maximum detected concentration is greater than a human health RBC.
- B—Analyte is a COPC because the maximum detected concentration is greater than the background . concentration.
- E—Analyte is an essential nutrient but its maximum concentration results in a daily dose that is greater than one-fifth of the RDA for children.
- **Oual**—Analyte is retained as a COPC because screening criteria used in the data evaluation were not available.
- Bio-Analyte is retained because of a high potential to bioaccumulate in fish (i.e., BAF greater than 100).

1.2.3.2 Evaluation of modeled concentrations for groundwater—future condition

As reported in Chap. 5 of Vol. 1, the Multimedia Environmental Pollutant Assessment System (MEPAS) model was used to estimate potential concentrations of selected COPCs in groundwater at the security fence and the DOE property boundary. Appendix B of this volume presents the results of the MEPAS model.

Exhibit 1.8 presents the maximum nonzero modeled concentrations of the COPCs at the PGDP fence boundary and the contaminant's sources, compares the concentrations to residential use human health RBCs, and reports the chemicals with maximum detected concentrations that exceed RBCs. As shown in Exhibit 1.8, the maximum concentrations of the analytes cobalt, neptunium-237, and plutonium-239 do not exceed their respective RBCs; therefore, these contaminants can be removed from the list of COPCs that may migrate from WAG 28 sites.

Exhibit 1.9 summarizes the sources and maximum nonzero modeled concentrations for contaminants that have a source within a WAG 28 site that exceeds an RBC. This exhibit is similar to Exhibit 1.8 except it shows all modeled sources of a contaminant. As shown in this table, there are four inorganic chemcials, one organic chemical, and one radionuclide that may migrate from sources in WAG 28 sites to the PGDP fence boundary at concentrations that exceed RBCs. There was only one contaminant, chromium, with multiple sources within a site, and it did not exceed its RBC.

			Residential use RBC ^d		
Contaminant ^a	Source ^b	Maximum concentration ^c	Cancer	Systemic toxicity	<u> </u>
Inorganic chemicals (mg/	L)				
Chromium	SWMU 194 UCRS soil	7.24E+1	NV	4.2E-3	ST
Cobalt	SWMU 193c UCRS soil	3.56E-2	NV	9.1E-2	None
Lithium	SWMU 194 UCRS soil	6.7E+1	NV	3.0E-2	ST
Manganese	SWMU 193c UCRS soil	5.11E+0	NV	6.7E-2	ST
Strontium	SWMU 194 UCRS soil	1.05E+1	NV	9.0E-1	ST
Organic chemicals (mg/L)				
Trichloroethene	AOC 204 UCRS soil	1.428E+1 ^f	1.4E-4	1.2E-3	Both
Radionuclides (pCi/L) ^g					
Neptunium-237	SWMU 99a UCRS soil	3.86E-2	1.31E-1	NV	None
Plutonium-239	SWMU 99a UCRS soil	1.23E-10	1.22E-2	NV	None
Technetium-99	SWMU 99a surface soil	1.81E+2	2.8E+1	NV	Cancer

Exhibit 1.8. Comparison between maximum nonzero modeled concentrations at the PGDP fence boundary and residential use RBCs

^a All contaminants with an identified source and a modeled concentration are listed.

^b Media for each site in which the source contributing the maximum modeled concentration is located.

^c Maximum modeled contaminant concentration among all sources modeled.

^d All residential use RBCs were taken from Table 1.10 in Appendix A. All cancer RBCs are based on a 40-year exposure; all systemic toxicity RBCs are based on chronic exposure by a child age 1-7 years. Both cancer and systemic toxicity RBCs integrate exposure through ingestion of water, inhalation of vapors emitted by water (showering and household use), and dermal contact with water (showering). Target risk for all cancer RBCs is 1E-7 because more than five contaminants are present. Target HI for all systemic toxicity RBCs is 0.1 because more than five contaminants are present. "NV" indicates an RBC for the endpoint is not available because toxicity information is lacking. The RBC for chromium is for exposure to Cr(VI). The RBCs for radionuclides include contributions from short-lived daughters.

"Cancer" indicates that the modeled concentration exceeds the cancer RBC.
 "ST" indicates that the modeled concentration exceeds the systemic toxicity RBC.
 "Both" indicates that the modeled concentration exceeds both the cancer and systemic toxicity RBC.
 "None" indicates that neither RBC is exceeded by the maximum modeled concentration.

^f The computed maximum concentration is greater than the designated initial concentration at the source (1.42E-7 mg/L). The current receptor is located too close to the source, creating a near-field condition that cannot be properly assessed by a flux boundary condition model; therefore, concentrations have been truncated to the initial dissolved concentration.

⁸ The RBCs for radionuclides include contributions from short-lived daughters.

			Residential Use RBC ^a		Exceed? ^e
Contaminant ^a .	- Source ^b	Maximum concentration ^c	Systemic Cancer toxicity		
Inorganic chemicals (mg/L)				
Chromium	SWMU 194 UCRS soil	7.24E+1	NV	4.2E-3	ST
· · · ·	SWMU 193a UCRS soil	3.803E+0	NV	4.2E-3	ST
	SWMU 193b surface soil	2.02E-3	NV	4.2E-3	None
	SWMU 99a surface soil	2.08E-18	NV	4.2E-3	None
	SWMU 99a UCRS soil	9.40E-20	NV	4.2E-3	None
Lithium	SWMU 194 UCRS soil	6.7E+1	NV	3.0E-2	ST
	SWMU 99a UCRS soil	4.686E+1	NV	3.0E-2	ST
	SWMU 193c UCRS soil	3.805E+1	NV	3.0E-2	ST
	SWMU 99a surface soil	5.632E+0	NV	3.0E-2	ST
	SWMU 193c surface soil	2.085E+0	NV	3.0E-2	ST
Manganese	SWMU 193c UCRS soil	5.11E+0	NV	6.7E-2	ST
Strontium	SWMU 194 UCRS soil	1.05E+1	NV	9.0E-1	ST
	SWMU 193c UCRS soil	7.453E+0	NV	9.0E-1	ST
	SWMU 99a UCRS soil	3.782E+0	NV	9.0E-1	ST
	SWMU 99a surface soil	2.214E+0	NV	9.0E-1	ST
	SWMU 193c surface soil	2.52E-1	NV	9.0E-1	None
Organic chemicals (mg/L)					
Trichloroethene	AOC 204 UCRS soil	1.428E+1 ^f	1.4E-4	1.2E-3	Both
Radionuclides (pCi/L) ^g					
Technetium-99	SWMU 99a surface soil	1.81E+2	2.8E+1	NV	Cancer

Exhibit 1.9. Summary of sources and maximum nonzero modeled concentrations for contaminants that have a source within WAG 28 exceeding a residential use RBC

^a Only contaminants that have a maximum modeled contaminant concentration over all sources that exceed either RBC are listed.

^b Maximum modeled concentration reported for sources within a site. Sites not listed do not contain a source of the contaminant.

^c Maximum modeled contaminant concentration for source.

^d All residential use RBCs were taken from Table 1.10 in Appendix A. All cancer RBCs are based on a 40-year exposure; all systemic toxicity RBCs are based on chronic exposure by a child age 1-7 years. Both cancer and systemic toxicity RBCs integrate exposure through ingestion of water, inhalation of vapors emitted by water (showering and household use), and dermal contact with water (showering). Target risk for all cancer RBCs is 1E-7 because more than five contaminants are present. Target HI for all systemic toxicity RBCs is 0.1 because more than five contaminants are present. "NV" indicates an RBC for the endpoint is not available because toxicity information is lacking.

"Cancer" indicates that the modeled concentration exceeds the cancer RBC.

"Both" indicates that the modeled concentration exceeds both the cancer and systemic toxicity RBC.

"ST" indicates that the modeled concentration exceeds the systemic toxicity RBC.

"Both" indicates that the modeled concentration exceeds both the cancer and systemic toxicity RBC.

"None" indicates that neither RBC is exceeded by the maximum modeled concentration.

^f The computed maximum concentration is greater than the designated initial concentration at the source (1.42E-7 mg/L). The current receptor is located too close to the source, creating a near-field condition that cannot be properly assessed by a flux boundary condition model; therefore, concentrations have been truncated to the initial dissolved concentration.

^g The RBCs for radionuclides include contributions from short-lived daughters.

The number of years from the present required to attain maximum concentrations, assuming current releases from each of the sources, is shown in Exhibit 1.10. The organic chemical, trichloroethene, if released from its sources today, will take 111 years to attain its maximum modeled concentration. The inorganic chemicals, with the exception of lithium (20–78 years), generally will take much longer to attain maximum modeled concentrations, with times ranging from 56 to 15,655 years from present. Risks from exposure to these chemicals are characterized in Sect. 1.5.

Contaminant ^a	ninant ^a Source ^b		Year ^d	
Inorganic chemicals (mg/L)				
Chromium	SWMU 194 UCRS soil	7.24E+1	3783	
	SWMU 193a UCRS soil	3.803E+0	5929	
	SWMU 193b surface soil	2.02E-3	5929	
	SWMU 99a surface soil	2.08E-18	9904-15,654	
	SWMU 99a UCRS soil	9.40E-20	9904-15,655	
Lithium	SWMU 194 UCRS soil	6.7E+1	20	
	SWMU 99a UCRS soil	4.686E+1	67	
	SWMU 193c UCRS soil	3.805E+1	49	
	SWMU 99a surface soil	5.632E+0	78	
	SWMU 193c surface soil	2.085E+0	46	
Manganese	SWMU 193c UCRS soil	5.11E+0	2655	
Strontium	SWMU 194 UCRS soil	1.05E+1	56	
	SWMU 193c UCRS soil	7.453E+0	9854-10,834	
	SWMU 99a UCRS soil	3.782E+0	8953	
	SWMU 99a surface soil	2.214E+0	8953	
	SWMU 193c surface soil	2.52E-1	985410,834	
Organic chemicals (mg/L)				
Trichloroethene	AOC 204 UCRS soil	1.428E+1 ^e	11 Ì	
Radionuclides (pCi/L) ^g				
Technetium-99	SWMU 99a surface soil	1.81E+2	1570	

Exhibit 1.10. Summary of years required to attain maximum modeled concentrations at the PGDP fence boundary for contaminant sources within WAG 28 that contribute maximum contaminant concentrations exceeding residential use RBCs

^a Only contaminants that have a maximum modeled contaminant concentration over all sources that exceed either RBC are listed.

^b Maximum modeled concentration reported for sources within a site. Site sectors that contain a source are listed.

^c Maximum modeled contaminant concentration for source.

^d All dates taken from MEPAS modeling results and are years from present.

^e The computed maximum concentration is greater than the designated initial concentration at the source (1.42E-7 mg/L). The current receptor is located too close to the source, creating a near-field condition that cannot be properly assessed by a flux boundary condition model; therefore, concentrations have been truncated to the initial dissolved concentration.

1.2.4 Evaluation of Data from Other Sources

This section describes results of the Phase I groundwater user survey, agricultural extension agent interviews, Kentucky Department of Fish and Wildlife Resources (KDFWR) information, deer range information, exposure unit information for workers, and site size information. This information was used to develop the exposure assessment in Sect. 1.3.

1.2.4.1 Groundwater user survey Phase I (CH2M Hill 1991a)

In response to the discovery of groundwater contamination in residential wells near PGDP, a survey of groundwater and surface water users in the vicinity of PGDP was conducted in February and March 1990. The two objectives of the survey were to (1) estimate the number of residents using water wells that may be affected by groundwater contamination originating at PGDP and (2) determine the number of surface water intakes on the Ohio River within 15 miles downstream of PGDP. The groundwater user survey included residences and businesses with wells within a 4-mile radius of the plant; therefore, this survey included parts of McCracken and Ballard counties in Kentucky and part of Massac County in Illinois. A questionnaire was mailed to local residents to identify well water users. State agencies and major industrial facilities were contacted to identify surface water users. The information provided by respondents was developed into a database, which is summarized in the following text.

A total of 1988 surveys were delivered, and 44 percent (872) of these were returned. Of the respondents, 58 percent used well water for some purpose. Eighty-four percent used well water as their sole water supply. Eighty-five percent used well water for drinking; 47 percent used well water for irrigation; 29 percent used well water for watering livestock; and 80 percent used well water for domestic uses such as laundry, washing cars, etc. The total depth of wells in the study area (i.e., the area investigated by this survey) was reported to range from 15 ft to 245 ft; however, 21 percent of residents did not report total depth. The most frequently reported total depth was 40 ft (26 respondents), followed by 30 ft (21 respondents) and 100 ft (20 respondents). Fifty-four percent of wells were reported to be 20–60 ft deep. Plastic and tile were the predominant construction materials; however, steel, brick, and concrete were also reported.

Unfortunately, the questionnaire used in this survey did not determine frequency of groundwater use. See Chap. 1 of Appendix 5 in the Methods Document for a reproduction of the questionnaire. As indicated earlier, these data were used qualitatively in the exposure assessment to develop the site conceptual model and reduce the level of uncertainty of the exposure assessment in the BHHRA.

1.2.4.2 Agricultural extension agent interviews

To gather site-specific agricultural information, the Agricultural Extension Agents for Ballard and McCracken counties were contacted in February 1994. Information on population, gardening, crop farming, livestock farming, and fish farming was requested. Summaries of the interviews are presented in Chap. 2 of Appendix 5 of the Methods Document. Data gathered from the agents were used qualitatively in the exposure assessment to develop the site conceptual model and reduce the level of uncertainty of the exposure assessment in the BHHRA.

1.2.4.3 KDFWR information

During the development of the site conceptual model, it was determined that wildlife may also serve as an important exposure pathway to humans. To determine the level of importance of this pathway, requests were made for reports on harvest of deer, ducks, geese, and turkey in Ballard and McCracken counties. Information on these game species was solicited because they are the most widely hunted animals in the area and require specific licenses and check-in procedures. Harvest information is provided in Chap. 3 of Appendix 5 of the Methods Document.

1.2.4.4 Site size information

To accurately represent exposure to contaminated soil or sediment in each of the sites, the size of each site was determined (see Exhibit 1.11). These sizes were subsequently integrated with the exposure

unit information presented in Sects. 1.2.4.5, 1.2.4.6, and 1.2.4.7 when calculating the daily intake or daily dose for each COPC. Methods used to integrate exposure unit size and site size are presented along with the exposure equations presented in Sect. 1.3.

Sites	Area (sq. ft)	Area (acres)
SWMU 99a	104,544	2.4
SWMU 99b	13,068	0.3
SWMU 193a	757,944	17.4
SWMU 193b	187,308	4.3
SWMU 193c	3,789,720	87.0
SWMU 194	1,816,307	41.7
AOC 204	492,090	11.3

Exhibit	1.11.	Areas of	WAG 28 sites
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1.2.4.5 Exposure unit information for workers

During the development of the site conceptual model, it was determined that the size of a site was directly proportional to the time that a worker would be directly exposed to potentially contaminated soil at a site. To account for this, an exposure unit representing the reasonable area that an industrial worker would occupy in a day's time was selected. This value was 0.5 acres as presented in Chap. 5 of Appendix 5 of the Methods Document.

1.2.4.6 Exposure unit information for residents

Similarly, it was determined that the size of a site was directly proportional to the time that a resident would be exposed to potentially contaminated soil at a site. An exposure unit representing the reasonable area that a rural resident would occupy in a day's time was selected. This value was the same as the area of the average residential garden in western Kentucky (0.25 acres). This area was determined from interviews with local agricultural extension agents as presented in Chap. 2 of Appendix 5 of the Methods Document.

1.2.4.7 Wildlife range information

It was determined that the size of a site was directly proportional to the time that a wildlife receptor would be exposed to potentially contaminated soil and vegetation at a site. The exposure unit size for deer was based on the average home range of deer in the United States, which is 494 acres. The means by which this value was determined is presented in Chap. 4 of Appendix 5 of the Methods Document. The exposure unit size for rabbit is 7.7 acres (EPA 1993a). This value was based on the average home ranges of cottontail rabbits, male and female, in Wisconsin and Pennsylvania. The exposure unit size for quail is 25.5 acres (EPA 1993a). This value was based on the average home ranges for individual quail, male and female, and coveys in Iowa, Illinois, and Tennessee.

1.2.5 Summary of COPCs

A general summary of COPCs in soil by depth class for each site and in RGA and McNairy Formation groundwater is presented in Exhibit 1.12. Table 1.9 contains a detailed summary listing the COPCs individually. In Table 1.9, analytes marked with an asterisk lack toxicity information [i.e., a toxicity value is not in the EPA's Integrated Risk Information System (IRIS) (EPA 1998a) or Health Effects Assessment Summary Tables (HEAST) (EPA 1998b) and is not available from the alternate approved sources listed in the Methods Document]. Finally, Table 1.10 summarizes each detected_ analyte, including the reason for the retention of an analyte as a COPC.

A comparison between the COPCs listed in Table 1.9 and the preliminary list of COPCs shown in Exhibit 1.7 shows that Table 1.9 is far more extensive than Exhibit 1.7; however, all COPCs listed in Exhibit 1.7 are included in Table 1.9.

		Analyte type			
Location	Medíum ^a	Metals	Organics		
SWMU 99a	Surface soil	4	19	6	
	Subsurface soil	11	84	6	
	RGA groundwater	18	4	2	
	McNairy groundwater	· 0	4	0	
SWMU 99b	Subsurface soil	4	1	0	
	RGA groundwater	8	1	1	
SWMU 193a	Surface soil	1	13	0	
	Subsurface soil	3	13	0	
	RGA groundwater	6	5	1	
	McNairy groundwater	1	3	2	
SWMU 193b	Surface soil	3	0	• 0	
	Subsurface soil	3	0	0	
	RGA groundwater	0	7	1	
	McNairy groundwater	0	2	Ō	
SWMU 193c	Surface soil	3	0	0	
	Subsurface soil	10	1	0	
	RGA groundwater	-0	2	0	
	McNairy groundwater	21	15	- 1	
SWMU 194	Subsurface soil	6	1	0	
AOC 204	Subsurface soil	0	6	0	
	RGA groundwater	0	9	0	

Exhibit 1.12 General summary of COPCs by location, medium, and analyte type

^a Media are listed by groups used in the risk assessment. A brief list is provided below. A complete discussion is found in Sect. 1.3 of this assessment.

Surface soil (0-1 ft bgs)-Receptors are the current and future industrial worker, future excavation worker, future rural resident, and future recreational user.

Subsurface soil (0-15 ft bgs)---The receptor is the future excavation worker.

· • •

RGA-The receptors are the future industrial worker and future on-site rural resident.

1.3 EXPOSURE ASSESSMENT

Exposure is the contact of an organism with a chemical or physical agent. The magnitude of exposure (i.e., dose) is determined by measuring or estimating the amount of an agent available at exchange boundaries (e.g., gut, skin) during a specified period. Exposure assessment is a process that uses information about the exposure setting and human activities to develop conceptual site models for current and potential future conditions. This section introduces the general methods used in exposure assessment, applies these methods to WAG 28 to develop a conceptual site model, and presents the doses for the COPCs resulting from this application.

The first step in the exposure assessment is to characterize the exposure setting. This includes describing the activities of the human population on or near the site that may affect the extent of exposure and the physical characteristics of the site. During this process, sensitive subpopulations that may be present at the site or that may be exposed to contamination migrating from the site are also considered to determine if the BHHRA should address these populations. Generally, site characterization results in a qualitative evaluation of the site and the surrounding population.

The second step in the exposure assessment is to identify exposure pathways. Exposure pathways describe the path a contaminant travels from its source to an individual. A complete exposure pathway includes all links between the source and the exposed population; therefore, a complete pathway consists of the source of release, a mechanism of release, a transport medium, a point of potential human contact, and an exposure route.

The third step in the exposure assessment is to calculate dose by quantifying the magnitude, frequency, and duration of exposure for the populations for the exposure pathways selected for quantitative evaluation. This step involves estimating exposure or representative concentrations for COPCs and quantifying pathway-specific intakes.

All exposure estimates in this BHHRA represent normalized exposure rates that are evaluated for sources of uncertainty such as variability in data, modeling results, and/or parameter assumptions. Specifically, in this BHHRA, the exposure estimate is an estimation of the reasonable maximum exposure (RME) that can be expected to occur under current or future site conditions. As defined in RAGS, an RME estimate is a conservative estimate of exposure that falls within the upper bound of the range of all possible exposure estimates. In situations where populations are exposed through multiple pathways, RME estimates are calculated for both individual and multiple pathways.

The focus of the exposure assessment for WAG 28 at PGDP is to determine chronic intake or dose. The chronic exposure estimate is used because it allows for estimation of health consequences that result from long-term or unrestricted exposure to contaminants at sources in WAG 28. Subchronic exposures receive less attention, because these exposures require the use of assumptions concerning restrictions on rates of contact with contaminated media.

1.3.1 Characterization of Exposure Setting

The first step in evaluating exposure is to characterize surface features, meteorology, geology, demography and land use, ecology, hydrology, and hydrogeology of the area inhabited by potential receptors. These aspects are fully discussed in Chaps. 1, 2, and 3 of Vol. 1 of this report. Physical descriptions and photographs (Figs. 1.2–1.8) of the WAG 28 sites are included within this exposure assessment to support later discussions of the conceptual model and its uncertainties. The following sections present physical descriptions of the sites. Two of the sites (i.e., SWMU 99 and SWMU 193) have been subdivided into areas within the site that best define potential releases and exposures.

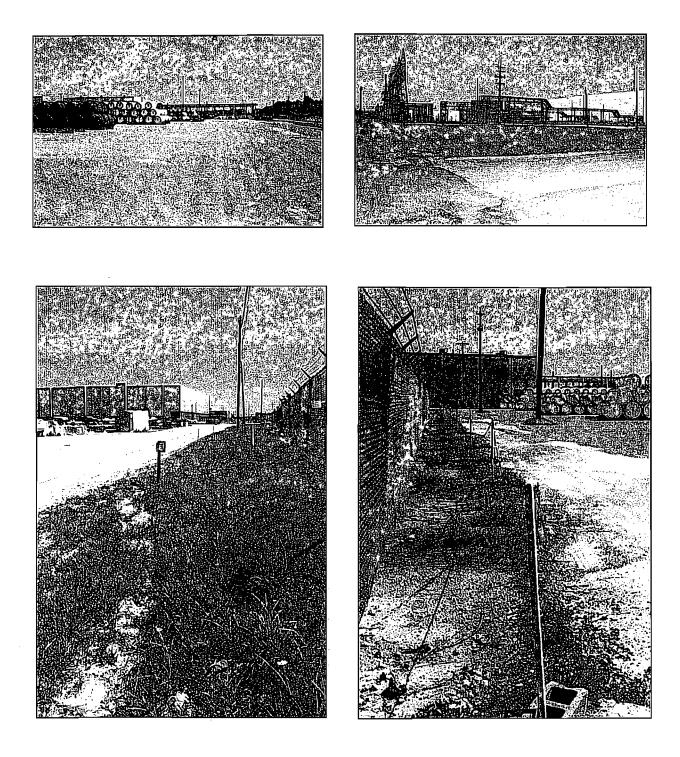
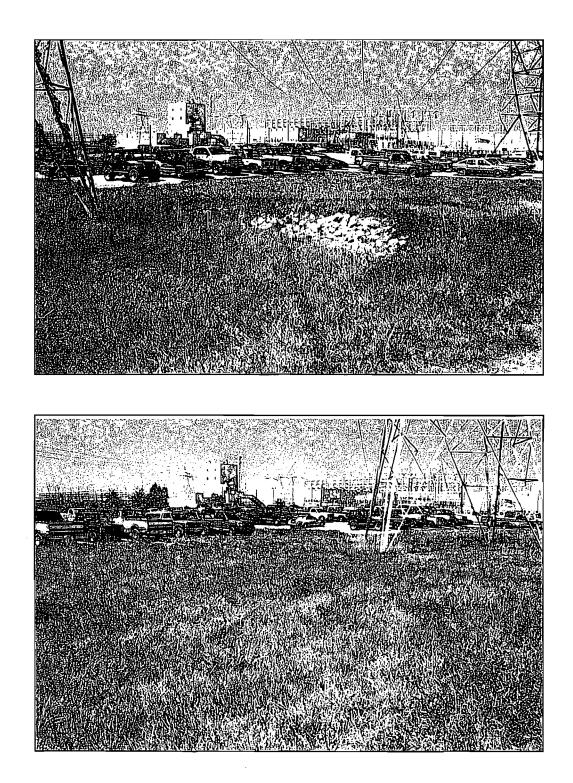


Figure 1.2. Photographs of SMWU 99a



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Figure 1.3. Photographs of SWMU 99b (Note: Only a small portion of grass in the foreground is within SWMU 99b.)

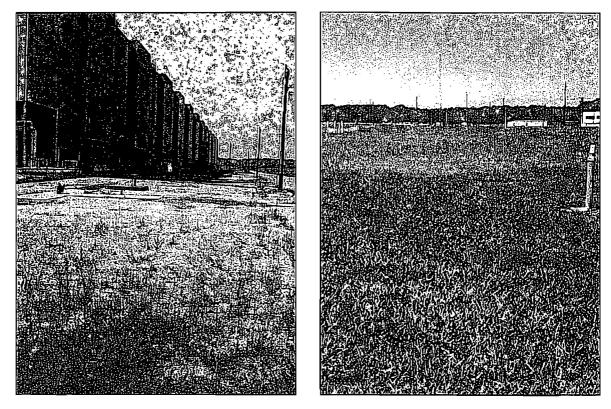


Fig. 1.4. Photographs of SWMU 193a

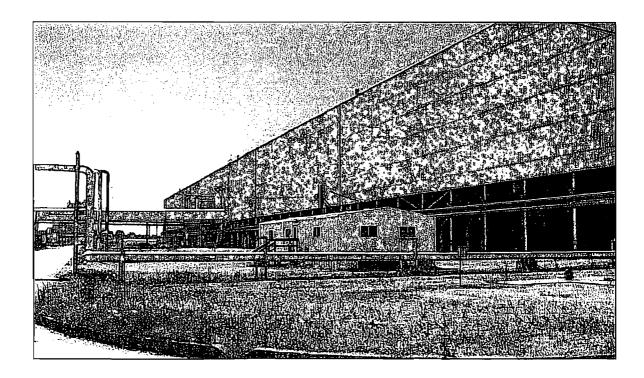


Fig. 1.5. Photograph of SWMU 193b

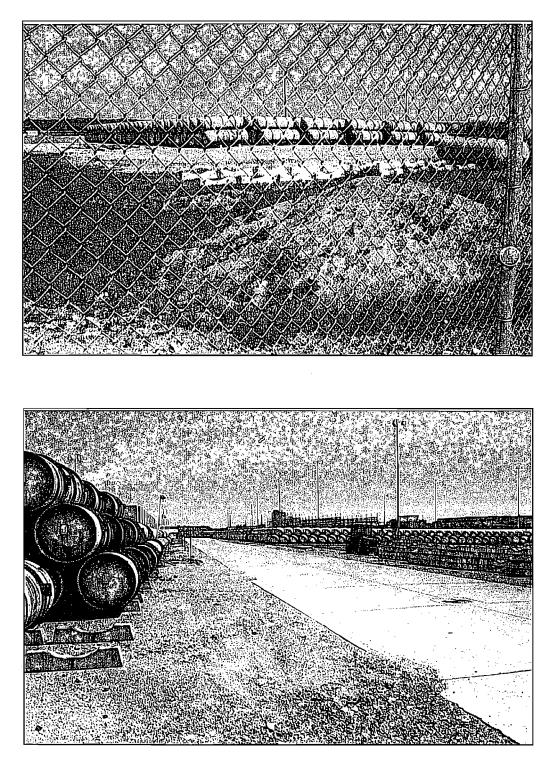


Fig. 1.6. Photographs of SWMU 193c

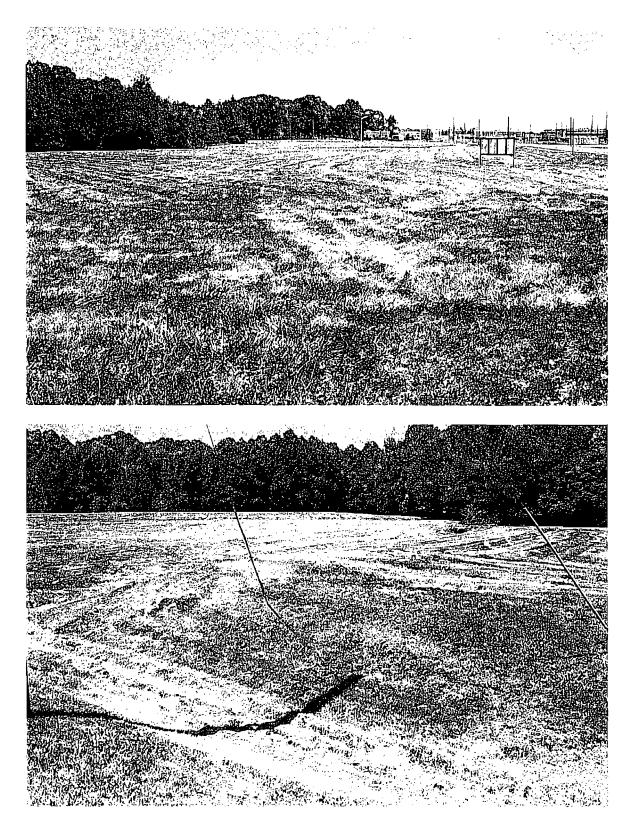


Fig. 1.7. Photographs of SWMU 194

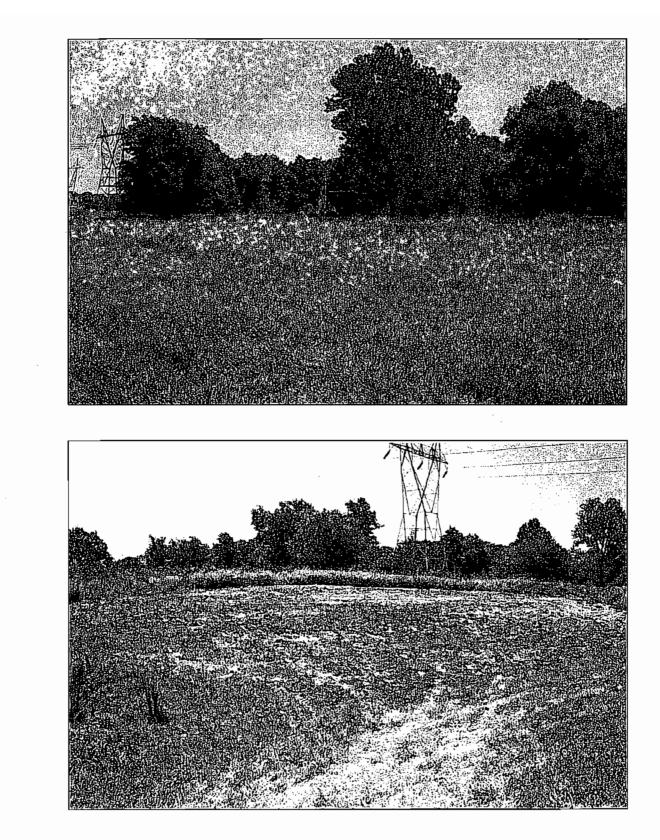


Fig. 1.8. Photographs of AOC 204

1.3.1.1 Physical description of SWMU 99a

SWMU 99a, site of the former C-745 Kellogg Buildings, is located along the eastern edge of PGDP, south of Building C-360, immediately north of Tennessee Avenue, and west of Patrol Road 3. The buildings were constructed in 1951 as support facilities during construction of the PGDP cascade facilities. Degreasing operations using trichloroethene possibly occurred on this site. The buildings have been demolished, and the area now serves as the C-746-C Classified Scrap Yard and the C-745-E uranium hexafluoride (UF₆) Cylinder Storage Yard. SWMU 99a was identified as a possible source area due to past practices on the site. At some time, a layer of gravel was placed over the soil on most of site to improve drainage, improve site access, and control weeds. SWMU 99a includes the area bound by Tennessee Avenue on the south, the PDGP security fence on the east, the security fence to Building C-360 on the north, and 18th Street on the west. The total area is approximately 2.4 acres. See Fig. 1.2 for photographs of SWMU 99a and Fig. 3.6 in Vol. 1 for detailed diagrams.

The percentages of each site covered by concrete/asphalt, gravel, or grass/trees/shrubs are presented in Exhibit 1.13. Approximately 40 percent of SWMU 99a is covered by concrete/asphalt and 60 percent by gravel.

	Area	Cover type and percent of total area covered ^a		
Site	(acres) ^b	Concrete/asphalt ^e	Gravel	Grass/trees/shrubs
SWMU 99a	2.4	40%	60%	0%
SMWU 99b	0.3	0%	80%	20%
SMWU 193a	17.4	0%	10%	90%
SWMU 193b	4.3	0%	100%	0%
SWMU 193c	87.0	15%	80%	5%
SWMU 194	41.7	0%	0%	100%
AOC 204	11.3	0%	0%	100%

^a All percentages are estimates.

^b Includes ditches.

^c Includes areas of buildings.

1.3.1.2 Physical description of SWMU 99b

SWMU 99b, a former septic tank and leaching field used by the Kellogg Buildings, is located immediately outside the east guard house of the plant. The tank and the associated field, which is connected to the Kellogg Buildings by a vitreous clay drain line, are located approximately 350-400 ft southeast of the building site in the gravel parking lot east of Patrol Road 3. Although lateral lines for the leaching field were found intact when encountered during construction activities in late 1994, they were not located during RI field activities. The suspected location is situated under a gravel-covered parking area between the contractor staging area to the north and AOC 204 to the south. The total area is approximately 0.3 acres. See Fig. 1.3 for photographs of SWMU 99b and Fig. 3.6 in Vol. 1 for detailed diagrams. An estimated 80 percent of SWMU 99b is covered by gravel and 20 percent by grass (Exhibit 1.13).



1.3.1.3 Physical description of SWMU 193a

SWMU 193a, the former Millwright Shop, is the outside perimeter of Building C-333 located in the western portion of SWMU 193 north of Michigan Avenue and west of 13th Street. The shop is no longer standing, and all that remains is a concrete pad. The site does not include Building C-333 but includes the property directly west of the building. The site is bound on the south by Michigan Avenue, on the west by Patrol Road 5, on the north by Ohio Avenue, and on the east by 13th Street. The area is drained by the plant storm drain system, which eventually exits the plant through Kentucky Pollutant Discharge Elimination System (KPDES) Outfall 009. The total area is approximately 17.4 acres. See Fig. 1.4 for photographs of SWMU 193a and Fig. 3.7 in Vol. 1 for detailed diagrams. An estimated 90 percent of SWMU 193a is covered by grass (Exhibit 1.13) and 10 percent by gravel.

1.3.1.4 Physical description of SWMU 193b

SWMU 193b, the former Pipe Fabrication Shop, is the outside northern perimeter of Building C-333 located in the northern portion of SMWU 193. The site does not include Building C-333 but includes the property directly north of the building. The site is bound on the south by Building C-333, on the north by Ohio Avenue, on the west by 13th Street, and on the east by 16th Street. The area is drained by the plant storm drain system, which eventually exits the plant through KPDES Outfall 009. The total area is approximately 4.3 acres. See Fig. 1.5 for photographs of SWMU 193b and Fig. 3.7 in Vol. 1 for detailed diagrams. An estimated 100 percent of SWMU 193b is covered by gravel (Exhibit 1.13).

1.3.1.5 Physical description of SMWU 193c

SWMU 193c is located on the south side of the C-333 building. The site formerly consisted of temporary buildings used during the construction of PGDP, including the electrical warehouse, general warehouse, sheet metal shop, light and heavy equipment shops, acetylene shop, paint shop, civil engineering testing laboratory, filling station, and steel fabrication shop. A leaching field was located in the southwest corner of the site. The leaching field consists of 4-in. drain tiles in shallow soil. Currently, the site is used to store UF_6 cylinders. The site is bound on the north by Michigan Avenue, on the south by Patrol Road 4, on the east by 21st Street, and on the west by Patrol Road 5. The area is drained by the plant storm drain system, which eventually exits the plant through KPDES Outfall 011. The total area is approximately 87.0 acres. See Fig. 1.6 for photographs of SWMU 193c and Fig. 3.7 in Vol. 1 for detailed diagrams. An estimated 15 percent of SWMU 193c is covered by concrete/asphalt, 80 percent by gravel, and 5 percent by grass (Exhibit 1.13).

1.3.1.6 Physical description of SWMU 194

SWMU 194 is located in the southwest portion of the plant directly outside the security fence. SWMU 194 was the site of the administrative portion of the McGraw construction facilities and consisted of an administration building (105,500 ft²), cafeteria (10,200 ft²), security guard headquarters (5,360 ft²), hospital (4,480 ft²), purchasing building (12,000 ft²), paper and stationary warehouse (3,900 ft²), a boiler house, and two leaching fields located west of Hobbs Road. All of the buildings have been demolished. The site is bound on the north by Curlee Road, on the south by Patrol Road 4, on the east by Patrol Road 5, and extends west of Hobbs Road. The total area is approximately 41.7 acres. See Fig. 3.9 for photographs of SWMU 194 and Fig. 4.19 in Vol. 1 for detailed diagrams. An estimated 100 percent of SMWU 194 is covered by grass (Exhibit 1.13).

1.3.1.7 Physical description of AOC 204

AOC 204 is located on the eastern side of PGDP and bound on the north and south by KPDES Outfalls 010 and 011 and on the east and west by Dyke Road and the security fence. It is suspected that AOC 204 was used as a staging area or construction debris burial ground associated with the original construction of the plant. The surface of AOC 204 is undulating, with elevations ranging from 364 to 382 ft above mean sea level. The area is covered with heavy vegetation and a young stand of trees. A small stand ditch (approximately 4 ft wide and 3 ft deep) is situated across the mound from north to south. The total area is approximately 11.3 acres. See Fig. 1.8 for photographs and Fig. 3.10 in Vol. 1 for detailed diagrams. An estimated 50 percent of AOC 204 is covered by grass and 50 percent by trees/shrubs (Exhibit 1.13).

1.3.2 Demography and Land Use

As indicated in the physical descriptions presented, current land use of all WAG 28 sites within the fence is industrial. Under current use, only plant workers and authorized visitors are allowed access to SWMUs 99a and 193a, b, and c because of security arrangements. SWMUs 99b and 194 and AOC 204 are located outside the security fence but within DOE property and are accessible to the public. As discussed in the PGDP *Site Management Plan* (DOE 1996b), foreseeable future land use of the area is expected to be industrial as well; however, alternative uses in the future are possible as shown by the current use of areas surrounding PGDP.

At present, both recreational and residential land uses occur in areas surrounding PGDP. Recreational use occurs in the Western Kentucky Wildlife Management Area (WKWMA). WKWMA is used primarily for hunting and fishing, but other activities include horseback riding, field trials, hiking, and bird watching. An estimated 5000 fishermen visit the area annually, according to KDFWR, manager of WKWMA. Residential use near the plant generally is rural residential and includes agricultural activities; however, more urban residential use occurs in the villages of Heath, Grahamville, and Kevil, which are within 3 miles of DOE property boundaries. The closest major urban area is the municipality of Paducah, Kentucky, which has a population of approximately 28,000 and is approximately 10 miles from PGDP. Other municipalities in the region near PGDP are Cape Girardeau, Missouri, which is approximately 40 miles west of the plant, and the cities of Metropolis and Joppa, Illinois, which are across the Ohio River from PGDP. Total population within a 40-mile radius of the plant is approximately 500,000, with about 50,000 people living within 10 miles, based on 1990 census data. The population of McCracken County, in which PGDP is located, is an estimated 63,000 people.

In the area near PGDP and in western Kentucky in general, the economy has historically been agriculturally based; however, industry has increased in recent years. PGDP is a major employer with approximately 1800 workers. Another major employer near PGDP is the TVA Shawnee Steam Plant that employs approximately 500 people.

1.3.3 Identification of Exposure Pathways

Exposure pathways describe how a contaminant travels from its source to an individual. A complete exposure pathway includes all links between the source and the exposed population. That is, a complete pathway consists of a source of release, a mechanism of release, a transport medium, a point of potential human contact, and an exposure route. Sources of release, mechanisms of release, and transport media are discussed completely in Chap. 5 of Vol. 1 of this report. The following discussions focus on points of potential human contact, types of receptors, and exposure routes.



1.3.3.1 Points of human contact—land use considerations

As discussed previously, sites in WAG 28 are located in or around heavily industrialized property. The current land use for sites within the security fence is industrial. According to KDEP and EPA agreement (Methods Document), this land use limits the current exposure medium for a receptor to the first 1 ft of surface soil. The current land use for sites outside the security fence is also industrial but may be used for recreational activities by trespassers.

The current land use at WAG 28 is expected to continue in the foreseeable future. That is, the most plausible future land use of the WAG 28 area is also industrial and recreational; however, uses of areas surrounding PGDP indicate that it would be prudent to examine a range of land uses to provide managers with estimates of the risk that may be posed to humans under these alternative uses. In addition, consideration of a range of land uses is consistent with requirements outlined in the Commonwealth of Kentucky's Risk Assessment Guidance (KDEP 1995). Alternative land uses considered in this assessment. in order of their plausibility, are industrial, excavation, recreational, and rural residential. As with industrial land use and per agreement with KDEP and EPA (Methods Document), soil exposure for the industrial worker (future conditions), rural resident, and recreational user is limited to the first 1 ft of surface soil; therefore, materials in lines or in line-bedding materials are assumed to be unavailable for direct contact for these land use scenarios. For the excavation worker, the first 15 ft of soil are assumed to be available for direct contact. The Methods Document directs that the excavation worker scenario consider soil to a depth of 10 ft. This assessment uses soil to a depth of 15 ft for the excavation worker. because many of the utility lines in the WAG 28 area are at or near this depth. In addition, per the site descriptions contained in Vol. 1 and per agreement with KDEP and EPA in the Methods Document, both the future industrial worker and future rural resident are assumed to use groundwater drawn from the RGA and McNairy Formation underlying sites in WAG 28.

The assessment assumes that residents are the individuals most likely to partake in recreational activities at WAG 28 and near PGDP. That is, in addition to exposure from rural residential activities, a resident may also be exposed during recreational activities. This assumption means that it is possible that the exposure of a rural resident may be greater than that reported in this BHHRA if the rural resident also receives exposure through the recreational routes of exposure. To address this issue, the reader may wish to combine the exposure values from the recreational user scenario with those from the rural resident scenario.

1.3.3.2 Potential receptor populations

As noted previously, the receptor populations are industrial workers under current conditions and industrial workers, excavation workers, recreational users, and rural residents under potential future conditions. Within these broad categories, the recreational users and rural residents contain age cohorts that require consideration (Methods Document). For the recreational users, the cohorts considered are the child (age 1–7 years), teen (age 8–20 years), and the adult (older than 21 years). For rural residents, the cohorts considered are children (age 1–7) and older individuals (termed adults in this assessment). The recreational user and the rural resident population may also contain sensitive subpopulations such as pregnant women, young children (age 0–1 year), the elderly, and the infirm. In this assessment, exposures to these subpopulations are not quantified, because much of the information needed is not available; however, these subpopulations are considered qualitatively in the uncertainty discussion included in this assessment. Also, as noted earlier, this assessment assumes that the recreational user is a rural resident who has repeated access to the study area. Recreational users not residing in the study area are not considered separately because nearby residents were determined to be the individuals most likely to take part in recreational activities at PGDP on a continual basis. In addition, the exposure assessment

determined that little useful information would be obtained by including a separate visiting recreationaluser in the assessment.

1.3.3.3 Delineation of exposure points and exposure routes

As previously discussed, human health risks are assessed by determining exposure points and exposure routes. Exposure points are locations where human receptors can contact contaminated media. Exposure routes are the processes by which human receptors contact contaminated media. The exposure routes considered during the exposure assessment per agreement with the regulatory agencies (Methods Document) are listed in the following paragraphs. This material also presents reasons for selecting or not selecting each exposure route for each of the potentially exposed populations. Not all exposure routes presented in the following list are quantitatively evaluated in the BHHRA; after all possible exposure routes were extensively reviewed, only the probable exposure routes were quantified.

- Ingestion of groundwater as a drinking water source—Residential and industrial use of groundwater is common in western Kentucky. Potential receptors for this pathway are rural residents and industrial workers.
- Inhalation of volatile constituents emitted while using groundwater—As noted, residential and industrial use of groundwater is common in western Kentucky. Rural residents and industrial workers are potential receptors for this exposure route.
- **Dermal contact with groundwater while showering**—As noted, residential and industrial use of groundwater is common in western Kentucky. Rural residents and industrial workers are potential receptors for this exposure route.
- External exposure to ionizing radiation emitted by constituents in groundwater while showering—As noted, residential and industrial use of groundwater is common in western Kentucky. Rural residents and industrial workers are potential receptors for this exposure route.
- Inhalation of volatile constituents while irrigating with groundwater—In the Midwest, irrigation of farmland with groundwater using center pivot irrigation is common. Rural residents are potential receptors for this exposure route.
- Incidental ingestion of soil (soil and waste)—Industrial processes at WAG 28 have contaminated the soil. Recreational users may ingest soil during recreational activities, and residents may ingest soil while gardening. Industrial workers may ingest soil while working outdoors, and excavation workers may ingest soil while digging. Recreational users, rural residents, industrial workers, and excavation workers are potential receptors for this exposure route.
- Dermal contact with soil (soil and waste)—Industrial processes at WAG 28 have contaminated the soil. Recreational users may get soil on their skin during recreational activities, and residents may get soil on their skin while gardening. Industrial workers may get soil on their skin while working outdoors, while excavation workers may get soil on their skin while digging. Recreational users, rural residents, industrial workers, and excavation workers are potential receptors for this exposure route.
- Inhalation of particulates emitted from soil (soil and waste)—Industrial processes at WAG 28 have contaminated the soil, and this soil may release particulates to the air when the soil is dry and disturbed. Recreational users may inhale these particulates during recreational activities, and residents may inhale these particulates while gardening. Industrial workers may inhale these particulates while working outdoors, and excavation workers may inhale these particulates while digging.

Recreational users, rural residents, industrial workers, and excavation workers are potential receptors for this exposure route.

- Inhalation of volatile constituents emitted from soil (soil and waste)—Industrial processes at WAG 28 have contaminated the soil. Some of these contaminants may be volatile and released to the air as vapors. Recreational users may inhale these vapors during recreational activities, and residents may inhale these vapors while gardening. Industrial workers may inhale these vapors while working outdoors, and excavation workers may inhale these vapors while digging. Recreational users, rural residents, industrial workers, and excavation workers are potential receptors for this exposure route.
- External exposure to ionizing radiation emitted by constituents in soil (soil and waste)— Industrial processes at WAG 28 have contaminated the soil. Radionuclides present in contaminated soil undergo decay and emit ionizing radiation. Recreational users may be exposed to this ionizing radiation during recreational activities, and residents may be exposed to it while gardening. Industrial workers may be exposed to ionizing radiation while working outdoors, and excavation workers may be exposed to it while digging. Recreational users, rural residents, industrial workers, and excavation workers are potential receptors for this exposure route.
- Incidental ingestion of water while swimming in privately owned fishponds filled with groundwater—Construction of fishponds was determined to be a viable future agricultural land use. The Agricultural Extension Agents for Ballard and McCracken counties noted that "pay-to-fish" lakes filled with groundwater exist in Ballard County and that the Agriculture Extension Office has actively promoted the construction of commercial ponds (see Chap. 2 of Appendix 5 of the Methods Document). Although the agents disagreed on how profitable this form of farming could be in western Kentucky, the presence of "pay-to-fish" lakes filled with groundwater in Ballard County indicates that aquaculture is a viable alternative rural residential land use in the study area. Because open bodies of water are often attractive for recreation, swimming and wading in these ponds by residents is reasonable to assume. Residents could incidentally ingest water while swimming. Rural residents are potential receptors for this exposure route.
- Dermal contact with water while swimming or wading in privately owned fishponds filled with groundwater—The rationale for considering ponds is presented in the previous paragraph. During recreational use (e.g., swimming or wading), residents would have dermal contact with water. Rural residents are potential receptors for this exposure route.
- External exposure to ionizing radiation emitted by constituents in water while swimming or wading in privately owned fishponds filled with groundwater—The rationale for considering ponds is presented previously. During recreational use, residents could be exposed to ionizing radiation emitted by radionuclides in water. Rural residents are potential receptors for this exposure route.
- Incidental ingestion of sediment while swimming or wading in privately owned fishponds filled with groundwater—The rationale for considering ponds is presented previously. During recreational activities, residents could incidentally ingest sediment contaminated by constituents in groundwater. Rural residents are potential receptors for this exposure route.
- Dermal contact with sediment while swimming or wading in privately owned fishponds filled with groundwater—The rationale for considering ponds is presented previously. During recreational use, residents could have dermal contact with sediment contaminated by constituents in groundwater. Rural residents are potential receptors for this exposure route.

- External exposure to ionizing radiation emitted by constituents in sediment while swimming orwading in privately owned fishponds filled with groundwater—The rationale for considering ponds is presented previously. During use, residents could be exposed to ionizing radiation emitted by radionuclides in sediment. Rural residents are potential receptors for this exposure route.
- Ingestion of fish raised in privately owned fishponds filled with groundwater—The fish raised in ponds could be exposed to contaminants in groundwater and may accumulate some contaminants in their edible tissues. These fish, caught in either a "pay-to-fish" or a commercial pond by residents, could reasonably be expected to be consumed. Rural residents are potential receptors for this exposure route.
- Incidental ingestion of surface water from creeks or ponds—Open bodies of water, such as Bayou Creek or settling ponds, are attractive for recreation (e.g., swimming and wading) and must be maintained. Although such bodies of water are not included in the assessment of the WAG 28 area, contaminants may migrate from WAG 28 to these areas. Incidental ingestion of water could occur while a person is swimming. Recreational users and industrial workers are potential receptors for this exposure route. Surface migration to off-site locations is not believed to be an important pathway of migration at WAG 28 as discussed in Chap. 5 of Vol. 1.
- Dermal contact with surface water while swimming or wading in creeks or ponds—The rationale for considering open bodies of water is presented in the previous paragraph. During recreational use, a person would have dermal contact with water. Although such bodies of water are not included in this assessment of the WAG 28 area, contaminants may migrate from WAG 28 to these areas. Recreational users and industrial workers are potential receptors for this exposure route. Surface migration to off-site locations is not believed to be an important pathway of migration at WAG 28 as discussed in Chap. 5 of Vol. 1.
- External exposure to ionizing radiation emitted by constituents in surface water while swimming or wading in creeks or ponds—The rationale for considering open bodies of water is presented previously. During recreational use, exposure to ionizing radiation emitted by radionuclides in water could occur. Although such bodies of water are not included in this assessment of the WAG 28 area, contaminants may migrate from WAG 28 to these areas. Recreational users and industrial workers are potential receptors for this exposure route. Surface migration to off-site locations is not believed to be an important pathway of migration at WAG 28 as discussed in Chap. 5 of Vol. 1.
- Incidental ingestion of sediment while swimming or wading in creeks or ponds—The rationale for considering open bodies of water is presented previously. During recreational use, a person could incidentally ingest sediment. Although such bodies of water are not included in this assessment of the WAG 28 area, contaminants may migrate from WAG 28 to these areas. Recreational users and industrial workers are potential receptors for this exposure route. Surface migration to off-site locations is not believed to be an important pathway of migration at WAG 28 as discussed in Chap. 5 of Vol. 1.
- Dermal contact with sediment while swimming or wading in creeks or ponds—The rationale for considering open bodies of water is presented previously. During recreational use, a person could have dermal contact with sediment. Although such bodies of water are not included in this assessment of the WAG 28 area, contaminants may migrate from WAG 28 to these areas. Recreational users and industrial workers are potential receptors for this exposure route. Surface migration to off-site locations is not believed to be an important pathway of migration at WAG 28 as discussed in Chap. 5 of Vol. 1.

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- External exposure to ionizing radiation emitted by constituents in sediment while swimming or wading in creeks or ponds—The rationale for considering open bodies of water is presented previously. During recreational use, a person could be exposed to ionizing radiation emitted by sediment. Although such bodies of water are not included in this assessment of the WAG 28 area, contaminants may migrate from WAG 28 to these areas. Recreational users and industrial workers are potential receptors for this exposure route. Surface migration to off-site locations is not believed to be an important pathway of migration at WAG 28 as discussed in Chap. 5 of Vol. 1.
- Ingestion of fish from creeks and ponds containing surface water—Fish living in Bayou Creek or settling ponds may accumulate contaminants in surface water in their edible tissues. Although such bodies of water are not included in this assessment of the WAG 28 area, contaminants may migrate from WAG 28 to these areas. Recreational users and residents may catch and consume fish from the potentially impacted surface water bodies. Potential receptors for this route of exposure are recreational users. Surface migration to off-site locations is not believed to be an important pathway of migration at WAG 28 as discussed in Chap. 5 of Vol. 1.
- Ingestion of vegetables and produce raised in contaminated soil (soil and waste)—As noted in Chap. 2 of Appendix 5 of the Methods Document, crop farming and gardening are common activities near PGDP, and this land use pattern may be expanded to the WAG 28 area in the future after the industrial infrastructure is removed. Because industrial use of WAG 28 has contaminated the soil, plants raised in this soil may accumulate these contaminants. Finally, humans may consume contaminated produce. Potential receptors for this route of exposure are rural residents.
- Ingestion of vegetables and produce irrigated with contaminated water—As noted in the previous paragraph, crop farming and gardening are common activities near PGDP, and this land use pattern may be expanded to the WAG 28 area in the future after the industrial infrastructure is removed. Because industrial use of WAG 28 has contaminated the groundwater, plants irrigated with contaminated groundwater may accumulate these contaminants. Finally, humans may consume contaminated produce. Potential receptors for this route of exposure are rural residents.
- Ingestion of beef from cattle contaminated by consuming vegetation (pasture and concentrates) irrigated with groundwater, consuming soil (soil and waste) contaminated through irrigation or industrial use while on pasture, and drinking groundwater—During interviews, Agricultural Extension Agents for Ballard and McCracken counties indicated that small-scale cow-calf operations are common in western Kentucky (see Chap. 2 of Appendix 5 of the Methods Document). They further noted that slaughtering feeder cattle for home consumption is common. In the study area, such beef may be contaminated by incidental ingestion of soil while on pasture, by ingestion of contaminated vegetation (pasture and concentrate), and by ingestion of contaminated groundwater. Residents may eat this beef. Potential receptors for this route of exposure are rural residents.
- Ingestion of dairy products (i.e., milk) from cows contaminated by consuming vegetation (pasture or concentrates) irrigated with groundwater, consuming soil (soil and waste) contaminated through industrial use while on pasture, and drinking groundwater—During interviews, Agricultural Extension Agents for Ballard and McCracken counties noted that dairy farming still occurs in their counties (see Chap. 2 of Appendix 5 of the Methods Document). Furthermore, the agents stated that these cattle are fed stored feed and are allowed to graze on pasture. As noted previously, the soil at WAG 28 is contaminated, and the vegetation may become contaminated through incidental ingestion of soil while on pasture, ingestion of contaminated vegetation, and ingestion of contaminated groundwater. Residents could in turn consume products made from milk from these cows. Potential receptors for this route of exposure are rural residents.

- Ingestion of pork from swine fed contaminated feed and groundwater—During interviews, Agricultural Extension Agents for Ballard and McCracken counties noted that both large commercial and small hog farms exist in their counties (see Chap. 2 of Appendix 5 of the Methods Document). Furthermore, they indicated that swine on both types of farms were fed locally raised feed and, on the smaller farms, farmers consumed that farm-raised pork. Swine raised may be contaminated through ingestion of contaminated feed and groundwater, and rural residents may eat this pork. Rural residents are potential receptors for this pathway.
- Ingestion of poultry given groundwater to drink—During interviews, Agricultural Extension Agents for Ballard and McCracken counties noted that commercial broiler production occurs in their counties but not near PGDP (see Chap. 2 of Appendix 5 of the Methods Document). Home flocks for both meat and eggs were noted as being uncommon. Furthermore, they stated that broilers were fed purchased feed (not locally raised), that normal resident time in poultry houses was two months, and that commercial distribution of the product occurs; however, the agents did note that the birds are most likely watered with groundwater. Broilers may become contaminated through ingestion of contaminated groundwater. For this exposure assessment, the receptor assumed to consume the contaminated poultry is the rural resident.
- Ingestion of game contaminated by ingestion of vegetation grown in contaminated soil (soil and waste) and ingestion of groundwater—As indicated in the Methods Document and discussed earlier, hunting of game is common around the study area. Potential game species include deer, rabbits, ducks, geese, quail, and wild turkey. Each of these species may be contaminated by ingestion of contaminated vegetation, soil, or groundwater. Potential receptors for this route of exposure are recreational users.

Thirty-one routes of exposure, including those that consider biota, are possible for WAG 28; however, not all of these routes are quantified in this assessment. The routes quantified are presented in Exhibit 1.14. The models and parameters used to quantify intakes of chemicals and radionuclides for the various exposure routes are presented in Tables 1.12–1.34. To determine the representative concentrations of COPCs in biota, the models in Tables 1.35–1.38 were used. Chemical-specific parameters used in these models, such as biotransfer factors, are listed in Table 1.39. Table 1.40 presents the representative concentrations of COPCs in biota derived from these models.

1.3.3.4 Rationale for elimination of exposure points/exposure routes

As noted previously, there are several potential routes of exposure that are not quantified in this assessment. The exposure routes not quantified and the reasons they were not selected are presented in the following discussion. This information is summarized in Table 1.41. Exhibit 1.15 presents the media and analyte classes retained for analysis.

Exposure route	Table ^a
Residential user	
Ingestion of groundwater as a drinking water source (SWMUs 99a,b; 193a,b,c; and AOC 204)	Table 1.12
Dermal contact with groundwater while showering (SWMUs 99a,b; 193a,b,c; and AOC 204)	Table 1.12 Table 1.13
Inhalation of volatiles in groundwater while showering (SWMUs 99a,b; 193a,b,c; and AOC 204)	Table 1.13 Table 1.14
AOC 204)	14010 1,14
Inhalation of volatiles in groundwater during household use (SWMUs 99a,b; 193a,b,c; and AOC 204)	Table 1.15
Incidental ingestion of surface soil (SWMUs 99a, and 193a,b,c)	Table 1.16
Dermal contact with surface soil (SWMUs 99a, and 193a,b,c)	Table 1.17
Inhalation of volatiles and particulates emitted from surface soil (SWMUs 99a, and 193a,b,c)	Table 1.18
External exposure to ionizing radiation emitted from surface soil (SWMUs 99a, and 193a,b,c)	Table 1.19
Ingestion of homegrown vegetables and produce irrigated with contaminated groundwater and/or grown in contaminated soil (SWMUs 99a,b; 193a,b,c; and AOC 204)	Table 1.20
Recreational user	
Ingestion of venison ranging in study area (SWMUs 99a, and 193a,b,c)	Table 1.21
Ingestion of rabbit ranging in study area (SWMUs 99a, and 193a,b,c)	Table 1.22
Ingestion of quail ranging in study area (SWMUs 99a, and 193a,b,c)	Table 1.23
Industrial worker (current worker—soil only, future worker—soil and groundwater)	
Ingestion of groundwater as a drinking water source (SWMUs 99a,b; 193a,b,c; and AOC 204)	Table 1.24
Dermal contact with groundwater while showering (SWMUs 99a,b; 193a,b,c; and AOC 204)	Table 1.25
Inhalation of volatile compounds in groundwater while showering (SWMUs 99a,b; 193a,b,c; and AOC 204)	Table 1.26
Incidental ingestion of surface soil (SWMUs 99a, and 193a,b,c)	Table 1.27
Dermal contact with surface soil (SWMUs 99a, and 193a,b,c)	Table 1.28
Inhalation of volatile compounds and particulates emitted from surface soil (SWMUs 99a, and 193a,b,c)	Table 1.29
External exposure to ionizing radiation emitted from surface soil (SWMUs 99a, and 193a,b,c)	Table 1.30
Excavation worker	
Incidental ingestion of surface and subsurface soil (SWMUs 99a,b; 193a,b,c; 194; and AOC 204)	Table 1.31
Dermal contact with surface and subsurface soil (SWMUs 99a,b; 193a,b,c; 194; and AOC 204)	Table 1.32
Inhalation of volatile compounds and particulates emitted from surface and subsurface soil (SWMUs 99a,b; 193a,b,c; 194; and AOC 204)	Table 1.33
External exposure to ionizing radiation emitted from surface and subsurface soil (SWMUs 99a,b; 193a,b,c; 194, and AOC 204)	Table 1.34

Exhibit 1.14. Exposure routes quantified, by location, in the baseline human health risk assessment

^a Table in Appendix A in which equation and exposure parameters are displayed.

Site	Metals	Organic compounds	Radionuclides
SWMU 99a			·····
Surface soil	Yes	Yes	Yes
Subsurface soil	Yes	Yes	Yes
On-site RGA groundwater	Yes	Yes	Yes
On-site McNairy groundwater	No	Yes	Yes
Off-site RGA groundwater	Yes	Yes	Yes
Off-site McNairy groundwater	No	Yes	Yes
SWMU 99b			
Subsurface soil	Yes	Yes	Yes
On-site RGA groundwater	Yes	Yes	Yes
Off-site RGA groundwater	Yes	Yes	Yes
SWMU 193a			
Surface soil	Yes	Yes	Yes
Subsurface soil	Yes	Yes	Yes
On-site RGA groundwater	Yes	Yes	Yes
On-site McNairy groundwater	Yes	Yes	Yes
Off-site RGA groundwater	Yes	Yes	Yes
Off-site McNairy groundwater	Yes	Yes	Yes
SWMU 193b	Yes	Yes	Yes
Surface soil	Yes	Yes	Yes
Subsurface soil	Yes	Yes	Yes -
On-site RGA groundwater	No	Yes	Yes
On-site McNairy groundwater	No	Yes	Yes
Off-site RGA groundwater	No	Yes	Yes
Off-site McNairy groundwater	No	Yes	Yes
SWMU 193c			
Surface soil	Yes	No	No
Subsurface soil	Yes	Yes	Yes
On-site RGA groundwater	No	Yes	No
On-site McNairy groundwater	Yes	Yes	Yes
Off-site RGA groundwater	No	Yes	No
Off-site McNairy groundwater	Yes	Yes	Yes
SWMU 194			
Subsurface soil	Yes	Yes	Yes
AOC 204			
Subsurface soil	No	Yes	Yes
On-site RGA groundwater	No	Yes	Yes
Off-site RGA groundwater	No	Yes	Yes

Exhibit 1.15 Media and analyte class retained for analysis in this BHHRA





Three exposure routes for external exposure to ionizing radiation were not quantified in the BHHRA:

- external exposure to groundwater while showering,
- external exposure to groundwater while swimming in a privately owned pond filled with groundwater, and
- external exposure to surface water while swimming or wading in creeks or ponds.

These routes were not quantified because radionuclide slope factors for external exposure to ionizing radiation emitted by radionuclides in water are currently not available from EPA, and the information needed to quantify these routes is not sufficient.

Four routes of exposure involving contact by recreational users with contaminated soil were not quantitatively evaluated in the BERA:

- incidental ingestion of contaminated surface soil by recreational users,
- dermal contact with contaminated surface soil by recreational users,
- inhalation of volatiles and particulates emitted from surface soil by recreational users, and
- external exposure to ionizing radiation emitted from surface soil by recreational users.

The exposure assessment and previous studies indicated that repeated contact by recreational users with soil at the sites in WAG 28 would be unlikely and exposure time would be minimal.

Six routes of exposure involving contacts with media in privately owned ponds filled with groundwater were not quantitatively evaluated in the BHHRA:

- incidental ingestion of groundwater while swimming or wading in a privately owned pond filled with groundwater,
- incidental ingestion of sediment while swimming or wading in a privately owned pond filled with groundwater,
- dermal contact with groundwater while swimming or wading in a privately owned pond filled with groundwater,
- dermal contact with sediment while swimming or wading in a privately owned pond filled with groundwater,
- external exposure to sediment while swimming in a privately owned pond filled with groundwater, and
- ingestion of fish raised in privately owned ponds filled with groundwater.

These routes were not quantified because the determination was made that these pathways would bebest quantified when considering the Groundwater Operable Unit (OU) as a whole. This decision is consistent with guidance in the Methods Document.

Six routes of exposure involving contact with media in open bodies of surface water were not quantitatively evaluated in the BHHRA:

- incidental ingestion of surface water while swimming or wading in creeks or ponds,
- incidental ingestion of sediment while swimming or wading in creeks or ponds,
- dermal contact with surface water while swimming or wading in creeks or ponds,
- dermal contact with sediment while swimming or wading in creeks or ponds,
- · external exposure to sediment while swimming or wading in creeks or ponds, and
- ingestion of fish from creeks or ponds containing contaminated surface water.

These routes were not quantified because no surface waters or sediments are present at WAG 28 sites; therefore, these are incomplete pathways.

Four routes of exposure involving ingestion of livestock products by a rural resident were not quantitatively evaluated in the BHHRA:

- ingestion of beef,
- ingestion of dairy products,
- ingestion of pork, and
- ingestion of poultry and eggs.

These were not quantified because it was determined that the industrial nature of WAG 28 sites would prevent livestock production in this area in the foreseeable future (DOE 1999). In addition, the belief is that the contaminant concentrations in soil may change markedly by the time the industrial infrastructure is removed, making any calculations using current contaminant concentration meaningless; however, the reader should recognize that past assessments at PGDP have shown that dose from the livestock pathways may be significant. The exclusion of the livestock production pathways and exposure routes is consistent with guidance in the Methods Document. In the current Methods Document, the assessor is directed to quantify these pathways only in assessment of integrator units (i.e., the groundwater, surface water, and surface soil integrator (OUs). The various pathways to be evaluated in BHHRAs for the PGDP will be reevaluated when the Methods Document is revised; however, it may be appropriate to change the Methods Document so that domestic livestock pathways are always assessed for the larger OU investigations.

One route involving inhalation of volatile organic compounds (VOCs) emitted by groundwater was not quantified:

• inhalation of volatiles emitted from groundwater during irrigation.

This route was not quantified because a qualitative evaluation in Baseline Risk Assessment and Technical Investigation Report for the Northwest Dissolved Phase Plume, Paducah Gaseous Diffusion Plant, Paducah, Kentucky (DOE 1994) indicates that the volume of air in which mixing could occur outdoors resulted in potential intakes that were very small and insignificant compared to those from ingestion. Second, the determination was made that the potential importance of vapor emission would be more conservatively estimated using the indoor pathways (i.e., inhalation of vapors while using groundwater in a shower).

1.3.3.5 Development of conceptual site models

Using the information presented in the previous sections, a conceptual site model was developed for WAG 28 sites. This conceptual site model (Fig. 1.9) illustrates all sources, pathways of migration, and routes of exposure for potential receptors in WAG 28. Site-specific conceptual site models have been developed for each of the sites under investigation and are provided in Figs. 1.10-1.16.

1.3.3.6 Calculation of representative concentrations of COPCs

The representative concentrations of COPCs in each medium under current conditions for each sector were determined before the intake models were used to calculate the CDIs used in the risk calculations. The representative concentrations for COPCs in surface soil, subsurface soil, and RGA and McNairy groundwater are presented in Table 1.11. The program used to calculate these values is SAS[®] Program 3 in Appendix C of this volume.

In all cases, the representative concentration for a COPC within a medium was the lesser of the maximum detected concentration of the COPC in the medium and the 95% upper confidence limit (UCL) on the arithmetic mean concentration of the COPC in the medium (EPA 1992a, DOE 1996a). In deriving the 95% UCL concentrations for COPCs expected at WAG 28, the surrogate concentration used for samples in which the COPC was not detected was the detection limit of the COPC in the medium. For COPCs not expected at WAG 28, the surrogate concentration used when calculating the 95% UCL concentration for samples in which the COPC was not detected was one-half the detection limit of the COPC in the medium. After surrogate concentrations were assigned and before calculating the representative concentration, the form of the distribution of the concentrations for each COPC within a medium was determined. In this analysis, the two distribution forms against which data were compared were the normal distribution and the lognormal distribution (EPA 1992a). The test used for the comparisons was the W-test contained in the Univariate Procedure of SAS[®] (SAS 1990). If data were determined to be normally distributed, the following equation was used to calculate the 95% UCL (EPA 1992a, DOE 1996a).

$$95\% \text{ UCL} = \overline{X} + \left[t \times \left(\frac{s}{\sqrt{n}} \right) \right]$$

where:

95% UCL = the upper 95 percent confidence limit on the mean

X = the arithmetic mean

t = the Student's t value for the appropriate number of degrees of freedom

s = the standard deviation of the sample data

n = the number of observations

If data were determined to be lognormally distributed, the following equation was used to calculate the 95% UCL (EPA 1992a).

95% UCL =
$$e\left[\overline{X} + (0.5 \times s^2) + \left(\frac{s \times H}{\sqrt{n-1}}\right)\right]$$

where:

95% UCL = the upper 95 percent confidence limit on the mean

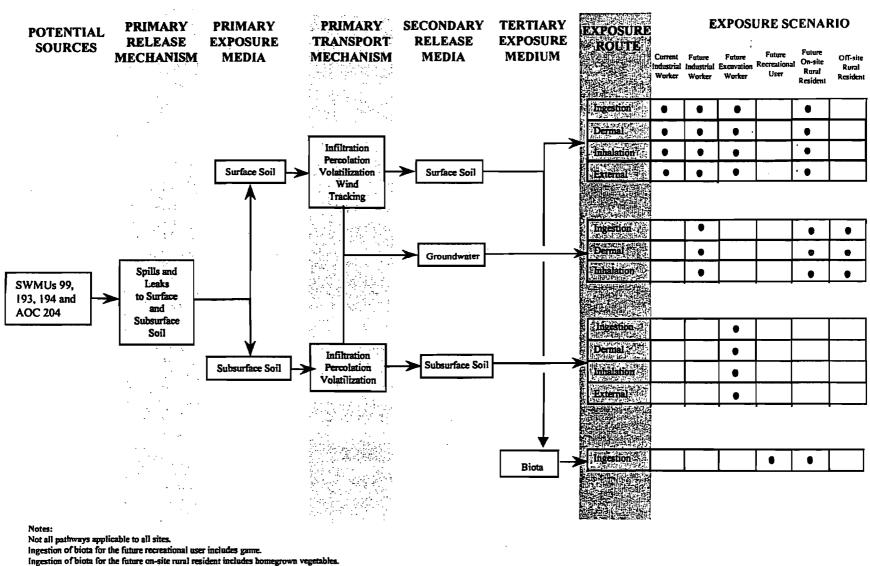
e = the base of the natural log

X = the arithmetic mean of the log-transformed values

 s^2 = the variance of the log-transformed sample data

H = the H-statistic

n = the number of observations

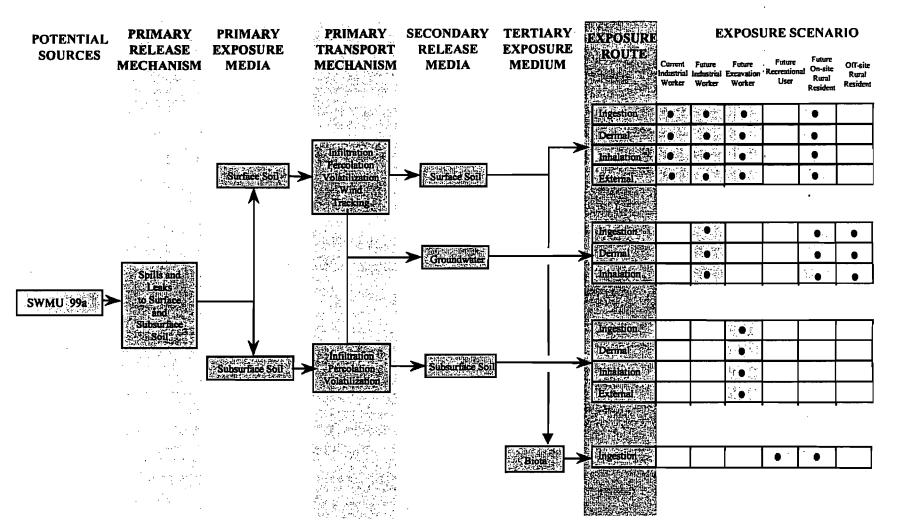


Inhalation of groundwater for the future rural resident includes inhalation of volatiles from showering and household use.

Inhalation of soil for all pathways includes inhalation of volatiles and inhalation of particulates.

Shaded boxes indicate routes and endpoints included in the WAG 28 risk assessment.

Fig. 1.9. Human health conceptual site model for sites at WAG 28



Not all pathways applicable to all sites.

Ingestion of biota for the future recreational user includes game.

Ingestion of biota for the future on-site rural resident includes homegrown vegetables.

Inhalation of groundwater for the future rural resident includes inhalation of volatiles from showering and household use.

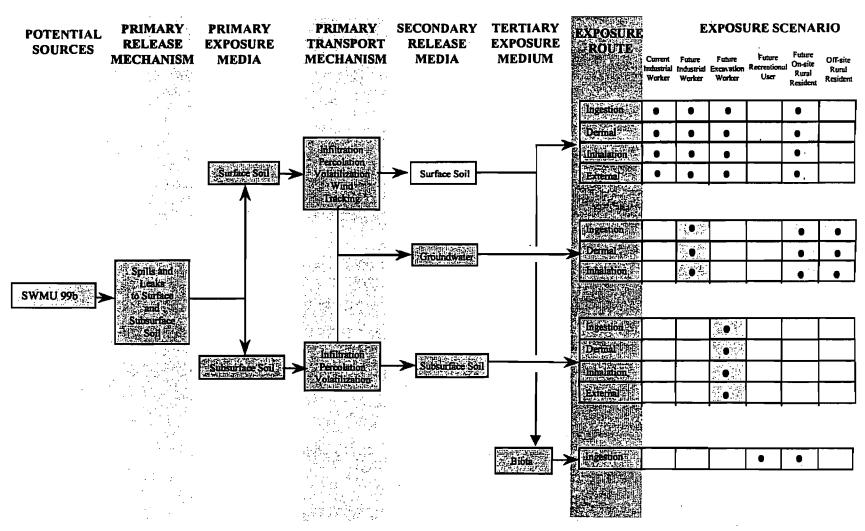
Inhalation of soil for all pathways includes inhalation of volatiles and inhalation of particulates.

Shaded boxes indicate routes and endpoints included in the WAG 28 risk assessment.

Fig. 1.10. Human health conceptual site model for SWMU 99a

.t





Not all pathways applicable to all sites.

Ingestion of biota for the future recreational user includes game.

Ingestion of biota for the future on-site rural resident includes homegrown vegetables.

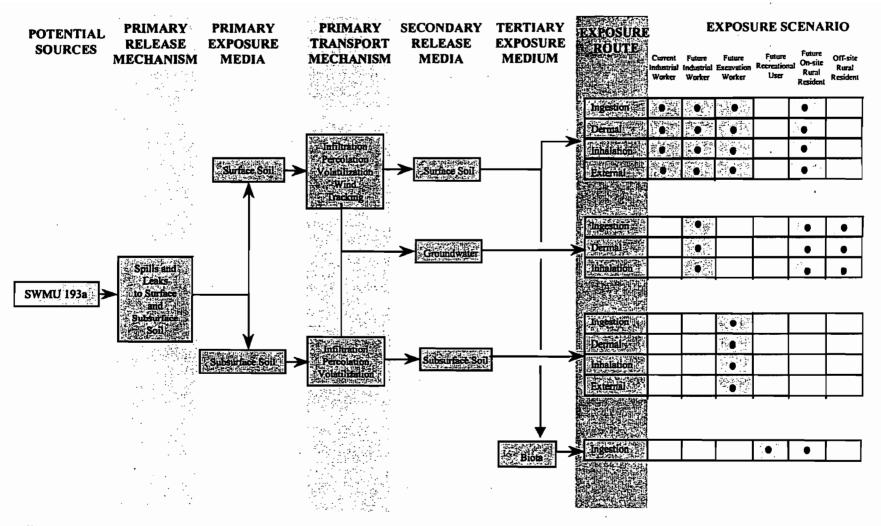
Inhalation of groundwater for the future rural resident includes inhalation of volatiles from showering and household use.

Inhalation of soil for all pathways includes inhalation of volatiles and inhalation of particulates.

Shades boxes indicate routes and endpoints included in the WAG 28 risk assessment.

Fig. 1.11. Human health conceptual site model for SWMU 99b

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Not all pathways applicable to all sites.

Ingestion of biota for the future recreational user includes game.

Ingestion of biota for the future on-site rural resident includes homegrown vegetables.

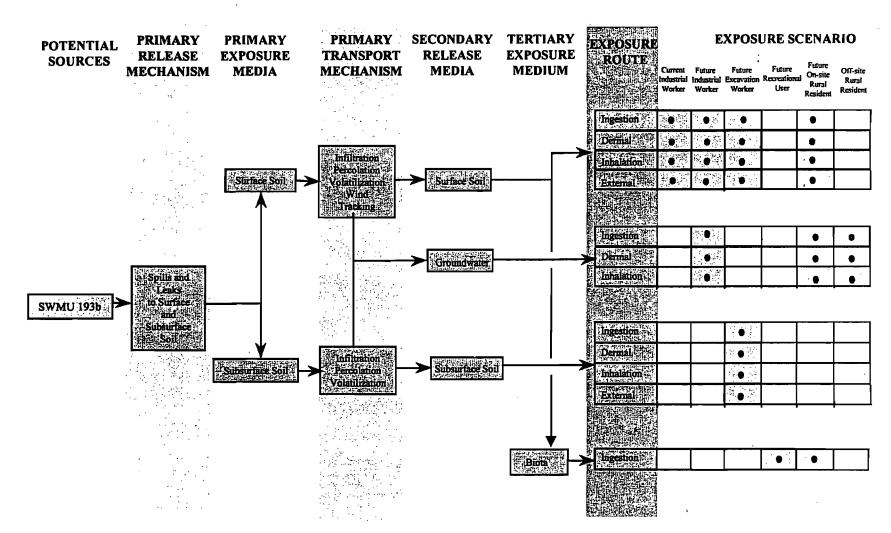
Inhalation of groundwater for the future rural resident includes inhalation of volatiles from showering and household use.

Inhalation of soil for all pathways includes inhalation of volatiles and inhalation of particulates.

Shaded boxes indicate routes and endpoints included in the WAG 28 risk assessment.



4



Not all pathways applicable to all sites.

Ingestion of biota for the future recreational user includes game.

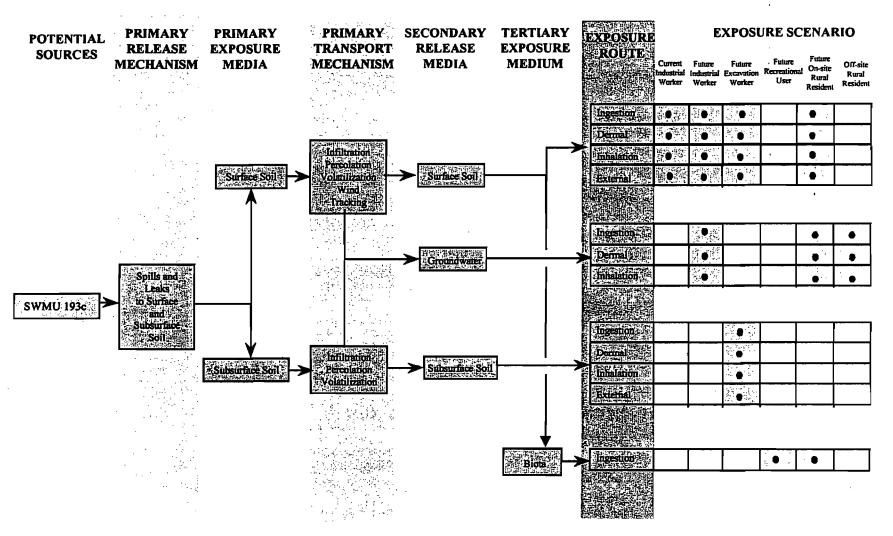
Ingestion of biota for the future on-site rural resident includes homegrown vegetables.

Inhalation of groundwater for the future rural resident includes inhalation of volatiles from showering and household use.

Inhalation of soil for all pathways includes inhalation of volatiles and inhalation of particulates.

Shaded boxes indicate routes and endpoints included in the WAG 28 risk assessment.

Fig. 1.13. Human health conceptual site model for SWMU 193b



Not all pathways applicable to all sites.

Ingestion of biota for the future recreational user includes game.

Ingestion of biota for the future on-site rural resident includes homegrown vegetables.

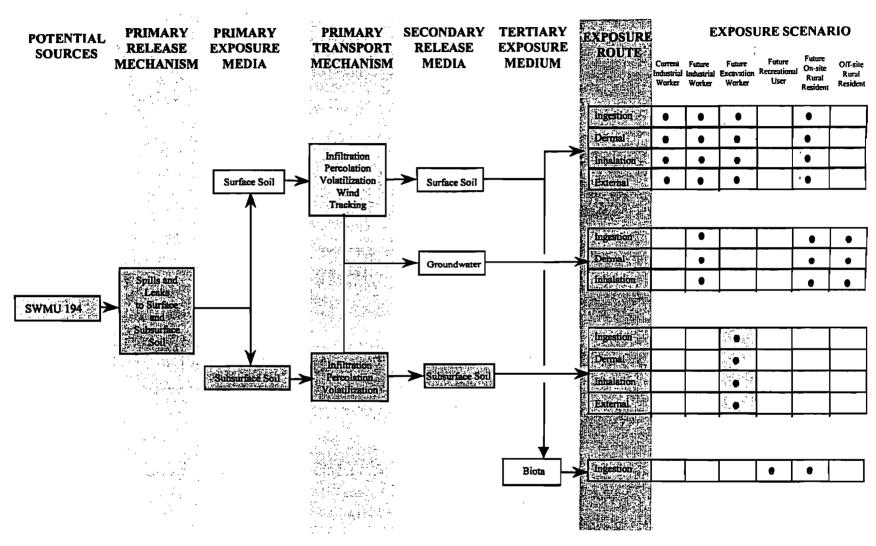
Inhalation of groundwater for the future rural resident includes inhalation of volatiles from showering and household use.

Inhalation of soil for all pathways includes inhalation of volatiles and inhalation of particulates.

Shaded boxes indicate routes and endpoints included in the WAG 28 risk assessment.

Fig. 1.14. Human health conceptual site model for SWMU 193c

3



Not all pathways applicable to all sites.

Ingestion of biota for the future recreational user includes game.

Ingestion of biota for the future on-site rural resident includes homegrown vegetables.

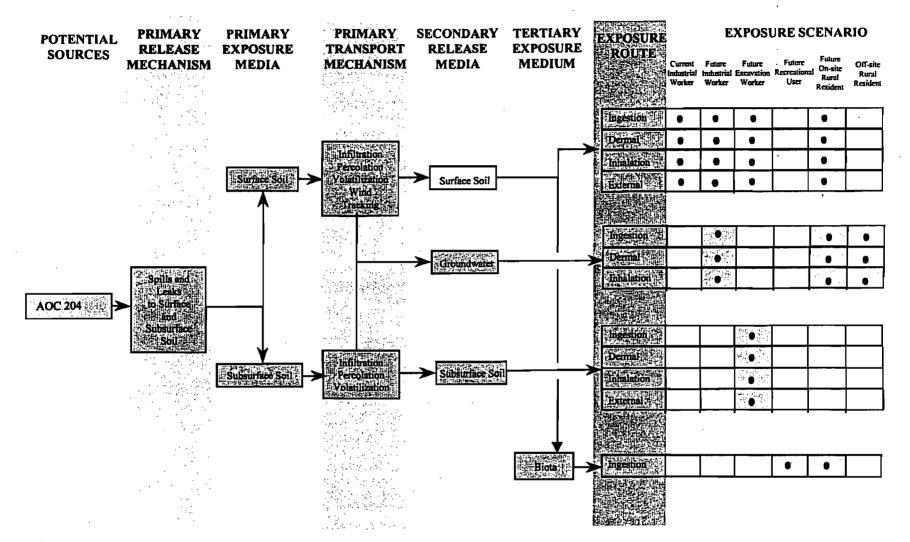
Inhalation of groundwater for the future rural resident includes inhalation of volatiles from showering and household use.

Inhalation of soil for all pathways includes inhalation of volatiles and inhalation of particulates.

Shaded boxes indicate routes and endpoints included in the WAG 28 risk assessment.

Fig. 1.15. Human health conceptual site model for SWMU 194

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Not all pathways applicable to all sites.

Ingestion of biota for the future recreational user includes game.

Ingestion of biota for the future on-site rural resident includes homegrown vegetables.

Inhalation of groundwater for the future rural resident includes inhalation of volatiles from showering and household use.

Inhalation of soil for all pathways includes inhalation of volatiles and inhalation of particulates.

Shaded boxes include routes and endpoints included in the WAG 28 risk assessment.



1-60

After the 95% UCL concentration of the COPC was determined, this value was compared to themaximum detected concentration of the COPC. As noted previously, the representative concentration of each COPC in each medium was the lessor of the maximum detected concentration and the appropriate 95% UCL concentration (RAGS).

1.3.3.7 Chronic daily intakes

Using the human exposure models, the conceptual site models, and the representative concentrations and uptake models, CDIs of each of the COPCs were determined. The SAS[®] program used to calculate the CDIs is Program 8 as described in Appendix C; these CDIs are presented in Tables 1.42–1.57. In this presentation, the CDIs used to estimate current systemic toxicity at current concentrations (i.e., noncarcinogenic effects) are presented first, and the values used to estimate current ELCR at current concentrations follow. Next, CDIs used to estimate future systemic toxicity at current concentrations are presented, and the values used to estimate future ELCR at current concentrations follow. Within each of these broad classifications, CDIs are presented by location, exposure scenario, and medium.

1.3.3.8 Summary of exposure assessment

Media available for contact at one or more of the sites are soil, groundwater, and biota. Industrial land use currently characterizes WAG 28. Current on-site receptors are industrial workers. Future potential on-site receptors are industrial workers, excavation workers, recreational users (children, teens, and adults), and rural residents (children and adults). Potential off-site human receptors are rural residents (children and adults).

Several potential routes of exposure exist. Routes quantified for the current and potential future industrial worker are ingestion of soil, dermal contact with soil, inhalation of volatile compounds and particulates emitted from soil, and external exposure to ionizing radiation emitted from soil. In addition, routes quantified for future industrial workers are ingestion of groundwater, dermal contact with groundwater, and inhalation of volatile compounds while showering.

Routes quantified for the potential future excavation worker are ingestion of surface and subsurface soil, dermal contact with surface and subsurface soil, inhalation of volatile compounds and particulates emitted from surface and subsurface soil, and external exposure to ionizing radiation emitted from surface and subsurface soil.

Routes quantified for the potential future recreational user are ingestion of venison, rabbit, and quail ranging in the study area, arising from hypothetical hunting activities. Even though game may currently visit sites outside the security fence (SWMU 194 and AOC 204), current recreational use was not assessed because surface soils (potentially contaminated medium to which game may be exposed) were not sampled at those sites. In developing the conceptual site model for WAG 28 sites, historical use suggested that the surface soils at SWMU 194 and AOC 204 were unlikely to be impacted. The conceptual model in the approved WAG 28 work plan (DOE 1998a) defined the potential sources of contamination in SWMU 194 and AOC 204 as being contained within subsurface soil (i.e., drainfields and buried debris pile). Because game ranging in these sites are unlikely to be exposed to potentially contaminated subsurface soil, the current and future recreational use scenarios were excluded from evaluation. Surface water and sediment exposure routes were not quantified, as these media do not exist on WAG 28 sites, and contaminant migration to off-site surface water is not a viable pathway. Sediment transport modeling was not undertaken to assess off-site contaminant transport via this pathway.

Routes quantified for the potential future on-site rural resident are ingestion of groundwater as a drinking water source, dermal contact with groundwater while showering, inhalation of volatiles in

groundwater while showering, inhalation of volatiles in groundwater during household use, incidental ingestion of soil, dermal contact with soil, inhalation of volatiles and particulates emitted from soil, external exposure to ionizing radiation emitted from soil, and ingestion of homegrown vegetables and produce raised in contaminated soil and/or irrigated with contaminated groundwater. Routes evaluated for the potential off-site rural resident are ingestion of groundwater as a drinking water source, dermal contact with groundwater while showering, inhalation of volatiles in groundwater while showering, and inhalation of volatiles in groundwater while showering, and inhalation of volatiles in groundwater during household use.

1.4 TOXICITY ASSESSMENT

This section summarizes the potential toxicological effects of the COPCs on exposed populations. Many of the toxicological effects summaries and nearly all of the toxicity values included in this section (except lead and a few others) obtained from information were drawn from http://risk.lsd.ornl.gov/tox/rap hp.shtml. This web site (DOE 1998c) is the Risk Assessment Information System (RAIS) prepared by the Toxicology and Risk Analysis Section (TARA) of Oak Ridge National Laboratory (ORNL) and the University of Tennessee for DOE. This site is a compilation of toxicity values taken from EPA's most recent IRIS database (EPA 1998a) and the HEAST database (EPA 1998b). For those chemicals not profiled in RAIS, a brief summary of information drawn from Agency for Toxic Substances and Disease Registry (ATSDR) or other library research sources is included in this section. Note that the last paragraph of each profile contains the toxicity values used in this BHHRA.

The toxicity information considered in the assessment of potential carcinogenic risks includes (1) a weight-of-evidence classification and (2) a slope factor. The weight-of-evidence classification qualitatively describes the likelihood that an agent is a human carcinogen, based on the available data from animal and human studies. A chemical may be placed in one of three groups to indicate its potential for carcinogenic effects: Group A, a known human carcinogen; Group B, a probable human carcinogen; and Group C, a possible human carcinogen. Group B is divided into Subgroups B1 and B2. Assignment of a chemical to Subgroup B1 indicates that the judgment that the chemical is a probable human carcinogen is based on limited human data, and assignment of a chemical to Subgroup B2 indicates that the judgment that the chemical is a probable human data are lacking or inadequate. Chemicals that cannot be classified as human carcinogens because of a lack of data are categorized in Group D, and those for which there is evidence of noncarcinogenicity in humans are categorized in Group E.

The slope factor for chemicals is defined as a plausible upperbound estimate of the probability of a response (i.e., development of cancer) per unit intake of a chemical over a lifetime (EPA 1989a). Slope factors are specific for each chemical and route of exposure. Slope factors are currently available for ingestion and inhalation pathways. The slope factors used for oral and inhalation routes of exposure for the COPCs considered in this report are shown in Table 1.58.

Toxicity values used in risk calculations also include the chronic reference dose (RfD), which is used to estimate the potential for systemic toxicity or noncarcinogenic risk. The chronic RfD is defined as "an estimate of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime" (EPA 1989a). RfD values are specific to the route of exposure. The RfDs used for oral and inhalation routes of exposure for the COPCs considered in this report are presented in Table 1.59.

For the dermal routes of exposure (i.e., dermal exposure to contaminated water during swimming or bathing or dermal contact with contaminated soil), it is necessary to consider the absorbed dose received by a receptor. This is reflected by the addition of an absorption coefficient in the equations used to calculate the CDI for these pathways. Because the CDI is expressed as an absorbed dose, it is necessary to use RfDs and slope factors that are also expressed in terms of absorbed dose. Currently, EPA has not produced lists of RfDs and slope factors based on absorbed dose. However, EPA has produced guidance concerning the estimation of absorbed dose RfDs and slope factors from administered dose RfDs and slope factors. This guidance is found in *Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual, Supplemental Guidance: Dermal Risk Assessment, Interim Guidance* (EPA 1992b) and states that to convert an administered dose slope factor to an absorbed dose slope factor, the administered dose slope factor is divided by the gastrointestinal absorption efficiency of the contaminant. Alternatively, to convert an administered dose RfD to an absorbed dose RfD, the administered dose RfD is multiplied by the gastrointestinal absorption efficiency of the contaminant. The absorbed dose slope factors and RfDs and the information used in their derivation are presented in Tables 1.60 and 1.61, respectively.

EPA has adopted a toxicity equivalency factor (TEF) methodology for carcinogenic PAHs on the Target Compound List as described in *Supplemental Guidance from RAGS: Region 4 Bulletins, Human Health Risk Assessment, Interim Guidance* (EPA 1995a). These TEFs are based on the potency of each compound relative to that of benzo(a)pyrene (BaP). Exhibit 1.16 lists the TEFs that were used to convert each PAH concentration to an equivalent concentration of BaP.

Carcinogenic PAH	TEF
Benzo(a)pyrene	1.0
Benzo(a)anthracene	0.1
Benzo(b)fluoranthene	0.1
Benzo(k)fluoranthene	0.01
Chrysene	0.001
Dibenzo(a,h)anthracene	1.0
Indeno(1,2,3-cd)pyrene	0.1

Exhibit 1.16. Toxicity equivalency factors^a (TEFs) used for carcinogenic PAHs

^a All TEFs taken from Supplemental Guidance from RAGS: Region 4 Bulletins, Human Health Risk Assessment (Interim Guidance) (EPA 1995a).

1.4.1 Inorganic Compounds

1.4.1.1 Aluminum (CAS 007429-90-5) (RAIS)

Aluminum is a silver-white flexible metal with a vast number of uses. It is poorly absorbed and efficiently eliminated; however, when absorption does occur, aluminum is distributed mainly in bone, liver, testes, kidneys, and brain (ATSDR 1990a).

Aluminum may be involved in Alzheimer's disease (dialysis dementia) and in Amyotrophic Lateral Sclerosis and Parkinsonism-Dementia Syndromes of Guam (Guam ALS-PD complex) (ATSDR 1990a, Goyer 1991). Aluminum content of brain, muscle, and bone increases in Alzheimer's patients. Neurofibrillary tangles are found in patients suffering from aluminum encephalopathy and Alzheimer's disease. Symptoms of "dialysis dementia" include speech disorders, dementia, convulsions, and myoclonus. People of Guam and Rota have an unusually high incidence of neurodegenerative diseases. The volcanic soil in the region of Guam where the high incidence of ALS-PD occurs contains high levels of aluminum and manganese. Neurological effects have also been observed in rats orally exposed to aluminum compounds.

The respiratory system appears to be the primary target following inhalation exposure to aluminum. Alveolar proteinosis has been observed in guinea pigs, rats, and hamsters exposed to aluminum powders (Gross et al. 1973). Rats and guinea pigs exposed to aluminum chlorohydrate exhibited an increase in alveolar macrophages, increased relative lung weight, and multifocal granulomatous pneumonia (Cavender et al. 1978). No decrease in reproductive capacity, hormonal abnormalities, or testicular histopathology wasobserved in male rats exposed to aluminum in drinking water for 90 days (Dixon et al. 1979).

However, male rats exposed to aluminum (as aluminum chloride) via gavage for 6 months exhibited decreased spermatozoa counts and sperm motility, and testicular histological and histochemical changes (Krasovskii et al. 1979).

Subchronic and chronic RfDs and reference concentrations (RfCs) have not been derived for aluminum.

Male rats exposed to drinking water containing aluminum (as aluminum potassium sulfate) for a lifetime exhibited increases in unspecified malignant and nonmalignant tumors (Schroeder and Mitchener 1975a), and similarly exposed female mice exhibited an increased incidence of leukemia (Schroeder and Mitchener 1975b). Rats and guinea pigs exposed via inhalation to aluminum chlorohydrate developed lung granulomas (Cavender et al. 1978), while granulomatous foci developed in similarly exposed male hamsters (Drew et al. 1974).

The EPA has not evaluated aluminum or aluminum compounds for carcinogenicity, and a weight-ofevidence classification is currently not assigned.

Subchronic and chronic RfDs and RfCs have not been officially released by EPA in IRIS or HEAST. In addition, EPA has not evaluated aluminum or its compounds for carcinogenicity, and a weight-ofevidence classification is currently not assigned. However, an oral RfD of 1.00E+0 mg/kg-day was used based on EPA 1996. The gastrointestinal absorption factor is 0.1, and the corresponding absorbed reference dose is 1.00E-01 mg/kg-day.

1.4.1.2 Ammonia (CAS 007664-41-7) (RAIS)

Ammonia is a colorless gas and a common molecule given off by living organisms and in water. It is used in making fertilizer, plastics, dyes, animal foods, glues, explosives and textiles. It is also used in the treatment and refining of metals. It may enter the environment through natural organic matter decomposition, run-off from agricultural fields or feedlots, municipal waste treatment plant discharges, oil refinery and chemical manufacturing effluents, or atmospheric fallout.

Acute toxic effects may include the death of animals, birds, or fish. Contact can cause severe skin burns and can cause severe burns of the eyes, leading to permanent damage. Breathing ammonia can irritate the mouth, nose, and throat. Higher levels may irritate the lungs, causing coughing and/or shortness of breath. Very high exposures can cause a buildup of fluid in the lungs (pulmonary edema), which can result in death.

Ammonia has not been evaluated for carcinogenicity and has not been tested for its ability to adversely affect reproduction. No RfDs are available for oral or dermal routes. The inhalation RfD value used in this BHHRA is 2.86E-2mg/kg-day and the inhalation RfC is 1.00E-1 mg/m³.

1.4.1.3 Antimony (CAS 007440-36-0) (RAIS)

Antimony is a naturally occurring metal that is used in various manufacturing processes. It exists in valence states of 3 and 5 (Budavari et al. 1989, ATSDR 1990b). Antimony is a common urban air pollutant (Beliles 1979). Exposure to antimony may be via inhalation, oral and dermal routes (ATSDR 1990b).

Antimony is sparingly absorbed following ingestion or inhalation (Felicetti et al. 1974a, Gerber et al. 1982, ATSDR 1990b). Both gastrointestinal and pulmonary absorption are a function of compound solubility. Antimony is transported in the blood, its distribution varying among species and dependent on its valence state (Felicetti et al. 1974b). Antimony is not metabolized but may bind to macromolecules and react covalently with sulfhydryl and phosphate groups (ATSDR 1990b). Excretion of antimony is primarily via the urine and feces, and is also dependent upon valence state (Cooper et al. 1968, Ludersdorf et al. 1987, ATSDR 1990b).

Acute oral exposure of humans and animals to high doses of antimony or antimony-containing compounds (antimonials) may cause gastrointestinal disorders (vomiting and diarrhea), respiratory difficulties, and death at extremely high doses (Bradley and Frederick 1941, Beliles 1979, ATSDR 1990b). Subchronic and chronic oral exposure may affect hematologic parameters (ATSDR 1990b). Long-term exposure to high doses of antimony or antimonials has been shown to adversely affect longevity in animals (Schroeder et al. 1970). Limited data suggest that prenatal and postnatal exposure of rats to antimony interferes with vasomotor responses (Marmo et al. 1987, Rossi et al. 1987).

Acute inhalation exposure of humans may cause gastrointestinal disorders (probably due to ingestion of airborne antimony) (ATSDR 1990b). Exposure of animals to high concentrations of antimony and antimonials (especially stibine gas) may result in pulmonary edema and death (Price et al. 1979). Long-term occupational exposure of humans has resulted in electrocardiac disorders, respiratory disorders, and possibly increased mortality (Renes 1953, Breiger et al. 1954). Antimony levels for these occupational exposure evaluations ranged from 2.2 to 11.98 mg Sb/m³. Based on limited data, occupational exposure of women to metallic antimony and several antimonials has reportedly caused alterations in the menstrual cycle and an increased incidence of spontaneous abortions (Belyaeva 1967). Reproductive dysfunction has been demonstrated in rats exposed to antimony trioxide (Belyaeva 1967).

No data were available indicating that dermal exposure of humans to antimony or its compounds results in adverse effects. However dermal application of high doses of antimony oxide (1,584 mg Sb/kg) resulted in the death of rabbits within one day (IBTL 1972). Eye irritation due to exposure to stibine gas and several antimony oxides has been reported for humans (Stevenson 1965, Potkonjak and Pavlovich 1983).

EPA (EPA 1998a, 1998b) calculated subchronic and chronic oral RfD based on decreased longevity and alteration of blood chemistry in rats chronically exposed to potassium antimony tartrate in the drinking water (5 ppm equivalent to 0.35 mg Sb/kg-day). An uncertainty factor of 1000 was applied: 10 for extrapolation from a lowest-observed-adverse-effect level (LOAEL) to a no-observed-adverse-effect level (NOAEL), 10 for extrapolation from animal data, and 10 for protection of sensitive populations.

The primary target organ for acute oral exposure to antimony appears to be the gastrointestinal tract (irritation, diarrhea and vomiting) and targets for long-term exposure are the blood (hematological disorders) and liver (mild hepatotoxicity) (ATSDR 1990b). Inhalation exposure to antimony affects the respiratory tract (pneumoconiosis and restrictive airway disorders), with secondary targets being the cardiovascular system (altered blood pressure and electrocardiograms) and kidneys (histological changes) (Renes 1953, Breiger et al. 1954). Only limited evidence exists for reproductive disorders due to antimony exposure (Belyaeva 1967).

Although some data indicate that long-term exposure of rats to antimony trioxide and trisulfide increased the incidence of lung tumors (Wong et al. 1979, Watt 1980, Groth et al. 1986, Bio/dynamics 1989), EPA has not evaluated antimony or antimonials for carcinogenicity and a weight-of-evidence classification is currently unavailable.

EPA has calculated subchronic and chronic oral RfDs of 4.00E-4 mg/kg-day based on decreasedlongevity and alteration of blood chemistry in rats chronically exposed to potassium antimony tartrate in drinking water. A chronic absorbed RfD of 8.00E-6 was calculated from the oral dose assuming a gastrointestinal absorption factor of 2 percent. A chronic inhalation RfD was not found. Although some data indicate that long-term exposure of rats to antimony trioxide and trisulfide increased the incidence of lung tumors, EPA has not evaluated antimony or antimonials for carcinogenicity, and a weight-ofevidence classification is currently unavailable.

1.4.1.4 Arsenic (CAS 007440-38-2) (RAIS)

The toxicity of inorganic arsenic (As) depends on its valence state (-3, +3, or +5) and also on the physical and chemical properties of the compound in which it occurs. Trivalent (As^{+3}) compounds are generally more toxic than pentavalent (As^{+5}) compounds, and the more water-soluble compounds are usually more toxic and more likely to have systemic effects than the less soluble compounds, which are more likely to cause chronic pulmonary effects if inhaled. One of the most toxic inorganic arsenic compounds is arsine gas (AsH_3) . Laboratory animals are generally less sensitive than humans to the toxic effects of inorganic arsenic. In addition, in rodents the critical effects appear to be immunosuppression and hepato-renal dysfunction, whereas in humans the skin, vascular system, and peripheral nervous system are the primary target organs.

Water-soluble inorganic arsenic compounds are absorbed through the gastrointestinal tract (> 90 percent) and lungs; distributed primarily to the liver, kidney, lung, spleen, aorta, and skin; and excreted mainly in the urine at rates as high as 80 percent in 61 hr following oral dosing (EPA 1984a, ATSDR 1989a, Crecelius 1977). Pentavalent arsenic is reduced to the trivalent form and then methylated in the liver to less toxic methylarsinic acids (ATSDR 1989a).

Symptoms of acute inorganic arsenic poisoning in humans are nausea, anorexia, vomiting, epigastric and abdominal pain, and diarrhea. Dermatitis (exfoliative erythroderma), muscle cramps, cardiac abnormalities, hepatotoxicity, bone marrow suppression and hematologic abnormalities (anemia), vascular lesions, and peripheral neuropathy (motor dysfunction, paresthesia) have also been reported (USAF 1990a, ATSDR 1989a, Franzblau and Lilis 1989, EPA 1984a, Armstrong et al. 1984, Hayes 1982a, Mizuta et al. 1956).

Oral doses as low as 20–60 g/kg-day have been reported to cause toxic effects in some individuals (ATSDR 1989a). Severe exposures can result in acute encephalopathy, congestive heart failure, stupor, convulsions, paralysis, coma, and death. The acute lethal dose to humans has been estimated to be about 0.6 mg/kg-day (ATSDR 1989a). General symptoms of chronic arsenic poisoning in humans are weakness, general debility and lassitude, loss of appetite and energy, loss of hair, hoarseness of voice, loss of weight, and mental disorders (Hindmarsh and McCurdy 1986). Primary target organs are the skin (hyperpigmentation and hyperkeratosis) (Terada et al. 1960, Tseng et al. 1968, Zaldivar 1974, Cebrian et al. 1983, Huang et al. 1985), nervous system (peripheral neuropathy) (Hindmarsh et al. 1977 and 1986, Valentine et al. 1982, Heyman et al. 1956, Mizuta et al. 1956, Tay and Seah 1975), and vascular system (Tseng et al. 1968, Borgano and Greiber 1972, Salcedo et al. 1984, Wu et al. 1989, Hansen 1990). Anemia, leukopenia, hepatomegaly, and portal hypertension have also been reported (Terada et al. 1960, Viallet et al. 1972, Morris et al. 1974, Datta 1976). In addition, possible reproductive effects include a high male to female birth ratio (Lyster 1977).

In animals, acute oral exposures can cause gastrointestinal and neurological effects (Heywood and Sortwell 1979). Oral lethal dose for 50 percent of a population (LD_{50}) values range from about 10 to 300 mg/kg (ASTDR 1989a, USAF 1990a). Low subchronic doses can result in immunosuppression (Blakely et al. 1980) and hepato-renal effects (Mahaffey et al. 1981, Brown et al. 1976, Woods and

Fowler 1977 and 1978, Fowler and Woods 1979, Fowler et al. 1979). Chronic exposures have also resulted in mild hyperkeratosis and bile duct enlargement with hyperplasia, focal necrosis, and fibrosis (Baroni et al. 1963, Byron et al. 1967). Reduction in litter size, high male/female birth ratios, and fetotoxicity without significant fetal abnormalities occur following oral exposures (Schroeder and Mitchener 1971, Hood et al. 1977, Baxley et al. 1981); however, parenteral dosing has resulted in exencephaly, encephaloceles, skeletal defects, and urogenital system abnormalities (Ferm and Carpenter 1968, Hood and Bishop 1972, Beaudoin 1974, Burk and Beaudoin 1977).

Acute inhalation exposures to inorganic arsenic can damage mucous membranes, cause rhinitis, pharyngitis and laryngitis, and result in nasal septum perforation (EPA 1984a). Chronic inhalation exposures, as occurring in the workplace, can lead to rhino-pharyno-laryngitis, tracheobronchitis, (Lundgren 1954); dermatitis, hyperpigmentation, and hyperkeratosis (Perry et al. 1948, Pinto and McGill 1953); leukopenia (Kyle and Pease 1965, Hine et al. 1977); peripheral nerve dysfunction as indicated by abnormal nerve conduction velocities (Feldman et al. 1979, Blom et al. 1985, Landau et al. 1977); and peripheral vascular disorders as indicated by Raynaud's syndrome and increased vasospastic reactivity in fingers exposed to low temperatures (Lagerkvist et al. 1986). Higher rates of cardiovascular disease have also been reported in some arsenic-exposed workers (Lee and Fraumeni 1969, Axelson et al. 1978, Wingren and Axelson 1985). Possible reproductive effects include a high frequency of spontaneous abortions and reduced birth weights (Nordström et al. 1978a, b). Arsine gas (AsH₃), at concentrations as low as 3–10 ppm for several hours, can cause toxic effects. Hemolysis, hemoglobinuria, jaundice, hemolytic anemia, and necrosis of the renal tubules have been reported in exposed workers (ACGIH 1986a, Fowler and Weissberg 1974).

Animal studies have shown that intratracheal instillation of inorganic arsenic, can cause pulmonary inflammation and hyperplasia (Webb et al. 1986, 1987), lung lesions (Pershagen et al. 1982), and immunosuppression (Hatch et al. 1985). Long-term inhalation exposures have resulted in altered conditioned reflexes and central nervous system (CNS) damage (Rozenshstein 1970). Reductions in fetal weight and in the number of live fetuses, and increases in fetal abnormalities due to retarded osteogenesis have been observed following inhalation exposures (Nagymjtenyi et al. 1985).

Subchronic and chronic RfCs for inorganic arsenic have not been derived.

Epidemiological studies have revealed an association between arsenic concentrations in drinking water and increased incidences of skin cancers (including squamous cell carcinomas and multiple basal cell carcinomas), as well as cancers of the liver, bladder, respiratory and gastrointestinal tracts (EPA 1987a, IARC 1987, Sommers and Manus 1953, Reymann et al. 1978, Dobson et al. 1965, Chen et al. 1985 and 1986). Occupational exposure studies have shown a clear correlation between exposure to arsenic and lung cancer mortality (IARC 1987, EPA 1991a). EPA (1991a) has placed inorganic arsenic in weight-of-evidence group A, human carcinogen. A drinking water unit risk of $5.0E-5(\mu g/L)^{-1}$ has been proposed (EPA 1991a) derived from drinking water unit risks for females and males that are equivalent to slope factors of 1.00E-3 ($\mu g/kg/day$)⁻¹ (females) and 2.00E-3 ($\mu g/kg/day$)⁻¹ (males) (EPA 1987a). For inhalation exposures, a unit risk of 4.30E-3 m³/µg (EPA 1991a) and a slope factor of 5.00E+1 (mg/kg-day)⁻¹ have been derived (EPA 1998b).

The RfD for chronic oral exposures, 3.00E-4 mg/kg-day, is based on a NOAEL of 8.00E-4 mg/kgday and a LOAEL of 1.40E-2 mg/kg-day for hyperpigmentation, keratosis, and possible vascular complications in a human population consuming arsenic-contaminated drinking water (EPA 1991a). Because of uncertainties in the data, EPA (1991a) states that "strong scientific arguments can be made for various values within a factor of 2 or 3 of the currently recommended RfD value." The subchronic RfD is the same as the chronic RfD, 3.00E-4 mg/kg-day (EPA 1998b). The absorbed reference dose value is 1.23E-4 mg/kg-day based on a GI absorption factor of 41 percent. EPA has placed inorganic arsenic in weight-of-evidence classification Group A, human carcinogen. Cancer slope factors for arsenic areavailable. The values used in the BHHRA are 1.50E+0, 5.00E+1, and 3.66E+0 (mg/kg-day)⁻¹ for the oral, inhalation, and dermal exposure routes, respectively. The slope factor for the dermal exposure route was calculated by assuming a gastrointestinal absorption factor of 41 percent.

1.4.1.5 Barium (CAS 007440-39-3) (RAIS)

The soluble salts of barium, an alkaline earth metal, are toxic in mammalian systems. They are absorbed rapidly from the gastrointestinal tract and are deposited in the muscles, lungs, and bone. Barium is excreted primarily in the feces.

At low doses, barium acts as a muscle stimulant and at higher doses affects the nervous system eventually leading to paralysis. Acute and subchronic oral doses of barium cause vomiting and diarrhea, followed by decreased heart rate and elevated blood pressure. Higher doses result in cardiac irregularities, weakness, tremors, anxiety, and dyspnea. A drop in serum potassium may account for some of the symptoms. Death can occur from cardiac and respiratory failure. Acute doses around 0.8 grams can be fatal to humans.

Subchronic and chronic oral or inhalation exposure primarily affects the cardiovascular system resulting in elevated blood pressure. A LOAEL of 0.51 mg barium/kg-day based on increased blood pressure was observed in chronic oral rat studies (Perry et al. 1983), whereas human studies identified a NOAEL of 0.21 mg barium/kg-day (Wones et al. 1990, Brenniman and Levy 1984). EPA used human data were used by the EPA to calculate a chronic and subchronic oral RfD of 7.00E-2 mg/kg-day (EPA 1998b, 1995b). In the Wones et al. study, human volunteers were given barium up to 10 mg/L in drinking water for 10 weeks. No clinically significant effects were observed. An epidemiological study was conducted by Brenniman and Levy in which human populations ingesting 2 to 10 mg/L of barium in drinking water were compared to a population ingesting 0 to 0.2 mg/L. No significant individual differences were seen; however, a significantly higher mortality rate from all combined cardiovascular diseases was observed with the higher barium level in the 65+ age group. The average barium concentration was 7.3 mg/L, which corresponds to a dose of 0.20 mg/kg-day. Confidence in the oral RfD is rated medium by EPA.

Subchronic and chronic inhalation exposure of human populations to barium-containing dust can result in a benign pneumoconiosis called "baritosis." This condition is often accompanied by an elevated blood pressure but does not result in a change in pulmonary function. Exposure to an air concentration of 5.2 mg barium carbonate/m³ for 4 hours/day for 6 months has been reported to result in elevated blood pressure and decreased body weight gain in rats (Tarasenko et al. 1977). Reproduction and developmental effects were also observed. Increased fetal mortality was seen after untreated females were mated with males exposed to 5.2 mg/m³ of barium carbonate. Similar results were obtained with female rats treated with 13.4 mg barium carbonate/m³. The NOAEL for developmental effects was 1.15 mg/m³ (equivalent to 0.8 mg barium/m³). EPA calculated an RfC of 5.00E-3 mg/m³ for subchronic and 5.00E-4 mg/m³ for chronic exposure based on the NOAEL for developmental effects (EPA 1998b). These effects have not been substantiated in humans or other animal systems.

EPA has not evaluated barium for evidence of human carcinogenic potential (EPA 1995b). No slope factors were used in BHHRA for barium.

Subchronic or chronic oral or inhalation exposure primarily affects the cardiovascular system, resulting in elevated blood pressure. An LOAEL of 5.10E-1 mg barium/kg-day based on increased blood pressure was observed in chronic oral rat studies, whereas human studies identified a NOAEL of 2.1E-1 mg/kg-day. EPA used human data to calculate a chronic and subchronic oral RfD of 7.00E-2 mg/kg-day.



EPA also has released an inhalation RfD of 1.43E-4 mg/kg-day. A gastrointestinal absorption factor of 7 percent was used to calculate an absorbed RfD of 4.90E-3 mg/kg-day.

1.4.1.6 Beryllium (CAS 007440-41-7) (RAIS)

Beryllium is present in the earth's crust, in emissions from coal combustion, in surface water and soil, and in house dust, food, drinking water, and cigarette smoke (EPA 1987b). However, the highest risk for exposure occurs among workers employed in beryllium manufacturing, fabricating, or reclamation industries (ATSDR 1988a). Workers encounter dusts and fumes of many different beryllium compounds; the current occupational standard for worker exposure to beryllium is 2 g/m³ during an 8-hour work shift (OSHA 1989).

Inhaled beryllium is absorbed slowly and localizes mainly in the lungs, bone, liver and kidneys (Stiefel et al. 1980, Reeves et al. 1967, Reeves and Vorwald 1967, Zorn et al. 1988, Tepper et al. 1961, Meehan and Smyth 1967). Ingested beryllium undergoes limited absorption and localizes in liver, kidneys, lungs, stomach, spleen, and the large and small intestines (Crowley et al. 1949, Furchner et al. 1973, Watanabe et al. 1985). Significant absorption of beryllium or its compounds through intact skin is unlikely because of its chemical properties (EPA 1987c). Beryllium per se is not biotransformed, but soluble salts may be converted to less soluble compounds in the lung (EPA 1987c). Most orally administered beryllium passes through the gastrointestinal tract unabsorbed and is excreted in the feces (Reeves 1965), whereas inhaled water-soluble beryllium salts are excreted mainly by the kidneys (Zom et al. 1988).

Limited data indicate that the oral toxicity of beryllium is low. No adverse effects were noted in mice given 5 ppm beryllium in drinking water in a lifetime bioassay (Schroeder and Mitchener 1975a,b). The dose (converted to 5.40E-1 mg/kg bw/day) was the NOAEL used in the calculation of the chronic oral RfD for beryllium of 5.00E-3 mg/kg-day (EPA 1991b).

In contrast, the toxicity of inhaled beryllium is well-documented. Humans inhaling "massive" doses of beryllium compounds (such as the water-soluble sulfate, fluoride, chloride, and oxide) may develop acute berylliosis (Constantinidis 1978). ATSDR (1988a) estimated that, based on existing data, the disease could develop at levels ranging from approximately 2–1000 g Be/m³. This disease usually develops shortly after exposure and is characterized by rhinitis, pharyngitis, and/or tracheobronchitis and may progress to severe pulmonary symptoms. The severity of acute beryllium toxicity correlates with exposure levels, and the disease is now rarely observed in the United States because of improved industrial hygiene (Zorn et al. 1988, Kriebel et al. 1988).

Humans inhaling beryllium may also develop chronic berylliosis which, in contrast to acute berylliosis, is highly variable in onset, is more likely to be fatal, and can develop in a few months to greater than 20 years after exposure (Constantinidis 1978, Hall et al. 1959, Kriebel et al. 1988). Chronic beryllium disease is a systemic disease that primarily affects the lungs and is characterized by the development of noncaseating granulomas. The disease most likely results from a hypersensitivity response to beryllium as evidenced by positive patch tests (Nishimura 1966) and positive lymphocyte transformation tests (Williams and Williams 1983) in exposed individuals. Granulomas may also appear in the skin, liver, spleen, lymph nodes, myocardium, skeletal muscles, kidney, bone, and salivary glands (Kriebel et al. 1988, Freiman and Hardy 1970).

Epidemiologic studies have suggested that beryllium and its compounds could be human carcinogens. In a study that covered 15 regions of the United States, Berg and Burbank (1972) found a significant correlation between cancers of the breast, bone, and uterus and the concentration and detection frequency of beryllium in drinking water. However, imperfect analytical and sampling methods used in the study prompted the EPA (1986a) to conclude that these results are not proof of cause-and-effectrelationships between cancer and beryllium in drinking water. Studies in workers exposed to beryllium, mostly via inhalation, have shown significant increases in observed over expected lung cancer incidences (Bayliss et al. 1971, Bayliss and Lainhart 1972, Bayliss and Wagoner 1977, Wagoner et al. 1980, Mancuso 1970, 1979, and 1980). EPA (1986b), in evaluating the total database for the association of lung cancer with occupational exposure to beryllium, noted several limitations but concluded that the results must be considered to be at least suggestive of a carcinogenic risk to humans. In laboratory studies, beryllium sulfate caused increased incidences of pulmonary tumors in rats and rhesus monkeys (Vorwald 1953, 1962, 1968; Vorwald et al. 1955 and 1966; Schepers et al. 1957; Reeves and Deitch 1969).

Based on sufficient evidence for animals and inadequate evidence for humans, beryllium has been placed in the EPA weight-of-evidence classification B2, probable human carcinogen (EPA 1991b). For inhalation exposure, the unit risk value is 2.40E-3 ($\mu g/m^3$)⁻¹, and the slope factor is 8.40E+0 (mg/kg-day)⁻¹ (EPA 1991b). For oral exposure, the unit risk value is 1.20E-4 ($\mu g/L$)⁻¹ and the slope factor is 4.30E+0 (mg/kg-day)⁻¹ (EPA 1991b).

A chronic oral RfDs of 2.00E-3 mg/kg-day, was used in this BHHRA. A gastrointestinal absorption factor of 1 percent was used to calculate absorbed dose RfDs of 2.00E-5 and 5.00E-5 mg/kg-day for chronic and subchronic exposures. The chronic inhalation RfD is 2.00E-2 mg/kg-day with a chronic inhalation RfC of 2.00E-2 (mg/m³). An oral, inhalation and absorbed dose slope factor of 4.30E+0, 8.40E+0, and 4.30E+2 (mg/kg-day)⁻¹ was used in this BHHRA, respectively. A gastrointestinal absorption factor of 1 percent was used to calculate an absorbed dose slope factor.

1.4.1.7 Cadmium (CAS 007440-43-9) (RAIS)

Cadmium is a naturally occurring metal that is used in various chemical forms in metallurgical and other industrial processes and in the production of pigments. Environmental exposure can occur via the diet and drinking water (ATSDR 1989b).

Cadmium is absorbed more efficiently by the lungs (30–60 percent) than by the gastrointestinal tract, the latter being a saturable process (Nordberg et al. 1985). Cadmium is transported in the blood and widely distributed in the body but accumulates primarily in the liver and kidneys (Goyer 1991). Cadmium burden (especially in the kidneys and liver) tends to increase in a linear fashion up to about 50 or 60 years of age, after which the body burden remains somewhat constant. Metabolic transformations of cadmium are limited to its binding to protein and nonprotein sulfhydryl groups and various macromolecules, such as metallothionein, which is especially important in the kidneys and liver (ATSDR 1989b). Cadmium is excreted primarily in the urine.

Acute oral exposure to 20–30 g has caused fatalities in humans. Exposure to lower amounts may cause gastrointestinal irritation, vomiting, abdominal pain, and diarrhea (ATSDR 1989b). An asymptomatic period of one-half to one hour may precede the onset of clinical signs. Oral LD₅₀ values in animals range from 63 to 1125 mg/kg, depending on the cadmium compound (USAF 1990b). Longer term exposure to cadmium primarily affects the kidneys, resulting in tubular proteinosis, although other conditions such as "itai-itai" disease may involve the skeletal system. Cadmium involvement in hypertension is not fully understood (Goyer 1991).

Inhalation exposure to cadmium and cadmium compounds may result in effects including headache, chest pains, muscular weakness, pulmonary edema, and death (USAF 1990b). The 1-minute and 10-minute lethal concentration of cadmium for humans has been estimated to be about 2500 and 250 mg/m³, respectively (Barrett et al. 1947, Beton et al. 1966). An 8-hour time-weighted-average (TWA) exposure level of 5 mg/m³ has been estimated for lethal effects of inhalation exposure to cadmium, and

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exposure to 1 mg/m³ is considered to be immediately dangerous to human health (Friberg 1950). Renal toxicity (tubular proteinosis) may also result from inhalation exposure to cadmium (Goyer 1991).

Chronic oral RfDs of 5.00E-4 and 1.00E-3 mg/kg-day have been established for cadmium exposure via drinking water and food, respectively (EPA 1998b). Both values reflect incorporation of an uncertainty factor of 10. The RfDs are based on an extensive database regarding toxicokinetics and toxicity in both human and animals, the critical effect being renal tubular proteinuria. Confidence in the RfD and database is high. Inhalation RfC values are currently not available.

The target organ for cadmium toxicity via oral exposure is the kidney (Goyer 1991). For inhalation exposure, both the lungs and kidneys are target organs for cadmium-induced toxicity (ATSDR 1989b, Goyer 1991).

There is limited evidence from epidemiologic studies for cadmium-related respiratory tract cancer (ATSDR 1989b). An inhalation unit risk of 1.80E-3 ($\mu g/m^3$)⁻¹ and an inhalation slope factor of 6.10E+0 (mg/kg-day)⁻¹ are based on respiratory tract cancer associated with occupational exposure (EPA 1998a). Based on limited evidence from multiple occupational exposure studies and adequate animal data, cadmium is placed in weight-of-evidence Group B1-probable human carcinogen.

Cadmium has two variations of toxicity values. The first variation is termed cadmium-water. An oral RfD of 5.00E-4 mg/kg-day was used in this BHHRA for cadmium-water. A gastrointestinal absorption factor of 1 percent was used to calculate an absorbed dose RfD of 5.00E-6 mg/kg-day for cadmium-water. No inhalation RfD is available; however, an inhalation reference dose of 5.71E-5 mg/kg-day was used based on EPA (1998b) for both diet and water exposures.

The second variation is termed cadmium-diet. Cadmium-diet is used for exposure to soil and food. An oral RfD of 1.00E-3 mg/kg-day was used in this BHHRA for cadmium-diet. A gastrointestinal absorption factor of 1 percent was used to calculate an absorbed dose RfD of 1.00E-5 mg/kg-day for cadmium-diet. The same inhalation RfD was used for cadmium-diet as for cadmium-water. The only slope factor available for cadmium was for inhalation, 6.10E+0 (mg/kg-day)⁻¹.

1.4.1.8 Chromium III (CAS 016065-83-1) and Chromium VI (CAS 018540-29-9) (RAIS)

Elemental chromium (Cr) does not occur in nature but is present in ores, primarily chromite (FeOCr₂O₃) (Hamilton and Wetterhahn 1988). Only two of the several oxidation states of chromium, Cr(III) and Cr(VI), are reviewed in this report based on their predominance and stability in the ambient environment and their toxicity in humans and animals.

Chromium plays a role in glucose and cholesterol metabolism and is thus an essential element to man and animals (Schroeder et al. 1962). Nonoccupational exposure to the metal occurs via the ingestion of chromium-containing food and water, whereas occupational exposure occurs via inhalation (Langard 1982, Pedersen 1982). Workers in the chromate industry have been exposed to estimated chromium levels of $10-50 \text{ g/m}^3$ for Cr(III) and $5-1000 \text{ g/m}^3$ for Cr(VI); however, improvements in the newer chrome-plating plants have reduced the Cr(VI) concentrations 10- to 40-fold (Stern 1982).

Chromium(III) is poorly absorbed, regardless of the route of exposure, whereas chromium(VI) is more readily absorbed (Hamilton and Wetterhahn 1988). Humans and animals localize chromium in the lung, liver, kidney, spleen, adrenals, plasma, bone marrow, and red blood cells (Langard 1982, ATSDR 1989c, Bragt and van Dura 1983, Hamilton and Wetterhahn 1988). There is no evidence that chromium is biotransformed, but Cr(VI) does undergo enzymatic reduction, resulting in the formation of reactive intermediates and Cr(III) (Hamilton and Wetterhahn 1988). The main routes for the excretion of chromium are via the kidneys/urine and the bile/feces (Guthrie 1982, Langard 1982).

Animal studies show that Cr(VI) is generally more toxic than Cr(III), but neither oxidation state is very toxic by the oral route. In long-term studies, rats were not adversely affected by approximately 1.90E+0 g/kg-day of chromic oxide [Cr(III)] (diet), 2.40E+0 mg/kg-day of Cr(III) as chromic chloride (drinking water), or 2.40E+0 mg/kg-day of Cr(VI) as potassium dichromate (drinking water) (Ivankovic and Preussmann 1975, MacKenzie et al. 1958).

The respiratory and dermal toxicity of chromium are well-documented. Workers exposed to chromium have developed nasal irritation (at $< 0.01 \text{ mg/m}^3$, acute exposure), nasal ulcers, perforation of the nasal septum (at approximately 2 g/m³, subchronic or chronic exposure) (Hamilton and Wetterhahn 1988, ATSDR 1989c, Lindberg and Hedenstierna 1983) and hypersensitivity reactions and "chrome holes" of the skin (Pedersen 1982, Burrows 1983, USAF 1990c). Among the general population, contact dermatitis has been associated with the use of bleaches and detergents (Love 1983).

Compounds of both Cr(VI) and Cr(III) have induced developmental effects in experimental animals that include neural tube defects, malformations, and fetal deaths (Iijima et al. 1983, Danielsson et al. 1982, Matsumoto et al. 1976).

The subchronic and chronic oral RfD value is 1.00E+0 mg/kg-day for Cr(III). The subchronic and chronic oral RfD values for Cr (VI) are 2.00E-2 and 5.00E-3 mg/kg-day, respectively (EPA 1991c, 1998b). The subchronic and chronic oral RfD values for Cr(VI) and Cr(III) are derived from NOAELs of 1.47 g/kg-day Cr(III) and 25 ppm of potassium dichromate (Cr[VI]) in drinking water, respectively (Ivankovic and Preussmann 1975, MacKenzie et al. 1958). The inhalation RfC values for both Cr(III) and Cr(VI) are currently under review by an EPA workgroup.

The inhalation of chromium compounds has been associated with the development of cancer in workers in the chromate industry. The relative risk for developing lung cancer has been calculated to be as much as 30 times that of controls (Hayes 1982, Leonard and Lauwerys 1980, Langard 1983). There is also evidence for an increased risk of developing nasal, pharyngeal, and gastrointestinal carcinomas (Hamilton and Wetterhahn 1988). Quantitative epidemiological data were obtained by Mancuso and Hueper (1951), who observed an increase in deaths (18.2 percent; p < 0.01) from respiratory cancer among chromate workers compared with 1.2 percent deaths among controls. In a follow-up study, conducted when more than 50 percent of the cohort had died, the observed incidence for lung cancer deaths had increased to approximately 60 percent (Mancuso 1975). The workers were exposed to $1-8 \text{ mg/m}^3$ /year total chromium. Mancuso (1975) observed a dose response for total chromium exposure and attributed the lung cancer deaths to exposure to insoluble [Cr(III)], soluble [Cr(VI)], and total chromium. The results of inhalation studies in animals have been equivocal or negative (Nettesheim et al. 1971, Glaser et al. 1986, Baetjer et al. 1959, Steffee and Baetjer 1965).

Based on sufficient evidence for humans and animals, Cr(VI) has been placed in the EPA weight-ofevidence classification A, human carcinogen (EPA 1991c). For inhalation exposure, the unit risk value is $1.20E-2 (\mu g/m^3)^{-1}$ and the slope factor is $4.10E+01 (mg/kg-day)^{-1}$ (EPA 1991c).

For estimation of risk from exposure to chromium, the toxicity values associated with Cr(VI) were used. Cr(III) values were not used because most analytical results were not specific for this ionic species. The uncertainty in using Cr(III) versus Cr(VI) in the risk assessment is discussed in Sect. 1.6.

An inhalation cancer slope factor for chromium of 4.10E+01 (mg/kg-day)⁻¹ was used in this BHHRA. No slope factors were available for oral and dermal routes. The oral and dermal RfDs used are

3.00E-3, and 6.00E-5 mg/kg-day, respectively. The dermal route RfD is based on the oral RfD and a gastrointestinal absorption factor of 2 percent. The inhalation RfD factor is 2.86E-5 mg/kg-day.

1.4.1.9 Cobalt (CAS 007440-48-4) (ATSDR)

Cobalt is a steel-gray, shiny, hard metal that occurs naturally in soil. Cobalt and cobalt-containing compounds are used widely in industry, and cobalt undergoes environmental redistribution through industrial processes, such as the burning of coal and oil and exhaust from cars. Cobalt is a component of Vitamin B_{12} .

Acute exposure to cobalt salts can lead to histological changes in the kidneys, lungs, liver, and adrenal glands. Cobalt is a sensitizer, and many occurrences of cobalt hypersensitivity have been documented in occupationally exposed individuals. The effects observed among cobalt-exposed workers include allergic dermatitis, eczema, and changes in white blood cells. Chronic inhalation exposure has produced hard-metal pneumoconiosis and other lung diseases in humans, as well as lung damage in experimental animals. Some evidence in humans suggests an association between high levels of cobalt exposure and cardiomyopathy (ATSDR 1990c).

When cobalt metal was tested in vitro, a weak mutagenic response was noted, probably due to cobalt complexes that formed. Cobalt has been reported to be genotoxic in other test systems but antimutagenic in bacteria. Adverse teratogenic and reproductive effects have been observed experimentally in animals; however, teratogenic or reproductive effects have not been reported in humans following oral, dermal, or inhalation exposure to cobalt (Angerer et al. 1988, ATSDR 1990c).

An oral RfD of 6.00E-2 mg/kg-day was used in this BHHRA. A gastrointestinal absorption factor of 8 percent was used to calculate an absorbed dose RfD of 4.80E-2 mg/kg-day. No inhalation RfD is used in this BHHRA. No slope factors were available.

1.4.1.10 Copper (CAS 007440-50-8)

Copper occurs naturally in elemental form and as a component of many minerals. Because of its high electrical and thermal conductivity, it is widely used in the manufacture of electrical equipment. Common copper salts, such as sulfate, carbonate, cyanide, oxide, and sulfide, are used as fungicides, as components of ceramics and pyrotechnics, for electroplating, and for numerous other industrial applications (ACGIH 1986b). Copper can be absorbed by the oral, inhalation, and dermal routes of exposure. It is an essential nutrient that is normally present in a wide variety of tissues (ATSDR 1990d, EPA 1987d).

In humans, ingestion of gram quantities of copper salts may cause gastrointestinal, hepatic, and renal effects with symptoms such as severe abdominal pain, vomiting, diarrhea, hemolysis, hepatic necrosis, hematuria, proteinuria, hypotension, tachycardia, convulsions, coma, and death (USAF 1990d). Gastrointestinal disturbances and liver toxicity have also resulted from long-term exposure to drinking water containing 2.2–7.8 mg Cu/L (Mueller-Hoecker et al. 1988, Spitalny et al. 1984). The chronic toxicity of copper has been characterized in patients with Wilson's disease, a genetic disorder causing copper accumulation in tissues. The clinical manifestations of Wilson's disease include cirrhosis of the liver, hemolytic anemia, neurologic abnormalities, and corneal opacities (Goyer 1991, ATSDR 1990d, EPA 1987d). In animal studies, oral exposure to copper caused hepatic and renal accumulation of copper, liver and kidney necrosis at doses of greater than or equal to 100 mg/kg-day and hematological effects at doses of 40 mg/kg-day (EPA 1986c; Haywood 1985, 1980; Rana and Kumar 1978; Gopinath et al. 1974; Kline et al. 1971).

Acute inhalation exposure to copper dust or fumes at concentrations of 0.075-0.12 mg Cu/m³ maycause metal fume fever with symptoms such as cough, chills, and muscle ache (USAF 1990d). Among the reported effects in workers exposed to copper dust are gastrointestinal disturbances, headache, vertigo, drowsiness, and hepatomegaly (Suciu et al. 1981). Vineyard workers chronically exposed to Bordeaux mixture (copper sulfate and lime) exhibit degenerative changes of the lungs and liver. Dermal exposure to copper may cause contact dermatitis in some individuals (ATSDR 1990d).

Oral or intravenous administration of copper sulfate increased fetal mortality and developmental abnormalities in experimental animals (Lecyk 1980, Ferm and Hanlon 1974). Evidence also indicates that copper compounds are spermicidal (ATSDR 1990d, Battersby et al. 1982).

An RfD for elemental copper is not available (EPA 1998b). However, EPA established an action level of 1300 μ g/L for drinking water (56 *Federal Register* 26460, June 7, 1991). Data were insufficient to derive an RfC for copper.

No suitable bioassays or epidemiological studies are available to assess the carcinogenicity of copper. Therefore, EPA (1991d) has placed copper in weight-of-evidence Group D, not classifiable as to human carcinogenicity.

No RfDs or slope factors are available for copper. However, a provisional oral RfD of 4.00E-2 mg/kg-day was used to estimate noncarcinogenic risk. This oral value was used with a gastrointestinal absorption factor of 30 percent to calculate an absorbed RfD of 1.20E-2.

1.4.1.11 Fluorine (Soluble Fluoride) (CAS 007782-41-4)

Fluoride is the soluble form of fluorine and is a naturally occurring compound. In surface water, levels of naturally occurring fluoride usually range from 0.01 to 1.5 mg/l, and the level of fluoride in soils is usually between 200 and 300 mg/kg. Fluorides are commonly added to municipal water supplies and toothpaste to aid in the prevention of dental cavities. Fluoride is also used to help make steel, chemicals, pesticides, ceramics, lubricants, and plastics.

Dermal exposure to fluorides (in the form of fluoride or hydrogen fluoride) may produce severe irritation. Teeth mottling occurs in children chronically exposed to fluoride at doses above 2 mg/kg during the development of their deciduous and permanent teeth. The skeletal system is the primary target system for intermediate and chronic exposures because of fluoride deposition. Humans chronically exposed to 2.4 to 6.0 mg/m³ had serious bone damage throughout their bodies. Exposure to high levels of fluoride may also cause disturbances in calcium metabolism which is necessary for the functional integrity of the voluntary and autonomic nervous system. Cardiac arrhythmias have been observed in fluoride poisonings.

The optimal level for water fluoridation is 0.7-1.2 mg/l, with primary and secondary contaminant levels of 4 and 2 mg/l, respectively (ATSDR 1991).

An oral cancer slope factor for fluoride is not available; therefore, neither the oral route nor the dermal route can be quantitatively assessed for carcinogenicity. In addition there is no inhalation cancer slope factor. The oral RfD used in the BHHRA is 6.00E-2 mg/kg-day. The dermal route RfD based on the oral RfD and a gastrointestinal absorption factor of 97 percent permits a value for the absorbed oral RfD of 5.80E-2 mg/kg-day (ATSDR 1991) to be derived.



1.4.1.12 Iron (CAS 007439-89-6)

Iron is one of the most abundant metals in the environment and is used in many industrial processes. It is an essential element in the human diet. More than 80 percent of the iron present in the body is involved in the support of red blood cell production. In addition, it is also an essential component of myoglobin and various enzymes. Iron deficiency is the most common cause of anemia (Goodman and Gilman 1985). Exposure to excessive levels of iron may cause gastrointestinal damage and dysfunction and enlargement of the liver and pancreas (Goodman and Gilman 1985).

No cancer slope factors for iron were found. Therefore, carcinogenicity due to exposure to iron is not included in the BHHRA. The oral RfD used in the BHHRA is 3.00E-1 mg/kg-day and is taken from RAIS. The dermal route RfD used in the BHHRA, based on the oral RfD and a gastrointestinal absorption factor of 15 percent, is 4.50E-2 mg/kg-day. An inhalation RfD for iron is not available, and based on the localized effects on the gastrointestinal tract as discussed previously, it would not be appropriate to extrapolate an inhalation RfD from the oral RfD.

1.4.1.13 Lead (CAS 007439-92-1) (RAIS)

Lead occurs naturally as a sulfide in galena. It is a soft, bluish-white, silvery-gray, malleable metal with a melting point of 327.5C. Elemental lead reacts with hot boiling acids and is attacked by pure water. The solubility of lead salts in water varies from insoluble to soluble, depending on the type of salt (IARC 1980, Goyer 1988, Budavari et al. 1989).

Lead is a natural element that is persistent in water and soil. Most of the lead in environmental media is of anthropogenic sources. The mean concentration is $3.9 \ \mu g/L$ in surface water and $0.005 \ \mu g/L$ in sea water. River sediments contain about 20,000 $\ \mu g/g$ and coastal sediments about 100,000 $\ \mu g/g$. Soil content varies with the location, ranging up to 30 $\ \mu g/g$ in rural areas, 3000 $\ \mu g/g$ in urban areas, and 20,000 $\ \mu g/g$ near point sources. Human exposure occurs primarily through diet, air, drinking water, and ingestion of dirt and paint chips (EPA 1989b,c and ATSDR 1993a).

The efficiency of lead absorption depends on the route of exposure, age, and nutritional status. Adult humans absorb about 10-15 percent of ingested lead, whereas children may absorb up to 50 percent, depending on whether lead is in the diet, dirt, or paint chips. More than 90 percent of lead particles deposited in the respiratory tract are absorbed into systemic circulation. Inorganic lead is not efficiently absorbed through the skin; consequently, this route does not contribute considerably to the total body lead burden (EPA 1986d).

Lead absorbed into the body is distributed to three major compartments: blood, soft tissue, and bone. The largest compartment is the bone, which contains about 95 percent of the total body lead burden in adults and about 73 percent in children. The half-life of bone lead is more than 20 years. The concentration of blood lead changes rapidly with exposure, and its half-life of only 25-28 days is considerably shorter than that of bone lead. Blood lead is in equilibrium with lead in bone and soft tissue. The soft tissues that take up lead are liver, kidneys, brain, and muscle. Lead is not metabolized in the body, but it may be conjugated with glutathione and excreted primarily in the urine (EPA 1986d, 1986e, ATSDR 1993a). Exposure to lead is evidenced by elevated blood lead levels.

The systemic toxic effects of lead in humans have been well-documented by the EPA (EPA 1986d-h, 1989c, 1990a) and ATSDR (1993a), who extensively reviewed and evaluated data reported in the literature up to 1991. The evidence shows that lead is a multitargeted toxicant, causing effects in the gastrointestinal tract, hematopoietic system, cardiovascular system, central and peripheral nervous systems, kidneys, immune system, and reproductive system. Overt symptoms of subencephalopathic CNS

effects and peripheral nerve damage occur at blood lead levels of 40–60 μ g/dL, and nonovert symptoms, such as peripheral nerve dysfunction, occur at levels of 30–50 μ g/dL in adults; no clear threshold is evident. Cognitive and neuropsychological deficits are not usually the focus of studies in adults, but there is some evidence of neuropsychological impairment (Ehle and McKee 1990) and cognitive deficits in lead workers with blood levels of 41–80 μ g/dL (Stollery et al. 1991).

Although similar effects occur in adults and children, children are more sensitive to lead exposure than are adults. Irreversible brain damage occurs at blood lead levels greater than or equal to $100 \ \mu g/dL$ in adults and at $80-100 \ \mu g/dL$ in children; death can occur at the same blood levels in children. Children who survive these high levels of exposure suffer permanent severe mental retardation.

As discussed previously, neuropsychological impairment and cognitive (IQ) deficits are sensitive indicators of lead exposure; both neuropsychological impairment and IQ deficits have been the subject of cross-sectional and longitudinal studies in children. One of the early studies reported IQ score deficits of four points at blood lead levels of $30-50 \mu g/dL$ and one to two points at levels of $15-30 \mu g/dL$ among 75 black children of low socioeconomic status (Schroeder and Hawk 1986).

Very detailed longitudinal studies have been conducted on children (starting at the time of birth) living in Port Pirie, Australia (Vimpani et al. 1985,1989; McMichael et al. 1988; Wigg et al. 1988; Baghurst et al. 1987, 1992), Cincinnati, Ohio (Dietrich et al. 1986, 1991, 1992, 1993), and Boston, Massachusetts (Bellinger et al. 1984, 1987a, 1987b, 1990, 1992; Stiles and Bellinger 1993). Various measures of cognitive performance have been assessed in these children. Studies of the Port Pirie children up to 7 years of age revealed IQ deficits in 2-year-old children of 1.6 points for each 10- μ g/dL increase in blood lead, deficits of 7.2 points in 4-year-old children, and deficits of 4.4 to 5.3 points in 7-year-old children, 5 years or younger, who lived in the Cincinnati, Ohio, area. In 6.5-year-old children, performance IQ was reduced by 7 points in children whose lifetime blood level exceeded 20 μ g/dL.

Children living in the Boston, Massachusetts, area have been studied up to the age of 10 years. Cognitive performance scores were negatively correlated with blood lead in the younger children in the high lead group (greater than or equal to $10 \ \mu g/dL$), and improvements were noted in some children at 57 months as their blood lead levels became lower. However, measures of IQ and academic performance in 10-year-old children showed a 5.8-point deficit in IQ and an 8.9-point deficit in academic performance as blood lead increased by 10 $\mu g/dL$ within the range of 1–25 $\mu g/dL$. Because of the large database on subclinical neurotoxic effects of lead in children, only a few of the studies have been included. However, EPA (EPA 1986e, 1990a) concluded that there is no clear threshold for neurotoxic effects of lead in children.

In adults, the cardiovascular system is a very sensitive target for lead. Hypertension (elevated blood pressure) is linked to lead exposure in occupationally exposed subjects and in the general population. Three large population-based studies have been conducted to study the relationship between blood lead levels and high blood pressure. The British Regional Heart Study (BRHS) (Pocock et al. 1984), the NHANES II study (Harlan et al. 1985, Pirkle et al. 1985, Landis and Flegal 1988, Schwartz 1991, EPA 1990a), and Welsh Heart Programme (Ellwood et al. 1988a, 1988b) comprise the major studies for the general population. The BRHS study showed that systolic pressure greater than 160 mm Hg and diastolic pressure greater than 100 mm Hg were associated with blood lead levels greater than 37 μ g/dL (Pocock et al. 1984). An analysis of 9933 subjects in the NHANES study showed positive correlations between blood pressure and blood lead among 12–74-year-old males but not females (Harlan et al. 1985, Landis and Flegal et al. 1988), 40-59-year-old white males with blood levels ranging from 7–34 μ g/dL (Pirkle et al. 1985), and males and females greater than 20 years old (Schwartz 1991). In addition, left ventricular

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hypertrophy was also positively associated with blood lead (Schwartz 1991). The Welsh study did not show an association among men and women with blood lead of 12.4 and 9.6 μ g/dL, respectively (Ellwood et al. 1988a, 1988b). Other smaller studies showed both positive and negative results. The EPA (EPA 1990a) concluded that increased blood pressure is positively correlated with blood lead levels in middle-aged men, possibly at concentrations as low as 7 μ g/dL. In addition, the EPA estimated that systolic pressure is increased by 1.5–3.0 mm Hg in males and 1.0–2.0 mm Hg in females for every doubling of blood lead concentration.

The hematopoietic system is a target for lead as evidenced by frank anemia occurring at blood lead levels of 80 μ g/dL in adults and 70 μ g/dL in children. The anemia is due primarily to reduced heme synthesis, which is observed in adults having blood levels of 50 μ g/dL and in children having blood levels of 40 μ g/dL. Reduced heme synthesis is caused by inhibition of key enzymes involved in the synthesis of heme. Inhibition of erythrocyte -aminolevulinic acid dehydrase (ALAD) activity (catalyzes formation of porphobilinogen from -aminolevulinic acid) has been detected in adults and children having blood levels of less than 10 μ g/dL. ALAD activity is the most sensitive measure of lead exposure, but erythrocyte zinc protoporphyrin is the most reliable indicator of lead exposure because it is a measure of the toxicologically active fraction of bone lead. The activity of another erythrocyte enzyme, pyrimidine-5-nucleotidase, is also inhibited by lead exposure. Inhibition has been observed at levels below 5 μ g/dL; no clear threshold is evident.

Other organs or systems affected by exposure to lead are the kidneys, immune system, reproductive system, gastrointestinal tract, and liver. These effects usually occur at high blood levels, or the blood levels at which they occur have not been sufficiently documented.

The EPA has not developed an RfD for lead because it appears that lead is a nonthreshold toxicant, and it is not appropriate to develop RfDs for these types of toxicants. Instead the EPA has developed the Integrated Exposure Uptake Biokenetic Model to estimate the percentage of the population of children up to 6 years of age with blood lead levels above a critical value, $10 \mu g/dL$. The model determines the contribution of lead intake from multimedia sources (diet, soil and dirt, air, and drinking water) on the concentration of lead in the blood. Site-specific concentrations of lead in various media are used when available; otherwise default values are assumed. The EPA has established a screening level of 400 ppm ($\mu g/g$) for lead in soil (EPA 1994a).

Inorganic lead and lead compounds have been evaluated for carcinogenicity by the EPA (EPA 1989b, c, 1993a). The data from human studies are inadequate for evaluating the potential carcinogenicity of lead. Data from animal studies, however, are sufficient based on numerous studies showing that lead induces renal tumors in experimental animals. A few studies have shown evidence for induction of tumors at other sites (cerebral gliomas; testicular, adrenal, prostate, pituitary, and thyroid tumors). A slope factor was not derived for inorganic lead or lead compounds.

As noted previously, neither slope factors nor RfDs for lead are available from the EPA. However, KYDEP has provided provisional RfDs for oral, dermal, and inhalation toxicity; they are 1.00E-7, 1.50E-8, and 2.86E-4 mg/kg-day, respectively. A gastrointestinal absorption factor of 15 percent can be derived from the oral and dermal RfDs. In addition, three classes of benchmarks are available and are used in this BHHRA. These are the benchmarks applied by the Integrated Exposure Uptake Biokinetic Model (10 μ g/dL); the EPA screening values of 400 mg/kg and 15 μ g/l for soil and water, respectively [Office of Solid Waste and Emergency Response (OSWER) Dir. No. 9344.4-12]; and the Commonwealth of Kentucky screening values of 20 mg/kg and 4 μ g/l for soil and water, respectively (KDEP 1995).

1.4.1.14 Lithium (CAS 007439-93-2)

Lithium is an alkali metal similar to magnesium and sodium in its properties (Birch 1988, Arena 1986) and has a molecular weight of 6.941 (Beliles 1994). It does not occur in nature in its free form but is found in minerals such as spodumene, petalite, and eucryptite (Beliles 1994). Lithium compounds are found in natural waters and in some foods. The average dietary intake is estimated to be about 2 mg per day (Beliles 1994).

Inorganic salts or oxides of lithium have many uses. Lithium carbonate is used extensively as a therapeutic agent in the treatment of manic depressive affective disorders (Ellenhorn and Barceloux 1988). Elemental lithium is a component of metal alloys; lithium hydride is used as a nuclear reactor coolant. Lithium hydroxide is used in alkaline storage batteries; lithium carbonate and lithium borate are used in the ceramic industry; and lithium chloride and fluoride are used in welding and brazing fluxes (Beliles 1994). Lithium forms covalent bonds in organometallic compounds such as lithium stearate. Organo-lithium compounds are used as multipurpose greases, particularly in the automotive industry (Beliles 1994).

Most common inorganic lithium compounds are water soluble to some extent: i.e., chloride, 454 g/L; carbonate, 13.3 g/L; hydroxide, 223 g/L; oxide, 66.7 g/L (Beliles 1994). Lithium hydride reacts with water to form a very basic solution of lithium hydroxide.

Soluble lithium compounds are readily absorbed through the gastrointestinal tract but not the skin; distribution is rapid to the liver and kidneys but slower to other organ systems (Jaeger et al. 1985). Lithium crosses the human placenta (ACGIH 1991) and can also be taken up by infants through breast milk. Lithium is not metabolized and is excreted primarily in the urine.

The oral toxicity of most lithium compounds is relatively low; oral LD₅₀ values for several compounds and animal species range from 422–1165 mg/kg. Case histories described by Gosselin et al. (1984) indicate that doses of 12–60 g (171–857 mg/kg-day for a 70 kg person) can result in coma, respiratory and cardiac complications, and death in humans. A single oral dose of 40 mg/kg produced toxic lithium blood levels in a patient with a history of prior lithium use (Marcus 1980). In contrast, for chronic therapeutic use, the standard dose of lithium carbonate is 1-2 g/day (14–28 mg/kg-day).

Signs and symptoms of lithium toxicity include anorexia; nausea; diarrhea; alopecia; weight gain; thirst; pretibial edema (sodium retention); polyuria; glycosuria; aplastic anemia; tremors; acne; muscle spasm; and, rarely, dysarthria, ataxia, impaired cognition, and pseudotumor cerebri (Arena 1986; Ellenhorn and Barceloux 1988). Toxic effects that may appear after prolonged therapeutic use may include neurological symptoms, changes in kidney function, hypothyroidism, and leukocytosis.

The nervous system is the primary target organ of lithium toxicity. Neurologic effects occurring during prolonged therapy often include minor effects on memory, motor activity, and associative productivity (Kocsis et al. 1993). Movement disorders (myoclonus, choreoathetosis), proximal muscle weakness, fasciculations, gait disturbances, incontinence, corticospinal tract signs, and a Parkinsonian syndrome (cogwheel rigidity, tremor) have been reported (Sansone and Ziegler 1985). Cases of severe lithium neurotoxicity, which may occur during chronic therapy as a result of increased lithium retention, may be characterized by disorientation, incoherence, paralysis, stupor, seizure, and coma (Hall et al. 1979). Permanent brain damage has occurred in several patients on long-term lithium therapy (Gosselin et al. 1984).

During chronic lithium therapy, changes in kidney function may appear as transient natriuresis, polydipsia/polyuria, nephrogenic diabetes insipidus, partial renal tubular acidosis, minimal change

disease, and nephrotic syndrome (Ellenhorn and Barceloux 1988). Degenerative changes may occur in the glomeruli or in the distal convoluted tubules or collecting ducts (Richman et al. 1980, Hestbech et al. 1977). In rare cases, acute renal failure may occur (Fenves et al. 1984).

Cohort studies indicate that the risk of major congenital malformations among women receiving lithium during early pregnancy is slightly higher (4–12 percent) than that among control groups (2–4 percent) (Cohen et al. 1994). Evidence also suggests that women on lithium therapy may have a higher risk of premature births. In animals, reproductive and developmental effects (decrease in litter size, decrease in live pups, reduced growth, and increased incidence of cleft palate) have been reported in rodents exposed to lithium salts during gestation (Marathe and Thomas 1986, Sechzer et al. 1992, Szabo 1970, Chernoff and Kavlock 1982). Subchronic and chronic oral RfDs have not been derived for lithium.

Limited information is available on the inhalation toxicity of lithium compounds. Lithium hydride is a respiratory tract irritant. In occupationally exposed workers, concentrations between 1 and 5.0 mg/m³ caused severe eye and nasal irritation as well as skin irritation; concentrations of 0.025 mg/m³ or less caused no adverse effects (Beliles 1994). In animal studies, concentrations above 10 mg/m3 for 4–7 hours resulted in inflammation of the eyes, partial sloughing of mucosal epithelium of the trachea, lesions of the nose and forepaws, and erosion of the nasal septum (Spiegl et al. 1956).

Lithium combustion aerosols are also respiratory tract irritants. In a study in which rats were exposed for 4 hours to an aerosol consisting of 80 percent lithium carbonate and 20 percent lithium hydroxide, signs of toxicity included anorexia, dehydration, respiratory difficulty, perioral and perinasal encrustation, ulcerative or necrotic laryngitis, focal to segmental ulcerative rhinitis often accompanied by squamous metaplasia, and in some animals, suppurative bronchopneumonia or aspiration pneumonia, probably secondary to laryngeal lesions (Greenspan et al. 1986). The LC₅₀ (after 14 days) was estimated to be 1700 mg/m³ for males and 2000 mg/m³ for females. In a second study in which rats were exposed for 4 hours to an aerosol containing mostly lithium monoxide, some lithium hydroxide, and 12 percent lithium carbonate, the LC₅₀ value (after 14 days) was 940 mg/m³ (Rebar et al. 1986). Four-hour exposure to an aerosol containing primarily lithium hydroxide with 23 percent lithium carbonate resulted in an LC₅₀ of 960 mg/m³ (Rebar et al. 1986).

Little information was found in the available literature on the carcinogenicity of lithium compounds. However, three patients on chronic lithium therapy developed leukemia, and one developed a thyroid tumor. Lithium has not been classified by EPA as to its potential carcinogenicity.

No information was found in the available literature on the subchronic, chronic, or developmental/reproductive toxicity of lithium compounds by the inhalation route. In addition, subchronic and chronic inhalation RfCs have not been derived for lithium.

The oral RfD used in this BHHRA is 2.00E-2 mg/kg-day. The dermal route RfD based on the oral RfD and a gastrointestinal absorption factor of 8 percent is 1.60E-2 mg/kg-day. No slope factors were found. Inhalation toxicity values were not available.

1.4.1.15 Manganese (CAS 007439-96-5) (RAIS)

Manganese is an essential trace element in humans that can elicit a variety of serious toxic responses upon prolonged exposure to elevated concentrations either orally or by inhalation. The CNS is the primary target. Initial symptoms are headache, insomnia, disorientation, anxiety, lethargy, and memory loss. These symptoms progress with continued exposure and eventually include motor disturbances, tremors, and difficulty in walking, symptoms similar to those seen with Parkinsonism. These motor difficulties are often irreversible. Based on human epidemiological studies, 0.8 mg/kg-day for drinking water exposure and 0.34 mg/m³ in air for inhalation exposure have been estimated LOAELs for CNS-effects.

Effects on reproduction (decreased fertility, impotence) have been observed in humans with inhalation exposure and in animals with oral exposure at the same or similar doses that initiate the central nervous system effects. An increased incidence of coughs, colds, dyspnea during exercise, bronchitis, and altered lung ventilatory parameters have also been seen in humans and animals with inhalation exposure. A possible effect on the immune system may account for some of these respiratory symptoms.

Because of the greater bioavailability of manganese from water, separate RfDs for water and diet were calculated. A chronic (EPA 1995c) and subchronic RfD (EPA 1998b) for drinking water has been calculated by EPA from a human NOAEL; the NOAEL was determined from an epidemiological study of human populations exposed for a lifetime to manganese concentrations in drinking water ranging from 3.6 to 2300 μ g/L (Kondakis et al. 1989). A chronic (EPA 1995c) and subchronic RfD (EPA 1998b) for dietary exposure has been calculated by EPA from a human NOAEL, which was determined from a series of epidemiological studies (Schroeder et al. 1966, WHO 1973, NRC 1989). Large populations with different concentrations of manganese in their diets were examined. No adverse effects that were attributable to manganese were seen in any of these groups. For both the drinking water and dietary values, the RfD was derived from these studies without uncertainty factors since manganese is essential in human nutrition and the exposure of the most sensitive groups was included in the populations examined. EPA (1995c) indicates that the chronic RfD values are pending change.

A RfC of 0.05 μ g/m³ (EPA 1995c) for chronic inhalation exposure was calculated from a human LOAEL of 0.05 mg/m³ for impairment of neurobehavioral function from an epidemiological study by Roels et al. (1992). The study population was occupationally exposed to airborne manganese dust with a median concentration of 0.948 mg/m³ for 0.2 to 17.7 years with a mean duration of 5.3 years. Neurological examinations, psychomotor tests, lung function tests, blood tests, and urine tests were used to determine the possible effects of exposure. The LOAEL was derived from an occupational-lifetime integrated respirable dust concentration of manganese dioxide expressed as mg manganese/m³ × years. Confidence in the inhalation RfC is rated medium by the EPA.

Some conflicting data exist on possible carcinogenesis following injections of manganese chloride and manganese sulfate in mice. However, the EPA weight-of-evidence classification is Group D, not classifiable as to human carcinogenicity based on no evidence in humans and inadequate evidence in animals (EPA 1995c).

As noted previously, no cancer slope factors for manganese are available. Therefore, carcinogenicity from exposure to manganese is not included in this BHHRA. The oral RfDs used are 4.60E-2 and 1.40E-1 mg/kg-day for the exposure through aqueous media and diet, respectively. The dermal route RfD based on the oral RfD for exposure to aqueous media and diet and a gastrointestinal absorption factor of 4 percent permits the derivation of absorbed RfD values of 1.84E-3 and 5.60E-03 mg/kg-day, respectively. The manganese RfD for inhalation exposure used is 1.43E-5 mg/kg-day for aqueous media and diet.

1.4.1.16 Mercury (CAS 007439-97-6) (RAIS)

Mercury is a naturally occurring element existing in multiple forms and in various oxidation states. It is used in a wide variety of products and processes. In the environment, mercury may undergo transformations among its various forms and among its oxidation states. Exposure to mercury may occur in both occupational and environmental settings, the latter primarily involving dietary exposure (ATSDR 1989d).



Absorption, distribution, metabolism, and excretion of mercury is dependent upon its form and oxidation state (ATSDR 1989d, Goyer 1991). Organic mercurials are more readily absorbed than are inorganic forms. An oxidation-reduction cycle is involved in the metabolism of mercury and mercury compounds by both animals and humans (ATSDR 1989d). The urine and feces are primary excretory routes. The elimination half-life is 35–90 days for elemental mercury and mercury vapor and about 40 days for inorganic salts (Goyer 1991).

Ingestion of mercury metal is usually without effect (Goldwater 1972). Ingestion of inorganic salts may cause severe gastrointestinal irritation, renal failure, and death with acute lethal doses in humans ranging from 1 to 4 g (ATSDR 1989d). Mercuric (divalent) salts are usually more toxic than are mercurous (monovalent) salts (Goyer 1991). Mercury is also known to induce hypersensitivity reactions such as contact dermatitis and acrodynia (pink disease) (Mathesson et al. 1980). Inhalation of mercury vapor may cause irritation of the respiratory tract, renal disorders, CNS effects characterized by neurobehavioral changes, peripheral nervous system toxicity, renal toxicity (immunologic glomerular disease), and death (ATSDR 1989d).

Toxicity resulting from subchronic and chronic exposure to mercury and mercury compounds usually involves the kidneys and/or CNS, the specific target and effect being dependent on the form of mercury (ATSDR 1989d). Organic mercury, especially methyl mercury, rapidly enters the CNS resulting in behavioral and neuromotor disorders (ATSDR 1989d, Goyer 1991). The developing CNS is especially sensitive to this effect, as documented by the epidemiologic studies in Japan and Iraq where ingestion of methyl mercury-contaminated food resulted in severe toxicity and death in adults and severe central nervous system effects in infants (Bakir et al. 1973, Amin-Zaki et al. 1974, Harada 1978, Marsh et al. 1987). Blood mercury levels of less than 10 μ g/dL and 300 μ g/dL corresponded to mild effects and death, respectively (Bakir et al. 1973). Teratogenic effects due to organic or inorganic mercury exposure do not appear to be well-documented for humans or animals, although some evidence exists for mercury-induced menstrual cycle disturbances and spontaneous abortions (Derobert and Tara 1950, Amin-Zaki et al. 1974, ATSDR 1989d).

A subchronic and chronic oral RfD of 1.00E-4 mg/kg-day for methyl mercury is based on a benchmark dose of 1.10 μ g/kg-day relative to neurologic developmental abnormalities in human infants (EPA 1998a, 1998b). A subchronic and chronic oral RfD of 3.00E-4 mg/kg-day for mercuric chloride is based on immunologic glomerulonephritis (EPA 1998a). An LOAEL of 6.30E-1 mg Hg/kg-day for mercuric chloride was identified (EPA 1987e). NOAELs were not available for oral exposure to inorganic mercury or methyl mercury. A subchronic and chronic inhalation RfC of 3.00E-4 mg Hg/m³ for inorganic mercury (EPA 1998a, 1998b) is based on neurological disorders (increased frequency of intention tremors) following long-term occupational exposure to mercury vapor (Fawer et al. 1983). The LOAELs for subchronic and chronic inhalation RfC for methyl mercury has not been determined.

No data were available regarding the carcinogenicity of mercury in humans or animals. EPA has placed inorganic mercury in weight-of-evidence classification D, not classifiable as to human carcinogenicity (EPA 1998a). Weight-of-evidence classifications of C (possible human carcinogen) have been assigned to mercuric chloride and methyl mercury by EPA (1998a) based upon limited evidence of carcinogenicity in rodents. No slope factors have been calculated.

The oral RfD used in this BHHRA is 3.00E-4 mg/kg-day. The absorbed RfD based on the oral RfD and a gastrointestinal absorption factor of 7 percent is 2.10E-5 mg/kg-day. The RfD for inhalation exposure used in the BHHRA is 8.57E-5 mg/kg-day.

1.4.1.17 Molybdenum (CAS 007439-98-7) (RAIS)

Molybdenum occurs naturally in various ores, the principal source being molybdenite (M_0S_2) (Stokinger 1981a). Molybdenum compounds are used primarily in the production of metal alloys. Molybdenum is considered an essential trace element; the provisional recommended dietary intake is 75–250 g/day for adults and older children (NRC 1989).

Water-soluble molybdenum compounds are readily taken up through the lungs and gastrointestinal tract, but insoluble compounds are not. Following absorption, molybdenum is distributed throughout the body with the highest levels generally found in the liver, kidneys, spleen, and bone (Wennig and Kirsch 1988). Limited data suggest that 25–50 percent of an oral dose is excreted in the urine, with small amounts also eliminated in the bile. Biological half-life may vary from several hours in laboratory animals to as much as several weeks in humans (Friberg and Lener 1986, Jarrell et al. 1980, Stokinger 1981a, Vanoeteren et al. 1982, Venugopal and Luckey 1978).

Data documenting molybdenum toxicity in humans are limited. The physical and chemical state of the molybdenum, route of exposure, and compounding factors such as dietary copper and sulfur levels may all affect toxicity. Mild cases of molybdenosis may be clinically identifiable only by biochemical changes (e.g., increases in uric acid levels due to the role of molybdenum in the enzyme xanthine oxidase). Excessive intake of molybdenum causes a physiological copper deficiency, and conversely, in cases of inadequate dietary intake of copper, molybdenum toxicity may occur at lower exposure levels.

There is no information available on the acute or subchronic oral toxicity of molybdenum in humans. In studies conducted in a region of Armenia where levels of molybdenum in the soil are high (77 mg Mo/kg), 18 percent of the adults examined in one town and 31 percent of those in another town were found to have elevated concentrations of uric acid in the blood and urine, increased blood xanthine oxidase activity, and gout-like symptoms such as arthralgia, articular deformities, erythema, and edema (Kovalskii et al. 1961). The daily molybdenum intake was estimated to be 10-15 mg. An outbreak of genu valgum (knock-knees) in India was attributed to an increase in Mo levels in sorgum, the main staple food of the region. The estimated daily Mo intake was 1.5 mg (Jarrell et al. 1980).

In animals, acutely toxic oral doses of molybdenum result in severe gastrointestinal irritation with diarrhea, coma, and death from cardiac failure. Oral LD_{50} values of 125 and 370 mg Mo/kg for molybdenum trioxide and ammonium molybdate, respectively, have been reported in laboratory rats (Venugopal and Luckey 1978). Subchronic and chronic oral exposures can result in gastrointestinal disturbances, growth retardation, anemia, hypothyroidism, bone and joint deformities, sterility, liver and kidney abnormalities, and death (Lloyd et al. 1976, Venugopal and Luckey 1978, Valli et al. 1969, Fairhall et al. 1945, Rana and Kumar 1980). Fatty degeneration of the liver occurred in rabbits dosed with 50 mg/kg-day for 6 months (Asmangulyan 1965) and in rats dosed with 5 mg/kg-day as ammonium molybdate for 1 year (Valjcuk and Sramko 1973). Male sterility was reported in rats fed diets containing 80 or 140 ppm molybdenum (Jeter and Davis 1954). Teratogenic effects have not been observed in mammals, but embryotoxic effects, including reduced weight gain, reduced skeletal ossification, nerve system demyelinization, and reduced survival of offspring have been reported (Wide 1984, Earl and Vish 1979, Schroeder and Mitchener 1971).

The chronic oral RfD for molybdenum and molybdenum compounds is 5.00E-3 mg/kg-day, based on biochemical indices in humans (EPA 1998b). The subchronic RfD is also 5.00E-3 mg/kg-day (EPA 1998b).

Information on the inhalation toxicity of molybdenum in humans following acute and subchronic exposures is not available. Studies of workers chronically exposed to molybdenum indicate a high

incidence of weakness, fatigue, headache, irritability, lack of appetite, epigastric pain, joint and muscle pain, weight loss, red and moist skin, tremor of the hands, sweating, and dizziness (Akopajan 1964, Ecolajan 1965, Walravens et al. 1979). Elevated levels of molybdenum in blood plasma and urine and high levels of ceruloplasmin and uric acid in blood serum were reported for workers exposed to molybdenum (8-hour TWA 9.5 mg molybdenum/m³) (Walravens et al. 1979). Occupational exposure to molybdenum may also result in increased serum bilirubin levels and decreased blood IgA/IgG ratios due to a rise in alpha-immunoglobulins (Avakajan 1966, 1968). Direct pulmonary effects of chronic exposure to molybdenum have been reported in only one study in which 3 of 19 workers exposed to molybdenum and MoO_3 (1–19 mg/m³) for 3–7 years were symptomatic and had X-ray findings indicative of pneumoconiosis (Mogilevskaya 1963). Adverse reproductive or developmental effects have not been observed in molybdenum workers (Metreveli et al. 1985).

In animal studies, inhalation exposures to molybdenum compounds have resulted in respiratory tract irritation, pulmonary hemorrhages, perivascular edema, and liver and kidney damage (Mogilevskaya 1963, Fairhall et al. 1945). Other effects reported in animals include diarrhea, muscle incoordination, loss of hair, loss of weight (Fairhall et al. 1945), changes in ECG, increased arterial blood pressure, increased serum lactate dehydrogenase, increased cardiac adrenaline and noradrenaline levels (Babayan et al. 1984), and inflammation of the uterine horns with necrotic foci and endometrial atrophy (Metreveli and Daneliya 1984). Some molybdenum compounds, such as molybdenum trioxide and sodium molybdate (Na_2MoO_4), are strong eye and skin irritants; however, others, such as calcium and zinc molybdates, are not primary irritants.

Subchronic and chronic RfC for molybdenum are not available.

Information on the oral or inhalation carcinogenicity of molybdenum compounds in humans was not available, and animal data indicate that molybdenum may have an inhibitory effect on esophageal (Luo et al. 1983, van Rensburg et al. 1986, Komada et al. 1990) and mammary carcinogenesis (Wei et al. 1987). However, intraperitoneal injections of MoO_3 in mice produced a significant increase in the number of lung adenomas per mouse and an insignificant increase in the number of mice bearing tumors (Stoner et al. 1976). Molybdenum is placed in EPA Group D, not classifiable as to carcinogenicity in humans (EPA 1990b) and calculation of slope factors is not possible.

A chronic oral RfD of 5.00 E-3 mg/kg-day was used in the BHHRA. The absorbed RfD based on the oral RfD and a gastrointestinal absorption factor of 38 percent is 1.90E-3 mg/kg-day (RAIS). No inhalation RfD was available.

1.4.1.18 Nickel (CAS 007440-02-0) (RAIS)

Nickel is a naturally occurring element that may exist in various mineral forms. It is used in a wide variety of applications including metallurgical processes and electrical components, such as batteries (ATSDR 1988b, USAF 1990e). Some evidence suggests that nickel may be an essential trace element for mammals.

The absorption of nickel is dependent on its physicochemical form, with water soluble forms being more readily absorbed. The metabolism of nickel involves conversion to various chemical forms and binding to various ligands (ATSDR 1988b). Nickel is excreted in the urine and feces with relative amounts for each route being dependent on the route of exposure and chemical form. Most nickel enters the body via food and water consumption, although inhalation exposure in occupational settings is a primary route for nickel-induced toxicity.

In large doses (> 0.5 g), some forms of nickel may be acutely toxic to humans when taken orally. (Daldrup et al. 1983, Sunderman et al. 1988). Oral LD_{50} values for rats range from 67 mg nickel/kg (nickel sulfate hexahydrate) to > 9000 mg nickel/kg (nickel powder) (ATSDR 1988b). Toxic effects of oral exposure to nickel usually involve the kidneys with some evidence from animal studies showing a possible developmental/reproductive toxicity effect (ATSDR 1988b, Goyer 1991).

Inhalation exposure to some nickel compounds will cause toxic effects in the respiratory tract and immune system (Smialowicz et al. 1984, 1985, 1987; ATSDR 1988b; Goyer 1991). Inhalation LC_{50} values for animals range from 0.97 mg nickel/m³ for rats (6-hour exposure) to 15 mg nickel/m³ for guinea pigs (time not specified) (USAF 1990e). Acute inhalation exposure of humans to nickel may produce headache, nausea, respiratory disorders, and death (Goyer 1991, Rendall et al. 1994). Asthmatic conditions have also been documented for inhalation exposure to nickel (Goyer 1991). Soluble nickel compounds tend to be more toxic than insoluble compounds (Goyer 1991). In addition, nickel carbonyl is known to be extremely toxic to humans upon acute inhalation exposure (Goyer 1991).

Data on nickel-induced reproductive/developmental effects in humans following inhalation exposure are equivocal. No clinical evidence of developmental or reproductive toxicity were reported for women working in a nickel refinery (Warner 1979), but Chashschin et al. (1994) reported possible reproductive and developmental effects in humans of occupational exposure to nickel (0.13–0.2 mg nickel/m³). Although not validated by quantitative epidemiologic data or statistical analyses, the authors reported an apparently abnormal increase in spontaneous and threatening abortions (16–17 percent in nickel-exposed workers versus 8–9 percent in nonexposed workers), and an increased incidence of non-specified structural malformations (17 percent versus 6 percent) was reported also. Furthermore, sensitivity reactions to nickel are well documented and usually involve contact dermatitis reactions resulting from contact with nickel-containing items such as cooking utensils, jewelry, coins, etc. (ATSDR 1988b).

A chronic (EPA 1995d) and subchronic (EPA 1998b) oral RfD of 2.00E-2 mg/kg-day for soluble nickel salts is based on changes in organ and body weights of rats receiving dietary nickel sulfate hexahydrate (5 mg/kg-day) for 2 years. A NOAEL and LOAEL of 5 mg/kg-day and 50 mg/kg-day, respectively, were reported in the key study (Ambrose et al. 1976). An uncertainty factor of 300 reflects interspecies extrapolation uncertainty, protection of sensitive populations, and a modifying factor of 3 for a database deficient in reproductive/developmental studies. An inhalation RfC for soluble nickel salts is under review by the RfD/RfC Work Group (EPA 1995d) and currently is not available.

The primary target organs for nickel-induced systemic toxicity are the lungs and upper respiratory tract for inhalation exposure and the kidneys for oral exposure (ATSDR 1988b, Goyer 1991). Other target organs include the cardiovascular system, immune system, and the blood.

Epidemiologic studies have shown that occupational inhalation exposure to nickel dust (primarily nickel subsulfate) at refineries has resulted in increased incidences of pulmonary and nasal cancer (NAS 1975, Enterline and Marsh 1982, ATSDR 1988b). Inhalation studies using rats have also shown nickel subsulfate or nickel carbonyl to be carcinogenic (Sunderman et al. 1959, Sunderman and Donnelly 1965, Ottolenghi et al. 1974). Based on these data, the EPA (1995d) has classified nickel subsulfate and nickel refinery dust in weight-of-evidence group A, human carcinogen. Carcinogenicity slope factors of 1.70E+0 and 8.40E-1 (mg/kg-day)⁻¹ and unit risks of 4.80E-4 (μ g/m³)⁻¹ and 2.40E-4 (μ g/m³)⁻¹ have been calculated for nickel subsulfide and nickel refinery dust, respectively (EPA 1998b, 1995d). Based on an increased incidence of pulmonary carcinomas and malignant tumors in animals exposed to nickel carbonyl by inhalation or by intravenous injection, this compound had been placed in weight-of-evidence Group B2, probable human carcinogen (EPA 1995d). No unit risk values were available for nickel carbonyl. Recent analyses of epidemiologic data, however, indicate that definitive identification of a specific



nickel compound as the causative agent is not yet possible (Easton et al. 1994, Langård 1994, Roberts et al. 1994).

No cancer slope factors for soluble nickel salts were found. Therefore, carcinogenicity due to exposure to soluble nickel salts is not included in the BHHRA. The oral RfD used in the BHHRA is 2.00E-2 mg/kg-day. The dermal route RfD used in the BHHRA, based on the oral RfD and a gastrointestinal absorption factor of 27 percent, is 5.40E-3 mg/kg-day. An inhalation RfD for soluble nickel salts was not found.

1.4.1.19 Silica (CAS 007631-86-9)

Information on the toxicity of silica was not found in the available literature. When information becomes available, it will be included in this report.

Neither slope factors nor RfDs for any route of exposure were found for silica. Therefore, neither carcinogenicity nor systemic toxicity resulting from silica exposure is included in the BHHRA.

1.4.1.20 Silver (CAS 007440-22-4) (RAIS)

Silver is a relatively rare metal that occurs naturally in the earth's crust and is released to the environment from various industrial sources. Human exposure to silver and silver compounds can occur orally, dermally, or by inhalation. Silver is found in most tissues but has no known physiologic function.

In humans, accidental or intentional ingestion of large doses of silver nitrate has produced corrosive damage of the gastrointestinal tract, abdominal pain, diarrhea, vomiting, shock, convulsions, and death (EPA 1985a). Respiratory irritation was noted following acute inhalation exposure to silver or silver compounds. Silver nitrate solutions are highly irritating to the skin, mucous membranes, and eyes (Stokinger 1981b).

Ingestion, inhalation, or dermal absorption of silver may cause argyria, the most common indicator of long-term exposure to silver or silver compounds in humans. Argyria is a gray or blue-gray, permanent discoloration of the skin and mucous membranes that is not a toxic effect per se but is considered cosmetically disfiguring. Chronic inhalation exposure of workers to silver oxide and silver nitrate dusts resulted in upper and lower respiratory irritation, deposition of granular silver-containing deposits in the eyes, impaired night vision, and abdominal pain (Rosenman et al. 1979). Mild allergic responses have been attributed to dermal contact with silver (ATSDR 1990e).

In long-term oral studies with experimental animals, silver compounds have produced slight thickening of the basement membranes of the renal glomeruli, growth depression, shortened lifespan, and granular silver-containing deposits in skin, eyes, and internal organs (Matuk et al. 1981; Olcott 1948, 1950). Hypoactivity was seen in rats subchronically exposed to silver nitrate in drinking water (Rungby and Danscher 1984).

An RfD of 5.00E-3 mg/kg-day for subchronic and chronic exposure was calculated from an LOAEL of 1.40E-2 mg/kg-day for argyria observed in patients receiving intravenous injections of silver arsphenamine (EPA 1998a,b). Data are presently insufficient to derive an RfC for silver (EPA 1998a).

Data adequate for evaluating the carcinogenicity of silver to humans or animals by ingestion, inhalation, or other routes of exposure were not found. Based on EPA guidelines, silver is placed in weight-of-evidence Group D, not classifiable as to human carcinogenicity (EPA 1998a).

The oral RfD used in this BHHRA is 5.00E-3 mg/kg-day. The dermal route RfD based on the oral-RfD and a gastrointestinal absorption factor of 18 percent is 9.00E-4 mg/kg-day. The RfD for inhalation exposure has not been determined.

1.4.1.21 Sulfate (CAS 014808-79-8) (RAIS)

The sulfate ion, SO₄, is one of the major anions occurring in natural waters (Daniels 1988). The majority of sulfates are soluble in water with the exception of lead, barium, and strontium sulfates. Thus, dissolved sulfate is considered to be a permanent solute of water (WHO 1984a).

The major health effect observed with sulfate ingestion is laxative action (Daniels 1988, NAS 1977), and the cation associated with the sulfate appears to have some effect on the salt's potency as a laxative (Daniels 1988). Sulfate itself slowly penetrates mammalian cellular membranes and is rapidly eliminated through the kidneys (WHO 1984a). Pursuant to the Safe Drinking Water Act, the EPA has proposed maximum contaminant level (MCL) goals of either 400 or 500 mg/L to protect infants (based on Chien et. al. 1968, Peterson 1951, and Moore 1952), and has identified a LOAEL of 630 mg/L based on diarrhea in infants receiving formula made with high-sulfate water (EPA 1990c). The Drinking Water Standards of the U.S. Public Health Service recommend that sulfate in water should not exceed 250 mg/L, except when no more suitable supplies are or can be made available.

Sulfates can contribute to an undesirable taste in water. The taste threshold for the sulfate ion in water is 300-400 mg/L (NAS 1977), and a guidance value of 400 mg/L based on aesthetic quality has been suggested (WHO 1984b). The current EPA national Secondary MCL for sulfate, based on organoleptic effects, is 250 mg/L (EPA 1990c).

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure for sulfate. Therefore, quantitative estimates of carcinogenicity and systemic toxicity due to sulfate exposure are not included in the BHHRA.

1.4.1.22 Tetraoxo-sulfate (CAS 012143-45-2)

Information on the toxicity of tetraoxo-sulfate (1-) was not found in the available literature. When information becomes available, it will be included in this report.

Neither slope factors nor RfDs for any route of exposure were found for tetraoxo-sulfate (1-). Therefore, neither carcinogenicity nor systemic toxicity resulting from tetraoxo-sulfate (1-) exposure is included in the BHHRA.

1.4.1.23 Thallium (CAS 007440-28-0) (RAIS)

This report is an update of the Toxicity Summary for Thallium (CAS Registry No. 7440-28-0). The original summary for this chemical was submitted in 1991. The update was performed by incorporating any new human health toxicity data published since the original submittal of the report. Pertinent pharmacokinetic, toxicologic, carcinogenic, and epidemiologic data were obtained through on-line searches of the TOXLINE database from 1991 through 1994. In addition, any changes to EPA-approved toxicity values (RfDs, RfCs, or cancer slope factors) from the IRIS (current as of December 1998) and/or the HEAST, Annual FY-94 and July Supplement No. 1, for this chemical were incorporated in this update.

Thallium, a naturally occurring elemental metal, is commonly found in minerals and as thallium salts. It can also be released into the environment from industrial sources. Atmospheric thallium

contaminates surface soils by deposition, allowing for the exposure of humans by oral, dermal, or inhalation routes. The most common nonoccupational sources of thallium exposure are contaminated food crops and tobacco. Although normally present in the urine of humans, elevated urine thallium concentrations have been associated with adverse health effects.

The primary targets of thallium toxicity are the nervous, integumentary, and reproductive systems. In humans, acute exposures produce paresthesia, retrobulbar neuritis, ataxia, delirium, tremors, and hallucinations. This implies central, peripheral, and autonomic nervous system involvement (Stokinger 1981c, de Groot and Van Heijst 1988, Kazantzis 1986). Human and animal chronic exposures result in alterations of the brain, spinal cord, and peripheral nerves (Stokinger 1981c, Manzo et al. 1983). In both humans and animals, alopecia is the most common indicator of long-term thallium poisoning (Stokinger 1981c, Manzo et al. 1983).

An increased incidence of congenital malformations was found in children of parents exposed to thallium through the consumption of home grown fruits and vegetables. However, a causal relationship between these effects and thallium exposure could not be confirmed (Dolgner et al. 1983). In animal studies, thallium compounds produced testicular effects in male rats and slight fetotoxicity and significant impairment of learning ability in the offspring of treated female rats (Formigli et al. 1986, Roll and Matthiaschk 1981, Bornhausen and Hagen 1984).

RfDs have been calculated for subchronic and chronic oral exposure to several thallium compounds. The values, derived from a single study where thallium treatment increased AST and LDH activities in rats, are based on NOAELs ranging from 2.30E-1 to 2.80E-1 mg/kg-day (EPA 1986i). The subchronic RfDs are 8.00E-04 (thallium sulfate, chloride, and carbonate) or 9.00E-4 mg/kg-day (thallium nitrate and acetate) (EPA 1998b), and the chronic RfDs are 8.00E-5 (thallium sulfate, chloride, and carbonate) or 9.00E-5 mg/kg-day (thallium nitrate and acetate) (EPA 1994b).

Data suitable for evaluating the carcinogenicity of thallium to humans or animals by ingestion, inhalation, or other routes of exposure were not found. Thallium sulfate, selenite, nitrate, chloride, carbonate, and acetate have been placed in EPA's weight-of evidence Group D, not classifiable as to human carcinogenicity based on inadequate human and animal data (EPA 1994b-g).

Neither slope factors nor chronic RfDs for any route of exposure were found for thallium. Therefore, neither carcinogenicity nor systemic toxicity due to thallium (soluble salt) exposure is included in the BHHRA. A gastrointestinal absorption factor of 15 percent is available for thallium-soluble salts.

1.4.1.24 Uranium (metal and soluble salts) (CAS 007440-61-1)

Uranium is a hard, silvery-white amphoteric metal and is a radioactive element. In its natural state it consists of three isotopes: uranium-234, uranium-235, and uranium-238. More than 100 uranium minerals exist; those of commercial importance are the oxides and oxygenous salts. The processing of uranium ore generally involves extraction then leaching either by an acid or a carbonate method. In addition, the metal may be obtained from its halides by fused salt electrolysis. The primary use of natural uranium is in nuclear energy as a fuel for nuclear reactors, in plutonium production, and as feeds for gaseous diffusion plants; it is also a source of radium salts. Uranium compounds are used in staining glass, glazing ceramics, and enameling; in photographic processes; for alloying steels; and as a catalyst for chemical reactions, radiation shielding, and aircraft counterweights (Sittig 1981).

The primary route of exposure to uranium metals and salts is through dermal contact. Uranium soluble compounds act as a poison to cause kidney damage under acute exposure and pneumoconiosis or pronounced blood changes under chronic exposure conditions. Furthermore, it is difficult to separate the

toxic chemical effects of uranium and its compounds from their radiation effects. The chronic radiationeffects are similar to those produced by ionizing radiation. Reports now confirm that carcinogenicity is related to dose and exposure time. Cancer of the lung, osteosarcoma, and lymphoma have all been reported (Sittig 1985a). An EPA weight-of-evidence classification for uranium metal was not located in the available literature.

The oral and dermal RfD for chronic exposures is 3.00E-3 and 2.55E-3 mg/kg-day, respectively for uranium. A gastrointestinal absorption factor of 85 percent was used. No slope factors are available for uranium.

1.4.1.25 Vanadium (CAS 007440-62-2 for metal) (RAIS)

Vanadium is a metallic element that occurs in six oxidation states and numerous inorganic compounds. Some of the more important compounds are vanadium pentoxide (V_2O_5), sodium metavanadate (NaVO₃), sodium orthovanadate (Na₃VO₄), vanadyl sulfate (VOSO₄), and ammonium vanadate (NH₄VO₃). Vanadium is used primarily as an alloying agent in steels and nonferrous metals (ATSDR 1990f). Vanadium compounds are also used as catalysts and in chemical, ceramic or specialty applications.

Vanadium compounds are poorly absorbed through the gastrointestinal system (0.5–2 percent of dietary amount) (NRCC 1980, ICRP 1960, Byrne and Kosta 1978), but slightly more readily absorbed through the lungs (20–25 percent) (ICRP 1960, Davies and Bennett 1983). Absorbed vanadium is widely distributed in the body, but short-term localization occurs primarily in bone, kidneys, and liver (Vouk 1979, Roshchin et al. 1980, Parker et al. 1980, Sharma et al. 1980, Wiegmann et al. 1982). In the body, vanadium can undergo changes in oxidation state interconversion of vanadyl (+4) and vanadate (+5) forms, and it can also bind with blood protein (transferin) (Harris et al. 1984). Vanadium is excreted primarily in the feces following oral exposures and primarily in the urine following inhalation exposures (Tipton et al. 1969, ATSDR 1990f).

The toxicity of vanadium depends on its physicochemical state, particularly on its valence state and solubility. Based on acute toxicity, pentavalent NH_4VO_3 has been reported to be more than twice as toxic as trivalent VCl_3 and more than 6 times as toxic as divalent VI_2 . Pentavalent V_2O_5 has been reported to be more than 5 times as toxic as trivalent V_2O_3 (Roshchin 1967). In animals, acutely toxic oral doses cause vasoconstriction, diffuse desquamative enteritis, congestion and fatty degeneration of the liver, congestion and focal hemorrhages in the lungs and adrenal cortex (Gosselin et al. 1984). Minimal effects seen after subchronic oral exposures to animals include diarrhea, altered renal function, and decreases in erythrocyte counts, hemogloblin, and hematocrit (Domingo et al. 1985, Zaporowska and Wasilewski 1991). In humans, intestinal cramps and diarrhea may occur following subchronic oral exposures. These studies indicate that, for subchronic and chronic oral exposures, the primary targets are the digestive system, kidneys, and blood.

RfDs for chronic oral exposures are: 7.00E-3 mg/kg-day for vanadium; 9.00E-3 mg/kg-day for vanadium pentoxide; 2.00E-2 mg/kg-day for vanadyl sulfate; and 1.00E-3 mg/kg-day for sodium metavanadate (EPA 1987f, 1991e, 1998b). The subchronic RfDs for these compounds are the same as the chronic RfDs, except for sodium metavanadate, which is 1.00E-2 mg/kg-day (EPA 1987f, 1991e, 1998b).

Inhalation exposures to vanadium and vanadium compounds result primarily in adverse effects to the respiratory system (Sax 1984, ATSDR 1990f). In laboratory studies, minimal effects (throat irritation and coughing) occurred after an 8-hour exposure to 0.1 mg V/m^3 (Zenz and Berg 1967). In studies on workers occupationally exposed to vanadium, the most common reported symptoms were irritation of the respiratory tract, conjunctivitis, dermatitis, cough, bronchospasm, pulmonary congestion, and bronchitis



(Symanski 1939; Sjoberg 1950, 1951, 1955, 1956; Vintinner et al. 1955; Lewis 1959; Tebrock and Machle 1968; Roshchin 1968; Kiviluoto et al. 1981). Quantitative data are insufficient to derive a subchronic or chronic inhalation RfC for vanadium or vanadium compounds.

There is little evidence that vanadium or vanadium compounds are reproductive toxins or teratogens. There is also no evidence that any vanadium compound is carcinogenic; however, very few adequate studies are available for evaluation. Vanadium has not been classified as to carcinogenicity by the EPA (1998b).

The oral RfD used in this BHHRA is 7.00E-3 mg/kg-day. The dermal route RfD based on the oral RfD and a gastrointestinal absorption factor of 1 percent is 7.00E-5 mg/kg-day. The RfD for inhalation exposure has not been determined. No slope factors are available for metallic vanadium.

1.4.1.26 Zinc (CAS 007440-66-6 for metal) (RAIS)

Zinc is used primarily in galvanized metals and metal alloys, but zinc compounds also have wide commercial applications as chemical intermediates, catalysts, pigments, vulcanization activators and accelerators in the rubber industry, ultraviolet stabilizers, and supplements in animal feeds and fertilizers. They are also used in rayon manufacture, smoke bombs, soldering fluxes, mordants for printing and dyeing, wood preservatives, mildew inhibitors, deodorants, antiseptics, and astringents (Lloyd 1984, ATSDR 1989e). In addition, zinc phosphide is used as a rodenticide.

Zinc is an essential element with RDA ranging from 5 mg for infants to 15 mg for adult males (NRC 1989).

Gastrointestinal absorption of zinc is variable (20-80 percent) and depends on the chemical compound as well as on zinc levels in the body and dietary concentrations of other nutrients (EPA 1984c). In individuals with normal zinc levels in the body, gastrointestinal absorption is 20-30 percent (ATSDR 1989e). Information on pulmonary absorption is limited and complicated by the potential for gastrointestinal absorption due to mucociliary clearance from the respiratory tract and subsequent swallowing. Zinc is present in all tissues with the highest concentrations in the prostate, kidney, liver, heart, and pancreas. Zinc is a vital component of many metalloenzymes such as carbonic anhydrase, which regulates CO₂ exchange (Stokinger 1981d). Homeostatic mechanisms involving metallothionein in the mucosal cells of the gastrointestinal tract regulate zinc absorption and excretion (ATSDR 1989e).

In humans, acutely toxic oral doses of zinc cause nausea, vomiting, diarrhea, and abdominal cramps and in some cases gastric bleeding (Elinder 1986, Moore 1978, ATSDR 1989e). Ingestion of zinc chloride can cause burning in the mouth and throat, vomiting, pharyngitis, esophagitis, hypocalcemia, and elevated amylase activity indicative of pancreatitis (Chobanian 1981). Zinc phosphide, which releases phosphine gas under acidic conditions in the stomach, can cause vomiting, anorexia, abdominal pain, lethargy, hypotension, cardiac arrhythmias, circulatory collapse, pulmonary edema, seizures, renal damage, leukopenia, and coma and death in days to weeks (Mack 1989). The estimated fatal dose is 40 mg/kg. Animals dosed orally with zinc compounds develop pancreatitis, gastrointestinal and hepatic lesions, and diffuse nephrosis.

Gastrointestinal upset has also been reported in individuals taking daily dietary zinc supplements for up to 6 weeks (Samman and Roberts 1987). There is also limited evidence that the human immune system may be impaired by subchronic exposures (Chandra 1984). In animals, gastrointestinal and hepatic lesions (Allen et al. 1983, Brink et al. 1959); pancreatic lesions (Maita et al. 1981, Drinker et al. 1927); anemia (ATSDR 1989e, Fox and Jacobs 1986, Maita et al. 1981); and diffuse nephrosis (Maita et al. 1981, Allen et al. 1983) have been observed following subchronic oral exposures. Chronic oral exposures to zinc have resulted in hypochromic microcytic anemia associated with-hypoceruloplasminemia, hypocupremia, and neutropenia in some individuals (Prasad et al. 1963, Porter et al. 1977). Anemia and pancreatitis were the major adverse effects observed in chronic animal studies (Aughey et al. 1977, Drinker et al. 1927, Walters and Roe 1965, Sutton and Nelson 1937). Teratogenic effects have not been seen in animals exposed to zinc; however, high oral doses can affect reproduction and fetal growth (Ketcheson et al. 1969, Schlicker and Cox 1967 and 1968, Sutton and Nelson 1937).

The RfD for chronic oral exposure to zinc is under review by EPA; the currently accepted RfD for both subchronic and chronic exposures is 3.00E-1 mg/kg-day based on clinical data demonstrating zincinduced copper deficiency and anemia in patients taking zinc sulfate for the treatment of sickle cell anemia (EPA 1998b). The chronic oral RfD for zinc phosphide is 3.0E-4 mg/kg-day (EPA 1991f), and the subchronic RfD is 3.00E-3 mg/kg-day (EPA 1998b).

Under occupational exposure conditions, inhalation of zinc compounds (mainly zinc oxide fumes) can result in a condition identified as "metal fume fever," which is characterized by nasal passage irritation, cough, rales, headache, altered taste, fever, weakness, hyperpnea, sweating, pains in the legs and chest, leukocytosis, reduced lung volume, and decreased diffusing capacity of carbon monoxide (ATSDR 1989e, Bertholf 1988). Inhalation of zinc chloride can result in nose and throat irritation, dyspnea, cough, chest pain, headache, fever, nausea and vomiting, and respiratory disorders such as pneumonitis and pulmonary fibrosis (ITII 1988, ATSDR 1989e, Nemery 1990). Pulmonary inflammation and changes in lung function have also been observed in inhalation studies on animals (Amur et al. 1982, Lam et al. 1985, Drinker and Drinker 1928).

Although "metal fume fever" occurs in occupationally exposed workers, it is primarily an acute and reversible effect that is unlikely to occur under chronic exposure conditions when zinc air concentrations are less than 8–12 mg/m³ (ATSDR 1989e). Gastrointestinal distress, as well as enzyme changes indicative of liver dysfunction, have also been reported in workers occupationally exposed to zinc (NRC 1979, Stokinger 1981d, EPA 1991f, Guja 1973, Badawy et al. 1987); however, it is unclear as to what extent these effects might have been caused by pulmonary clearance and subsequent gastrointestinal absorption. Consequently, there are no clearly defined toxic effects that can be identified as resulting specifically from pulmonary absorption following chronic low level inhalation exposures. Animal data for chronic inhalation exposures are not available.

An inhalation RfC has not been derived for zinc or zinc compounds (EPA 1998b).

No case studies or epidemiologic evidence has been presented to suggest that zinc is carcinogenic in humans by the oral or inhalation route (EPA 1991f). In animal studies, zinc sulfate in drinking water or zinc oleate in the diet of mice for a period of one year did not result in a statistically significant increase in hepatomas, malignant lymphomas, or lung adenomas (Walters and Roe 1965); however, in a 3-year, 5-generation study on tumor-resistant and tumor-susceptible strains of mice, exposure to zinc in drinking water resulted in increased frequencies of tumors from the F_0 to the F_4 generation in the tumor-resistant strain (from 0.8 to 25.7 percent versus 0.0004 percent in the controls) and higher tumor frequencies in two tumor-susceptible strains (43.4 percent and 32.4 percent versus 15 percent in the controls) (Halme 1961).

Zinc is placed in weight-of-evidence Group D, not classifiable as to human carcinogenicity due to inadequate evidence in humans and animals (EPA 1991f).

The oral RfD used in this BHHRA is 3.00E-1 mg/kg-day. The dermal route RfD based on the oral RfD and a gastrointestinal absorption factor of 20 percent is 6.00E-2 mg/kg-day. The RfD for inhalation exposure has not been determined.



1.4.2 Organic Compounds

1.4.2.1 1,1,2-Trichloroethane (CAS 000079-00-5) (RAIS)

1,1,2-Trichloroethane, also known as vinyl trichloride, is a nonflammable liquid that is used in the manufacture of 1,1-dichloroethene; as a solvent for fats, waxes, resins, and alkaloids; and in organic synthesis. (Budavari et al. 1989, EPA 1980a).

1,1,2-Trichloroethane is released to the environment as a result of anthropogenic activity. The chemical has been identified in the United States at 45 of 1177 hazardous waste sites on the National Priorities List. Based on release patterns of related chemicals, it is estimated that 70–90 percent of the total release is to air, 10–30 percent to land, and a few percent to water. Removal of 1,1,2-trichloroethane from the atmosphere is thought to occur by reaction with photochemically produced hydroxyl radicals (estimated half-life 49 days) and from washout by precipitation; however, most of the 1,1,2-trichloroethane removed by washout is expected to reenter the atmosphere by volatilization. If released to soil, 1,1,2-trichloroethane is expected to partially leach into groundwater and to partially volatilize. In surface water, volatilization is the primary removal process (ATSDR 1989f).

1,1,2-Trichloroethane is rapidly absorbed, widely distributed in organs and tissues, and extensively metabolized. Major metabolites include chloroacetic acid, S-carboxymethylcysteine, and thiodiacetic acid. 1,1,2-Trichloroethane and/or its metabolites are primarily excreted through the lungs and urine (Morgan et al. 1970, 1972; Kronevi et al. 1977; Mitoma et al. 1985a).

Very limited human data were available to evaluate the toxicity of 1,1,2-trichloroethane. The chemical exerts a narcotic action at "low" concentrations and is irritating to the eyes and mucous membranes of the respiratory tract. When in contact with skin, 1,1,2-trichloroethane may cause cracking and erythema (IARC 1979a).

The oral LD₅₀ for mice (378-491 mg/kg) (White et al. 1985a) indicates that, in animals, the acute oral toxicity of 1,1,2-trichloroethane is moderate. 1,1,2-Trichloroethane is a CNS depressant, inducing sedation in mice at oral doses of 378 mg/kg (White et al. 1985a) and drowsiness, incoordination, and narcosis in dogs at 289 mg/kg (Wright and Schaffer 1932). Male and female CD-1 mice ingesting 384 mg/kg in drinking water for 90 days exhibited alterations in serum enzyme and hepatic microsomal enzyme activities, indicating adverse liver effects. In addition, depressed immune function in both sexes and decreased hemoglobin and hematocrit values in females were noted (Sanders et al. 1985b). Decreased survival was reported in female B6C3F₁ mice exposed to 1.95E+2 or 3.90E+2 mg/kg day for 78 weeks (NCI 1978a).

Bonnet et al. (1980) reported an inhalation LC_{50} of 1654 ppm for rats exposed to 1,1,2-trichloroethane for 6 hours, while another study found that a single 7-hour exposure to 250 or 500 ppm resulted in the death of more than half of the exposed female rats, with surviving animals exhibiting marked liver and kidney damage (Torkelson 1994). As noted previously, 1,1,2-trichloroethane is a CNS depressant inducing narcosis; death results from respiratory arrest (ACGIH 1991). In mice, a concentration of 3750 ppm for 30 minutes produced CNS depression and significantly increased liver enzyme activity within 18 minutes and death in half the animals within 10 hours (Gehring 1968). No adverse effects were observed in rats, guinea pigs, and rabbits exposed to 15 ppm for 7 hours/day, 5 days/week for 6 months, but female rats exposed to 30 ppm (16 exposures; 7 hours/day, 5 days/week) exhibited minor hepatic effects (Torkelson 1994). Repeated topical applications of 0.1 mL 1,1,2-trichloroethane produced erythema, edema, fissuring, and scaling of rabbit and guinea pig skin (Wahlberg 1984).

An oral RfD for subchronic exposure of 4E-2 (EPA 1998b) and for chronic exposure (EPA 1998a) to-1,1,2-trichloroethane was calculated based on a NOAEL of 3.90E-0 mg/kg-day and a LOAEL of 44 mg/kg-day from a 90-day drinking water study with mice (White et al. 1985a, Sanders et al. 1985). Clinical chemistry alterations indicative of liver damage were identified as critical effects. An inhalation RfC for 1,1,2-trichloroethane is under review by EPA (EPA 1998a).

No epidemiologic studies or case reports addressing the carcinogenicity of 1,1,2-trichloroethane in humans were available. In a rodent bioassay, 1,1,2-trichloroethane was administered by gavage to Osborne-Mendel rats (46 or 92 mg/kg-day) and B6C3F₁ mice (195 or 390 mg/kg-day), 5 days/week for 78 weeks (NCI 1978a). No effects on tumor development were noted in rats. Treated mice had significantly (p < 0.01) increased incidences of hepatocellular carcinomas. The tumor incidences in treated males were 37 percent and 76 percent in the low- and high-dose groups, respectively, compared with 10 percent in vehicle controls, and 33 percent and 89 percent in females, respectively, compared to no observed tumors in vehicle controls. An increased incidence of adrenal pheochromocytomas was also observed in male and female mice. In a cancer initiation/promotion study with rats, 1,1,2-trichloroethane did not exhibit tumor initiating or promoting activity (Story et al. 1986).

Based on EPA guidelines, 1,1,2-trichloroethane was assigned to weight-of-evidence Group C, possible human carcinogen. For oral exposure, the slope factor is $5.70E-2 (mg/kg-day)^{-1}$, and the unit risk for drinking water is 1.60E-6 (μ g/L)⁻¹ (EPA 1998a). The inhalation slope factor and unit risk are $5.70E-2 (mg/kg-day)^{-1}$ (EPA 1998b) and 1.60E-5 (μ g/m³)⁻¹, respectively (EPA 1998a).

The oral, dermal, and inhalation cancer slope factors used in the BHHRA for 1,1,2-trichloroethane are 5.70E-2, 7.04E-2, and 5.70E-2 (mg/kg-day)⁻¹, respectively. The oral and dermal RfDs used in the BHHRA are 4.00E-3 and 3.24E-3 mg/kg-day. An inhalation RfD was not found, and based on the localized effects discussed previously, it would not be appropriate to extrapolate an inhalation RfD from the oral RfD. Both the dermal cancer slope factor and the dermal RfD were derived from their respective oral toxicity value using a gastrointestinal absorption factor of 81 percent.

1.4.2.2 1,1-Dichloroethane (CAS 000075-34-3) (RAIS)

1,1-Dichloroethane is used primarily as an intermediate in manufacturing vinyl chloride and 1,1,1-trichloroethane; it is also used as a cleaning agent and degreaser and as a solvent for plastics, oils, and fats (ATSDR 1990g).

The available evidence indicates that 1,1-dichloroethane can be readily absorbed following inhalation and oral exposures (ATSDR 1990g). The anesthetic effects of 1,1-dichloroethane are evidence that the chemical reaches the CNS. Acetic acid is a major metabolite, and 2,2-dichloroethanol, chloroacetic acid, and dichloroacetic acid are minor metabolites (McCall et al. 1983). In animal studies, orally administered 1,1-dichloroethane was excreted primarily in expired air as the unmetabolized chemical (Mitoma et al. 1985b).

No information is available on the oral toxicity of 1,1-dichloroethane to humans. In animals, a drinking water concentration of up to 2500 mg/L for 52 weeks caused no adverse effects in male mice (Klaunig et al. 1986), and maximum gavage doses of 764 mg/kg-day (male Osborne-Mendel rats), 950 mg/kg (female Osborne-Mendel rats), 2885 mg/kg (male B6C3F₁ mice), and 3331 mg/kg (female B6C3F₁ mice), 5 days/week for 78 weeks (3 weeks on, 1 week off) resulted in no histopathological changes (NCI 1978b). A subchronic oral RfD of 1.00E+0 mg/kg-day and a chronic oral RfD of 1.00E-1 mg/kg-day (based on an inhalation study in rats and route-to-route extrapolation) are listed in HEAST (EPA 1998b); however, an oral RfD is currently not found in IRIS. EPA reassessment of the oral RfD is pending (EPA 1998b).

At high vapor concentrations (26,000 ppm), 1,1-dichloroethane induces anesthesia and can cause cardiac arrhythmia in humans, but no fatalities have occurred (ATSDR 1990g). Adverse effects following subchronic or chronic exposures to humans have not been reported. In animal studies, 1,1-dichloroethane did not cause developmental or reproductive effects but did delay rib ossification in rats (Schwetz et al. 1974a). Kidney damage was observed in cats exposed to 2025 mg/m³ (6 hours/day, 5 days/week) for 13 weeks followed by 4050 mg/m³ for an additional 13 weeks; however, similar effects were not seen in rats, rabbits, or guinea pigs. A subchronic RfC of 5 mg/m³ and a chronic RfC of 0.5 mg/m³ are listed in HEAST (EPA 1998b). These RfCs are based on the adverse renal effects in cats following subchronic inhalation exposure. An RfC for 1,1-dichloroethane is not currently on IRIS, although an EPA reassessment of the compound is pending (EPA 1993b).

1,1-Dichloroethane is placed in Group C, possible human carcinogen (EPA 1993b), based on no human data and limited evidence of carcinogenicity in two animal species (rats and mice), as shown by an increased incidence of mammary gland adenocarcinomas and hemangiosarcomas in female rats and an increased incidence of hepatocellular carcinomas and benign uterine polyps in mice (EPA 1993b). Slope factors and unit risks have not been calculated. The RfD used in this BHHRA is 1.00E-1 mg/kg-day for the oral and dermal routes and 1.43E-1 mg/kg-day for the inhalation route. The dermal was derived from the oral toxicity value using a gastrointestinal absorption factor of 100 percent.

1.4.2.3 1,1-Dichloroethylene (CAS 000075-35-4) (RAIS)

1,1-Dichloroethylene, also known as 1,1-dichloroethene and vinylidine chloride, is a colorless liquid that is used primarily in the production of polyvinylidine chloride (PVC) copolymers and as an intermediate for synthesis of organic chemicals. The major application for PVC copolymers is the production of flexible films for food packaging such as Saran® wrap (ATSDR 1993b).

1,1-Dichloroethylene does not occur naturally (IARC 1986a) but is found in the environment due to releases associated with its production and transport and with the production of its polymers. Because of its high volatility, releases to the atmosphere are the greatest source of ambient 1,1-dichloroethylene. Smaller amounts are released to surface waters and soils (ATSDR 1993b). Loss of 1,1-dichloroethylene from water and soils is primarily due to volatilization. In the atmosphere, reaction with photochemically generated hydroxyl radicals is expected to be the predominant removal mechanism (EPA 1987g). Human exposure to 1,1-dichloroethylene is potentially highest in workplace settings and in the vicinity of hazardous waste sites where the compound may contaminate environmental media (ATSDR 1993b).

The primary effect of acute exposure to high concentrations (approximately 4000 ppm) of 1,1-dichloroethylene vapor in humans is CNS depression, which may progress to unconsciousness (Gosselin et al. 1984). Occupational exposure has been reported to cause liver dysfunction in workers (Tierney et al. 1979). 1,1-Dichloroethylene is irritating when applied to the skin, and prolonged contact can cause first degree burns (Tierney et al. 1979). Direct contact with the eyes may cause conjunctivitis and transient corneal injury (IARC 1986a).

In experimental animals, the liver and kidneys are target organs for the toxic effects of 1,1-dichloroethylene. Subchronic oral exposure for 90 days to 1,1-dichloroethylene in drinking water produced slight hepatotoxic effects at 200 ppm (Rampy et al. 1977), and chronic oral exposure to drinking water for 2 years produced hepatocellular changes in males at greater than or equal to 100 ppm and in females at greater than or equal to 50 ppm (Quast et al. 1983). Gavage administration of 10 mg/kg-day, 5 days/week for 2 years produced chronic inflammation of the kidney in male and female rats and liver necrosis in male and female mice (NTP 1982b). Exposure by inhalation to 55 ppm 1,1-dichloroethylene, 6 hours/day, 5 days/week for up to 1 year produced fatty liver changes in rats and focal degeneration and necrosis in mice (Lee et al. 1977).

In a three-generation study, no treatment-related effects on reproduction or neonatal development---were seen in male and female Sprague-Dawley rats administered up to 200 ppm of 1,1-dichloroethylene in the drinking water (Nitschke et al. 1983). However, inhalation exposure during gestation produced increased resorptions and minor skeletal alterations in rodents at concentrations that caused maternal toxicity. These effects were reported in rats and mice at greater than or equal to 15 ppm (Short et al. 1977) and in rats and rabbits at greater than or equal to 80 ppm and greater than or equal to 160 ppm, respectively (Murray et al. 1979a).

An oral RfD of 9.00E-3 mg/kg-day was derived for chronic exposure (EPA 1998a) and subchronic exposure to 1,1-dichloroethylene (EPA 1998b), based on liver lesions seen in rats in a 2-year drinking water study (Quast et al. 1983). The oral RfD is currently under review and may be subject to change. An inhalation RfC for 1,1-dichloroethylene is under review (EPA 1998a).

An epidemiology study using a small cohort found no association between the occurrence of cancer or cancer mortality and exposure to 1,1-dichloroethylene (Ott et al. 1976). Oral carcinogenicity bioassays (drinking water or gavage exposures) with experimental animals gave generally negative results (NTP 1982b; Quast et al. 1983; Maltoni et al. 1984, 1985). In one inhalation study (Maltoni et al. 1985), statistically significant increases in renal adenocarcinomas were noted in male Swiss mice exposed to 25 ppm for 12 months. Also observed were statistically significant increases in mammary gland carcinomas in females and lung tumors in both sexes. Results of other inhalation studies with rats, mice, and hamsters have been negative (Hong et al. 1981, Maltoni et al. 1984, Quast et al. 1986).

Based on EPA guidelines, 1,1-dichloroethylene was assigned to weight-of-evidence Group C, possible human carcinogen. For oral exposure, the slope factor is $6.00E-1 \text{ (mg/kg-day)}^{-1}$, and the unit risk is 1.70E-5 (ug/L)⁻¹ (EPA 1998a). The inhalation slope factor and unit risk are $1.20E+0 \text{ (mg/kg-day)}^{-1}$ and $5.00E-5 \text{ m}^3/\mu \text{g}$ (EPA 1998a), respectively.

The oral, dermal, and inhalation cancer slope factors used in the BHHRA for 1,1-dichloroethene are 6.00E-1, 1.20E+0, and 1.20E+0 (mg/kg-day)⁻¹, respectively. The oral, dermal and inhalation RfDs used in the BHHRA was 9.00E-3 mg/kg-day for all three routes. Both the dermal cancer slope factor and the dermal RfD were derived from their respective oral toxicity value using a gastrointestinal absorption factor of 100 percent.

1.4.2.4 1,2,4-Trichlorobenzene (CAS 000120-82-1)

There are no carcinogenic slope factors used in the BHHRA for 1,2,4-trichlorobenzene due to it being assigned a weight-of-evidence class D.

A chronic oral RfD of 1.00E-2 mg/kg-day is used in this risk assessment for 1,2,4-trichlorobenzene. The dermal route chronic RfD used in the BHHRA is 9.70E-3 mg/kg-day and the inhalation RfD is 5.71E-2. The chronic inhalation RfC is 2.00E-1 mg/m³.

1.4.2.5 1,2-Dichlorobenzene (CAS 000095-50-1)

There are no carcinogenic slope factors used in the BHHRA for 1,2-dichlorobenzene due to it being assigned a weight-of-evidence class D.

A chronic oral reference dose of 9.00E-2 mg/kg-day is used in this risk assessment for 1,2-dichlorobenzene. The dermal route chronic RfD used in the BHHRA is 7.20E-2 mg/kg-day. The chronic inhalation RfC is 2.00E-1 mg/m³, and the inhalation RfD is 5.70E-2 mg/kg-day.



1.4.2.6 1,2-Dichloroethane (CAS 000107-06-2) (RAIS)

1,2-Dichloroethane is used primarily in the manufacture of vinyl chloride, as well as in the synthesis of tetrachloroethylene, trichloroethylene, 1,1,1-trichloroethane, vinylidene chloride, aziridines, and ethylenediamines (USAF 1989a, ATSDR 1992a). It is added to gasoline as a lead-scavenging agent, and, in the past, has been used as a metal degreasing agent; a solvent; and a fumigant for grain, upholstery, and carpets. It has also been used in paints, coatings, adhesives, varnishes, finish removers, soaps, and scouring agents (USAF 1989a, ATSDR 1992a).

1,2-Dichloroethane is expected to be highly mobile in most soils and, consequently, contamination of groundwater is possible. Adsorption to soil particles is low, particularly for soils with a low organic carbon content. Volatilization from soils and surface waters may be an important transport process. Microbial biodegradation is not expected to be significant.

1,2-Dichloroethane is absorbed through the lungs, gastrointestinal system, and skin (ATSDR 1992a). It is distributed throughout the body but may be concentrated in adipose tissue. The compound can also accumulate in breast milk (Urusova 1953) and may cross the placenta (Withey and Karpinski 1985, Vozovaya 1977). Metabolism of 1,2-dichloroethane most likely involves conjugation with glutathione (ATSDR 1992a). Urinary metabolites are likely to include thiodiglycolic acid, chloroacetic acid, and N-acetyl-S-carboxymethyl-L-cysteine (NTP 1991). Excretion occurs primarily through elimination of soluble urinary metabolites (Reitz et al. 1982, Spreafico et al. 1980).

Bronchitis, hemorrhagic gastritis and colitis, hepatocellular damage, renal tubular necrosis, CNS depression, and histopathological changes in the brain have been reported in cases of acute oral poisoning of humans (ATSDR 1992a, NIOSH 1976). Animal data indicate that short-term exposures may produce immune system deficiencies (Munson et al. 1982a), and subchronic or chronic oral exposures may affect the liver or kidney (NTP 1991, Alumot et al. 1976). Subchronic or chronic oral RfDs for 1,2-dichloroethane have not been adopted by EPA (EPA 1993c); however, a provisional RfD of 0.03 mg/kg-day has been calculated by the Superfund Health Risk Technical Support Center (EPA 1994h) from a NOAEL of 26 mg/kg-day for rats tested in a subchronic gavage study (NTP 1991). Use of this value in risk assessment reports for specific sites must be approved by the Support Center.

Acute inhalation exposures to 1,2-dichloroethane (75-125 ppm) can result in irritation of the eyes, nose and throat, dizziness, nausea, vomiting, increasing stupor, cyanosis, rapid pulse, delirium, anesthesia, partial paralysis, loss of tactile sense, degenerative changes in the myocardium, abnormal electroencepthalogram, liver and kidney damage, pulmonary edema, and hemorrhages throughout the body (NIOSH 1976, CEC 1986, ATSDR 1992a, Nouchi et al. 1984). Short-term exposures to animals have resulted in CNS depression (inactivity or stupor, tremors, uncertain gait, narcosis); pulmonary congestion; renal tubular degeneration; fatty degeneration of the liver and, less commonly, necrosis and hemorrhage of the adrenal cortex; chronic splenitis; fatty infiltration of the myocardium; and immunodeficiency (Spencer et al. 1951, Heppel et al. 1946, Storer et al. 1984a, Sherwood et al. 1987). Chronic occupational exposure to 1,2-dichloroethane may result in CNS effects including irritability, sleeplessness, and decreased heart rate; loss of appetite; nausea; vomiting; epigastric pain, as well as irritation of the mucous membranes; and liver and kidney impairment (NIOSH 1976). Subchronic or chronic inhalation exposures to animals resulted in pathological lesions in the kidney, liver, heart, lungs, and testes (Heppel et al. 1946, Spencer et al. 1951, Cheever et al. 1990). A subchronic or chronic inhalation RfC for 1,2-dichloroethane has not been adopted and verified by EPA (EPA 1993c); however, a provisional RfC of 5.00E-3 mg/m³ has been calculated by the Superfund Health Risk Technical Support Center (EPA 1994h) from a LOAEL (gastrointestinal disturbances and liver and gallbladder disease) of 10 mg/m³ for occupationally exposed workers (Kozik 1957). Use of this value in risk assessment reports for specific sites must be approved by the Support Center.

1,2-Dichloroethane is classified by EPA in Group B2 as a probable human carcinogen by both the ______ oral and inhalation exposure routes, based on evidence for the induction of several types of tumors in rats and mice. Male rats treated by gavage with 1,2-dichloroethane exhibited increased incidences of fibromas of the subcutaneous tissue; hemangiosarcomas of the spleen, liver, pancreas, and adrenal gland; and squamous-cell carcinomas of the forestomach. Female rats treated by gavage developed mammary adenocarcinomas. Increased incidences of hepatocellular carcinomas and pulmonary adenomas were observed in male mice treated by gavage, and increased incidences of mammary adenocarcinomas, pulmonary adenocarcinomas, and endometrial polyps and sarcomas were observed in female mice (NCI 1978b). Mice treated by topical application of 1,2-dichloroethane exhibited an increased incidence of lung papillomas (Van Duuren et al. 1979). The oral slope factor for 1,2-dichloroethane is 9.10E-2 (mg/kg-day)⁻¹, and the drinking water unit risk is 2.60E-6 ($\mu g/L$)⁻¹. The inhalation slope factor is 9.10E-2 (mg/kg-day)⁻¹, and the inhalation unit risk is 2.60E-5 m³/ μg (EPA 1993c, 1998b).

The oral, dermal, and inhalation cancer slope factors used in the BHHRA for 1,2-dichloroethane are 9.10E-2 (mg/kg-day)⁻¹. The inhalation RfD used in the BHHRA is 2.86E-3 mg/kg-day. Oral and dermal RfDs were not found. A gastrointestinal absorption factor of 100 percent was used to derive the dermal slope factor.

1.4.2.7 cis- and trans-1,2-Dichloroethene (CAS 000156-59-2 and CAS 000156-60-5) (RAIS)

1,2-Dichloroethene exists in two isomeric forms, *cis*-1,2-dichloroethene and *trans*-1,2-dichloroethene, that are colorless, volatile liquids with a slightly acrid odor. Although not used extensively in industry, 1,2-dichloroethene is used in the production of other chlorinated solvents and as a solvent for dyes, perfumes, and lacquers (Sax and Lewis 1989, Budavari et al. 1989). Humans are exposed to 1,2-dichloroethene primarily by inhalation, but exposure can also occur by oral and dermal routes.

Limited information exists on the absorption, distribution, and excretion of 1,2-dichloroethene in either humans or animals. In vitro studies have shown that the mixed function oxidases will metabolize 1,2-dichloroethene; the final metabolic products are dependent on the initial isomer of 1,2-dichloroethene (Costa and Ivanetich 1984, Henschler 1977, Liebman and Ortiz 1977).

Information on the toxicity of 1,2-dichloroethene in humans and animals is limited. Workers exposed to 1,2-dichloroethene have been reported to suffer from drowsiness, dizziness, nausea, fatigue, and eye irritation (ATSDR 1990h). Acute and subchronic oral and inhalation animal studies of *trans*-1,2-dichloroethene and acute inhalation animal studies of *cis*-1,2-dichloroethene suggest that the liver is the primary target organ. The toxicity is expressed in increased activities of liver-associated enzymes, fatty degeneration, and necrosis (McCauley et al. n.d., Barnes et al. 1985, Freundt et al. 1977). Secondary target organs include the CNS and lung.

Based on an unpublished study describing decreased hemoglobin and hematocrits in rats treated by gavage for 90 days, EPA (1998a,b) assigned a subchronic and chronic oral RfD for *cis*-1,2-dichloroethene of 1.00E-01 mg/kg-day and 1.00E-02 mg/kg-day, respectively. The RfDs were derived from a NOAEL/LOAEL of 32 mg/kg-day. An inhalation RfC for *cis*-1,2-dichloroethene has not been derived.

Subchronic and chronic RfDs of 2.00E-01 mg/kg-day and 2.00E-02 mg/kg-day, respectively, for *trans*-1,2-dichloroethene have been calculated (EPA 1998a,b). The RfDs were derived from a LOAEL of 1.75E+2 mg/kg-day that was based on increased serum alkaline phosphatase activity in mice that received *trans*-1,2-dichloroethene in their drinking water (EPA 1998a,b). An RfC for *trans*-1,2-dichloroethene has not been derived.



No information was available concerning the chronic, developmental, or reproductive toxicity of *cis*-1,2-dichloroethene or *trans*-1,2-dichloroethene. No cancer bioassays or epidemiological studies were available to assess the carcinogenicity of 1,2-dichloroethene. EPA (1998b) has placed both *cis*-1,2-dichloroethene and *trans*-1,2-dichloroethene in weight-of-evidence Group D, not classifiable as to human carcinogenicity, based on the lack of human or animal carcinogenicity data and on essentially negative mutagenicity data. Oral and inhalation slope factors have not been calculated for these isomers.

No cancer slope factors for *cis*- or *trans*-1,2-dichloroethene are available; therefore, carcinogenicity from exposure could not be quantified in the BHHRA. The oral and dermal chronic RfDs for *cis*-1,2-dichloroethene used in the BHHRA are 1.00E-2 and 1.00E-2 mg/kg-day, respectively. The oral and dermal chronic RfDs for *trans*-1,2-dichloroethene used in the BHHRA are 2.00E-2 and 2.00E-2 mg/kg-day, respectively and the inhalation RfD was also 2.00E-2 mg/kg-day. The dermal RfD for *trans*- and *cis*-1,2-dichloroethene was derived from the oral toxicity value using a gastrointestinal absorption factor of 100 percent.

1.4.2.8 1,3-Dichlorobenzene (CAS 000541-73-1)

There are no carcinogenic slope factors used in the BHHRA for 1,3-dichlorobenzene due to it being assigned a weight-of-evidence classification of D. No reference doses are available.

1.4.2.9 1,4-Dichlorobenzene (CAS 000106-46-7) (RAIS)

1,4-Dichlorobenzene, also referred to as para-DCB, p-DCB, paracide, Paramoth®, Parazene®, PDB, and Santochlor®, has a benzene ring with two chlorine atoms attached at the 1 and 4 carbon atoms; it does not occur naturally (ATSDR 1993c). 1,4-Dichlorobenzene is used to make mothballs, deodorant blocks used in restrooms, and in animal holding facilities to control odors (ATSDR 1993c). It also has applications in fumigants, insecticides, lacquers, paints, and seed disinfection products (Leber and Benya 1994). Of the 1300 sites on the EPA National Priorities List, 1,4-dichlorobenzene has been identified on at least 244 sites. Drinking water samples from U.S. surface water sources, environmental hazardous waste sites, and food have been reported to contain 1,4-dichlorobenzene (ATSDR 1993c).

Detectable concentrations of 1,4-dichlorobenzene were found in adipose tissue and blood samples taken from Tokyo residents (Morita and Ohi 1975, Morita et al. 1975). A national survey of various volatile organic chemicals demonstrated 1,4-dichlorobenzene in the three adipose tissues sampled. In addition, studies have shown that babies can receive 1,4-dichlorobenzene from mother's milk (ATSDR 1993c). 1,4-Dichlorobenzene is absorbed by experimental animals via inhalation, gavage, or subcutaneous injection (Hawkins et al. 1980). Data from oral administration of 1,4-dichlorobenzene to rabbits indicated oxidation to 2,5-dichlorophenol, which was found in the urine as a conjugate of glucuronic and sulfuric acids (Azouz et al. 1955). Other metabolites identified in the blood and urine of rats were 2,5-dichlorophenyl methyl sulfoxide and 2,5-dichlorophenyl methyl sulfone.

Severe hypochromic, microcytic anemia with excessive polychromasia, marginal nuclear hypersegmentation of the neutrophils, and a small number of red blood cells with Heinz bodies developed in a pregnant woman (21 years old) who consumed 1–2 blocks of 1,4-dichlorobenzene toilet air freshener per week throughout her pregnancy (Campbell and Davidson 1970). A 19-year-old female who consumed 4–5 moth pellets containing 1,4-dichlorobenzene on a daily basis for 2.5 years developed symmetrical, well-demarcated areas of increased pigmentation over various parts of her body, which disappeared over a 4-month period after discontinuing the ingestion (Frank and Cohen 1961).

In rats, 13-week gavage studies resulted in decreased hematocrit levels, red blood cell counts, and hemoglobin concentrations at 300 mg/kg-day (NTP 1987a). Oral administration of 1200 and

Rats exposed via inhalation to 96-341 ppm of 1,4-dichlorobenzene intermittently for 5-7 months had cloudy swelling and degeneration of hepatic parenchymal cells in the central zone of the liver. Increased liver weights in the male and/or female rats occurred above 96 ppm (Hollingsworth et al. 1956). During a 2-generation study, adult rats exposed to 538 ppm exhibited tremors, ataxia, and hyperactivity; decreased grooming behavior; and an unkempt appearance (Tyl and Neeper-Bradley 1989). Both generations of offspring in the 538 ppm group had lower body weights at lactation day 4, and average litter size and survival were decreased. Selected animals from the first filial generation still had reduced body weights at 5 weeks postexposure.

No epidemiologic studies or case reports addressing the carcinogenicity of 1,4-dichlorobenzene in humans were available. In a 2-year study, female rats and male and female mice were gavaged with 300 and 600 mg/kg-day, and male rats were gavaged with 150 and 300 mg/kg-day (NTP 1987a). Nephropathy, epithelial hyperplasia of the renal pelvis, mineralization of the collecting tubules in the renal medulla, and focal hyperplasia of the renal tubular epithelium were noted in male rats receiving 150 and 300 mg/kg-day. Female rats gavaged with 300 and 600 mg/kg-day had an increased incidence of nephropathy and minimal hyperplasia of the renal pelvis or tubules. The following tumors were described as being present in the animals: renal tubular adenocarcinomas in male rats (controls, 2 percent; low dose, 6 percent; high dose, 14 percent), a marginal increase in mononuclear cell leukemia in male rats (control. 10 percent; low dose, 14 percent; high dose, 22 percent), hepatocellular carcinomas in male mice (controls, 28 percent; low dose, 22.5 percent; high dose, 64 percent) and in female mice (controls, 10 percent; low dose, 10.4 percent; high dose, 38 percent), and hepatocellular adenomas in male mice (controls, 10 percent; low dose, 26.2 percent; high dose, 32 percent) and in female mice (controls, 20 percent; low dose, 12.5 percent; high dose, 42 percent). In this National Toxicology Program (NTP) study, the tumor incidence in female controls was higher than the historical control. In both male and female mice, hepatocellular degeneration with resultant initiation of tissue repair was present. These findings resulted in a speculation by NTP (1987a) that 1,4-dichlorobenzene was acting as a tumor promotor for liver tumors in male and female mice.

RfCs of 2.5 mg/m³ (0.42 ppm) for subchronic inhalation exposure (EPA 1998b) and 0.8 mg/m³ (0.13 ppm) for chronic inhalation exposure for 1,4-dichlorobenzene were derived (EPA 1998a) based on increased liver weights in the P1 males exposed via inhalation to 1,4-dichlorobenzene from the study of Tyl and Neeper-Bradley (1989). The NOAEL was 301 mg/m³ (50 ppm). The LOAEL was 902 mg/m³ (150 ppm) (EPA 1998a). 1,4-Dichlorobenzene has been classified as Group C, possible carcinogen to humans (EPA 1998b). For oral exposure, the slope factor was 2.40E-2 (mg/kg-day)⁻¹, and the unit risk was 6.80E-7 (μ g/L)⁻¹ (EPA 1998b).

An oral slope factor of 2.40E-2 (mg/kg-day)⁻¹ was used for 1,4-dichlorobenzene. For inhalation exposure, the inhalation unit risk is not available. The absorbed dose slope factor is 2.67E-2 (mg/kg-day)⁻¹.

An oral RfD is not available for 1,4-dichlorobenzene. The dermal route RfD is also not available. The inhalation RfC is $8.0E-1 \text{ mg/m}^3$ and the RfD is 2.29E-1 mg/kg-day.

1.4.2.10 2,4,5-Trichlorophenol (CAS 000095-95-4)

There are no carcinogenic slope factors used in the BHHRA for 2,4,5-trichlorophenol due to a lack of carcinogenic toxicity information.

An oral and inhalation RfD of 1.00E-1 mg/kg-day is used in this risk assessment for 2,4,5-trichlorophenol. The dermal route RfD used in the BHHRA is 5.00E-2 mg/kg-day. An inhalation RfC for 2,4,5-trichlorophenol is not available.

1.4.2.11 2,4,6-Trichlorophenol (CAS 000088-06-2)

There are no RfDs used in the BHHRA for 2,4,6-trichlorophenol due to a lack of noncarcinogenic toxicity information.

An oral slope factor of 1.10E-2 (mg/kg-day)⁻¹ is used in this risk assessment for 2,4,6-trichlorophenol. The dermal route slope factor used in the BHHRA is 2.20E-2 (mg/kg-day)⁻¹. The inhalation slope factor is 1.00E-2 (mg/kg-day)⁻¹. The inhalation unit risk is 3.10E-7 (μ g/L)⁻¹. The inhalation unit risk is 3.1E-6 (μ g/m³)⁻¹.

1.4.2.12 2,4-Dinitrotoluene (CAS 000121-14-2) (RAIS)

The chemical 2,4-Dinitrotoluene (2,4-DNT; 1-methyl-2,4-dinitrobenzene; CAS Reg. No. 121-14-2) is a yellow crystalline solid and one of six possible chemical forms of dinitrotoluene (DNT). Technical grade DNT (t-DNT) is typically composed of 78 percent 2,4-DNT, 19 percent 2,6-DNT, and small amounts of 3,4-DNT, 2,3-DNT, and 2,5-DNT (Dunlap 1978). 2,4-DNT is primarily used as a chemical intermediate in the manufacture of polyurethanes but also serves as a component of military and commercial explosives, as an intermediate in dye processes (Etnier 1987, Hawley 1981), and as a propellant additive (Sears and Touchette 1982).

The DNTs are absorbed through the gastrointestinal tract, respiratory tract, and skin in most species (EPA 1986j). The initial acute toxic effects of 2,4-DNT in humans include methemoglobinemia, cyanosis, and headache. Symptoms indicative of neurotoxicity are impaired reflexes, tremors, nystagmus, dizziness, and sleepiness (EPA 1980b). Subchronic and chronic oral toxicity studies with experimental animals indicate that the blood, liver, nervous system, and reproductive system are targets affected by 2,4-DNT. These effects were generally observed at doses of 5 mg/kg-day in rats and at 10 mg/kg-day in dogs. The most common hematological findings were methemoglobinemia, anemia, reticulocytosis, and an increase in Heinz bodies. Hepatotoxic effects included liver discoloration and proliferative alterations of hepatocytes and bile duct epithelium. Neuromuscular effects, ranging from tremors and ataxia to convulsions, were more severe in dogs than in rodents. Reproductive effects consisted of decreased spermatogenesis, testicular atrophy, and ovarian dysfunction (Lee et al. 1985; Ellis et al. 1985, 1979; Lee et al. 1978b).

The major route of exposure to DNT in the occupational setting is by inhalation. Effects reported in workers exposed to t-DNT and/or 2,4-DNT included ischemic heart disease, hematological effects characterized by cyanosis, anemia, and leukocytosis, and neurological effects such as dizziness, insomnia, nausea, and tingling pains in extremities (Levine et al. 1986, McGee et al. 1942). The evidence for potential reproductive effects (reduction of sperm counts) in male workers exposed to a mixture of DNT isomers and diaminotoluene is equivocal (Hamill et al. 1982, Ahrenholz 1980).

An oral RfD of 2.00E-03 mg/kg-day has been calculated for chronic (EPA 1995e) and subchronic exposure to 2,4-DNT (EPA 1998b), based on a NOAEL of 0.2 mg/kg-day derived from a chronic oral study with dogs conducted by Ellis et al. (1985). Data are inadequate for the calculation of an inhalation RfC (EPA 1995e).

An association between DNT exposure and increased risk of hepatobiliary cancer was found in a retrospective mortality study involving 4989 workers exposed to DNT (isomer composition not specified)

and 7436 unexposed controls at an U.S. Army munitions facility (Stayner et al. 1993). The carcinogenic --activity of 2,4-DNT and t-DNT has been studied in several chronic bioassays and in less than lifetime studies (Leonard et al. 1987, CIIT 1982, Ellis et al. 1979, NCI 1978c). 2,4-DNT (containing small amounts of 2,6-DNT) induced an increased incidence of hepatocellular carcinomas and subcutaneous tumors in rats and renal tumors in male mice (Ellis et al. 1979). In two rat studies, t-DNT induced hepatocellular carcinomas (Leonard et al. 1987, CIIT 1982). However, conclusions drawn from the isomer-specific carcinogenicity study by Leonard et al. (1987) and tumor-initiation/promotion assays by Popp and Leonard (1982) suggest that 2,6- rather than 2,4-DNT is the primary hepatocarcinogen in t-DNT. Although EPA has not evaluated pure 2,4-DNT for evidence of human carcinogenic potential, the dinitrotoluene mixture (containing 2,4-DNT and 2,6-DNT) was classified as a B2 chemical carcinogen, probable human carcinogen (EPA 1995f). A slope factor of 6.8E-1 (mg/kg-day)⁻¹ was calculated for oral exposure to the dinitrotoluene mixture. The drinking water unit risk is 1.9E-5 ($\mu g/L$)⁻¹ (EPA 1995f).

The oral and dermal cancer slope factors used in the BHHRA are 6.80E-1 and 8.00E-1 (mg/kg-day)⁻¹, respectively. An inhalation cancer slope factor was not found. The oral and dermal RfDs used in the BHHRA are 2.00E-3 and 1.70E-3 mg/kg-day, respectively. The inhalation RfD is 2.00E-3 mg/kg-day. When calculating the dermal route RfD and slope factor from the oral value, a gastrointestinal absorption factor of 85 percent was used.

1.4.2.13 2,6-Dinitrotoluene (CAS 000606-20-2) (RAIS)

2,6-Dinitrotoluene (2,6-DNT; 2-methyl-1,3-dinitrobenzene; CAS Reg. No. 606-20-2) is a pale yellow crystalline solid and one of six possible chemical forms of dinitrotoluene (DNT). Technical grade DNT (t-DNT) is typically composed of 78 percent 2,4-DNT, 19 percent 2,6-DNT, and small amounts of 3,4-DNT, 2,3-DNT, and 2,5-DNT (Dunlap 1978). DNT is primarily used as a chemical intermediate in the manufacture of polyurethanes. It is also used as a component of military and commercial explosives, as an intermediate in dye processes (Etnier 1987, Hawley 1981), and as a propellant additive (Sears and Touchette 1982).

The DNTs are absorbed through the gastrointestinal tract, respiratory tract, and skin in most species (EPA 1986j). Human data regarding potential health effects of 2,6-DNT are very limited. A significant increase in the death rate due to ischemic heart disease has been associated with occupational exposure to t-DNT (Levine et al. 1986). The evidence for potential reproductive effects (reduction of sperm counts) in male workers exposed to a mixture of DNT isomers is equivocal (Hamill et al. 1982, Ahrenholz 1980).

Oral subchronic toxicity studies with rats, mice, and dogs indicate that the blood, liver, and reproductive system are targets affected by 2,6-DNT in all three species (Lee et al. 1976). These effects were generally observed at doses of 35 mg/kg-day in rats, 51 mg/kg-day in mice, and 20 mg/kg-day in dogs. The primary hematologic effect in all three species was methemoglobinemia with sequelae such as Heinz bodies, reticulocytosis, anemia, and extramedullary hematopoiesis. Also seen in all three species were bile duct hyperplasia, decreased spermatogenesis, and testicular atrophy. In addition, dogs exhibited neurotoxic effects (incoordination, weakness, tremors, and paralysis) as well as inflammatory and degenerative kidney changes.

According to EPA (1998b), available data are inadequate for the calculation of a RfD or RfC for 2,6-DNT.

In a 1-year carcinogenesis bioassay, 2,6-DNT at oral doses of 7 and 14 mg/kg-day, respectively, produced hepatocellular carcinomas in 85 percent and 100 percent of male rats. t-DNT, containing about 76 percent 2,4-DNT and 19 percent 2,6-DNT, also yielded a positive hepatocarcinogenic response (Leonard et al. 1987). In another study on the effects of t-DNT, dietary doses of 14 mg/kg-day induced

The oral and dermal cancer slope factors used in the BHHRA are 6.80E-1 and 8.0E-1 (mg/kg-day)⁻¹, respectively. An inhalation cancer slope factor was not found. The oral and dermal chronic RfDs used in the BHHRA are 1.00E-3 and 8.50E-4 mg/kg-day, respectively. The inhalation RfD was 1.00E-3 mg/kg-day. When calculating the dermal route RfD and slope factor from the oral value, a gastrointestinal absorption factor of 85 percent was used.

1.4.2.14 2-Chloronaphthalene (CAS 000091-58-7)

There are no carcinogenic slope factors used in the BHHRA for 2-chloronaphthalene due to a lack of carcinogenic toxicity information.

An oral RfD of 8.00E-2 mg/kg-day is used in this risk assessment for 2-chloronaphthalene. The dermal route RfD used in the BHHRA is 4.00E-2 mg/kg-day. An inhalation RfC is not available.

1.4.2.15 2-Hexanone (methyl butyl ketone) (CAS 000591-78-6) (ATSDR)

2-Hexanone, also known as methyl n-butyl ketone, is a flammable, colorless liquid with a pungent acetone-like odor. It is known to occur in nature in very low concentrations (HSDB 1993). 2-Hexanone has been used as a solvent for lacquers, dye printing, ink and paint thinners, resins, oils, fats and waxes (HSDB 1993, Klaassen et al. 1986). 2-Hexanone is no longer made in the United States, and its uses have been restricted because of its harmful health effects (ATSDR 1990i). 2-Hexanone is a waste product of wood pulping, coal gasification, and oil shale operations (ATSDR 1990i). 2-Hexanone is very soluble in water and soil. Biodegradation may occur slowly in water and soil, but bioconcentration is not expected (ATSDR 1990i).

Inhalation, ingestion, and dermal absorption are possible routes of exposure. The most common effect of 2-hexanone is weight loss, or in the case of developing animals, decreased weight gain. Neurological effects have been observed in humans that were occupationally exposed to 2-hexanone, and animal studies have shown neurological effects as well as possible hematological and reproductive effects (ATSDR 1990i). 2-Hexanone applied to the eyes of rabbits resulted in moderate corneal necrosis, and when applied to skin, it caused irritation (ATSDR 1990i).

Neither slope factors nor RfDs for any route of exposure were found for 2-hexanone. Therefore, neither carcinogenicity nor systemic toxicity due to 2-hexanone exposure is included in the BHHRA.

1.4.2.16 2-Methyl-4,6-dinitrophenol (CAS 000534-52-1)

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure for 2-methyl-4,6-dinitrophenol. Therefore, quantitative estimates of carcinogenicity and systemic toxicity due to 2-methyl-4,6-dinitrophenol exposure is not included in the BHHRA.

1.4.2.17 2-Methylnaphthalene (CAS 000091-57-6)

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure for 2-methylnaphthalene. Therefore, quantitative estimates of carcinogenicity and systemic toxicity due to 2-methylnaphthalene exposure is not included in the BHHRA.

1.4.2.18 2-Nitroaniline (CAS 000088-74-4)

There are no carcinogenic slope factors used in the BHHRA for 2-nitroaniline due to a lack of carcinogenic toxicity.

The oral, inhalation and dermal RfDs are 6.00E-5, 571E-5 and 3.00E-5 mg/kg-day respectively.

1.4.2.19 2-Nitrophenol (CAS 000088-75-5)

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure for 2-nitrophenol. Therefore, quantitative estimates of carcinogenicity and systemic toxicity due to 2-nitrophenol exposure are not included in the BHHRA.

1.4.2.20 3,3'-Dichlorobenzidine (CAS 000091-94-1)

An oral slope factor of 4.50E-1 (mg/kg-day)⁻¹ was used for 3,3'-dichlorobenzidine. For inhalation exposure, the inhalation unit risk is not available. The absorbed dose slope factor is 9.00E- $1(mg/kg-day)^{-1}$. The oral unit risk is 1.30E-5 (μ g/L)⁻¹.

There are no RfDs or RfCs used in the BHHRA for 3,3'-dichlorobenzidine due to a lack of systemic toxicity and noncarcinogenic risk information.

1.4.2.21 3-Nitroaniline (CAS 000099-09-2)

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure for 3-niroaniline. Therefore, quantitative estimates of carcinogenicity and systemic toxicity due to 3-niroaniline exposure are not included in the BHHRA.

1.4.2.22 4,4'-DDD (CAS 000072-54-8)

An oral slope factor of 2.40E-1 (mg/kg-day)⁻¹ was used for 4,4'-DDD. For inhalation exposure, the inhalation unit risk is not available. The absorbed dose slope factor is 3.43E-1 (mg/kg-day)⁻¹. The oral unit risk is 6.90E-6 (µg/L)⁻¹.

There are no RfDs or RfCs used in the BHHRA for 4,4'-DDD due to a lack of systemic toxicity and noncarcinogenic risk information.

1.4.2.23 4,4'-DDE (CAS 000072-55-9)

An oral slope factor of 3.40E-1 (mg/kg-day)⁻¹ was used for 4,4'-DDE. For inhalation exposure, the inhalation unit risk is not available. The absorbed dose slope factor is 4.86E-1 (mg/kg-day)⁻¹. The oral unit risk is 9.70E-6 (μ g/L)⁻¹.

There are no RfDs or RfCs used in the BHHRA for 4,4'-DDE due to a lack of systemic toxicity and noncarcinogenic risk information.

1.4.2.24 4,4'-DDT (CAS 000050-29-3)

An oral slope factor of 3.40E-1 (mg/kg-day)⁻¹ was used for 4,4'-DDT. For inhalation exposure, the inhalation slope factor is 3.40E-1 (mg/kg-day)⁻¹, and the inhalation unit risk is 9.70E-5 m³/ μ g. The absorbed dose slope factor is 4.86E-1 (mg/kg-day)⁻¹.

An oral RfD of 5.00E-4 mg/kg-day is used in this risk assessment for 4,4'-DDT. The dermal route RfD used in the BHHRA is 3.50E-4 mg/kg-day. The inhalation RfC is not available.

1.4.2.25 4-Bromophenyl-phenylether (CAS 000101-55-3)

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure for 4-bromophenyl-phenylether. Therefore, quantitative estimates of carcinogenicity and systemic toxicity due to 4-bromophenyl-phenylether exposure are not included in the BHHRA.

1.4.2.26 4-Chloro-3-methylphenol (CAS 000059-50-7)

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure for 4-chloro-3-methylphenol. Therefore, quantitative estimates of carcinogenicity and systemic toxicity due to 4-chloro-3-methylphenol exposure are not included in the BHHRA.

1.4.2.27 4-Chlorophenyl-phenylether(CAS 007005-72-3)

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure for 4-chlorophenyl-phenylether. Therefore, quantitative estimates of carcinogenicity and systemic toxicity due to 4-chlorophenyl-phenylether exposure are not included in the BHHRA.

1.4.2.28 4-Nitroaniline (CAS 000100-01-6)

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure for 4-nitroaniline. Therefore, quantitative estimates of carcinogenicity and systemic toxicity due to 4-nitroaniline exposure are not included in the BHHRA.

1.4.2.29 Acenaphthene (CAS 000083-32-9) (RAIS)

Acenaphthene, also known as 1,2-dihydroacenaphthylene or 1,8-ethylenenaphthalene, is a tricyclic aromatic hydrocarbon that occurs in coal tar. It is used as a dye intermediate, in the manufacture of some plastics, and as an insecticide and fungicide (EPA 1980c). Acenaphthene has been detected in cigarette smoke, automobile exhausts, and urban air; in effluents from petrochemical, pesticide, and wood preservative industries (EPA 1980c); and in soils, groundwater, and surface waters at hazardous waste sites (ATSDR 1990j).

No absorption data are available for acenaphthene; however, by analogy to structurally related PAHs, it would be expected to be absorbed from the gastrointestinal tract and lungs (EPA 1988a). The anhydride of naphthalic acid was identified as a urinary metabolite in rats treated orally with acenaphthene (Chang and Young 1943).

Although a large body of literature exists on the toxicity and carcinogenicity of PAHs, primarily benzo(a)pyrene, toxicity data for acenaphthene are limited. Acenaphthene is irritating to the skin and mucous membranes of humans and animals (Sandmeyer 1981, Knobloch et al. 1969). Acute toxicity data for animals include oral LD_{50} s of 10 g/kg for rats and 2.1 g/kg for mice (Knobloch et al. 1969) and an

intraperitoneal LD_{50} of 600 mg/kg for rats (Reshetyuk et al. 1970). Oral exposure of rats to daily ---2-g doses of acenaphthene for 32 days produced peripheral blood changes, mild liver and kidney damage, and pulmonary effects (Knobloch et al. 1969). Subchronic oral exposure to acenaphthene at doses of greater than or equal to 350 mg/kg for 90 days produced increased liver weights, hepatocellular hypertrophy, and increased cholesterol levels in mice. Reproductive effects included decreased ovary weights at doses of greater than or equal to 350 mg/kg and decreased ovarian and uterine activity as well as smaller and fewer corpora lutea at 700 mg/kg-day (EPA 1989e). Adverse effects on the blood, lungs, and glandular tissues were reported in rats exposed daily to 12 mg/m³ of acenaphthene for 5 months (Reshetyuk et al. 1970).

An RfD of 6E-1 mg/kg-day for subchronic oral exposure (EPA 1998b) and 6E-2 mg/kg-day for chronic oral exposure to acenaphthene (EPA 1998a) was calculated from a NOAEL of 175 mg/kg-day from a 90-day gavage study with mice. The critical effect was hepatotoxicity. Data were insufficient to derive an inhalation RfC for acenaphthene (EPA 1998a and b).

No oral bioassays were available to assess the carcinogenicity of acenaphthene. A limited inhalation study in which rats were exposed to 12 mg/m³ acenaphthene for 5 months and observed an additional 8 months provided no evidence of carcinogenicity (Reshetyuk et al. 1970). EPA has not assigned a weight-of-evidence classification for carcinogenicity to acenaphthene (EPA 1998a and b).

No cancer slope factors were used in the BHHRA for acenaphthene. The oral, inhalation and dermal RfDs used in the BHHRA are 6.00E-2, 6.00E-2 and 1.86E-2 mg/kg-day, respectively. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 31 percent was used.

1.4.2.30 Acenaphthylene (CAS 000208-96-8)

Neither slope factors nor RfDs for any route of exposure were found for acenaphthylene. Therefore, neither carcinogenicity nor systemic toxicity due to acenaphthylene exposure is included in the BHHRA.

1.4.2.31 Acetone (CAS 000067-64-1) (RAIS)

Acetone is a clear, colorless, highly flammable liquid with a vapor pressure of 182 mm Hg at 20°C (Morgott 1993). It is completely miscible in water and soluble in organics such as benzene and ethanol (ATSDR 1994). Its log octanol-water partitioning coefficient has been estimated to be -0.24 (ATSDR 1994). Acetone is used primarily as a solvent and chemical intermediate, and it is also found in some consumer products such as nail polish remover (Inoue 1983, Kumai et al. 1983).

Acetone may be released into the environment as stack emissions and/or fugitive emissions and in wastewater effluents from facilities involved in its production and use as a chemical intermediate and solvent (HSDB 1995). Acetone is also a natural metabolic by-product found in and released from plants and animals. Much of the acetone released into the environment will volatilize into the atmosphere where it will be subject to photo-oxidation (average half-life is 22 days). Volatilization from surface waters is moderately rapid (estimated half-life about 20 hours from a model river). If released onto the ground, acetone will both volatilize and leach into the soil, and relatively little will be adsorbed to soil particles (HSDB 1995). Acetone has been detected in groundwater and drinking water.

Acetone can be absorbed through the lungs, digestive tract, and the skin (Morgott 1993). It is rapidly transported throughout the body and is not preferentially stored in any body tissue (Morgott 1993). The liver is the major organ of acetone metabolism, and excretion occurs mainly through the lungs and in the urine.

Acute toxic effects following ingestion of 50 mL or more may include ataxia, sedation, and coma; --respiratory depression; gastrointestinal disorders (vomiting and hematemesis); hyperglycemia and ketonemia; acidosis; and hepatic and renal lesions (Krasavage et al. 1982). Ingestion of 10-20 mL (7.9-15.8 g) generally is not toxic (HSDB 1995), and consumption of 20 g/day for several days resulted in only slight drowsiness (Morgott 1993). The minimum lethal dose for a 150-lb man is estimated to be 100 mL (79.1 g). No information is available on the subchronic or chronic oral toxicity to humans. In animal studies, subchronic oral exposures were associated with kidney damage and hematological changes.

The RfD for chronic oral exposures, 0.1 mg/kg-day (EPA 1995g), is based on increased liver and kidney weights and nephrotoxicity in rats (EPA 1986k). The subchronic oral RfD of 1 mg/kg-day (EPA 1998b) is based on the same rodent study.

Information on the inhalation toxicity of acetone to humans is derived from occupational and laboratory studies. Typical symptoms of inhalation exposure are CNS depression and irritation of the mucous membranes of the eyes, nose, and throat (Morgott 1993). CNS effects can range from subtle neurobehavioral changes to narcosis depending on the magnitude and length of exposure. Neurobehavioral changes have been reported at concentrations as low as 237 ppm (574 mg/m³) (Dick et al. 1989). Irritant effects have been reported at concentrations of 500 ppm (1210 mg/m³) and higher. Transient effects were reported in workers exposed to 600-2150 ppm (1452-5203 mg/m³) (EPA 1995g). Extremely high concentrations (> 29 g/m³) can cause dizziness, confusion, unsteadiness, and unconsciousness (ATSDR 1994). Prolonged occupational exposures to acetone vapors have not been associated with chronic systemic disorders (Morgott 1993).

Studies have shown that acetone vapor concentrations in excess of 8000 ppm (19.36 mg/m³) are generally required to produce signs of CNS depression in animals, but concentrations as low as 500 ppm (1210 mg/m³) may cause subtle behavioral changes (Morgott 1993, ATSDR 1994). Little information is available on subchronic or chronic inhalation toxicity in animals.

An inhalation RfC has not been derived for acetone (EPA 1995g).

Animal data indicate that acetone is not teratogenic; however, adverse reproductive effects may occur at high concentrations. Drinking water concentrations equal to doses > 3 g/kg-day during pregnancy were associated with spermatogenic effects, reduced reproductive index, and decreased pup survival of rodents (Larsen et al. 1991, EHRT 1987). Inhalation exposure to 11,000 ppm resulted in reduction in maternal body weight gain, a decrease in uterine and extragestational weight gain, and a significant reduction in fetal weight of rats but no adverse effects on reproduction or development (Mast et al. 1988). In the latter study, incidence of malformations was not increased by exposure to acetone.

No evidence is available that suggests acetone is carcinogenic in humans or animals (Morgott 1993). Negative results have been reported in occupational exposure studies and in rodent skin painting studies. Although acetone has not been tested in a 2-year rodent bioassay, in vitro tests for mutagenicity, chromosome damage, and deoxyribonucleic acid (DNA) interaction indicate that acetone is not genotoxic except under severe conditions (Morgott 1993). Acetone is classified by EPA in weight-of-evidence Group D, not classifiable as to human carcinogenicity (EPA 1995g).

There are no carcinogenic slope factors used in the BHHRA for acetone due to a lack of carcinogenic toxicity information.

1.4.2.32 Aldrin (CAS 000309-00-2)

An oral slope factor of 1.70E+1 (mg/kg-day)⁻¹ was calculated for aldrin. For inhalation exposure, the inhalation unit risk is 4.90E+0 m³/g. The absorbed dose slope factor is 3.40E+1 (mg/kg-day)⁻¹.

An oral and inhalation RfD of 3.00E-5 mg/kg-day is used in this risk assessment for aldrin. The dermal route RfD used in the BHHRA is 1.50E-5 mg/kg-day. The inhalation RfC is not available.

1.4.2.33 alpha-BHC (CAS 000319-84-6)

There are no carcinogenic slope factors used in the BHHRA for alpha-benzene hexachloride (BHC) due to a lack of carcinogenic toxicity information.

An oral RfD of 6.30E+0 mg/kg-day is used in this risk assessment for alpha-BHC. The dermal route RfD used in the BHHRA is 6.49E+0 mg/kg-day. The inhalation RfC is 1.80E+0 mg/m³.

1.4.2.34 alpha-Chlordane (CAS 005103-71-9)

The oral, inhalation and dermal slope factors for alpha-chlordane and gamma-chlordane used in the BHHRA were 3.50E-1, 1.30E+0 and 7.0E+0 (mg/kg-day)⁻¹.respectively. The oral and dermal route RfDs were 5.00E-4 mg/kg-day and 2.5E-4 mg/kg-day respectively and the inhalation RfD was 2.00E-4 mg/kg-day.

1.4.2.35 Anthracene (CAS 000120-12-7) (RAIS)

Anthracene, also referred to as paranaphthalene or green oil, is a PAH derived from coal tar and is primarily used as an intermediate in the production of dyes. It has also been used in the production of smoke screens. Anthracene is ubiquitous in the environment as a product of incomplete combustion of fossil fuels. Although a large body of literature exists on the toxicity and carcinogenicity of a number of PAHs, toxicity data for anthracene are limited.

Evidence indicates that anthracene is absorbed following oral and dermal exposure. Targets for anthracene toxicity are the skin, hematopoietic system, lymphoid system, and gastrointestinal tract. Adverse dermatologic effects have been observed in humans and animals in conjunction with acute and subchronic exposure to anthracene. In humans, anthracene may cause acute dermatitis with symptoms of burning, itching, and edema. Prolonged dermal exposure produces pigmentation, cornification of skin surface layers, and telangiectasis (Volkova 1983). Anthracene is photosensitizing, potentiating skin damage elicited by exposure to ultraviolet (UV) radiation (EPA 1987h, Dayhaw-Barker et al. 1985, Forbes et al. 1976). Hematologic toxicity was observed in patients receiving intraperitoneal injections of anthracene-containing chemotherapeutic agents (Falkson et al. 1985) and in rats exposed to anthracene by oral gavage and by inhalation (Volkova 1983). Mice receiving subcutaneous injections of anthracene exhibited adverse lymphoid effects (Hoch-Ligeti 1941). Long-term use of anthracene-containing laxatives produced melanosis of the colon and rectum (Badiali et al. 1985). Human exposure to anthracene has also been associated with headache, nausea, loss of appetite, inflammation of the gastrointestinal tract, slow reactions, and weakness (Volkova 1983).





An RfD of 3 mg/kg-day for subchronic oral exposure and 0.3 mg/kg-day for chronic oral exposure to anthracene was calculated from a NOAEL of 1000 mg/kg-day derived from a 90-day gavage study with mice (EPA 1989e). Data were insufficient to derive an inhalation RfC for anthracene (EPA 1991h and i).

Carcinogenicity bioassays with anthracene generally gave negative results. Studies involving oral administration (Druckrey and Schmahl 1955, Schmahl 1955) or intrapulmonary implantation in rats (Stanton et al. 1972) or implantation into the brain of rabbits (Russell 1947) provided no evidence of carcinogenicity. Negative results were also obtained when anthracene was tested in mice by skin application (Wynder and Hoffman 1959a, Pollia 1939, Kennaway 1924a and b) and in mouse-skin initiation assays (LaVoie et al. 1979, Scribner 1973). However, skin application of anthracene followed by exposure to UV radiation or visible light induced a high incidence of skin tumors in mice (Heller 1950).

Based on no human data and inadequate data from animal bioassays, EPA (1991h and i) has placed anthracene in weight-of-evidence Group D, not classifiable as to human carcinogenicity.

No cancer slope factors were used in the BHHRA for anthracene. The oral and dermal RfDs used in the BHHRA are 3.00E-1 and 2.28E-1 mg/kg-day, respectively. The inhalation RfD was 3.00E-1 mg/kg-day. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 76 percent was used.

1.4.2.36 Benz(a)anthracene (CAS 000056-55-3) (RAIS)

Benz(a)anthracene, along with a number of other PAHs, are natural products produced by the incomplete combustion of organic material. The arrangement of the aromatic rings in the benz(a)anthracene molecule gives it a "bay region" often correlated with carcinogenic properties. In general, the bay-region PAHs and some of their metabolites are known to react with cellular macromolecules, including DNA, which may account for both their toxicity and carcinogenicity. The inducible mixed-function oxidase enzymes oxidize benz(a)anthracene to form metabolites with increased water solubility that can be efficiently excreted in the urine. A minor product of this oxidation, a bay-region diol epoxide, reacts readily with DNA and has been shown to be highly carcinogenic (EPA 1980d, 1984d; Jerina et al. 1977).

The toxic effects of benz(a)anthracene and similar PAHs are primarily directed toward tissues that contain proliferating cells. Animal studies indicate that exposure to bay-region PAHs can damage the hematopoietic system, leading to progressive anemia as well as agranulocytosis (Robinson et al. 1975, Cawein and Sydnor 1968). The lymphoid system can also be affected, resulting in lymphopenia. Toxic effects have been observed in the rapidly dividing cells of the intestinal epithelium, spermatogonia and resting spermatocytes in the testis and primary oocytes of the ovary (Philips et al. 1973; Mackenzie and Angevine 1981; Kraup 1970; Ford and Huggins 1963; Mattison and Thorgeirsson 1977; EPA 1980d, 1984d). Most of these effects have occurred following both oral and parenteral exposure. Epithelial proliferation and cell hyperplasia in the respiratory tract have been reported following subchronic inhalation exposure (Reznik-Schuller and Mohr 1974, Saffiotti et al. 1968). However, because of the lack of quantitative data, neither a RfD nor RfC have been derived (EPA 1991j).

The primary concern with benz(a)anthracene exposure is its potential carcinogenicity. There is no unequivocal, direct evidence of the carcinogenicity of the compound to humans. However, benz(a)anthracene and other known carcinogenic PAHs are components of coal tar, soot, coke oven emissions, and tobacco smoke. There is adequate evidence of its carcinogenic properties in animals. Oral exposures of mice to benz(a)anthracene have resulted in hepatomas, pulmonary adenomas and forestomach papillomas (Klein 1963, Bock and King 1959, EPA 1991j). The EPA weight-of-evidence classification is B2, probable human carcinogen, for both oral and inhalation exposure based on adequate ---animal evidence and no human evidence (EPA 1991j). A slope factor has not been derived specifically for benz(a)anthracene by EPA (EPA 1991j). However, an oral slope factor of 7.3 (mg/kg-day)⁻¹ has been calculated for benzo(a)pyrene based on the incidence of stomach tumors in mice treated with benzo(a)pyrene (Neal and Rigdon 1967; EPA 1980d, 1984d, 1992c). A drinking water unit risk of 2.1E-4 (µg/L)⁻¹ has also been calculated for benzo(a)pyrene (EPA 1992c).

The oral, dermal, and inhalation cancer slope factors used in the BHHRA for benz(a)anthracene are 7.30E-1, 2.35E+0, and 3.10E-1 (mg/kg-day)⁻¹, respectively. These were derived from the values for benzo(a)pyrene using the relative potency factors recommended by EPA. The dermal slope factor was derived from the oral slope factor using a gastrointestinal absorption factor of 31 percent. No RfDs for benz(a)anthracene were found; therefore, noncancer effects due to exposure to benz(a)anthracene could not be estimated in the BHHRA.

1.4.2.37 Benzene (CAS 000071-43-2) (RAIS)

Benzene is absorbed via ingestion, inhalation, and skin application. Experimental data indicate that animals can absorb up to 95 percent of oral doses and that humans can absorb up to 80 percent of inhaled benzene (after 5 minutes of exposure) (Sabourin et al. 1987, Srobova et al. 1950). Humans may absorb benzene vapors through the skin as well as the lungs; of the total dose absorbed by the two routes, an estimated 22–36 percent, enters the body through the skin (Susten et al. 1985).

Autopsy of a youth who died while sniffing benzene revealed that the chemical was distributed to the urine, stomach, bile, liver, kidney, abdominal fat, and brain (Winek and Collum 1971). The depots for benzene and its metabolites in animals are similar to those in humans and, in addition, include the fetus and placenta, bone marrow, Zymbal gland, and oral and nasal cavities (Ghantous and Danielsson 1986, Rickert et al. 1979, Low et al. 1989).

Numerous studies indicate that the metabolism of benzene is required for its toxicity (Kalf et al. 1987). The liver is the main site for the metabolism of benzene; the bone marrow, a minor site (ATSDR 1992b). Phenol, hydroquinone, catechol, and benzene oxide are the major metabolites (Kalf et al. 1987, Snyder 1987). The metabolite(s) of benzene that are responsible for its toxicity have not been positively identified, but likely candidates include muconaldehyde, quinones, and free radicals generated by oxidizing enzymes (Henderson et al. 1989, Snyder 1987).

Benzene is eliminated either unchanged in expired air or as metabolites in the urine (ATSDR 1992b). The proportions of the administered dose excreted by each route and the half-times for excretion are dependent on route, dose, and duration of exposure.

Lethal oral doses of benzene are estimated to be 10 mL in humans; oral LD_{50} values for benzene in rats range from 0.93 to 5.96 g/kg (Cornish and Ryan 1965, Withey and Hall 1975). These data indicate that benzene is of low acute toxicity (O'Bryan and Ross 1988).

Limited data show that nonlethal oral doses of benzene can impact the nervous, hematological, and immunological systems. Ingested benzene produces symptoms of neurotoxicity at acute doses of 2 mL for humans and 325 mg/kg for rats (Thienes and Haley 1972, Clayton and Clayton 1981, Cornish and Ryan 1965). A 4-week exposure of mice to greater than or equal to 8 mg of benzene/kg/day in drinking water induced synthesis and catabolism of monoamine neurotransmitters and produced dose-related decreases in red blood cell parameters and lymphocyte numbers (Hsieh et al. 1988). Rats and mice that were treated with benzene by gavage for 103 weeks developed a dose-related lymphocytopenia (LOAEL, 25 mg/kg-

Inhalation of benzene vapor concentrations of 20,000 ppm for 5-10 minutes can be fatal to humans; death results from CNS depression (Clayton and Clayton 1981). The estimated LD₅₀ value for the rat is 13,700 ppm (Drew and Fouts 1974).

As with orally administered benzene, the targets for nonlethal concentrations of inhaled benzene include the nervous, hematological, and immunological systems. Neurological symptoms in humans may appear at exposure concentrations of 700 ppm (Clayton and Clayton 1981). In animals, 1 week of exposure to 300 ppm induced behavioral effects (Drew and Fouts 1974), and 1–4 weeks of exposure to benzene concentrations ranging from 21–50 ppm suppressed the bone marrow (NOAEL, 10 ppm) (Cronkite et al. 1985, Toft et al. 1982), the cellular immune response (NOAEL, 10 ppm) (Rosenthal and Snyder 1985), and the humoral immune response (LOAEL, 50 ppm) (Aoyama 1986).

Subchronic and chronic exposures to benzene vapors induce a progressive depletion of the bone marrow and dysfunction of the hematopoietic system. Early symptoms of bone marrow depression include leukopenia, anemia or thrombocytopenia, or a combination of the three (Snyder 1984). A group of workers exposed to benzene concentrations of 30 and 150 ppm for 4 months to 1 year had increased incidences of pancytopenia (Aksoy et al. 1971, Aksoy et al. 1972, Aksoy and Erdem 1978). A group of patients who had been exposed to benzene concentrations of 150–650 ppm for 4 months to 15 years exhibited severe blood dyscrasias and 8 of the 32 patients died with thrombocytopenic hemorrhage and infection (Aksoy et al. 1972). The human data are supported by animal data showing bone marrow suppression in mice and rats exposed to benzene concentrations ranging from 10 ppm for 24 weeks to 300 ppm for 13 weeks (Baarson et al. 1984, Ward et al. 1985).

Benzene may also have long-term effects on the CNS. Workers exposed to benzene for 0.5-4 years exhibited EEG changes and atypical sleep activity consistent with neurotoxicity (Kellerova 1985). Others exposed to benzene concentrations of 210 ppm for 6-8 years had peripheral nerve damage (Baslo and Aksoy 1982).

In humans, benzene crosses the placenta and is present in the cord blood in amounts equal to those in maternal blood (Dowty et al. 1976); however, studies of the effects of benzene on human reproduction and development have been confounded by the presence of other chemicals in the environment (USAF 1989b). Benzene does produce developmental effects (fetal toxicity, but not malformations) in the offspring of treated animals, mostly at maternally toxic doses (Nawrot and Staples 1979, Seidenberg et al. 1986, Keller and Snyder 1988).

RfDs and RfCs for benzene have not been established. An oral risk assessment for benzene will be reviewed by an EPA work group, and an inhalation risk assessment is currently under review (EPA 1992d).

Benzene is carcinogenic in humans and animals by inhalation and in animals by the oral route of exposure. Occupational exposure to benzene has been associated mainly with increased incidences of acute myeloblastic or erythroblastic leukemias and chronic myeloid and lymphoid leukemias among workers (Aksoy 1989). Workers at risk were exposed in one study to 8-hour TWA concentrations ranging from 10 to 100 ppm (Rinsky et al. 1981) and in another to 8-hour TWA concentrations ranging from less than 2 to greater than 25 ppm (Ott et al. 1978). In a historical prospective mortality study of chemical workers, Yin et al. (1987) described a dose-response relationship between exposure to benzene and lymphatic and hematopoietic cancers, which adds strength to the association between exposure in the workplace and cancer development. Studies in animals have demonstrated an association between oral

and inhalation exposure to benzene and the development of a variety of tumors, including lymphoma and ----carcinomas of the Zymbal gland, oral cavity, mammary gland, ovaries, lung, and skin (Huff et al. 1989, Maltoni et al. 1989). In one study, C57Bl/BNL mice had increased incidences of leukemia, lymphoma, and solid tumors after exposure to 300 ppm for only 16 weeks (Cronkite et al. 1985, Cronkite 1986).

Based on several studies of increased incidence of nonlymphocytic leukemia from occupational exposure, increased incidence of neoplasia in rats and mice exposed by inhalation and gavage, and some supporting data, benzene has been placed in the EPA weight-of-evidence classification A, human carcinogen (EPA 1991d).

The oral and inhalation slope factors for benzene are 2.90E-2 $(mg/kg-day)^{-1}$, and the oral and inhalation unit risk values are 8.30E-7 $(\mu g/L)^{-1}$ and 8.3E-6 $(\mu g/m^3)^{-1}$, respectively, based on the studies of Ott et al. (1978), Rinsky et al. (1981), and Wong et al. (EPA 1992d, 1998b). No oral and dermal RfDs were found. The inhalation RfD is 1.71E-3 mg/kg-day.

1.4.2.38 Benzo(a)pyrene (CAS 000050-32-8) (RAIS)

Benzo(a)pyrene is a PAH that can be derived from coal tar. Benzo(a)pyrene occurs ubiquitously in products of incomplete combustion of fossil fuels and has been identified in ambient air, surface water, drinking water, wastewater, and char-broiled foods (IARC 1983a). Benzo(a)pyrene is primarily released to the air and removed from the atmosphere by photochemical oxidation and dry deposition to land or water. Biodegradation is the most important transformation process in soil or sediment (ATSDR 1990k).

Benzo(a)pyrene is readily absorbed following inhalation, oral, and dermal routes of administration (ATSDR 1990k). Following inhalation exposure, benzo(a)pyrene is rapidly distributed to several tissues in rats (Sun et al. 1982, Weyand and Bevan 1986). The metabolism of benzo(a)pyrene is complex and includes the formation of a proposed ultimate carcinogen, benzo(a)pyrene 7,8 diol-9,10-epoxide (IARC 1983a). The major route of excretion is hepatobiliary followed by elimination in the feces (EPA 1991k).

No data are available on the systemic (noncarcinogenic) effects of benzo(a)pyrene in humans. In mice, genetic differences appear to influence the toxicity of benzo(a)pyrene. Subchronic dietary administration of 120 mg/kg benzo(a)pyrene for up to 180 days resulted in decreased survival due to hematopoietic effects (bone narrow depression) in a "nonresponsive" strain of mice (i.e., a strain whose cytochrome P-450 mediated enzyme activity is not induced as a consequence of PAH exposure). No adverse effects were noted in "responsive" mice (i.e., a strain capable of inducing increased cytochrome P-450 mediated enzyme activity as a consequence of PAH exposure) (Robinson et al. 1975). Immunosuppression has been reported in mice administered daily intraperitoneal injections of 40 or 160 mg/kg of benzo(a)pyrene for 2 weeks, with more pronounced effects apparent in "nonresponsive" mice (Blanton et al. 1986, White et al. 1985b). In utero exposure to benzo(a)pyrene has produced adverse developmental/reproductive effects in mice. Dietary administration of doses as low as 10 mg/kg during gestation caused reduced fertility and reproductive capacity in offspring (Mackenzie and Angevine 1981), and treatment by gavage with 120 mg/kg-day during gestation caused stillbirths, resorptions, and malformations (Legraverend et al. 1984). Similar effects have been reported in intraperitoneal injection studies (ATSDR 1990k). Neither a RfD nor a RfC has been derived for benzo(a)pyrene.

Numerous epidemiologic studies have shown a clear association between exposure to various mixtures of PAHs containing benzo(a)pyrene (e.g., coke oven emissions, roofing tar emissions, and cigarette smoke) and increased risk of lung cancer and other tumors. However, each of the mixtures also contained other potentially carcinogenic PAHs; therefore, it is not possible to evaluate the contribution of benzo(a)pyrene to the carcinogenicity of these mixtures (IARC 1983a, EPA 1991k). An extensive database is available for the carcinogenicity of benzo(a)pyrene in experimental animals. Dietary

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administration of benzo(a)pyrene has produced papillomas and carcinomas of the forestomach in mice (Neal and Rigdon 1967), and treatment by gavage has produced mammary tumors in rats (McCormick et al. 1981) and pulmonary adenomas in mice (Wattenberg and Leong 1970). Exposure by inhalation and intratracheal instillation has resulted in benign and malignant tumors of the respiratory and upper digestive tracts of hamsters (Ketkar et al. 1978, Thyssen et al. 1981). Numerous topical application studies have shown that benzo(a)pyrene induces skin tumors in several species, although mice appear to be the most sensitive species. Benzo(a)pyrene is a complete carcinogen and also an initiator of skin tumors (IARC 1973a, EPA 1991k). Benzo(a)pyrene has also been reported to induce tumors in animals when administered by other routes, such as intravenous, intraperitoneal, subcutaneous, intrapulmonary, and transplacental.

Based on EPA guidelines, benzo(a)pyrene was assigned to weight-of-evidence group B2, probable human carcinogen. For oral exposure, the slope factor and unit risk are 7.30E+0 (mg/kg-day)⁻¹ and 2.10E-4 (ug/L)⁻¹, respectively (EPA 1998a).

The oral, dermal, and inhalation cancer slope factors used in the BHHRA for benzo(a)pyrene are 7.30, 2.35E+1, and 3.10E+0 (mg/kg-day)⁻¹, respectively. The dermal slope factor was derived from the oral slope factor using a gastrointestinal absorption factor of 31 percent. No RfDs for benzo(a)pyrene were found; therefore, noncancer effects due to exposure to benzo(a)pyrene could not be estimated in the BHHRA.

1.4.2.39 Benzo(b)fluoranthene (CAS 000205-99-2) (RAIS)

Benzo(b)fluoranthene, a crystalline solid with a chemical formula of $C_{20}H_{12}$ and a molecular weight of 252.32 (Lide 1991), is a PAH with one five-membered ring and four six-membered rings. There is no commercial production or known use of this compound (IARC 1983b). Benzo(b)fluoranthene is found in fossil fuels and occurs ubiquitously in products of incomplete combustion. It has been detected in mainstream cigarette smoke; urban air; gasoline engine exhaust; emissions from burning coal and from oil-fired heating; broiled and smoked food; oils and margarine (IARC 1983b); and in soils, groundwater, and surface waters at hazardous waste sites (ATSDR 1990j).

No absorption data were available for benzo(b)fluoranthene; however, by analogy to structurally related PAHs, primarily benzo(a)pyrene, it would be expected to be absorbed from the gastrointestinal tract, lungs, and skin (EPA 1991k). Major metabolites of benzo(b)fluoranthene formed in vitro in rat liver include dihydrodiols and monohydroxy derivatives (Amin et al. 1982) and monohydroxy derivatives in mouse epidermis (Geddie et al. 1987).

No data were found concerning the acute, subchronic, chronic, developmental, or reproductive toxicity of benzo(b)fluoranthene. No data were available for the derivation of an oral RfD or inhalation RfC (EPA 1998a).

No long-term oral or inhalation bioassays were available to assess the carcinogenicity of benzo(b)fluoranthene. Benzo(b)fluoranthene was tested for carcinogenicity in dermal application, lung implantation, subcutaneous injection, and intraperitoneal injection studies. Dermal applications of 0.01-0.5 percent solutions of benzo(b)fluoranthene for life produced a high incidence of skin papillomas and carcinomas in mice (Wynder and Hoffmann 1959b). In initiation-promotion assays, the compound was active as an initiator of skin carcinogenesis in mice (LaVoie et al. 1982a, Amin et al. 1985a). Sarcomas and carcinomas of the lungs and thorax were seen in rats receiving single lung implants of 0.1-1 mg benzo(b)fluoranthene (Deutsch-Wenzel et al. 1983). Newborn mice receiving 0.5 umol benzo(b)fluoranthene via intraperitoneal injection developed liver and lung tumors (LaVoie et al. 1987),

and mice administered three subcutaneous injections of 0.6 mg benzo(b)fluoranthene developed injection .-- site sarcomas (IARC 1983).

Based on no human data and sufficient evidence for carcinogenicity in animals, EPA has assigned a weight-of-evidence classification of B2, probable human carcinogen, to benzo(b)fluoranthene (EPA 1998a).

The oral, dermal, and inhalation cancer slope factors used in the BHHRA for benzo(b)fluoranthene are 7.30E-1, 2.35E+0, and 3.10E-1 (mg/kg-day)⁻¹, respectively. The inhalation unit risk is 8.80E-2 m³/ μ g. These were derived from the values for benzo(a)pyrene using the relative potency factors recommended by EPA. The dermal slope factor was derived from the oral slope factor using a gastrointestinal absorption factor of 31 percent. No RfDs for benzo(b)fluoranthene were found; therefore, noncancer effects due to exposure to benzo(b)fluoranthene could not be estimated in the BHHRA.

1.4.2.40 Benzo(g,h,i)perylene (CAS 000191-24-2) (RAIS)

Benzo(g,h,i)perylene, also known as 1,12-benzoperylene, is a PAH with six aromatic rings. There is no known commercial production or use of benzo(g,h,i)perylene. It occurs naturally in crude oils and is present ubiquitously in products of incomplete combustion and in coal tar (EPA 1987i).

No absorption data were available for benzo(g,h,i)perylene; however, by analogy to other PAHs, primarily benzo(a)pyrene, it would be expected to be absorbed from the gastrointestinal tract, lungs, and skin (EPA 1991k).

No human or animal data were available to evaluate the toxicity of benzo(g,h,i)perylene. Because of the lack of data, EPA has not derived an oral RfD or inhalation RfC (EPA 1998a).

No oral or inhalation bioassays were available to assess the carcinogenicity of benzo(g,h,i)perylene. Negative results were reported in dermal application studies (Hoffmann and Wynder 1966, Van Duuren and Goldschmidt 1976) and in initiation-promotion assays for skin tumorigenesis in mice (Hoffmann and Wynder 1966, Van Duuren et al. 1970). However, when benzo(g,h,i)perylene was administered simultaneously with benzo(a)pyrene to the skin of mice, an increased incidence of skin tumors was observed compared to the tumor incidence in mice treated with benzo(a)pyrene alone, indicating possible cocarcinogenic activity of benzo(g,h,i)perylene (Van Duuren et al. 1973). Although a few pulmonary tumors were observed in Osborne-Mendel rats when benzo(g,h,i)perylene was administered as single lung implants of greater than or equal to 83 mg (Deutsch-Wenzel et al. 1983), the tumors may have been caused by impurities in the test compound (IARC 1983c). In subcutaneous injection studies, benzo(g,h,i)perylene did not produce injection site tumors in mice (Muller 1968).

Based on no human data and inadequate data with experimental animals, EPA has classified benzo(g,h,i)perylene in weight-of-evidence Group D, not classifiable as to human carcinogenicity (EPA 1998a).

Neither slope factors nor RfDs for any route of exposure were found for benzo(g,h,i)perylene. Therefore, neither carcinogenicity nor systemic toxicity due to benzo(g,h,i)perylene exposure is included in the BHHRA.

1.4.2.41 Benzo(k)fluoranthene (CAS 000207-08-9) (RAIS)

Benzo(k)fluoranthene, a crystalline solid with a chemical formula of $C_{20}H_{12}$ and a molecular weight of 252.32 (Lide 1991), is a PAH with one five-membered and four six-membered rings. There is no

commercial production or known use of this compound (IARC 1983b). Benzo(k)fluoranthene is found in fossil fuels and occurs ubiquitously in products of incomplete combustion (IARC 1983b) and in soils, groundwater, and surface waters at hazardous waste sites (ATSDR 1990j).

No absorption or excretion data were available for benzo(k)fluoranthene; however, by analogy to structurally related PAHs, primarily benzo(a)pyrene, it would be expected to be absorbed from the gastrointestinal tract, lungs, and skin (EPA 1991k). Rat liver microsomes have been shown to metabolize benzo(k)fluoranthene to dihydrodiol, 8,9-dihydro-8,9-dihydroxy benzo(k)fluoranthene (LaVoie et al. 1980).

No data were found concerning the acute, subchronic, chronic, developmental, or reproductive toxicity of benzo(k)fluoranthene. Because of a lack of toxicity data, an oral RfD or inhalation RfC have not been derived (EPA 1998a).

No long-term oral or inhalation bioassays were available to assess the carcinogenicity of benzo(k)fluoranthene. Benzo(k)fluoranthene was tested for carcinogenicity in dermal application, subcutaneous injection, lung implantation, and intraperitoneal injection studies. Dermal applications of 0.5 percent solutions of benzo(k)fluoranthene for life produced only a few skin papillomas in mice (Wynder and Hoffmann 1959b), but in initiation-promotion assays, benzo(k)fluoranthene was active as an initiator of skin carcinogenesis (LaVoie et al. 1982a, Amin et al. 1985b). Injection site sarcomas developed in mice given three subcutaneious injections of 0.6 mg benzo(k)fluoranthene (Lacassagne et al. 1963), and dose-related increases of epidermoid carcinomas of the lungs were reported in rats receiving single lung implants of 0.16–4.15 mg benzo(k)fluoranthene (Deutsch-Wenzel et al. 1983). In a short-term assay, hepatic and lung tumors occurred in newborn mice receiving 2.1 umol benzo(k)fluoranthene via intraperitoneal injection (LaVoie et al. 1987).

Based on no human data and sufficient evidence for carcinogenicity in animals, EPA has assigned a weight-of-evidence classification of B2, probable human carcinogen, to benzo(k)fluoranthene (EPA 1998a).

The oral, dermal, and inhalation cancer slope factors used in the BHHRA for benzo(k)fluoranthene are 7.30E-2, 2.35E-1, and 3.10E-2 (mg/kg-day)⁻¹, respectively. These were derived from the values for benzo(a)pyrene using the relative potency factors recommended by EPA. The dermal slope factor was derived from the oral slope factor using a gastrointestinal absorption factor of 31 percent. No RfDs for benzo(k)fluoranthene were found; therefore, noncancer effects due to exposure to benzo(k)fluoranthene could not be estimated in the BHHRA.

1.4.2.42 beta-BHC (CAS 000319-85-7)

An oral slope factor of 1.80E+0 $(mg/kg-day)^{-1}$ was used for beta-BHC. For inhalation exposure, the inhalation unit risk is 5.30E-1 m³/µg. The absorbed dose slope factor is 1.98E+0 $(mg/kg-day)^{-1}$.

There are no RfDs or RfCs used in the BHHRA for beta-BHC due to a lack of systemic toxicity and noncarcinogenic risk information.

1.4.2.43 bis(2-Chloroethoxy)methane (CAS 000111-91-1)

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure for bis(2-chloroethoxy)methane. Therefore, quantitative estimates of carcinogenicity and systemic toxicity due to bis(2-chloroethoxy)methane exposure is not included in the BHHRA.

1.4.2.44 bis(2-Chloroethyl)ether (CAS 000111-44-4)

An oral slope factor of 1.1E+0 (mg/kg-day)⁻¹ was used for bis(2-chloroethyl)ether. For inhalation exposure, the inhalation unit risk is 3.30E-4 (μ g/m³)⁻¹. The absorbed dose slope factor is 2.20E+0 (mg/kg-day)⁻¹.

There are no RfDs or RfCs used in the BHHRA for bis(2-chloroethyl)ether due to a lack of systemic toxicity and noncarcinogenic risk information.

1.4.2.45 bis(2-Chloroisopropyl)ether (CAS 039638-32-9)

There are no slope factors used in the BHHRA for bis(2-chloroisopropyl)ether due to a lack of carcinogenic risk information.

A chronic oral RfD of 4.00E-2 mg/kg-day was used for bis(2-chloroisopropyl)ether. The chronic absorbed RfD is 2.00E-2 mg/kg-day and the inhalation RfD is 4.00E-2 mg/kg-day.

1.4.2.46 bis(2-Ethylhexyl)phthalate (CAS 000117-81-7) (RAIS)

bis(2-Ethylhexyl)phthalate is a colorless, oily liquid that is extensively used as a plasticizer in a wide variety of industrial, domestic, and medical products. It is an environmental contaminant and has been detected in groundwater, surface water, drinking water, air, soil, plants, fish, and animals (Sittig 1985b, Sandmeyer and Kirwin 1978). It is rapidly absorbed from the gastrointestinal tract primarily as mono(2-ethylhexyl)phthalate (Pollack et al. 1985, Teirlynck and Belpaire 1985). The diester can be absorbed through the skin and from the lungs (Elsisi et al. 1989, Pegg 1982). It is rapidly metabolized in the blood and tissues to the monoester, which can be excreted as a glucuronide conjugate or further hydrolyzed to phthalic acid and excreted (Kluwe et al. 1982, Albro et al. 1982).

Animal studies have indicated that the primary target organs are the liver and kidneys (Carpenter et al. 1953, EPA 1987j,k); however, higher doses are reported to result in testicular effects and decreased hemoglobin and packed cell volume (Kluwe et al. 1982, Gray et al. 1977). The primary intracellular effects of bis(2-ethylhexyl)phthalate in the liver and kidneys are an increase in the smooth endoplasmic reticulum and a proliferation in the number and size of peroxisomes (Kluwe et al. 1982, Reddy and Lalwani 1983, Tomaszewski et al. 1986). An epidemiological study reported no toxic effects from occupational exposure to air concentrations of bis(2-ethylhexyl)phthalate up to 0.16 mg/m³ (Thiess et al. 1978). Other studies on occupational exposures to mixtures of phthalate esters containing bis(2-ethylhexyl)phthalate have reported polyneuritis and sensory-motor polyneuropathy with decreased thrombocytes, leukocytes, and hemoglobin in some exposed workers (Milkov et al. 1973, Gilioli et al. 1978). Developmental toxicity studies with rats and mice have shown that bis(2-ethylhexyl)phthalate is fetotoxic and teratogenic when given orally during gestation (Wolkowski-Tyl et al. 1984a, 1984b; Shiota and Mima 1985). Oral exposure has also been shown to result in decreased sperm count in rats (Siddipui and Srivastava 1992).

An RfD of 2.00E-2 mg/kg-day for both subchronic and chronic oral exposure was calculated from a LOAEL of 19 mg/kg-day based on increased relative liver weight in guinea pigs given 0, 19, or 64 mg bis(2-ethylhexyl)phthalate/kg/day for 12 months in their diet (Carpenter et al. 1953, EPA 1998a,b). An RfC for inhalation exposure is not available (EPA 1998b).

bis(2-Ethylhexyl)phthalate is known to induce the proliferation of peroxisomes, which has been associated with carcinogenesis (Rao and Reddy 1991). Dose-dependent, statistically significant increases in the incidences of hepatocellular carcinomas and combined carcinomas and adenomas were seen in mice and rats exposed to bis(2-ethylhexyl)phthalate in their diet for 103 weeks (Kluwe et al. 1982). An \cdots increased incidence of neoplastic nodules and hepatocellular carcinomas was also reported in rats (Rao et al. 1990).

Based on EPA guidelines, bis(2-ethylhexyl)phthalate was assigned to weight-of-evidence Group B2, probable human carcinogen, on the basis of an increased incidence of liver tumors in rats and mice. A carcinogenicity slope factor (q_1^*) of 0.014 $(mg/kg-day)^{-1}$ for oral exposure was based on the combined incidence of hepatocellular carcinomas and adenomas in male mice (Kluwe et al. 1982, EPA 1998b). A drinking water unit risk of 4.0E-7 $(\mu g/L)^{-1}$ was calculated based on the q_1^* . A quantitative estimation of carcinogenic risk from inhalation exposure is not available (EPA 1998b).

The oral and dermal cancer slope factors used in the BHHRA for bis(2-ethylhexyl)phthalate are 1.40E-2 and 7.35E-2 (mg/kg-day)⁻¹, respectively. An inhalation cancer slope factor was not found: however, based on the whole body effects discussed previously, the oral slope factor, 1.40E-2 (mg/kg-day)⁻¹, is used as a surrogate inhalation slope factor in the uncertainty discussion in Sect. 1.6. The oral and dermal RfDs used in the BHHRA are 2.00E-2 and 3.80E-3 mg/kg-day, respectively. A inhalation RfD was not found; however, based on the whole body effects discussed previously, the oral RfD, 2.00E-2 mg/kg-day, is used as a surrogate inhalation RfD in the uncertainty discussion in Sect. 1.6. When calculating both the dermal route cancer slope factor and dermal route RfD from their respective oral values, a gastrointestinal absorption factor of 19 percent was used.

1.4.2.47 Bromodichloromethane (CAS 000075-27-4)

Bromodichloromethane is a colorless liquid that boils at 89.2°–90.6°C. It is soluble in water, alcohol, ether, acetone, benzene, and chloroform. Bromodichloromethane is used in the synthesis of organic chemicals and as a reagent in laboratory research (EPA 1980a, Sittig 1985a). It has also been used to separate minerals and salts, as a flame retardant, in fire extingishers, and as a solvent for waxes, fats, and resins (HSDB 1995).

There is sufficient evidence for the carcinogenicity of bromodichloromethane in experimental animals (NTP 1987b). When administered by gavage, bromodichloromethane increased the incidences of tubular cell adenomas and adenocarcinomas in the kidney and adenocarcinomas and adenomatous polyps in the large intestine in rats of both sexes. When administered by gavage, bromodichloromethane increased the incidences of tubular cell adenomas and adenocarcinomas in the kidney of male mice and increased the incidences of hepatocellular adenomas and carcinomas in female mice (NTP 1987b, ATSDR 1989).

The oral and dermal and inhalation RfDs for bromodichlormethane are 2.00E-2 mg/kg-day, 1.96E-2 mg/kg-day, and 2.00E-2 mg/kg-day respectively. The oral and dermal slope factors used in the BHHRA are 6.20E-2 and 6.33E-2 (mg/kg-day)⁻¹, respectively. An inhalation slope factor was not found.

1.4.2.48 Butyl benzyl phthalate (CAS 000085-68-7)

Butyl benzyl phthalte is also known as BBP; n-butyl benzyl phthalate; 1,2-benzenedicarboxylic acid butyl phenylmethyl ester; benzyl butyl phthalate; benzyl n-butyl phthalate; butyl phenylmethyl 1,2-benzenedicarboxylate; santicizer 160; palatinol bb; sicol 160; and unimoll bb.

No cancer slope factors were used in the BHHRA for butyl benzyl phthalate. The oral and dermal RfDs used in the BHHRA are 2.00E-1 and 1.22E-1 mg/kg-day, respectively. An inhalation RfD equivalent to the oral RfD of 2.00E-1 mg/kg-day was used. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 61 percent was used.

1.4.2.49 Carbon tetrachloride (CAS 000056-23-5) (RAIS)

Humans are sensitive to carbon tetrachloride intoxication by oral, inhalation, and dermal routes. Oral and inhalation exposure to high concentrations of carbon tetrachloride results in acute CNS effects including dizziness, vertigo, headache, depression, confusion, incoordination and, in severe cases, respiratory failure, coma, and death. Gastrointestinal problems, including nausea, abdominal pain, and diarrhea, often accompany these narcotic effects. Liver and kidney damage can appear after the acute symptoms subside. All symptoms can occur following a single oral or inhalation exposure. Milder narcotic effects followed by liver and kidney damage have been reported following dermal exposure. Although an inhalation exposure of about 1000 ppm for a few minutes to hours will cause the narcotic effects in 100 percent of the population, large variations in sensitivity are seen. Alcohol intake greatly increases human sensitivity to carbon tetrachloride; consequently, exposure to 250 ppm for 15 minutes can be life- threatening to an alcoholic.

Subchronic and chronic exposure to doses as low as 10 ppm can result in liver and kidney damage (Sax and Lewis 1989, ATSDR 1989g). Lung damage has also been reported in animals and humans but is not route specific and is believed to be secondary to kidney damage (Sax and Lewis 1989). Prolonged exposure has been observed to cause visual effects in both humans and animals. Changes in the visual field, reduced corneal sensitivity, subnormal dark adaption, and changes in color perception have been reported in humans exposed by inhalation to a minimum concentration of 6.4 ppm, 1 hour/day for an average of 7.7 years. Increased hepatic enzyme activities indicative of liver damage have also been observed (Smyth et al. 1936, Barnes and Jones 1967, Moeller 1973, ATSDR 1989g).

Maternal toxicity and fetotoxic effects have been reported in rats following oral or inhalation exposure to carbon tetrachloride during gestation (Wilson 1954, Schwetz et al. 1974a). Repeated inhalation exposure of male rats to carbon tetrachloride concentrations of 200 ppm or greater has been reported to cause degeneration of the testicular germinal epithelium as well as severe liver and kidney damage (Adams et al. 1952).

A subchronic reference dose (RfD_s) of 0.007 mg/kg-day has been calculated for oral exposure from a NOAEL of 0.71 mg/kg-day determined in a 12-week rat study (EPA 1998b). Significantly higher doses caused minimal liver damage (Bruckner et al. 1986). A dose of 7.1 mg/kg-day was considered a LOAEL. A chronic reference dose (RfD_c) of 0.0007 mg/kg-day was calculated by adding an additional uncertainty factor of 10 to account for the use of a subchronic study. EPA (1998a) rates confidence in the oral RfD values as medium.

EPA is currently developing RfD_c or RfD_s for inhalation exposure.

Although data for the carcinogenicity of carbon tetrachloride in humans are inconclusive, there is ample evidence in animals that the chemical can cause liver cancer. Hepatocellular carcinomas have been induced in hamsters, rats, and mice after oral carbon tetrachloride treatment for 16–76 weeks. Liver tumors have also been demonstrated in rats following inhalation exposure, but the doses were not quantitatively established. The EPA weight-of-evidence classification for both oral and inhalation exposure is B2, probable human carcinogen, based on adequate animal evidence. Carcinogenicity slope factors of 0.13 (mg/kg-day)⁻¹ for oral exposure and 0.053 (mg/kg-day)⁻¹ for inhalation exposure have been calculated from the oral exposure experiments with hamsters, rats, and mice (EPA 1998a, 1998b; Della Porta et al. 1961; Edwards et al. 1942; NCI 1976a, 1976b; and Weisburger 1977). A drinking water unit risk of 3.7E-6 (μ g/L)⁻¹ and an inhalation unit risk of 1.5E-5 (μ g/m³)⁻¹ have also been calculated by EPA (1998a).



The oral and dermal cancer slope factors used in the BHHRA for carbon tetrachloride are 1.30E-1 and $2.00E-1 (mg/kg-day)^{-1}$, respectively. An inhalation cancer slope factor of $5.30E-2 (mg/kg-day)^{-1}$ is used. The oral and dermal RfDs used in the BHHRA are 7.00E-4 and 4.55E-4 mg/kg-day, respectively. A provisional inhalation RfD of 5.71E-4 mg/kg-day was used to estimate the noncarcinogenic hazard from the inhalation pathway. When calculating both the dermal route cancer slope factor and dermal route RfD from their respective oral values, a gastrointestinal absorption factor of 65 percent was used.

1.4.2.50 Chloroform (CAS 000067-66-3) (RAIS)

Chloroform is a colorless, volatile liquid that is widely used as a general solvent and as an intermediate in the production of refrigerants, plastics, and pharmaceuticals (Torkelson et al. 1976, IARC 1979b). Chloroform is rapidly absorbed from the lungs and the gastrointestinal tract and, to some extent, through the skin. It is extensively metabolized in the body with carbon dioxide as the major end product. The primary sites of metabolism are the liver and kidneys. Excretion of chloroform occurs primarily via the lungs, either as unchanged chloroform or as carbon dioxide (ATSDR 1989h).

Target organs for chloroform toxicity are the liver, kidneys, and CNS. Liver effects (hepatomegaly, fatty liver, and hepatitis) were observed in individuals occupationally exposed to chloroform (Bornski et al. 1967). Several subchronic and chronic studies by the oral or inhalation routes of exposure documented hepatotoxic effects in rats, mice, and dogs (Palmer et al. 1979, Munson et al. 1982, Heywood et al. 1979). Renal effects were reported in rats and mice following oral and inhalation exposures (Roe et al. 1979, Reuber 1979, Torkelson et al. 1976), but evidence for chloroform-induced renal toxicity in humans is sparse. Chloroform is a CNS depressant, inducing narcosis and anesthesia at high concentrations. Lower concentrations may cause irritability, lassitude, depression, gastrointestinal symptoms, and frequent and burning urination (ATSDR 1989h).

Developmental toxicity studies with rodents indicate that inhaled and orally administered chloroform is toxic to dams and fetuses. Possible teratogenic effects were reported in rats and mice exposed to chloroform by inhalation (Schwetz et al. 1974c, Murray et al. 1982b). Chloroform may cause sperm abnormalities in mice and gonadal atrophy in rats (Palmer et al. 1979, Reuber 1979, Land et al. 1981).

An RfD of 1.00E-2 mg/kg-day for subchronic and chronic oral exposure was calculated from a LOAEL of 15 mg/kg-day based on fatty cyst formation in the liver of dogs exposed to chloroform for 7.5 years (Heywood et al. 1979). Development of an inhalation RfC is presently under review (EPA 1992e).

Epidemiological studies indicate a possible relationship between exposure to chloroform present in chlorinated drinking water and cancer of the bladder, large intestine, and rectum. Chloroform is one of several contaminants present in drinking water, but it has not been identified as the sole or primary cause of the excess cancer rate (ATSDR 1989h, EPA 1985b). In animal carcinogenicity studies, positive results included increased incidences of renal epithelial tumors in male rats, hepatocellular carcinomas in male and female mice, and kidney tumors in male mice (Jorgensen et al. 1985, Roe et al. 1979, NCI 1976c).

Based on EPA guidelines, chloroform was assigned to weight-of-evidence Group B2, probable human carcinogen, on the basis of an increased incidence of several tumor types in rats and in three strains of mice. The carcinogen slope factor (q_1°) for chloroform is 6.1E-3 $(mg/kg-day)^{-1}$ for oral exposure (EPA 1992e) and 8.1E-2 $(mg/kg-day)^{-1}$ for inhalation exposure (EPA 1998b). An inhalation unit risk of 2.3E-5 $(\mu g/m^3)^{-1}$ is based on hepatocellular carcinomas in mice in an oral gavage study (EPA 1992e).

The oral and dermal cancer slope factors used in the BHHRA for chloroform are 6.10E-3 and 3.05E-2 (mg/kg-day)⁻¹, respectively. An inhalation cancer slope factor of 8.10E-2 (mg/kg-day)⁻¹ is used.

The oral and dermal RfDs used in the BHHRA are 1.00E-2 and 2.00E-3 mg/kg-day, respectively. An---inhalation RfD is not used. When calculating both the dermal route cancer slope factor and dermal route RfD from their respective oral values, a gastrointestinal absorption factor of 20 percent was used.

1.4.2.51 Chrysene (CAS 000218-01-9) (RAIS)

Chrysene, a PAH, is a ubiquitous environmental contaminant formed primarily by the incomplete combustion of organic compounds. Although present in coal and oil, the presence of chrysene in the environment is the result of anthropogenic activities such as coal combustion and gasification; gasoline exhaust; diesel and aircraft exhaust; and emissions from coke ovens, wood burning stoves, and waste incineration (IARC 1983c, ATSDR 19901). Chrysene is not produced or used commercially, and its use is limited strictly to research applications.

Little information on the absorption, distribution, metabolism, and excretion of chrysene in humans is available. Animal studies have shown that approximately 75 percent of the administered chrysene may be absorbed by oral, dermal, or inhalation routes (Grimmer et al. 1988, Modica et al. 1983, Chang 1943). Following its absorption, chrysene is preferentially distributed to highly lipophilic regions of the body, most notably adipose and mammary tissue (Bartosek et al. 1984, Modica et al. 1983). Phase I metabolism of chrysene, whether in the lung, skin, or liver, is mediated by the mixed function oxidases. The metabolism results in the formation of 1,2-, 3,4-, and 5,6-dihydrodiols as well as the formation of 1-, 3-, and 4-phenol metabolites (Sims 1970, Nordquist et al. 1981, Jacob et al. 1982 and 1987). Additional Phase I metabolism of chrysene 1,2-dihydrodiol forms chrysene 1,2-dihydrodiol-3,4-epoxide and 9-hydroxychrysene 1,2-diol-3,4-oxide. These metabolites were shown to have mutagenic and alkylating activity (Hodgson et al. 1983, Wood et al. 1977, Wood et al. 1979). Phase II metabolism of chrysene results in the formation of glucuronide and sulfate ester conjugates; however, glutathione conjugates of diol- and triol-epoxides are also formed (Sims and Grover 1974 and 1981, Hodgson et al. 1986, Robertson and Jernström 1986). Hepatobiliary secretion with elimination in the feces is the predominant route of excretion (Schlede et al. 1970, Grimmer et al. 1988).

Human or animal systemic, developmental, and reproductive health effects following exposure to chrysene were not identified. Because of the lack of systemic toxicity data, the RfD and the RfC for chrysene have not been derived (EPA 1994i and 1998b). Target organs have not been described, although chrysene may induce immunosuppression similar to certain other PAHs. Oral and inhalation carcinogenic bioassays were not identified. In mouse skin painting studies, chrysene was an initiator of papillomas and carcinomas. In addition, intraperitoneal injections of chrysene have induced liver adenomas and carcinomas in male CD-1 and BLU/Ha Swiss mice. Although oral and inhalation slope factors have not been derived, EPA (1994i and 1998b) has classified chrysene in weight-of-evidence Group B2, probable human carcinogen, based on the induction of liver tumors and skin papillomas and carcinomas following treatment and the mutagenicity and chromosomal abnormalities induced in in vitro tests.

The oral, dermal, and inhalation cancer slope factors used in the BHHRA for chrysene are 7.30E-3, 2.35E-2, and 3.10E-3 (mg/kg-day)⁻¹, respectively. The inhalation unit risk is 8.80E-7 m³/ μ g. These were derived from the values for benzo(a)pyrene using the relative potency factors recommended by EPA. The dermal slope factor was derived from the oral slope factor using a gastrointestinal absorption factor of 31 percent. No RfDs for chrysene were found; therefore, noncancer effects due to exposure to chrysene could not be estimated in the BHHRA.

1.4.2.52 cis-1,2-Dichloroethene

See Sect. 1.4.2.7.

1.4.2.53 cis-1,3-Dichloropropene (CAS 010061-01-5)

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure for cis-1,3-dichloropropene. Therefore, quantitative estimates of carcinogenicity and systemic toxicity due to cis-1,3-dichloropropene exposure is not included in the BHHRA.

1.4.2.54 delta-BHC (CAS 000319-86-8)

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure for delta-BHC. Therefore, quantitative estimates of carcinogenicity and systemic toxicity due to delta-BHC exposure is not included in the BHHRA.

1.4.2.55 Dibenz(a,h)anthracene (CAS 000053-70-3) (RAIS)

Dibenz(a,h)anthracene is a PAH with five aromatic rings. No commercial production or use of dibenz(a,h)anthracene is known. It occurs as a component of coal tars, shale oils, and soots (IARC 1985) and has been detected in gasoline engine exhaust, coke oven emissions, cigarette smoke, charcoal broiled meats, vegetation near heavily traveled roads, and surface water and soils near hazardous waste sites (ATSDR 1993d, IARC 1983e).

Dibenz(a,h)anthracene is poorly absorbed from the gastrointestinal tract and is primarily excreted via feces (Chang 1943). Following absorption, dibenz(a,h)anthracene is distributed to various tissues, with highest accumulation in the liver and kidneys (Daniel et al. 1967). Dibenz(a,h)anthracene is metabolized by mixed function oxidases to dihydrodiols. Epoxidation of the 3,4-dihydrodiol may lead to the formation of a diol-epoxide, the putative ultimate carcinogenic metabolite of dibenz(a,h)anthracene (Buening et al. 1979a).

No human studies were available to evaluate the toxicity of dibenz(a,h)anthracene. In animals, depressed immune responses were observed in mice following single or multiple subcutaneous injections of dibenz(a,h)anthracene (White et al. 1985b). Weekly subcutaneous injections of 0.05 percent dibenz(a,h)anthracene for 40 weeks produced lymphoid tissue changes, decreased spleen weights, and liver and kidney lesions in mice (Hoch-Ligeti 1941). Weekly intramuscular injections of 20 mg/kg promoted the development of arteriosclerotic plaques in chickens (Penn and Snyder 1988).

EPA has not derived an oral RfD or inhalation RfC for dibenz(a,h)anthracene (EPA 1998a).

No epidemiologic studies or case reports addressing the carcinogenicity of dibenz(a,h)anthracene in humans were available. In animals, dibenz(a,h)anthracene has produced tumors by different routes of administration, having both local and systemic carcinogenic effects.

After oral administration, dibenz(a,h)anthracene produced tumors at several sites. Male and female mice fed dibenz(a,h)anthracene (0.85 mg/day for males, 0.76 mg/day for females) in an aqueous olive oil emulsion developed pulmonary adenomatosis, alveologenic carcinomas of the lung, hemangioendotheliomas of the pancreas and mesentery/abdominal lymph nodes, and mammary carcinomas (females) after 200 days (Snell and Stewart 1962). A single oral dose of 1.5 mg dibenz(a,h)anthracene in polyethylene glycol produced a low incidence of forestomach papillomas in mice (Berenblum and Haran 1955). Mammary carcinomas developed in mice treated by gavage with a total dose of 15 mg over a 15-week period (Biancifiori and Caschera 1962).

Carcinogenic as well as tumor-initiating activity of dibenz(a,h)anthracene has been demonstrated in topical application studies with mice. Repeated dermal application of 0.001-0.01 percent solutions

produced a high incidence of skin papillomas and carcinomas in mice (Wynder and Hoffmann 1959b, ----Van Duuren et al. 1967). In initiation-promotion assays, the compound was active as an initiator of skin carcinogenesis in mice (Buening et al. 1979a, Platt et al. 1990). However, no skin tumors were observed in Syrian golden hamsters that received topical dibenz(a,h)anthracene applications over a 10-week period (Shubik et al. 1960): Injection site sarcomas developed in mice injected subcutaneously with dibenz(a,h)anthracene (Pfeiffer 1977). In newborn mice, a single subcutaneous injection of dibenz(a,h)anthracene induced local sarcomas and lung adenomas (Platt et al. 1990), and three intraperitoneal injections induced a high incidence of pulmonary tumors (Buening et al. 1979a). A number of earlier studies have also demonstrated the carcinogenicity of dibenz(a,h)anthracene when administered by various parenteral routes in several animal species (IARC 1973b).

Based on no human data and sufficient evidence for carcinogenicity in animals, EPA has assigned dibenz(a,h)anthracene a weight-of-evidence classification of B2, probable human carcinogen (EPA 1998a).

The oral, dermal, and inhalation cancer slope factors used in the BHHRA for dibenz(a,h)anthracene are 7.30E+0, 2.35E+1, and 3.10E+0 (mg/kg-day)⁻¹, respectively. The inhalation unit risk is 8.80E-4 m³/µg. These were derived from the values for benzo(a)pyrene using the relative potency factors recommended by EPA. The dermal slope factor was derived from the oral slope factor using a gastrointestinal absorption factor of 31 percent. No RfDs for dibenz(a,h)anthracene were found; therefore, noncancer effects because of exposure to dibenz(a,h)anthracene could not be estimated in the BHHRA.

1.4.2.56 Dibenzofuran (CAS 000132-64-9)

There are no carcinogenic slope factors used in the BHHRA for dibenzofuran due to a lack of carcinogenic toxicity information.

An oral reference dose of 4.00E-3 mg/kg-day is used in this risk assessment for dibenzofuran. The dermal route RfD used in the BHHRA is 2.00E-3 mg/kg-day. The inhalation RfC is 1.40E-2 mg/m³.

1.4.2.57 Dieldrin (CAS 000060-57-1)

An oral slope factor of 1.6E+1 (mg/kg-day)⁻¹ was calculated for dieldrin. For inhalation exposure, the inhalation unit risk is 4.60E-3 m³/µg. The absorbed dose slope factor is 3.20E+1 (mg/kg-day)⁻¹.

An oral RfD of 5.00E-5 mg/kg-day is used in this risk assessment for dieldrin. The dermal route RfD used in the BHHRA is 2.50E-5 mg/kg-day. The inhalation RfD is 5.00E-5 mg/kg-day.

1.4.2.58 Di-n-butyl phthalate (CAS 84-74-2)

Di-n-butyl phthalate is also known as DBP; dibutyl phthalate; n-butylphthalate; 1,2-benzenedicarboxylic acid dibutyl ester; phthalic acid dibutyl ester; o-benzenedicarboxylic acid, dibutyl ester; benzene-o-dicarboxylic acid di-n-butyl ester; dibutyl 1,2-benzenedicarboxylate; celluflex dpb; elaol; hexaplas m/b; palatinol c; polycizer dbp; PX 104; staflex dbp; witcizer 300; benzenedicarboxylic acid, dibutyl ester; and dibutyl-o-phthalate.

No cancer slope factors were used in the BHHRA for di-n-butyl phthalate. The oral and dermal RfDs used in the BHHRA are 1.00E-1 and 1.00E-1 mg/kg-day, respectively. An inhalation RfD was not found. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 100 percent was used.



1.4.2.59 Di-n-octylphthalate (CAS 000117-84-0)

Di-n-octylphthalate is also known as bis(n-octyl) phthalate, DNOP; dinopol NOP; n-dioctyl phthalate; 1,2-benzenedicarboxylic acid dioctyl ester; n-octyl phthalate; 1,2-benzenedicarboxylic acid, dioctyl ester; benzenedicarboxylic acid, di-n-octyl ester; vinicizer 85; dioctyl o-benzenedicarboxylate; celluflex dop; polycizer 162; and PX-138.

There are no cancer slope factors used in the BHHRA for di-n-octylphthalate. The oral and dermal RfDs used in the BHHRA are 2.00E-2 and 1.80E-2 mg/kg-day, respectively. The inhalation RfD is 2.00E-2 mg/kg-day. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 90 percent was used.

1.4.2.60 Endosulfan I (CAS 000959-98-8)

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure therefore, quantitative estimates of carcinogenicity and systemic toxicity due to Endosulfan I exposure is not included in the BHHRA. An oral RfD of 6.00E-3 mg/kg-day was used in the BHHRA. The inhalation RfD was 6.00E-3 mg/kg-day and the dermal route RfD was 3.00E-3 mg/kg-day.

1.4.2.61 Endosulfan II (CAS 033213-65-9)

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure for Endosulfan II. Therefore, quantitative estimates of carcinogenicity and systemic toxicity due to Endosulfan II exposure is not included in the BHHRA. An oral RfD of 6.00E-3 mg/kg-day was used in the BHHRA. The inhalation RfD was 6.00E-3 mg/kg-day and the dermal route RfD was 3.00E-3 mg/kgday.

1.4.2.62 Endosulfan sulfate (CAS 001031-07-8)

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure for endosulfan sulfate. Therefore, quantitative estimates of carcinogenicity and systemic toxicity due to endosulfan sulfate exposure is not included in the BHHRA. An oral RfD of 6.00E-3 mg/kg-day was used in the BHHRA. The inhalation RfD was 6.00E-3 mg/kg-day and the dermal route RfD was 3.00E-3 mg/kg-day.

1.4.2.63 Endrin (CAS 000072-20-8)

There are no carcinogenic slope factors used in the BHHRA for endrin due to a lack of carcinogenic toxicity information.

An oral RfD of 3.00E-4 mg/kg-day is used in this risk assessment for endrin. The dermal route RfD used in the BHHRA is 6.00E-6 mg/kg-day. The inhalation RfC is not available.

1.4.2.64 Endrin ketone (CAS 053494-70-5)

No toxicity information was found (e.g., slope factors, RfDs, RfCs) for any route of exposure for endrin ketone. Therefore, quantitative estimates of carcinogenicity and systemic toxicity due to endrin ketone exposure is not included in the BHHRA.

1.4.2.65 Ethylbenzene (CAS 000100-41-4) (RAIS)

Ethylbenzene is a colorless, flammable liquid with a pungent odor (Cavender 1994). The water solubility of ethylbenzene is 0.014 g/100 mL, and its vapor pressure is 10 mm Hg at 25°C (Budavari et al. 1989). Ethylbenzene is commonly used as a solvent, chemical intermediate in the manufacture of styrene and synthetic rubber, and as an additive in some automotive and aviation fuels (Cavender 1994). Occupational exposure to ethylbenzene may occur during production and conversion to polystyrene and during production and use of mixed xylenes (Fishbein 1985). The public can be exposed to ethylbenzene in ambient air as a result of releases from vehicle exhaust and cigarette smoke (Fishbein 1985).

Ethylbenzene can be absorbed through the lungs, digestive tract, and skin (Fishbein 1985). It also crosses the placenta (Cavender 1994). The liver is the major organ of ethylbenzene metabolism. In humans the major metabolites of ethylbenzene are mandelic acid (64–70 percent) and phenylglyoxylic acid (25 percent) (Bardodej and Bardodejova 1970, Fishbein 1985, Cavender 1994); however, these compounds are only minor metabolites in laboratory animals (EPA 1995h). Excretion occurs primarily in the urine (NTP 1992, Climie et al. 1983).

Ingestion of sublethal amounts of ethylbenzene is likely to cause CNS depression, oro-pharyngeal and gastric discomfort, and vomiting (HSDB 1995); however, specific experimental data are not available. Animal studies indicate that the primary target organs following chronic oral exposures are likely to be the liver and kidney. The oral RfD for chronic exposures is 0.1 mg/kg-day, based on increased weight and histopathological changes in the liver and kidneys of rats (EPA 1996b).

Acute exposures to high atmospheric concentrations of ethylbenzene may cause eye and respiratory tract irritation and CNS effects (e.g., coordination disorders, dizziness, vertigo, narcosis, convulsions, pulmonary irritation, and conjunctivitis) (Ivanov 1962). Concentrations of 1000 ppm (434 mg/m³) can be highly irritating to the eyes of humans (Yant et al. 1930); the threshold for eye irritation has been reported to be 200 ppm (879 mg/m³) (Grant 1986). No evidence is available to suggest that occupational exposures to ethylbenzene result in chronic toxic effects (Fishbein 1985); however, histopathological changes in the liver and kidney have been observed in experimental animals following prolonged inhalation exposures. Laboratory studies also indicate that exposure to ethylbenzene (4340 mg/m³) during gestation results in adverse developmental effects in rats (skeletal variants) and rabbits (reduced number of live offspring per litter). The NOAEL for developmental effects was reported to be 434 mg/m³. The inhalation RfC for chronic exposures is 1 mg/m³, based on developmental effects (EPA 1996b).

No epidemiological information is available on the potential carcinogenicity of ethylbenzene in humans following oral or inhalation exposures. A statistically significant increase in total malignant tumors was observed in female rats dosed orally with ethylbenzene (Maltoni et al. 1985); however, because of study limitations, these results cannot be considered conclusive. Although ethylbenzene has been tested by NTP in a two-year rodent bioassay, the results of that study are not yet available (NTP 1995). Ethylbenzene is placed by EPA in Group D, not classifiable as to human carcinogenicity, based on a lack of data in humans and animals (EPA 1996b).

There are no carcinogenic slope factors used in the BHHRA for ethyl benzene due to a lack of carcinogenic toxicity information.

No cancer slope factors were used in the BHHRA for ethylbenzene. The oral, inhalation, and dermal RfDs used in this BHHRA are 1.00E-1, 2.86E-1, and 9.7E-2 mg/kg-day, respectively. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 97 percent was used.

1.4.2.66 Fluoranthene (CAS 000206-44-0) (RAIS)

Fluoranthene is a PAH that can be derived from coal tar. Occurring ubiquitously in products of incomplete combustion of fossil fuels, fluoranthene has been identified in ambient air; surface, drinking, and waste water; and in char-broiled foods. Currently, there is no commercial production or use of this compound (IARC 1983f).

Fluoranthene can be absorbed through the skin following dermal exposure (Storer et al. 1984b) and, by analogy to structurally related PAHs, would be expected to be absorbed from the gastrointestinal tract and lungs (EPA 1988b). An in vitro study identified 2-methylfluoranthene and 3-methylfluoranthene and their dihydrodiols as metabolites of fluoranthene (La Voie et al. 1982b).

Although a large body of literature exists on the toxicity and carcinogenicity of PAHs, primarily benzo(a)pyrene, toxicity data for phenanthrene are very limited. No human data were available that addressed the toxicity of fluoranthene. Acute toxicity data for animals include an oral LD_{50} of 2000 mg/kg for rats; a dermal LD_{50} of 3180 mg/kg for rabbits (Smyth et al. 1962); and an intravenous LD_{50} of 100 mg/kg for mice (RTECS 1993). Subchronic oral exposure to fluoranthene at doses of greater than or equal to 250 mg/kg produced nephropathy, increased liver weights, and increased liver enzyme levels in rats (EPA 1988b). A single intraperitoneal injection of fluoranthene to pregnant rats caused an increased rate of embryo resorptions (Irvin and Martin 1987). Fluoranthene was photosensitizing, enhancing erythema elicited by ultraviolet radiation in guinea pig skin (Kochevar et al. 1982) and was irritating to the eyes of rabbits (Grant 1986).

An RfD of 4.00E-01 mg/kg-day for subchronic oral exposure and 4.00E-02 mg/kg-day for chronic oral exposure to fluoranthene was calculated from a NOAEL of 125 mg/kg-day and a LOAEL of 250 mg/kg-day derived from a 13-week gavage study with mice (EPA 1998a and b). The critical effects were nephropathy, increased liver weights, and changes in clinical and hematological parameters. Data were insufficient to derive an inhalation RfC for fluoranthene (EPA 1998a and b).

No oral or inhalation bioassays were available to assess the carcinogenicity of fluoranthene. Bioassays by other exposure routes generally gave negative results. Studies involving topical application to the skin of mice (Horton and Christian 1974, Hoffmann et al. 1972, Wynder and Hoffmann 1959b, Suntzeff et al. 1957) and subcutaneous injection in mice (Shear 1938) provided no evidence of carcinogenicity. Fluoranthene was also inactive in mouse skin initiation and promotion assays (Van Duuren and Goldschmidt 1976, Hoffmann et al. 1972). However, fluoranthene has been shown to be active as a cocarcinogen when applied with benzo(a)pyrene to mice by skin application (Van Duuren and Goldschmidt 1976) and was active as a complete carcinogen in a short-term lung tumor assay with newborn mice (Busby et al. 1984).

Based on no human data and inadequate data from animal bioassays, EPA (1998a, 1998b) has placed fluoranthene in weight-of-evidence group D, not classifiable as to human carcinogenicity.

The oral and dermal RfDs used in the BHHRA are 4.00E-2 and 1.24E-2 mg/kg-day, respectively. the inhalation RfD is 4.00E-2 mg/kg-day. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 31 percent was used. No cancer slope factors are available.

1.4.2.67 Fluorene (CAS 000086-73-7)

Fluorene is also known as 9H-fluorene; o-biphenylenemethane; diphenylenemethane; 2,2'-methylenebiphenyl; o-biphenylmethane; 2,3-benzindene; and alpha-diphenylenemethane-9H-fluorene.

No cancer slope factors were used in the BHHRA for fluorene. The oral and dermal RfDs used in the ----BHHRA are 4.00E-2 and 2.00E-2 mg/kg-day, respectively. The inhalation RfD was 4.00E-2mg/kg-day. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 50 percent was used.

1.4.2.68 gamma-BHC (Lindane) (CAS 000058-89-9)

An oral slope factor of 1.30E+0 (mg/kg-day)⁻¹ was calculated for gamma-BHC. For inhalation exposure, the inhalation unit risk is not available. The absorbed dose slope factor is 1.34E+0 (mg/kg-day)⁻¹.

An oral RfD of 3.0E-4 mg/kg-day is used in this risk assessment for gamma-BHC. The dermal route RfD used in the BHHRA is 2.91E-4 mg/kg-day. The inhalation RfC is 1.05E-3 mg/m³.

1.4.2.69 gamma-Chlordane (CAS 005103-74-2)

See Sect. 1.4.2.34.

1.4.2.70 Heptachlor (CAS 000076-44-8) (RAIS)

Heptachlor, a cyclodiene insecticide, was extensively used until the 1970s for control of a variety of insects. At the present time, its only permitted commercial use in the United States is fire ant control in power transformers. Heptachlor is converted to heptachlor epoxide and other degradation products in the environment. The epoxide degrades more slowly and, as a result, is more persistent than heptachlor. Both heptachlor and heptachlor epoxide are bioconcentrated in terrestrial and aquatic organisms. Heptachlor is subject to long-range transport and removal from the atmosphere by wet deposition (ATSDR 1993e, Leber and Benya 1994).

Heptachlor is absorbed from the gastrointestinal tract, lungs, and skin. It is distributed to various tissues with highest levels occurring in adipose tissue. Transplacental transfer to the fetus has been reported (EPA 19861). Metabolism produces primarily heptachlor epoxide, which is more toxic than its parent compound. Heptachlor and its metabolites are eliminated primarily via feces (Tashiro and Matsumara 1978).

The primary adverse health effects associated with heptachlor are CNS and liver effects. For humans, acute oral exposure has resulted in abnormal behavior, hyperirritability, tremors, and convulsions (Leber and Benya 1994). Various CNS effects such as hyperexcitability, incoordination, tremors, muscle spasms, and seizures have also been reported in animals following acute and subchronic oral exposure (Akay and Alp 1981, Buck et al. 1959, EPA 1985c). Oral LD₅₀ values for rabbits, rats, sheep, and calves are 2000, 90–160, 50, and 20 mg/kg, respectively (IARC 1979c, Leber and Benya 1994). Although hepatic effects have not been reported in humans, chronic dietary exposure of rodents to 10 ppm heptachlor or to 10 ppm of a 25:75 mixture of heptachlor/heptachlor epoxide for 18 months has produced increased liver weights, liver lesions, and decreased body weight gains (Velsicol Chemical Corporation 1955, IRDC 1973).

Other effects reported in humans include blood dyscrasias as a result of exposure to heptachlor during home termite treatment (Epstein and Ozonoff 1987) and increased mortality from cerebrovascular disease in workers manufacturing pesticides. However, cardiovascular effects were not seen in a cohort of pesticide applicators with potentially high exposures to heptachlor (Wang and MacMahon 1979a and b). Reduced fertility, increased resorptions, and decreased survival of offspring was noted in rats fed diets containing 0.25 mg/kg-day for 60 days prior to mating, with treatment continuing through gestation for

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the females (Green 1970). Reduced fertility and an increased incidence of cataracts, particularly in ----offspring, was reported in rats fed 6 mg/kg-day over an 18-month period (Mestitzova 1967).

An oral RfD of 5E-4 mg/kg-day for subchronic (EPA 1998b) and chronic exposure (EPA 1998a) to heptachlor was calculated based on a NOAEL of 0.15 mg/kg-day and a LOAEL of 0.25 mg/kg-day from a 2-year dietary study with rats (Velsicol Chemical Corporation 1955). Increased relative liver weight was identified as the critical effect. An inhalation RfC for heptachlor has not been derived.

Existing epidemiological studies on heptachlor are inadequate to establish a clear assessment of heptachlor exposure and human risk of developing cancer. Large-scale occupational cohort studies on workers engaged in the manufacture of heptachlor and pesticide applicators have not identified significantly increased cancer deaths (Wang and McMahon 1979a and b). Several bioassays have shown that heptachlor can cause liver cancer in mice. Bioassays with rats were generally negative. Benign liver tumors and hepatocellular carcinomas developed in both sexes of C3H mice fed 10 ppm heptachlor for 2 years; hepatocellular carcinomas developed in both sexes of $B6C3F_1$ mice fed 6–18 ppm technical grade heptachlor for 80 weeks; and nodular hyperplasia benign hepatomas and hepatocellular carcinomas developed in CD-1 mice fed 5 ppm (both sexes) or 10 ppm (males) of a 25:75 heptachlor/heptachlor epoxide mixture for 18 months (Epstein 1976, NCI 1977a).

Based on EPA guidelines, heptachlor was assigned to weight-of-evidence group B2, probable human carcinogen. For oral exposure, the slope factor is 4.50 (mg/kg-day)⁻¹ and the unit risk is 1.3E-4 (µg/L)⁻¹ (EPA 1998a). The inhalation slope factor and unit risk are 4.50 (mg/kg-day)⁻¹ (EPA 1998b) and 1.30E-3 m³/µg (EPA 1998a), respectively.

An oral slope factor of 4.50E+0 (mg/kg-day)⁻¹ was calculated for heptachlor. For inhalation exposure, the inhalation unit risk is 1.30E-3 m³/µg. The absorbed dose slope factor is 6.25E+0 (mg/kg-day)⁻¹.

An oral RfD of 5.00E-4 mg/kg-day is used in this risk assessment for heptachlor. The dermal route RfD used in the BHHRA is 3.60E-4 mg/kg-day. The inhalation RfC is 1.3E+0 mg/m³.

1.4.2.71 Heptachlor epoxide (CAS 001024-57-3) (RAIS)

Heptachlor epoxide, an oxidation product of the cyclodiene insecticide heptachlor, is not produced commercially in the United States and is not known to occur naturally (ATSDR 1993e, IARC 1979c). In the environment, heptachlor is converted to the epoxide, a chemical that degrades more slowly and, as a result, is more persistent than heptachlor. Both compounds adsorb strongly to sediments and are bioconcentrated in terrestrial and aquatic organisms; biomagnification of both is significant. Heptachlor epoxide has been identified in at least 87 of the 1300 hazardous waste sites on EPA's National Priorities List (NPL) (ATSDR 1993e).

In the body, heptachlor epoxide is formed by epoxidation of heptachlor. It is distributed to various tissues with highest levels occurring in adipose tissues where it may persist for prolonged periods. Heptachlor epoxide has been found in human fat, milk, and also in blood and fat of stillborn infants, indicating transplacental transfer to the fetus (IARC 1979c, EPA 1986)).

No studies were available regarding the toxic effects in humans after exposure to heptachlor epoxide. In laboratory animals, the liver and CNS are the primary target organs for heptachlor epoxide toxicity. Acute oral LD_{50} s for rats, mice, and rabbits range from 39 to 144 mg/kg (ATSDR 1993e), indicating moderate acute oral toxicity. Hypoactivity, ruffled fur, and increased mortality occurred in mice given a single oral dose of 30 mg/kg of a 25:75 heptachlor:heptachlor epoxide mixture (Arnold et al.

1977), and muscle spasms in the head and neck region and convulsive seizures were observed in young --- calves fed 2.5 mg/kg-day of a heptachlor epoxide preparation for 3 days (Buck et al. 1959). Increased liver weights and hepatocytomegaly were reported in male and female CD-1 mice fed a diet containing 1–10 ppm of a 25:75 heptachlor:heptachlor epoxide mixture for 18 months (IRDC 1973). Increased liver weights were also seen in dogs administered diets containing 0.5–7.5 ppm heptachlor epoxide for 60 weeks (Dow Chemical Company 1958).

An oral RfD of 1.3E-5 mg/kg-day for subchronic (EPA 1998b) and chronic exposure (EPA 1998a) to heptachlor epoxide was calculated based on a LOAEL of 0.0125 mg/kg-day from a 60-week dietary study with dogs (Dow Chemical Company 1958). Increased relative liver weight was identified as the critical effect. An inhalation RfC for heptachlor epoxide has not been derived.

No epidemiological studies or case reports addressing the carcinogenicity of heptachlor epoxide in humans were available. Studies with laboratory animals demonstrated that heptachlor epoxide causes liver cancer in mice and rats. Liver carcinomas developed in C3H mice fed 10 ppm heptachlor epoxide for 2 years (Davis 1965). Hepatic hyperplasia, hyperplastic nodules, and liver carcinomas developed in CD-1 mice fed 0.1–10 ppm of a 25:75 heptachlor:heptachlor epoxide mixture for 18 months (IRDC 1973) and in CFN rats fed 0.5–10 ppm heptachlor epoxide for 108 weeks (Epstein 1976).

Based on EPA guidelines, heptachlor epoxide was assigned to weight-of-evidence Group B2, probable human carcinogen (EPA 1998a). For oral and inhalation exposure, the slope factor is 9.1 (mg/kg-day)⁻¹ (EPA 1985c). The unit risk is 2.60E-4 (μ g/L)⁻¹ for oral exposure and 2.60E-3 (μ g/m³)⁻¹ for inhalation exposure (EPA 1998a).

An oral slope factor of 9.10E+0 (mg/kg-day)⁻¹ was calculated for heptachlor epoxide. For inhalation exposure, the inhalation unit risk is 2.6E-3 (μ g/m³)⁻¹. The absorbed dose slope factor is 1.26E+1 (mg/kg-day)⁻¹.

An oral RfD of 1.30E-5 mg/kg-day is used in this risk assessment for heptachlor epoxide. The dermal route RfD used in the BHHRA is 9.30E-6 mg/kg-day. The inhalation RfC is not available.

1.4.2.72 Hexachlorobenzene (CAS 000118-74-1)

An oral slope factor of 1.60E+0 (mg/kg-day)⁻¹ was calculated for hexachlorobenzene. For inhalation exposure, the inhalation unit risk is 4.60E-4 m³/ μ g. The absorbed dose slope factor is 3.20E+0 (mg/kg-day)⁻¹.

An oral RfD of 8.00E-4 mg/kg-day is used in this risk assessment for hexachlorobenzene. The dermal route RfD used in the BHHRA is 4.00E-4 mg/kg-day and the inhalation route RfD is 8.00E-4 mg/kg-day. The inhalation RfC is not available.

1.4.2.73 Hexachlorobutadiene (CAS 000087-68-3)

An oral slope factor of 7.80E-2 $(mg/kg-day)^{-1}$ was calculated for hexachlorobutadiene. For inhalation exposure, the inhalation unit risk is 2.20E-5 m³/µg. The absorbed dose slope factor is 1.56E-1 $(mg/kg-day)^{-1}$.

An oral RfD of 2.00E-4 mg/kg-day is used in this risk assessment for hexachlorobutadiene. The dermal route RfD used in the BHHRA is 1.00E-4 mg/kg-day and the inhalation RfD is 2.00E-4 mg/kg-day The inhalation RfC is not available.



1.4.2.74 Hexachlorocyclopentadiene (CAS 000077-47-4)

There are no carcinogenic slope factors used in the BHHRA for hexachlorocyclopentadiene due to a lack of carcinogenic toxicity information.

An oral RfD of 7.00E-3 mg/kg-day is used in this risk assessment for hexachlorocyclopentadiene. The dermal and inhalation route RfDs used in the BHHRA are 3.5E-3 mg/kg-day and 7.00E-3 mg/kg-day respectively. The inhalation RfC is 7.00E-5 mg/m³.

1.4.2.75 Hexachloroethane (CAS 000067-72-1)

An oral slope factor of 1.40E-2 $(mg/kg-day)^{-1}$ was calculated for hexachloroethane. For inhalation exposure, the inhalation unit risk is 4.00E-3 m³/µg. The absorbed dose slope factor is 2.80E-2 $(mg/kg-day)^{-1}$.

An oral RfD of 1.00E-3 mg/kg-day is used in this risk assessment for hexachloroethane. The dermal route RfD used in the BHHRA is 5.00E-4 mg/kg-day and the inhalation route RfD is 1.00E-3 mg/kg-day. The inhalation RfC is not available.

1.4.2.76 Indeno(1,2,3-cd)pyrene (CAS 000193-39-5) (RAIS)

Indeno(1,2,3-cd)pyrene, a crystalline solid with a chemical formula of $C_{22}H_{12}$ and a molecular weight of 276.3, is a PAH. There is no commercial production or known use of this compound (IARC 1983g). Indeno(1,2,3-c,d)pyrene is found in fossil fuels and occurs ubiquitously in products of incomplete combustion (IARC 1983g) and has been identified in soils, groundwater, and surface waters at hazardous waste sites (ATSDR 1990j).

No absorption data were available for indeno(1,2,3-cd)pyrene; however, by analogy to structurally related PAHs, primarily benzo(a)pyrene, it would be expected to be absorbed from the gastrointestinal tract, lungs, and skin (EPA 1991k). In vivo metabolites identified in mouse skin include the *trans*-1,2-dihydrodiol and 8- and 9-hydroxy forms of indeno(1,2,3-cd)pyrene (Rice et al. 1986). Similar metabolites were formed in vitro in rat liver microsomes (Rice et al. 1985).

No data were found concerning the acute, subchronic, chronic, developmental, or reproductive toxicity of indeno(1,2,3-cd)pyrene. Because of a lack of toxicity data, an oral RfD or inhalation RfC has not been derived (EPA 1998a).

No long-term oral or inhalation bioassays were available to assess the carcinogenicity of indeno(1,2,3-cd)pyrene. The compound was tested for carcinogenicity in dermal application, lung implant, subcutaneous injection, and intraperitoneal injection studies. Dermal application of 0.1-0.5 percent solutions of indeno(1,2,3-cd)pyrene in acetone produced skin papillomas and carcinomas in mice (Hoffmann and Wynder 1966). In initiation-promotion assays, indeno(1,2,3-cd)pyrene was active as an initiator of skin carcinogenesis (Hoffmann and Wynder 1966, Rice et al. 1986). Dose-related increases of epidermoid carcinomas of the lungs were reported in rats receiving single lung implants of 0.16-4.15 mg indeno(1,2,3-cd)pyrene (Deutsch-Wenzel et al. 1983). Injection site sarcomas developed in mice given three subcutaneous injections of 0.6 mg indeno(1,2,3-cd)pyrene (Lacassagne et al. 1963). The compound was not tumorigenic when newborn mice received 2.1 mol indeno(1,2,3-cd)pyrene via intraperitoneal injection (LaVoie et al. 1987).

Based on no human data and sufficient evidence for carcinogenicity in animals, EPA has assigned a ---weight-of-evidence classification of B2, probable human carcinogen, to indeno(1,2,3-cd)pyrene (EPA 1998a).

The oral, dermal, and inhalation cancer slope factors used in the BHHRA for ideno(1,2,3-cd)pyrene are 7.30E-1, 2.35E+0, and 3.10E-1 (mg/kg-day)⁻¹, respectively. The inhalation unit risk is 8.80E-5 m³/ μ g. These were derived from the values for benzo(a)pyrene using the relative potency factors recommended by EPA. The dermal slope factor was derived from the oral slope factor using a gastrointestinal absorption factor of 31 percent. No RfDs for ideno(1,2,3-cd)pyrene were found; therefore, noncancer effects due to exposure to ideno(1,2,3-cd)pyrene could not be estimated in the BHHRA.

1.4.2.77 Methoxychlor (CAS 000072-43-5)

There are no carcinogenic slope factors used in the BHHRA for methoxychlor because of a lack of carcinogenic toxicity information.

An oral RfD of 5.00E-3 mg/kg-day is used in this risk assessment for methoxychlor. The dermal route RfD used in the BHHRA is 2.50E-3 mg/kg-day and the inhalation route RfD is 5.00E-3 mg/kg-day. The inhalation RfC is not available.

1.4.2.78 Methylene chloride (CAS 000075-09-2) (RAIS)

Methylene chloride, also known as dichloromethane, is a colorless, volatile liquid with a penetrating, ether-like odor. In industry, methylene chloride is widely used as a solvent in paint removers, degreasing agents, and aerosol propellants; as a polyurethane foam-blowing agent; and as a process solvent in the pharmaceutical industry. The compound is also used as an extraction solvent for spice oleoresins, hops, and caffeine (ATSDR 1989i, IARC 1986b).

Methylene chloride is readily absorbed from the lungs, the gastrointestinal tract, and to some extent through the skin. Metabolism of methylene chloride produces CO_2 and CO, which readily binds with blood hemoglobin to form carboxyhemoglobin (CO-Hb). The primary adverse health effects associated with methylene chloride exposure are CNS depression and mild liver effects. Neurological symptoms described in individuals occupationally exposed to methylene chloride included headaches, dizziness, nausea, memory loss, paresthesia, tingling hands and feet, and loss of consciousness (Welch 1987). Major effects following acute inhalation exposure include fatigue, irritability, analgesia, narcosis, and death (ATSDR 1989i). CNS effects have also been demonstrated in animals following acute exposure to methylene chloride (Weinstein et al. 1972, Berger and Fodor 1968).

Impaired liver function has been associated with occupational exposure to methylene chloride (Welch 1987). Liver effects have also been documented in a number of inhalation studies with laboratory animals. Subchronic exposure of rats, mice, dogs, and monkeys caused mild hepatic effects such as cytoplasmic vacuolization and fatty changes (EPA 1983, Haun et al. 1972, Weinstein and Diamond 1972, Heppel et al. 1944). Hepatocellular foci, fatty changes, and necrosis were reported following chronic inhalation exposure of rats and mice (Nitschke et al. 1988a, NTP 1986a). Chronic oral exposure to methylene chloride via drinking water resulted in histopathological alterations of the liver in rats and mice (NCA 1983). In addition, inhalation exposure of rats caused nonspecific degenerative and regenerative changes in the kidneys (EPA 1983, Haun et al. 1972).

An RfD, and oral RfD_c of 6E-2 mg/kg-day for methylene chloride has been calculated by EPA (1998a,b). This value is based on a NOAEL of 5.85 mg/kg-day derived from a chronic drinking water

study with rats (NCA 1983). This same study was adapted for the derivation of the subchronic and ---- chronic RfC of 3E+0 mg/m³ (NOAEL, 694.8 mg/m³) (EPA 1998b).

Studies of workers exposed to methylene chloride have not recorded a significant increase in cancer cases above the number of cases expected for nonexposed workers (Hearne et al. 1987, Ott et al. 1983, Friedlander et al. 1978). However, long-term inhalation studies with rats and mice demonstrated that methylene chloride causes cancer in laboratory animals. Mice exposed via inhalation to high concentrations of methylene chloride (2000 or 4000 ppm) exhibited a significant increase of malignant liver and lung tumors compared with nonexposed controls (NTP 1986a). Rats of both sexes exposed to concentrations of methylene chloride ranging from 500 to 4000 ppm showed increases of benign mammary tumors (Nitschke et al. 1988a, NTP 1986a, Burek et al. 1984). An inhalation study with rats and hamsters revealed sarcomas of the salivary gland in male rats, but not in female rats or hamsters (Burek et al. 1984). Liver tumors observed in rats and mice that ingested methylene chloride in drinking water for 2 years provided suggestive evidence of carcinogenicity (NCA 1983). Based on inadequate evidence of carcinogenicity in humans and on sufficient evidence in animals, EPA (1998a) has placed methylene chloride in weight-of-evidence group B2, probable human carcinogen. A slope factor and unit risk of 7.5E-3 (mg/kg-day)⁻¹ and 2.1E-7 (ug/L)⁻¹, respectively (EPA 1998a), was derived for oral exposure to methylene chloride. The inhalation unit risk is 4.7E-7 m³/µg (EPA 1998a).

The oral and dermal cancer slope factors used in the BHHRA are 7.50E-3 and 7.89E-3 (mg/kg-day)⁻¹, respectively. An inhalation cancer slope factor of 1.65E-3 (mg/kg-day)⁻¹ was used. The oral and dermal RfDs used in the BHHRA are 6.00E-2 and 5.70E-2 mg/kg-day, respectively. An inhalation RfD of 8.57E-01 mg/kg-day was used. When calculating both the dermal route cancer slope factor and dermal route RfD from their respective oral values, a gastrointestinal absorption factor of 95 percent was used.

1.4.2.79 Napthalene (CAS 000091-20-3) (RAIS)

Naphthalene, a white solid with a characteristic odor of mothballs, is a PAH composed of two fused benzene rings. The principal end use of naphthalene is as raw material for the production of phthalic anhydride. It is also used as an intermediate for synthetic resins, celluloid, lampblack, smokeless powder, solvents, and lubricants. Naphthalene is used directly as a moth repellant, insecticide, anthelmintic, and intestinal antiseptic (ATSDR 1990m, EPA 1986m).

Naphthalene can be absorbed by oral, inhalation, and dermal routes of exposure and can cross the placenta in amounts sufficient to cause fetal toxicity. The most commonly observed effect of naphthalene toxicity following acute oral or inhalation exposure in humans is hemolytic anemia associated with decreased hemoglobin and hematocrit values, increased reticulocyte counts, presence of Heinz bodies, and increased serum bilirubin levels (ATSDR 1990m). Hemolytic anemia has been observed in an infant dermally exposed to naphthalene (Schafer 1951) and in infants whose mothers were exposed to naphthalene during pregnancy (Anziulewicz et al. 1959, Zinkham and Childs 1958). Infants and individuals having a congenital deficiency of erythrocyte glucose-6-phosphate dehydrogenase are especially susceptible to naphthalene-induced hemolytic anemia (Wintrobe et al. 1974).

Acute oral and subchronic inhalation exposure of humans to naphthalene has resulted in neurotoxic effects (confusion, lethargy, listlessness, vertigo), gastrointestinal distress, hepatic effects (jaundice, hepatomegaly, elevated serum enzyme levels), renal effects, and ocular effects (cataracts, optical atrophy). Cataracts have been reported in individuals occupationally exposed to naphthalene (Ghetti and Mariani 1956) and in rabbits and rats exposed orally to naphthalene (Van Heyningen and Pirie 1976, Fitzhugh and Buschke 1949). A number of deaths have been reported following intentional ingestion of naphthalene-containing mothballs (ATSDR 1990m). The estimated lethal dose of naphthalene is 5–15 g

for adults and 2-3 g for children. Naphthalene is a primary skin irritant and is acutely irritating to the eyes ---- of humans (Sandmeyer 1981).

Increased mortality, clinical signs of toxicity, kidney and thymus lesions, and signs of anemia were observed in rats treated by gavage with 400 mg/kg of naphthalene for 13 weeks (NTP 1980a). No adverse effects occurred at 50 mg/kg. Transient clinical signs of toxicity were seen in mice exposed by gavage to 53 mg/kg for 13 weeks (NTP 1980b). Subchronic oral exposure to 133 mg/kg-day for 90 days produced decreased spleen weights in female mice (Shopp et al. 1984). Reduced numbers of pups/litter were observed when naphthalene was administered orally to pregnant mice (Pflasterer et al. 1985). Negative results in a 2-year feeding study with rats receiving 10–20 mg naphthalene/kg-day (Schmahl 1955) and equivocal results in a mouse lung tumor bioassay (Adkins et al. 1986) suggest that naphthalene is not a potential carcinogen.

An RfD_s and oral RfD_c of 2E-2 mg/kg-day for naphthalene has been calculated by EPA (1998b). These values are based on a NOAEL of 50 mg/kg-day derived from a subchronic oral toxicity study with rats (NTP 1980a). The RfD is currently under review by EPA and may be subject to change (EPA 1998b). A RfC for chronic inhalation exposure has not been derived by EPA. Available cancer bioassays were insufficient to assess the carcinogenicity of naphthalene. Therefore, EPA (1991m,1998b) has placed naphthalene in weight-of-evidence Group D, not classifiable as to human carcinogenicity.

No cancer slope factors were used in the BHHRA for naphthalene. The oral inhalation and dermal RfDs used in the BHHRA are 2E-2, 8.57E-4 and 1.6E-2 mg/kg-day, respectively. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 80 percent was used.

1.4.2.80 N-nitroso-di-n-propylamine (CAS 000621-64-7)

N-nitroso-di-n-propylamine is also known as n-nitrosodipropylamine; n-Nitroso-n-propyl-1propanamine; dipropylnitrosamine; DPNA; NDPA; di-n-propylnitrosamine; nitrosodipropylamine; N-nitroso-n-dipropylamine; nitrous dipropylamide; and DPN.

The oral and dermal cancer slope factors used in the BHHRA are 7.00E+0 and 2.80E+1 (mg/kg-day)⁻¹, respectively. An inhalation cancer slope factor was not found. No RfD's were found for n-nitroso-di-n-propylamine. When calculating the dermal route cancer slope factor from the oral value, a gastrointestinal absorption factor of 25 percent was used.

1.4.2.81 N-nitrosodiphenylamine (CAS 000086-30-6)

N-nitrosodiphenylamine is also known as diphenyl, n-nitrosoamine; n-nitroso-n-phenylaniline; diphenylnitrosamine; Redax; N-nitroso-N-phenylbenzenamine; nitrosodiphenylamine; vulcatard; nitrous diphenylamide; N,N-diphenylnitrosamine; curetard a; delac j; naugard tjb; NDPHA; retarder j; TJB; vulcalent a; vulcatard a; vultrol; and phenyl-N-nitrosoamine.

The oral and dermal cancer slope factors used in the BHHRA are 4.90E-3 and 1.96E-2 (mg/kg-day)⁻¹, respectively. Inhalation cancer slope factors were not found. No RfDs were found for N-nitrosodiphenylamine. When calculating the dermal route cancer slope factor from the oral value, a gastrointestinal absorption factor of 25 percent was used.



1.4.2.82 Pentachlorophenol (CAS 000087-86-5) (RAIS)

Pentachlorophenol, a man-made organic biocide, is often contaminated with other toxic organic chemicals such as chlorinated phenols, dioxins, and dibenzofurans (Williams 1982, USAF 1989c, ATSDR 1992c).

Pentachlorophenol is readily absorbed following oral or inhalation exposure and is widely and rapidly distributed throughout the body (Wagner et al. 1991, ATSDR 1992c, Jorens and Schepens 1993). Human and animal studies have provided evidence indicating that pentachlorophenol is metabolized to various conjugated metabolites. Both the parent compound and the conjugates are excreted in the urine (Braun et al. 1979).

Assessing the potential toxicity of technical (commercial) grade pentachlorophenol is complicated by the presence of the toxic impurities that are usually present, and the effects resulting from occupational exposure are often difficult to attribute to a specific route of exposure (Jorens and Schepens 1993). The effects in humans following acute oral exposure include increased heart and respiratory rates, elevated temperature, increased basal metabolic rate, and death (29 and 401 mg/kg) (RTECS 1989).

Human fatalities and toxic effects including tachycardia, jaundice, and other hematologic alterations have been reported for acute and subchronic occupational (e.g., sawmill workers, herbicide sprayers) inhalation exposures to pentachlorophenol. Upper respiratory tract inflammation and bronchitis were reported for sawmill workers chronically exposed to pentachlorophenol (Baader and Bauer 1951, Menon 1958, ATSDR 1992c). However, dose-terms for these exposures were not available, and concurrent exposures to other chemicals make definitive assessments impossible.

Data regarding the dermal exposure of humans to pentachlorophenol are anecdotal or equivocal, lack dose terms, and are compromised by concurrent exposures to other chemicals, including the known contaminants in technical-grade pentachlorophenol. Acute exposure to 0.4 percent pentachlorophenol produced localized irritation (Bevenue et al. 1967), and subchronic exposures have caused chloracne (Baader and Bauer 1951, O'Malley et al. 1990) and possibly renal damage (ATSDR 1992c). Dermal lesions including pemphigus and chronic urticaria have been reported for humans chronically exposed to pentachlorophenol-treated wood (Lambert et al. 1986). There currently are no definitive data regarding reproductive toxicity in humans exposed to pentachlorophenol.

Acute oral exposure of animals to pentachlorophenol affects the liver, kidneys, cardiovascular system, and the peripheral and CNS. Oral LD_{50} values for laboratory animals range from 27 to 230 mg/kg (Borzelleca et al. 1985, USAF 1989c, ATSDR 1992c). Definitive data regarding the effects of subchronic or chronic oral exposure of humans to pentachlorophenol are not available. However, subchronic exposure (1–8 months) of rats to pentachlorophenol at doses ranging from 5 to 40 mg/kg-day has produced cardiovascular, hematotoxic, renal, hepatic, and immunologic responses (Schwetz et al. 1974b, 1978; USAF 1989c; ATSDR 1992c). Evidence of reproductive/developmental toxicity (increased resorptions, embryolethality, embryotoxicity, and teratogenicity) have also been observed in rats given pentachlorophenol during gestation (Larsen et al. 1975, 1976; Schwetz et al. 1978).

Because the most significant acute toxic effect of pentachlorophenol is elevated metabolism, a specific target organ or tissue is difficult to identify. However, for subchronic and chronic exposures, toxicity data indicate that the liver, kidney, and cardiovascular system are targets for some of the toxic effects of pentachlorophenol.

Both the RfD_c and RFD_s for pentachlorophenol are 3.00E-02 mg/kg-day based on a NOAEL of ----3 mg/kg-day and a LOAEL of 10 mg/kg-day for histopathologic findings in the liver and kidneys of rats given pentachlorophenol in the diet for 2 years (EPA 1998a, 1998b; Schwetz et al. 1978).

The RfC for pentachlorophenol is under review (EPA 1998a).

Based upon increased incidences of hepatocellular adenomas and carcinomas, adrenal medulla pheochromocytomas, malignant pheochromocytomas, and hemangiosarcomas/hemangiomas in mice, pentachlorophenol is classified by the EPA as a probable human carcinogen weight-of-evidence classification B2 and has an oral slope factor of 1.2E-01 (mg/kg-day)⁻¹ and an oral unit risk of 3.0E-06 (μ g/L)⁻¹ (EPA 1998a, 1998b). The potential carcinogenicity of pentachlorophenol following inhalation exposure has not been evaluated.

An oral slope factor of 1.2E-1 $(mg/kg-day)^{-1}$ was calculated for pentachlorophenol. For inhalation exposure, the inhalation unit risk is not available. The absorbed dose slope factor is 1.2E-1 $(mg/kg-day)^{-1}$.

An oral RfD of 3.0E-2 mg/kg-day is used in this risk assessment for pentachlorophenol. The dermal route RfD used in the BHHRA is 3.0E-2 mg/kg-day and the inhalation RfD is also 3.0E-2 mg/kg-day. The inhalation RfC is not available.

1.4.2.83 Phenanthrene (CAS 000085-01-8) (RAIS)

Phenanthrene is a PAH that can be derived from coal tar. Currently, there is no commercial production or use of this compound (EPA 19871). Phenanthrene is ubiquitous in the environment as a product of incomplete combustion of fossil fuels and wood and has been identified in ambient air, surface and drinking water, and foods (EPA 1988a, IARC 1983h).

Phenanthrene is absorbed following oral and dermal exposure (Storer et al. 1984b, Chang 1943). Data from structurally related PAHs suggest that phenanthrene would be absorbed from the lungs (EPA 19871). Metabolites of phenanthrene identified in in vivo and in vitro studies indicate that metabolism proceeds by epoxidation at the 1-2, 3-4, and 9-10 carbons, with dihydrodiols as the primary metabolites (Nordquist et al. 1981, Chaturapit and Holder 1978, Sims 1970, Boyland and Sims 1962, Boyland and Wolf 1950).

Although a large body of literature exists on the toxicity and carcinogenicity of PAHs, primarily benzo(a)pyrene, toxicity data for phenanthrene are very limited. No human data were available that addressed the toxicity of phenanthrene. Single intraperitoneal injections of phenanthrene produced slight hepatotoxicity in rats (Yoshikawa et al. 1985). Data regarding the subchronic, chronic, developmental, or reproductive toxicity in experimental animals by any route of exposure could not be located in the available literature.

Data were insufficient to derive an oral RfD or inhalation RfC for phenanthrene (EPA 1988a). The chemical is not currently listed in IRIS or HEAST (EPA 1998a,b).

No inhalation bioassays were available to assess the carcinogenicity of phenanthrene. A single oral dose of phenanthrene did not induce mammary tumors in rats (Huggins and Yang 1962), and a single subcutaneous injection did not result in treatment-related increases in tumor incidence in mice (Steiner 1955). Neonate mice administered intraperitoneal or subcutaneous injections of phenanthrene also did not develop tumors (Buening et al. 1979b). No skin tumors were reported in two skin painting assays with mice (Roe and Grant 1964, Kennaway 1924b). Phenanthrene was also tested in several mouse skin initiation-promotion assays. It was active as an initiator in one study (Scribner 1973), inactive as an

initiator in four others (LaVoie et al. 1981, Wood et al. 1979, Roe 1962, Salaman and Roe 1956), and inactive as a promoter in one study (Roe and Grant 1964).

Based on no human data and inadequate data from animal bioassays, EPA (1998a and 1987l) has placed phenanthrene in weight-of-evidence Group D, not classifiable as to human carcinogenicity.

Neither slope factors nor RfDs for any route of exposure were found for phenanthrene. Therefore, neither carcinogenicity nor systemic toxicity due to phenanthrene exposure is included in the BHHRA.

1.4.2.84 Polychlorinated biphenyl (CAS 001336-36-3) (RAIS)

Aroclor-1254 is a PCB mixture containing approximately 21 percent $C_{12}H_6Cl_4$, 48 percent $C_{12}H_5Cl_5$. 23 percent $C_{12}H_4Cl_6$, and 6 percent $C_{12}H_3Cl_7$ with an average chlorine content of 54 percent (USAF 1989d). PCBs are inert, thermally and physically stable, and have dielectric properties. In the environment, the behavior of PCB mixtures is directly correlated to the degree of chlorination. Aroclor-1254 is strongly sorbed to soil and remains immobile when leached with water; however, the mixture is highly mobile in the presence of organic solvents (USAF 1989d). PCBs are resistant to chemical degradation by oxidation or hydrolysis. However, biodegradation, especially of lower chlorinated PCBs, can occur (USAF 1989d). PCBs have high bioconcentration factors, and because of lipophilicity, especially of highly chlorinated congeners, tend to accumulate in the fat of fish, birds, mammals, and humans (ATSDR 1995).

PCBs are absorbed after oral, inhalation, or dermal exposure and are stored in adipose tissue. The location of the chlorine atoms on the phenyl rings is an important factor in PCB metabolism and excretion. The major route of PCB excretion is in the urine and feces; however, more important is the elimination in human milk. Metabolites are predominately found in urine and bile, while small amounts of the parent compound are found in the feces. Biliary excretion appears to be the source of fecal excretion (ATSDR 1995).

Accidental human poisonings and data from occupational exposure to PCBs suggest initial dermal and mucosal disturbances followed by systemic effects that may manifest themselves several years postexposure. Initial effects are enlargement and hypersecretion of the Meibomian gland of the eye, swelling of the eyelids, pigmentation of the fingernails and mucous membranes, fatigue, and nausea. These effects were followed by hyperkeratosis; darkening of the skin; acneform eruptions; edema of the arms and legs; neurological symptoms, such as headache and limb numbness; and liver disturbance (USAF 1989d).

Hepatotoxicity is a prominent effect of Aroclor-1254 that has been well characterized (EPA 1995j). Effects included hepatic microsomal enzyme induction, increased serum levels of liver-related enzymes indicative of hepatocellular damage, liver enlargement, lipid deposition, fibrosis, and necrosis. Groups of 16 adults (11.1 +/-4.1 years at study initiation) female rhesus monkeys ingested gelatin capsules containing 0, 0.005, 0.02, 0.04, or 0.08 mg/kg-day Aroclor-1254 daily for more than 5 years (Arnold et al. 1993a and b, Truelove et al. 1990). Treated monkeys were observed to have increases in the incidence of inflamed and/or prominent Meibomian glands; increased incidences of ocular exudate; changes in finger and/or toe nails; decreases in IgG and IgM antibody levels; decreases in the percent of helper T-lymphocytes; increases in suppressor T-lymphocyte count; a decrease in helper/suppressor ratio; and decreases in reticulocyte count, serum cholesterol, total bilirubin, and alpha-1+ alpha-2-globulins. An chronic oral RfD of 2E-05 mg/kg-day for Aroclor-1254 was calculated from a LOAEL of 0.0005 mg/kg-day derived from the above study (EPA 1995j). The oral RfDs is 5E-05 mg/kg-day (EPA 1995i).

Data are suggestive but not conclusive concerning the carcinogenicity of PCBs as a group in --humans. EPA has not determined a weight-of-evidence classification or slope factor for Aroclor-1254 specifically. However, hepatocellular carcinomas in three strains of rats and two strains of mice have led EPA (1995j) to classify PCBs as Group B2, probable human carcinogen. Carcinogenicity slope factors of 2E+0 (high risk), 4.00E-1 (low risk), and 7E-2 (lowest risk) (mg/kg-day)⁻¹ have been dervied for PCBs as a group, based on an increase of hepatocellular tumors in female Sprague-Dawley rats treated with Aroclor-1260. A drinking water unit risk of 1E-5 (μ g/L)⁻¹ (low risk) for PCBs was calculated based on the q₁* (EPA 1995j).

An oral slope factor of 2.00E+0 (mg/kg-day)⁻¹ was used for Aroclor-1016, -1221, -1232, -1242, -1248, 1254, and -1260. For inhalation exposure, the inhalation unit risk is $5.71E-4 (\mu g/m^3)^{-1}$ was used for these. The absorbed dose slope factor is 2.22E+0 (mg/kg-day)⁻¹.

An oral RfD of 7.00E-5 and 2.00E-5 mg/kg-day is used in this risk assessment for Aroclor-1016 and 1254 respectivly. The dermal route RfD for Aroclor 1016 used in the BHHRA is 6.30E-5 mg/kg-day. The inhalation RfC is 2.45E-4 mg/m³.

There are no RfDs or RfCs used in the BHHRA for Aroclor, -1221, -1232, -1242, and -1248 because of a lack of systemic toxicity and noncarcinogenic risk information.

1.4.2.85 Pyrene (CAS 000129-00-1) (RAIS)

Pyrene, also referred to as benzo(def)phenanthrene and -pyrene, is a PAH that can be derived from coal tar. Currently, there is no commercial production or use of this compound. Pyrene is ubiquitous in the environment as a product of incomplete combustion of fossil fuels and has been identified in surface and drinking water, numerous foods, and ambient air (EPA 1988a and 1987m, IARC 1983i).

Although a large body of literature exists on the toxicity and carcinogenicity of PAHs, toxicity data for pyrene are limited. No human data were available that addressed the toxicity of pyrene. Subchronic oral exposure to pyrene produced nephropathy, decreased kidney weights, increased liver weights, and slight hematological changes in mice (TRL 1989) and produced fatty livers in rats (White and White 1939). A single intraperitoneal injection of pyrene produced swelling and congestion of the liver and increased serum aspartate amino transferase (AST) and bilirubin levels in rats (Yoshikawa et al. 1985). No data were available concerning the toxic effects of inhalation exposure to pyrene or data regarding teratogenicity or other reproductive effects by any route of exposure.

An RfD of 3E-1 mg/kg-day for subchronic (EPA 1998b) and 3E-2 mg/kg-day for chronic oral exposure (EPA 1998a) to pyrene was calculated from a NOAEL of 75 mg/kg-day in a 13-week gavage study with mice (TRL 1989). Data were insufficient to derive an inhalation RfC for pyrene (EPA 1998a,b).

No oral or inhalation bioassays were available to assess the carcinogenicity of pyrene. Studies involving other routes of exposure (intratracheal, dermal, and subcutaneous) generally gave negative results. Intratracheal administration of pyrene in combination with Fe_2O_3 particles did not induce turnors in hamsters (Sellakumar and Shubik 1974). Skin painting assays evaluating complete carcinogenesis in mice (Van Duuren and Goldschmidt 1976, Horton and Christian 1974, Roe and Grant 1964, Wynder and Hoffman 1959b); or initiating (Roe and Grant 1964); or promoting capacity (Wood et al. 1980, Scribner 1973, Salaman and Roe 1956) have been negative or inconclusive. Mice injected subcutaneously with pyrene did not develop turnors (Shear and Leiter 1941), but there is evidence that pyrene enhances the turnorigenicity of topically applied benzo(a)pyrene (Slaga et al. 1979, Van Duuren and Goldschmidt 1976, Goldschmidt et al. 1973).

Based on no human data and inadequate data from animal bioassays, EPA (1998a,b) has placed -pyrene in weight-of-evidence Group D, not classifiable as to human carcinogenicity.

No cancer slope factors were used in the BHHRA. The oral and dermal RfDs used in the BHHRA are 3.00E-2 and 9.30E-3 mg/kg-day, respectively. An inhalation reference dose of 3.00E-2 mg/kg-day was also used. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 31 percent was used.

1.4.2.86 Tetrachloroethylene (CAS 000127-18-4) (RAIS)

Tetrachloroethylene is a halogenated aliphatic hydrocarbon with a vapor pressure of 17.8 mm Hg at 25°C (EPA 1982). The chemical is used primarily as a solvent in industry and, less frequently, in commercial dry-cleaning operations (ATSDR 1990n). Occupational exposure to tetrachloroethylene occurs via inhalation, resulting in systemic effects, and via dermal contact, resulting in local effects. Exposure to the general population can occur through contaminated air, food, and water (ATSDR 1990n).

The respiratory tract is the primary route of entry for tetrachloroethylene (NTP 1986b, EPA 1988c). The chemical is rapidly absorbed by this route and reaches an equilibrium in the blood within 3 hours after the initial exposure (Hake and Stewart 1977). Tetrachloroethylene is also significantly absorbed by the gastrointestinal tract, but not through the skin (Koppel et al. 1985, ATSDR 1990n). The chemical accumulates in tissues with high lipid content, where the half-life is estimated to be 55 hours (Stewart 1969, ATSDR 1990n), and has been identified in perirenal fat, brain, liver, placentofetal tissue, and amniotic fluid (Savolainen et al. 1977). The proposed first step for the biotransformation of tetrachloroethylene is the formation of an epoxide thought to be responsible for the carcinogenic potential of the chemical (Henschler and Hoos 1982, Calabrese and Kenyon 1991). Tetrachloroethylene is excreted mainly unchanged through the lungs, regardless of route of administration (NTP 1986b). The urine and feces comprise secondary routes of excretion (Monster et al. 1979, Ohtsuki et al. 1983). The major urinary metabolite of tetrachloroethylene, trichloroacetic acid, is formed via the cytochrome P-450 system (ATSDR 1990n).

The main targets of tetrachloroethylene toxicity are the liver and kidney by both oral and inhalation exposure and the CNS by inhalation exposure. Acute exposure to high concentrations of the chemical (estimated to be greater than 1500 ppm for a 30-minute exposure) may be fatal to humans (Torkelson and Rowe 1981a). Chronic exposure causes respiratory tract irritation, headache, nausea, sleeplessness, abdominal pains, constipation, cirrhosis of the liver, hepatitis, and nephritis in humans; and microscopic changes in renal tubular cells, squamous metaplasia of the nasal epithelium, necrosis of the liver, and congestion of the lungs in animals (Chmielewski et al. 1976, Coler and Rossmiller 1953, Stewart et al. 1970, von Oettingen 1964, Stewart 1969, NTP 1986b).

Some epidemiology studies have found an association between inhalation exposure to tetrachloroethylene and an increased risk for spontaneous abortion, idiopathic infertility, and sperm abnormalities among dry-cleaning workers, but others have not found similar effects (Kyyronen et al. 1989, van der Gulden and Zielhuis 1989). The adverse effects in humans are supported in part by the results of animal studies in which tetrachloroethylene induced fetotoxicity (but did not cause malformations) in the offspring of treated dams (Schwetz et al. 1975, Beliles et al. 1980, Nelson et al. 1980).

RfDs for subchronic and chronic oral exposure to tetrachloroethylene are 1E-1 mg/kg-day and 1E - 2 mg/kg-day, respectively (Buben and O'Flaherty 1985, EPA 1990d and 1998b). These values are based on hepatotoxicity observed in mice given 100 mg tetrachloroethylene/kg body weight for 6 weeks and a NOAEL of 20 mg/kg.

Epidemiology studies of dry-cleaning and laundry workers have demonstrated excesses in mortality ---due to various types of cancer, including liver cancer, but the data are regarded as inconclusive because of various confounding factors (Lynge and Thygesen 1990, EPA 1988c). The tenuous finding of an excess of liver tumors in humans is strengthened by the results of carcinogenicity bioassays in which tetrachloroethylene, administered either orally or by inhalation, induced hepatocellular tumors in mice (NCI 1977b, NTP 1986b). The chemical also induced mononuclear cell leukemia and renal tubular cell tumors in rats. Tetrachloroethylene was negative for tumor initiation in a dermal study and for tumor induction in a pulmonary tumor assay (Van Duuren et al. 1979, Theiss et al. 1977).

Although EPA's Science Advisory Board recommended a weight-of-evidence classification of C-B2 continuum (C = possible human carcinogen; B2 = probable human carcinogen), the agency has not adopted a current position on the weight-of-evidence classification (EPA 1992f). In an earlier evaluation, tetrachloroethylene was assigned to weight-of-evidence Group B2, probable human carcinogen, based on sufficient evidence from oral and inhalation studies for carcinogenicity in animals and no or inadequate evidence for carcinogenicity to humans (NCI 1977b, NTP 1986b, EPA 1987n). The unit risk and slope factor values for tetrachloroethylene have been withdrawn from IRIS and HEAST. The upper bound risk estimates from the 1985 Health Assessment Document (EPA 1985d) as amended by inhalation values from the 1987 addendum (EPA 1987n) have not yet been verified by the IRIS-CRAVE Workgroup. For oral exposure, the slope factor is 2.00E-3 (mg/kg-day)⁻¹; the unit risk ranges from 2.90E-7 to 9.50E-7 m³/µg with a geometric mean of 5.80E-7 m³/µg (EPA 1987n). When the agency makes a decision about weight-of-evidence, the CRAVE-IRIS verification will be completed and the information put on IRIS (EPA 1992f).

The oral and dermal cancer slope factors used in the BHHRA for tetrachloroethene are 5.20E-2 and 5.20E-2 (mg/kg-day)⁻¹, respectively. An inhalation cancer slope factor of 2.00E-3 (mg/kg-day)⁻¹ is used. The oral and dermal RfDs used in the BHHRA are 1.00E-2 and 1.00E-2 mg/kg-day, respectively. An inhalation RfD of 1.71E-1 mg/kg-day is used. When calculating the dermal route cancer slope factor from the oral value, a gastrointestinal absorption factor of 100 percent was used.

1.4.2.87 Toxaphene (CAS 008001-35-2)

An oral slope factor of 1.1E+0 (mg/kg-day)⁻¹ was used for toxaphene. For inhalation exposure, the inhalation unit risk is 3.2E-4 (μ g/m³)⁻¹. The absorbed dose slope factor is 2.2E+0 (mg/kg-day)⁻¹.

There are no RfDs or RfCs used in the BHHRA for toxaphene because of a lack of systemic toxicity and noncarcinogenic risk information.

1.4.2.88 trans-1,2-Dichloroethene (CAS 000156-60-5)

See Sect. 1.4.2.7.

1.4.2.89 trans-1,3-Dichloropropene (CAS 010061-02-6)

Information on the toxicity of *trans*-1,3-dichloropropene was not found in the available literature. When information becomes available, it will be included in this report.

Neither slope factors nor RfDs for any route of exposure were found for *trans*-1,3-dichloropropene. Therefore, neither carcinogenicity nor systemic toxicity resulting from *trans*-1,3-dichloropropene exposure is included in the BHHRA.

1.4.2.90 Trichloroethene (CAS 000079-01-6) (RAIS)

Trichloroethene is an industrial solvent used primarily in metal degreasing and cleaning operations. trichloroethene can be absorbed through the lungs, mucous membranes, gastrointestinal tract, and the skin. Trichloroethene is extensively metabolized in humans to trichloroacetic acid and trichloroethanol, as well as to several minor metabolites, with most of the absorbed dose excreted in urine (ATSDR 1989j, EPA 1985e).

Human and animal data indicate that exposure to trichloroethene can result in toxic effects on a number of organs and systems, including the liver, kidney, blood, skin, immune system, reproductive system, CNS, and cardiovascular system. In humans, acute inhalation exposure to trichloroethene causes CNS symptoms such as headache, dizziness, nausea, and unconsciousness (EPA 1985e). Among the reported effects from occupational exposure studies are fatigue, light-headedness, sleepiness, vision distortion, abnormal reflexes, tremors, ataxia, nystagmus, increased respiration, as well as neurobehavioral or psychological changes. Cardiovascular effects include tachycardia, extrasystoles, EKG abnormalities, and precordial pain (Landrigan et al. 1987, Grandjean et al. 1955, Milby 1968). The use of trichloroethene as an anesthetic has been associated with cardiac arrhythmias (EPA 1985e).

Cases of severe liver and kidney damage, including necrosis, have been reported in humans following acute exposure to trichloroethene (Defalque 1961), but these effects generally are not associated with long-term occupational exposures. In animals, trichloroethene has produced liver enlargement with hepatic biochemical and/or histological changes (Nomiyama et al. 1986, Kjellstrand et al. 1981 and 1983, Stott et al. 1982, Tucker et al. 1982) and kidney enlargement, renal tubular alterations and/or toxic nephropathy (NTP 1982a, 1986c, 1988). Also observed in animals were hematological effects (Tucker et al. 1982, Mazza and Brancaccio 1967) and immunosuppression (Sanders et al. 1982). Inhalation studies with rats indicate that trichloroethene is a developmental toxicant causing skeletal ossification anomalies and other effects consistent with delayed maturation (Healy et al. 1982, Dorfmueller et al. 1979). Trichloroethene may cause dermatitis and dermographism (EPA 1985e).

EPA (EPA 1992g) is presently reviewing RfDs and RfCs for subchronic and chronic oral and inhalation exposure to trichloroethene.

Epidemiologic studies have been inadequate to determine if a correlation exists between exposure to trichloroethene and increased cancer risk. Chronic oral exposure to trichloroethene increased the incidences of hepatocellular carcinomas in mice and renal adenocarcinomas and leukemia in rats (NTP 1988, Maltoni et al. 1986, NTP 1986c, 1982a; NCI 1976d). Chronic inhalation exposure induced lung and liver tumors in mice and testicular Leydig cell tumors in rats (Maltoni et al. 1986, 1988; Fukuda et al. 1983, Bell et al. 1978). Although EPA's Science Advisory Board recommended a weight-of-evidence classification of C-B2 continuum (C = possible human carcinogen; B2 = probable human carcinogen), the agency has not adopted a current position on the weight-of-evidence classification (EPA 1992h). In an earlier evaluation, trichloroethene was assigned to weight-of-evidence Group B2, probable human carcinogen, based on tumorigenic responses in rats and mice for both oral and inhalation exposure and on inadequate data in humans (EPA 1987o and 1990e). Carcinogen slope factors are 1.1E-2 (mg/kg-day)⁻¹ and 6.0E-3 (mg/kg-day)⁻¹ for oral and inhalation exposure, respectively. The corresponding unit risks are $3.2E-7 (\mu g/L)^{-1}$ and $1.7E-6 (\mu g/m^3)^{-1}$, respectively (EPA 1992h).

The oral, dermal, and inhalation cancer slope factors used in this BHHRA for trichloroethene are 1.10E-2, 7.33E-2, and 6.00E-3 (mg/kg-day)⁻¹, respectively. The oral, dermal, and inhalation RfDs are not available for trichloroethene. However, a provisional oral RfD for trichloroethene of 6.00E-3 mg/kg-day was used to estimate the noncarcinogenic hazard from the ingestion pathway. When calculating both the

dermal route cancer slope factor and dermal route RfD from their respective oral values, a gastrointestinal ---- absorption factor of 15 percent was used.

1:4.2.91 Vinyl chloride (CAS 000075-01-4) (RAIS)

Vinyl chloride (CAS Reg. No. 75-01-4), a colorless gas, is a halogenated aliphatic hydrocarbon with the empirical formula of C_2H_3Cl . It is used primarily as an intermediate in the manufacture of PVC; limited quantities are used as a refrigerant and as an intermediate in the production of chlorinated compounds (ATSDR 1989k).

Vinyl chloride is rapidly absorbed from the gastrointestinal tract and lungs. Metabolism of vinyl chloride occurs primarily in the liver via oxidation by hepatic microsomal enzymes to polar compounds that can be conjugated with glutathione and/or cysteine. These covalently bound metabolites are then excreted in the urine (EPA 1980e and 1985f).

In humans and animals, vinyl chloride is a CNS depressant, inducing narcosis and anesthesia at high concentrations (Torkelson and Rowe 1981b, Patty et al. 1930). Nonneoplastic toxic effects observed in workers exposed by inhalation to vinyl chloride include hepatotoxicity, acroosteolysis and scleroderma, and Raynaud's syndrome, a vascular disorder of the extremities. Also reported were abnormalities of CNS function, high blood pressure, and occasional pulmonary effects (ATSDR 1989k, EPA 1985f, Lloyd et al. 1984, Langauer-Lewowicka et al. 1983, Waxweiler et al. 1977). The evidence for potential developmental effects in humans (increased fetal loss and birth defects) is equivocal (ATSDR 1989k, Waxweiler et al. 1977, Infante et al. 1976). Occupational exposure to vinyl chloride has been associated with reduced sexual function in both sexes and gynecological effects in women (Makarov 1984, Makarov et al. 1984).

For the oral route of exposure, the primary target organ of vinyl chloride toxicity in animals is the liver. Chronic oral administration of 1.7–14.1 mg/kg-day of vinyl chloride induced dose-related increases in nonneoplastic lesions of the liver of rats (Feron et al. 1981). In addition to the CNS, target organs for inhalation exposure include the liver, kidneys, lungs, spleen, and testes. Subchronic inhalation studies with rodents documented hepatic effects at concentrations as low as 50 ppm (Sokal et al. 1980) and degenerative changes of the liver and kidneys at greater than or equal to 500 ppm (Torkelson et al. 1961). Exposure to higher concentrations caused proliferative changes in the lungs of mice (Suzuki 1980), extensive liver and kidney damage in rats and guinea pigs, cerebral and cerebellar nephrosis in rats, and degeneration of the spleen in guinea pigs (Prodan et al. 1975, Viola et al. 1971). Subchronic exposure of rats to 100 ppm vinyl chloride produced significantly decreased testes weights and testicular regeneration (Bi et al. 1985). Evidence of developmental toxicity was seen in rats exposed to vinyl chloride during the first trimester of gestation (Ungvary et al. 1978).

Neither an oral RfD nor an inhalation RfC have been derived for vinyl chloride (EPA 1998b).

The carcinogenicity of vinyl chloride in humans has been demonstrated in a number of epidemiological studies and case reports, many of which associated occupational exposure to vinyl chloride to the development of angiosarcomas of the liver. In addition to liver cancer, exposure to vinyl chloride also has been linked to an increased risk of lung, brain, hematopoietic, and digestive tract cancers (EPA 1985f, Heldaas et al. 1984, IARC 1979d, Byren et al. 1976, Waxweiler et al. 1976, Monson et al. 1974). Vinyl chloride has been shown to be carcinogenic in numerous animal studies. Inhalation exposure to vinyl chloride induced an increased incidence of liver angiosarcomas; kidney nephroblastomas; and lung, brain, and forestomach tumors in rodents (Maltoni et al. 1980, 1981; Feron et al. 1981; Hong et al. 1981; Suzuki 1978; Lee et al. 1977, 1978a). Oral administration of vinyl chloride induced liver, lung, and kidney tumors in rodents (Feron et al. 1981, Maltoni 1977). Angiosarcomas observed in offspring of rats



EPA has classified vinyl chloride as a Group A chemical, human carcinogen (EPA 1985f). A slope factor of 1.9E+0 (mg/kg-day)⁻¹ and a drinking water unit risk of 5.4E-5 (μ g/L)⁻¹ was calculated for oral exposure to vinyl chloride (EPA 1998b). For inhalation exposure, the slope factor and inhalation unit risk are 3.0E-1 (mg/kg-day)⁻¹ and 8.4E-5 (μ g/m³)⁻¹, respectively. The oral slope factor and inhalation unit risk are currently under review and may be subject to change (EPA 1998b).

An oral slope factor of 1.9E+0 (mg/kg-day)⁻¹ was calculated for vinyl chloride. For inhalation exposure, the slope factor is 3.0E-1 (mg/kg-day)⁻¹. A gastrointestinal absorption factor of 100 percent was used to derive an absorbed dose slope factor of 1.90E+0. No RfDs were available.

1.4.2.92 Xylene (CAS 000075-01-4) (RAIS)

Xylene (dimethylbenzene) is a colorless, flammable liquid that is used as a solvent in the printing, rubber, and leather industries and as a cleaner and paint thinner. It occurs naturally in petroleum and coal tar. Xylene is absorbed following oral, dermal, or inhalation exposure; can be stored in adipose tissue; and is eliminated in the urine after conjugation with glycine.

Human exposure to xylene by either oral or inhalation routes can cause death due to respiratory failure accompanied by pulmonary congestion (Sandmeyer 1981). Nonlethal levels of xylene vapor may cause eye (Carpenter et al. 1975), nose, and throat (ATSDR 1993f) irritation, and contact with liquid may result in dermatitis (Sittig 1985a). Chronic occupational exposure to xylene has been associated with headaches, chest pain, electrocardiographic abnormalities, dyspnea, cyanosis of hands, fever, leukopenia, malaise, impaired lung function, and confusion (Hipolito 1980).

Long-term gavage studies with mixed xylenes in laboratory animals resulted in decreased body weight gain in male rats given 500 mg/kg-day and hyperactivity in male and female mice given 1000 mg/kg-day (NTP 1986d). An oral RfD_c of 2 mg/kg-day for mixed xylenes was calculated from a NOAEL of 250 mg/kg-day derived from a chronic gavage study with rats (EPA 1998a). The critical effects were hyperactivity, decreased body weight, and increased mortality (males). An RfD of 2 mg/kg-day is also reported for the *m*- and *o*-xylene isomers (EPA 1998b).

Inhalation of 3000 mg/m³ of the o-, p-, or m-xylene isomer by rats on gestation days 7-14 resulted in decreased fetal weights, skeletal anomalies, and altered fetal enzyme activities (Hood and Ottley 1985). Rib anomalies and cleft palate occurred in mouse fetuses following maternal oral exposure of 2.06 mg/kg-day of mixed xylenes on gestation days 6-15 (Marks et al. 1982). EPA (1998a) is reviewing an inhalation RfC.

Oral (NTP 1986d) and topical (Berenblum 1941, Pound 1970) carcinogenic studies with xylene in laboratory animals gave negative results. EPA (1998a) has placed xylene in weight-of-evidence Group D, not classifiable as to human carcinogenicity. No significant increase in tumor incidence was observed in rats or mice of both sexes following oral administration of technical grade xylene.

There are no carcinogenic slope factors used in the BHHRA for xylene because of a lack of carcinogenic toxicity information.

An oral RfD of 2.0E+0 mg/kg-day is used in this risk assessment for xylene. The dermal route RfD used in the BHHRA is 1.84E+0 mg/kg-day. The inhalation RfC is not available.

1.4.3 Radionuclides

Radionuclides are unstable atoms of chemical elements that will emit charged particles or energy or both to achieve a more stable state. These charged particles are termed "alpha and beta radiation"; energy is termed "neutral gamma rays." Interaction of these charged particles (and gamma rays) with matter will produce ionization events, or radiation, which may cause living cell tissue damage. Because the deposition of energy by ionizing radiation is a random process, sufficient energy may be deposited (in a critical volume) within a cell and result in cell modification or death. In addition, ionizing radiation has sufficient energy that interactions with matter will produce an ejected electron and a positively charged ion (known as free radicals) that are highly reactive and may combine with other elements, or compounds within a cell, to produce toxins or otherwise disrupt the overall chemical balance of the cell. These free radicals can also react with DNA, causing genetic damage, cancer induction, or even cell death.

Radionuclides are characterized by the type and energy level of the radiation emitted. Radiation emissions fall into two major categories: particulate (electrons, alpha particles, beta particles, and protons) or electromagnetic radiation (gamma and X rays). Therefore, EPA classifies all radionuclides as Group A carcinogens based on their property of emitting ionizing radiation and on the extensive weight-of-evidence provided by epidemiological studies of humans with cancers induced by high doses of radiation. Alpha particles are emitted at a characteristic energy level for differing radionuclides. The alpha particle has a charge of +2 and a comparably large size. Alpha particles have the ability to react (and/or ionize) with other molecules, but they have very little penetrating power and lack the ability to pass through a piece of paper or human skin. However, alpha-emitting radionuclides are directly ionizing and deposit their energy in dense concentrations [termed high linear energy transfer (high LET)], resulting in short paths of highly localized ionization reactions. The probability of cell damage increases as a result of the increase in ionization events occurring in smaller areas; this may also be the reason for increased cancer incidence caused by inhalation of radon gas. In addition, the cancer incidence in smokers may be directly attributed to the naturally occurring alpha emitter, polonium-210, in common tobacco products.

Beta emissions generally refer to beta negative particle emissions. Radionuclides with an excess of neutrons achieve stability by beta decay. Beta radiation, like alpha radiation, is directly ionizing but, unlike alpha activity, beta particles deposit their energy along a longer track length (low-LET), resulting in more space between ionization events. Beta-emitting radionuclides can cause injury to the skin and superficial body tissue but are most destructive when inhaled or ingested. Many beta emitters are similar chemically to naturally occurring essential nutrients and will therefore tend to accumulate in certain specific tissues. For example, strontium-90 is chemically similar to calcium and, as a result, accumulates in the bones, where it causes continuous exposure. The health effects of beta particle emissions depend upon the target organ. Those seeking the bones would cause a prolonged exposure to the bone marrow and affect blood cell formation, possibly resulting in leukemia, other blood disorders, or bone cancers. Those seeking the liver would result in liver diseases or cancer, while those seeking the thyroid would cause thyroid and metabolic disorders. In addition, beta radiation may lead to damage of genetic material (DNA), causing hereditary defects.

Gamma emissions are the energy that has been released from transformations of the atomic nucleus. Gamma emitters and X rays behave similarly but differ in their origin: gamma emissions originate in nuclear transformations, and X rays result from changes in the orbiting electron structure. Radionuclides that emit gamma radiation can induce internal and external effects. Gamma rays have high penetrating ability in living tissue and are capable of reaching all internal body organs. Without such sufficient shielding as lead, concrete, or steel, gamma radiation can penetrate the body from the outside and does not require ingestion or inhalation to penetrate sensitive organs. Gamma rays are characterized as low-LET radiation, as is beta radiation; however, the behavior of beta radiation differs from that of gamma radiation in that beta particles deposit most of their energy in the medium through which they pass, while gamma rays often escape the medium because of higher energies, thereby creating difficulties in determining actual internal exposure. For this reason, direct whole-body measurements are necessary to detect gamma radiation, while urine/fecal analyses are usually effective in detecting beta radiation.

People receive gamma radiation continuously from naturally occurring radioactive decay processes going on in the earth's surface, from radiation naturally occurring inside their bodies, from the atmosphere as fallout from nuclear testing or explosions, and from space or cosmic sources. Cesium-137 (from nuclear fallout) decays to barium-137, the highest contributor to fallout-induced gamma radiation. Beta radiation from the soil is a less penetrating form of radiation but has many contributing sources. Potassium-40, cesium-137, lead-214, and bismuth-214 are among the most common environmental beta emitters. Tritium is also a beta emitter but contributes little to the soil beta radiation because of the low energy of its emission and its low concentration in the atmosphere. Alpha radiation is also emitted by the soil but is not measurable more than a few centimeters from the ground surface. The majority of alpha emissions are attributable to radon-222 and radon-220 and their decay products. This contributes to what is called background exposure to radiation.

The general health effects of radiation can be divided into stochastic (related to dose) and nonstochastic (not related to dose) effects. The risk of development of cancer from exposure to radiation is a stochastic effect. Examples of nonstochastic effects include acute radiation syndrome and cataract formation, which occur only at high levels of exposures.

Radiation can damage cells in different ways. It can cause damage to DNA within the cell, and the cell either may not be able to recover from this type of damage or may survive but function abnormally. If an abnormally functioning cell divides and reproduces, a tumor or mutation in the tissue may develop. The rapidly dividing cells that line the intestines and stomach and the blood cells in bone marrow are extremely sensitive to this damage. Organ damage results from the damage caused to the individual cells. This type of damage has been reported with doses of 10-500 rads (0.1-5.0 gray, in SI units). Acute radiation sickness is seen only after doses of greater than 50 rads (0.5 gray), which is a dose rate usually achieved only in a nuclear accident.

When the radiation-damaged cells are reproductive cells, genetic damage can occur in the offspring of the person exposed. The developing fetus is especially sensitive to radiation. The type of malformation that may occur is related to the stage of fetal development and the cells that are differentiating at the time of exposure. Radiation damage to children exposed in the womb is related to the dose the pregnant mother receives. Mental retardation is a possible effect of fetal radiation exposure.

The most widely studied population that has had known exposure to radiation is the atomic bomb survivors of Hiroshima and Nagasaki, Japan. Data indicate an increase in the rate of leukemia and cancers in this population. However, the rate at which cancer incidence is significantly affected by low radiation exposures, such as results of exposure to natural background and industrially contaminated sites, is still undergoing study and is uncertain. In studies conducted to determine the rate of cancer and leukemia increase, as well as genetic defects, several radionuclides must be considered.

1.4.3.1 Cesium-137 (CAS 010045-97-3)

Cesium occurs in nature as cesium-133 in the aluminosilicates, pollucite (a hydrated silicate of aluminum and cesium) and lepidolite; in the borate, rhodizite; and in other sources (Budavari et al. 1989, Klaassen et al. 1986). Cesium-137 is one of the artificial isotopes of cesium and is one of the principle radionuclides present in reactor effluent under normal operations. Cesium-137 may also be produced in nuclear and thermonuclear explosions, through which it would be a primary contributor to human

exposure through fallout radiation, assimilation through the food chain, or beta dose to the skin (Budavari --- et al. 1989, Klaassen et al. 1986). In addition, Cesium-137, along with strontium-90, is one of the most important fission products that was widely distributed in near-surface soils because of historical weapons testing. Measurable concentrations still exist today, almost exclusively in the upper 15 cm of soil; these concentrations decrease roughly exponentially with depth.

Cesium-137 may also have important roles in medical treatments (a teletherapy source or intercavity or interstitial radiation source in treatment of malignancies) and as an encapsulated energy source (Budavari et al. 1989, Casarett 1968). Cesium-137 decays to and reaches radioactive equilibrium with its daughter product, barium-137m (Budavari et al. 1989, Casarett 1968). Barium-137m is a very short-lived gamma emitter that can contribute to external gamma exposure (Budavari et al. 1989).

Oral, inhalation, and external exposure cancer slope factors used in the BHHRA for cesium-137 are 3.16E-11 risk/pCi, 1.91E-11 risk/pCi, and 2.09E-06 [(risk × g)/(pCi × yr)], respectively. For cesium-137, the cancer slope factor used in the BHHRA includes risks posed by short-lived decay products in addition to that posed by the parent radionuclide. Oral, dermal, and inhalation RfDs are not available for this element; therefore, systemic toxicity because of exposure to cesium is not quantified in the BHHRA.

1.4.3.2 Neptunium-237 (CAS 013994-20-2)

Specific literary information for neptunium-237 is limited. However, available literature states that during neutron bombardment, neptunium-237 breaks down to plutonium-238, which produces small masses of high capacity energy that is useful for satellites and spacecraft (Moskalev et al. 1979).

The most common route of neptunium-237 exposure is inhalation of aerosols. According to studies conducted on rats, acute effects include injury to the liver and kidney and circulation disorders. Long-term effects include osteosarcomas and lung cancer. Extremely high doses cause immediate or premature death by destruction of the lungs (Moskalev et al. 1979).

Oral, inhalation, and external exposure cancer slope factors used in the BHHRA for neptunium-237 are 3.00E-10 risk/pCi, 3.45E-08 risk/pCi, and 4.62E-07 [(risk × g)/(pCi × yr)], respectively. A dermal cancer slope factor was not calculated because this route of exposure is not considered significant for radionuclides and is not evaluated in the BHHRA. Oral, dermal, and inhalation RfDs are not available for this element; therefore, systemic toxicity due to exposure to neptunium is not quantified in the BHHRA.

1.4.3.3 Radon-222 (CAS 014859-67-7)

Radon belongs to the noble gases and is the heaviest known gas. It is colorless and odorless at standard temperature and pressure. When cooled below the freezing point, radon exhibits a brilliant phosphorescence that becomes yellow as the temperature is lowered and orange-red at the temperature of liquid air.

Radon is formed naturally in soil, groundwater, and air as a daughter product in the decay chain of NORM uranium found in the earth's crust. Radon-222 has a half-life of 3.82 days and decays through alpha emission at 5.590 MeV to pollonium-219. Excessive radon buildup in basements of homes from the surrounding soils, rocks, and groundwater is an inhalation hazard, both from direct inhalation and from inhalation of absorbed radon and daughter products on dust particles.

To derive the inhalation slope factor for radon-222 plus daughter products, EPA's Office of Radiation and Indoor Air (ORIA) uses a slightly different risk model and set of exposure assumptions, including an inhalation rate of 2.2E+04 L/day; 50 percent equilibrium for decay products; and a risk

coefficient of 2.36E-4 cases per working level month (WLM). A more detailed description of ORIA's radon risk assessment methodology is provided in the EPA CRAVE Summary Sheet, "Inhaled Radon-222 and Its Short Half-Life Decay Products."

The inhalation slope factor derived for radon-222 plus daughter products used in this BHHRA is 7.57E-12 [(risk \times g)/(pCi \times yr)]. Oral, dermal, and external exposure cancer slope factors were not calculated because these routes of exposure are not considered significant. Oral, dermal, and inhalation RfDs are not available for this element; therefore, systemic toxicity is not quantified in the BHHRA.

1.4.3.4 Technetium-99 (CAS 014133-76-7)

Technetium is a radioactive element that occurs in a number of isotopic forms. Technetium is found in some extraterrestrial material (i.e., stars); however, no appreciable amounts have been found in nature due to the relatively short half-lives of its radioactive isotopes (Kutegov et al. 1968). While no isotopes of technetium, technetium-97 and technetium-98 have half-lives of 2.6E-6 and 1.5E-6 years, respectively. The third isotope, technetium-99, has a half-life of 2.12E-5 years. None, however, possesses a half-life sufficiently long to allow technetium to occur naturally (Boyd 1959). Technetium is made artificially for industrial use, and natural technetium, particularly technetium-99, has been identified and isolated from the spontaneous fission of uranium, as well as other fissionable material, or via the irradiation of molybdenum (Venugopal and Luckey 1978b, Clarke and Podbielski 1988).

Technetium is an emitter of beta particles of low specific activity (Boyd 1959). It does not release nuclear energy at a rate sufficient to make the element attractive for the conventional application of radioactivity (Boyd 1959). Technetium-99 is the only long-lived isotope that is readily available and is the isotope on which most of the chemistry of technetium is based. Although gamma radiation has not been associated with technetium-99, the secondary X rays may become important with larger amounts of the element.

Oral, inhalation, and external exposure cancer slope factors used in this BHHRA for technetium-99 are 1.40E-12 risk/pCi, 2.89E-12 risk/pCi, and 6.19E-13 [(risk \times g)/(pCi \times yr)], respectively. A dermal cancer slope factor was not calculated because this route of exposure is not evaluated in this BHHRA. Oral, dermal, and inhalation RfDs are not available for this element; therefore, systemic toxicity due to exposure to technetium-99 is not quantified.

1.4.3.5 Thorium-234 (CAS 15065-10-8)

Thorium is a naturally occurring, radioactive metal. Small amounts of thorium are present in all rocks, soil, aboveground and underground water, plants, and animals. These small amounts of thorium contribute to the weak background radiation for such substances. Soil commonly contains an average of about 6 ppm of soil. Rocks in some underground mines may also contain thorium in a more concentrated form. After these rocks are mined, thorium is usually concentrated and changes into thorium dioxide or other chemical forms. Thorium-bearing rock that has had most of the thorium removed from it is called "depleted" ore or tailings (ATSDR 1990o).

Thorium is a metallic element of the actinide series. It exists in several isotopic forms. The isotope thorium-232 is a naturally occurring element that is radioactive. It decays through the emission of a series of alpha and beta particles, gamma radiation, and the formation of daughter products, finally yielding the stable isotope of lead, lead-208. Isotopes thorium-234 and thorium-230 are produced during the decay of naturally occurring uranium-238, the isotope thorium-228 during the decay of thorium-232, and the isotopes thorium-231 and thorium-227 during the decay of uranium-235. Of these naturally produced isotopes of thorium, only thorium-232, thorium-230, and thorium-228 have long enough half-lives to be

environmentally significant. More than 99.99 percent of natural thorium is thorium-232; the rest is_--thorium-230 and thorium-228 (ATSDR 19900).

Thorium is used to make ceramics, lantern mantles, and metals used in the aerospace industry and in nuclear reactions. Thorium can also be used as a fuel for generating nuclear energy. More than 30 years ago, thorium oxides were used in hospitals to make certain kinds of diagnostic X-ray photographs (ATSDR 19900).

Because thorium is found almost everywhere, most people in the United States eat some thorium with their food every day. Normally, little of the thorium in lakes, rivers, and oceans gets into fish or seafood used commercially. More thorium may be found near uncontrolled hazardous waste sites that contain thorium that might not have been disposed of properly. Consequently, people living near one of these sites may be exposed to slightly more thorium as a result of inhaling windblown dust containing thorium or eating food grown in soil contaminated with thorium. Larger-than-normal amounts of thorium might also enter the environment through accidental releases from thorium processing plants (ATSDR 1990o).

Breathing dust contaminated with thorium is the primary pathway for thorium exposure to the body. A large portion of this dustborne thorium will be eliminated by normal bodily functions (urine/feces); however, a small amount of thorium will be taken up by the blood and subsequently transmitted to the bones. Breathing thorium dust may cause an increased chance of developing lung disease and cancer of the lung or pancreas many years after being exposed. Changes in genetic material have also been shown to occur in workers who breathed thorium dust. Liver diseases and effects on the blood have been found in people injected with thorium to take special X rays. Many types of cancer have been shown to occur in these people many years after thorium was injected in their bodies. Because thorium is radioactive and may be stored in bone for a long time, bone cancer is also a potential concern for people exposed to thorium. Animal studies have shown that breathing in thorium may result in lung damage. Other studies in animals suggest drinking massive amounts of thorium can cause death from metal poisoning. The presence of large amounts of thorium, such as radium and thoron, which is an isotope of radon. Thorium is not known to cause birth defects or to affect childbearing abilities (ATSDR 1990o).

Oral, inhalation, and external exposure cancer slope factors used in the BHHRA for thorium-228 and its short-lived daughter products are 2.31E-10 risk/pCi, 9.68E-08 risk/pCi, and 6.20E-06 [(risk \times g)/(pCi \times yr)], respectively. The slope factors for thorium-228 include ingrowth of daughters. Oral, inhalation, and external exposure cancer slope factors used in the BHHRA for thorium-230 are 3.75E-11 risk/pCi, 1.72E-08 risk/pCi, and 4.40E-11 [(risk \times g)/(pCi \times yr)], respectively. Oral, inhalation, and external exposure cancer slope factors used in the BHHRA for thorium-234 are 1.93E-11 risk/pCi, 1.90E-11 risk/pCi, and 3.50E-09 [(risk \times g)/(pCi \times yr)], respectively. A dermal cancer slope factor was not calculated because this route of exposure is not considered significant for radionuclides and is not evaluated in the BHHRA. Oral, dermal, and inhalation RfDs are not available for this element; therefore, systemic toxicity due to exposure to thorium is not quantified in the BHHRA.

1.4.3.6 Uranium (CAS 013966-29-5 for uranium-234, CAS 15117-96-1 for uranium-235, and CAS 07440-61-1 for uranium-238)

Uranium is a mildly radioactive element that occurs widely in the earth's crust. It is found in all soils, most rocks, and, in lesser concentrations, in water, vegetation, and animals, including humans. Uranium emits a low level of alpha particles and a much lower level of gamma rays. Alpha particles are unable to penetrate skin but can travel short distances in the body if ingested or inhaled. Consequently, uranium represents a significant carcinogenic hazard only when taken into the body, where alpha particle energy is absorbed by small volumes of tissue. Although the penetrating (gamma) radiation of uranium is not considered to be significant (ATSDR 19891), one of its daughter radionuclides is a strong gamma emitter. Therefore, gamma radiation may be a concern in areas containing uranium.

Natural uranium contains the uranium isotopes uranium-238 (which averages 99.27 percent of total uranium mass), uranium-235 (0.72 percent), and uranium-234 (0.0056 percent), each of which undergoes radioactive decay. Natural uranium, therefore, contains the radionuclide daughter products from the decay of uranium-238 and uranium-235 (Bowen 1979, ATSDR 19891).

Uranium is a radioactive element, but it is also a metallic element. Toxicological effects from the ingestion of uranium are the result of the action of uranium as a metal and its radioactive properties. The primary toxic chemical effect of uranium is seen in kidney damage. Studies in rabbits, mice, and dogs showed effects on the kidney to be dose-related. Fetal skeletal abnormalities and fetal death were found in pregnant mice exposed to 6 mg/kg or uranyl acetate dihydrate.

The primary human exposure studies to uranium have been studies of uranium miners or uranium factory workers. These studies have shown an increase in lung cancer deaths among these workers, which may be attributable to the decay of uranium into radon and its daughters. These workers are exposed to high levels of uranium dust and fumes and other radioactive elements in confined conditions (ATSDR 1989)).

Oral, inhalation, and external exposure cancer slope factors used in the BHHRA for uranium-234 are 4.44E-11 risk/pCi, 1.40E-08 risk/pCi, and 2.14E-11 [(risk \times g)/(pCi \times yr)], respectively. Oral, inhalation, and external exposure cancer slope factors used in the BHHRA for uranium-235 are 4.70E-11 risk/pCi, 1.30E-08 risk/pCi, and 2.65E-07 [(risk \times g)/(pCi \times yr)], respectively. The slope factors for uranium-235 include ingrowth of daughters. Oral, inhalation, and external exposure cancer slope factors used in the BHHRA for uranium-238 are 6.20E-11 risk/pCi, 1.24E-08 risk/pCi, and 6.57E-08 [(risk \times g)/(pCi \times yr)], respectively. The slope factors used in the BHHRA for uranium-238 are 6.20E-11 risk/pCi, 1.24E-08 risk/pCi, and 6.57E-08 [(risk \times g)/(pCi \times yr)], respectively. The slope factors for uranium-238 include ingrowth of daughters. A dermal cancer slope factor was not calculated because this route of exposure is not considered significant for radionuclides and is not evaluated in the BHHRA. Oral, dermal, and inhalation RfDs are not available for this element; therefore, systemic toxicity due to exposure to neptunium is not quantified in the BHHRA.

1.4.4 Chemicals for Which No EPA Toxicity Values Are Available

Oral RfD values exist for all of the inorganic COPCs included in the WAG 28 BHHRA except ammonia, silica, sulfate, tetraxo-sulfate, and thallium. Provisional values were found for copper and lead. Oral RfDs exist for all of the organic COPCs included in the WAG 28 BHHRA except 1,4-dichlorobenzene, 2,4,6-trichlorophenol, 1,3-dichlorobenze, 2-hexanone. 1,2-dichloroethane, 2-methyl-4,6-dinitrophenol, 2-methylnaphthalene, 2-nitroaniline, 2-nitrophenol, 3,3-dichlorobenzidine, 3-nitroaniline, 4,4'-DDD, 4,4'-DDE, 4,4'-DDT, 4-bromophenyl-phenylether, 4-chloro-3-methylphenol, 4-chlorophenyl-phenyl ether, 4-nitroaniline, acenaphthylene, alpha chlordane, benz(a)anthracene, benzene, benzo(a)pyrene, benzo(b)fluoroanthene, benzo(g,h,i)perylene, benzo(k)fluoranthene, beta bromodichloromethane, BHC, bis(2-chloroethoxy)methane, bis(2-chloroethyl)ether, chrysene. cis-1,3-dichloropropene, delta BHC, dibenzo(a,h)anthracene, dieldrin, endosulfan I & II, endosulfan endrin ketone, gamma-chlordane, indeno(1,2,3-cd)pyrene, n-nitroso-di-n-propylamine, sulfate. n-nitrosodiphenylamine, phenanthrene, PCBs 1221, 1232, 1242, 1248, and 1260, toxaphene, trans-1,3-dichloropropene, and vinyl chloride.

All the inorganic COPCs, except ammonia, barium, beryllium, cadmium, managanese, and mercury, lack inhalation RfD values. Cadmium has a value for cadmium (diet) based on both diet and water exposures. Lead has a provisional inhalation RfD value. Of the organics, the following do not have

inhalation RfD values: acenaphthylene, PBCs 1221, 1232, 1242, 1248, and 1260, benz(a)anthracene, --benzo(g,h,i)perylene, benzo(k)fluoranthene benz(a)pyrene, benzo(b)fluoranthene, bis(2-chloroethyoxy)methane, bis(2-chloroethyl)ether, 4-bromophenyl phenyl, bromodichloromethane 4-chloro-3-methylphenol, 4-chlorophenyl phenyl ether, chrysene, 4,4'-DDD, 4,4'-DDE, dibenz(a,h)anthracene, 3,3-dichlorobenzidine, 4,6-dinitrophenol, alpha hexachlorocyclohexane, beta indeno(1,2,3-cd)pyrene, hexachlorocyclohexane, delta hexachlorocyclohexane, 2-hexanone, 3-nitroaniline, 4-nitroaniline, 2-nitrophenol, n-nitroso-di-n-propylamine, n-nitrosodiphenylamine, phenanthrene, toxaphene, 2,4,6-trichlorophenol, and vinyl chloride. EPA is currently developing inhalation RfD values for several of these compounds and recommends that, until these values have been verified, the noncarcinogenic effects of inhalation of substances without EPA-derived RfC values be evaluated qualitatively.

Absorbed dose RfD values exist for all of the inorganic COPCs included in the WAG 28 BHHRA except ammonia, copper, silica, sulfate, tetraxo-sulfate, and thallium. The value for lead is a provisional value. Absorbed dose RfDs exist for all of the organic COPCs included in the WAG 28 BHHRA except 1.2-dichloroethane, 1,3-dichlorobenze, 1,4-dichlorobenzene, 2,4,6-trichlorophenol, 2-hexanone. 2-methyl-4,6-dinitrophenol, 2-methylnaphthalene, 2-nitroaniline, 2-nitrophenol, 3,3-dichlorobenzidine, 3-nitroaniline, 4,4'-DDD, 4,4'-DDE, 4,4'-DDT, 4-bromophenyl-phenylether, 4-chloro-3-methylphenol, 4-chlorophenyl-phenyl ether, 4-nitroaniline, acenaphthylene, alpha chlordane, benz(a)anthracene, benzene, benzo(a)pyrene, benzo(b)fluoroanthene, benzo(g,h,I)perylene, benzo(k)fluoranthene, beta BHC, bis(2-chloroethoxy)methane. bis(2-chloroethyl)ether, bromodichloromethane. chrysene. cis-1,3-dichloropropene, delta BHC, dibenzo(a,h)anthracene, dieldrin, endosulfan I & II, endosulfan endrin ketone, gamma-chlordane, indeno(1,2,3-cd)pyrene, n-nitroso-di-n-propylamine. sulfate. n-nitrosodiphenylamine, phenanthrene, PCBs 1221, 1232, 1242, 1248, and 1260, toxaphene, trans-1,3-dichloropropene, and vinyl chloride.

Oral slope factors for inorganic compounds are only available for arsenic and beryllium. Oral slope factors do not currently exist for 41 of the 93 inorganic COPCs included in this assessment. The organic compounds without oral slope factors are acenapthene, acenaphthylene, acetone, anthracene, beno(g,h,i)perylene, bis(2-chloroethoxy)methane, 4-bromophenyl phenyl ether, di-n-butylphthalate, butyl benzyl phthlate, 4-chlorophenyl phenyl ether, beta-chloronapthalene, dibensofuran, 1,2-dichlorobenzene, 1,3-dichlorobenzene, 1,1-dichloroethane, cis- and trans-1,2-dichloroethylene, 2-methyl-4,6-dinitrophenol, endrin. ethylbenzene. fluoranthene. fluorene. delta hexachlorocyclohexane. endosulfan. hexachlorcyclopentadiene, 2-hexanone, methoxychlor, 2-methylnaphthalene, naphthalene, 2-nitroaniline, 3-nitroaniline, 4-nitroaniline, 2-nitrophenol, di-n-octyl phthalate, phenanthrene, pyrene, 1,2,4-trichlorobenzene, 2,4,5-trichlorophenol, and xylene.

EPA-approved inhalation slope factors are available for only a few of the COPCs. Inorganic COPCs with inhalation slope factors are arsenic, beryllium, cadmium, and chromium. Organic COPCs with approved inhalation slope factors are 1,1,2-trichloroethane. 1,1-dichloroethene, 1,2-dichloroethane, benz(a)anthracene, benzene, 1,3-dichloropropene, 2,4,6-trichlorophenol. 4.4'DDT. aldrin, benzo(a)pyrene. benzo(b)fluoroanthene. benzo(k)fluoranthene, bis(2-chloroethyl)ether, carbon tetrachloride, alpha and gamma chlorodane, chloroform, chrysene, dibenzo(a,h)anthracene, dieldrin, epoxide, hexachlorobenzene, hexachlorobutadiene, alpha and beta heptachlor, heptachlor hexachlorocyclohexane, hexachloroethane, indeno(1,2,3-cd)pyrene, methylene chloride, PCBs, 1016, 1221, 1232, 1242, 1248, 1254, and 1260, tetrachloroethene, toxaphene, 1,1,2-trichloroethane, trichloroethene, 2,4,6-trichlorophenol, and vinyl chloride.

Fifty-four COPCs have absorbed dose slope factors. Two of these are the inorganics arsenic and beryllium. Fifty-two are organic compounds (these are identical to those analytes having oral slope

factors). All radionuclide COPCs except radon-222 have oral, inhalation, and external exposure slope factors. Radon-222 has an inhalation slope factor.

1.4.5 Uncertainties Related to Toxicity Information

Standard EPA RfDs and slope factors were used to estimate potential noncarcinogenic and carcinogenic health effects from exposure to chemical contaminants detected at WAG 28. Considerable uncertainty is associated with the methodology applied to derive slope factors and RfDs. EPA working groups review all relevant human and animal studies for each compound and select the studies pertinent to the derivation of the specific RfD and slope factor. These studies often involve data from experimental studies in animals, high exposure levels, and exposures under acute or occupational conditions. Extrapolation of these data to humans under low-dose, chronic conditions introduces uncertainties. The magnitude of these uncertainties is addressed by applying uncertainty factors to the dose response data for each applicable uncertainty. These factors are incorporated to provide a margin of safety for use in human health assessments.

The dose-response relationship between cancer and ionizing radiation has been evaluated in many reports. Derivation of risk factors is extrapolated from the cancer risk established using the Japanese Atomic Bomb Survivors database and a relative risk projection model. EPA methodology for estimating radionuclide carcinogenic risks is currently being reevaluated.

1.4.6 Summary of Toxicity Assessment

A breakdown of the COPCs and their available toxicity information by site is provided in the following sections.

1.4.6.1 SWMU 99a COPC toxicity summary

SWMU 99a surface soil contains 29 COPCs (4 inorganics, 19 organics, and 6 radionuclides). All of the radionuclides and the inorganics have some toxicity information. Three of the 19 organics do not have toxicity information. Subsurface soil contains 101 COPCs (11 inorganics, 84 organics, and 6 radionuclides). All the radionuclides have toxicity information. Ten of the 11 inorganics have toxicity information, and 81 of the 101 organics do. RGA groundwater contains 24 COPCs (18 inorganics, 4 organics, and 2 radionuclides). There is toxicity information for all the radionuclides, 14 of the 18 inorganics and for all of the organics. McNairy groundwater contains four COPCs (four organics) and four out of the four have toxicity information.

1.4.6.2 SWMU 99b COPC toxicity summary

SWMU 99b subsurface soil contains five COPCs (four inorganics and one organic), all of which have toxicity information. RGA groundwater contains 10 COPCs (8 inorganics, 1 organic, and 1 radionuclide). The radionuclide and the organic both have toxicity information as do five of the eight inorganics. Surface soil and McNairy groundwater were not assessed.

1.4.6.3 SWMU 193a COPC toxicity summary

SWMU 193a surface soil contains 14 COPCs (1 inorganic and 13 organics). Of these, the inorganic has toxicity information available as do 12 of the 13 organics. The subsurface soil contains 16 COPCs (three organics and 13 inorganics). All three inorganics have toxicity information, and 12 of the 13 organics have toxicity information. RGA groundwater contains 12 COPCs (6 inorganics, 5 organics, and 1 radionuclide). Four of the six inorganics and five of the five organics have toxicity information.

1.4.6.4 SWMU 193b COPC toxicity summary

SWMU 193b surface soil contains three COPCs (three inorganics), all three of which have toxicity information. The subsurface soil also contains three COPCs (three inorganics), which have toxicity information. RGA groundwater contains eight COPCs (seven organics and one radionuclide). The radionuclide in this media and all of the seven organics have toxicity information. McNairy groundwater contains two COPCs (two organics), both of which have toxicity information.

1.4.6.5 SWMU 193c COPC toxicity summary

SWMU 193c surface soil contains three COPCs (three inorganics), and there is toxicity information on all three. The subsurface soil contains 11 COPCs (10 inorganics and one organic), with toxicity information on all 11. RGA groundwater contains two COPCs (two organics), both of which have toxicity information. McNairy groundwater contains 37 COPCs (21 inorganics, 15 organics, and one radionuclide). Eighteen of the 21 inorganice have toxicity information, while 14 of the organics do. There is toxicity information on the radionuclide also.

1.4.6.6 SWMU 194 COPC toxicity summary

SWMU 194 subsurface soil contains seven COPCs (six inorganics and one organic), and all seven have toxicity information. Surface soil and groundwater were not assessed.

1.4.6.7 AOC 204 COPC toxicity summary

AOC 204 subsurface soil contains six COPCs (six organics), and all six have toxicity information. RGA groundwater contains nine COPCs (nine organics), and they all have toxicity information.⁴

1.5 RISK CHARACTERIZATION

Risk characterization is the final step in the risk assessment process. In this step, the information from the exposure and toxicity assessments is integrated to quantitatively estimate both carcinogenic health risks and noncarcinogenic hazard potential. For this assessment, risk is defined as the lifetime probability of excess cancer incidence for carcinogens and the estimate of exposure levels that may lead to toxic effects for noncarcinogens.

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1.5.1 Determination of Potential for Noncancer Effects

In this risk assessment, the numeric estimate of the potential for noncancer effects posed by a single chemical within one pathway of exposure is derived as the ratio of the CDI of a chemical from a single pathway to the appropriate RfD. This ratio is also referred to as a hazard quotient (HQ). This value is calculated as shown in the following equation:

$$HQ = \frac{CDI}{RfD}$$

where:

HQ = the hazard quotient, dimensionless

CDI = the CDI of a particular chemical, mg/kg-day

RfD = the chronic RfD for a particular chemical and pathway, mg/kg-day

(Note: Use of RfCs is similar for the inhalation pathway.)

Care was taken when performing this calculation to ensure that the proper RfD was used for each CDI. For CDIs that reflect ingestion, the RfD used was that for administered dose. For CDIs that reflect absorption, as in dermal contact, the RfD used was that for absorbed dose. Finally, for CDIs that reflect inhalation exposure, the RfD used was that for inhalation. Similarly, the RfD appropriate for the duration of exposure was used. For all adult exposures, the period of exposure was greater than seven years; therefore, the chronic RfD was used. For all exposures to children, regardless of duration, the chronic RfD was used (RAGS, Methods Document). Generally, only chronic RfDs were used for adults because this assessment only considered lifetime exposures.

If several chemicals may reach a receptor through a common pathway, guidance (RAGS, Methods Document) recommends adding the HQs of all chemicals reaching the receptor through the common pathway to calculate a pathway HI. This is represented by the following equation:

Pathway HI =
$$HQ_1 + HQ_2 + HQ_3 + ... + HQ_n$$

where:

Pathway HI = the sum of the individual chemical HQs, dimensionless HQ_1 to HQ_n = the individual chemical HQs relevant to the pathway, dimensionless

Similarly, guidance (RAGS, Methods Document) recommends summing the pathway HIs for all pathways relevant to an individual receptor to develop a total HI. The total HI is not an estimate of the systemic toxicity posed by all contaminants that may reach the receptor, but it can be used to estimate whether a toxic effect may result if all contaminants reaching the receptor have additive effects over all pathways. This is represented in the following equation:

where:

Total HI = the sum of all pathways relevant to a single receptor, dimensionless HI_1 to HI_n = the individual pathway HIs

The HQ, the pathway HI, and the total HI do not define a dose-response relationship. That is, the magnitude of the HQ or HI does not represent a statistical probability of incurring an adverse effect. If the HQ is less than 1, the estimated exposure to a substance may be judged to be below a level that could present a toxic effect. If the HQ is greater than 1, a toxic effect may or may not result depending on the assumptions used to develop the CDI and assumptions used in deriving the RfD. Similarly, if the pathway HI is less than 1, the estimated exposure to multiple chemicals contributing to the pathway HI should not be expected to present a toxic effect. If the pathway HI is greater than 1, exposure may or may not result in a toxic effect depending on what assumptions were used to develop the pathway and how the chemicals included in the pathway interact. Finally, if the total HI is less than 1, the estimated exposure to multiple chemicals to result in a toxic effect. If the total HI is greater than 1, a toxic effect. If the total HI is less than 1, the estimated exposure to multiple should not be expected to result in a toxic effect. If the total HI is less than 1, the estimated exposure to multiple chemicals than 1, the estimated exposure to multiple chemicals over multiple pathways should not be expected to result in a toxic effect. If the total HI is greater than 1, a toxic effect may or may not result depending on the rigor used to develop the conceptual site model for all pathways and the interaction between pathways and individual chemicals.

After summing within and across pathways, the risk was further evaluated if the sum was greater than 1. In this evaluation, chemicals with similar effects were segregated to determine whether the HQs of these chemicals also summed to a value greater than 1. This evaluation was performed because if the sum of the HQs of chemicals with common effects is greater than 1, there is greater confidence in stating that exposure to several chemicals within a pathway or across several pathways may lead to a toxic effect. This and other uncertainties related to this method of determining the potential for systemic toxicity are discussed in more detail in Sect. 1.6.

1.5.2 Determination of ELCR

Estimates of the potential for cancer induction are measured by calculating estimates of ELCR. Generally, ELCR is defined as the incremental increase in the probability that a receptor may develop cancer if the receptor is exposed to chemicals or radionuclides or both. ELCRs developed using the following procedures are specific for the conceptual site model used to define the routes and magnitude of exposure. The magnitude of the ELCRs may vary markedly if the exposure assumptions used to develop the conceptual site model are varied.

1.5.2.1 Chemical excess lifetime cancer risk

The numeric estimate of the ELCR resulting from exposure to a single chemical carcinogen is derived by multiplying the CDI for a particular pathway by the slope factor appropriate to that pathway. The resulting value is referred to as a chemical-specific ELCR. This value is calculated as shown in the following equation:

$Chemical - specific ELCR = CDI \times SF$

where:

Chemical-specific ELCR = an estimate of the excess lifetime probability of developing cancer, dimensionless

CDI = the CDI of the chemical mg/kg-day

SF = the slope factor for the specific chemical (mg/kg-day)⁻¹

(Note: Use of unit risk toxicity values is similar for the inhalation pathway.)

As with the calculation used to derive HQs, care was taken when performing this calculation to ensure that the proper slope factor was used for each CDI. For CDIs that reflect ingestion, the slope factor was that for an administered dose. For CDIs that reflect absorption, the slope factor was that for absorbed ---- dose. Finally, for CDIs that reflect inhalation exposure, the slope factor was that for inhalation.

If several chemicals may reach a receptor through a common pathway, guidance (RAGS, Methods Document) recommends adding the chemical-specific ELCRs of all chemicals reaching the receptor through the common pathway to calculate a pathway ELCR. This is represented by the following equation:

Pathway ELCR = $ELCR_1 + ELCR_2 + ELCR_3 + ... + ELCR_n$

where:

Pathway ELCR = the sum of the chemical-specific ELCRs, dimensionless $ELCR_1$ to $ELCR_n$ = the chemical-specific ELCRs relevant to the pathway, dimensionless

Similarly, guidance (RAGS, Methods Document) recommends combining the pathway ELCRs for all pathways relevant to an individual receptor to develop a total ELCR. The total ELCR is not an actuarial estimate of an individual developing cancer, but it can be used to estimate the total ELCR that may result if all contaminants reaching the receptor have additive effects over all pathways. This is represented in the following equation:

where:

Total ELCR = the sum of all pathways relevant to a single receptor, dimensionless $ELCR_{P1}$ to $ELCR_{P2}$ = the individual pathway ELCRs

The chemical-specific ELCR, the pathway ELCR, and total ELCR define a dose-response relationship, unlike the HQ, the pathway HI, and the total HI. That is, the ELCRs represent a statistical probability of the increased risk that cancer will develop in receptors exposed under the assumptions used in the calculation of the CDI; however, like pathway HI and total HI, additional evaluation of the risk characterization should be performed if the total ELCR exceeds 1E-4. If the total ELCR exceeds 1E-4, chemicals contributing to the ELCR should be segregated by common effect. This analysis is performed to decrease the uncertainty in the risk presentation and increase the confidence of any subsequent risk management decision. This and other uncertainties related to this method of calculating ELCR are discussed in more detail in Sect. 1.6.

1.5.2.2 Radionuclide excess lifetime cancer risk

Calculation of cancer risk due to radionuclide exposure through ingestion or inhalation is conceptually similar to calculation of risks for chemical carcinogens. In performing this calculation, ELCR is calculated by multiplying the intake of the radionuclide by the route-specific cancer slope factor. This is represented by the following equation:

Radionuclide - specific $ELCR = CDI \times SF$

where:

Radionuclide-specific ELCR = an estimate of the excess lifetime probability of developing cancer, dimensionless

CDI = the ingestion and inhalation CDI of the radionuclide, pCi

SF = the ingestion and inhalation slope factor for the specific radionuclide, risk/pCi

(Note: For external exposure, the units for CDI and SF are pCi-year/g and risk-g/pCi-year, respectively.)

As with the calculation used to derive chemical-specific ELCRs, care was taken when performing this calculation to ensure that the proper slope factor was used for each CDI. For CDIs that reflect ingestion, the slope factor was that for ingestion. Similarly, for CDIs that reflect inhalation exposure, the slope factor was that for inhalation.

Both the pathway ELCR for radionuclides and the total ELCR from exposure to multiple radionuclides within a pathway and across multiple pathways, respectively, are calculated as illustrated for chemical carcinogens in Sect. 1.5.2. These equations will not be presented in this risk assessment. The uncertainties related to this method of determining ELCR from exposure to radionuclides are discussed in detail in Sect. 1.6.

In this risk assessment, ELCRs due to exposure to chemicals and radionuclides were summed within pathways and across all pathways to indicate the potential health risk to a receptor that may be exposed to radionuclides and chemicals over all pathways. The uncertainties associated with combining radionuclide and chemical ELCRs are discussed in detail in Sect. 1.6.

1.5.3 Risk Characterization for Current Land Use Scenarios at Current Concentrations

This section presents the risk associated with current land use (i.e., industrial) at WAG 28 sites. Exhibits and discussion in this section provide the total HI or ELCR for each site and list the major exposure routes and constituents contributing to the total HI or ELCR. Land use scenarios of concern, pathways of concern, and chemicals of concern (COCs) are discussed in Sects. 1.5.7.1, 1.5.7.2, and 1.5.7.3, respectively.

The information summarized in the exhibits and discussion in this section are presented in full in Tables 1.62 and 1.63. Table 1.62 presents the systemic toxicity for each site for the current industrial worker. Table 1.63 presents the ELCR for each site for the current industrial worker. In each table, the risk for each contaminant within each pathway, the risk for each contaminant across all pathways, the risk for each pathway, and the total risk across all pathways are presented. The program used to calculate the risk values is SAS[®] Program 10 described in Appendix C of this volume.

1.5.3.1 Systemic toxicity

Exhibit 1.17 summarizes the HIs for exposure routes from soil for the current industrial worker for each site with and without lead included as a COPC. The total scenario HI (i.e., "Site total" in Exhibit 1.17) is less than 1 for SWMUs 99a (0.526) and 193a (0.432), between 1 and 10 for SWMU 193b (5.25), and greater than 1000 for SWMU 193c (3620). The scenario total HIs for some sites at WAG 28 are large because of the presence of lead at concentrations greater than background. If, when present, hazards from lead are excluded from consideration, the total location HI becomes less than 1 for SMWU 193c (0.194). For each location, with or without lead, the driving exposure route is dermal contact with soil. For each location, the inhalation exposure route contributes insignificantly to the location total HI.

Exhibit 1.18 lists the contaminants contributing more than 1 percent of the total systemic toxicity for the current industrial worker exposed to soil at each site where the total systemic toxicity exceeds 1 without lead included as a COPC. Including lead as a COPC in this table would mask the contribution from the other COPCs. Lead contributes more than 99 percent of the systemic toxicity for SWMU 193c when included. HIs at SWMUs 99a, 193a, and 193c are less than 1. For SWMU 193b, beryllium, chromium, and vanadium are the primary contaminants.

	E			
Scenario and site	Incidental ingestion	Dermal contact	Inhalation of vapors/particles	Site total ^b
Current industrial worker				
SWMU 99a (soil)	< 0.1 (< 0.1)	0.517 (0.517)	< 0.1 (< 0.1)	0.526 (0.526)
% of total	1% (1%)	98% (98%)	< 1% (< 1%)	
SWMU 193a (soil)	< 0.1 (< 0.1)	0.43 (0.43)	< 0.1 (< 0.1)	0.432 (0.432)
% of total	< 1% (< 1%)	> 99% (> 99%)	< 1% (< 1%)	
SWMU 193b (soil)	< 0.1 (< 0.1%)	5.23 (5.23)	< 0.1% (< 0.1%)	5.25 (5.25)
% of total	< 1% (< 1%)	> 99% (> 99%)	< 1% (< 1%)	
SWMU 193c (soil)	122 (< 0.1)	3500 (0.193)	< 0.1 (< 0.1)	3620 (0.194)
% of total	3% (< 1%)	> 99% (> 99%)	< 1% (< 1%)	

Exhibit 1.17. Exposure route summary for the current use scenario-systemic toxicity^a

^a The value for the individual exposure routes and the site total, in parenthesis, is without lead as a COPC.

^b Current convention is to use one significant digit for presentation of hazard indices. Three significant digits are used here when the HI is greater than 0.1 to enable the reader to match the numbers reported in the exhibit with those in its associated risk characterization table. Additionally, use of three significant digits, when the exposure route value is greater than 0.1, allows the reader to sum the route values and check the location total. The scenario totals without lead are presented in Tables 1.75–1.81.

Exhibit 1.18. Driving contaminants summary for current use scenario—systemic toxicity
without lead included as a COPC

Scenario and site Driving contaminants over all exposure routes		Site total	
Current industrial worker			
SWMU 99a (soil)	HI < 1	0.526	
SWMU 193a (soil)	HI < 1	0.432	
SWMU 193b (soil)	Beryllium (3%), chromium (60%), vanadium (37%)	5.25	
SWMU 193c (soil)	HI < 1	0.194	

Notes: HI < 1 indicates that total scenario hazard index is less than 1; therefore, analytes are not listed. Lead contributes more than 99% of the systemic toxicity for each site when included.

1.5.3.2 Excess lifetime cancer risk

Exhibit 1.19 summarizes the excess cancer risks for exposure routes for the current industrial worker exposed to soil at each site. The total ELCR for SWMU 193c is less than 1E-6. The total ELCRs for SWMUs 99a (3.1E-4), 193a (1.5E-5), and 193b (5.1E-4) are greater than 1E-6. For SWMUs 99a, 193a, and 193b, the exposure route contributing most to ELCR is dermal contact with soil.

Exhibit 1.20 lists the contaminants contributing more than 1 percent of the total ELCR for the current industrial worker exposed to soil at each site. The driving contaminants at SWMU 99a are beryllium, PAHs, and radionuclides, PAHs at SWMU 193a, and beryllium at SWMU 193b.

1.5.4 Risk Characterization for Potential Future Land Use Scenarios at Current Concentrations

This section presents hazards and risks for future land uses (i.e., industrial, rural, recreational, residential, and excavation) for WAG 28 sites. The exhibits at the end of this section provide the total HI or ELCR for each site and list the exposure routes and COPCs contributing most to total HI or ELCR.



	Exposure routes for soil				
Scenario and site	Incidentai ingestion	Dermal contact	Inhalation of vapors/particles	External exposure	Site total
Current industrial worker					
SWMU 99a (soil)	4.8E-6	2.5E-4	5.8E-8	5.3E-5	3.1E-4
% of total	2%	81%	< 1%	17%	
SWMU 193a (soil)	5.4E-7	1.5E-5	1.4E-9	NV	1.5E-5
% of total	4%	97%	< 1%	NV	
SWMU 193b (soil)	1.2E-6	5.1E-4	2.7E-9	NV	5.1E-4
% of total	< 1%	100%	< 1%	NV	
SWMU 193c (soil)	NV	NV	1.7E-10	NV	1.7E-10
% of total	. NV	NV	100%	NV	

Exhibit 1.19. Exposure route summary for the current use scenario—excess lifetime cancer risk

Note: NV indicates that a value is not available.

Exhibit 1.20. Driving contaminants summary for current use scenario—excess lifetime cancer risk

Scenario and site	Driving contaminants over all exposure routes	Location total	
Current industrial worker	• • •		
SWMU 99a (soil)	Beryllium (70%), PAHs (12%), cesium-137 (3%), neptunium-237 (9%), uranium-238 (5%)	3.1E-4	
SWMU 193a (soil)	PAHs (99.9%)	1.5E-5	
SWMU 193b (soil)	Beryllium (100%)	5.1E-04	
SWMU 193c (soil)	ELCR < 1E-6	1.7E-10	

Note: ECLR < 1E-6 indicates that total scenario ELCR is less than 1E-6; therefore, analytes are not listed.

Complete presentations of the information summarized in this section are given in Tables 1.64–1.74. Table 1.64 presents the systemic toxicity for the future industrial worker at current concentrations. Tables 1.65 and 1.66 present the risk summaries for systemic toxicity for the future adult and child rural residents at current concentrations, respectively. Tables 1.67, 1.68, and 1.69 present the risk summaries for systemic toxicity for the adult, teen, and child recreational users at current concentrations, respectively. Table 1.70 presents the risk summaries for systemic toxicity for the future excavation worker at current concentrations. Table 1.71 presents the ELCR for the future industrial worker at current concentrations. Table 1.72 presents the risk summaries for ELCR for the future rural resident at current concentrations. Table 1.73 presents the risk summaries for ELCR for the future rural resident at current concentrations. Table 1.74 presents the risk summaries for ELCR for the future rural resident at current concentrations. Table 1.74 presents the risk summaries for ELCR for the future excavation worker at current concentrations. In each table, the risk for each contaminant within each pathway, the risk for each contaminant across all pathways, the risk from each pathway, and the total risk across all pathways are presented. The program used to calculate the risk values in these tables is SAS[®] Program 10 described in Appendix C of this volume.

1.5.4.1 Systemic toxicity

Future Industrial Worker at Current Concentrations. Exhibit 1.21 summarizes the HIs for exposure routes for the future industrial worker for each site with and without lead included as a COPC. For sites where lead was not detected, the total HI for exposure to McNairy groundwater at SWMU 193b is less than 1, whereas the HIs for the six sites are less than 10 [SWMUs 99a McNairy (1.64), 99b RGA (7.00), 193a RGA (1.64), 193a McNairy (4.69), 193b RGA (1.74), and 193c RGA (1.46)]. The HI for AOC 204 is greater than 10 (33.30). For sites at which lead was detected, total HIs for groundwater exceed 1000 for SWMUs 99a RGA (8150) and 193c McNairy (25,100) with lead included as a COPC; however, excluding lead at these sites reduces the HIs to less than 10 for both sites [99a RGA (5.11) and 193c McNairy (9.92)]. The driving exposure route for both water sources for all sites except AOC 204 is ingestion of groundwater. The driving exposure route at AOC 204 is dermal contact with groundwater.

The results for exposure to soil presented in this exhibit are the same as those for the current industrial worker (Exhibit 1.17). The total scenario HI is less than 1 for SWMUs 99a (0.526) and 193a (0.432), between 1 and 10 for SWMU 193b (5.25), and greater than 1000 for SWMU 193c (3620); however, as before, the scenario total HIs for some sites at WAG 28 are large because of the presence of lead at concentrations greater than background. Thus, if hazard from lead is not considered, the total location HI is less than 1 for SMWU 193c (0.194). For each location, the driving exposure route is dermal contact with soil. Also, for each location, the inhalation exposure route contributes insignificantly to the location total HI.

Exhibit 1.22 lists the contaminants contributing more than 1 percent of the total systemic toxicity for the future industrial worker for each site where the total systemic toxicity exceeds 1, excluding lead as a COPC. Including lead as a COPC in this exhibit would mask the contribution from the other COPCs. Lead contributes more than 99 percent of the systemic toxicity for SWMUs 99a RGA and 193c McNairy, when included as a COPC. Various metals and trichloroethene (or its degradation products) are the driving contaminants in groundwater.

The results for exposure to soil presented in this exhibit are the same as those for the current industrial worker (see Exhibit 1.18.). Exhibit 1.22 summarizes the contaminants contributing more than 1 percent of the total systemic toxicity for the future industrial worker for each site where the total systemic toxicity exceeds 1 without lead included as a COPC. Including lead as a COPC in this table would mask the contribution from the other COPCs. Lead contributes more than 99 percent of the systemic toxicity for SWMU 193c when included. HIs at SWMUs 99a, 193a, and 193c are less than 1. For SWMU 193b, beryllium, chromium, and vanadium are the primary contaminants.

Future On-site Rural Resident at Current Concentrations. Exhibit 1.23 summarizes the HIs for exposure routes for the future child on-site rural resident for each site with and without lead included as a COPC. Although results for the future adult on-site rural resident were calculated and are presented in Table 1.65, these results are not summarized here because the child is the most sensitive receptor for systemic toxicity for this scenario.

The total HIs for all sites are greater than 1, irrespective of lead. The HI for SWMU 193b McNairy (2.69) is less than 10. HIs are above 10 but less than 300 at several other sites [SWMUs 99a McNairy (53.1), 99b RGA (208), 193a RGA (28.6), 193a McNairy (59.9), 193b RGA (55.5), 193c RGA (80.7), and AOC 204 (279)]. Total HIs for exposure to RGA or McNairy groundwater with lead included as a COPC are greater than 1000 for SWMUs 99a RGA (90,600) and 193c McNairy (278,000); however, exclusion of lead as a COPC reduces these HIs to 97.3 and 103, respectively. The driving exposure route for SWMUs 99a McNairy, 99b RGA, 193a RGA, 193b RGA, 193b McNairy, 193c RGA, and 193c



	Exposure routes				
Scenario and site	Ingestion	Dermai contact	Inhalation of vapors	total	
Future Industriai worker					
Exposure routes for groundwater					
SWMU 99a RGA	7960 (3.72)	193 (0.77)	0.616 (0.616)	8150 (5.11)	
% of total	98% (73%)	2% (15%)	< 1% (12%)	~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~~	
SWMU 99a McNairy	0.874 (0.874)	0.284 (0.284)	0.482 (0.482)	1.64 (1.64)	
% of total	53% (53%)	17% (17%)	29% (29%)	. ,	
SWMU 99b RGA	3.79 (3.79)	1.36 (1.36)	1.85 (1.85)	7.00 (7.00)	
% of total	54% (54%)	19% (19%)	26% (26%)		
SWMU 193a RGA	1.35 (1.35)	0.14 (0.14)	0.152 (0.152)	1.64 (1.64)	
% of total	82% (82%)	9% (9%)	9% (9%)	. ,	
SWMU 193a McNairy	4.48 (4.48)	0.113 (0.113)	<0.1 (<0.1)	4.69 (4.69)	
% of total	96% (96%)	2% (2%)	2% (2%)		
SWMU 193b RGA	0.911 (0.911)	0.328 (0.328)	0.504 (0.504)	1.74 (1.74)	
% of total	52% (52%)	19% (19%)	29% (29%)		
SWMU 193b McNairy	< 0.1 (< 0.1)	< 0.1 (< 0.1)	< 0.1 (< 0.1)	< 0.1 (< 0.1)	
% of total	57% (57%)	12% (12%)	31% (31%)		
SWMU 193c RGA	0.875 (0.875)	0.105 (0.105)	0.478 (0.478)	1.46 (1.46)	
% of total	60% (60%)	7% (7%)	33% (33%)	· · ·	
SWMU 193c McNairy	24,500 (8.48)	593 (1.39)	< 0.1 (,0.1)	25,100 (9.92)	
% of total	98% (86%)	2% (14%)	< 1% (< 1%)		
AOC 204 RGA	14.3 (14.3)	18.3 (18.3)	0.725 (0.725)	33.3 (33.3)	
% of total	43% (43%)	55% (55%)	2% (2%)	-	
Exposure routes for soll ^d					
SWMU 99a (soil)	< 0.1 (< 0.1)	0.517 (0.517)	< 0.1 (< 0.1)	0.526 (0.526)	
% of total	1% (1%)	98% (98%)	< 1% (< 1%)	, -	
SWMU 193a (soil)	< 0.1 (< 0.1)	0.43 (0.43)	< 0.1 (< 0.1)	0.432 (0.432)	
% of total	< 1% (< 1%)	> 99% (> 99%)	< 1% (< 1%)		
SWMU 193b (soil)	< 0.1 (< 0.1)	5.23 (5.23)	< 0.1 (< 0.1)	5.25 (5.25)	
% of total	< 1% (1%)	> 99% (> 99%)	< 1% (< 1%)		
SWMU 193c (soil)	122 (< 0.1)	3500 (0.193)	< 0.1 (< 0.1)	3620 (0.194)	
% of total	3% (< 1%)	97% (> 99%)	< 1% (< 1)		

Exhibit 1.21.	Exposure route summary for future use scenario-systemic
	toxicity for the future industrial worker ^{a,b,c}

* The values for the individual exposure routes and the site total, in parenthesis, are without lead as a COPC.

^b Current convention is to use one significant digit for presentation of hazard indices. Three significant digits are used here to enable the reader to match the numbers reported in the exhibit with those in its associated risk characterization table. Additionally, use of three significant digits, when the exposure route value is greater than 0.1, allows the reader to sum the route values and check the location total.

^c Risks from use of water drawn from the RGA were calculated separately from those for water drawn from the McNairy Formation.

^d Risks from exposure to soil are identical to risk for the current industrial worker.

Scenario and site	Driving contaminants over all exposure routes	Site tota
Future industrial worker		
Exposure routes for ground	water	
SWMU 99a RGA	Aluminum (2%), arsenic (4%), chromium (10%), iron (15%), manganese (8%), vanadium (14%), trichloroethene (42%)	5.11
SWMU 99a McNairy	Carbon tetrachloride (4%), trichloroethene (84%), cis-1,2-dichloroethene (11%)	1.64
SWMU 99b RGA	Chromium (3%), trichloroethene (94%)	. 7.0
SWMU 193a RGA	Fluoride (4%), iron (62%), trichloroethene (33%)	1.64
SWMU 193a McNairy	Iron (94%), cis-1,2-dichloroethene (6%)	4.69
SWMU 193b RGA	Carbon tetrachloride (8%), trichloroethene (90%)	1.74
SWMU 193b McNairy	HI < 1	0.076
SWMU 193c McNairy	Aluminum (4%), antimony (33%), arsenic (4%), cadmium (10%), chromium (6%), iron (20%), manganese (3%), vanadium (16%)	9.92
SWMU 193c RGA	1,2-Dichloroethene (65%), trichloroethene (35%)	1.46
AOC 204 RGA	1,1-Dichloroethane (2%), PCB-1254 (88%), tetrachloroethene (4%), trichloroethene (5%)	33.3
Exposure routes for soil ^a	÷	
SWMU 99a (soil)	HI < 1	0.526
SWMU 193a (soil)	HI < 1	0.432
SWMU 193b (soil)	Beryllium (3%), chromium (60%), vanadium (37%)	5.25
SWMU 193c (soil)	HI < 1	0.194

Exhibit 1.22. Driving contaminants summary for future use scenario—systemic toxicity for the future industrial worker without lead as a COPC

÷.

Notes: HI < I indicates that total scenario hazard index is less than 1; therefore, analytes are not listed. Lead contributes more than 99% of the systemic toxicity for each site when included.

^a Risks from exposure to soil are identical to risk for the current industrial worker.

Scenario and site		Dermal	Inhalation	Ingestion of	- Site
	Ingestion	contact	vapors ^d	vegetables	total
Future child on-site rural r	resident				
Exposure routes for ground	dwater				
SWMU 99a RGA	53,800 (25.2)	518 (2.06)	49.36 (49.36)	36,300 (20.7)	90,600 (97.3)
% of total	59% (26%)	< 1%(2)	< 1% (51%)	40% (21%)	
SWMU 99a McNairy	5.9 (5.9)	0.762 (0.762)	38,66 (38.66)	7.81 (7.81)	53.1 (53.1)
% of total	11% (11%)	1% (1%)	73% (73%)	15% (15%)	
SWMU 99b RGA	25.6 (25.6)	3.64 (3.64)	148.5 (148.5)	30.4 (30.4)	208 (208)
% of total	12% (12%)	2% (2%)	71% (71%)	15% (15%)	
SWMU 193a RGA	9.09 (9.09)	0.376 (0.376)	12.23 (12.23)	6.93 (6.93)	28.6 (28.6)
% of total	32% (32%)	1% (1%)	43% <u>(</u> 43%)	24% (24%)	
SWMU 193a McNairy	30.3 (30.3)	0.302 (0.302)	7.546 (7.546)	21.7 (21.7)	59.9 (59.9)
% of total	51% (51%)	< 1% (< 1%)	13% (13%)	36% (36%)	
SWMU 193b RGA	6.16 (6.16)	0.88 (0.88)	40.41(40.41)	8.03 (8.03)	55.5 (55.5)
% of total	11% (11%)	2% (2%)	73% (73%)	15% (15%)	
SWMU 193b McNairy	0.295 (0.295)	< 0.1 (< 0.1)	1.911 (1.911)	0.454 (0.454)	2.69 (2.69)
% of total	11% (11%)	< 1% (< 1%)	71% (71%)	17% (17%)	
SWMU 193c RGA	5.92 (5.92)	0.282 (0.282)	38.33 (38.33)	36.1 (36.1)	80.7 (80.7)
% of total	7% (7%)	< 1% (< 1%)	48% (48%)	45% (45%)	
SWMU 193c McNairy	165,000 (57.3)	1590 (3.73)	3.844 (3.844)	111,000 (37.7)	278,000 (103)
% of total	59% (56%)	< 1% 4%)	< 1% (3%)	40% (37%)	
AOC 204 RGA	96.6 (96.6)	49.0 (49.0)	58.1 (58.1)	75.5 (75.5)	279 (279)
% of total	35% (35%)	18% (18%)	21% (21)	27% (27%)	
Exposure routes for soli					
SWMU 99a (soil)	0.20 (0.20)	3.03 (3.03)	< 0.1 (< 0.1)	14.0 (14.0)	17.2 (17.2)
% of total	1%(1%)	18%(18%)	< 1%(< 1%)	81%(81%)	-
SWMU 193a (soil)	< 0.1 (< 0.1)	2.52 (2.52)	< 0.1 (< 0.1)	3.68 (3.68)	6.25 (6.25)
% of total	< 1% (< 1%)	<u>4</u> 0% (40%)	< 1% (< 1%)	59% (59%)	
SWMU 193b (soil)	0.524 (0.524)	30.7 (30.7)	< 0.1 (< 0.1)	35.5 (35.5)	66.7 (66.7)
% of total	< 1% (< 1%)	46% (46%)	< 1% (< 1%)	53% (53%)	· · ·
SWMU 193c (soil)	3300 (< 0.1)	20,500 (1.13)	< 0.1(< 0.1)	224,000 (1.88)	247,000 (3.04)
% of total	1% (<1%)	8% (37%)	< 1% (< 1%)	90% (62%)	

Exhibit 1.23. Exposure route summary for the future use scenario—systemic toxicity for the future child on-site rural resident^{a,b,e}

^a The values for the individual exposure routes and the site total, in parenthesis, are without lead as a COPC.

^b Current convention is to use one significant digit for presentation of hazard indices. Three significant digits are used here to enable the reader to match the numbers reported in the exhibit with those in its associated risk characterization table. Additionally, use of three significant digits, when the exposure route value is greater than 0.1, allows the reader to sum the route values and check the location total.

^c Risks from use of water drawn from the RGA were calculated separately from those for water drawn from the McNairy Formation.

^d Risks from inhalation while showering are combined with risks from inhalation during household use for groundwater pathways.

McNairy is inhalation of vapors. The driving exposure route for SWMU 193a McNairy is ingestion of --groundwater. The driving exposure route for AOC 204 is ingestion of groundwater, but dermal contact, inhalation of vapors, and ingestion of vegetables contribute significantly to total exposure. The driving exposure route with lead included as a COPC for SWMU 99a is ingestion of groundwater, whereas the driving exposure route excluding lead as a COPC is inhalation of vapors. With the exception of AOC 204, dermal contact accounts for less than 5 percent of total exposure.

For exposure to soil, total HIs for several sites are greater than 1 but less than 100 [99a (17.2), 193a (6.25), and 193b (66.7)]. The HI for SWMU 193c (247,000) is greater than 1000; however, excluding lead as a COPC reduces the HI at SWMU 193c to 3.04. The driving exposure route at all sites is ingestion of vegetables. The exposure route with the second greatest impact is dermal contact. Ingestion of soil and inhalation of vapors and particulates contribute 1 percent or less to total exposure at all sites with or without lead included as a COPC.

Exhibit 1.24 lists the contaminants contributing more than 1 percent of the total systemic toxicity for the future child on-site rural resident for each site where the total systemic toxicity exceeds 1, excluding lead as a COPC. Including lead as a COPC in this exhibit would mask the contribution from the other COPCs. Lead contributes more than 99 percent of the systemic toxicity for SWMUs 99a RGA, SWMU 193c McNairy, and SWMU 193c soil when included. Trichloroethene (or its degradation products) are the primary contaminants in both groundwater formations at most sites; however, metal contaminants are also present in significant amounts in RGA groundwater at SWMUs 99a and 193a and in McNairy groundwater at SWMUs 193a and 193c. PCBs are significant contaminants in RGA groundwater at AOC 204.

Scenario and site	Driving contaminants over ail exposure routes	Site total
Future child on-site rural	resident	 -
Exposure routes for groun	dwater	
SWMU 99a RGA	Arsenic (2%), chromium (5%), iron (9%), manganese (3%), vanadium (6%), trichloroethene (68%)	97.3
SWMU 99a McNairy	Carbon tetrachloride (5%), trichloroethene (80%), cis-1,2-dichloroethene (13%)	53.1
SWMU 99b RGA	trichloroethene (98%)	208
SWMU 193a RGA	Fluoride (2%), iron (40%), trichloroethene (58%)	28.6
SWMU 193a McNairy	Iron (82%), cis-1,2-dichloroethene (17%)	59.9
SWMU 193b RGA	Carbon tetrachloride (9%), trichloroethene (88%)	55.5
SWMU 193b McNairy	Trichloroethene (47%), cis-1,2-dichloroethene (53%)	2.69
SWMU 193c RGA	1,2-Dichloroethene (80%), trichloroethene (20%)	80.7
SWMU 193c McNairy	Aluminum (4%), antimony (32%), arsenic (4%), cadmium (7%), chromium (5%), iron (21%), manganese (2%), vanadium (14%), carbon tetrachloride (2%)	103
AOC 204 RGA	1,1-Dichloroethane (9%), PCB-1254 (66%), tetrachloroethene (5%), trichloroethene (19%)	279
Exposure routes for soil		
SWMU 99a (soil)	Barium (19%), beryllium (4%), chromium (28%), zinc (4%), PCBs (44%)	17.2
SWMU 193a (soil)	Chromium (99%)	6.25
SWMU 193b (soil)	Beryllium (3%), chromium (68%), vanadium (30%)	66.7
SWMU 193c (soil)	Chromium (91%), zinc (9%)	3.04

Exhibit 1.24. Dri	ving contaminants summary for future use scenario—systemic toxicit	у
for the	e future child on-site rural resident without lead as a COPC	

Note: Lead contributes more than 99% of the systemic toxicity for each site, with lead as a COPC, when included.

The results for exposure to soil are also presented in Exhibit 1.24. Lead contributes more than ---99 percent of the systemic toxicity for 193c when included as a COPC. Metals, particularly chromium, are the primary contaminants at all sites. PCBs contribute significantly to contamination at SWMU 99a.

Future Recreational User at Current Concentrations. Exhibit 1.25 summarizes the HIs for exposure routes for the future child recreational user (see Table 1.69) with and without lead included as a COPC. Although results for the future adult and teen recreational user were calculated and are presented in Tables 1.67 and 1.68, these results are not summarized here because the child is the most sensitive receptor for systemic toxicity for this scenario.

Scenario and site	Ingestion of deer	Ingestion of rabbit	Ingestion of quail	Site total
Future child recreational user				
SWMU 99a (soil)	< 0.1 (< 0.1)	< 0.1 (< 0.1)	< 0.1 (< 0.1)	< 0.1 (< 0.1)
% of total	13% (13%)	59% (59%)	28% (28%)	
SWMU 193a (soil)	< 0.1 (< 0.1)	< 0.1 (< 0.1)	< 0.1 (< 0.1)	< 0.1 (< 0.1)
% of total	32% (32%)	68% (68%)	< 1% (< 1%)	
SWMU 193b (soil)	< 0.1 (< 0.1)	< 0.1 (< 0.1)	< 0.1 (< 0.1)	< 0.1 (< 0.1)
% of total	17% (17%)	82% (82%)	< 1% (< 1%)	
SWMU 193c (soil)	5.09 (< 0.1)	2.08 (< 0.1)	< 0.1 (< 0.1)	7.21 (< 0.1)
% of total	71% (72%)	29% (28%)	< 1% (< 1%)	

Exhibit 1.25. Exposure route summary for the future use scenario—systemic toxicity for the future child recreational user^{a,b}

^a The values for the individual exposure routes and the site total, in parenthesis, are without lead as a COPC.

Current convention is to use one significant digit for presentation of hazard indices. Three significant digits are used here to enable the reader to match the numbers reported in the exhibit with those in its associated risk characterization table. Additionally, use of three significant digits, when the exposure route value is greater than 0.1, allows the reader to sum the route values and check the location total.

When lead is included as a COPC, the HI for ingestion of game at SWMU 193c is 7.21. Excluding lead as a COPC reduces the HI to less than 1. The HIs for all remaining sites are less than 1. The driving exposure route at SWMU 193c is ingestion of deer.

Exhibit 1.26 lists the contaminants contributing more than 1 percent of the total systemic toxicity for the future child recreational user for those locations where the total HI exceeds 1. Lead contributes more than 99 percent of the systemic toxicity for SWMU 193c when included as a COPC. No HIs exceed 1 when lead is excluded as a COPC.

Exhibit 1.26. Driving contaminants summary	y for future use scenario—systemic toxicity
for the future child recreational	user without lead as a COPC

Scenario and site	Driving contaminants over all exposure routes	Site total	
Future child recreational user			
SWMU 99a (soil)	HI < 1	0.003	
SWMU 193a (soil)	HI < 1	0.007	
SWMU 193b (soil)	HI < 1	0.004	
SWMU 193c (soil)	HI < 1	0.004	

Notes: HI < 1 indicates that total scenario hazard index is less than 1; therefore, analytes are not listed.

Lead contributes more than 99% of the systemic toxicity for each site, with lead as a COPC, when included.

Future Excavation Worker. Exhibit 1.27 summarizes the total HIs for exposure routes for the ---future excavation worker at each site with and without lead as a COPC. The HIs for SWMUs 99b, 193a, and AOC 204 are less than 1, while the HI for SWMU 193b is 1.75. The HIs are greater than 1000 for three sites with lead included as a COPC [SWMUs 99a (2510), 193c (1890), and 194 (2190)]; however, excluding lead as a COPC reduces the HI to less than 1 for SWMU 194 and to 1.46 and 2.09 for SWMUs 99a and 193c, respectively. For each location, the driving exposure route is dermal contact with soil. The inhalation exposure route contributes less than 1 percent to the total HI with the exception of AOC 204 (10 percent).

	E			
Scenario and site	Incidental	Dermal	Inhalation of	- Site
	ingestion	contact	vapors/particles	total
Future excavation worker				
SWMU 99a (soil)	628 (0.196)	1880 (1.22)	< 0.1 (< 0.1)	2510 (1.46)
% of total	25% (13%)	75% (83%)	< 1% (< 1%)	
SWMU 99b (soil)	<0.1 (< 0.1)	0.491 (0.491)	< 0.1 (< 0.1)	0.569 (0.569)
% of total	14% (14%)	86% (86%)	< 1% (< 1%)	
SWMU 193a (soil)	< 0.1 (< 0.1)	0.435 (0.435)	< 0.1 (< 0.1)	0.471 (0.471)
% of total	8% (8%)	93% (93%)	< 1% (< 1%)	
SWMU 193b (soil)	< 0.1 (< 0.1)	1.69 (1.69)	<0.1 (< 0.1)	1.75 (1.75)
% of total	3% (3%)	97% (97%)	<1% (< 1%)	
SWMU 193c (soil)	473 (0.248)	1410 (1.84)	< 0.1 (< 0.1)	1890 (2.09)
% of total	25% (12%)	75% (88%)	< 1% (< 1%)	
SWMU 194 (soil)	549 (< 0.1)	1640 (0.528)	< 0.1 (< 0.1)	2190 (0.570)
% of total	25% (7%)	75% (93%)	< 1% (< 1%)	
AOC 204 (soil)	< 0.1 (< 0.1)	< 0.1 (< 0.1)	< 0.1 (< 0.1)	< 0.1 (< 0.1)
% of total	37% (37%)	53% (53%)	10% (10%)	

Exhibit 1.27.	Exposure route summary for future use scenario—systemic toxicity
	for the future excavation worker ^{a,b}

^a The values for the individual exposure routes and the site total, in parenthesis, are without lead as a COPC.

^b Current convention is to use one significant digit for presentation of hazard indices. Three significant digits are used here to enable the reader to match the numbers reported in the exhibit with those in its associated risk characterization table. Additionally, use of three significant digits, when the exposure route value is greater than 0.1, allows the reader to sum the route values and check the location total.

Exhibit 1.28 summarizes the contaminants contributing more than 1 percent of the total systemic toxicity for the future excavation worker for each site. Lead contributes more than 99 percent of total exposure at SWMUs 99a, 193c, and 194 when it is included as a COPC. Metals are the primary driving contaminants at all sites where the HI is greater than 1.

Scenario and site	Driving contaminants over all exposure routes	Site total
Future excavation worker	-	
SWMU 99a (soil)	Aluminum (6%), antimony (35%), arsenic (5%), barium (3%), beryllium (2%), cadmium (2%), chromium (16%), manganese (14%), 2-nitroaniline (8%), PCBs (4%)	1.46
SWMU 99b (soil)	HI < 1	0.569
SWMU 193a (soil)	HI < 1	0.471
SWMU 193b (soil)	Beryllium (3%), chromium (59%), vanadium (37%)	1.75
SWMU 193c (soil)	Aluminum (5%), beryllium (3%), cadmium (3%), chromium (28%), iron (31%), manganese (17%), vanadium (14%)	2.09
SWMU 194 (soil)	HI<1	0.57
AOC 204 (soil)	HI < 1	0.0131

Exhibit 1.28 Driving contaminants summary for future use scenario—systemic toxicity for the future excavation worker without lead as a COPC

Notes: HI < 1 indicates that total scenario hazard index is less than 1; therefore, analytes are not listed. Lead contributes more than 99% of the systemic toxicity for SWMUs 99a, 193c, and 194, with lead as a COPC, when included.

1.5.4.2 Excess lifetime cancer risk

Future Industrial Worker at Current Concentrations. Exhibit 1.29 summarizes the total ELCRs for exposure routes for the future industrial worker. The ELCR for exposure to McNairy groundwater at SWMU 193b (8.4E-7) is less than 1E-6. The ELCRs for exposure to either RGA or McNairy groundwater at five sites are between 1E-6 and 1E-4 [SWMUs 99a McNairy (7.6E-5), 193a RGA (2.6E-5), 193a McNairy (1.1E-6), 193b RGA (4.4E-5), and 193c RGA (1.0E-5)]. The total ELCRs for exposure to RGA or McNairy groundwater at four sites are greater than 1E-4 [SWMUs 99a RGA (5.6E-4), 99b (2.6E-4), 193c McNairy (4.2E-4), and AOC 204 RGA (1.3E-3)]. The driving exposure route for both water sources at most sites is ingestion of groundwater; however, inhalation of vapors or dermal contact plays a significant role at some sites. The driving exposure route at SWMU 99b RGA is inhalation of vapors, and at AOC 204 RGA, dermal contact.

The results for exposure to soil presented in this exhibit are the same as those for the current industrial worker (Exhibit 1.19). Sites with ELCRs greater than 1E-6 are SWMUS 99a (3.1E-4), 193a (1.5E-5), and 193b (5.1E-4). For SWMUs 99a, 193a, and 193b, the exposure route contributing most to ELCR is dermal contact with soil. At these sites, inhalation of vapors accounts for less than 1 percent of total exposure. The ELCR is less than 1E-6 for SWMU 193c (1.7E-10).

Exhibit 1.30 summarizes the contaminants contributing more than 1 percent of the total ELCR for the future industrial worker for each site. For both groundwater sources, metals, trichloroethene and its degradation products, and radionuclides are the driving contaminants. The results for exposure to soil presented in this exhibit are the same as those for the current industrial worker (see Exhibit 1.20). The driving contaminants at SWMU 99a are beryllium, PAHs, and radionuclides. The driving contaminants at SWMU 193a are PAHs. The driving contaminant at SWMU 193b is beryllium.

Future On-site Rural Resident at Current Concentrations. Exhibit 1.31 summarizes the ELCRs for the future on-site rural resident. The total ELCRs for use of water drawn from either RGA or McNairy groundwater exceed 1E-4 at all sites except SWMU 193b McNairy (1.2E-5). The dermal exposure route contributes least to ELCRs. Ingestion of vegetables contributes greater than 90 percent at SWMU193a for



	Exposure routes				
Scenario and site	Ingestion	Dermal contact	Inhalation of vapors	total	
Future industrial worker					
Exposure routes for groundwat	er				
SWMU 99a RGA	2.5E-4	6.8E-5	2.4E-4	5.6E-4	
% of total	44%	12%	43%		
SWMU 99a McNairy	4.0E-5	7.3E-6	2.9E-5	7.6E-5	
% of total	52%	10%	38%		
SWMU 99b RGA	8.0E-5	3.1E-5	1.5E-4	2.6E-4	
% of total	31%	12%	57%		
SWMU 193a RGA	1.3E-5	1.1E-5	2.4E-6	2.6E-5	
% of total	48%	42%	9%		
SWMU 193a McNairy	9.8E-7	5.5E-8	4.3E-8	1.1E-6	
% of total	9 1%	5%	4%		
SWMU 193b RGA	2.6E-5	8.1E-6	1.0E-5	4.4E-5	
% of total	59%	18%	23%		
SWMU 193b McNairy	5.0E-7	1.9E-7	1.5E-7	8.4E-7	
% of total	59%	23%	18%		
SWMU 193c RGA	6.2E-6	2.4E-6	1.9E-6	1.0E-5	
% of total	59%	23%	18%		
SWMU 193c McNairy	3.0E-4	6.4E-5	5.9E-5	4.2E-4	
% of total	71%	15%	14%		
AOC 204 RGA	5.3E-4	7.0 E-4	1.0E-4	1.3E-3	
% of total	40%	53%	8%		

Exhibit 1.29. Exposure route summary for future use scenario-excess lifetime cancer risk for the future industrial worker^a

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Scenario and slte	Ingestion	Dermal contact	Inhalation of vapors	External exposure	Site total
Exposure routes for soil ^b					-
SWMU 99a (soil)	4.8E-6	2.5E-4	5.8E-8	5,3E-5	3.1E-4
% of total	2%	81%	< 1%	17%	
SWMU 193a (soil)	5.4E-7	1.5E-5	1.4E-9	NV	1.5E-5
% of total	4%	97%	< 1%	NV	
SWMU 193b (soil)	1.2E-6	5.1E-4	2.7E-9	NV	5.1E-4
% of total	<1%	100%	< 1%	NV	
SWMU 193c (soil)	NV	NV	1.7E-10	NV	1.7E-10
% of total			100%	NV	

Notes: NV indicates that a value is not available.

Risks from use of water drawn from the RGA were calculated separately from those for water drawn from the McNairy Formation.
 Risks from exposure to soil are identical to risks for the current industrial worker.

Scenario and site	Driving contaminants over all exposure routes			
Future industrial worker				
Exposure routes for groundy	vater	•		
SWMU 99a RGA	GA Arsenic (5%), beryllium (38%), 1,1-dichloroethene (14%), trichloroethene (8%), radon-22 (35%)			
SWMU 99a McNairy	1,1-Dichloroethene (61%), carbon tetrachloride (2%), trichloroethene (37%)	7.6E-5		
SWMU 99b RGA	Trichloroethene (53%), radon-222 (48%)	2.6E-4		
SWMU 193a RGA	1,1-Dichloroethene (3%), pentachlorophenol (45%), trichloroethene (42%), bis (2-ethylhexyl) phthalate (3%), technetium-99 (6%)	2.6E-5		
SWMU 193a McNairy	Trichloroethene (22%), technetium-99 (30%), uranium-238 (48%)	1.1E-6		
SWMU 193b RGA	1,1-Dichloroethene (16%), carbon tetrachloride (8%), trichloroethene (74%)	4.4E-5		
SWMU 193b McNairy	ELCR < 1E-6	8.4E-7		
SWMU 193c RGA	Trichloroethene (100%)	1.0E-5		
SWMU 193c McNairy	Arsenic (15%), beryllium (54%), 1,1-dichloroethene (3%), vinyl chloride (16%), radon-222 (11%)	4.2E-4		
AOC 204 RGA	1,1-Dichloroethene (13%), PCBs (63%), tetrachloroethene (21%), trichloroethene (3%)	1.3E-3		
Exposure routes for soil				
SWMU 99a (soil)	PAHs (12%), cesium-137 (3%), neptunium-237 (9%), uranium-238 (5%)	3.1E-4		
SWMU 193a (soil)	PAHs (99.9%)	1.5E-5		
SWMU 193b (soil)	Beryllium (100%)	5.1E-4		
SWMU 193c (soil)	ELCR < 1E-6	1.7E-10		

Exhibit 1.30. Driving contaminants summary for future use scenario—excess lifetime cancer risk for the future industrial worker

Note: ECLR < 1E-6 indicates that total scenario ELCR is less than 1E-6; therefore, analytes are not listed.

		Exposu	ire routes		
Scenario and site	Ingestion	Dermal contact	Inhalation of vapors ^b	Ingestion of vegetables	Site totai
Future on-site rural reside	ent				
Exposure routes for groun	ndwater				
SWMU 99a RGA	1.4E-3	1.7E-4	2.5E-3	1.6E-3	5.6E-3
% of total	24%	3%	45%	28%	
SWMU 99a McNairy	2.2E-4	1.9E-5	1.2E-3	3.0E-4	1.7E-3
% of total	12%	1%	70%	17%	
SWMU 99b RGA	4.3E-4		1.3E-3	4.5E-4	2.3E-3
% of total	19%	4%	58%	20%	
SWMU 193a RGA	6.7E-5	2.9E-5	1.0E-4	2.2E-3	2.4E-3
% of total	3%	1%	4%	92%	
SWMU 193a McNairy	4.2E-6	1.4E-7	1.75E-6	4.1E-4	4.1E-4
% of total	1%	< 1%	< 1%	99%	
SWMU 193b RGA	1.4E-4	2.1E-5	4.15E-4	4.5E-4	1.0E-3
% of total	14%	2%	40%	44%	
SWMU 193b McNairy	2.7E-6	4.9E-7	6.2E-6	2.8E-6	1.2E-5
% of total	22%	4%	50%	23%	
SWMU 193c RGA	3.4E-5	6.1E-6	7.8E-5	3.5E-5	1.5E-4
% of total	22%	4%	50%	23%	
SWMU 193c McNairy	1.6E-3	1.6E-4	6.4E-4	1.6E-3	4.0E-3
% of total	41%	4%	16%	39%	
AOC 204 RGA	2.9E-3	1.8E-3	4.15E-3	6.2E-3	> 1E-2
% of total	19%	12%	28%	41%	

Exhibit 1.31. Exposure route summary for the future use scenario-excess lifetime cancer risk for the future on-site rural resident^a

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Scenario and site	Ingestion	Dermal contact	Inhalation of vapors	Ingestion of vegetables	External exposure	Site total
SWMU 99a (soil)	3.5E-5	7.5E-4	3.6E-7	> 1E-2°	3.6E-4	> 1E-2°
% of total	<1%	< 1%	<1%	99%	< 1%	
SWMU 193a (soil)	5.5E-6	4.4E-5	9.2E-9	6.6E-4	NV	7.1E-4
% of total	< 1%	6%	< 1%	93%	NV	
SWMU 193b (soil)	1.2E-5	1.5E-3	1.8E-8	1.5E-3	NV	3.0E-3
% of total	< 1%	50%	<1%	49%	NV	
SWMU 193c (soil)	NV	NV	1.1E-9	NV	NV	1.1E-9
% of total			100%		NV	

NV indicates that a value is not available. Notes:

* Risks from use of water drawn from the RGA were calculated separately from those for water drawn from the McNairy Formation.

^b Risks from inhalation while showering are combined with risks from inhalation during household use for groundwater pathways. • The ELCR is approximate because the linearized multistage model returns imprecise values at risks > 1E-2.



either groundwater formation. Inhalation of vapors contributes 50 percent or more to risks at SWMUs 99a -----McNairy, 193b McNairy, and 193c RGA. Ingestion of groundwater, inhalation of vapors, and ingestion of vegetables contribute significantly to risks at the remaining sites.

Also shown in this exhibit are ELCRs from exposure to soil. The ELCRs are greater than 1E-4 at all sites except SWMU 193c (1.1E-9). Notably, the ECLR for SWMU 99a is > 1E-2. The driving exposure route at SWMUs 99a and 193a is ingestion of vegetables. The driving exposure routes at 193b are dermal contact and ingestion of vegetables.

Exhibit 1.32 lists the contaminants contributing more than 1 percent of the total ELCR for the future rural resident for each site. For both groundwater sources, metals, organics, and radionuclides contribute to ELCRs. Technetium-99 is the driving contaminant in SWMU 193a RGA and McNairy groundwater. Organics contribute to the majority of risk at the remaining sites, most notably the chlorinated alkenes such as trichloroethene. For soils, PAHs account for nearly 100 percent of the risks at SWMU 193a. Beryllium is the driving contaminant at SWMU 193b. Technetium-99 is the driving contaminant at SWMU 193b.

While an ELCR of > 1E-2 may be considered a potential threat to human health, as it is markedly in excess of EPA's range of concern, this last finding may be biased by two samples that are atypical of SWMU 99a as a whole. During the WAG 28 RI, a collapsed section of road (Tennessee Avenue) located at or beyond the perimeter of SWMU 99a exposed a section of drainpipe that is believed to have drained the area beneath SWMU 99a. A backhoe was used to excavate the top of the pipe, and two samples were collected. While the samples were actually collected several feet below grade, they were classified as surface samples in this risk assessment because the area from which they were collected became available for direct contact, unlike subsurface soil taken from a soil boring. Assessing these samples as surface soil was the most conservative approach. Of these two samples, one drove the risk (082-014), while the other was essentially a nondetect (082-015) (see Table 4.30 in Vol. 1). This limited sampling in and around the exposed drainpipe, which is located at or beyond the perimeter of the site, is associated with a high degree of uncertainty in the ELCR calculated for SWMU 99a. This issue is explored further in Sect. 1.6.1^{*}.6

Future Recreational User at Current Concentrations. Exhibit 1.33 summarizes the ELCRs for exposure routes for the future recreational user. The total ELCR is less than 1E-6 for SWMU 193c (4.4E-8). The total ELCRs exceed 1E-6 but are less than 1E-4 for SWMUs 99a (2.7E-6) and 193a (3.6E-6). The driving exposure route for all sites is ingestion of rabbit.

Exhibit 1.34 presents the contaminants driving ELCR for those locations where the total scenario risk exceeds 1E-6. The contaminants driving the ELCR for ingestion of game at SWMUs 99a and 193a are PAHs.

Future Excavation worker. Exhibit 1.35 summarizes the ELCRs for exposure routes for the future excavation worker. The total scenario ELCRs are greater than 1E-6 for every site and greater than 1E-4 for each site except one [SWMUs 99a (2.1E-4), 99b (2.1E-4), 193a (1.7E-4), 193b (1.7E-4), 193c (1.7E-4), 194 (3.1E-4) and AOC 204 (1.1E-6)].

Exhibit 1.36 lists the contaminants contributing more than 1 percent of the total ELCR for each location. Beryllium is the driving contaminant at SWMUs 99b, 193a, 193b, 193c, and 194. Metals, organics, and radionuclides contribute to the ELCR at SWMU 99a, with beryllium as the primary contaminant. PCBs and organics contribute to the ELCR at AOC 204, with PCBs as the driving contaminants.

Scenario and site	Driving contaminants over all exposure routes	Site tota	
Future on-site rural resident	· · · · · · · · · · · · · · · · · · ·		
Exposure routes for groundwa	ater		
SWMU 99a RGA	Arsenic (4%), beryllium (26%), 1,1-dichloroethene (40%), trichloroethene(11%), radon-222 (9%), technetium-99 (9%)	5.6E-3	
SWMU 99a McNairy	1,1-Dichloroethene (75%), trichloroethene (24%)	1.7E-3	
SWMU 99b RGA	Trichloroethene (87%), radon-22 (13%)	2.3E-3	
SWMU 193a RGA	Pentachlorophenol (2%), trichloroethene (7%), technetium-99 (90%)	2.4E-3	
SWMU 193a McNairy	Technetium-99 (98%)	4.1E-4	
SWMU 193b RGA	1,1-Dichloroethene (20%), carbon tetrachloride (5%), trichloroethene (46%), technetium-99 (29%)	1.0E-3	
SWMU 193b McNairy	Trichloroethene (100%)	1.2E-5	
SWMU 193c RGA	Trichloroethene (100%)	1.5E-4	
SWMU 193c McNairy	Arsenic (14%), beryllium (39%), 1,1-dichloroethene (8%), vinyl chloride (34%), radon-222 (3%)	4.0E-3	
AOC 204 RGA	1,1-Dichloroethene (33%), PCBs (52%), tetrachloroethene (11%), trichloroethene (3%)	> 1E-2 ^a	
Exposure routes for soil	· · · · · · · · · · · · · · · · · · ·		
SWMU 99a (soil)	Technetium-99 (96%)	> 1E-2*	
SWMU 193a (soil)	PAHs (99.9%)	7.1E-4	
SWMU 193b (soil)	Beryllium (100%)	3.0E-3	
SWMU 193c (soil)	ELCR < 1E-6	1.1E-9	

Exhibit 1.32. Driving contaminants summary for future use scenario—excess lifetime cancer risk for the future on-site rural resident

Notes: ECLR < 1E-6 indicates that total scenario ELCR is less than 1E-6; therefore, analytes are not listed.

^a The ELCR is approximate because the linearized multistage model returns imprecise values at risks > 1E-2.

		Exposure routes		- Site total
Scenario and site	Ingestion of deer	Ingestion of rabbit	Ingestion of quail	
Future recreational user				
SWMU 99a (soil)	4.7E-7	1.9E-6	2.9E-7	2.7E-6
% of total	18%	72%	11%	
SWMU 193a (soil)	1.1E-6	2.0E-6	4.2E-7	3.6E-6
% of total	31%	57%	12%	-
SWMU 193b (soil)	8.6E-9	3.5E-8	2.7E-10	4.4E-8
% of total	20%	80%	< 1%	
SWMU 193c (soil)	NV	NV	NV	NV
% of total	NV	NV	NV	

Exhibit 1.33. Exposure route summary for the future use scenario---excess lifetime cancer risk for the future recreational user

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Note: NV indicates that a value is not available.

Scenario and site	Driving contaminants over all exposure routes	Site total					
Future recreational user							
SWMU 99a (soil)	PAHs (71%), PCBs (20%), technetium-99 (3%), uranium-238 (4%)	2.7E-6					
SWMU 193a (soil)	PAHs (100%)	3.6E-6					
SWMU 193b (soil)	ELCR < 1E-6	4.4E-8					
SWMU 193c (soil)	NV	NV					

Exhibit 1.34. Driving contaminants summary for future use scenario—excess lifetime cancer risk for the future recreational user

Notes: NV indicates that a value is not available.

ELCR < 1E-6 indicates that total scenario ELCR is less than 1E-6; therefore, analytes are not listed.

	Exposure routes for soil				
Scenario and site	Incidental ingestion	Dermal contact	Inhalation of vapors/particles	External exposure	Site total
Future excavation worker					
SWMU 99a (soil)	4.6E-5	1.3E-4	4.3E-7	3.3E-5	2.1E-4
% of total	21%	63%	< 1%	16%	
SWMU 99b (soil)	1.1E-5		6.7E-9	NV	2.1E-4
% of total	5%	95%	< 1%	NV	
SWMU 193a (soil)	7.2E-6	1.6E-4	1.0E-9	NV	1.7E-4
% of total	4%	96%	< 1%	NV	•
SWMU 193b (soil)	3.7E-6	1.7E-4	8.7E-10	NV	1.7E-4
% of total	2%	98%	< 1%	NV	
SWMU 193c (soil)	3.6E-6	1.6E-4	4.9E-10	NV	1.7E-4
% of total	2%	98%	<1%	NV	
SWMU 194 (soil)	6.8E-6	3.1 E-4	2.5E-10	NV	3.1E-4
% of total	2%	98%	< 1%	NV	
AOC 204 (soil)	4.6E-7	4.9E-7		NV	1.1E-6
% of total	43%	46%	10%	NV	

Exhibit 1.35. Exposure route summary for future use scenario—excess lifetime cancer risk for the future excavation worker

Note: NV indicates that a value is not available.

Exhibit 1.36. Driving contaminants summary for future use scenario—excess lifetime
cancer risk for the future excavation worker

Scenario and site	Driving contaminants over all exposure routes	Site total	
Future excavation worker			
SWMU 99a (soil)	Arsenic (5%), beryllium (35%), PAHs (21%), N-nitrosodi-n-propylamine (8%), PCBs (2%), cesium-137 (3%), neptunium-237 (11%), uranium-238 (7%)	2.1E-4	
SWMU 99b (soil)	Arsenic (7%), beryllium (93%)	2.1E-4	
SWMU 193a (soil)	Beryllium (91%), PAHs (9%)	1.7E-4	
SWMU 193b (soil)	Beryllium (100%)	1.7E-4	
SWMU 193c (soil)	Beryllium (100%)	1.7E-4	
SWMU 194 (soil)	Beryllium (100%)	3.1E-4	
AOC 204 (soil)	1,1-Dichloroethene (17%), PCBs (57%), tetrachloroethene (17%), trichloroethene (10%)	1.1 E-6	

1.5.5 Risk Characterization for Potential Land Use Scenarios at Modeled Concentrations

This section discusses the potential risks to a resident using RGA groundwater contaminated by migration of chemicals from sources within WAG 28. As discussed in Sect. 1.2.3.1 of this BHHRA, the point of exposure to which contaminants were modeled was the PGDP security fence boundary. Information about the methods used in the model is presented in Chap. 5 of Vol. 1 of this report. Complete modeling results are presented in Appendix B of this volume.

Exhibit 1.37 presents the chemical-specific HIs from exposure to the maximum modeled concentrations of contaminants in the RGA at the point of exposure for household use of water by a rural resident. As shown in Exhibit 1.37, there are five chemicals with chemical-specific HIs that exceed 0.1. These chemicals and their sources are as follows:

- SWMU 99a UCRS soil—lithium, strontium
- SWMU 99a surface soil—lithium, strontium
- SWMU 193a UCRS soil—chromium
- SWMU 193c UCRS soil—lithium, manganese, strontium
- SWMU 193c surface soil—lithium
- SWMU 194 UCRS soil-chromium, lithium, strontium
- AOC 204 UCRS soil-trichloroethene

Exhibit 1.38 presents the chemical-specific ELCRs from exposure to maximum modeled concentrations of contaminants in the RGA at the point of exposure for household use of water by a rural resident. As shown in Exhibit 1.38, there is one organic compound with a chemical-specific ELCR that exceeds 1E-6. This chemical and its source is as follows:

AOC 204 UCRS soil—trichloroethene

As shown in Exhibit 1.10, a single receptor may be exposed to the maximum modeled concentration of more than one chemical in a lifetime. Specifically, the inorganic compounds lithium and strontium have transport times that may allow a single receptor to be exposed to the maximum modeled concentration of each within a lifetime. If such exposure occurred, the total risk to the receptor could be the sum of the risks from the maximum modeled concentrations over all sources for each. The combined HI is 224.

1.5.6 Risk Characterization of Lead

The potential hazards of exposure to lead were determined using KDEP-provided provisional reference dose values for the metal. In addition, risks to exposed children were estimated using EPA's Integrated Exposure Uptake Biokinetic (IEUBK) model, and the RME concentrations of lead in soil and groundwater samples were compared to KDEP and EPA screening values. These additional procedures were pursued to address uncertainties associated with the use of the provisional reference dose provided by KDEP, to meet the requirements of EPA, and to conform to the Methods Document.

As presented in Appendix F of this volume, applying the biokinetic model for lead indicates that the concentration of the element in the McNairy formation at SWMU 193c (250 μ g/L) and in the RGA groundwater at SWMU 99a (81.3 μ g/L) would result in a greater than 5 percent probability of a child having blood lead levels greater than 10 μ g/dL (84 percent probability for SWMU 193c McNairy and 38 percent for SWMU 99a RGA). These findings are consistent with the respective lead-driven HIs of 278,000 and 90,600, as calculated for a future on-site rural resident child exposed to contaminants in these aquifers.

Contaminant ^a	. Source ^b	Maximum concentration ^c	Systemic toxicity ^d	HIe
Inorganic chemicals (mg/L)				
Chromium	SWMU 194 UCRS soil	7.24E+1	4.2E-3	1720
· · · ·	SWMU 193a UCRS soil	3.803E+0	4.2E-3	90.5
	SWMU 193b surface soil	2.02E-3	4.2E-3	HI < 0.1
	SWMU 99a surface soil	2.08E-18	4.2E-3	HI < 0.1
	SWMU 99a UCRS soil	9.40E-20	4.2E-3	HI < 0.1
Lithium	SWMU 194 UCRS soil	6.7E+1	3.0E-2	223
	SWMU 99a UCRS soil	4.686E+1	3.0E-2	156
	SWMU 193c UCRS soil	3.805E+1	3.0E-2	127
	SWMU 99a surface soil	5.632E+0	3.0E-2	18.8
	SWMU 193c surface soil	2.085E+0	3.0E-2	6.95
Manganese	SWMU 193c UCRS soil	5.11E+0	6.7E-2	7.63
Strontium	SWMU 194 UCRS soil	1.05E+1	9.0E-1	1.17
	SWMU 193c UCRS soil	7.453E+0	9.0E-1	0.828
	SWMU 99a UCRS soil	3.782E+0	9.0E-1	0.420
	SWMU 99a surface soil	2.214E+0	9.0E-1	0.246
	SWMU 193c surface soil	2.52E-1	9.0E-1	HI < 0.1
Organic chemicals (mg/L)				
Trichloroethene	AOC 204 UCRS soil	1.428E+1 ^f	1.2E-3	1190
Radionuclides (pCi/L)				
Technetium-99	SWMU 99a surface soil	1.81E+2	NV	NT

Exhibit 1.37. Estimated hazard indices for a rural resident from exposure to maximum modeled concentrations from sources within WAG 28

^a Only contaminants that have a maximum modeled contaminant concentration over all sources that exceed either the cancer or systemic toxicity RBC are listed.

^b Maximum modeled concentration reported for sources within a site. Only sites that contain a source of the contaminant are listed.

^c Maximum modeled contaminant concentration for source.

^d All residential use RBCs were taken from Table 1.5 in Appendix A. All systemic toxicity RBCs are based on chronic exposure of a child age 1-7 years and integrate exposure through ingestion of water, inhalation of vapors emitted by water (showering and household use), and dermal contact with water (showering). Target HI for all systemic toxicity RBCs is 0.1 because more than five contaminants are present. "NV" indicates that an RBC for the endpoint is not available because toxicity information is lacking.

• Value calculated by dividing the maximum contaminant concentration by the RBC and multiplying by the target HI of 0.1. "NT" indicates that the contaminant is not a systemic toxicant or does not have a systemic-toxicity-based RBC because a reference dose for the systemic toxicity endpoint is lacking.

^f The computed maximum concentration is greater than the designated initial concentration at the source (1.42E-7 mg/L). The current receptor is located too close to the source, creating a near-field condition that cannot be properly assessed by a flux boundary condition model; therefore, concentrations have been truncated to the initial dissolved concentration.

Contaminant ^a	Source ^b	Maximum concentration ^c	Cancer ^d	ELCR
Organic chemicals (mg/L)				
Chromium	SWMU 194 UCRS soil	7.24E+1	NV	NC
	SWMU 193a UCRS soil	3.803E+0	NV	NC
· · ·	SWMU 193b surface soil	2.02E-3	NV	NC
	SWMU 99a surface soil	2.08E-18	NV	NC
	SWMU 99a UCRS soil	9.40E-20	NV	NC
Lithium	SWMU 194 UCRS soil	6.7E+1	NV	NC
	SWMU 99a UCRS soil	4.686E+1	NV	NC
· · · ·	SWMU 193c UCRS soil	3.805E+1	NV	NC
	SWMU 99a surface soil	5.632E+0	NV	NC
	SWMU 193c surface soil	2.085E+0	NV	NC
Manganese	SWMU 193c UCRS soil	5.11E+0	NV	NC
Strontium	SWMU 194 UCRS soil	1.05E+1	NV	NC
	SWMU 193c UCRS soil	7.453E+0	NV	NC
	SWMU 99a UCRS soil	3.782E+0	NV	NC
	SWMU 99a surface soil	2.214E+0	NV	NC
	SWMU 193c surface soil	2.52E-1	NV	NC
Organic chemicals (mg/L)				
Trichloroethene	AOC 204 UCRS soil	1.428E+1 ^f	1.4E-4	1.02E-2
Radionuclides (pCi/L)				
Technetium-99	SWMU 99 surface soil	1.81E+2	2.8E+1	6.46E-7

Exhibit 1.38. Estimated excess lifetime cancer risks for a rural resident from exposure to maximum modeled concentrations from sources within WAG 28

^a Only contaminants that have a maximum modeled contaminant concentration over all sources that exceed either the cancer or systemic toxicity RBC are listed.

^b Maximum modeled concentration reported for sources within a site. Only sites that contain a source of the contaminant are listed.

^c Maximum modeled contaminant concentration for source.

^d All residential use RBCs were taken from Table 1.5 in Appendix A. Cancer RBCs are based on a 40-year exposure. The cancer RBCs integrate exposure through ingestion of water, inhalation of vapors emitted by water (showering and household use), and dermal contact with water (showering). Target risk for cancer RBCs is 1E-7 because more than five contaminants are present. "NV" that indicates an RBC for the endpoint is not available because toxicity information is lacking.

Value calculated by dividing the maximum contaminant concentration by the RBC and multiplying by the target risk of 1 E-7. "NC" indicates that the contaminant is not a carcinogen or does not have a cancer-based RBC because a cancer endpoint toxicity value is lacking.

¹ The computed maximum concentration is greater than the designated initial concentration at the source (1.42E-7 mg/L). The current receptor is located too close to the source, creating a near-field condition that cannot be properly assessed by a flux boundary condition model; therefore, concentrations have been truncated to the initial dissolved concentration.



The RME lead concentrations in SWMU 193c McNairy and SWMU 99a RGA are also greater than the KDEP and EPA screening level concentrations for this element (4 and 15 μ g/L, respectively); therefore, when these findings are considered together, there is qualitative agreement on the potential hazards of prevailing lead concentrations in the groundwater at these sites.

Where the element was detected in surface or subsurface soil, lead-driven HIs of greater than 1000 contrast with very low probabilities (< 0.02 percent) of children having blood lead levels greater than $10\mu g/dL$, as determined by the IEUBK model. Furthermore, lead concentrations in subsurface soil at SWMUs 99a, 193c, and 194 do not exceed the soil screening values specified by either agency; however, as illustrated in Exhibit 1.39, the concentration of lead in surface soil at SWMU 193c exceeds the KDEP benchmark but not that of EPA (20 < 24.9 < 400 mg/kg).

1.5.7 Identification of Land Use Scenarios, Pathways, Media, and Chemicals of Concern

This section identifies the land use scenarios of concern, pathways of concern (POCs), media of concern (MOC), and COCs for sites in WAG 28. This section evaluates all land use scenarios to identify those land use scenarios, contaminants, and pathways to consider when choosing appropriate remedial actions. Sect. 1.8 presents RGOs for each location and land use combination using the information compiled here.

To determine land use scenarios of concern, risk characterization results for total systemic toxicity (total HI) and total risk (total ELCR) for each land use scenario at each location are compared to benchmarks of 1 and 1E-6 for HI and ELCR, respectively. Land use scenarios with total HIs exceeding the benchmark of 1 are deemed land use scenarios of concern for systemic toxicity. Land use scenarios with total ELCR exceeding the benchmark of 1E-6 are deemed land use scenarios of concern for ELCR. To determine COCs, the chemical-specific HI and ELCR contributed by each COPC across all pathways within a land use scenario of concern are compared to benchmarks of 0.1 and 1E-6 for chemical-specific HI and ELCR, respectively. COPCs with chemical-specific HIs or ELCRs that exceed these benchmarks are deemed COCs for that land use scenarios of concern. To determine POCs, the exposure route HI and ELCR, respectively. Exposure routes with exposure route HI and ELCRs are deemed to benchmarks of 0.1 and 1E-6 for chemical-specific HI and ELCR across all COPCs within the land use scenarios of concern are compared to benchmarks of 0.1 and 1E-6 for exposure route HI and ELCR, respectively. Exposure routes with exposure route HIs and ELCRs that exceed these benchmarks of 0.1 and 1E-6 for exposure route HI and ELCR, respectively. Exposure routes with exposure route HIs and ELCRs that exceed these benchmarks are deemed POCs for that land use scenario of concern. MOCs are determined by examining the POCs and selecting any medium that appears in a POC as an MOC.

1.5.7.1 Land use scenarios of concern

As noted previously, if the total HI or total risk for a land use scenario exceeds 1 or 1E-6, respectively, then that land use scenario is a land use scenario of concern for the location. Exhibit 1.40 presents the land uses of concern for each location. The results include contributions from lead.

As shown in Exhibit 1.40, at no site are all the land use scenarios of concern, even when including the contribution of lead. The future recreational user land use scenario is of concern only at SWMU 193c, and only when lead is included as a COPC. Future industrial use and on-site rural residential use of RGA groundwater are land use scenarios of concern at all sites where groundwater was assessed with or without considering contributions from lead. Excavation worker exposure to subsurface soil is a land use scenario of concern at all sites regarding cancer risk.

	Representative	KDEP screening		3	
Location	concentration	value	Exceed?	value	Exceed
Groundwater (µg/L)					
SWMU 99a RGA	81	4	Yes	15	Yes
SWMU 99a McNairy	-	4	-	15	-
SWMU 99b RGA	-	4		15	
SWMU 193a RGA	-	4	_	15	-
SWMU 193a McNairy	-	4	-	15	-
SWMU 193b RGA	·	4	-	15	_
SWMU 193b McNairy	-	4	_	15	-
SWMU 193c RGA	. –	4	-	15	_
SWMU 193c McNairy	250	4	Yes	15	Yes
AOC 204 RGA	_	4	_	15	. –
Surface soil (mg/kg) ^b					
SWMU 99a	-	20	_	400	-
SWMU 193a	_	20	-	400	_
SWMU 193b	_	20	-	400	_
SWMU 193c	24.9	20	Yes	400	No
Subsurface soil (mg/kg) ^c					
SWMU 99a	18.1	20	No	400	No
SWMU 99b	-	20	-	400	_
SWMU 193a	-	20	· _	400	_
SWMU 193b	-	20	-	400	-
SWMU 193c	13.6	20	No	400	No
SWMU 194	15.8	20	No	4 00	No
AOC 204	-	20	_	400	_

Exhibit 1.39. Comparison of representative concentrations^a of lead against regulatory screening values

Notes: "-" indicates that lead was not a COPC for that location; therefore, a representative concentration is not available.

^a By definition (EPA 1992a), the representative concentration or the representative exposure concentration is the average contaminant concentration within an area; however, as shown in Sect. 1.2.3.1, this value is actually the lesser of the maximum detected concentration and the 95% UCL of the mean concentration.

^b Surface soil collected from 0-1 ft bgs.

^c Subsurface soil collected from 0-15 ft bgs.





	Site						
Scenario	SWMU			SWMU		SWMU	AOC
· · ·	<u>99a</u>	99b	193a	193b	193c	194	_ 204
Systemic toxicity ^a							
Current industrial worker							
Exposure to soil	-	NA	-	Xb	Xc	NA	NA
Future industrial worker							
Exposure to soil	_	NA	-	Xb	Xc	NA	NA
Exposure to RGA groundwater	Xď	Хр	Xp	Xb	Xp	NA	Xp
Exposure to McNairy groundwater	ХÞ	NA	Xp	-	\mathbf{X}^{d}	NA	NA
Future on-site rural resident ^a							
Exposure to soil	X۶	NA	Xp	Xb	Xď	NA	NA
Exposure to RGA groundwater	Xd	Xb	Xb	Xb	Xb	NA	Xb
Exposure to McNairy groundwater	Xb	NA	Xb	Xb	Xď	NA	NA
Off-site rural resident							
Exposure to groundwater ^e	Xe	_	Xe		Xe	Xť	X۴
Future recreational user ^a							
Exposure to soil	-	NA	-	-	X°	NA	NA
Future excavation worker							
Exposure to soil	X⁴	-	-	Xb	Xď	X	-
Excess lifetime cancer risk							
Current industrial worker				-			
Exposure to soil	x	NA	· X	х	-	NA	NA
Future industrial worker							
Exposure to soil	x	NA	х	х	-	NA	NA
Exposure to RGA groundwater	х	х	Х	х	х	NA	х
Exposure to McNairy groundwater	x	NA	х	-	х	NA	·NA
Future on-site rural resident ^f							
Exposure to soil	x	NA	х	х	-	NA	NA
Exposure to RGA groundwater	x	х	х	х	х	NA	Х
Exposure to McNairy groundwater	Х	NA	x	х	x	NA	NA
Off-site rural resident							
Exposure to groundwater ^e	-		-	-	-	· _	Xe
Future recreational user							
Exposure to soil	х	NA	х	-	-	NA	NA
Future excavation worker	_						
Exposure to soil	х	х	х	х	х	х	х

Exhibit 1.40. Selection of land uses of concern

Notes: Scenarios where risk exceeded the benchmark levels (HI of 1/ELCR of 1E-6) are marked with an "X." Scenarios where risk did not exceed a benchmark level are marked with a "-."

"NA" indicates that the scenario/land use combination is not appropriate.

^a For systemic toxicity regarding the future recreational user and the future on-site rural resident, the results for a child are presented.

^b These scenarios are of concern even though lead was undetected.

^c If contribution from lead is not considered, the total HI falls below 1, and the scenario is not of concern.

^d Lead is present, and the scenario is of concern whether or not the element is included in the assessment.

^e Based on the results of contaminant transport modeling, "X" indicates the location contains a source of unacceptable off-site contamination.

^f For excess lifetime cancer risk regarding the future recreational user and the future on-site rural resident, the values are for lifetime exposure.

1.5.7.2 Chemicals of concern

Only those contaminants whose chemical-specific ELCRs summed across all pathways within a land use scenario of concern are greater than or equal to 1E-6 or whose HQs summed across all pathways are greater than or equal to 0.1 are COCs. The COCs in soil across all land use scenarios for systemic toxicity are summarized in Exhibit 1.41. In this exhibit, contaminants that are COCs within a scenario of concern and have chemical-specific HIs greater than 1 are marked with a solid cell. Contaminants that are COCs within a scenario of concern and have chemical-specific HIs between 0.1 and 1 are marked with an "X." Contaminants that are not COCs within a scenario are not marked (i.e., cell left blank). Similar information for COCs in soil for ELCR is shown in Exhibit 1.43. In this exhibit, all COCs in soil across all land use scenarios for ELCR are summarized. Contaminants that are COCs within a scenario of concern and have chemical-specific ELCRs greater than 1E-4 are marked with a solid cell (see Exhibit 1.43). Contaminants that are COCs within a scenario of concern and have a chemical-specific ELCRs between 1E-6 and 1E-4 are marked with an "X." Contaminants that are not COCs within a scenario are not marked (i.e., cell left blank). Finally, Exhibits 1.42 and 1.44 present the COCs in RGA and McNairy groundwater over all land use scenarios. The markings used in these exhibits are the same as those used in Exhibits 1.41 and 1.43, respectively.

As shown in Exhibit 1.41, there are 16 COCs for systemic toxicity in soil in all of WAG 28. Of these COCs, 12 are inorganic and 4 are organic compounds. There are no COCs for systemic toxicity in soil at SWMU 99b. There are 12 COCs for systemic toxicity in soil at SWMU 99a, 9 inorganics and 3 organics. There is one COC for systemic toxicity in soil at SWMU 193a, and it is an inorganic compound. There are three COCs for systemic toxicity in soil at 193b, all of which are inorganic compounds. There are eight COCs for systemic toxicity in soil at SWMU 193c, all of which are inorganic compounds. There are six COCs for systemic toxicity in soil at SWMU 194, all of which are inorganic compounds. There are is one COC for systemic toxicity in soil at SWMU 194, all of which are inorganic compounds. There are is one COC for systemic toxicity in soil at AOC 204, and it is an organic compound.

As shown in Exhibit 1.42a, there are 26 COCs for systemic toxicity in RGA groundwater in all of WAG 28. There are 18 COCs for systemic toxicity in RGA groundwater at SWMU 99a, 15 inorganic and 3 organic compounds. There are five COCs for systemic toxicity in RGA groundwater at SWMU 99b, four inorganics and one organic compound. There are four COCs for systemic toxicity in RGA groundwater at SWMU 193a, two inorganic and two organic compounds. There are five COCs for systemic toxicity in RGA groundwater at SWMU 193a, two inorganic and two organic compounds. There are five COCs for systemic toxicity in RGA groundwater at SWMU 193b, all of which are organic compounds. There are two COCs for systemic toxicity in RGA groundwater at SWMU 193b, all of which are organic compounds. There are organic compounds. There are seven COCs for systemic toxicity in RGA groundwater at AOC 204, one inorganic and six organic compounds. Groundwater was not sampled at SWMU 194.

As shown in Exhibit 1.42b, there are 26 COCs for systemic toxicity in McNairy groundwater in all of WAG 28. There are four COCs for systemic toxicity in McNairy groundwater at SWMU 99a, all of which are organic compounds. There are three COCs for systemic toxicity in McNairy groundwater at SWMU 193a, one inorganic and two organic compounds. There are two COCs for systemic toxicity in McNairy groundwater at SWMU 193b, both of which are organic compounds. There are 25 COCs for systemic toxicity in McNairy groundwater at SWMU 193c, 15 inorganic compounds, 9 organic compounds, and 1 radionuclide.

Sites		S	WM	1U 9	9a				SV	VM	U 9	9b		Т	S	WN	1U	193	a			SV	M	U 19	3b		Γ	S	wM	U 19	93c	,		S	WM	IV 1	94				A0(C 20	4	
and scenarios Chemicals of potential concern ⁴	Current Industrial worker	Future Industriai worker	Future excavation worker		dent	Off-site rural resident	Current Industrial worker	Current Industrial WOrker Deters to to determine to the	r uture Inqustrial Worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current Industrial worker	Future Industrial worker	Future excavation worker		Future recreational user	Future on-site rural resident	Off-site rural resident	Current Industrial worker	Ruture industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current industrial worker	Future industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current industrial worker	Future Industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current industrial worker	Future industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident
2-Nitroaniline			X				Ι	Ι																					I						<u> </u>	ļ		L		<u> </u>]	
Aluminum																															l				X	<u> </u>	1	<u> </u>			l	<u> </u>	<u> </u>	
Antimony			X																									l]						[1		<u> </u>	<u> </u>
Barium		[Ι			Τ	Τ	Τ		-		Γ	1	Γ	Τ	Τ	Т	T																									
Beryllium			<u> </u>	1	X	T			T				Γ		Γ	Τ	T	T	T		X	X						1	Ι	ĺ				1	X									
Chromium			x	Ι			- 	I	I	T					Γ	T	Ι										X	X	X						X									
Iron				Γ	Т			Τ						1		Τ	Τ		Π										X			l								i		,		
Lead					Τ	1		Τ	Т	I			Γ	1	Γ	Т		T	Ι		1					Γ																		
Lithium									Τ						Γ		Γ		Ī										I]							
Manganese			x]										1		I											X											<u> </u>			<u> </u>	
PCB-1016				Γ																						ĺ		Ŀ							ĺ									
PCB-1254			Γ	Γ			Τ	Т	Τ	Τ				 		Ι	Ι	T	Ī							1											ļ							
Strontium				Ī	Ī	X	_	Τ	Τ	T			Ī		Γ	Т	Τ	T	1							1			I			X	Γ]			1							
Trichloroethene				T	1	Τ	-	Τ	T	T			Γ	1	Γ	Τ										Γ	Ι		Ι		I	I	Ī	1			Ι	I	ľ		Γ			
Vanadium			[Τ	1	T	1		T	1				Γ	Γ	Τ	Τ	T			ĺ		X						x	Γ	I	I	Ι	1			I	Ĩ	I				Ī	
Zinc				1	X	I		l	Ť	1					Γ	T	T	T	T		1						Γ	Γ	I		x	Γ	Ι	Τ			Ī	I	[Ι	

Exhibit 1.41. Chemicals of concern for systemic toxicity in soil at each site

Notes: "X" indicates that the chemical of potential concern is a chemical of concern, and chemical-specific HI is between 0.1 and 1 for the scenario. Solid cell indicates that the chemical of potential concern is a chemical of concern, and chemical-specific HI is greater than 1 for the scenario. Blank cell indicates that the chemical of potential concern is not a chemical of concern for the scenario.

^a Only chemicals of potential concern that have a chemical-specific HI greater than 1 for one or more land use scenarios of concern are listed.



Exhibit 1.42a. Chemicals of concern for systemic toxicity in RGA groundwater at each site

Sites		S	WM	IU 9	9a			S	WM	U 9	9b			S	WM	U 1	93a		1	sv	VM	U 19)3b			S	WM	Ū 19	3c			S	WM	U 19	94 ^b .				AOC	204	4	
And scenarios Chemicals of potential concern*	Current industrial worker	Future Industrial worker	Future excavation worker		Future on-site rural resident	Off-site rural resident	Current Industrial worker	Future industrial worker	Future excavation worker	Future recreational user	Future on-slte rural resident	Off-site rural resident	Current Industrial worker	Future industrial worker			lent	Off-site rural resident	Current Industrial worker	Future Industrial worker	Future excavation worker		Future on-site rural resident	Off-site rural resident	Current Industrial worker	Future Industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current Industrial worker	Future Industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current industrial worker	Future industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident
1,1-Dichloroethane			<u> </u>	Γ										1	Ī	T		1			Γ	1																x				
1,1-Dichloroethene				Γ		Γ	1			[I	1	Γ	1	Γ		1		1	X	1							Ī											
1,2-Dichloroethene				Ī	1	Γ	T							Γ	Γ	Τ	T	1	1	T	 	Γ	T	1		X	I															
Acetone		—		1	\square	Ť						Í		1	t	Ť	1	Ť	1	Ť	1	T	x	T	1	Γ	T	[[[T	\square	
Aluminum		x	1	1							-		1	1	\mathbf{T}	1	1	1		1	1	T	T	1		T	1	1	1	Γ		1										
Arsenic		x		1			1							\square	1	T	1	1		1		\square		\square		1	T	—		1		[
Barium	-		—	1	x	1					x	i		1-	1	Ť	1	Ť	—	T	Ī	İ	1	1		Ī	1			1									T			
Beryllium			1	1	x	1								1	1	1	T	1	1	1	1	1	T	\square	1	1	1		1	1		 	1									
Carbon tetrachloride			Ī	1	1	1								İ	1	1	1	\mathbf{T}		x		Ī				Γ	1			1									T		\square	
Chromium		x	[1				X				[1	1	T	1			1		Î	1	1	1	[[T		Π	
cis-1,2-Dichloroethene			[1	X	1							·	1	1-	1	x	T	 	T		Γ	x	1	<u> </u>	[1			[Ĩ	X	
Cobalt			ĺ	Í	x	Ť						[1	Ì	Ì	T	1	1	Ţ		Î	Ī	ĺ		1				Ī							Ī		T			
Copper			[1	X	1							·		1		Τ	Γ	-				1	Γ	1																	
Fluoride	********			1	Γ	1								Ī	1	T	x	1		T	 	Γ	1	1	1	Γ	Γ		-	[
Iron		x	Ī	1							x	[1			1	1	[Ī	Ī	1	1	Γ													T	1		
Lead				Γ								[1	Γ	1	1	T	T		Ι	I	-	1	Τ	1				-	[
Lithium		Γ	1	1	X					1		1		1	T	1	1	1	1	1	Ī	T	Ī	1	1	1	1	1	1													
Manganese		x	T	1							x	1	1	Ī	1	T	1	1		1	1	1	1	1	1-		Γ_												1			\square
Мегсигу	[1	T	T	x	T	· · · · ·			—		—	1	1	1	T	1	T	1	T	1	T	1	1	1	T	Γ	1		1		<u> </u>								T	[]	
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Tetrachloroethene		1		1	T	\square	1	—	—	1	1	-	1	1	1-	T	1	T	-	1	<u> </u>	1	1	\mathbf{T}	1	1	1	1		1	\square											
Trichloroethene				1			1		—	Í		İ	·	x	1	1			1		[1		· · · ·		x		-									-	· · · · · · · · · · · ·				
Vanadium		x	1	1			1	1		1	[—		İ	t	Ť	1	1	1	1			Ī	1	1	T T	1															
Zinc	†	T	1	\square	x	1	1-	1-	\vdash					1-	1	t-	1	1	†	1			1	\square	t					<u> </u>												

Notes: "X" indicates that the chemical of potential concern is a chemical of concern, and chemical-specific HI is between 0.1 and 1 for the scenario. Solid cell indicates that the chemical of potential concern is a chemical of concern, and chemical-specific HI is greater than 1 for the scenario. Blank cell indicates that the chemical of potential concern is not a chemical of concern for the scenario.

^a Only chemicals of potential concern that have a chemical-specific HI greater than 1 for one or more land use scenarios of concern are listed.
 ^b Groundwater not sampled at SWMU 194.

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Sites		e.	SI/M	IU 9	0			61	wм	TT 00	3F-p			51	ww	U 1	02.		1	61		U 19	36		1	67	VMI	U 19	30		· ·	SV	VMI	U 19	4 ^b				400	204	4 ^b	
and scenarios	<u> </u>	3	44 141 1	10 9	721 	1	\vdash	3	TAT AN	0 93	1	1		101	1 V 1 V 1	1	100	1		1	1 141	1	50			31	A TAT		<u> </u>				1			[1	<u>,</u>	1	1	1	
Chemicals of potential concern*	Current Industrial worker	Future industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current industrial worker	Puture industrial worker	Puture escavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current Industrial worker	Future Industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current Industrial worker	Future Industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current Industrial worker	Future Industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current Industrial worker	Future Industrial worker	Future excavation worker	Future recreational user	Future on-slte rural resident	Off-site rural resident	Current industrial worker	Future industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident
1,1-Dichloroethene					X	l	1									Ι	1			I									X													
1,2-Ddichloroethane				l	Ī	Τ	T	Γ			l					Ī	I	Τ		T		1							Х									Ľ		ļ	L	ļ
1,1,2-Trichloroethane					Γ	I		[Γ							1		I									X]	<u> </u>	<u> </u>	
Aluminum			<u> </u>			Τ		Γ			<u> </u>					Τ	Τ	Τ	1	Τ		1				X										ĺ						
Antimony		İ	1	1	1	1		1				1			t	1	1	1	1	1			1	[1													l	Ī		Ī	
Arsenic			[1	İ	İ										1	1	T		Τ				[X																
Barium				1	1	1								1	Ī	Γ	İ	1		Ì	1	Ī	Ī	I					Χ													
Beryllium		1		Ī												1	1	1	1	T		1							Х	[
Benzene		İ	Ī	1	[İ									Ī	Ì	T	T	1	1		Í	Γ						Х													
Cadmium				1	1	1									1	Ī		1		T		Ī	ĺ			х	•															
carbon tetrachloride				Ī													1			Τ		Γ	Γ																	[
Chloroform			l			Ī								[Ι		Ī	1			l							X													<u> </u>
Chromium			Γ	Ι	Γ											Γ	I		Γ							X															<u> </u>	<u> </u>
cis-1,2-Dichloroethene		X												X															х										1		Į	į
Cobalt																													Х												Į	1
Iron																									1														1		1	
Lead																																						L	<u> </u>		_	ļ
Manganese																										X													L	<u> </u>		
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Uranium						[1										Ι		Ι		[х				l					[Ī	[Π	
Vanadium		Γ	Γ	Γ	Γ	T	1	Γ	Γ	I	Π		<u> </u>	[1	1	Τ	I	1	T		Γ																[1	[Ī	

Exhibit 1.42b. Chemicals of concern for systemic toxicity in McNairy groundwater at each site

Notes: "X" indicates that the chemical of potential concern is a chemical of concern, and chemical-specific HI is between 0.1 and 1 for the scenario. Solid cell indicates that the chemical of potential concern is a chemical of concern, and chemical-specific HI is greater than 1 for the scenario. Blank cell indicates that the chemical of potential concern is not a chemical of concern for the scenario.

^a Only chemicals of potential concern that have a chemical-specific HI greater than 1 for one or more land use scenarios of concern are listed.

^b McNairy groundwater not sampled at this site.

As shown in Exhibit 1.43, there are 24 COCs for ELCR in soil in all of WAG 28. There are ----23 COCs for ELCR in soil at SWMU 99a, 2 inorganic compounds, 15 organic compounds, and 6 radionuclides. There are two COCs for ELCR in soil at SWMU 99b, both of which are inorganic compounds. There are six COCs for ELCR in soil at SWMU 193a, one inorganic and five organic compounds. There is one COC for ELCR in soil at SWMUs 193b, 193c, and 194, and it is an inorganic compound. Beryllium is a COC at all sites, except AOC 204. There is one COC for ELCR in soil at AOC 204, and it is an organic compound.

As shown in Exhibit 1.44a, there are 15 COCs for ELCR in RGA groundwater in all of WAG 28. There are seven COCs for ELCR in RGA groundwater at SWMU 99a, two inorganic compounds, three organic compounds, and two radionuclides. There are two COCs for ELCR in RGA groundwater at SWMU 99b, one organic compound and one radionuclide. There are five COCs for ELCR in RGA groundwater at SWMU 193a, four organic compounds and one radionuclide. There are five COCs for ELCR in RGA groundwater at SWMU 193b, four organic compounds and one radionuclide. There are five COCs for ELCR in RGA groundwater at SWMU 193b, four organic compounds and one radionuclide. There are five COCs for ELCR in RGA groundwater at SWMU 193b, four organic compounds and one radionuclide. There is one COC for ELCR in RGA groundwater at SWMU 193c, and it is an organic compound. There are eight COCs for ELCR in RGA groundwater at AOC 204, all of which are organic compounds. Groundwater was not sampled at SWMU 194.

As shown in Exhibit 1.44b, there are 16 COCs for ELCR in McNairy groundwater in all of WAG 28. There are three COCs for ELCR in McNairy groundwater at SWMU 99a, all of which are organic compounds. There are three COCs for ELCR in McNairy groundwater at SWMU 193a, one organic compound and two radionuclides. There is one COC for ELCR in McNairy groundwater at SWMU 193b, and it is an organic compound. There are 14 COCs for ELCR in McNairy groundwater at SWMU 193c, 2 inorganic compounds, 11 organic compounds , and 1 radionuclide.

Combining the results from Exhibits 1.41 and 1.43 and considering the magnitude of the chemicalspecific HIs and ELCRs, the following COCs can be considered "priority COCs" (COCs with a chemicalspecific HI or ELCR that exceeds 1 or 1E-4, respectively) in soil for the current use and most likely future use scenario (i.e., industrial use):

- SWMU 99a—beryllium
- SWMU 99b—surface soil not assessed
- SWMU193a—none
- SWMU 193b—chromium, vanadium, and beryllium
- SWMU 193c—lead
- SWMU 194—surface soil not assessed
- AOC 204—surface soil not assessed

Combining the results from Exhibits 1.41 and 1.43 and considering the magnitude of the chemicalspecific HIs and ELCRs, the following COCs can be considered "priority COCs" (COCs with a chemicalspecific HI or ELCR that exceeds 1 or 1E-4, respectively) in soil for the future excavation worker:

- SWMU 99a—lead
- SWMU 99b—beryllium
- SWMU 193a—beryllium
- SWMU 193b—beryllium and chromium
- SWMU 193c—beryllium and lead
- SWMU 194-beryllium and lead
- ACO 204—none



																																		_			-					
Sites		S	WM	IU 9	9a	-		S	WM	U 9	9b			S	WM	<u>U 1</u>	<u>3a</u>			SV	VM	U 19	<u>3b</u>			SV	VM.	<u>U 19</u>	<u>3c</u>			S	WM	<u>U 1</u>	94		L		<u>AO(</u>	20	4	,
and scenarios	worker	rker	orker	user	resident	nt	vorker	orker	orker	user	resident	nt	vorker	orker	orker	user	resident	t	rorker	orker	orker	user	resident	nt	vorker	orker	orker/	user	resident	nt	vorker	orker	/orker	user	resident	nt	vorker	orker	vorker	user	resident	nt
Chemicals of potential concern*	Current Industrial w	Future Industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current Industrial worker	Future Industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current Industrial worker	Future Industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current Industrial worker	Future Industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current industrial worker	Future Industrial worker	Future excavation worker	Future recreational user	Future on-site rural resident	Off-site rural resident	Current Industrial worker	Future industrial worker	Future excavation worke	Future recreational user	Future on-site rural resident	Off-site rural resident	Current industrial worker	Future Industrial worker	Future excavation worker	Future recreational user	Future on-slte rural resident	Off-site rural resident
Arsenic		ļ	X	ļ		ļ	ļ	 	x		 	ļ		_	ļ	ļ	ļ	ļ	ļ	ļ			 	 			ļ	 		 		 	 	ļ		ļ		ļ			 	ļ
Benz(a)anthracene		x					ļ	ļ						ļ	ļ	ļ	X	ļ	ļ	ļ		ļ	ļ	 			L	ļ		ļ		ļ	ļ	ļ							 	ļ
Benzo(a)pyrene		X		ļ			ļ	ļ					x	x	<u> X</u>	X	2	ļ	ļ	ļ			 	Ļ				ļ		.		ļ	ļ	ļ	ļ							
Benzo(b)fluoranthene	x	x	X	L			L								<u> </u>	<u> </u>	x	Į	ļ				[Ļ				 		Ļ		ļ		L								
Benzo(k)fluoranthene					x										L								Į							.												
Beryllium			х																													l										
bis(2-Chloroethyl)ether			X												L		l															L		<u> </u>					<u>.</u>			
Cesium-137	х	x	X		x																																					
Chrysene					X																																					
dibenz(a,h)anthracene	X	X	X										X	x	x	X							L	<u>i</u>																		
Dieldrin			x													l	L																									
Hexachlorobenzene			x																																							
Indeno(1,2,3-cd)pyrene	X		X											•			X																									
Neptunium-237	x	X	X												[[
N-nitrosodi-n-propylanine			x																																							
РСВ-1016		Γ	Ι				Γ								Γ	Γ		1						Ī																		
PCB-1254		Γ	Ī		x	Γ	1								[1	Γ	Ι	[[I																		
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Thorium-234		l	x	T			1								1	1	1	1																								
Toxaphene	1	Ī	x	T	I	1	1.	1	1		 				İ	1	1	Ī																								
Trichloroethene	†	Ī	İ			1	1	1	Ī		1				İ	1	Ī	Ī	1					1																		
Uranium-234		1	x				—		1		1					[\square	
Uranium-238	x	x	4				1	[1		1				1	1		1	<u> </u>					1																		

Exhibit 1.43. Chemicals of concern for ELCR in soil at each site

Notes: "X" indicates that the chemical of potential concern has been selected as a chemical of concern, and the pathway-specific ELCR is between 1E-6 and 1E-4. Solid cell indicates that the chemical of potential concern is a chemical of concern, and chemical-specific ELCR is greater than 1E-4 for the scenario. Blank cell indicates that the chemical of potential concern is not a chemical of concern for the scenario.

^a Only chemicals of potential concern that have a chemical-specific ELCR greater than 1E-6 for one or more land use scenarios of concern are listed.

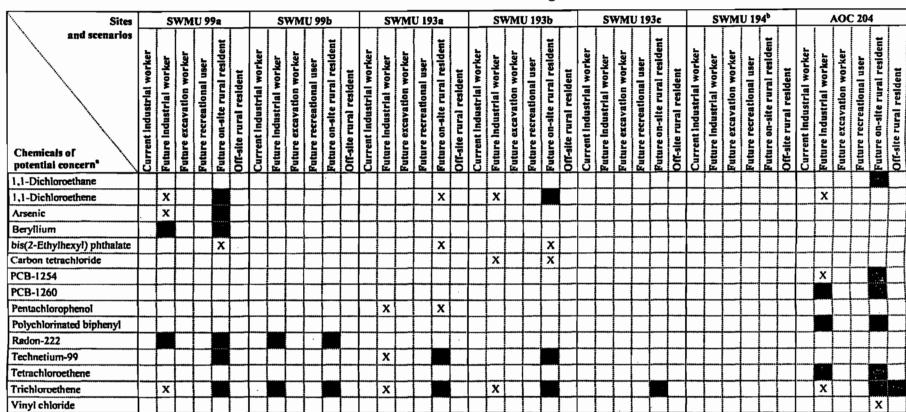


Exhibit 1.44a. Chemicals of concern for ELCR in RGA groundwater at each site

Notes: "X" indicates that the chemical of potential concern has been selected as a chemical of concern, and the pathway-specific ELCR is between 1E-6 and 1E-4. Solid cell indicates that the chemical of potential concern is a chemical of concern, and chemical-specific ELCR is greater than 1E-4 for the scenario. Blank cell indicates that the chemical of potential concern is not a chemical of concern for the scenario.

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^a Only chemicals of potential concern that have a chemical-specific ELCR greater than 1E-6 for one or more land use scenarios of concern are listed.

^b Groundwater not sampled at SWMU 194.

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Exhibit 1.44b. Chemicals of concern for ELCR in McNairy groundwater at each site

Notes: "X" indicates that the chemical of potential concern has been selected as a chemical of concern, and the pathway-specific ELCR is between 1E-6 and 1E-4. Solid cell indicates that the chemical of potential concern is a chemical of concern, and chemical-specific ELCR is greater than 1E-4 for the scenario. Blank cell indicates that the chemical of potential concern is not a chemical of concern for the scenario.

^a Only chemicals of potential concern that have a chemical-specific ELCR greater than 1E-6 for one or more land use scenarios of concern are listed.

^b McNairy groundwater not sampled at this site.

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Combining the results from Exhibits 1.41 and 1.43 and considering the magnitude of the chemicalspecific HIs and ELCRs, the following COCs can be considered "priority COCs" (COCs with a chemicalspecific HI and ELCR that exceeds 1 or 1E-4, respectively) in soil for the future on-site rural resident:

- SWMU 99a—barium, beryllium, chromium, benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, dibenz(a,h)anthracene, indeno(1,2,3,-cd)pyrene, PCB-1016, PCB-1254, neptunium-237, technetium-99, thorium-234, uranium-234, and uranium-238
- SWMU 99b— surface soil not assessed
- SWMU193a—chromium, benzo(a)pyrene, and dibenz(a,h)anthracene
- SWMU 193b-beryllium, chromium, and vanadium
- SWMU 193c---chromium and lead
- SWMU 194--- surface soil not assessed
- AOC 204 surface soil not assessed

Combining the results from Exhibits 1.41 and 1.43 and considering the magnitude of the chemicalspecific HIs and ELCRs, the following COCs can be considered "priority COCs" (COCs with an HI or ELCR that exceeds 1 or 1E-4, respectively) in soil for the future recreational user:

- SWMU 99a-none
- SWMU 99b—surface soil not assessed
- SWMU193a-none
- SWMU 193b—none
- SWMU 193c—lead
- SWMU 194—surface soil not assessed
- AOC 204—surface soil not assessed

Combining the results from Exhibits 1.42a and 1.44a and considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs can be considered "priority COCs" (COCs with a chemical-specific HI or ELCR that exceeds 1 or 1E-4, respectively) in RGA groundwater for the most likely future use at the following sites (i.e., industrial use):

- SWMU 99a—beryllium, lead, trichloroethene, and radon-222
- SWMU 99b—trichloroethene and radon-222
- SWMU193a-iron
- SWMU 193b—trichloroethene
- SWMU 193c—none
- SWMU 194-RGA groundwater not assessed
- AOC 204—PCB-1254, PCB-1260, PCBs, tetrachloroethene, and trichloroethene

Combining the results from Exhibits 1.42a and 1.44a and considering the magnitude of the chemicalspecific HIs and ELCRs, the following COCs can be considered "priority COCs" (COCs with a chemicalspecific HI or ELCR that exceeds 1 or 1E-4, respectively) in RGA groundwater for on-site rural residential use in the home at the following sites:

- SWMU 99a—aluminum, arsenic, beryllium, chromium, iron, lead, manganese, vanadium, 1,1-dichloroethene trichloroethene, radon-222, and technetium-99
- SWMU 99b-chromium, trichlororethene, and radon-222
- SWMU193a—iron, trichloroethene, and technetium-99
- SWMU 193b-1,1-dichloroethene, carbon tetrachloride, trichloroethene, and technetium-99
- SWMU 193c—1,2-dichloroethene and trichloroethene

- SWMU 194—RGA groundwater not assessed
- AOC 204—1,1-dichloroethane, 1,1-dichloroethene, PCB-1254, PCB-1260, PCBs, tetrachloroethene, and trichloroethene

Combining the results from Exhibits 1.42b and 1.44 and considering the magnitude of the chemicalspecific HIs and ELCRs, the following COCs can be considered "priority COCs" (COCs with a chemicalspecific HI or ELCR that exceeds 1 or 1E-4, respectively) in McNairy groundwater for the most likely future use at the following sites (i.e., industrial use):

- SWMU 99a—trichloroethene
- SWMU 99b—McNairy groundwater not assessed
- SWMU193a—iron
- SWMU 193b—none
- SWMU 193c—antimony, beryllium, iron, lead, and vanadium
- SWMU 194—McNairy groundwater not assessed
- AOC 204—McNairy groundwater not assessed

Combining the results from Exhibits 1.42b and 1.44a and b and considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs can be considered "priority COCs" (COCs with a chemical-specific HI or ELCR that exceeds 1 or 1E-4, respectively) in McNairy groundwater at the following sites for future on-site rural residential use at the following sites:

- SWMU 99a—carbon tetrachloride, cis-1,2-dichloroethene, 1,1-dichloroethene, and trichloroethene
- SWMU 99b—McNairy groundwater not assessed
- SWMU193a—cis-1,2-dichloroethene, iron, and technetium-99
- SWMU 193b—cis-1,2-dichloroethene and trichloroethene
- SWMU 193c—aluminum, antimony, arsenic, beryllium, cadmium, carbon tetrachloride, chromium, iron, lead, manganese, molybdenum, vanadium, 1,1-dichloroethene, vinyl chloride, and radon-222
- SWMU 194—McNairy groundwater not assessed
- AOC 204—McNairy groundwater not assessed

The following chemicals are "priority COCs" for off-site use of groundwater (i.e., rural residential use in the home). These chemicals are all COCs that may migrate from a source at a site in WAG 28 to an off-site location and present a chemical-specific HI or ELCR to the rural resident that is greater than 1 or 1E-4, respectively.

- SWMU 99a—lithium
- SWMU 99b—none
- SWMU193a—chromium
- SWMU 193b—none
- SWMU 193c—lithium and manganese
- SWMU 194—chromium, lithium, and strontium
- AOC 204 trichloroethene

1.5.7.3 Pathways of concern

Only those pathways with a pathway HI for adults or children greater than 0.1 or a pathway ELCR greater than 1E-6 across all contaminants within a land use scenario of concern are POCs. The POCs for each land use scenario of concern are presented in Exhibit 1.45. Inhalation of vapors and particulates from soil and ingestion of quail are not POCs. All other exposure routes are found in POCs in at least one site.

1.5.7.4 Media of concern

MOCs are those media that appear in at least one POC. Based on the information presented in Sect. 1.5.7.3 and Exhibit 1.45, the following media are of concern for each site:

- SWMU 99a-surface soil, subsurface soil, RGA groundwater, and McNairy groundwater
- SWMU 99b—subsurface soil and RGA groundwater
- SWMU193a—surface soil, subsurface soil, RGA groundwater, and McNairy groundwater
- SWMU 193b—surface soil, subsurface soil, RGA groundwater, and McNairy groundwater
- SWMU 193c---surface soil, subsurface soil, RGA groundwater, and McNairy groundwater
- SWMU 194—subsurface soil
- AOC 204—RGA groundwater

1.5.8 Summary of Risk Characterization

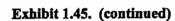
Tables 1.75–1.81 present summaries of the risk characterizations for WAG 28 sites. Each of these tables present land use scenarios of concern, COCs, and POCs. Along with this information, each table lists the risk posed to a receptor under each land use scenario of concern, the percent of risk each POC contributes to the total risk, and the percent of risk each COC contributes to the total risk. The tables that summarize the results for systemic toxicity do not include contributions from lead, because to do so would make the contributions from the other COCs appear meaningless.

In addition to the summary tables, Appendix E of this volume provides a more detailed summary of the risk assessment. In this appendix, the COPCs, CDIs, slope factors, RfDs, ELCRs, HIs, toxic effects, cancer classification, and total risk by pathway, land use, and site are tabulated. These tables allow for a direct check of the risk calculations discussed in this section. In addition, if additional toxicity information becomes available, these tables will allow for easy recalculation of risk for each COPC, pathway, and land use scenario at each site. The information in the tables in Appendix E was used to construct the risk characterization and summary tables presented earlier in this section.

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Exhibit 1.45. Pathways of concern at each site with lead included as a COPC

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Notes: "X" indicates that the pathway has been selected as a pathway of concern, and the pathway-specific HI is between 0.1 and 1 for the scenario and ELCR is between 1E-6 and 1E-4.

Solid cell indicates that the pathway has been selected as a pathway of concern, and the pathway-specific HI is greater than 1 for the scenario and ELCR greater than 1E-4.

Blank cell indicates that the pathway of potential concern is not a pathway of concern for the scenario.

^a Only land uses of concern that have pathway-specific HIs greater than 0.1 and ELCRs greater than 1E-6 are listed.

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1.6 UNCERTAINTY IN THE RISK ASSESSMENT

Uncertainties are associated with each step of the risk assessment process. The potential effect of the uncertainties on the final risk characterization must be considered when interpreting the results of the risk characterization because a number of assumptions are made during the risk assessment. Types of uncertainty to be considered are divided into four broad categories, including those associated with data, the exposure assessment, the toxicity assessment, and the risk characterization. Specific uncertainties in each of these broad categories are discussed in the following sections, with the magnitude of the effect of the uncertainty on the risk characterization being categorized as either small, moderate, or large. Uncertainties categorized as small do not affect the risk estimates by more than one order of magnitude; while uncertainties categorized as large may affect the risk estimate by more than two orders of magnitude.

In evaluating these uncertainties and their estimated effect on the risk estimates, it should be remembered that the following uncertainties are neither independent nor mutually exclusive; therefore, the total effect of all uncertainties discussed in the following sections on the risk estimates (i.e., total ELCRs and HIs) is not necessarily the sum of the estimated effects.

1.6.1 Uncertainties Associated with Data and Data Evaluation

Several uncertainties are associated with the data set and the selection of COPCs. Specific uncertainties discussed in the following sections are (1) selection of COPCs, (2) determination of exposure point concentrations under current and future conditions, (3) use of concentrations from total versus filtered samples for inorganic compounds in groundwater, and (4) use of concentrations from borehole versus monitoring well samples.

1.6.1.1 Selection of COPCs

Uncertainty in the selection of COPCs is derived from several sources. The first uncertainty relating to the selection of COPCs is the retention of infrequently detected or infrequently analysed for chemicals in the list of COPCs. As seen in Table 1.9, several of the chemicals retained in the list of COPCs were detected in less than 10 percent of the samples. Of greatest concern is that some of these COPCs are retained as COCs. Table 1.82 presents the differences in total ELCR estimates and total HIs when the chemicals detected in less than 10 percent of the samples are retained as COPCs and when they are deleted from the COPC list. These data indicate that, in most cases (67/77 comparisons), the infrequently detected COPCs had no effect on the risk or hazard estimates. Even where a difference was noted, for example, for a future rural resident child exposed to soil at SWMU 99a, eliminating the HQs for the infrequently detected PCBs 1016 and 1254 (each detected in 1/16 samples) resulted in a reduction in the total HI that fell within the range applicable to a small contribution to uncertainty (approximately 5-fold); therefore, the estimated effect of this uncertainty on the risk estimates as a whole is small (less than one order of magnitude).

The second uncertainty relating to selection of COPCs is that temporal patterns in detection of analytes were not considered when selecting COPCs. If temporal patterns were considered, the final risk results in this BHHRA may have been quite different depending on the times at which risks were estimated; however, in the time frame considered in this BHHRA (40 years), the assumed effect of this uncertainty on the risk estimates is small.

The third uncertainty relating to selection of COPCs concerns the quantitation limits used for some analytes. For many organic analytes and some radionuclides, the quantitation limit exceeds a

concentration or activity that may result in a significant health effect. For example, for RGA groundwater at SWMU 193a, 35 organic compounds and 2 radionuclides have quantitation limits for at least one sample that exceed their residential use ELCR RBCs (see Table 1.83). Similarly, in subsurface soil at AOC 204, six radionuclides and one organic compound have quantitation limits for at least one sample that exceed the residential use ELCR RBC (see Table 1.83). Because the quantitation limits exceed the RBCs, these chemicals and radionuclides could be present in amounts that contribute to risk but not be retained as COPCs for quantitative evaluation; however, because many of these components in RGA groundwater at SWMU 193a tend to be unrelated to processes at WAG 28, the effect of this uncertainty on the risk estimates for this scenario is likely to be small.

A fourth uncertainty relating to the selection of COPCs is the inclusion of common laboratory contaminants in the COPC list. Table 1.84 presents the difference in total ELCR estimates and total HI when common laboratory contaminants are retained as COPCs and when they are deleted from the COPC list. This table indicates that common laboratory contaminants COPCs had virtually no effect on risk or hazard estimates; therefore, the estimated effect of the uncertainty on risk estimates is small.

A fifth uncertainty relating to the selection of COPCs is based on the fact that, in this risk assessment, contaminant concentrations were not compared to concentrations found in blank samples. Typically, common laboratory contaminants and other analytes may be deleted from the COPC list if they are also detected in blank samples at appropriate concentrations (RAGS). While not performing this test ensures that all "hits" for compounds that may be potential laboratory contaminants are treated as site contaminants, it is actually impossible to distinguish unequivocally between the environmental medium and the laboratory-based extraction procedures and processes as the potential source of the observed contamination; however, the effect of this uncertainty is estimated to be small because, in general, common laboratory contaminants such as acetone, methylene chloride, and the phthalates appear to be present only at low concentrations and do not contribute significantly to risk (see Table 1.84).

A sixth uncertainty relating to the selection of COPCs is the use of a toxicity screen to determine the final COPC list. In this BHHRA, the maximum detected concentrations of analytes within each medium at each site were compared to residential human health risk-based screening criteria. The residential risk-based screening levels were used per regulatory agreement (Methods Document). Analytes with maximum concentrations less than these screening criteria were removed from the list of COPCs. The derivation of these criteria is explained in detail in Sect. 1.2.

To examine what effect the toxicity screen may have had on the COPC list and on the resulting risk estimates, hazard and risk contributions from analytes removed on the basis of this screen were calculated. Thus, these marginal hazard and risk contributions are the estimated increases in the final hazard and risk estimates that would be seen if the analytes removed from the list of COPCs had been retained. Exhibit 1.46 presents the marginal contributions to total HI and total ELCR, by medium and location, for those areas in which the rural residential scenario was assessed. As illustrated in this exhibit, the marginal contribution of the analytes removed from the COPC list is negligible; therefore, the estimated effect of this uncertainty on the final risk estimates is small.

In Table 1.85, the background concentrations in soil used for this screen are compared to their respective medium-specific human health risk-based screening criteria discussed in Sect. 1.2. As shown in Table 1.85, several of the background concentrations used in the BHHRA are greater than their respective risk-based screening criteria. Note that the target HI and ELCR used for chemicals when calculating the screening criteria are 0.1 and 1E-7, respectively, and that the target ELCR for radionuclides is 1E-6.

		Total ELC	R	_	Total hazard	index
Site	Total ELCR	Marginal contribution	Total with analytes added	Total HI	Marginal contribution	Total with analytes added
Future on-site rural re	esident					
SWMU 99a (RGA)	5.6E-3	0	5.6E-3	90,600	< 1	90,600
SWMU 99a (McN)	1.8E-3	6.9E-7	1.8E-3	53.1	< 1	53.1
SWMU 99a (soil)	>1E-2ª	0	> 1E-2ª	17.2	< 1	17.2
SWMU 99b (RGA)	2.3E-3	6.9E-7	2.3E-3	208	< 1	208
SWMU 193a (RGA)	2.4E-3	2.7E-7	2.4E-3	28.6	< 1	28.6
SWMU 193a (McN)	4.2E-4	1.4E-6	4.2E-4	59.9	< 1	59.9
SWMU 193a (soil)	7.1E-4	8.0E-8	7.1E-4	6.25	< 1	6.25
SWMU 193b (RGA)	1.0E-3	0	1.0E-3	55.5	< 1	55.5
SWMU 193b (McN)	1.2E-5	0	1.2E-5	2.69	0	2.69
SWMU 193b (soil)	3.0E-3	0	3.0E-3	66.7	< 1	66.7
SWMU 193c (RGA)	1.5E-4	0	1.5E-4	80.7	0	80.7
SWMU 193c (McN)	4.0E-3	9.8E-7	4.0E-3	278,000	< 1	278,000
SWMU 193c (soil)	1.1E-9	0	1.1E-9	247,000	< 1	247,000
AOC 204 (RGA)	> 1E-2 ^a	0	>1E-2 ^a	279	< 1	279

Exhibit 1.46. Marginal ELCR and HI contributions of analytes removed from the COPC list on the basis of the toxicity screen with lead included as a COPC

Note: All HI values are for the child.

The ELCR is approximate because the linearized multistage model returns imprecise values at risks > 1E-2.

The results presented in this table indicate that if analytes were not removed from the COPC list on the basis of the background screen, the final risk estimates would be larger; however, because this screen relied on a comparison of the maximum detected concentration of each analyte in a medium to the selected soil background concentration, erroneous removal of analytes from the list of COPCs is unlikely; therefore, removing contaminants from the COPC list on the basis of a background screen is likely to have had no more than a small effect on the final risk estimates.

Unlike soil, a background screen for groundwater was not used when developing the list of COPCs. Such a screen was omitted because the background concentration data for groundwater, which have been used in earlier risk assessments at the PGDP, were determined to be suspect during recent discussions with regulatory agencies because of changes in sampling methods. Generally, these changes in sampling methods led to inappropriate comparisons between analyte concentrations in site samples and the background concentrations; however, after the preparation of the D0 draft of this risk assessment, an internal draft of new site-wide background data for groundwater became available (Bonczek 1999). Although preliminary, these data have been used herein to screen COPCs whose concentration or activity in groundwater fell below their prevailing concentrations in areas not impacted by PGDP. Comparing the overall risks or hazards for the future industrial and residential scenarios with and without the contribution of these screened COPCs gives a measure of the degree of uncertainty bounding the risk and hazard determinations when a background groundwater screen is omitted. As seen in Exhibit 1.47, the overwhelming majority of comparisons return the same value for the ELCR or HI whether or not the background screen is in place. Where a difference is noted, for example, in the ELCR for the future onsite rural resident exposed to RGA groundwater at SWMU 99b, the difference is less than an order of magnitude (2.6E-3 versus 2.0E-3); therefore, the effect of this uncertainty is small.



Location	HI – groundwater screen (default)	HI + groundwater screen	ELCR – groundwater screen (default)	ELCR + groundwater screen
Future industrial worker				
SWMU 99a (RGA)	5.11	5.11	5.6E-4	5.6E-4
SWMU 99a (McNairy)	1.64	1.64	7.6E-5	7.6E-5
SWMU 99b (RGA)	7.0	6.93	2.6E-4	1.4E-4
SWMU 193a (RGA)	1.64	1.64	2.6E-5	2.6E-5
SWMU 193a McNairy)	4.69	4.69	1.1E-6	1.1E-6
SWMU 193b (RGA)	1.74	1.74	4.4E-5	4.4E-5
SWMU 193b McNairy)	< 1	<1	8.4E-7	8.4E-7
SWMU 193c (RGA)	1.46	1.46	1.1E-5	1.1E-5
SWMU 193c (McNairy)	9.92	9.92	4.2E-4	3.8E-4
AOC 204 (RGA)	<u>3</u> 3.3	33.3	1.3E-3	1.3E-3
Future on-site rural resid	lent adult			
SWMU 99a (RGA)	NA	NA	5.6E-3	5.6E-3
SWMU 99a (McNairy)	NA	NA	1.8E-3	1.8E-3
SWMU 99b (RGA)	NA	NA	2.6E-3	2.0E-3
SWMU 193a (RGA)	NA	NA	2.4E-3	2.4E-3
SWMU 193a (McNairy)	NA	NA	4.2E-4	4.2E-4
SWMU 193b (RGA)	NA	NA	1.0E-3	1.0E-3
SWMU 193b (McNairy)	NA	NA	1.2E-5	1.2E-5
SWMU 193c (RGA)	NA	NA	1.5E-4	1.5E-4
SWMU 193c (McNairy)	NA	NA	4.0E-3	3.9E-3
AOC 204 (RGA)	NA	NA	> 1E-2ª	> 1 E-2 ª

Exhibit 1.47. ELCR and HIs for the future industrial worker and future on-site rural resident exposed to groundwater with lead excluded as a COPC—the effect of omitting COPCs with concentrations below site-wide background concentrations

Notes: NA = Not Applicable

• The absolute value is omitted because a risk of > 1E-2 as calculated by the linearized multistage model may be imprecise.

1.6.1.2 Determination of exposure point concentrations—current conditions

The uncertainty in the calculated exposure point concentrations under current conditions cannot be quantified for this BHHRA. Although sampling data came from sources of known quality, and the data set was generated from samples collected and analyzed using EPA-approved protocols, the lack of validation for some data could have resulted in the retention of erroneous analyte concentrations; however, because the risk estimates are driven for the most part by contaminants determined from earlier investigations to be present in the WAG 28 sites, the effect of this uncertainty on the final risk estimates is believed to be small.

1.6.1.3 Determination of exposure point concentrations—future conditions

In calculating the exposure point concentrations under future conditions at WAG 28 sites, the concentrations of COPCs are assumed to be constant throughout the exposure period. That is, the risk assessment does not consider that concentrations of some contaminants may be lower or higher in the future because of processes such as degradation and attenuation; however, because a substantial proportion of the contaminants driving risk at the sites is not expected to degrade significantly throughout a lifetime, the overall effect of this uncertainty is estimated to be small.

Uncertainty also surrounds the risk to receptors as COPCs in media at WAG 28 sites migrate to groundwater below the sites and are transported off site. As noted in Chap. 5 of Vol. 1, to address this uncertainty, the MEPAS model was used to estimate potential concentrations of selected COPCs in groundwater at the security fence and the DOE property boundary. A complete presentation of the results of the MEPAS model is. in Appendix B of this volume. While the MEPAS model can estimate contaminant transport though multiple media, this model does not consider the presence of all possible contaminants and the geochemical interactions that may occur. Additionally, the model estimates contaminant concentrations assuming that the receiving groundwater is uncontaminated, in light of which the contaminant concentrations estimated for groundwater may differ from the actual concentrations; therefore, risk estimates generated using the results of the MEPAS model should be considered as screening estimates and should only be used to direct future modeling efforts as needed. If risk managers wish to consider the potential risk to an off-site user exposed to on-site contaminant concentrations, a value is available. Generally, the effect of the MEPAS modeling uncertainties on the risk estimates is considered moderate.

1.6.1.4 Use of concentrations from total versus filtered samples

In this BHHRA, all analyte concentrations in water came from the analyses of unfiltered or total samples. The use of data from analyses of total samples is consistent with current EPA guidance (RAGS) but introduces an additional uncertainty to the BHHRA for some water use pathways. The magnitude of the effect of this uncertainty upon the risk estimates is difficult to determine because it is not known to what extent the quality of water (in terms of total solids) from a residential well could vary from the quality of water collected during the recent sampling effort; however, because the samples used in this BHHRA came from both monitoring wells and boreholes, some samples did have high solid content. Exhibit 1.48 addresses these groundwater issues by comparing residential use risk estimates calculated using results from all unfiltered groundwater samples and the subset of 0.45μ filtered samples.

	All unfiltered	water samples	0.45 µ Filtered	water samples only
Site	HI	ELCR	HI	ELCR
Future industrial worker				
SWMU 99a (RGA)	27.4	1.3E-3	< 1	ND
SWMU 193a (RGA)	< 1	1.9E-5	ND	ND
SWMU 103a (McN)	< 1	1.4E-6	ND	ND
SWMU 193b (RGA)	< 1	2.0E-5	ND	ND
AOC 204 (RGA)	2.44	5.1E-5	ND	ND
Future on-site rural resident				
SWMU 99a (RGA)	314	> 1E-2 ^a	2.56	ND
SWMU 193a (RGA)	5.25	2.7E-3	ND	ND
SWMU 193a (McN)	< 1	7.4E-4	ND	ND
SWMU 193b (RGA)	18.4	3.8E-4	ND	ND
AOC 204 (RGA)	75.8	7.6E-4	ND	ND

Exhibit 1.48. Comparison of filtered and unfiltered	d water sample	5
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Notes: HI is for the child for the future on-site rural resident scenario.

All HI estimates exclude lead as a COPC. Only current data are used in this analysis.

ND = No Data.

* The absolute value is omitted because a risk of > 1E-2 as calculated by the linearized multistage model may be imprecise.

Though insufficient filtered groundwater specimens were available to allow a thorough WAG 28wide comparison to be made, the HI estimates calculated using unfiltered water from all samples of RGA groundwater at SWMU 99a differed from HI estimates calculated using only filtered samples by more than one order of magnitude (Exhibit 1.48). These results infer that the risk estimates could be markedly different if concentrations from filtered samples had been used in the risk assessment. This conclusion contrasts with that reached in the earlier uncertainty analysis in Baseline Risk Assessment and Technical Investigation Report for the Northwest Dissolved Phase Plume, Paducah Gaseous Diffusion Plant (DOE 1994, page 5-95), where it was determined that the risks from the total and filtered concentrations of manganese, a primary risk driver in that assessment, differed by less than an order of magnitude. In summary, the uncertainty in water sampling is at least moderate in this assessment depending upon which comparisons are investigated.

1.6.1.5 Use of concentrations from boreholes versus monitoring wells

Exhibit 1.49 presents the determinations of SWMU-specific RGA groundwater risks and hazards when the data are broken out according to the means of collection (i.e., borehole probe versus monitoring wells). Where sufficient data were available to make such a comparison (i.e., for SWMUs 99a and 193a), no prevailing trends could be discerned among the data that would suggest an effect related to sampling strategy. For example, for each site, the total ELCRs for the borehole probe samples were closely similar to those obtained from the monitoring wells. By contrast, the SWMU-specific HIs differed markedly according to the sample collection technique, though with no obvious bias. Accordingly, the potential impact of the means of collecting the groundwater samples (boreholes versus monitoring wells) on the overall risks and hazards when all classes of contaminants are considered together is impossible to determine from the HIs but is likely to be small based on the ELCR data. Restricting this analysis to the effect of metals might have resulted in more substantial and consistent differences among the samples because of their greater contribution to turbidity.

1.6.1.6 Excluding "hot-spot" soil samples from risk characterization of SWMU 99a

Exhibit 1.50 demonstrates the effect on the risk calculations of excluding two surface soil samples from the overall determination of cancer risk and systemic toxicity for receptors exposed to soil at SWMU 99a. The two samples (082-014 and 082-015) were obtained during the excavation of a collapsed drainpipe at the southwest corner of SWMU 99a; therefore, these two samples potentially represent contaminant transport via surface runoff from the Classified Scrap Yard and may be atypical of the waste management practices and prevailing levels of contamination at SWMU 99a. Sample (082-014) displayed high radioactivity (2730 pCi/g for total beta activity), ascribed to the presence of technetium-99.

For this SWMU, the radionuclide is a risk driver for the hypothetical future on-site rural resident, most notably when the receptor is exposed to biota growing in soil contaminated at current levels; however, Exhibit 1.50 shows that, for most receptors and exposure scenarios, few if any significant differences in the overall determinations of risk or hazard are evident, whether the contribution of this "hot-spot" value to the overall CDI is included or excluded. By contrast, the overall levels of risk to the future on-site rural resident differ by a factor of approximately 50 (> 1E-2 versus 3.2E-3), driven primarily by the pathway featuring consumption of vegetables grown in contaminated soil. Overall, small to moderate uncertainty is associated with the risk characterization from including these samples as surface soil from SWMU 99a, the variability depending on the exposure scenario and operative pathways.

· •	All borehole	probe samples	All wel	l samples
Site	HI	ELCR	HI	ELCR
Future industrial worker	•			
SWMU 99a (RGA)	6.74	4.0E-4	1.54	3.3E-4
SWMU 99a (McN)	2.44	5.1E-5	ND	ND
SWMU 99b (RGA)	ND	ND	7.00	2.6E-4
SWMU 193a (RGA)	< 1	1.8E-5	8.26	1.5E-4
SWMU 193a (McN)	< 1	1.0E-6	4.44	5.8E-7
SWMU 193b (RGA)	1.74	4.4E-5	ND	ND
SWMU 193b (McN)	<1	8.4E-7	ND	ND
SWMU 193c (RGA)	1.46	1.1E-5	ND	ND
AOC 204 (RGA)	2.44	5.1E-5	ND	ND
Future on-site rural resid	ient			
SWMU 99a (RGA)	122	4.5E-3	37.9	4.6E-3
SWMU 99a (MCN)	53.1	1.8E-3	ND	ND
SWMU 99b (RGA)	ND	ND	208	2.3E-3
SWMU 193a (RGA)	4.12	2.6E-3	237	2.9E-3
SWMU 193a (McN)	10.7	4.4E-4	49.9	8.5E-6
SWMU 193b (RGA)	55.5	1.0E-3	ND	ND
SWMU 193b (McN)	2.69	1.2E-5	ND	ND
SWMU 193c (RGA)	80.7	1.5E-4	ND	ND
AOC 204 (RGA)	75.8	7.6E-4	ND	ND

Exhibit 1.49. Comparison of the total HIs and ELCRs from groundwater samples recovered from borehole probes versus monitoring wells with lead excluded as a COPC

Notes: HI is for the child for the future on-site rural resident scenario. All HI estimates exclude lead as a COPC. ND = No Data.

Scenario	HI + "hot-spots"	HI - "hot-spots"	ELCR + "hot-spots"	ELCR - "hot-spots"
Current/future industrial worker	<1	<1	3.1E-4	2.6E-4
Future excavation worker	1.46	1.46	2.1E-4	1.7E-4
Future recreational worker	< 1	< 1	2.7E-6	2.4E-6
Future on-site rural resident	17.2	14.0	> 1E-2 ^a	3.2E-3

Exhibit 1.50. Comparison of ELCRs and HIs with and without "hot-spot" samples excluding lead as a COPC

Notes: HIs for the future on-site rural resident are for a child

^a The absolute value is omitted because a risk of > 1E-2 as calculated by the linearized multistage model may be imprecise.



The potential impact on human health that could arise from contact with the contaminants (predominantly radionuclides) at locations 082-014 and 082-015 was further analyzed by comparing the concentrations/activities of contaminants in each sample individually with (1) surface soil background levels, (2) industrial human health risk-based screening criteria, and (3) residential soil screening levels for the protection of groundwater. As illustrated in Exhibit 1.51, one or more contaminant-specific benchmark values are exceeded for each screening category evaluated.

Retained analytes after comparison to benchmarks				
Analytes	Background	Industri		
082-014	(surface soil)	ELCR	HI	Residential SSLs
PCB-1016	ND	exceeds	complies	exceeds
α-activity	ND	ND	ND	ND
β-activity	ND	ND	ND	ND
Cesium-137	Exceeds	exceeds	ND	ND
Neptunium-237	Exceeds	exceeds	ND	ND
Technetium-99	Exceeds	exceeds	ND	ND
Thorium-234	ND	exceeds	ND	ND
Uranium-234	ND	complies	ND	ND
Uranium-238	Exceeds	exceeds	ND	ND
082-015				
α-activity	ND	ND	ND	ND
β-activity	ND	ND	ND	ND

Exhibit 1.51 Screen of individual "hot-spot" samples at SWMU 99a against background concentrations/activities, industrial use risk-based concentrations, and EPA residential soil screening levels for protection of groundwater

Notes: ND = No Data. (A contaminant-specific benchmark value was unavailable for this parameter.)

1.6.2 Uncertainties Associated with Exposure Assessment

Uncertainties associated with the exposure assessment are from five sources, namely (1) biota fate and transport modeling, (2) use of the RME scenario, (3) development of the conceptual site model and selection of pathways, (4) use of default values when estimating dermal absorbed dose, and (5) use of conservative exposure values for the excavation worker and industrial worker scenarios. Each of these uncertainties is discussed in the following paragraphs.

1.6.2.1 Uncertainties in biota fate and transport modeling

Modeling was used to estimate chemical concentrations and radionuclide activities in biota. Although the models used in this assessment are industry standards (Methods Document), their output contains a considerable amount of uncertainty. To ensure that these models generated values that were unlikely to underestimate dose (i.e., were conservative values), default modeling parameters were used in all cases; however, the use of conservative values may result in risk estimates that overestimate the actual risk.

To examine this uncertainty, risk estimates that included and omitted the biota exposure routes were compiled. Exhibit 1.52 displays these results, in which all estimates of HI are for the future child on-site rural resident, and the effects of lead have been removed. This exhibit shows that the effect of this uncertainty on the assessment is small for groundwater and soil at most sites; however, at SWMU 99a, the

surface soil ELCRs differ by more than two orders of magnitude when values with and without the biota_ pathways are compared (> 1E-2 versus 1.1E-3). Notwithstanding this exception, most media-specific HIs and ELCRs for the future on-site rural resident differ by less than an order of magnitude when the biota pathways are excluded.

·		ural resident with ngestion	Future on-site rural resident without biota ingestion	
Site	HI	ELCR	HI	ELCR
SWMU 99a (RGA)	97.3	5.6E-3	76.6	4.1E-3
SWMU 99a (McN)	53.1	1.8E-3	45.3	1.5E-3
SWMU 99a (soil)	17.2	$> 1E-2^{a}$	3.24	1.1E-3
SWMU 99b (RGA)	208	2.3E-3	178	1.8E-3
SWMU 193a (RGA)	28.6	2.4E-3	21.7	2.0E-4
SWMU193a (McN)	59.9	4.2E-4	38.2	6.2E-6
SWMU 193a (soil)	6.25	7.1E-4	2.57	4.9E-5
SWMU 193b (RGA)	55.5	1.0E-3	47.4	5.8E-4
SWMU 193b (McN)	2.69	1.2E-5	2,23	9.4E-6
SWMU 193b (soil)	66.7	3.0E-3	31.2	1.5E-3
SWMU 193c (RGA)	80.7	1.5E-4	44.5	1.2E-4
SWMU 193c (McN)	103	4.0E-3	64.9	2.4E-3
SWMU 193c (soil)	3.04	1.1 E-9	1.16	1.1E-9
AOC 204 (RGA)	279	$> 1E-2^{a}$	204	8.9E-3

Exhibit 1.52. Effect of omitting biota pathways

Note: HI values are for the child, with lead excluded as a COPC.

* The ELCR is approximate because the linearized multistage model returns imprecise values at risks > 1E-2.

The effect of this uncertainty on the results for the future recreational user exposed to game foraging in soil is much greater. As discussed in Sect. 1.3, the only pathways assessed for the future recreational user exposed to game foraging in soil were the biota pathways; therefore, the recreational land use scenario would not have been evaluated for any site if the biota pathways were ignored.

1.6.2.2 Uncertainties in use of reasonable maximum exposure scenarios

For each exposure pathway modeled, assumptions were made about the number of times per year an activity could occur, routes of exposure, and rate of intake of contaminated media. Because site-specific data were not available for many parameters, EPA and Commonwealth of Kentucky defaults were used (Methods Document). Because most of these defaults are conservative to prevent the underestimation of risk, the resulting risk estimates tend to be conservative. Generally, when several upper bound values are combined, the derived value tends to exceed the level of exposure that may be reasonable at a site. In consideration of this problem, attention should be focused not on the fact that any individual dose model is overly conservative, because most are not, but on the fact that if results from several conservative dose models are combined, the resulting total dose may be overestimated.

To examine the potential effect of this uncertainty, risks for the residential scenario were estimated using average values for all exposure parameters. All exposure parameters used in this assessment were taken from the preliminary review draft of *EPA*'s Superfund's Standard Default Exposure Factors for the Central Tendency and Reasonable Maximum Exposure, Preliminary Review Draft (EPA 1993d).

Appendix G of this volume presents this report. In this assessment, the exposure pathways evaluated were identical to those used in the RME scenario. Similarly, the exposure equations, chemical concentrations, radionuclide activities, and toxicity values were identical to those used for the RME scenario. The results of this assessment are presented in Exhibit 1.53, in which, as noted, the HIs are for child exposures. HIs based on average exposure defaults differed by less than a factor of two from those based on RME values, while ELCRs developed from average exposure defaults differed from RME-based estimates by a factor of approximately five. Therefore, the effect of this uncertainty was small for estimates of systemic toxicity and ELCR.

Site	HI (RME)	HI (average)	ELCR (RME)	ELCR (average)
SWMU 99a (RGA)	97.3	71.8	5.6E-3	1.2E-3
SWMU 99a (McNairy)	53.1	37.1	1.8E-3	3.8E-4
SWMU 99a (soil)	17.2	11.4	> 1E-2 ^a	> 1E-2ª
SWMU 99b (RGA)	208	146	2.3E-3	4.9E-4
SWMU 193a (RGA)	28.6	21.5	2.4E-3	5.0E-4
SWMU 193a (McNairy)	59.9	48.1	4.2E-4	8.6E-5
SWMU 193a (soil)	6.25	4.16	7.1E-4	1.5E-4
SWMU 193b (RGA)	55.5	38.7	1.0E-3	2.2E-4
SWMU 193b (McNairy)	2.69	1.88	1.2E-5	2.7E-6
SWMU 193b (soil)	66.7	44.4	3.0E-3	6.3E-4
SWMU 193c (RGA)	80.7	55.5	1.5E-4	3.3E-5
SWMU 193c (McNairy)	103	83.9	4.0E-3	9.0E-4
SWMU 193c (soil)	3.04	2.02	1.1E-9	2.3E-10
AOC 204 (RGA)	279	213	> 1E-2ª	3.3E-3

Exhibit 1.53. Comparison of results using average exposure parameters to calculate dose versus RME parameters to calculate dose for the future on-site rural resident

Notes: All HIs are for the child, with lead excluded as a COPC.

^a The ELCR is approximate because the linearized multistage model returns imprecise values at risks > 1E-2.

1.6.2.3 Uncertainties related to development of the site conceptual models and selection of pathways

Generally, the level of uncertainty in the development of site conceptual models is small. Data used to develop these models were from several previous investigations of the site and from local experts; however, some of the uncertainties related to specific scenarios deserve additional explanation. These uncertainties are the consideration or lack of consideration of specific pathways for some scenarios, the lack of consideration of a separate intruder/infrequent recreational user scenario, and the summation of risks across areas and across scenarios.

An uncertainty related to assessment of specific pathways is the consideration of groundwater ingestion by the future industrial worker and future rural resident. Use of groundwater as drinking water and water for showering was assumed in the assessment. These exposure routes were included to provide risk managers with additional information about the potential risk posed by groundwater at WAG 28 sites; however, PGDP presently does not use groundwater, and there are no plans to use groundwater at the site in the future.

In this assessment surface soil samples were not evaluated at SWMU 194, SWMU 99b, and AOC 204. Neither were groundwater samples taken from SWMU 194. Thus, it was not possible to

evaluate risk from combined exposure to surface soil and groundwater at every site; however,... Exhibit 1.54 addresses this issue for WAG 28 sites with available data by reporting the sum of risks from soil and groundwater exposures for the future industrial worker and future rural resident (HIs do not include lead as a COPC.) As shown in this exhibit, the effect of this uncertainty is small.

Another uncertainty related to specific pathways is the lack of consideration of ingestion of livestock or products from livestock raised in contaminated areas. If the industrial infrastructure was removed, some WAG 28 sites are large enough to supply sufficient pasture for beef and dairy cows. Based on the results of other risk assessments performed for PGDP (DOE 1994), incorporation of this pathway into the risk characterization would have increased the risk to the rural resident at this unit; however, because the ECLR and systemic hazard to the rural resident at these units are already very high, the incorporation of the livestock pathway into the risk characterization would not have changed the final selection of the rural resident as a land use scenario of concern at this unit. The effect of this uncertainty for all units is small.

The lack of consideration of a separate intruder/infrequent recreational user scenario in the risk assessment did not impact the results reported in the risk characterization because the results from this scenario would have been reported separately. Direct exposure by a recreational user to contaminated media was assessed, and the addition of a separate intruder/infrequent recreational user scenario would add little to the risk results.

1.6.2.4 Uncertainties related to use of default values when estimating dermal absorbed dose

In this assessment, the default dermal absorption factors for soil provided by the Commonwealth of Kentucky in its *Risk Assessment Guidance* (KDEP 1995) were used in most cases because chemical-specific absorption values were unavailable. In this guidance, the absorption factors, which estimate the percentage of a contaminant in soil or sediment crossing the skin and entering the body, are 5 percent for inorganic compounds, 10 percent for semivolatile organic compounds, and 25 percent for volatile organic compounds. These factors are much higher than those recommended by EPA Region 4 (EPA 1995a), which are 0.1 percent for inorganic compounds and 1 percent for organic compounds.

The effect of using the Commonwealth of Kentucky's default values for dermal absorption versus the EPA Region 4 values is illustrated in Exhibit 1.55. This exhibit compares the systemic hazards and ELCRs from the dermal contact exposure route for WAG 28 sites when these estimates are derived using the Commonwealth of Kentucky's default values and EPA Region 4 values, respectively. Values in Exhibit 1.55 are for an excavation worker exposed to subsurface soil and for a current industrial worker exposed to surface soil, in each case without lead included as a COPC. As shown in this exhibit, the overall effect of this uncertainty at each SWMU is small to moderate for both ELCRs and HIs, which, when the EPA Region 4 values are used, fall within or below the EPA range of concern.

1.6.2.5 Uncertainties related to use of default values for the excavation worker and industrial worker exposure scenario

The Commonwealth of Kentucky guidance (KDEP 1995) recommends using 185 days per year and 25 years for the exposure frequency and the exposure duration, respectively, for the excavation worker. These values probably exceed the real values for WAG 28 because the excavation scenario typically represents a soil removal action associated with construction of a foundation or excavation of contaminated soil. Exhibit 1.56 compares the site-specific risks and hazards calculated using the KDEP exposure parameters to those derived using site-specific durations without lead as a COPC.



	F	uture industria	l worker ^a		
	McNairy	RGA	Soil	McNairy + soil	RGA + soil
ELCR					
SWMU 99a ^a	7.6E-5	5.6E-4	3.1E-4	3.9E-4	8.7E-4
SWMU 99b ^b	ND	2.6E-4	ND	NA	2.6E-4
SWMU 193a	1.1E-6	2.6E-5	1.5E-5	2.6E-5	4.1E-5
SWMU 193b	8.4E-7	4.4E-5	5.1E-4	5.1E-4	5.5E-4
SWMU 193c	4.2E-4	1.0E-5	1.7E-10	4.2E-4	1.0E-5
AOC 204	ND	1.3E-3	ND	NA	1.3E-3
HI		· · · · · · · · · · · · · · · · · · ·			
SWMU 99a ^a	1.64	5.11	<1	1.64	5.11
SWMU 99b ^b	ND	7.0	ND	NA	7.0
SWMU 193a	4.69	1.64	< 1	4.69	1.64
SWMU 193b	< 0.1	1.74	5.25	5.25	7.0
SWMU 193c	9.92	1.46	<1	9.92	1.46
AOC 204	ND	33.3	ND	NA	33.3
		Future rural re	esident ^b		
	McNairy	RGA	Soil	McNairy + soil	RGA + soil
ELCR					
SWMU 99a ^a	1.7E-3	5.6E-3	>1E-2°	>1E-2 ^c	> 1E-2°
SWMU 99b ^b	ND	2.3E-3	ND	NA	2.3E-3
SWMU 193a	4.1E-4	2.4E-3	7.1E-4	1.1E-3	3.1E-3
SWMU 193b	1.2E-5	1.0E-3	3.0E-3	3.0E-3	4.0E-3
SWMU 193c	4.0E-3	1.5E-4	1.1E-9	4.0E-3	1.5E-4
AOC 204	ND	>1E-2°	ND	NA	> 1E-2°
HI					
SWMU 99aª	53.1	97.3	17.2	70.3	114.5
SWMU 99b ^b	ND	208	ND	NA	208
SWMU 193a	59.9	28.6	6.25	66.2	34.9
SWMU 193b	2.69	55.5	66.7	69.4	122.2
SWMU 193c	103	80.7	3.04	106	11.7
AOC 204	ND	279	ND	NA	279

Exhibit 1.54. Presentation of groundwater risks plus soil risks for the future industrial worker and future on-site rural resident at WAG 28

Notes: All HIs exclude lead as a COPC.

ND = No Data.

NA = Not Applicable.

^a Results are taken from Exhibits 1.21 and 1.29.
 ^b Results are taken from Exhibits 1.23 and 1.31.
 ^c The ELCR is approximate because the linearized multistage model returns imprecise values at risks > 1E-2.

Site	HI (Kentucky)	HI (EPA)	ELCR (Kentucky)	ELCR (EPA)
•		Surface soil		
SWMU 99a	< 1	< 1	3.1E-4	6.7E-5
SWMU 193a	<1	< 1	1.5E-5	2.0E-6
SWMU 193b	5.25	< 1	5.1E-4	1.1E-5
SWMU 193c	< 1	< 1	1.7E-10	1.7E-10
	S	ubsurface Soil		
SWMU 99a	1.46	< 1	2.1E-4	8.8E-5
SWMU 99b	< 1	< 1	2.1E-4	1.5E-5
SWMU 193a	< 1	< 1	1.7E-4	1.1E-5
SWMU 193b	1.75	< 1	1.7E-4	7.1E-6
SWMU 193c	2.09	< 1	1.7E-4	6.9E-6
SWMU 194	< 1	< 1	3.1E-4	1.3E-5
AOC 204	<1	< 1	1.1E-6	8.0E-7

Exhibit 1.55. Effect of using Commonwealth of Kentucky defaults for dermal absorption versus EPA Region 4 defaults for the current industrial worker

For this BHHRA, the number of days and years to complete the excavation was set to maintain the exposure frequency as close to, but not over, 250 days per year, with the exposure duration set to maintain the smallest whole number of years possible. This was the most conservative approach. As shown, HIs fluctuate less than an order of magnitude for each site, although the effect of this uncertainty is more variable for the ELCRs. Thus, the effect of using KDEP versus site-specific exposure parameters for an excavation worker is small for systemic toxicity; however, examples of all categories of uncertainty (small, medium, and large) are evident in the ELCR comparisons.

Site	HI default	HI site-specific	ELCR default	ELCR site-specific
SWMU 99a	1.46	1.10	2.1E-4	1.3E-5
SWMU 99b	< 1	<1	2.1E-4	1.6E-6
SWMU 193a	< 1	<1	1.7E-4	7.4E-5
SWMU 193b	1.75	2.35	1.7E-4	1.8E-5
SWMU 193c	2.09	2.77	1.67E-4	3.6E-4
SWMU 194	< 1	<1	3.1E-4	3.3E-4
AOC 204	< 1	<1	1.1E-6	3.0E-7

Exhibit 1.56. Effect of using Commonwealth of Kentucky defaults versus site-specific exposure parameters for the future excavation worker

Notes: Values for the HI are with lead excluded as a COPC.

Another uncertainty affecting the excavation worker scenario concerns the toxicity values used for the scenario. For the excavation worker calculations, toxicity values based on chronic exposure were used. By definition, chronic exposures are those longer than 7 years in length, and subchronic exposures are those less than 7 years in length (RAGS). For the excavation worker scenario, toxicity values based on subchronic exposure may have been more appropriate; however, chronic values were used for the excavation scenario to remain consistent with KDEP exposure duration (i.e., 25 years) and because subchronic values are lacking for many chemicals. Because the differences between subchronic and chronic toxicity values for systemic toxicity are one order of magnitude (RAGS) or less, the effect of this uncertainty on the risk assessment is small. For this assessment, site-specific exposure parameters for the current industrial worker were not ... used, although site-specific exposure parameters for general site maintenance (16 days a year for exposure frequency and 25 years for exposure duration) have been estimated for other locations at PGDP. Exhibit 1.57 presents a comparison of risk results using KDEP default exposure parameters and PGDP site-specific exposure parameters, with lead excluded as a COPC as before. As shown, the effect of using KDEP exposure parameters for a current industrial worker is small to moderate for ELCR and systemic toxicity; however, of particular note in this analysis is that the site-specific ELCRs now fall within or below EPA's range of concern.

SWMU	HI default	HI site-specific	ELCR default	ELCR site-specific
SWMU 99a (soil)	< 1	<1	3.1E-4	2.0E-5
SWMU 193a (soil)	< 1	< 1	1.5E-5	9.9E-7
SWMU 193b (soil)	5.25	< 1	5.1E-4	3.3E-5
SWMU 193c (soil)	< 1	< 1	1.7E-10	1.1E-11

Exhibit 1.57.	Effect of using Commonwealth of Kentucky defaults versus site-specific
	exposure parameters for the current industrial worker

Notes: All HIs are calculated with lead excluded as a COPC.

While Exhibit 1.57 shows that risks are decreased when exposure at the sites is set to the rates used for general maintenance of other sites at PGDP, these risks may not represent the actual risks associated with exposure at WAG 28 sites because the potential rates of exposure may differ from site to site, based on differing use patterns. For example, SWMUs 99a and 193c are UF₆ cylinder yards at which work may be occurring on most days throughout the year. Similarly, as grassy perimeters to buildings, SWMUs 193a and 193b offer ready though unspecified access to workers on a daily basis. Thus, the risks and hazards calculated using the Commonwealth of Kentucky defaults (250 days a year for exposure frequency and 25 years for exposure duration) may reflect the more accurate use pattern and resulting ELCR/HI.

1.6.3 Uncertainties Associated with Toxicity Assessment

Uncertainties related to the toxicity assessment are from the following three sources: uncertainty because of lack of toxicity values for some chemicals, uncertainty in the calculation of toxicity values by EPA, and uncertainty in the calculation of absorbed dose toxicity values from administered dose toxicity values. Each of these is discussed in the following paragraphs.

1.6.3.1 Uncertainties because of lack of toxicity values for some chemicals

Uncertainties due to lack of toxicity values for some chemicals result from two sources in this BHHRA; these are the uncertainty from the use of provisional or withdrawn values and the uncertainty from extrapolating a toxicity value for an administered dose (oral) to an inhalation dose.

The uncertainty from the use of provisional or withdrawn values has a significant effect on the results of the BHHRA. Some COPCs do not have approved toxicity values, so a provisional or withdrawn value is used. The most notable of these COPCs is lead. This provisional reference dose toxicity value was provided by KDEP in a comment package on the WAG 17 RI/BRA. As discussed in Sect. 1.5, the systemic toxicity posed by lead dominates all land use scenarios at those sites where lead was detected. For better interpretation of the HIs for the rest of the COPCs in the BHHRA, results with and without contributions from lead are provided throughout this BHHRA. Generally, there is no consistent pattern to

the effect of using these provisional and withdrawn values on the final risk estimates; however, while most risk and hazard estimates fluctuate within narrow limits, the risk to some receptors at certain sites differes by several orders of magnitude. For example, risk to an excavation worker exposed to subsurface soil at SWMUs 193b, 193c, and 194 is reduced by six orders of magnitude when contaminants with provisional or withdrawn toxicity values are excluded from the risk assessment. Table 1.86 presents these results.

In the past, there was uncertainty in the selection of the appropriate toxicity value for PCBs (e.g., Aroclor 1254, 1260, etc.) because of (1) difficulty in identifying specific Aroclors in a mixture, (2) different rates of decay among the Aroclors in environmental media, and (3) the effects of weathering processes on the congener-specific "fingerprint" over time, a process making the Aroclors appear to be more chlorinated than they really are. To address these concerns and to ensure that the risk numbers for Aroclors are suitably conservative, KDEP requires that all PCBs be evaluated as Aroclor 1260. This assessment is consistent with KDEP guidance because oral slope factors for all Aroclors were assumed to be equal to 2.0 (mg/kg-day)⁻¹, consistent with recent EPA guidance (EPA 1996c). Results for exposures to multiple Aroclors are summed to generate a total PCB-specific value; therefore, unlike earlier assessments performed at PGDP in which the effect of uncertainty in the selection of toxicity values for PCBs on the final risk values may have been moderate, the effect of this uncertainty in the current assessment is likely to be small.

Including inhalation toxicity values extrapolated from toxicity values based on orally administered doses in the risk characterization does not significantly affect the results of the BHHRA. EPA guidance does not recommend extrapolating between oral and inhalation toxicity values (RAGS) because of the differing path a chemical entering through the lungs must follow before exerting its effect compared to that of a chemical entering via the gut; however, examination of this form of extrapolation as an uncertainty in assessments for PGDP was requested by the regulatory community. Previous work at PGDP, in which this effect was examined quantitatively, determined that including extrapolated inhalation toxicity values in the risk characterization resulted in insignificant changes in the final risk estimates; therefore, the estimated effect of this uncertainty on risk results is small.

1.6.3.2 Uncertainties in deriving toxicity values

Standard EPA RfDs and slope factors were used to estimate potential noncarcinogenic and carcinogenic health effects from exposure to chemicals. Considerable uncertainty is associated with the method applied to derive slope factors and RfDs. EPA has working groups that review all relevant human and animal studies for each compound and select studies pertinent to the derivation of the specific RfD and slope factor. These studies often involve data from experimental studies in animals, high exposure levels, and exposures under acute or occupational conditions. Extrapolation of these data to humans under low-dose, chronic conditions introduces uncertainties. The magnitude of these uncertainties is addressed by applying uncertainty factors to the dose response data for each applicable uncertainty. These factors are incorporated to provide a margin of safety for use in human health risk assessments. The effect of uncertainties in calculation of chemical toxicity values is moderate.

Unlike the uncertainty associated with chemical toxicity values, the uncertainty associated with radionuclide toxicity values is small. The dose-response relationship between cancer and ionizing radiation has been evaluated in many reports and is well established. In addition, unlike toxicity values for chemicals, risk factors for radionuclides are extrapolated from the cancer risk established using the Japanese Atomic Bomb Survivors database and a relative risk projection model; therefore, these values are based on human data and are expected to contribute minimally to uncertainty.

1.6.3.3 Uncertainties due to calculation of absorbed dose toxicity values from administered dose toxicity values

Uncertainty exists in the validity of the calculations used to convert an administered dose toxicity value to an absorbed dose. Of greatest importance is the lack of consideration of point-of-contact effects in this calculation. For example, some organic analytes (e.g., PAHs) can cause a toxic or carcinogenic response in skin. This effect is not considered in the calculation of absorbed dose toxicity values from administered dose toxicity values using EPA protocols. Similarly, the administered dose response for many chemicals relies on the delivery of a high concentration of contaminants to the liver via the portal system after ingestion. This effect is not seen if a contaminant is absorbed through the skin because of the larger distribution space for the contaminant absorbed through the skin; however, even with these uncertainties, the effect of the uncertainty in calculation of absorbed dose toxicity values from administered dose toxicity values upon the risk estimates is estimated to be small.

1.6.4 Uncertainties Associated with Risk Characterization

Two uncertainties are related to risk characterization. The first is the method used to combine HQs and chemical-specific ELCRs over pathways and combine pathway HIs and ELCRs to calculate total HI and ELCR. The second is the uncertainty added to the assessment by combining risks from chemicals and radionuclides. These uncertainties are discussed in the following sections.

1.6.4.1 Combining chemical-specific risk values and pathway risk values

The primary uncertainty in risk characterization is the method used to combine HQs and chemicalspecific ELCRs across pathways and to combine pathway HIs and ELCRs to calculate total HI and ELCR.

The method used to calculate pathway HIs and ELCRs in the BHHRA follows EPA protocols (RAGS, Methods Document). This guidance calls for the simple addition of HQs and chemical-specific ELCRs to calculate pathway HIs and ELCRs, respectively. This method assumes that all effects between chemicals are additive. EPA makes this assumption because information concerning the effect of chemical mixtures is lacking. The following limitations of this approach for systemic toxicity effects have been reported by EPA in RAGS:

- Little is known about the effects of chemical mixtures; although additivity is assumed, the interaction of multiple chemicals could possibly be synergistic or antagonistic.
- The RfDs and RfCs do not have equal accuracy or precision and are not based on the same severity of effects.
- Dose additivity is most properly applied to compounds that induce the same effect by the same mechanism of action. While the approach recommended by EPA is a useful screening-level approach, the cumulative systemic toxicity could be overestimated for chemicals that act by different mechanisms and/or on different target organs.

The effect of this uncertainty on the estimate of systemic toxicity depends on how many contaminants drive systemic toxicity and whether the contaminants have different endpoints. In this BHHRA, many contaminants do drive systemic toxicity for most scenarios, and these contaminants do have differing endpoints; however, as shown in Exhibits 1.18–1.36, individual contaminants alone contribute significant levels of risk for some exposure scenarios. The effect of this uncertainty on HIs is small.

EPA has reported specific limitations for this approach in regard to chemical carcinogenesis. (RAGS):

- Cancer risks (i.e., ELCRs) are based on slope factors that represent an upper 95th percentile estimate of potency; the upper 95th percentiles of probability distributions are not strictly additive. Summing these risks can result in an overly conservative estimate of lifetime ELCR.
- Cancer risks may not be additive. By analogy to systemic toxicity effects, the endpoints may differ, and mechanisms of effect may vary.
- Not all slope factors contain the same weight-of-evidence for human carcinogenicity. As explained in Sect. 1.4, EPA recognizes this by placing weight-of-evidence classifications on all slope factors. Those contaminants with a weight-of-evidence classification of A should probably receive more attention in the selection of a remedial design than contaminants with a B or C classification. Similarly, a contaminant with a B classification should probably receive greater attention than one with a C classification. The simple combination of ELCRs does not take this hierarchy into account.

The uncertainties involved in combining chemical-specific ELCRs and pathway ELCRs are considerable; however, the effect of these uncertainties on the total ELCRs presented in the BHHRA is small because a single chemical dominates the pathway ELCR for most pathways at some SWMUs. In such circumstances, the potential effect of mixtures is reduced.

1.6.4.2 Combining risks from chemicals with those from radionuclides

Uncertainty associated with adding risks from chemical exposure to those from exposure to radionuclides arises from two sources. First, as noted in Sect. 1.4, the slope factors used to characterize the risk from chemicals are derived differently from the slope factors used to characterize risk from radionuclides. This difference may result in estimates of chemical exposure risks that may be considered to be upper-bound risk estimates and estimates of radionuclide exposure risks that may be considered to be central tendency (i.e., "best") estimates; therefore, combining chemical exposure and radionuclide exposure risk estimates to estimate total risk for a land use scenario may place too much emphasis on chemical exposure risk. Second, the mechanism by which chemicals may cause cancer may vary from the mechanism by which radionuclides may cause cancer (see Sect. 1.4). This difference in mechanism of action inflates the uncertainties discussed in Sect. 1.6.4.1 that assume cancer risks are additive. Overall, the effect of this uncertainty on the total risk value for each land use scenario is small because, as discussed in Sect. 1.6.4.1, generally one COC drives the risks at the sites assessed. At sites where there are multiple chemicals and radionuclides driving risk, the effect of this uncertainty could be moderate.

1.6.5 Summary of Uncertainties

As shown in the previous sections, risk estimates may vary if different assumptions are used in deriving risk estimates or if better information is available for some parameters. The following text summarizes the estimated effects of each uncertainty mentioned previously.

Two uncertainties with an effect estimated to be large are the use of the provisional toxicity values for lead systemic toxicity and use of KDEP default values when calculating dermal absorbed dose for total HI. Because the uncertainty regarding the RfD for lead was identified as being large and easy to quantify, the summary discussions in this RI are more detailed than for some of the other uncertainties. This discussion is not meant to imply that the authors believe the provisional toxicity value for lead provided by the KDEP is incorrect.



Another uncertainty considered to be large at some sites is the use of site-specific exposure values on systemic toxicity and ELCR for the excavation worker.

Following is a list of uncertainties with effects estimated to be moderate:

- underestimation of risk due to migration of groundwater to off-site receptors
- use of total water samples versus filtered
- calculation of toxicity values for chemicals
- combination of chemical and radiological ELCRs
- use of quantitation limits that exceed human health RBCs
- use of groundwater data from samples collected from borehole versus monitoring wells
- inclusion of biota exposure pathways
- use of KDEP dermal absorption values instead of EPA values on the total systemic toxicity and ELCR
- use of site-specific exposure values on systemic toxicity and ELCR for the current industrial worker
- use of provisional and withdrawn toxicity values on the systemic toxicity and ELCR
- exclusion of "hot-spot" soil samples from the risk characterization of SWMU 99a
- use of site-specific exposure values on systemic toxicity and ELCR for the excavation worker
- determination of chemical toxicity values

Following is a list of uncertainties with effects estimated to be small:

- inclusion of infrequently detected COPCs
- inclusion of infrequently analyzed for COPCs
- lack of determination of temporal patterns in data
- use of quantitation limits that exceed human health RBCs
- inclusion of common laboratory contaminants in the data
- lack of analyte comparison to blanks
- contribution of analytes removed based on a toxicity screen
- removal of analytes based on comparison to background values
- lack of approved groundwater background concentrations
- determination of exposure points for current concentrations
- determination of exposure points for future concentrations
- inclusion of biota exposure pathways
- use of RME default exposure values instead of central tendency exposure values
- evaluation of groundwater separately from soil in future land use scenarios
- omission of livestock in future rural resident land use scenario
- omission of an intruder/infrequent recreator land use scenario
- use of KDEP dermal absorption values instead of EPA values on the total systemic toxicity and ELCR
- use of site-specific exposure values on systemic toxicity and ECLR for the excavation worker
- use of site-specific exposure values on systemic toxicity and ELCR for the current industrial worker
- use of chronic toxicity values for the excavation worker land use scenario
- use of provisional and withdrawn toxicity values on the total systemic toxicity and ELCR
- selection of toxicity values for PCBs
- use of inhalation toxicity values extrapolated from oral toxicity values
- determination of radionuclide toxicity values
- use of absorbed toxicity values calculated from administered toxicity values

- combination of risk from chemicals and radionuclides in pathways
- combination of pathway risks to determine land use scenario risk
- use of groundwater data from samples collected from boreholes versus monitoring wells
- exclusion of "hot-spot" soil samples from the risk characterization of SWMU 99a

These uncertainties are summarized in Table 1.87.

1.7 CONCLUSIONS AND SUMMARIES

This section summarizes the results of the risk assessment and draws conclusions from the results. The primary purpose of this section is to provide a concise summary of each of the risk assessment steps without the use of tables, extensive explanations, or justifications. This section also includes a series of observations in which the results of the risk assessment are combined with the uncertainties in the risk assessment.

1.7.1 Chemicals of Potential Concern

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COPCs were selected from data collected in the recently completed WAG 28 field investigation and previous investigations. This data set was screened to produce a final list of COPCs ordered by medium and depth of sampling. The media considered were soil and groundwater. The depths considered for soil were surface soil (samples collected from 0–1 ft bgs), subsurface soil (samples collected from 0–15 ft bgs), and other (samples collected from more than 16 ft bgs). For groundwater, the depths considered were uCRS groundwater, RGA groundwater, and McNairy Formation groundwater. Of these groups, one soil group and one water group are not assessed directly in the risk assessment; these are other soils and UCRS groundwater. While not assessed directly, these groups are assessed indirectly because they serve as sources of contamination to underlying groundwater in the contaminant transport modeling.

Through a series of screening steps that follow regulatory agency-approved procedures, the data sets were reduced to a list of COPCs for each medium and site.

SWMU 99a surface soil contains 29 COPCs (4 inorganics, 19 organics, and 6 radionuclides); subsurface soil contains 101 COPCs (11 inorganics, 84 organics, and 6 radionuclides); RGA groundwater contains 24 COPCs (18 inorganics, 4 organics, and 2 radionuclides); and McNairy groundwater contains 4 COPCs (4 organics).

SWMU 99b subsurface soil contains 5 COPCs (4 inorganics and 1 organic); RGA groundwater contains 10 COPCs (8 inorganics, 1 organic, and 1 radionuclide). Surface soil and McNairy groundwater were not assessed.

SWMU 193a surface soil contains 14 COPCs (1 inorganic and 13 organics); subsurface soil contains 16 COPCs (3 organics and 13 inorganics); RGA groundwater contains 12 COPCs (6 inorganics, 5 organics, and 1 radionuclide); and McNairy groundwater contains 6 COPCs (1 inorganic, 3 organics, and 2 radionuclides).

SWMU 193b surface soil contains 3 COPCs (3 inorganics); subsurface soil contains 3 COPCs (3 inorganics); RGA groundwater contains 8 COPCs (7 organics and 1 radionuclide); and McNairy groundwater contains 2 COPCs (2 organics).

SWMU 193c surface soil contains 3 COPCs (3 inorganics); subsurface soil contains 11 COPCs (10 inorganics and 1 organic); RGA groundwater contains 2 COPCs (2 organics); and McNairy groundwater contains 37 COPCs (21 inorganics, 15 organics, and 1 radionuclide).

SWMU 194 subsurface soil contains 7 COPCs (6 inorganics and 1 organic). Surface soil and groundwater were not assessed.

AOC 204 subsurface soil contains 6 COPCs (6 organics); RGA groundwater contains 9 COPCs (9 organics).



1.7.2 Exposure Assessment

Historical information and newly collected data were used to develop a conceptual site model for WAG 28 sites. After consideration of all data, the scenarios selected for assessment were the industrial worker, excavation worker, recreational user, and rural resident. The current land use scenario was determined to be industrial, and the most plausible future land use scenario was also determined to be industrial. Another future land use determined to be likely was excavation. A less likely future land use scenario was recreational. The least likely land use scenario was determined to be residential. Routes of exposure for each scenario are presented in the following text:

Current industrial worker

- ingestion of soil
- dermal contact with soil
- inhalation of vapors and particulates emitted from soil
- external exposure to ionizing radiation emitted from soil

Future industrial worker

- ingestion of soil
- dermal contact with soil
- inhalation of vapors and particulates emitted from soil
- external exposure to ionizing radiation emitted from soil
- ingestion of groundwater
- dermal contact with groundwater while showering
- inhalation of vapors emitted by groundwater while showering

Future excavation worker

- ingestion of soil
- dermal contact with soil
- inhalation of vapors and particulates emitted from soil
- external exposure to ionizing radiation emitted from soil

Future recreational user

- ingestion of venison
- ingestion of rabbit
- ingestion of quail

Future on-site rural resident

- ingestion of soil
- dermal contact with soil
- inhalation of vapors and particulates emitted from soil
- external exposure to ionizing radiation emitted from soil
- ingestion of groundwater
- dermal contact with groundwater while showering
- inhalation of vapors emitted by groundwater during household use

- inhalation of vapors emitted by groundwater while showering
- ingestion of vegetables

Off-site rural resident (at PGDP security fence)

- ingestion of groundwater
- dermal contact with groundwater while showering
- inhalation of vapors emitted by groundwater during household use
- inhalation of vapors emitted by groundwater while showering

After selection of the exposure routes, CDIs were calculated for each medium using standard exposure models. Most parameters used in models were default values; however, site-specific information, especially for the biota pathways, was included.

1.7.3 Toxicity Assessment

The toxicity values used in the risk assessment were those approved by EPA or recommended by KDEP. After toxicity information was compiled, it was determined that the majority of the COPCs had a toxicity value available for one or more routes of exposure.

1.7.4 Risk Characterization

Risks were characterized by combining the CDIs calculated during the exposure assessment with the toxicity values collected during the toxicity assessment. As a result of this characterization, it was determined that there were risks associated with exposure to soil and groundwater at sites within WAG 28. Where lead was present as a COPC, HIs for a receptor/land use combination were determined with the metal both included and excluded from the assessment (Exhibit 1.17). This approach reveals which sites pose a potential threat to human health in the absence of this element and permits the identification of other COPC drivers. Significant results of the risk characterization by area are presented in the following text.

1.7.4.1 Land use scenarios of concern

On-site land use scenario. Current and future industrial workers exposed to soil levels at SWMU 193c are subject to a significant threat of systemic toxicity (HI = 3620), due in large measure to the presence of lead; however, when lead is excluded from the assessment, the remaining contaminants at this site are present in insufficient quantities to characterize the industrial worker exposed to soil as a land use of concern (HI = 0.194). Industrial workers exposed to prevailing levels of contamination in soil at SWMUs 193a and 99a (where lead was undetected) are not subject to significant threat of systemic toxicity. Accordingly, this is not a land use of concern at these sites. By contrast, at another site where lead was undetected (SWMU 193b), industrial workers exposed to soil are subject to significant threat of systemic toxicity due to the cumulative effect of other contaminants (HI = 5.25).

For subsurface soil where lead is present, an exposed excavation worker represents a land use of concern at SWMUs 99a and 193c irrespective of whether lead is included in the assessment. By contrast, eliminating lead from consideration at SWMU 194 causes the HI to fall below the level of concern for this scenario (2190 to 0.57). Lead was undetected in the subsurface soil at other sites within WAG 28; however, an HI of 1.75 for this scenario is sufficient to characterize the excavation worker exposed to subsurface soil as a land use scenario of concern at SWMU 193b. The scenario is not a land use of concern at SWMU 193b, 193a, or AOC 204.



A future on-site resident child in contact with soil will be exposed to contaminants at levels sufficient to designate this exposure pathway as a land use of concern at all sites; however, the presence of lead in soil at SWMU 193c contributes to a markedly higher HI for this receptor compared to when the contaminant is excluded from the assessment (247,000 versus 3.04).

For current and future industrial workers exposed to soil at SWMUs 99a, 193a, and 193b (but not 193c), significant ELCRs have been computed, thereby designating this exposure route/receptor combination as a land use of concern. Similarly, the future excavation worker exposed to subsurface soil represents a land use of concern at all sites.

In this risk assessment, a number of exposure pathways feature exposure to contaminants in groundwater, principally on the part of a future industrial worker or hypothetical on-site rural resident. For the former receptor, modeled exposure to contaminants in groundwater designates this pathway as a land use of concern at all sites and formations other than McNairy groundwater at SWMU 193b. When lead is present (in the RGA aquifer at SWMU 99a or in McNairy groundwater at SWMU 193c), a profound impact on the HI is evidenced for this receptor (8150 versus 5.11 for RGA groundwater at SWMU 99a, and 25,100 versus 9.92 for McNairy groundwater at SWMU 193c). A similar overall pattern of threat of systemic toxicity to the future on-site rural resident is seen at all sites (including McNairy groundwater at SWMU 193b).

Exposure of the future industrial worker to groundwater represents a significant ELCR at all sites and aquifers except McNairy groundwater at SWMU 193b and at all sites without exception for the future on-site rural resident, thereby justifying these exposure pathway/receptor combinations as land uses of concern.

Most soil exposure pathways for the future recreational user are considered not to be land uses of concern with the exception of SWMU 193c, an exposure route representing a threat of systemic toxicity to this receptor, driven, in large part, by the presence of lead. Evidence of this exposure pathway as a borderline land use of concern is also provided by a significant ELCR to this receptor through exposure to soil at SWMUs 99a and 193a.

Off-site land use scenario. Residential use of RGA groundwater containing contaminants migrating from WAG 28 sites was determined to be a land use of concern.

1.7.4.2 Chemicals of concern

On-site land uses. There are 16 COCs for systemic toxicity in soil in all of WAG 28. Of these COCs, 12 are inorganic and 4 are organic compounds. There are no COCs for systemic toxicity in soil at SWMU 99b. There are 12 COCs for systemic toxicity in soil at SWMU 99a, 9 inorganics and 3 organics. There is one COC for systemic toxicity in soil at SWMU 193a, and it is an inorganic compound. There are three COCs for systemic toxicity in soil at SWMU 193b, all of which are inorganic compounds. There are eight COCs for systemic toxicity in soil at SWMU 193c, all of which are inorganic compounds. There are six COCs for systemic toxicity in soil at SWMU 194, all of which are inorganic compounds. There is one COC for systemic toxicity in soil at SWMU 194, all of which are inorganic compounds. There is one COC for systemic toxicity in soil at AOC 204, and it is an organic compound.

There are 26 COCs for systemic toxicity in RGA groundwater in all of WAG 28. There are 18 COCs for systemic toxicity in RGA groundwater at SWMU 99a, 15 inorganic and 3 organic compounds. There are five COCs for systemic toxicity in RGA groundwater at SWMU 99b, four inorganics and one organic compound. There are four COCs for systemic toxicity in RGA groundwater at SWMU 193a, two inorganic and two organic compounds. There are five COCs for systemic toxicity in RGA groundwater at SWMU 193a, two inorganic and two organic compounds. There are five COCs for systemic toxicity in RGA groundwater at SWMU 193b, all of which are organic compounds. There are two COCs for systemic toxicity in RGA

groundwater at SWMU 193c, both of which are organic compounds. There are seven COCs for systemic_toxicity in RGA groundwater at AOC 204, one inorganic and six organic compounds. Groundwater was not sampled at SWMU 194.

There are 26 COCs for systemic toxicity in McNairy groundwater in all of WAG 28. There are four COCs for systemic toxicity in McNairy groundwater at SWMU 99a, all of which are inorganic compounds. There are three COCs for systemic toxicity in McNairy groundwater at SWMU 193a, one inorganic and two organic compounds. There are two COCs for systemic toxicity in McNairy groundwater at SWMU 193b, both of which are organic compounds. There are 25 COCs for systemic toxicity in McNairy groundwater at SWMU 193b, both of which are organic compounds. There are 25 COCs for systemic toxicity in McNairy groundwater at SWMU 193c, 15 inorganic compounds, 9 organic compounds, and 1 radionuclide.

There are 24 COCs for ELCR in soil in all of WAG 28. There are 23 COCs for ELCR in soil at SWMU 99a, 2 inorganic compounds, 15 organic compounds, and 6 radionuclides. There are two COCs for ELCR in soil at SWMU 99b, both of which are inorganic compounds. There are six COCs for ELCR in soil at SWMU 193a, one inorganic and five organic compounds. There is one COC for ELCR in soil at SWMUs 193b, 193c, and 194, and it is an inorganic compound. Beryllium is a COC at all sites, except AOC 204. There is one COC for ELCR in soil at AOC 204, and it is an organic compound.

There are 15 COCs for ELCR in RGA groundwater in all of WAG 28. There are seven COCs for ELCR in RGA groundwater at SWMU 99a, two inorganic compounds, three organic compounds, and two radionuclides. There are two COCs for ELCR in RGA groundwater at SWMU 99b, one organic compound and one radionuclide. There are five COCs for ELCR in RGA groundwater at SWMU 193a, four organic compounds and one radionuclide. There are five COCs for ELCR in RGA groundwater at SWMU 193b, four organic compounds and one radionuclide. There are five COCs for ELCR in RGA groundwater at SWMU 193b, four organic compounds and one radionuclide. There are five COCs for ELCR in RGA groundwater at SWMU 193b, four organic compounds and one radionuclide. There are eight COCs for ELCR in RGA groundwater at SWMU 193c, and it is an organic compound. There are eight COCs for ELCR in RGA groundwater at AOC 204, all of which are organic compounds. Groundwater was not sampled at SWMU 194.

There are 16 COCs for ELCR in McNairy groundwater in all of WAG 28. There are three COCs for ELCR in McNairy groundwater at SWMU 99a, all of which are organic compounds. There are three COCs for ELCR in McNairy groundwater at SWMU 193a, one organic compound and two radionuclides. There is one COC for ELCR in McNairy groundwater at SWMU 193b, and it is an organic compound. There are 14 COCs for ELCR in McNairy groundwater at SWMU 193c, two inorganic compounds, 11 organic compounds, and one radionuclide.

Considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs can be considered "priority COCs" (COCs with a chemical-specific HI or ELCR that exceeds 1 or 1E-4, respectively) in soil for the current use and most likely future use scenarios (i.e., industrial use):

- SWMU 99a—beryllium
- SWMU 99b—surface soil not assessed
- SWMU193a none
- SWMU 193b—chromium, vanadium, and beryllium
- SWMU 193c-lead
- SWMU 194—surface soil not assessed
- AOC 204—surface soil not assessed

Considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs can be considered "priority COCs" (COCs with a chemical-specific HI or ELCR that exceeds 1 or 1E-4, respectively) in soil for the future excavation worker:

- SWMU 99a—lead
- SWMU 99b—beryllium
- SWMU193a—beryllium
- SWMU 193b—beryllium and chromium
- SWMU 193c—beryllium and lead
- SWMU 194—beryllium and lead
- AOC 204—none

Considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs can be considered "priority COCs" (COCs with a chemical-specific HI or ELCR that exceeds 1 or 1E-4, respectively) in soil for the future on-site rural resident:

- SWMU 99a—barium, beryllium, chromium, benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, dibenz(a,h)anthracene, indeno(1,2,3,-cd)pyrene, PCB-1016, PCB-1254, neptunium-237, technetium-99, thorium-234, uranium-234, and uranium-238
- SWMU 99b—surface soil not assessed
- SWMU193a—chromium, benzo(a)pyrene, and dibenz(a,h)anthracene
- SWMU 193b-beryllium, chromium, and vanadium
- SWMU 193c-chromium and lead
- SWMU 194—surface soil not assessed
- AOC 204—surface soil not assessed

Considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs can be considered "priority COCs" (COCs with a chemical-specific HI or ELCR that exceeds 1 or 1E-4, respectively) in soil for the future recreational user:

- SWMU 99a—none
- SWMU 99b—surface soil not assessed
- SWMU193a-none
- SWMU 193b—none
- SWMU 193c—lead
- SWMU 194—surface soil not assessed
- AOC 204—surface soil not assessed

Considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs can be considered "priority COCs" (COCs with a chemical-specific HI or ELCR that exceeds 1 or 1E-4, respectively) in RGA groundwater for the most likely future use at the following sites (i.e., industrial use):

- SWMU 99a-beryllium, lead, trichloroethene, and radon-222
- SWMU 99b—trichloroethene and radon-222
- SWMU193a—iron
- SWMU 193b-trichloroethene
- SWMU 193c—none
- SWMU 194—RGA groundwater not assessed
- AOC 204—PCB-1254, PCB-1260, PCBs, tetrachloroethene, and trichloroethene

Considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs can be ______ considered "priority COCs" (COCs with a chemical-specific HI or ELCR that exceeds 1 or 1E-4, respectively) in RGA groundwater for on-site rural residential use in the home at the following sites:

- SWMU 99a—aluminum, arsenic, beryllium, chromium, iron, lead, manganese, vanadium, 1,1 dichloroethane, trichloroethene, radon-222, and technetium-99
- SWMU 99b—chromium, trichlororethene, and radon-222
- SWMU193a—iron, trichloroethene, and technetium-99
- SWMU 193b—1,1-dichloroethene, carbon tetrachloride, trichloroethene, and technetium-99
- SWMU 193c-1,2-dichloroethene and trichloroethene
- SWMU 194—RGA groundwater not assessed
- AOC 204—1,1-dichloroethane, 1,1-dichloroethene, PCB-1254, PCB-1260, PCBs, tetrachloroethene, and trichloroethene

Considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs can be considered "priority COCs" (COCs with a chemical-specific HI or ELCR that exceeds 1 or 1E-4, respectively) in McNairy groundwater for the most likely future use at the following sites (i.e., industrial use):

- SWMU 99a-trichloroethene
- SWMU 99b-McNairy groundwater not assessed
- SWMU193a—iron
- SWMU 193b-none
- SWMU 193c-antimony, beryllium, iron, lead, and vanadium
- SWMU 194—McNairy groundwater not assessed
- AOC 204—McNairy groundwater not assessed

Considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs can be considered "priority COCs" (COCs with a chemical-specific HI or ELCR that exceeds 1 or 1E-4, respectively) in McNairy groundwater at the following sites for the future on-site rural resident:

- SWMU 99a—carbon tetrachloride, cis-1,2-dichloroethene, 1,1-dichloroethene, and trichloroethene
- SWMU 99b—McNairy groundwater not assessed
- SWMU193a—cis-1,2 dichloroethene, iron, and technetium-99
- SWMU 193b—cis-1,2 dichloroethene and trichloroethene
- SWMU 193c—aluminum, antimony, arsenic, beryllium, cadmium, carbon tetrachloride, chromium, iron, lead, manganese, molybdenum, vanadium, 1,1-dichloroethene, vinyl chloride, and radon-222
- SWMU 194—McNairy groundwater not assessed
- AOC 204—McNairy groundwater not assessed

When all sites are considered, the priority COCs contributing 10 percent or more to the total HI or ELCR in soil samples at one or more of the sites are ranked below according to the number of sites at which the contaminant is a priority COC:

beryllium	(6/7)	iron	(1/7)
chromium	(4/7)	technetium-99	(1/7)
PAHs	(2/7)	neptunium-234	(1/7)
PCBs	(2/7)	barium	(1/7)
vanadium	(2/7)	1,1-dichloroethene	(1/7)
manganese	(2/7)	tetrachloroethene	(1/7)
antimony	(1/7)	trichloroethene	(1/7)

When all sites are considered, the priority COCs contributing 10 percent or more to the total HI or ELCR in groundwater samples at one or more of the sites are ranked below according to the number of sites at which the contaminant was a priority COC:

trichloroethene	(6/6)	PCBs	(1/6)
cis-1,2-dichloroethene	(3/6)	tetrachloroethene	(1/6)
1,1-dichloroethene	(3/6)	PCB-1254	(1/6)
radon-222	(3/6)	antimony	(1/6)
iron	(3/6)	cadmium	(1/6)
technetium-99	(2/6)	chromium	(1/6)
beryllium	(2/6)	pentachlorophenol	(1/6)
vanadium	(2/6)	arsenic	(1/6)
1,2-dichloroethene	(1/6)	uranium-238	(1/6)
vinyl choride	(1/6)		

Off-site land uses. The following chemicals are priority COCs for off-site use of groundwater (i.e., rural residential use in the home). These chemicals are all COCs that may migrate from a source at a site in WAG 28 to an off-site location and present a chemical-specific HI or ELCR to the off-site rural resident that is greater than 1 or 1E-4, respectively.

- SWMU 99a—lithium
- SWMU 99b—none
- SWMU193a—chromium
- SWMU 193b—none
- SWMU 193c---lithium and manganese
- SWMU 194—chromium, lithium, and strontium
- AOC 204--trichloroethene

1.7.4.3 Pathways of concern

On-site land uses. Only those pathways with a pathway HI for adults or children greater than 0.1 or a pathway ELCR greater than 1E-6 across all contaminants within a land use scenario of concern are POCs. The POCs for each land use scenario of concern are presented in Exhibit 1.45 in Sect. 1.5. Inhalation of vapors and particulates from soil and ingestion of quail are not POCs at any site. All other exposure routes are found in a POC in at least one site.

Off-site land uses. Risks from exposure through individual exposure routes were not included in the assessment of off-site use of groundwater; however, the assumption is that each of the exposure routes included in the calculation of the RBCs used in the assessment are POCs. These are ingestion of water, dermal contact with water, inhalation of vapors emitted by water during showering, and inhalation of vapors emitted by water during household use.

1.7.4.4 Media of concern

MOCs are those media that appear in at least one POC. The following are MOCs at each site:

- SWMU 99a—surface soil, subsurface soil, RGA groundwater, and McNairy groundwater
- SWMU 99b—subsurface soil and RGA groundwater
- SWMU193a—surface soil, subsurface soil, RGA groundwater, and McNairy groundwater
- SWMU 193b—surface soil, subsurface soil, RGA groundwater, and McNairy groundwater
- SWMU 193c—surface soil, subsurface soil, RGA groundwater, and McNairy groundwater

- SWMU 194—subsurface soil
- AOC 204—RGA groundwater

1.7.5 Observations

This section presents observations based on the risk results and uncertainties presented in the previous sections.

Use of the provisional lead RfDs provided by KDEP results in total HIs that exceed 1000 for those locations where the maximum detected concentration of lead in soil exceeded its background concentration. The detection of lead in groundwater samples drawn from WAG 28 sites also resulted in HIs in excess of 1000. However, when this provisional value is excluded from the risk characterization, total HIs are markedly reduced because the hazards due to lead can no longer be explicitly considered. Because of the uncertainty in the results arising from the use of the provisional lead RfD, a better understanding of the risks presented by lead may be gained by comparing the representative exposure concentrations of lead in soil and groundwater to screening levels from KDEP and EPA. In these comparisons (see Exhibit 1.39), the concentrations of lead in RGA and McNairy Formation groundwater at all sites exceed both the KDEP and EPA screening levels. The concentrations of lead in surface soil at SWMU 193c exceeds the KDEP screening level but not that of EPA. Concentrations of lead in surface soil at all sites in WAG 28 are below KDEP and EPA screening levels.

Dermal contact with soil poses considerable threat to human health, with most of this risk coming from contact with metals in soil (primarily beryllium, chromium, and vanadium). In fact, for all land use scenarios evaluated, the systemic toxicity and the ELCR posed through the soil dermal exposure route exceeds that posed by the ingestion route. As illustrated in Sect. 1.6, this is a direct result of using dermal absorption factors (ABS values) that exceed gastrointestinal absorption values and may be too conservative. In such circumstances, risk estimates from the dermal exposure route may be unrealistic and exceed the real risk posed by this route of exposure. Although chemical-specific ABS values were used when available, default ABS values were used for most chemicals because chemical-specific values are lacking. Chemical-specific ABS values were available for PCBs, cadmium, and carbon disulfide and used in this BHHRA. Remedial decisions based on the dermal contact with soil exposure route should be carefully considered because of the uncertainty associated with risk from this exposure route.

The current use scenario, the industrial worker, has total HIs exceeding de minimus levels at two sites, SWMUs 193b and 193c; however, if lead is excluded as a COPC, SWMU 193c is no longer of concern. The driving contaminants at SWMU 193b contributing more than 10 percent to the total HI are chromium and vanadium, with dermal contact as the driving pathway.

The total ELCRs for the current industrial worker exceed de minimus levels at SWMUs 99a, 193a, and 193b. The driving contaminant at SWMU 99a is beryllium, with dermal contact as the driving pathway. The driving contaminants at SWMU 193a are PAHs, with dermal contact as the driving pathway. The driving contaminant at SWMU 193b is beryllium, with dermal contact as the driving pathway.

The most plausible future use scenario, the future industrial worker, has total HIs and ELCRs exceeding de minimus levels at all sites except SWMU 194, for which this scenario/land use combination did not apply. As discussed in the BHHRA, the future industrial land use scenario is identical to the current industrial land use scenario except that the future industrial land use scenario also evaluates use of RGA groundwater. Addition of groundwater as a medium of exposure adds significantly to the risk for

this scenario. If groundwater contribution is removed from the risk totals, the driving pathways are identical to the current industrial use scenario.

The driving contaminants contributing to more than 10 percent of total HIs at SWMU 99a (excluding lead) in RGA groundwater are trichloroethene, chromium, iron, and vanadium, with ingestion as the primary pathway. The driving contaminant contributing to more than 10 percent of total HIs at SWMU 99b is trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at SWMU 193a are iron and trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at SWMU 193b is trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at SWMU 193b is trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at SWMU 193c are 1,2-dichloroethene and trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at SWMU 193c are 1,2-dichloroethene and trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at SWMU 193c are 1,2-dichloroethene and trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at SWMU 193c are 1,2-dichloroethene and trichloroethene, with ingestion as the primary pathway. The driving contaminants contributing to more than 10 percent of total HIs at AOC 204 are PCBs, with dermal contact and ingestion as the primary pathways.

The driving contaminants contributing to more than 10 percent of total ELCR for the future industrial worker exposed to RGA groundwater at SWMU 99a are 1,1-dichloroethene, beryllium, and radon-222, with incidental ingestion and inhalation of vapors and particulates as the primary pathways. The driving contaminants contributing to more than 10 percent of total ELCR at SWMU 99b are trichloroethene and radon-222, with inhalation as the primary pathway. The driving contaminants contributing to more than 10 percent of total ELCR at SWMU 99b are trichloroethene, with ingestion and dermal contact as the primary pathways. The driving contaminants contributing to more than 10 percent of total ELCR at SWMU 193a are pentachlorophenol and trichloroethene, with ingestion and dermal contact as the primary pathways. The driving contaminants contributing to more than 10 percent of total ELCR at SWMU 193b are trichloroethene and 1,1-dichloroethene, with ingestion as the primary pathway. The driving contaminant contributing to more than 10 percent of total ELCR at SWMU 193b are trichloroethene and 1,1-dichloroethene, with ingestion as the primary pathway. The driving contaminant contributing to more than 10 percent of total ELCR at SWMU 193b are trichloroethene and 1,1-dichloroethene, with ingestion as the primary pathway. The driving contaminant contributing to more than 10 percent of total ELCR at SWMU 193c is trichloroethene, with ingestion as the driving pathway. The driving contaminants contributing to more than 10 percent of total ELCR at AOC 204 are PCBs, tetrachloroethene, and 1,1-dichloroethene, with dermal contact as the primary pathway.

The COCs for analytes migrating from sources in WAG 28 soil and groundwater as determined by risk estimates for off-site residential groundwater users are chromium, lithium, manganese, strontium, technetium-99, and trichloroethene.

Exhibits 1.58–1.61 summarize the effect of multiple uncertainties on the risk estimates for the most likely current and future use at WAG 28 sites (i.e., industrial). Exhibits 1.58 and 1.59 present a quantitative comparison of systemic toxicity estimates as various uncertainties are addressed. Exhibits 1.60 and 1.61 present similar information for ELCR. Specific uncertainties addressed in these exhibits are (1) the use of the provisional lead RfD (Exhibits 1.58 and 1.59 only), (2) inclusion of analytes that were infrequently detected, (3) inclusion of laboratory contaminants, (4) the use of provisional and withdrawn toxicity and carcinogenicity benchmark values in the toxicity assessment, and (5) the use of EPA Region 4 dermal absorption values (soil exposure only). In addition, the last column in each exhibit presents the total hazard or risk that results when the itemized uncertainties are addressed simultaneously. These data are intended to provide potentially more realistic quantitative lower bound risk estimates for the current and future industrial worker at each location, thereby representing additional values available to risk management decision makers. The true but unknown values for the risk or hazard might be expected to fall between the two extremes.

Location	Default HI ^a	Default HI w/o lead ^a	Default HI minus infequently detected analytes w/o lead ^b	Default HI minus common laboratory contaminants w/o lead ^c	Default HI minus analytes with provisional or withdrawn toxicity values w/o lead ^d	Region 4 absorption factors w/o lead ^e	Lower bound HI ^f
SWMU 99a (soil)	<]	<1	<1	<1	<1	<1	<1
SWMU 193a (soil)	<]	<1	<1	<1	<1	<1	<1
SWMU 193b (soil)	5.25	<1	5.25	5.25	<1	<]	<]
SWMU 193c (soil)	3620	<1	<1	<1	<1	<1	<]

Exhibit 1.58. Quantitative summary of uncertainties for the current industrial worker—systemic toxicity

^a These values are identical to the values presented in Exhibit 1.17.

^b These values are identical to the values presented in Table 1.82.

^c These values are identical to the values presented in Table 1.84.

^d These values are identical to the values presented in Table 1.86.

^e These values are identical to the values presented in Exhibit 1.55.

^f These values were derived omitting contributions from lead, infrequently detected analytes, and compounds for which only provisional or withdrawn toxicity values are available and using EPA Region 4 dermal absorption factors.

Exhibit 1.59. Quantitative summary of uncertainties for the future industrial worker—systemic toxicity

Location	Default HIª	Default HI w/o lead ^a	Default HIs minus infrequently detected analytes w/o lead ^b	Default HI minus laboratory contaminants w/o lead ^e	Default HI minus analytes with provisional or withdrawn toxicity values w/o lead ^d	Lower bound HI*
SWMU 99a (RGA)	8,150	5.11	5.11	5.11	2.61	2.6
SWMU 99a (McNairy)	1.64	1.64	1.64	1.64	<1	<]
SWMU 99b (RGA)	7.00	7.00	7.00	7.00	2.22	2.2
SWMU 193a (RGA)	1.64	1.64	1.63	1.63	<1	<1
SWMU 193a (McNairy)	4.69	4.69	4.43	4.69	<1	<1
SWMU 193b (RGA)	1.74	1.74	1.74	1.73	<1	<]
SWMU 193b (McNairy)	<1	<1	<1	<1	<]	<]
SWMU 193c (RGA)	1.46	1.46	1.46	1.46	1.09	1.09
SWMU 193c (McNairy)	25,100	9.92	9.92	9.92	7.55	7.5
AOC 204 (RGA)	33.3	33.3	33.3	33.3	32.1	32.1

^a These values are identical to the values presented in Exhibit 1.21.

^b These values are identical to the values presented in Table 1.82.

^c These values are identical to the values presented in Table 1.84.

^d These values are identical to the values presented in Table 1.86.

• These values were derived omitting contributions from lead, infrequently detected analytes, and compounds for which only provisional or withdrawn toxicity values are available and using EPA Region 4 dermal absorption factors.

Location	Default ELCR ^e	Default ELCR minus infequently detected analytes ^b	Default ELCR minus common iaboratory contaminants ^c	Default ELCR minus analytes with provisional or withdrawn toxicity values ^d	ELCR computed using EPA Region 4 absorption factors ^e	Lower bound ELCR ^f
SWMU 99a (soil)	3.1E-4	3.0E-4	3.1E-4	7.5E-5	6.7E-5	5.8E-5
SWMU 193a (soil)	1.5E-5	1.5E-5	1.5E-5	9.2E-6	2.0E-6	1.2E-6
SWMU 193b (soil)	5.1E-4	5.1E-4	5.1E-4	2.7E-9	1.1E-5	2.7E-9
SWMU 193c (soil)	1.7E-10	1.7E-10	1.7E-10	1.7E-10	1.7E-10	1.7E-10

Exhibit 1.60. Quantitative summary of uncertainties for the current industrial worker—excess lifetime cancer risk

^a These values are identical to the values presented in Exhibit 1.19.

^b These values are identical to the values presented in Table 1.82.

^c These values are identical to the values presented in Table 1.84.

^d These values are identical to the values presented in Table 1.86.

* These values are identical to the values presented in Exhibit 1.55.

^f These values were derived omitting infrequently detected analytes, laboratory contaminants, and those contaminants for which only provisional or withdrawn toxicity values are available and using EPA Region 4 dermal absorption values.

Location	Default ELCR*	Default ELCRs minus Infrequently detected analytes ^b	Defauit ELCR minus laboratory contaminants ^e	Default ELCR minus analytes with provisional or withdrawn toxicity values ^d	ELCR computed using EPA Region 4 dermal toxicity values	Lower bound ELCR [*]
SWMU 99a (RGA)	5.6E-4	5.6E-4	5.6E-4	3.1E-4	NA	3.1E-4
SWMU 99a (McNairy)	7.6E-5	7.6E-5	7.6E-5	5.3E-5	NA	5.3E-5
SWMU 99b (RGA)	2.6E-4	2.6E-4	2.6E-4	1.5E-4	NA	1.5E-4
SWMU 193a (RGA)	2.6E-5	1.4E-5	2.6E-5	1.7E-5	NA	3.6E-6
SWMU 193a (McNairy)	1.1E-6	1.1E-6	1.1 E-6	8.8E-7	NA	8.8E-7
SWMU 193b (RGA)	4.4E-5	4.4E-5	4.3E-5	1.7E-5	NA	1.7E-5
SWMU 193b (McNairy)	8.4E-7	8.4E-7	8.4E-7	1.5E-7	NA	1.5E-7
SWMU 193c (RGA)	1.0E-5	1.0E-5	1.0E-5	1.9E-6	NA	1.9E-6
SWMU 193c (McNairy)	4.2E-4	4.2E-4	4.2E-4	2.0E-4	NA	2.0E-4
AOC 204 (RGA)	1.3E-3	1.3 × 10 ⁻³	1.3×10^{-3}	1.0E-3	NA	1.0E-3

Exhibit 1.61.	Quantitative summary of uncertainties for the future industrial				
worker-excess lifetime cancer risk					

Notes: NA = Not Applicable.

* These values are identical to the values presented in Exhibit 1.29.

^b These values are identical to the values presented in Table 1.82.

^c These values are identical to the values presented in Table 1.84.

^d These values are identical to the values presented in Table 1.86.

^e These values were derived omitting infrequently detected analytes, laboratory contaminants, and those contaminants for which only provisional or withdrawn toxicity values are available and using EPA Region 4 dermal absorption values

In Exhibits 1.58 and 1.59, the HI estimates for both current and future industrial worker exposure to soil calculated using the default exposure rates (column 1) vary markedly from the lower bound estimates (last column) for those locations where lead was included as a COPC, and the provisional lead RfD was used. For those locations, omitting lead from the list of COPCs decreases the HIs by about four orders of magnitude. By contrast, other uncertainties investigated in both Exhibits 1.58 and 1.59 have little effect on the HI estimates. For the current industrial worker exposed to surface soil at SWMUs 99a, 193a, 193b and 193c, the lower-bound estimates of HI are all less than the de minimis level established in the Methods Document (i.e., HI = 1). For the future industrial worker, the lower bound HI estimates still exceed an HI of 1 at several locations in RGA and McNairy groundwater.

As shown in Exhibit 1.60, most of the ELCR estimates calculated for the current industrial worker using the default exposure rates (column 1) differ from the lower bound estimates (last column) to varying extents. Thus, the numerical comparisons vary from "no change" (a ratio of 1) to differences of over five orders of magnitude. Where changes occur, the uncertainties that appear to make the most significant contribution are the omission of compounds with provisional and withdrawn carcinogenicity benchmarks and the use of EPA Region 4 dermal absorption factors instead of KYDEP defaults. Notwithstanding these changes, the lower bound ELCRs remain within the EPA's range of concern for two of the four sites under consideration.

By contrast to soil exposure, Exhibit 1.61 shows that the ELCR estimates for the future industrial worker exposure to groundwater under default and lower bound conditions do not vary greatly. In general, the changes are less than one order of magnitude, with the resulting lower bound ELCR estimates still exceeding the de minimis level at some sites.

1.8 REMEDIAL GOAL OPTIONS

This section presents RGOs for the COCs identified in Sect. 1.5 and the methods used to calculate the RGOs. These RGOs should not be interpreted as being cleanup goals but as risk-based values that may be used to guide the development of cleanup goals by risk managers. Cleanup goals will be determined in the feasibility study. RGOs were calculated for each medium at each location. For pathways involving contributions from more than one medium (i.e., ingestion of vegetables), the RGOs were calculated for each medium by setting the contributions from all other media to zero; this allowed for accurate determination of RGOs by medium. Where ingestion rates differed between adults and children, the more conservative child ingestion rates were used. In addition, maximum contaminant levels (MCLs) are presented in the tables developed as part of this section. MCLs are not cleanup criteria. The National Contingency Plan (CERCLA) notes that reduction of contaminant concentrations below MCLs may be required if multiple contaminants are present or if contaminants may reach a receptor through exposure routes not considered in the development of MCLs; therefore, risks for use of contaminated groundwater must be presented in addition to a simple screen against MCLs so that risk managers can make decisions.

1.8.1 Calculation of RGOs

Recent EPA guidance directs that RGOs are to be calculated for all COCs identified in a BRA. The COCs identified in this risk assessment, their RGOs, and MCLs are presented in Table 1.88. The SAS[®] program used to calculate these RGOs is presented Sect. 8 of Appendix C of this volume.

EPA guidance (EPA 19911) directs that RGOs for each COC are to be calculated by rearranging the equations used to calculate each COC's HQ or chemical-specific ELCR so that the equation can be used to solve for a concentration of the COC that will result in target total HIs of 0.1, 1.0, and 3.0 and target total ELCRs of 1E-4, 1E-5, and 1E-6. Here, the target total HI is defined as the sum of a COC's HQs over all pathways of concern, and the target total ELCR is defined as the sum of a COC's chemical-specific ELCRs over all pathways of concern. Although rearranging the risk equations and solving for a concentration is one approach to calculating RGOs, it is simpler to use the fact that risk is calculated in this risk assessment by linearly combining a series of exposure factors and toxicity factors with each analyte's environmental concentration; therefore, the risk posed by an analyte at any given concentration is directly related to the risk posed by that analyte at any other concentration. This relationship is illustrated in the following equation:

Concentration	RGO
Risk	Target Risk

where:

Concentration = the exposure concentration for the medium Risk = the risk posed by exposure to the contaminated medium RGO = the remedial goal option Target Risk = one of the values listed above

1.8.2 Presentation of RGOs

The equation developed in the previous section was applied for each COC. The RGOs developed for all land use scenarios of concern, POCs, and COCs for WAG 28 are presented in Table 1.88. In addition, this table presents the representative exposure concentration used in the BHHRA and, for groundwater, each COC's MCL. The MCLs were taken from RAIS (DOE 1998c). RGOs for sources of off-site groundwater contamination are not presented because these rely on the fate and transport modeling

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discussed in Sect. 5 of Vol. 1 and Appendix B of this volume. These RGOs will be developed after this modeling is refined as needed in the feasibility study; however, the RGOs for groundwater in the off-site location are presented.

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2. BASELINE ECOLOGICAL RISK ASSESSMENT

The primary purpose of the BERA is to determine whether adverse ecological effects may occur or are occurring as a result of exposure to one or more stressors at WAG 28. The BERA follows general guidance provided in *Guidelines for Ecological Risk Assessment* (EPA 1998) and consists of the following elements:

- problem formulation (Sect. 2.1)
- exposure assessment (Sect. 2.2)
- effects assessment (Sect. 2.3)
- risk characterization (Sect. 2.4)

Because WAG 28 sites vary in terms of potential exposure media and pathways, receptor populations, and contaminant migration pathways, the general objectives of each step of the BERA are provided as introductory material. Site-specific information is then provided for each site in subsequent sections. Tables supporting the risk assessment are located in Appendix A of Vol. 4.

Because only abiotic data are available for sites in WAG 28, the BERA evaluated existing media data only. Additional lines of evidence (e.g., media toxicity testing and biological surveys) were not collected and do not appear to be necessary at this stage because much of the WAG 28 area is used for industry. SWMU 194 and AOC 204, which are located outside the PGDP security fence, provide suitable habitat for terrestrial biota; however, complete exposure pathways are not expected at these sites because potential contamination is contained within subsurface soils.

2.1 PROBLEM FORMULATION

Ecological risk assessments begin with a problem formulation phase that defines the scope of the assessment in terms of (1) environmental description, (2) evaluation of the adequacy of available data for identifying chemicals of potential ecological concern (COPECs), (3) assessment endpoints, (4) potential receptor populations, (5) identification of potential exposure pathways, and (6) development of a conceptual site model. The problem formulation phase determines the following:

- analytes (e.g., inorganics, organics, and radionuclides);
- media (e.g., surface water, sediment, and soil);
- routes of exposure (e.g., ingestion of water/soil, inhalation of volatile organics and/or particulates, ingestion of contaminated food items, and dermal contact with contaminated media); and
- categories of receptors (e.g., plants, invertebrates, herbivores, omnivores, carnivores, and vermivores) (Suter et al. 1995).

The result of the problem formulation phase is a conceptual site model that is based on an integration of the information gathered.

2.1.1 Environmental Description

PGDP is situated between Big Bayou Creek to the west and Little Bayou Creek to the east. The confluence of these two creeks is a marsh approximately 3 miles north (downgradient) of PGDP with ultimate discharge to the Ohio River. PGDP is located on a local drainage divide with surface water flow



to the east and northeast toward Little Bayou Creek and to the west and northwest toward Big Bayou Creek. Most of the flow in the creeks is from process effluents from PGDP (Energy Systems 1990).

The pH of soils and surface water in the Bayou drainage system verges on acidity. Stream alkalinity and pH are periodically low. Soil pH is strongly acidic and low in buffering capacity. In addition, the pH of rainfall in the region has been reported to be as low as 3.5 (Birge et al. 1989). The entire PGDP is above the historical high water floodplain of the Ohio River (CH2M Hill 1991a).

2.1.1.1 Aquatic communities

The aquatic communities in and around the PGDP area include Little Bayou Creek and Big Bayou Creek (both perennial streams), the North-South Diversion Ditch, and other smaller drainage areas (Fig. 3.4 of Vol. 1). In addition, approximately 13 fishing ponds are located primarily in the WKWMA. Aquatic habitats are used by muskrat, raccoon, and beaver, as well as many species of water birds, including wood duck, geese, heron, bald eagle, and other species of migratory birds. The dominant fish populations include several species of sunfish (especially bluegill and green sunfish) as well as bass and catfish. Bluegill, green and longear sunfish, and stoneroller dominate the shallow areas of the two creeks. Largemouth bass, bluegill and, to a lesser extent, green sunfish dominate ponds. Aquatic habitats do not currently exist at sites in WAG 28.

2.1.1.2 Terrestrial communities

The terrestrial component of the ecosystem includes the plants and animals that use the habitat for food, reproduction, and protection and is described by the dominant vegetation groups that characterize the community. Because much of PGDP's terrestrial habitat is managed for multiple uses, forest and shrub tracts alternate with fence rows and transitional edge habitats (ecotones) along roads and transmission-line corridors. In addition to upland terrestrial communities, a number of wetland communities exist at PGDP.

No quantitative surveys of terrestrial wildlife near PGDP were conducted as part of RI activities; however, observations by staff ecologists during site investigations and information from WKWMA and Ballard County Wildlife Management Area (BCWMA) staff have provided a qualitative description of wildlife communities likely to inhabit terrestrial communities. Rabbits, mice, and a variety of other small mammals frequent open herbaceous areas. Birds include red-winged blackbirds, quail, sparrows, and predators such as hawks and owls. In ecotones (including fence rows, low shrub, and young forests), a variety of wildlife is present, including opossum, vole, mole, raccoon, and deer. Birds typical in ecotones include red-winged blackbird, loggerhead shrike, mourning dove, bobwhite quail, turkey, cardinal, and meadowlark. Several groups of coyotes also reside in the vicinity of PGDP. In mature forests, squirrels, various songbirds, and great horned owls may be present. The primary game species occupying the area are deer, turkey, opossum, rabbit, raccoon, squirrel, quail, and mourning dove.

A detailed description of WAG 28 sites is provided in Chap. 3 of Vol. 1 and in Sect. 1.3.1 of this volume. Sites in WAG 28 that are inside the security fence are generally highly industrialized and provide minimal habitat for ecological receptors. See Figs. 1.2–1.8 in Chap. 1 of this volume for photographs. Sites in WAG 28 that are outside the security fence are open grassy areas and lightly forested areas that provide potential habitat for ecological receptors. While exposures at most sites inside the fence are unlikely (due to gravel/asphalt cover) and are not evaluated under current conditions (with the exception of SWMU 193a), the possibility of exposures in the future at pertinent sites are evaluated, assuming that industrial controls are no longer present and that the sites develop suitable habitat for terrestrial plants and wildlife. Exposures at sites outside the fence (SWMU 194 and AOC 204), which provide suitable habitat, are considered unlikely because potential contamination is contained within subsurface soils.

SWMU 99a, site of the former C-745 Kellogg Buildings, is located along the eastern edge of PGDP,... south of Building C-360, immediately north of Tennessee Avenue, and west of Patrol Road 3. The buildings were constructed in 1951 as support facilities during construction of the PGDP cascade facilities. Degreasing operations using trichloroethene possibly occurred on this site. The buildings have been demolished, and the area now serves as the C-746-C Classified Scrap Yard and the C-745-E Uranium Hexafluoride (UF₆) Cylinder Storage Yard. SWMU 99a was identified as a possible source area due to past practices on the site. At some time, a layer of gravel was placed over the soil on most of the site to improve drainage, improve site access, and control weeds. SWMU 99a includes the area bound by Tennessee Avenue on the south, the PDGP security fence on the east, the security fence to Building C-360 on the north, and 18th Street on the west. The total area is approximately 2.4 acres. Approximately 40 percent of SWMU 99a is covered by concrete/asphalt and 60 percent by gravel; therefore, this site provides no current suitable habitat for ecological receptors. An evaluation of potential future exposures was conducted for this site and assumed current soil concentrations and development of suitable wildlife habitat.

SWMU 99b, a former septic tank and leaching field used by the Kellogg Buildings, is located immediately outside the east guard house of the plant. The tank and the associated field, which is connected to the Kellogg Buildings by a vitreous clay drain line, are located approximately 350-400 ft southeast of the building site in the gravel parking lot east of Patrol Road 3. Although lateral lines for the leaching field were found intact when encountered during construction activities in late 1994, they were not located during RI field activities. The suspected location is situated under a gravel-covered parking area between the contractor staging area to the north and AOC 204 to the south. The total area is approximately 0.3 acres. An estimated 80 percent of SWMU 99b is covered by gravel and 20 percent by grass. Surface soil was not a medium of concern at this site because the leach lines were below the surface of a gravel-covered parking lot. No ecological exposures were expected; therefore, the site was eliminated from further evaluation during problem formulation.

SWMU 193a, the former Millwright Shop, is the outside perimeter of Building C-333 located in the western portion of SWMU 193 north of Michigan Avenue and west of 13th Street. The shop is no longer standing, and all that remains is a concrete pad. The site does not include Building C-333 but includes the property directly west of the building. The site is bound on the south by Michigan Avenue, on the west by Patrol Road 5, on the north by Ohio Avenue, and on the east by 13th Street. The area is drained by the plant storm drain system, which eventually exits the plant through Kentucky Pollutant Discharge Elimination System (KPDES) Outfall 009. The total area is approximately 17.4 acres. An estimated 90 percent of SWMU 193a is covered by grass and 10 percent by gravel. Though sites inside the security fence are generally highly industrialized, the amount of grass coverage at SWMU 193a provides potential ecological habitat, and ecological evaluations of current and future exposures were conducted for this site.

SWMU 193b, the former Pipe Fabrication Shop, is the outside northern perimeter of Building C-333 located in the northern portion of SMWU 193. The site does not include Building C-333 but includes the property directly north of the building. The site is bound on the south by Building C-333, on the north by Ohio Avenue, on the west by 13th Street, and on the east by 16th Street. The area is drained by the plant storm drain system, which eventually exits the plant through KPDES Outfall 009. The total area is approximately 4.3 acres. An estimated 100 percent of SWMU 193b is covered by gravel; therefore, this site provides no current suitable habitat for ecological receptors. An evaluation of potential future exposures was conducted for this site and assumed current soil concentrations and development of suitable wildlife habitat.

SWMU 193c is located on the south side of the C-333 building. The site formerly consisted of temporary buildings used during construction of PGDP, including the electrical warehouse, general warehouse, sheet metal shop, light and heavy equipment shops, acetylene shop, paint shop, civil

engineering testing laboratory, filling station, and steel fabrication shop. A leaching field was located in the southwest corner of the site. The leaching field consists of 4-in. drain tiles in shallow soil. Currently, the site is used to store UF₆ cylinders. The site is bound on the north by Michigan Avenue, on the south by Patrol Road 4, on the east by 21st Street, and on the west by Patrol Road 5. The area is drained by the plant storm drain system, which eventually exits the plant through KPDES Outfall 011. The total area is approximately 87.0 acres. An estimated 15 percent of SWMU 193c is covered by concrete/asphalt, 80 percent by gravel, and 5 percent by grass; therefore, this site provides no current suitable habitat for ecological receptors. An evaluation of potential future exposures was conducted for this site and assumed current soil concentrations and development of suitable wildlife habitat.

SWMU 194 is located in the southwest portion of the plant directly outside the security fence. SWMU 194 was the site of the administrative portion of the McGraw construction facilities and consisted of an administration building (105,500 ft²), cafeteria (10,200 ft²), security guard headquarters (5,360 ft²), hospital (4,480 ft²), purchasing building (12,000 ft²), paper and stationary warehouse (3,900 ft²), a boiler house, and two leaching fields located west of Hobbs Road. All of the buildings have been demolished. The site is bound on the north by Curlee Road, on the south by Patrol Road 4, on the east by Patrol Road 5, and extends west of Hobbs Road. The total area is approximately 41.7 acres. An estimated 100 percent of SMWU 194 is covered by grass. For the BERA, the conceptual model defined in the approved WAG 28 work plan (DOE 1998a) defined the potential sources of contamination in SWMU 194 as being contained within subsurface soil (i.e., drainfields). Consequently surface soils are not impacted and do not require an ecological evaluation to be performed.

AOC 204 is located on the eastern side of PGDP and bound on the north and south by KPDES Outfalls 010 and 011 and on the east and west by Dyke Road and the security fence. It is suspected that AOC 204 was used as a staging area or construction debris burial ground associated with the original construction of the plant. The surface of AOC 204 is undulating, with elevations ranging from 364 to 382 ft above mean sea level. The area is covered with heavy vegetation and a young stand of trees. The total area is approximately 11.3 acres. An estimated 50 percent of AOC 204 is covered by grass and 50 percent by trees/shrubs. Surface soil was not a medium of concern at AOC 204, and surface soil samples were not evaluated. Therefore, no ecological evaluation was conducted for this site. For the BERA, the conceptual model defined in the approved WAG 28 work plan (DOE 1998a) defined the potential sources of contamination in AOC 204 as being contained within subsurface soil (i.e., buried debris pile). Consequently surface soils are not impacted and do not require an ecological evaluation to be performed.

2.1.2 Data Evaluation

2.1.2.1 Ecological data evaluation considerations

For the BERA, the data evaluation steps described in RAGS, Part A, Chap. 5 (EPA 1989a) (as they apply to data collected at PGDP and as modified by recent regulatory agency comments) and EPA (1998c) were followed when developing COPECs. Environmental data evaluated for the BERA were collected during the WAG 28 RI field activities and historical data collected during previous investigations of the sites. Investigations during the WAG 28 RI were considered to provide additional characterization to areas of previous investigation for WAG 28 sites. Only surface soil samples (less than 1 ft bgs) were evaluated in assessing ecological risk from soils.

Analyte concentrations below the detection limit were assigned a value of 0 (zero) if all samples at a location were below the detection limit. These analytes were subsequently dismissed from the list of COPECs and from the assessment. Analyte concentrations below the detection limit were assigned a value of one-half the detection limit if one or more samples at a site were above the detection limit. These values were then used to calculate the 95% UCL.

For ecological risk evaluation, exposure concentrations that are deemed to be safe are referred to as ecotoxicological benchmarks. Benchmark values are updated regularly with the addition of new chemicals, analysis of new data, and receipt of new direction from regulators. The site-specific exposure concentrations that are compared to benchmarks are related to the characteristics of the receptor. In general, a concentration is used that represents a reasonable maximum exposure based on the characteristics of the medium and receptor. A fundamental distinction that should be made is between receptors that average their exposure spatially and those that are constantly exposed. For example, nonmotile receptors (e.g., plants) are more likely to be exposed to maximum contaminant concentrations, whereas motile species (e.g., wildlife) move through the environment and are more likely to be exposed to an average of contaminant concentrations. The following text describes the derivation of the RME for receptors in this BERA:

- Terrestrial wildlife move across a site, potentially consuming soil, vegetation, or prey from locations that vary in their degree of contamination. The 95% UCL on the mean surface soil concentration is the appropriate conservative estimate unless it exceeds the maximum detected concentration, in which case the maximum value is used as the RME.
- Surface soil contaminant concentrations are relatively constant over time, thus immobile or nearly
 immobile plants and invertebrates are constantly exposed. The RME for these receptors is the
 maximum observed concentration. That is, some organisms occupy that maximally contaminated soil
 or would occupy it if it were not toxic; therefore, exceedance of ecotoxicological benchmarks at any
 location implies a potential risk to some receptors.

Comparison to benchmarks requires specification of individual wildlife species. The chosen species should include potentially sensitive representatives of trophic groups and vertebrate classes that are potentially exposed to contaminants at the site. In some cases, there are no appropriate toxicity data available for a chemical/receptor combination. For these cases, the chemical cannot be eliminated, and its toxicity cannot be addressed. Such chemicals are retained in a separate category to determine the need for media toxicity testing and to prevent elimination from further consideration of the media in which the chemicals occur (Suter et al. 1995).

Comparing the site media concentrations to benchmarks identifies contaminant concentrations potentially toxic to the endpoint biota. Many of the analytes commonly identified during an RI also occur naturally in the environment. The concentrations of these analytes found at local background sites are normally assumed to be nonhazardous. To ensure that risk management decisions are based on the risks posed by site contaminants, the background constituents are differentiated from the site-associated contaminants. For each area, the detected, naturally occurring inorganic and radionuclide analytes are compared to background values. Synthetic organic compounds should not be present in reference samples; therefore, any synthetic organic compounds that are detected and validated are considered above background.

Background concentrations are available for soils (DOE 1997). The comparison of analyte concentrations and radionuclide activities in site samples and background samples involves only inorganic analytes and naturally occurring radionuclides.

2.1.2.2 Selection of COPECs for WAG 28

Summary statistics (frequency of detection and mean and maximum concentrations) for the analytes detected in soil at sites in WAG 28 are provided in Table 2.1. Table 2.1 also includes site-specific RMEs that are the lower of the 95% UCLs and the maximum detected concentrations. Essential nutrients and





analytes with maximum concentrations within background limits were eliminated from further consideration in the analysis. Those with concentrations above background or with no background values were carried through the assessment.

2.1.3 Ecological Assessment Endpoints

Assessment endpoints are valued ecological resources or entities that are to be protected. Should these environmental characteristics be significantly affected by site contamination, the need for remediation may be indicated (Suter 1989; EPA 1992a, 1998c). Measurement endpoints are quantitative summaries of a measurement or series of measurements that are related to effects on an assessment endpoint (Suter 1989; EPA 1992a, 1998c). For example, if the assessment endpoint is fish abundance in a stream suspected of being affected by a waste site, the stream can be sampled, and fish abundance (the corresponding measurement endpoint) can be measured directly.

The complete definition of an assessment endpoint includes a subject (e.g., soil invertebrates) as well as a level of effects (e.g., reduction in species richness or abundance) that are used to determine whether an impact has occurred (Suter 1993). Guidance for choosing levels of effects on endpoint properties that may constitute grounds for remedial action has not been promulgated on a national basis for ecological risk assessment as it has been for human health risk assessment. Therefore, these levels of effects must be inferred on the basis of analysis of historical federal and state EPA practice as well as that of other state regulatory agencies (Suter et al. 1995).

If the assessment endpoint is not readily observable, the measurement endpoint may be a surrogate for the assessment endpoint. For example, if the assessment endpoint is fish abundance in a stream that may receive future discharges from a waste site, the effect of these discharges on fish abundance cannot be measured directly. Instead, future contaminant concentrations in the stream must be modeled and then compared to standard toxicity data. The characteristics of good assessment endpoints are identified in EPA's field and laboratory manual for ecological assessment of hazardous waste sites and EPA's *Framework for Ecological Risk Assessment* (EPA 1992i). These characteristics are ecological and societal relevance, susceptibility to hazards at the site, and accessibility to prediction and measurement (Suter 1989; EPA 1992a, 1998c).

Six terrestrial populations (plants, soil invertebrates, herbivorous mammals, omnivorous mammals, vermivorous mammals, and carnivorous mammals) with characteristics that meet one or more of the criteria for good assessment endpoints were chosen for the BERA. The following paragraphs discuss the reasons for which each type of representative receptor population was chosen for evaluation in the BERA.

Species richness, abundance, or primary production of plants within the terrestrial community were chosen as assessment endpoints for the evaluation of terrestrial exposure to contaminants in soil because terrestrial plant communities are (1) ecologically significant because the plant community provides habitat for terrestrial animal species, (2) societally significant because the plant community provides habitat for terrestrial game species, (3) susceptible to hazards at the site because plants are immobile and receive their nutrients and water from a fixed area of the soil medium and would thereby be directly exposed to contaminants in that medium, and (4) accessible to prediction (toxicity data are available) and measurement (through biological surveys and/or toxicity testing).

Abundance of soil invertebrates was chosen as an assessment endpoint for the evaluation of terrestrial exposure to contaminants in soil because soil invertebrate species are (1) ecologically significant because they consume fresh organic material and leave partially decomposed products in their excreta, which are then further decomposed by soil microbes, (2) susceptible to hazards at the site because they inhabit the soil medium and are thereby directly exposed to any contaminants in that medium; and

(3) accessible to prediction (toxicity data are available) and measurement (through biological surveys, and/or toxicity testing).

Species richness or abundance of herbivorous mammals within the terrestrial community was chosen as an assessment endpoint for the evaluation of terrestrial exposure to contaminants in soil (incidental ingestion) and in their diet (uptake of contaminants into vegetation) because herbivorous mammals (e.g., meadow vole) are (1) of ecological significance as consumers of vegetation and prey for carnivores, (2) are susceptible to hazards at the site because they eat plants growing at the site, and (3) accessible to prediction (toxicity data are available) and measurement (through biological surveys).

Species richness or abundance of omnivorous small mammals within the terrestrial community was chosen as an assessment endpoint for the evaluation of terrestrial exposure to contaminants in soil (incidental ingestion) and in their diet (uptake of contaminants into vegetation and invertebrates) because omnivorous mammals (e.g., white-footed mouse) are (1) ecologically significant because they are prey for many other species, (2) susceptible to hazards at the site because they have home ranges small enough that their activities can be associated with a specific site, and (3) accessible to prediction (toxicity data are available) and measurement (through biological surveys).

Species richness or abundance of vermivorous small mammals within the terrestrial community was chosen as an assessment endpoint for the evaluation of terrestrial exposure to contaminants in soil (incidental ingestion) and in their diet [bioaccumulation/biomagnification in earthworms (the principal prey species)] because vermivorous mammals (e.g., short-tailed shrews) are (1) ecologically significant because they are prey for other species, (2) susceptible to hazards at the site because they have home ranges small enough that their activities can be associated with a specific site, and (3) accessible to prediction (toxicity data are available) and measurement (through biological surveys).

Species richness or abundance of carnivorous mammals within the terrestrial community was chosen as an assessment endpoint for the evaluation of terrestrial exposure to contaminants in soil (incidential ingestion) and in their diet (bioaccumulation/biomagnification in prey) because carnivorous mammals (e.g., long-tailed weasel) (1) are ecologically significant because they prey on small mammals (2) are susceptible to hazards at the site because contaminants may accumulate in their prey, (3) frequent grassy areas in pursuit of prey, and (4) are accessible to prediction (toxicity data are available) and measurement (through biological surveys).

The mammalian wildlife endpoints are assumed to represent other wildlife (i.e., birds) with similar modes of exposure. While there may be taxonomic differences in sensitivity to various chemicals, exposures for the mammalian receptors, particularly the shrew, are anticipated to be greater than comparable birds because of their potential close association with the sites (more restricted movements), residential status (nonmigratory) and, in the case of the shrew, higher expected soil ingestion rates. In addition, this suite of endpoints was agreed upon during the data quality objective process for WAG 28.

2.1.4 Identification of Potentially Exposed Receptors

To evaluate assessment endpoints, a representative set of receptor species is selected. The sections that follow discuss the selection of these representative receptors and their relationship to the particular medium which they are used to evaluate. Effects on species that are not included explicitly in the representative assessment receptor set are nonetheless considered implicitly in the evaluation because no species exists in isolation from the community of which it is a part.

The principal assessment endpoints involve effects to the receptor population or community rather than the individual level of biological organization unless the assessment is concerned with effects on a threatened and endangered (T&E) species or a set of species of special concern or habitat of special concern. In these cases, assessment endpoints are defined at the individual level because of the high degree of legal and societal concern with which these species or habitats are regarded. Assessments of effects at higher levels of biological organization (i.e., communities and ecosystems) must primarily address physical disturbance because there is little information on toxic effects at these levels. An additional difficulty associated with assessment of effects at higher levels of organization is that available toxicity data are generally inconsistent; however, functional system redundancy tends to buffer ecosystem processes from toxic effects, and the higher level taxa used as endpoint receptor species tend to integrate the effects on ecosystem processes.

2.1.4.1 Endpoint receptor species

Terrestrial endpoint receptor species are discussed individually in the following list:

Vascular Plants. Terrestrial vascular plant populations were selected as representative receptor populations because plants are immobile and receive their nutrients and water from a fixed area of the soil medium; therefore, potential contaminant exposure can be associated with a specific site. In addition, benchmarks are available for evaluation.

Soil Macroinvertebrates. Soil macroinvertebrates are representative of animals that live in intimate contact with the soil environment. The earthworm (*Lumbricus* sp.) was selected as the representative receptor species for soil invertebrates because benchmark values are available for evaluation.

Herbivorous Mammalian Wildlife. Herbivorous wildlife includes species that subsist primarily on plant material. The meadow vole (*Microtus pennsylvania*) was selected as a receptor species because home ranges potentially could incorporate most or all sites in WAG 28.

Omnivorous Mammalian Wildlife. Omnivorous animals subsist on both plant and animal material. The white-footed mouse (*Peromyscus leucopus*) was selected as the omnivorous receptor species because it is common on most sites and has a range small enough that its activities can be associated with a specific site. In addition, benchmark values are available for evaluation.

Vermivorous Mammalian Wildlife. Vermivorous animals subsist primarily on earthworms. The use of these species for evaluation is a natural extension of the use of earthworms as the representative receptor species for soil invertebrates. The short-tailed shrew (*Blarina brevicauda*) was selected as the vermivorous receptor species because it is common at most sites, has a home range small enough that its activities can be associated with a specific site, and has benchmark values available for evaluation. In addition, exposure for this receptor is likely to be higher than for most other small mammals because of its high metabolic rate, high percentage of invertebrates in its diet, and high soil ingestion rates.

Carnivorous Mammalian Wildlife. Carnivorous animals subsist on other mammals. The longtailed weasel (*Mustela frenata*) was selected as the carnivorous receptor species because it is most likely to live in both grassy and wooded habitats. While the home ranges for all carnivores tends to be larger than many individual sites, the long-tailed weasel has a smaller home range than other mammalian carnivores likely to occur at the facility.

Migratory birds were not designated as representative receptor species, because these species use the site far less frequently than the resident mammals evaluated.

2.1.4.2 Special endpoints

The COE (1994a, 1994b) documents, Environmental Investigations at the Paducah Gaseous Diffusion Plant and Surrounding Area, McCracken County, Kentucky, Vol. II, Wetland Investigation and Vol. III, Threatened and Endangered Species, were consulted to determine the potential for occurrence of two categories of special ecological endpoints considered by the BERA—floodplains and wetlands (special habitat) and T&E species (special receptors). In addition, a request was submitted to the Kentucky State Nature Preserves Commision (KSNPC), National Hertiage Program, for the most current state listing of rare, T&E species within the PGDP property boundaries. Two federally listed or candidate T&E species have been reported at PGDP. These are the Indiana bat (Myotis sodalis) and copper belly water snake (Nerodia erythrogaster neglecta). Based on the most current information from the KSNPC, nine occurrences of four plant and animal species and no occurrences of the exemplary natural communities that are monitored by KSNPC are reported as occurring within the Heath, Kentucky, U.S. Geological Service quandrangle. These listed or special concern species are the cream wild indigo (Baptisia bracteata var. leucophaea), compass plant (Silphium laciniatum var. robinsonii), redspotted sunfish (Lepomis miniatus), and northern crawfish frog (Rana areolata circulosa). No site-specific wetland or T&E species surveys have been conducted for WAG 28 sites.

2.1.5 Identification of Potential Exposure Pathways

2.1.5.1 Terrestrial

The following three potential terrestrial exposure pathways are considered in this BERA:

- Plants are in intimate association with the analyte-containing growth medium (soil), which is the major potential source of exposure. The analytes associated with the soil solution are in physical contact with plant roots in the soil and may enter the root with soil water. Plants are in turn eaten by herbivores.
- Earthworms, as representatives of soil-dwelling macroinvertebrates, are in direct contact with contaminant-containing soil. The outer cuticle is in contact with analytes associated with soil particles and in soil solution, and the earthworm gut is in contact with soil as it is ingested during feeding. Earthworms may then be eaten by first-order predators (e.g., shrew).
- Terrestrial wildlife may also consume contaminated soil by incidental ingestion while feeding and/or burrowing. It is not believed that wildlife receptors receive significant exposure via inhalation or dermal contact. Because such species are fur-covered, little if any direct exposure to dermal surfaces can occur. Exposure could occur through grooming or inhalation of dust, but these exposure routes are accounted for as incidental ingestion of soil. Omitting dermal contact as an exposure route to be quantitatively evaluated is a practice that is widely accepted in the field of ecological risk analysis. Further, exposure parameters and toxicity values for dermal exposure are generally not available. Inhalation of contaminated air contributes minimally to overall exposures at sites such as those in WAG 28 where VOCs are not significant contaminants of surface soils.

2.1.6 Conceptual Site Model

The ecological conceptual site model graphically represents the relationships between the contaminant sources and the endpoint receptors. It integrates the information in the other sections of the problem formulation step.

A generalized conceptual site model of possible exposure pathways for ecological receptors at sites in WAG 28 is provided in Fig. 2.1. Most sites currently provide limited ecological habitat (grass and small shrubs). In the future, it is possible habitat quality will improve, allowing the establishment of a greater diversity of species. The discussion in this section concerns current and hypothetical future exposures. Note that only terrestrial exposures are evaluated in this assessment, because aquatic habitats do not presently occur in WAG 28.

Surface soils at WAG 28 sites may have been contaminated during plant operations. Contaminants present in surface soils may leach into subsurface soils and then into the groundwater underlying the site. Contaminants in soils may also have been historically transported to nearby creeks via surface runoff.

Earthworms may be exposed to contaminants in surface soil through soil ingestion and direct contact with soil. Terrestrial plants may be exposed to contaminants in surface soil through direct contact of the roots with the soil and through root uptake. The short-tailed shrew may be exposed to contaminants in surface soil through incidental ingestion of soil and through ingestion of earthworms that may bioaccumulate contaminants in their tissues. The white-footed mouse may be exposed to contaminants in surface soil through incidental ingestion of soil and through ingestion of both plants and earthworms that may bioaccumulate contaminants in their tissues. Meadow voles may be exposed to contaminants in surface soil through incidental ingestion of soil and through ingestion of plants that may bioaccumulate contaminants in their tissues. Meadow voles may be exposed to contaminants in surface soil through incidental ingestion of soil and through ingestion of plants that may bioaccumulate contaminants in their tissues. Long-tailed weasels may be exposed to contaminants in surface soil through incidental ingestion of soil and through ingestion of plants that may bioaccumulate contaminants in their tissues. Long-tailed weasels may be exposed to contaminants in surface soil through incidental ingestion of soil and through ingestion of soil and through incidental ingestion of soil and through ingestion of soil and through incidental ingestion of soil and through ingestion of soil and through incidental ingestion of soil and through ingestion of soil and through incidental ingestion of soil and through ingestion of soil and through incidental ingestion of soil and through ingestion of soil and through incidental ingestion of soil and through ingestion of soil and through incidental ingestion of soil and through ingestion of small mammals.

2.2 EXPOSURE ASSESSMENT

This section describes the current and future modes of exposure that may occur at WAG 28 sites, the methods used to estimate exposure, and the available exposure data for the BERA.

2.2.1 Routes and Mechanisms of Chemical Transport and Transformation

Analytes in surface soil may be transported downward through the soil by the percolation of rainwater and may be transported off site by wind or surface water runoff. Physico-chemical changes (e.g., pH) or microbial activity may cause the release of bound analytes or their degradation products. The presence of other analytes in the soil may have a synergistic or antagonistic effect on associated analytes; however, for the BERA, the conservative assumption is that soil invertebrates, plants, and terrestrial wildlife are exposed to measured levels of analytes in surface soil.

Assuming that there are no accidental or additional releases to PGDP watersheds, chemical concentrations to which terrestrial plant and wildlife receptors may be exposed should decrease over time. Contaminants in surface soil may decrease because of leaching or natural degradation. Metals may adsorb to mineral as well as organic components of soil. Therefore, it is assumed that soil contaminant concentrations will not increase in the future and will likely decrease. For a conservative evaluation of future exposures, it is assumed that future surface soil concentrations will be similar to current concentrations.

2.2.2 Description of Exposure Models

Exposure models for specific exposure pathways are the same for each site where they are applicable. Exposure of soil invertebrates and terrestrial plants is evaluated based on a comparison of measured media concentrations to appropriate benchmarks; therefore, explicit exposure models for these receptors are not required. Exposure models are used to evaluate contaminant intake for terrestrial wildlife. Methods and models used in the BERA are described in the following sections.

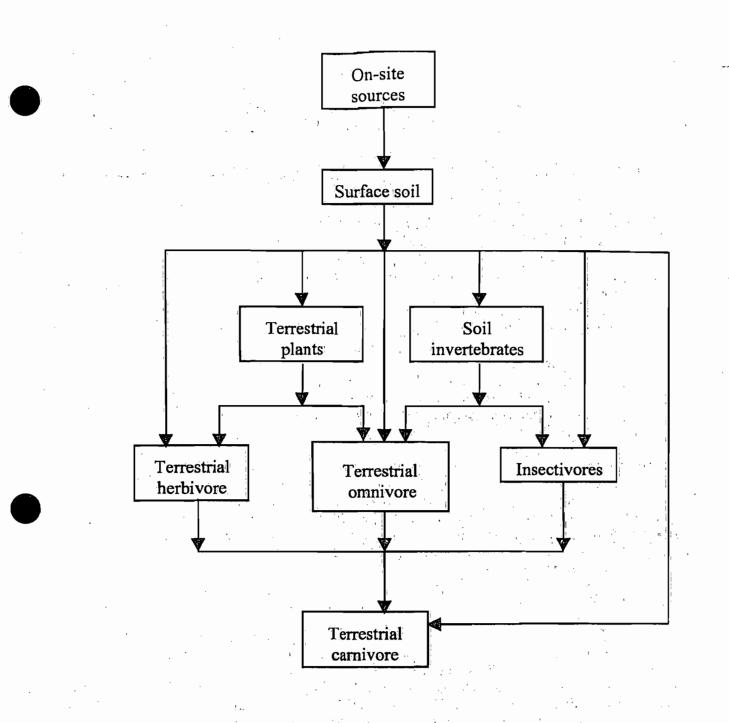


Fig. 2.1. Generalized ecological conceptual site model for WAG 28

المنافع من المالية في المراجع من الأمور منه المعنون من معنون من من عن من عن المراجع من المراجع من المراجع من ال يعاد الميجاد الله المنافع المراجع من المعنون منه بلا من المعنولية المراجع من من يوني من المعنون من المعنون من ا المنافع من المراجع من المراجع من المعنون من المعنون من من من المراجع من من يوني من من المراجع من المراجع من الم

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2.2.2.1 Nonradionuclide exposures

The potential daily contaminant intake from all potential sources for terrestrial wildlife is estimated using the following generalized equation (Sample and Suter 1994):

$$Intake = \left[IR_{food} \left(C_{plant} * P_{plant} + C_{invert} * P_{invert} + C_{mamm} * P_{mamm}\right) + \left(C_{soll} * IR_{soll}\right)\right] / BW$$

where:

Intake = estimated daily dose in mg/kg/day for the receptor IR_{food} = daily food ingestion rate (kg/day) C_{plant} = analyte concentration in plants (mg/kg, wet weight) ($C_{soil} \times B_v$) C_{invert} = analyte concentration in soil invertebrates (mg/kg, wet weight) [$C_{soil} \times$ uptake factor (terrestrial)] C_{mamm} = analyte concentration in small mammalian prey (mg/kg, wet weight) [$C_{soil} \times$ soil-totissue uptake factor or BAF × ($C_{plant} \times P_{plant} + C_{invert} \times P_{invert} + C_{soil} \times P_{soil}$)] C_{soil} = analyte concentration in soil (mg/kg, dry weight) IR_{soil} = rate of ingestion of soil (kg/day) P_{plant} = fraction of plant material in the receptor's diet (unitless) P_{invert} = fraction of plant material in the receptor's diet (unitless) P_{plant} = fraction of plant material in the receptor's diet (unitless)

BW = body weight (kg)

Parameter values required to estimate analyte exposure for terrestrial wildlife are presented in Tables 2.2–2.5. Body weights and food ingestion rates for each endpoint receptor species were obtained from Sample and Suter (1994). Because surface water is not available at the sites under investigation, ingestion of drinking water is not considered in the BERA. Furthermore, contaminant doses from drinking water are generally minor relative to doses from food and soil. Soil ingestion rates were obtained from the open literature for all species. It is assumed that all food and soil ingested by wildlife originate from the contaminated area.

2.2.2.2 Radiological exposures

In the BERA, it is assumed that all parts of an organism are exposed equally to radionuclide energies. While ecological receptors are exposed to radiation from natural sources, doses were only quantified for radiation from nuclides detected at WAG 28 sites. Radiation dose rates (mrad/day) from radionuclide exposures were calculated for plants, earthworms, and representative terrestrial wildlife species using methodology adapted from Blaylock et al. (1993) and Baker and Soldat (1992). Dose rates from internal exposures via ingestion of food and soil and inhalation of dust were evaluated, as were dose rates from external exposures via soil.

The representative terrestrial wildlife species selected as endpoints for the radiological assessment were the same as those for the chemical data assessment (i.e., terrestrial plants, earthworm, meadow vole, white-footed mouse, short-tailed shrew, and long-tailed weasel). Life history parameters used in the radiological assessment were identical to those used for the chemical data assessment (Tables 2.2–2.5). In addition, it was necessary to assume species-specific values for fraction of time spent above and below ground. The short-tailed shrew, white-footed mouse, meadow vole, and long-tailed weasel were assumed to spend 75 percent of their time above ground and 25 percent below the soil surface in dens or burrows.

The general methodology and the equations specific to each exposure route used in estimation of dose rates for biota are described in this section. Equations used in this assessment estimate the daily dose

under current conditions. Dose from alpha, beta, and gamma emissions (only beta and gamma for external exposures of earthworms and plants and only gamma for external exposures of wildlife receptors) were calculated for each radionuclide, including the dose rates from all short-lived daughter products. Dose from each radionuclide was then summed over all exposure routes and all radionuclides to arrive at the overall dose received for each receptor at each site.

External Exposures—Direct Radiation from Soil. The equation for estimating aboveground external dose rates (mrad/day) for terrestrial receptors exposed to contaminated soil uses dose coefficients published by Eckerman and Ryman (1993). Dose rate reduction factors are used to account for the fraction of time the receptor spends above ground. Dose coefficients assume the source region is a smooth plane (Eckerman and Ryman 1993), but this is rarely the case in a terrestrial habitat. A representative average dose reduction factor for ground roughness is 0.7 (Eckerman and Ryman 1993). For the shrew and mouse, relatively small mammals that are effectively much closer than 1 m to the source, an elevation correction factor of 2 was applied to account for the increased dose expected at ground level relative to the effective height of a standard human used to derive the dose coefficients. For plants, it was assumed that the dose represents that to the reproductive part of the plant with an effective height similar to that of the standard human. The following equation is for aboveground dose from external exposures for a plant or wildlife receptor:

 $\mathbf{D}_{abovegrd} = \mathbf{F}_{above} \mathbf{F}_{ruf} \sum \mathbf{C}_{soil,i} \mathbf{D} \mathbf{F}_{grd,i} \mathbf{CFb} \mathbf{ECF}$

 $D_{abovegrd}$ = external dose rate to receptor from aboveground exposures to contaminated soil (mrad/day)

 F_{above} = dose rate reduction factor accounting for the fraction of time the receptor spends above ground (unitless)

 F_{ruf} = dose rate reduction factor accounting for ground roughness (unitless) [Representative average of 0.7 Eckerman and Ryman (1993) used for this assessment]

 $C_{\text{soil:}i} = \text{activity of radionuclide } i \text{ in surface soil (pCi/g)}$

where:

 $DF_{grd,i}$ = dose coefficient for radionuclide *i* in soil contaminated to depth of 15 cm [(Table III.6, Eckerman and Ryman (1993)] (Sv/s per Bq/m³)

CFb = conversion factor to change Sv/s per Bq/m³ to mrad g/pCi d. equals 5.12E+14

ECF = the elevation correction factor to adjust dose coefficients to value representative of effective height of animal above ground (unitless)

Dose from alpha radiation is not a concern for external sources, as alpha radiation lacks penetrating power. The effective dose coefficients from Eckerman and Ryman (1993) incorporate both high energy beta and gamma emissions. Radionuclide-specific parameters are provided in Table 2.6. The lower of the 95% UCL and the maximum detected concentration in surface soil within a site was used in estimating the dose from external exposures.

Belowground exposures are calculated assuming immersion in a continuous soil medium. Dose coefficients were unavailable for the immersion scenario, so exposures were modeled as dose to soil adjusted for absorption by a small volume of tissue. The exposure fraction reflects the fraction of time the receptor spends below ground. Only gamma radiations with energies greater than 0.01 MeV were evaluated for wildlife receptors as those with lower energies are unlikely to penetrate skin. Both beta and gamma radiations were evaluated for earthworms. The following equation is for belowground external exposures of earthworms and wildlife receptors:

 $D_{belowgrd} = 1.05 F_{below} \sum C_{soll} \in CFa$

where:

 $D_{belowgrd}$ = external dose rate to earthworm or wildlife receptor in burrow from contaminated soil (mrad/day)

1.05 = conversion factor to account for immersion in soil versus water (Estimated value; Keith Eckerman, Health Sciences Research Division, Oak Ridge National Laboratory, personal communication, June 1996)

 F_{below} = dose rate reduction factor accounting for the fraction of time the receptor spends below ground (unitless)

 $C_{\text{soil, i}} = \text{activity of radionuclide } i \text{ in surface soil (pCi/g)}$

 ϵ_i = energy for beta or gamma emissions by nuclide *i* (MeV/nt)

CFa = conversion factor to convert MeV/nt to g mrad/pCi/d (5.12E-2)

Internal Exposures—Ingestion. Wildlife receptors may receive internal radiation doses after ingesting contaminated prey or soil or after inhaling contaminated dust. Blaylock et al. (1993) provides an equation for estimating the internal dose to fish contaminated with radionuclides. This equation can be modified to address consumers eating a variety of prey types and ingesting soil as well as plants and invertebrates taking up contaminants directly from the soil:

$$\mathbf{D}_{ing} = \sum \mathbf{QF} \mathbf{C}_{tissue} \in_{i} \mathbf{CFa} \mathbf{AF}$$

where:

 D_{ing} = internal dose rate received after ingestion of contaminated prey and soil (mrad/day) QF = quality factor to account for the greater biological effectiveness of alpha particles (20 for alpha; 1 for beta and gamma emissions; unitless) C_{tissue} = activity (pCi/g) of radionuclide *i* in tissue of organism ϵ_i = energy for alpha, beta, or gamma emissions by nuclide *i* (MeV/nt) CFa = conversion factor to convert MeV/nt to g mrad/pCi/day (5.12E-2) AF = absorption factor (unitless)

Radionuclide activity in tissue was determined a number of ways depending upon data availability. Measured plant, earthworm, and small mammal data were unavailable. Soil-to-tissue uptake factors were available for a number of analytes. When they were available, tissue concentrations were calculated as discussed in Sect. 2.2.3.3. When soil-to-tissue uptake factors were unavailable for wildlife receptors, literature-derived food-to-tissue transfer factors were used to obtain terrestrial biota tissue concentrations.

When uptake factors were unavailable for specific radionuclides, values were derived from those for related isotopes. Uptake factors used in this assessment are provided in Table 2.7. It was assumed that uptake of radionuclides from ingested food and soil was similar.

Absorbed energy fractions for alpha radiations were assumed as unity for all receptors. Absorption fractions for beta radiations were assumed as unity for wildlife receptors, but beta absorption fractions for large insects from Blaylock et al. (1993) were used for plants (assuming small reproductive parts of greatest concern) and earthworms. Absorption fractions for gamma radiations for plants and earthworms were also obtained from those for large insects presented in Blaylock et al. (1993). Absorption fractions for gamma radiations derived for infant or 1-yr-old humans using the methodology described in Cristy and Eckerman (1987) were used for wildlife receptors. Table 2.6 presents absorption factors used for each receptor-radionuclide combination evaluated in this report.

Dose from internal exposures was calculated for alpha, beta, and gamma energies of each radionuclide. Energies were obtained from Eckeman and Ryman (1993) and are provided in Table 2.6. Because different types of radiation differ in their relative biological effectiveness per unit of absorbed

dose, a quality factor derived from data on humans is normally applied (NCRP 1987). A quality factor of 1 is used for beta and gamma radiation and 20 for alpha radiation (Blaylock et al. 1993).

Internal Exposures—Inhalation. Wildlife species using burrows receive an additional internal dose from inhalation of dust originating from contaminated soil. Intake of radionuclide i by inhalation is estimated as follows (DOE 1995c):

where:

 D_{inh} = internal dose rate from inhalation of contaminated soil (mrad/day) F_{exp} = dose reduction factor for fraction of time receptor spends below ground (unitless) A = mass of respirable dust per volume of air breathed [0.1 g/m³ (DOE 1995)] AD = air density [1200 g/m³ (Eckerman and Ryman 1993)] ε_i = alpha, beta, or gamma radiation energies for radionuclide *i* (MeV) CFa = conversion factor to go from MeV to mrad g/pCi/d (5.12E-2) AF = absorption factor (unitless)

 $\mathbf{D}_{inh} = \mathbf{QF} \ \mathbf{F}_{below} \sum \mathbf{C}_{soll,i} \ \mathbf{A} \ \frac{1}{\mathbf{AD}} \in_i \mathbf{CFa} \ \mathbf{AF}$

Healy (1980) suggests that 0.0001 g/m³ is used as a conservative value when addressing human exposures to dust. Because burrowing animals are likely to spend a greater portion of their time in a confined space (burrow) than humans and are physically closer to the soil surface, an air mass loading of 0.1 g/m³ was selected as a conservative estimate of the mass of respirable dust (A) to which these animals may be exposed.

Total internal exposures are obtained by adding ingestion and inhalation dose rates over all radionuclides, including all short-lived daughter products.

2.2.3 Quantification of Exposure

2.2.3.1 Plant exposure

Vegetation is exposed to analytes that have been deposited in the soil. Some metal elements are more readily available for uptake by plants from soil pore water than others. Availability depends on a number of factors including the solubility of the source compound and interactions with soil constituents (e.g., organic material and clay) as well as interactions with other analytes. Organic analytes may interact strongly with soil organic matter and therefore be of limited availability for plant uptake. The use of reported concentrations for the BERA is consistent with the application of benchmarks that are derived from literature values representing essentially total (or added) soil metal concentrations.

A comprehensive analysis of the exposure of plants to the inorganic and organic analytes in the soils at WAG 28 sites requires information about important soil parameters (e.g., types and quantities of clay and organic components, pH, moisture content) and characteristics of the analyte compounds (e.g., water solubility, lipid solubility); however, because only partial information is available, it is assumed that the reported concentrations of the analytes are available for plant uptake at any one time, and analytes will therefore be assessed for potential negative impact on plant growth at reported concentrations.

Analytes have distinctive vertical distributions in soil that reflect interactions between the soil and the analyte as controlled by the chemical and physical nature of the soil and the quantity and chemical nature of the analyte. The exposure of plant roots to an analyte in the soil depends on the aforementioned abiotic factors and growth characteristics of individual plants, such as rooting depth and density. Because only partial information is available, it is assumed that the plants are rooted entirely in the zone from

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which the soil was sampled for analysis (the top 15 cm), and all plants rely on that zone for their immediate water and nutrient requirements. Therefore, the RME for terrestrial plants in the soil medium is the maximum detected concentration.

2.2.3.2 Earthworm exposure

The same abiotic soil and chemical factors considered in the quantification of exposure of plants to soil contaminants are applicable to the quantification of exposure of earthworms to analytes in the soil. Earthworms may be exposed to analytes by dermal contact with soil and pore water and ingestion of soil. It is not possible to distinguish between and quantify the exposure to earthworms by these two pathways. It is assumed that earthworms spend their entire life span in soil with analyte levels represented by those in the soils sampled for analyses. Therefore, maximum detected concentrations are used to assess potential negative impacts of contaminated soils on earthworm populations.

2.2.3.3 Terrestrial wildlife exposure

Because data on the analyte concentrations in vegetation (a primary food of white-footed mice and meadow voles), earthworms (primary food of short-tailed shrews), and small mammals (primary food of long-tailed weasels) were not available, these values were estimated using soil-to-plant, soil-toearthworm, or soil-to-small mammal uptake factors or food-to-tissue biotransfer factors obtained from the following literature. Soil-to-plant uptake factors for inorganic analytes were obtained from Efroymson et al. (1998), International Atomic Energy Agency (IAEA) (1994), National Council on Radiation Protection and Measurement (NCRP) (1989), or Baes et al. (1984), in that order. Soil-to-invertebrate uptake factors were obtained from Sample et al. (1998a), Menzie et al. (1992), or Beyer and Stafford (1993). Soil-to-small mammal uptake factors were obtained from Sample et al. (1998b). Food-to-tissue biotransfer factors for estimating mammal tissue concentrations were obtained from IAEA (1994), NCRP (1989), or Baes et al. (1984). When available, regression equations provided by Efroymson et al. (1998) and Sample et al. (1998a,b) were used to estimate tissue concentrations. These equations describe the relationship between tissue concentration and soil concentration for plants, invertebrates, and small mammals for a number of inorganics (Tables 2.8, 2.9, and 2.10). Travis and Arms (1988) report that uptake factors for organic chemicals in vegetation are inversely proportional to the square-root of the Kow, and mammalian biotransfer factors for organic chemicals are directly proportional to Kow. Kow values were used to estimate plant uptake and mammalian biotransfer factors for organic chemicals. Uptake factors and biotransfer factors are presented in Table 2.11.

To estimate contaminant exposure experienced by short-tailed shrews, the following assumptions were made:

- body weight = 0.015 kg
- food ingestion = 0.009 kg/day (fresh weight)
- soil ingestion = 0.00117 kg/day (dry weight)
- diet consists 100 percent of earthworms or soil invertebrates
- contaminant concentration in earthworms is representative of that in other invertebrate prey

To estimate contaminant exposure experienced by the white-footed mouse, the following assumptions were made:

- body weight = 0.022 kg
- food ingestion = 0.0034 kg/day (fresh weight)
- soil ingestion = 0.000068 kg/day (dry weight)

- diet consists 50 percent of earthworms or soil invertebrates and 50 percent herbaceous plant material
- contaminant concentration in earthworms is representative of that in other invertebrate prey

To estimate contaminant exposure experienced by the meadow vole, the following assumptions were made:

- body weight = 0.044kg
- food ingestion = 0.036 kg/day (fresh weight)
- soil ingestion = 0.00086 kg/day (dry weight) (2.4% of diet)
- diet consists 100 percent of herbaceous plant material

To estimate contaminant exposure experienced by the long-tailed weasel, the following assumptions were made:

- body weight = 0.15 kg
- food ingestion = 0.02 kg/day (fresh weight)
- soil ingestion = 0.003 kg/day (dry weight) (2.8 percent of diet)
- diet consists 100 percent of small mammals

Life history data and references for each receptor are provided in Tables 2.2–2.5. Point estimates of exposure to contaminants in each site were estimated for each endpoint using the RME (Table 2.1), the assumptions outlined above, uptake factors from Tables 2.7–2.11, and the exposure models described in Sect. 2.2.2.

2.3 EFFECTS ASSESSMENT

Ecological effects assessment involves the identification of known effects of contaminants on representative receptor populations through the use of conventional toxicity data, ambient media toxicity tests, and biological survey data. Because media toxicity and biological survey data are not available for the BERA, expected media concentrations must be compared with conventional toxicity data. This section discusses the toxicological evidence used in the risk characterization section (Sect. 2.4) to evaluate risks to terrestrial plants and animals.

In the chemical toxicity section (Sect. 2.3.1), the types, development, and interpretation of appropriate toxicological benchmarks are discussed. Conventional toxicity data consist of published values for toxicity of contaminants to test species; these data are generally not readily useful for ecological risk assessment. They are used in development of toxicological benchmarks applied in the risk assessment to determine if biological effects are likely. By comparing contaminant concentrations detected in a medium at a site to benchmarks for that medium, the likelihood that contaminants may pose a risk can be estimated. Toxicological benchmarks for plants and earthworms are presented in Table 2.1. Toxicological benchmarks for terrestrial wildlife receptors are provided in Table 2.12. It should be noted that additional lines of evidence such as biological surveys and soil toxicity testing are generally desired for a BRA, but such data have not been collected. This is recognized as an uncertainty in the evaluation of potential risks to ecological receptors.

2.3.1 Evaluation of COPECs

The procedures for screening COPECs in surface soil are described in the following paragraphs. The results of the current and hypothetical future exposure assessment for WAG 28 sites are then discussed in subsequent sections. Chemicals that occur at concentrations that are safe for ecological receptors can be excluded as COPECs. Exposure concentrations that are deemed to be safe are referred to as

2.3.1.1 Chemical toxicity data for terrestrial biota

Contaminant exposures experienced by terrestrial biota are compared to toxicological benchmarks to assess potential ecological effects. Toxicological benchmarks for plants and soil invertebrates were obtained from Efroymson et al. (1997a,b). Total exposure estimates for wildlife are compared to NOAELS and LOAELs derived according to the methods outlined by Sample et al. (1996). Only studies of the effects of long-term, chronic oral exposures, whether in food or water, were used. To make the LOAELs relevant to possible population effects, preference was given to studies that evaluated effects on reproductive parameters. In the absence of a reproduction endpoint, studies that considered effects on growth, survival, and longevity were used.

2.3.1.2 Effects data for organisms exposed to radionuclides

The IAEA recommends limiting the radionuclide dose for terrestrial organisms to 100 mrad/day (IAEA 1992). Studies evaluating reproductive success and survival were used to determine the dose limit. Species-specific effects data were not available, so 100 mrad/day was selected as the threshold dose for all representative wildlife receptors. A dose rate of this magnitude is unlikely to cause observable changes in terrestrial animal populations (IAEA 1992). Higher dose rates may result in impaired reproduction or reduced survivorship. A dose rate of 1 rad/day is generally considered protective of plant and invertebrate populations (IAEA 1992; Barnthouse 1995) based on studies of productivity and community characteristics. This dose rate is unlikely to cause observable changes in terrestrial plant populations (IAEA 1992). Higher dose rates may result in reduced productivity or changes in species composition within communities; therefore, 1 rad/day was selected as the threshold dose for effects on plant and invertebrate populations. Invertebrates tend to be less radiosensitive than plants or animals, and indirect responses to radiation-induced vegetation changes appear more critical than direct effects from radiation (IAEA 1992).

2.4 RISK CHARACTERIZATION

Risk characterization is the phase of ecological risk assessment in which information concerning exposure (Sect. 2.2) and information concerning potential effects of exposure (Sect. 2.3) are integrated to estimate risks (the likelihood of effects given the exposure). Standard risk characterization in ecological risk assessment is performed by a weight-of-evidence analysis. The principal lines of evidence concerning effects are single chemical toxicity data that indicate the toxic effects of the concentrations measured in site media, media toxicity data that indicate whether the contaminated media are toxic under controlled conditions, and biological survey data that indicate the actual state of the receiving environment. Media toxicity data and biological surveys were not available for this evaluation; therefore, the assessment is based on single-chemical toxicity data only. The result is a BERA that is a conservative estimate of site risk.

The following limitations that may induce either false positive or false negative results must be considered when interpreting the results of the BERA:

- Combined toxic effects of analytes (synergistic or antagonistic) are not considered.
- Analysis of ambient media may miss periods of high concentrations in temporally variable media or locally high concentrations in spatially variable media.

For some analytes, limits of detection may be above toxic concentrations.

Additional uncertainties involved in calculating and interpreting risk to biota based on singlechemical toxicity data are discussed in detail in Sect. 2.5.

Risk characterization is performed for each assessment endpoint by (1) screening contaminants against toxicological benchmarks, (2) estimating effects of contaminants retained by the analysis, and (3) listing and discussing the uncertainties in the assessment.

Many of the analytes commonly identified during an RI also occur naturally in the environment. The concentrations of these analytes found at local background (reference) sites are normally assumed to be nonhazardous. To ensure that risk management decisions are based on the risks posed by site-related contaminants, the detected, naturally occurring inorganic and radionuclide analytes are compared to background values. Synthetic organic compounds are anthropogenic in origin; therefore, any synthetic organic compound that is detected and validated is considered above background.

In the screening against benchmarks portion of risk characterization, the analyte concentrations measured in abiotic media (soil) or estimated doses in the case of wildlife receptors are compared to ecotoxicological benchmarks to derive HQs by the following formula:

HQ = media concentration or exposure dose/toxicological benchmark

HQs greater than 1 suggest that the chemical is potentially hazardous to the endpoint biota. HQs less than 1 suggest that the chemical is nonhazardous and does not need to be considered further. HQ calculations are performed by medium for each endpoint receptor population.

Toxicological benchmark values derived for contaminants found at PGDP and the Oak Ridge Reservation (Sample et al. 1996; Efroymson et al. 1997a,b) are used in this assessment. Benchmark development is an ongoing process, and although the methods for derivation remain the same, the most current benchmarks are used. The ecological risks posed by contaminants are discussed as they relate to each endpoint population, including multiple pathways of exposure (e.g., food, soil) when applicable. Benchmarks are not available for all chemical/receptor combinations. Results of the comparison of surface soil concentrations to benchmarks for plants and invertebrates are reported in Table 2.1. Results for terrestrial wildlife are in Table 2.14. Table 2.13 provides a summary of benchmark exceedences for those chemicals detected above background levels.

Because adverse effects associated with radionuclides in soil were evaluated differently than nonradiological contaminants, the results and characterization of potential ecological risks because of radionuclides are discussed in separate sections.

2.4.1 SWMU 99a

This site is covered by gravel and asphalt/concrete. Current exposure of terrestrial biota to surface soil is not evaluated; however, existing surface soil data are used to estimate potential future risks, assuming suitable habitat develops at the site over time.

Fourteen inorganic and 19 organic analytes (including 15 PAHs and 3 PCBs) were detected in surface soil from SWMU 99a (Table 2.1). Barium, beryllium, chromium, nickel, and zinc are the only inorganics that exceed background levels. Background values are not available for lithium or stontium.

Assuming current surface soil concentrations represent future surface soil concentrations at this site, widespread effects on terrestrial biota are not expected. However, barium is present at a concentration 12× background at Station 099-014 and may pose a risk to plants in this location. Barium concentrations at 12 other samples within this site are near or below background levels and below levels of concern for plants.

Estimated doses from exposure to radionuclides in soil are below recommended dose rate limits for wildlife, but dose rates for plants and soil invertebrates are higher than the recommended dose rate limit of 1 rad/day. Technetium-99 is the radionuclide of concern.

2.4.1.1 Risks to plants

Barium, chromium, and zinc were the only analytes detected above background that exceed toxicological benchmarks for plants (Table 2.13). Barium is present at 2470 mg/kg at Station 099-014, exceeding background levels by 12× and exceeding the benchmark for phytotoxicity by almost a factor of 5. However, barium at other stations in this site is found at concentrations more than an order of magnitude lower and well below levels posing a risk to plants. The maximum zinc concentration is only $2.5 \times$ background, and the mean concentration is below background (Table 2.1), suggesting risks are unlikely. While chromium exceeds the benchmark for plants in this area, confidence in the chromium benchmark is low because limited data are available for derivation of the value (Efroymson et al. 1997a). In addition, the studies used to derive the benchmark value investigated the effects of chromium added to soil as Cr(VI). Chromium(VI) is more soluble and available to plants than Cr(III) and is considered the more toxic form (Smith et al. 1989). In soils with a normal eH and pH range, Cr(VI) is likely to be reduced to the less available Cr(III). Thus, there is considerable uncertainty in determining whether chromium concentrations result in toxicity to plants. Given the low HQs and concentrations near or below background (with the exception of one isolated high concentration of barium), it appears that while there may be risks to individual plants in the areas of highest concentration, future risks to the terrestrial plant community at this site are likely to be low.

2.4.1.2 Risks to earthworms

Chromium and zinc were the only analytes detected above background that exceed toxicological benchmarks for soil invertebrates (Table 2.13). Confidence in the chromium benchmark for soil invertebrates is low (Efroymson et al. 1997b). In addition, the benchmark is based on Cr(VI). Chromium(VI) can pass through cell membranes more easily than can Cr(III), but without a better understanding of chromium transformations, transport, and reactions within cells, it is difficult to separate effects of different forms (Efroymson et al. 1997b). Van Gestel (1992) reported 32 ppm Cr(III) reduced earthworm growth by 30 percent. The maximum chromium concentration in SWMU 99a is approximately $1.5 \times$ this value, but the mean and 95% UCL concentrations are less than a third. While there is considerable uncertainty regarding effects of chromium on earthworms, chromium at SWMU 99a may not be a significant concern. Zinc only slightly exceeds the soil invertebrate benchmark (by less than a factor of 2), and the mean zinc concentration is below background. Given the low HQs and concentrations near or below background, it appears that while there may be risks to individual plants in the areas of highest concentration, future risks to the soil invertebrate community at this site are likely to be low.

2.4.1.3 Risks to terrestrial wildlife

No analytes exceed LOAELs for wildlife receptors at this site (Table 2.13). Barium results in estimated daily doses slightly exceeding NOAELs for shrews and meadow voles (Table 2.14), but as discussed for plants, one sample location has a barium concentration more than an order of magnitude

higher than other stations. Risks to terrestrial wildlife populations from future exposures to surface soil at SWMU 99a are not expected.

2.4.1.4 Risks to terrestrial organisms from radiological exposures

Estimated dose rates for all terrestrial wildlife receptors are well below the effects threshold of 100 mrad/day for wildlife (Table 2.15). Estimated dose rates for plants and soil invertebrates exceed their associated effects threshold of 1 rad/day. The risk driver in this case is the internal dose from technetium-99. Technetium-99 was detected at only 3 of 16 sample locations in SWMU 99a, and the maximum detected activity came from one of two samples (082-014 and 082-015) recovered during excavation of a drain pipe.

2.4.2 SWMU 193a

This SWMU is inside the security fence in a highly industrialized area, but because it is mostly grass-covered, both current and future exposures are evaluated. Current exposures assume ecological receptors reside at the site.

Thirteen inorganics and 14 organics (10 PAHs and 4 phthalates) were detected in SWMU 193a surface soil. Chromium is the only inorganic detected above background levels. Background concentrations are unavailable for lithium and strontium.

Risks to terrestrial receptors are not expected from current or future exposures at this site. No radionuclides were detected, and only chromium, for which toxicological benchmarks are likely highly conservative, exceeds levels of potential concern.

2.4.2.1 Risks to plants

Chromium is the only analyte in SWMU 193a surface soil that exceeds background concentrations and toxicological benchmarks for plants (Table 2.13). As discussed for SMWU 99a, confidence in the chromium benchmark is low because limited data are available for derivation of the value (Efroymson et al. 1997a). In addition, the studies used to derive the benchmark value investigated the effects of chromium added to soil as Cr(VI). Chromium(VI) is more soluble and available to plants than Cr(III) and is considered the more toxic form (Smith et al. 1989). Thus, there is considerable uncertainty in determining whether chromium concentrations result in toxicity to plants. The site currently supports grassy vegetation. Adverse effects on plants do not appear likely at this site.

2.4.2.2 Risks to earthworms

Chromium concentrations exceed benchmarks for soil invertebrates (Table 2.13). Confidence in the chromium benchmark is low because it is based on only five reported concentrations resulting in toxicity to earthworms (Efroymson et al. 1997b). The relative toxicity of Cr(III) versus Cr(VI) to earthworms is not clear.

2.4.2.3 Risks to terrestrial wildlife

No analytes exceed background levels and NOAELs or LOAELs for terrestrial wildlife exposed to contaminants in surface soil at SWMU 193a (Tables 2.13 and 2.14). Therefore, no risks are anticipated from current or future exposures for these receptors.





2.4.2.4 Risks to terrestrial organisms from radiological exposures

No radionuclides were detected in samples from SWMU 193a; therefore, there is no risk anticipated to ecologicals receptors from radionuclides at this site.

2.4.3 SWMU 193b

This area is covered by gravel, providing no habitat for ecological receptors; however, future exposures to surface soil contaminants at this location are evaluated assuming current soil concentrations represent those in the future and suitable habitat develops over time.

Thirteen inorganics and one organic (toluene) were detected in surface soil at SWMU 193b. Beryllium, chromium, and vanadium are the only inorganics that exceed background levels. Background values are unavailable for lithium and strontium.

Potential future risks from exposure of plants, soil invertebrates, and wildlife to chromium or vanadium were identified, but there is considerable uncertainty associated with the benchmarks available for chromium.

2.4.3.1 Risks to plants

Two analytes (chromium and vanadium) exceed background and benchmarks for plants exposed to contaminants in surface soil at this site (Tables 2.1 and 2.13). As noted in the discussion for SWMU 99a, confidence in the chromium benchmark is low, and it is based on the more soluble and toxic Cr(VI). Similarly, confidence in the vanadium benchmark is also low as no primary reference data describing toxicity of vanadium to plants grown in soil were found; however, Kabata-Pendias and Pendias (1984) reported unspecified toxic effects to plants grown in soil with 50 ppm added vanadium (Efroymson et al. 1997b). The maximum detected concentration of vanadium in SWMU 193b is 65 ppm.

While there is low confidence in benchmarks for both chromium and vanadium, concentrations of both analytes are elevated at this site and may pose future risks to terrestrial plants.

2.4.3.2 Risks to earthworms

Chromium concentrations exceed benchmarks for soil invertebrates (Table 2.13). Confidence in the chromium benchmark is low because it is based on only five reported concentrations resulting in toxicity to earthworms (Efroymson et al. 1997b). The relative toxicity of Cr(III) versus Cr(VI) to earthworms is not clear. However, chromium concentrations in surface soil at SWMU 193b are higher than at other WAG 28 sites and may pose a future risk to soil invertebrates.

2.4.3.3 Risks to terrestrial wildlife

Vanadium is the only analyte that exceeds both background levels and LOAELs for a wildlife receptor. The only LOAEL exceeded is that for the short-tailed shrew, and the exposure concentration (based on the maximum detected concentration because there are only two samples from this site) results in an HQ only slightly greater than 1 (Table 2.13). Vanadium also exceeds NOAEL-based benchmarks for shrews, meadow voles, and long-tailed weasels (Table 2.14), and chromium exceeds the NOAEL for the shrew. Exceedance of the vanadium LOAEL for the shrew indicates potential risks from future exposure of terrestrial wildlife receptors to SWMU 193b surface soil, assuming vanadium concentrations remain the same or higher than current conditions and suitable habitat develops on the site.

2.4.3.4 Risks to terrestrial organisms from radiological exposures

No radionuclides were detected in samples from SWMU 193b; therefore, there is no risk anticipated to ecological receptors from radionuclides at this site.

2.4.4 SWMU 193c

This site is almost entirely covered with gravel and asphalt/concrete. It provides no current habitat for ecological receptors; however, future exposures to surface soil contaminants at this location are evaluated, assuming current soil concentrations represent those in the future and suitable habitat develops over time.

Twelve inorganics and no organics were detected in surface soil from SWMU 193c. Copper, lead, and zinc are the only inorganics that exceed background levels. Background values are not available for boron, lithium, or strontium.

Potential future risks from exposure of plants to boron, chromium, lead, and zinc and exposure of soil invertebrates to chromium were identified, but there is considerable uncertainty associated with the benchmarks available for boron and chromium.

2.4.4.1 Risks to plants

Maximum boron, chromium, lead, and zinc concentrations at SWMU 193c exceed benchmarks for phytotoxicity (Table 2.1); however, chromium is within background levels. As discussed previously, confidence in the chromium benchmark is low, and it is based on the more soluble and toxic Cr(VI). Chromium concentrations in SWMU 193c are low relative to other WAG 28 sites, and chromium was detected in only three of five samples. Confidence in the benchmark for boron is also low due to the limited number of studies available. Boron was detected in only one of five samples. Lead was also detected at only one of five sample locations at this site at a concentration less than 2× background, resulting in an HQ only slightly higher than unity. While the maximum zinc concentration exceeds the zinc benchmark, the 95% UCL concentration is less than half the benchmark value, suggesting that if there is a risk of adverse effects from zinc, it is unlikely to be a widespread concern at this site.

There appears to be a risk of potential adverse effects from future exposure of plants to SWMU 193c surface soil concentrations, but there is considerable uncertainty due to the toxicological data available for boron and chromium and to the near background, low HQ levels of lead and zinc.

2.4.4.2 Risks to earthworms

Chromium concentrations exceed benchmarks for soil invertebrates (Table 2.13); however, chromium is within background levels. Confidence in the chromium benchmark is low because it is based on only five reported concentrations resulting in toxicity to earthworms (Efroymson et al. 1997b). The relative toxicity of Cr(III) versus Cr(VI) to earthworms is not clear. Furthermore, chromium concentrations in surface soil from SWMU 193c are lower than at other WAG 28 sites, and chromium was detected in only three of five samples. It is unlikely that chromium in SWMU 193c surface soil will pose a future risk to soil invertebrates.

2.4.4.3 Risks to terrestrial wildlife

No analytes exceed NOAELs or LOAELs for terrestrial wildlife receptors at SWMU 193c. Therefore, no risks are anticipated should future exposures occur at this site.

2.4.4.4 Risks to terrestrial organisms from radiological exposures

Radioisotope data were not collected from SWMU 193c.

2.5 UNCERTAINTIES

The uncertainties associated with any BERA are extensive; however, the primary sources of uncertainty are (1) the paucity of ecologically relevant data necessary to estimate site-specific HQs, resulting in over-reliance on system models and default variables, (2) the necessity to evaluate risk to biota based only on the single-chemical toxicity data line of evidence, and (3) extrapolation from current to future conditions. The following sections discuss the uncertainties involved in the BERA.

2.5.1 Risks to Plants

The following are factors that create uncertainty in assessing the risk to plants posed by the COPECs in soils:

- Use of maximum detected concentration as exposure concentration—Because plants are immobile, the maximum detected contaminant concentration was used as the exposure concentration. While this is appropriate for evaluating potential risks to individual plants exposed to the maximum concentration, exposures for plants in other parts of the site may be overestimated.
- Bioavailability of elements—Soil sample extraction methods may remove quantities of elements and compounds greater than those available to plants. The double-acid extraction method used for RI sampling removes the exchangeable fraction of metals, thereby resulting in a concentration that reflects the total potential pool of contaminants, not that to which the plant is exposed at any one time. Under field conditions, these contaminants are in the soil solution and available for uptake in concentrations reflecting a dynamic equilibrium between the solid and liquid phase; therefore, it is difficult to assess the types of interactions that may occur between contaminants and plant roots under field conditions. This is confounded by the concentration- and species-dependent synergistic and antagonistic interactions between metals during uptake by roots and once inside plants. The analytical techniques also fail to differentiate between species of metals present in the soil (particularly arsenic, chromium, and mercury) that pose variable toxicity to plants and other life forms. Without specific analyses for these forms, or soil chemical and physical data sufficient to evaluate the probable occurrence of the species, it is not possible to accurately assess the risk posed by these contaminants at the site.
- Variable response to toxicants—Information on toxicity of contaminants to specific plant species and growth stages is generally not available. There is a considerable amount of variability between plant species and plant growth stages in tolerance to specific contaminants and combinations of contaminants. The literature from which benchmarks were derived is not based on experiments using plants found in ecosystems representative of site-specific conditions. It is difficult to extrapolate from agricultural crops in early growth stages, which are used in most of the published literature, to trees and other natural vegetation found on site.
- Multiple contaminant exposure—Because of a lack of understanding of the complex interactions between contaminants, benchmark levels are necessarily derived from experiments in which plants are exposed to single contaminants. Exposure to site soils involves multiple contaminant exposure that may not be adequately assessed on the basis of literature-derived, single contaminant benchmarks.

• Benchmark availability—Toxicity data for derivation of toxicological benchmarks were not available for all chemical-receptor combinations. While it is possible that chemicals without benchmarks could be a concern, there is no way to evaluate possible effects without site-specific toxicity testing. However, benchmarks are available for many priority contaminants.

2.5.2 Risks to Earthworms

The following are factors that create uncertainty in assessing the risk to soil macroinvertebrates (earthworms) posed by the chemicals in soils:

• Use of maximum detected concentration as exposure concentration—Because earthworms are relatively immobile, the maximum detected contaminant concentration was used as the exposure concentration. While this is appropriate for evaluating potential risks to individuals exposed to the maximum concentration, exposures for earthworms in other parts of the site may be overestimated.

- **Bioavailability of elements**—This factor is discussed in the previous section that focused on the uncertainty associated with evaluating risks to plants. Soil sample extraction methods may remove quantities of elements and compounds from the soil greater than those to which earthworms are actually exposed.
- Variable response to contaminants—There is variability between earthworm species and growth stages in terms of tolerance to specific contaminants and combinations of contaminants. The literature from which benchmarks were derived is not based on experiments using earthworm species known to be representative of those occurring in site soils.
- Multiple contaminant exposure—Toxicity benchmark concentrations are derived from experiments in which earthworms are exposed to single contaminants; however, multiple contaminant exposure occurs in most soils. This multiple exposure may not be adequately assessed on the basis of literature-derived benchmarks.
- **Benchmark availability**—Toxicity data for derivation of toxicological benchmarks were not available for all chemical-receptor combinations. While it is possible that chemicals without benchmarks could be a concern, there is no way to evaluate possible effects without site-specific toxicity testing. However, benchmarks are available for many priority contaminants.

2.5.3 Risks to Wildlife

The following are factors that create uncertainty in assessing the risk to terrestrial wildlife posed by the COPECs in soils:

- **Bioavailability of elements**—Bioavailability of contaminants was assumed to be comparable between soils from WAG 28 sites and the diets used in literature toxicity tests. Because bioavailability may not be comparable, exposure estimates based upon contaminant concentrations may either under- or overestimate the actual contaminant exposure.
- Extrapolation from published toxicity data—To estimate toxicity of contaminants at WAG 28 sites, it was necessary to extrapolate from studies performed on test species (i.e., mice and rats). While it was assumed that toxicity could be estimated as a function of body size, the accuracy of the estimate is not known. For example, shrews may be more or less sensitive to contaminants than mice because of factors other than metabolic rate.



Additional extrapolation uncertainty exists for those contaminants for which data consisted of tests that were subchronic in duration. An uncertainty factor of 10 was used to estimate chronic LOAELs. The uncertainty factor of 10 may either over- or underestimate the actual subchronic-chronic relationship.

Toxicity of PCBs to wildlife was evaluated using toxicity data from studies on Aroclor-1254. Because toxicity of PCB congeners can vary dramatically, the applicability of data for Aroclor-1254 is unknown; however, higher chlorinated forms such as Aroclor-1254 tend to be more toxic than lower chlorinated forms, so this assumption is generally conservative.

- Variable food ingestion—While food ingestion by wildlife was assumed to be similar to that reported for the same or related species in other locations, the validity of this assumption cannot be determined. Food ingestion by wildlife may be greater or less than that reported in the literature, resulting in either an increase or decrease in contaminant exposure.
- Water ingestion—Because PGDP outfalls border SWMU 99b and AOC 204, outfall water may be a source of drinking water for area wildlife; however, exposures related to drinking water were expected to be minor relative to other exposure pathways, so they were excluded from the exposure models in this BERA. This exclusion could result in an underestimate of risk to wildlife if the drinking water pathway is more significant than expected. Water from these outfalls is being evaluated in the PGDP surface water integrator unit.
- Subsurface soil exposures—Wildlife exposures were only evaluated for surface soil (0-1 ft bgs). While this accounts for the majority of likely exposures for most wildlife receptors, burrowing animals could be exposed to soils below 1 ft. If concentrations of some contaminants are greater below 1 ft bgs, doses to burrowing animals from exposure to these soils may be greater than those determined using surface soil alone.
- **Multiple contaminant exposure**—While wildlife is exposed to multiple contaminants concurrently, published toxicological values only consider effects experienced by exposures to single contaminants. Because some contaminants to which wildlife is exposed can interact antagonistically, single contaminant studies may overestimate their toxic potential. Similarly, for those contaminants that interact additively or synergistically, single contaminant studies may underestimate their toxic potential.
- Metal speciation—Toxicity of metal species varies dramatically depending upon the valence state or form (organic or inorganic) of the metal. For example, arsenic (III), chromium (VI), and methyl mercury are more toxic than arsenic (V), chromium (III), and inorganic mercury, respectively. The available data on the contaminant concentrations in media do not report which species or form of contaminant was observed. Because benchmarks used for comparison represented the more toxic species/forms of the metals (particularly for arsenic, chromium, and mercury), if the less toxic species/form of the metal was actually present, potential toxicity at the sites may be overestimated.
- Uptake factors—Soil-to-biota or food-to-biota uptake factors specific to WAG 28 were unavailable. Therefore it was assumed that the uptake factors derived from published studies were applicable. Because of potential differing geologies and histories between the study areas and WAG 28, the factors from published studies may over- or underestimate the actual biota concentrations. Uncertainties associated with literature-derived uptake factors may also result in over- or underestimates of actual biota concentrations.

- LOAELs versus NOAELs—Potential risks to wildlife were assessed using LOAELs with the intent of focusing the BERA on contaminants that exceed levels associated with adverse effects. LOAELs were selected as the benchmark most likely to indicate actual effects. The use of NOAELs is more conservative, but exceedance of a NOAEL does not indicate that risks are likely, only that contaminant concentrations exceed levels that did not result in effects in previous studies. The use of NOAELs is more appropriate in screening assessments. There is some uncertainty in evaluating contaminants that exceed a NOAEL but are below the corresponding LOAEL. Thus, the assessment may underestimate potential risks if actual adverse effects levels are between the NOAEL and LOAEL values.
- **Benchmark availability**—Toxicity data for derivation of toxicological benchmarks were not available for all chemical-receptor combinations. While it is possible that chemicals without benchmarks could be a concern, there is no way to evaluate possible effects without site-specific toxicity testing. However, benchmarks are available for many priority contaminants.

2.6 SUMMARY AND CONCLUSIONS

Lack of quality habitat in the industrial setting of WAG 28 sites within the fence boundaries limits exposure of ecological receptors at most sites under current conditions (with the exception of SWMU 193a). However, potential future risks are assessed, assuming conditions change so that suitable habitat becomes available for ecological receptors.

Chemical and radionuclide contaminants are evaluated for surface soils from SWMUs 99a, 193a, 193b, and 193c. Detectable concentrations that exceed background are evaluated for the potential of inducing adverse ecological effects to a representative set of receptor species that potentially could inhabit the WAG 28 area. Tables 2.13 and 2.15 summarize COPECs that are identified, based on the results of screening contaminant concentrations against ecological benchmarks.

Six nonradionuclide COPECs, all inorganics, exceed background and benchmarks for at least one receptor group (Table 2.13). The inorganics are boron, barium, chromium, lead, vanadium, and zinc. Similarly, chromium and lead are near background levels (maximum of 1.05 and 1.53× background, respectively). Confidence in the benchmarks for boron and chromium is low. Potential risks from chromium are largely based on chromium being present as the more toxic Cr(VI) rather than the more likely Cr(III); however, chromium exceeds benchmarks for plants and soil invertebrates at all four sites, with the highest concentrations occuring at SWMUs 99a and 193b. Barium is only a potential concern for plants at SWMU 99a, and the concern is driven by a maximum detected concentration more than an order of magnitude higher than other detects in that SWMU. Lead is only a concern for plants in SWMU 193c, but the lead concentration is near background levels. Zinc is a potential concern for plants at SWMU 193c and plants and soil invertebrates at SWMU 99a but, as with lead, concentrations are near background levels. Vanadium is a potential concern for plants and wildlife at SWMU 193b. The potential for adverse effects to ecological receptors exposed to chemicals in surface soil from WAG 28 sites is low.

Estimated doses from exposure to radionuclides in soil are below recommended dose rate limits for all receptors in all sites except for plants and soil invertebrates at SWMU 99a, in which technetium-99 is the radionuclide of concern.

The purpose of this assessment was to evaluate the likelihood that adverse ecological effects may occur or are occurring as a result of exposures at WAG 28. Under current conditions, complete exposure pathways are not expected for terrestrial biota, except at SWMU 193a, and even this area is within the industrialized portion of the plant. Thus, this evaluation focuses on hypothetical future exposures, assuming loss of industrial controls and buildings and development of a larger area of suitable habitat. Analytes that are retained as COPECs may require further study to determine if adverse ecological effects are likely if decisions for remedial actions are based on ecological concerns. Uncertainty concerning the future condition, the bioavailability or form of various metals (e.g., boron, barium, chromium, lead, vanadium, and zinc), and use of only one line of evidence (comparison of exposures to single-chemical toxicity values) may lead to an overestimate of potential future ecological risks.

A summary of analytes of potential concern and receptors potentially at risk should future exposures occur is presented below by site and in Table 2.13.

• SWMU 99a—While chromium and zinc exceed benchmarks for plants and soil invertebrates and barium exceeds benchmarks for plants, potential risks to plant and soil invertebrate communities from future exposure to surface soil in this SWMU appear low. The barium risk is due to a location (Station 099014) where the concentration is more than an order of magnitude higher than at other stations. Zinc is near background levels and results in low exceedances of benchmarks. There is considerable uncertainty in the benchmark for chromium, which is based on the more toxic Cr(VI) rather than the more likely Cr(III).

Estimated doses from exposure to radionuclides in soil are below recommended dose rate limits for all receptors in all sites except for plants and soil invertebrates at SWMU 99a, in which technetium-99 is the radionuclide of concern based on its occurrence in a single sample.

- SWMU 193a—Risks to terrestrial receptors are not expected from current or future exposures at this SMWU. No radionuclides were detected, and only chromium, for which the toxicological benchmark is likely highly conservative, exceeds levels of potential concern for plants and soil invertebrates.
- SWMU 193b—Potential future risks from exposure of plants, soil invertebrates, and wildlife to chromium or vanadium are identified. While there is considerable uncertainty associated with the benchmark available for chromium, concentrations of both chromium and vanadium are elevated relative to other areas in WAG 28, indicating a greater potential to cause adverse effects.
- SWMU 193c—Potential future risks from exposure of plants to boron, chromium, lead, and zinc and exposure of soil invertebrates to chromium are identified, but there is considerable uncertainty associated with the benchmarks available for boron and chromium. Lead and zinc are near background levels, and chromium concentrations are lower in this SWMU than in other areas in WAG 28. Lower chromium concentrations relative to other areas in WAG 28 do not necessarily equate with no risk, but potential risks from chromium are lower.

3. REFERENCES

ACGIH (American Conference of Governmental Industrial Hygienists) 1991. Documentation of the Threshold Limit Values and Biological Exposure Indices, 6th ed. Cincinnati, OH.

ACGIH 1986a. "Documentation of TLVs. Arsine," p. 39, Cincinnati, OH.

ACGIH 1986b. "Copper," p. 146, in Documentation of the Threshold Limit Values and Biological Exposure Indices, 5th ed., Cincinnati, OH.

Adams, E. M., Spencer, H. C., Rowe, V. K., McCollister, D. D., and Irish, D. D. 1952. "Vapor Toxicity of Carbon Tetrachloride Determined by Experiments on Laboratory Animals," Arch. Ind. Hyg. Occup. Med. 6:50-66.

Adkins, B., Van Stee, E. W., Simmons, J. E., and Eustis, S. 1986. "Oncogenic Response Of Strain A/J Mice To Inhaled Chemicals," J. Toxicol. Environ. Health 17:311-322.

Ahrenholz, S. H. 1980. Health Hazard Evaluation Determination Report No. HE 79-113-728, Olin Chemical Company, Brandenburg, Kentucky, for National Institute for Occupational Safety and Health, Cincinnati, OH.

Akay, M. T., and Alp, U. 1981. "The Effects of BHC and Heptachlor on Mice," *Hacette Bull. Nat. Sci. Eng.* 10:11-22.

Akopajan, O. A. 1964. "Some Biochemical Shifts in the Bodies of Workers in Contact With Molybdenum-Containing Dust," in Information on the 2nd Scientific Conference of the Institute of Labor Hygiene and Occupational Diseases on Problems of Labor Hygiene and Occupational Pathology. Erevan 65-67.

Aksoy, M. 1989. "Hematotoxicity and Carcinogenicity of Benzene," Environ. Health Perspect. 82:193-197.

Aksoy, M., and Erdem, S. 1978. "Followup Study on the Mortality and the Development of Leukemia in 44 Pancytopenic Patients with Chronic Benzene Exposure," *Blood* 52:285-292.

Aksoy, M., Erdem, S., DinCol, G., et al. 1972. "Details of Blood Changes in 32 Patients with Pancytopenia Associated with Long-term Exposure to Benzene," Br. J. Ind. Med. 29:56-64.

Aksoy, M., Erdem, S., Akgun, T., et al. 1971. "Haematological Effects of Chronic Benzene Poisoning in 217 Workers," Br. J. Ind. Med. 28:296-302.

Albro, P. W., Corbett, J. T., Schroeder, J. L., Jordan, S., and Matthews, H. B. 1982. "Pharmacokinetics, Interactions with Macromolecules and Species Differences in Metabolism of DEHP," *Environ. Health Perspect.* 45:19–25.

Allen, J. G., Master, H. G., Peet, R. L., et al. 1983. "Zinc Toxicity in Ruminants," J. Comp. Pathol. 93:363-377.

Alumot, E. O., Nachtomi, E., Mandel, E., et al. 1976. "Tolerance and Acceptable Daily Intake of Chlorinated Fumigants in the Rat Diet," Food Cosmet. Toxicol. 14:105-110.

Ambrose, A. M., Larson, P. S., Borzelleca, J. R., and Hennigar, G. R., Jr. 1976. "Long-term Toxicologic Assessment of Nickel in Rats and Dogs," J. Food. Sci. Technol. 13:181-187.



- Amin, S., LaVoie, E. J., and Hecht, S. S. 1982. "Identification of Metabolites of Benzo(b)fluoranthene." Carcinogenesis 3:171-174.
- Amin, S., Huie, K., and Hecht, S. S. 1985a. "Mutagenicity and Tumor Initiating Activity of Methylated Benzo(b)fluoranthene," Carcinogenesis 6:1023-1025.
- Amin, S., Hussain, N., Balanikas, G., et al. 1985b. "Mutagenicity and Tumor Initiating Activity of Methylated Benzo(k)fluoranthenes," *Cancer Lett.* 26:343-347.
- Amin-Zaki, L., Elhassani, S., Majeed, M. A., Clarkson, T. W., Doherty, R. A., and Greenwood, M. 1974. "Intra-uterine Methylmercury Poisoning," *Pediatrics* 54:587-595.
- Amur, M., McCarthy, J., and Gill, M. 1982. "Respiratory Response of Guinea Pigs to Zinc Oxide Fumes," Am. Ind. Hyg. Assoc. J. 43:887-889.
- Angerer, J., and Heinrich, R. 1988. "Cobalt," pp. 251-264, in Handbook on Toxicity of Inorganic Compounds, Seiler, H. G., Sigel, H., and Sigle, A., eds., Marcel Dekker, Inc., New York.
- Anziulewicz, J. A., Dick, H. J., and Chiarulli, E. E. 1959. "Transplacental Naphthalene Poisoning," Am. J. Obstet. Gynecol. 78:519-521.
- Aoyama, K. 1986. "Effects of Benzene Inhalation on Lymphocyte Subpopulations and Immune Response in Mice," *Toxicol. Appl. Pharmacol.* 85:92–101.
- Arena, J. M. (ed.) 1986. Poisoning: Toxicology, Symptoms, Treatments, Charles C. Thomas, Springfield, IL.
- Armstrong, C. W., Stroube, R. B., Rubio, T., Siudyla, E. A., and Miller, G. B. 1984. "Outbreak of Fatal Arsenic Poisoning Caused by Contaminated Drinking Water," *Arch. Environ. Health.* 39:276–279.
- Arnold, D. L., Bryce, R., Stapley, R., et al. 1993a. "Toxicological Consequences of Aroclor 1254 Ingestion by Female Rhesus (Macaca mulatta) Monkeys, Part 1A: Prebreeding Phase-Clinical Health Findings. Food. Chem. Toxicol. 31:799-810.
- Arnold, D. L., Bryce, R., Karpinski, K., et al. 1993b. "Toxicological Consequences of Aroclor 1254 Ingestion by Female Rhesus (Macaca mulatta) Monkeys, Part 1B: Prebreeding Phase-Clinical and Analytical Laboratory Findings. Food. Chem. Toxicol. 31:811-824.
- Arnold, D. W., Kennedy, G. L., Jr., Keplinger, M. L., et al. 1977. "Dominant Lethal Studies with Technical Chlordane, HCS-3260, and Heptachlor:Heptachlor Epoxide," J. Toxicol. Environ. Health 2:547-555.
- Asimangulyan, T. A. 1965. "Determination of the Maximum Permissible Concentration of Molybdenum in Open Bodies of Water," Gig. Sanit. 30:4-6.
- ATSDR (Agency for Toxic Substances and Disease Registry) 1995. Toxicological Profile for Polychlorinated Biphenyls. Draft for Public Comment (Update), U.S. Public Health Service, Atlanta, GA.
- ATSDR 1994. Toxicological Profile for Acetone, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1993a. Toxicological Profile for Lead, Update, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1993b. Toxicological Profile for 1,1-Dichloroethylene, Update, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1993c. Toxicological Profile for 1,4-Dichlorobenzene, ATSDR/TP-92/10, U.S. Public Health Service, Atlanta, GA.

- ATSDR 1993d. Toxicological Profile for Polycyclic Aromatic Hydrocarbons (PAHs), Draft for Public-Comment, Update, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1993e. Toxicological Profile for Heptachlor/Heptachlor Epoxide, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1993f. Toxicological Profile for Xylenes, Draft for Public Comment, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1992a. Toxicological Profile for 1,2-Dichloroethane, Update (Draft), U.S. Public Health Service, Atlanta, GA.
- ATSDR 1992b. Toxicological Profile for Benzene, Draft for Public Comment, Update, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1992c. Toxicologic Profile for Pentachlorophenol, Update (Draft), U.S. Public Health Service, Atlanta, GA.
- ATSDR 1991. Toxicological Profile for Fluoride, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1990a. Toxicological Profile for Aluminum, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1990b. Toxicological Profile for Antimony, Draft, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1990c. Toxicological Profile for Cobalt, Draft, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1990d. Toxicological Profile for Copper, ATSDR/TP-90-08, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1990e. Toxicological Profile for Silver, ATSDR/TP-90-24, U.S. Public Health Service, Atlanta, GA.

ATSDR 1990f. Toxicological Profile for Vanadium, DRAFT, U.S. Public Health Service, Atlanta, GA.

- ATSDR 1990g. Toxicological Profile for 1,1-Dichloroethane, ATSDR/TP-90-12, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1990h. Draft Toxicological Profile for 1,2-Dichloroethene, U.S. Public Health Service, Atlanta, GA.

ATSDR 1990i. Toxicological Profile for 2-Hexanone, U.S. Public Health Service, Atlanta, GA.

- ATSDR 1990j. Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Acenaphthene, Acenaphthylene, Anthracene, Benzo(a)anthracene, Benzo(a)pyrene, Benzo(b)fluoranthene, Benzo(g,i,h)perylene, Benzo(k)fluoranthene, Chrysene, Dibenzo(a,h)anthracene, Fluoranthene, Fluorene, Indeno(1,2,3-c,d)pyrene, Phenanthrene, Pyrene, ATSDR/TP-90-20, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1990k. Toxicological Profile for Benzo(a)pyrene, ATSDR/TP-88-05, U.S. Public Health Service, Atlanta, GA.
- ATSDR 19901. Toxicological Profile for Chrysene, ATSDR/TP-88/11, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1990m. Toxicological Profile for Naphthalene and 2-Methylnaphthalene, ATSDR/TP-90-18, U.S. Public Health Service, Atlanta, GA.

- ATSDR 1990n. Toxicological Profile for Tetrachloroethylene, U.S. Public Health Service, Atlanta, GA.
- ATSDR 19900. Toxicological Profile for Thorium, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1989a. Toxicological Profile for Arsenic, ATSDR/TP-88/02, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1989b. Toxicological Profile for Cadmium, ATSDR/TP-88/08, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1989c. Toxicological Profile for Chromium, ATSDR/TP-88/10, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1989d. Toxicological Profile for Mercury, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1989e. Toxicological Profile for Zinc, ATSDR/TP-89-25, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1989f. Toxicological Profile for 1,1,2-Trichloroethane, ATSDR/TP-89/24, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1989g. Toxicological Profile for Carbon Tetrachloride, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1989h. Toxicological Profile for Chloroform, ATSDR/TP-88/09, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1989i. Toxicological Profile for Methylene Chloride, ATSDR/TP-88-18, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1989j. Toxicological Profile for Trichloroethylene, ATSDR/TP-88/24, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1989k. Toxicological Profile for Vinyl Chloride, ATSDR/TP-88-25, U.S. Public Health Service, Atlanta, GA.
- ATSDR 19891. Draft Toxicological Profile for Uranium and Compounds, U.S. Public Health Service, Atlanta, GA.ATSDR 1988a. Toxicological Profile for Beryllium, ATSDR/TP-88/09, U.S. Public Health Service, Atlanta, GA.
- ATSDR 1988b. Toxicological Profile for Nickel, ATSDR/TP-88/19, U.S. Public Health Service, Atlanta, GA.
- Aughey, E., Grant, L., Furman, B. L., and Dryden, W. F. 1977. "The Effects of Oral Zinc Supplementation in the Mouse," J. Comp. Pathol. 87:1-14.
- Avakajan, M. A. 1966. "The Functional Condition of the Liver in Workers in the Copper and Molybdenum Industry" in Information on the 2nd Scientific Conference of the Institute of Labor Hygiene and Occupational Pathology, Aiastan: Erevan. (Ref. Zh. Otd. Vyp. Farm. Khim. Sredstva Toksikol. No. 154789).
- Avakajan, M. A. 1968. [Title not given]. Ref. Zh. Otd. Vyp. Farm. Khim. Sredstva Toksikol. No. 254748.
- Axelson, O., Dahlgren, E., Jasson, C. D., and Rehnlund, S. O. 1978. "Arsenic Exposure and Mortality: A Case-Referent Study from a Swedish Copper Smelter," Br. J. Ind. Med. 35:8-15.
- Azouz, W. M., Parke, D. V., and Williams, R. T. 1955. "Studies in Detoxication. The Metabolism of Halogenobenzenes and Ortho- and Para-dichlorobenzenes," *Biochem. J.* 59:410-415.

Baader, E. W., and Bauer, H. J. 1951. "Industrial Intoxication Due to Pentachlorophenol," Ind. Med. Surg. 20:286-290.

- Baarson, K., Snyder, C. A., and Albert, R. E. 1984. "Repeated Exposures of C57B1 Mice to 10 ppm Inhaled Benzene Markedly Depressed Erythropoietic Colony Formation," *Tox. Lett.* 20:337-342.
- Babayan, E. A., Nazaretyan, R. A., Martirosyan, A. S., and Darbinyan, G. V. 1984. "State of the Cardiovascular System in Rats Exposed to Acute and Chronic Action of Metallic Molybdenum Aerosol," pp. 15–19 (Abst), Aktual. Vopr. Gig. Tr. Profpatol. Prom-sti. Sel'sk. Khoz. M. Ya. Eglite, ed.
- Badawy, N. B. A., El Salam, M. A., Eissa, S. M., et al. 1987. "Studies on Some Enzymatic Activities Among Zinc Workers," pp. 77-79, in *Heavy Metals in the Environment, 6th International* Conference, New Orleans, September 1987, Vol. 2, Lindberg, S. E., and Hutchinson, T. C., eds., CEP Consultants, Edinburgh.
- Badiali, D., Marcheggiano, A., Pallone, F., et al. 1985. "Melanosis of the Rectum in Patients with Chronic Constipation," *Dis. Colon Rectum* 28:241-245.
- Baetjer, A. M., Lowney, J. F., Stefee, H., and Budacz, V. 1959. "Effect of Chromium on Incidence of Lung Tumors in Mice and Rats," Arch. Ind. Health 20:124-135.
- Baes, C. F., III, Sharp, R. D., Sjoreen, A. L., and Shor, R. W. 1984. A Review and Analysis of Parameters for Assessing Transport of Environmentally Released Radionuclides Through Agriculture, ORNL-5786, Health and Safety Research Division, Oak Ridge National Laboratory, Oak Ridge, TN.
- Baghurst, P. A., Tong, S. L., McMichael, A. J., et al. 1992. "Environmental Exposure to Lead and Children's Intelligence at the Age of Seven Years. The Port Pirie Cohort Study," New Eng. J. Med. 327:1279-1284.
- Baghurst, P. A., Robertson, E. F., McMichael, A. J., et al. 1987. "The Port Pirie Cohort Study: Lead Effects on Pregnancy Outcome and Early Childhood Development," *Neurotoxicology* 8:395-401.
- Baker, D. A., and Soldat, J. K. 1992. Methods for Estimating Doses to Organisms from Radioactive Materials Released into the Aquatic Environment, PNL-8150, Pacific Northwest Laboratory, Richland, WA.
- Bakir, F., Kamluji, S. F., Amin-Zaki, L., et al. 1973. "Methylmercury Poisoning in Iraq," Science 181:230-241.
- Bardodej, Z., and Bardodejova, E. 1970. "Biotransformation of Ethylbenzene, Styrene, and Alphamethylstyrene in Man," Am. Indust. Hyg. Assoc. J. 31:206-209.

Barnes, D. W., Sanders, V. W., White, K. L., Shopp, G. M., and Munson, A. E. 1985. "Toxicology of trans-1,2-Dichloroethylene in the Mouse," Drug and Chem. Toxicol. 8(5):373-392.

Barnes, R., and Jones, R. C. 1967. "Carbon Tetrachloride Poisoning," Am. Ind. Hyg. Assoc. J. 28:557-560.

Barnthouse, L. W. 1995. Effects of Ionizing Radiation on Terrestrial Plants and Animals: A Workshop Report, ORNL/TM-13141, Oak Ridge National Laboratory, Oak Ridge, TN.

Baroni, C., van Esch, G. J., and Saffiotti, U. 1963. "Carcinogenesis Tests of Two Inorganic Arsenicals," Arch. Environ. Health 7:668-674.

- Barrett, H. M., Irwin, D. A., and Semmons, E. 1947. "Studies on the Toxicity of Inhaled Cadmium. I. The Acute Toxicity of Cadmium Oxide by Inhalation," J. Ing. Hyg. Toxicol. 29:279.
- Bartosek, I., Guaitani, A., Modica, R., Fiumem, M., and Urso, R. 1984. "Comparative Kinetics of Oral Benz(a)anthracene, Chrysene and Triphenylene in Rats: Study with Hydrocarbon Mixtures," *Toxicol. Lett.* 23:333-339.
- Baslo, A., and Aksoy, M. 1982. "Neurological Abnormalities in Chronic Benzene Poisoning: A Study of Six Patients with Aplastic Anemia and Two with Preleukemia," *Environ. Res.* 27:457-465.
- Battersby, S., Chandler, J. A., and Morton, M. S. 1982. [Title not given.] Fertil. Steril. 37:230-235.
- Baxley, M. N., Hood, R. D., Vedel, G. C., Harrison, W. P., and Szczech, G. M. 1981. "Prenatal Toxicity of Orally Administered Sodium Arsenite in Mice," *Bull. Environ. Contam. Toxicol.* 26:749-756.
- Bayliss, D. L., and Lainhart, W. S. 1972. "Mortality Patterns in Beryllium Production Workers," presented at the Am. Ind. Hygiene Assoc. Conf., OSHA Exhibit No. 66, Docket No. H005.
- Bayliss, D. L., and Wagoner, J. K. 1977. "Bronchogenic Cancer and Cardio-Respiratory Disease Mortality Among White Males Employed in a Beryllium Production Facility," OSHA Beryllium Hearing, 1977, Exhibit 13.F.
- Bayliss, D. L., Lainhart, W. S., Crally, L. J., Ligo, R., Ayer, H., and Hunter, F. 1971. "Mortality Patterns in a Group of Former Beryllium Workers," pp. 94–107, in *Tran. 334rd Ann. Meeting ACGIH*, Toronto, Canada.
- Beaudoin, A. R. 1974. "Teratogenicity of Sodium Arsenate in Rats," Teratology 10:153-158.
- Beliles, R. P. 1979. "The Lesser Metals," pp. 547-615, in *Toxicity of Heavy Metals in the Environment*, Oehme, F. W., ed., Marcel Dekker Co., New York.
- Beliles, R. P. 1994. "Lithium," in *Patty's Industrial Hygiene and Toxicology*, 4th ed., Vol. II, Part C, Clayton, G. D., and Clayton, F. E., eds., John Wiley and Sons, Inc., New York.
- Beliles, R. P., Bursisk, D. J., and Meclec, F. J. 1980. Teratogenic, Mutagenic Risk Of Workplace Contaminants: Trichloroethylene, Perchloroethylene And Carbon Disulfide. Unpublished studies U.S. Dept. of Health, Education and Welfare, Contract 210-77-0047, Litton Bionetics Inc., Kensington, MD.
- Bell, Z. G., Olson, K. H., and Benja, T. J. 1978. Final Report of Audit Findings of the Manufacturing Chemists Association: Administered Trichloroethylene Chronic Inhalation Study at Industrial Biotest Laboratories, Inc., Decatur, IL. (Unpublished)
- Bellinger, D. C., Needleman, H. L., Leviton, A., et al. 1984. "Early Sensory-Motor Development and Prenatal Exposure to Lead," Neurobehav. Toxicol. Teratol. 6:387-402.
- Bellinger, D. C., Stiles, K. M., and Needleman, H. L. 1992. "Low-level Lead Exposure, Intelligence and Academic Achievement: A Long-term Follow-up Study," *Pediatrics* 90:855-561. (abstract: BIOSIS/93/07320)
- Bellinger, D., Leviton, A., and Sloman, J. 1990. "Antecedents and Correlates of Improved Cognitive Performance in Children Exposed In Utero to Low Levels of Lead," Environ. Health Perspect. 89:5-11.
- Bellinger, D., Leviton, A., and Waternaux, C. 1987a. "Longitudinal Analyses of Prenatal and Postnatal Lead Exposure and Early Cognitive Development," 316:1038-1043.

- Bellinger, D., Sloman, J., Leviton, A., et al. 1987b. "Low-level Lead Exposure and Child Development:... Assessment at Age 5 of a Cohort Followed From Birth," pp. 49-53, in Int. Conf.; Heavy Metals in the Environment, Vol. 1, September 1987, New Orleans, LA, CEP Consultants, Ltd., Edinburgh, United Kingdom.
- Belyaeva, A. O. 1967. "The Effect of Antimony on Reproduction," Gig. Truda. Prof. Zabol. 11:32.
- Berenblum, I. 1941. "The Cocarcinogenic Action of Croton Resin." Cancer Res. 1:44-48.
- Berenblum, I., and Haran, N. 1955. "The Influence of Croton Oil and of Polyethylene Glycol-400 on Carcinogenesis in the Forestomach of the Mouse," *Cancer Res.* 15:510-516.
- Berg, J. W., and Burbank, F. 1972. "Correlations Between Carcinogenic Trace Metals in Water Supply and Cancer Mortality," Ann. N.Y. Acad. Sci. 199:249-261.
- Berger, M., and Fodor, G. G. 1968. "CNA Disorders Under the Influence of Air Mixtures Containing Dichloromethane," Zentralbl. Bakteriol. 215:417.
- Bertholf, R. L. 1988. "Zinc," pp. 787-800, in Handbook on Toxicity of Inorganic Compounds, Sigel, H., and Seiler, H. G., eds., Marcel Dekker, Inc., New York.
- Beton, D. C., Andrews, G. S., Davies, H. J., Howless, L., and Smith, G. F. 1966. "Acute Cadmium Fume Poisoning: Five Cases with One Death from Renal Necrosis," *Br. J. Ind. Med.* 23:292.
- Bevenue, A., Wilson, J., Casarett, L. J., et al. 1967. "A Survey of Pentachlorophenol Content in Human Urine," Bull. Environ. Contam. Toxicol. 2:319-332.
- Beyer, W. N., and Stafford, C. 1993. "Survey and Evaluation of Contaminants in Earthworms and Soils from Dredged Material at Confined Disposal Facilities in the Great Lakes Region," *Environ. Monit.* Assess. 24:151-165.
- Bi, W., Wang, Y., Huang, M., and Meng, D. 1985. "Effect of Vinyl Chloride on Testis in Rats," Ecotoxicol. Environ. Saf. 10:281-289.
- Biancifiori, C., and Caschera, F. 1962. "The Relation Between Pseudo-Pregnancy and the Chemical Induction of Mammary and Ovarian Tumors in BALB/c Mice," Br. J. Cancer 16:722-730.
- Bio/Dynamics 1989. A One-year Inhalation Toxicity Study of Antimony Trioxide in the Rats (With a One-year Recovery Period)- Month 18 Interim Report, Bio/dynamics, Inc., for the Antimony Oxide Industry Assoc., Washington, DC.
- Birch, N. J. 1988. "Lithium," in Handbook on Toxicity of Inorganic Compounds, Seiler, H. G., and Sigel, H. eds., Marcel Dekker, Inc., New York.
- Birge, W. J., Black, J. A., and Short, T. M. 1989. Biological Monitoring Program for the Paducah Gaseous Diffusion Plant Two-Year Report, Paducah, KY.
- Blakely, B. R., Sisodia, C. S., and Mukkur, T. K. 1980. "The Effect of Methylmercury, Tetraethyl Lead, and Sodium Arsenite on the Humoral Immune Response in Mice," *Toxicol. Appl. Pharmacol.* 52:245-254.
- Blaylock, B. G., Frank, M. L., and O'Neal, B. R. 1993. Methodology for Estimating Radiation Dose Rates to Freshwater Biota Exposed to Radionuclides in the Environment, ES/ER/TM-78, Oak Ridge National Laboratory, Oak Ridge, TN.
- Blanton, R. H., Lyte, M., Myers, M. J., and Bick, P. H. 1986. "Immunomodulation by Polyaromatic Hydrocarbons in Mice and Murine Cells," *Cancer Res.* 46:2735-2739.

- Blom, S., Lagerkvist, B., and Linderholm, H. 1985. "Arsenic Exposure to Smelter Workers: Clinical and Neurophysiological Studies," Scand. J. Work Environ. Health 11:265-270.
- Bock, F. G., and King, D. W. 1959. "A Study of the Sensitivity of the Mouse Forestomach Toward Certain Polycyclic Hydrocarbons," J. Natl. Cancer Inst. 23:833-839.
- Bomski, H., Sobolweska, A., and Strakowski, A. 1967. "Toxic Damage to the Liver by Chloroform in Chemical Industry Workers," Arch. Gewerbepathol. Gewerbehyg. 24:127-134.
- Bonczek, R. 1999. Background concentrations of naturally occurring inorganic chemicals and selected radionuclides in the Regional Gravel Aquifer and McNairy Formation at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky. Draft. Prepared by Center for Information Studies, the University of Tennessee, Knoxville, Tennessee and Scientific Applications International Corporation, Oak Ridge, Tennessee.
- Bonnet, P., Francin, J. M., Gradiski, D., et al. 1980. "Determination of the Median Lethal Concentration of Principal Chlorinated Aliphatic Hydrocarbons in the Rat," Arch. Mal. Prof. Med. Trav. Secur. Soc. 41:317-321.
- Borgono, J. M., and Greiber, R. 1972. "Epidemiological Study of Arsenism in the City of Antofagasta," in *Trace Substances in Environmental Health*, Vol. 5, Hemphill, D. C., ed., University of Missouri, Columbia, MS.
- Bornhausen, M., and Hagen, U. 1984. "Operant Behavior Performance Changes in Rats After Prenatal and Postnatal Exposure to Heavy Metals," *IRCS Med. Sci.* 12:805–806.
- Borzelleca, J. F., Hayes, J. R., Condi, L. W., et al. 1985. "Acute Toxicity of Monochlorophenols, Dichlorophenols and Pentachlorophenols," *Toxicol. Lett* 29:39-42.
- Bowen, H. J. M. 1979. Environmental Chemistry of the Elements, Academic Press: London.
- Boyd, G. E. 1959. "Technetium and Promethium," J. Chem. Ed. 36(1):3-14.
- Boyland, E., and Wolf, G. 1950. "Metabolism of Polycyclic Compounds. 6. Conversion of Phenanthrene into Dihydroxydihydrophenanthrene," *Biochem. J.* 47:64-69.
- Boyland, E., and Sims, P. 1962. "Metabolism of Polycyclic Compounds. The Metabolism of Phenanthrene in Rabbits and Rats: Dihydro-dihydroxy Compounds and Related Glucosiduronic Acids," Biochem. J. 84:571-582.
- Bradley, W. R., and Frederick, W. G. 1941. "The Toxicity of Antimony Animals Studies," Ind. Med. 10:15-22.
- Bragt, P. C., and van Dura, E. A. 1983. "Toxicokinetics of Hexavalent Chromium in the Rat After Intratracheal Administration of Chromates of Different Solubilities," Ann. Occup. Hyg. 27:315-322.
- Braun, W. H., Blau, G. E., and Chenoweth, M. B. 1979. "The Metabolism/Pharmacokinetics of Pentachlorophenol in Man, and a Comparison with the Rat and Monkey," pp. 289–296, in Deichmann, W. E., ed., *Toxicology and Occupational Medicine*, New York, Amsterdam, Oxford, Elsevier/North-Holland.
- Breiger, H., Semisch, C. W., Stasney, J., and Piatnek, D. A. 1954. "Industrial Antimony Poisoning," Indust. Med. Health 23:521-523.
- Brenniman, G. R., and Levy, P. S. 1984. "High Barium Levels in Public Drinking Water and Its Association with Elevated Blood Pressure," pp. 231-249, in Advances in Modern Toxicology IX, Calabrese, E. J., ed., Princeton Scientific Publications, Princeton, NJ.

- Brink, M. F., Becker, D. E., Terrill, S. W., et al. 1959. "Zinc Toxicity in the Weanling Pig," J. Anim. Sci. 18:836-842.
- Brown, M. M., Rhyne, B. C., Goyer, R. A., and Fowler, B. A. 1976. "Intracellular Effects of Chronic Arsenic Administration on Renal Proximal Tubule Cells," J. Toxicol. Environ. Health 1:505-514.
- Bruckner, J. V., MacKenzie, W. F., Muralidhara, S., Luthra, U., Kyle, G. M., and Aconta, D. 1986. "Oral Toxicity of Carbon Tetrachloride: Acute, Subacute and Subchronic Studies in Rats," Fund. Appl. Toxicol. 6(1):16-34.
- Buben, J. A., and O'Flaherty, E. J. 1985. "Delineation of the Role of Metabolism in the Hepatotoxicity of Trichloroethylene and Perchloroethylene: A Dose-effect Study," *Toxicol. Appl. Pharmacol.* 78:105-122.
- Buck, W. B., Radeleff, R. D., Jackson, J. B., et al. 1959. "Oral Toxicity Studies with Heptachlor and Heptachlor Epoxide in Young Calves," J. Econ. Entomol. 52:1127-1129.
- Budavari, S., O'Neil, M. J., Smith, A., and Heckelman, P. E., eds. 1989. The Merck Index. An Encyclopedia of Chemcials, Drugs, and Biologicals, 11th ed., Merck and Co., Inc., Rahway, NJ.
- Buening, M. K., Levin, W., Wood, A. W., et al. 1979a. "Tumorigenicity of the Dihydrodiols of Dibenzo(a,h)anthracene on Mouse Skin and in Newborn Mice," Cancer Res. 39:1310-1314.
- Buening, M. K., Levin, W., Karle, J. M., et al. 1979b. "Tumorigenicity of Bay-Region Epoxides and Other Derivatives of Chrysene and Phenanthrene in Newborn Mice," *Cancer Res.* 39:5063-5068.
- Burek, J. D., Nitschke, K. D., Bell, T. J., et al. 1984. "Methylene Chloride: A Two-Year Inhalation Toxicity and Carcinogenicity Study in Rats and Hamsters," *Fund. Appl. Toxicol.* 4:30-47.

Burk, D., and Beaudoin, A. R. 1977. "Arsenate-Induced Renal Agenesis in Rats," Teratology 16:247-260.

- Burrows, D., ed. 1983. Chromium: Metabolism and Toxicity, pp. 137-163, CRC Press, Inc., Boca Raton, FL.
- Busby, W. F., Jr., Goldman, M. E., Newberne, P. M., and Wogan, G. N. 1984. "Tumorigenicity of Fluoranthene in a Newborn Mouse Lung Adenoma Bioassay," *Carcinogenesis* 5:1311-1316.
- Byren, D., Engholm, G., Englund, A., and Westerholm, P. 1976. "Mortality and Cancer Morbidity in a Group of Swedish VCM and PVC Production Workers," *Environ. Health Perspect.* 17:167–170.
- Byrne, A. R., and Kosta, L. 1978. "Vanadium in Foods and in Human Body Fluids and Tissues," Sci. Total Environ. 10:17-30.
- Byron, W. R., Bierbower, G. W., Brouwer, J. B., and Hansen, W. H. 1967. "Pathologic Changes in Rats and Dogs from Two-Year Feeding of Sodium Arsenite or Sodium Arsenate," *Toxicol. Appl. Pharmacol.* 10:132-147.
- Calabrese, E. J., and Kenyon, E.M. 1991. Air Toxics and Risk Assessment, pp. 531-535, Lewis Publishers, Chelsea, MI.
- Campbell, D. M., and Davidson, R. J. 1970. "Toxic Haemolytic Anemia in Pregnancy Due to a Pica for Paradichlorobenzene," J. Obstet. Gynaec. Br. Commonw. 77:657-659.
- Carpenter, C. P., Kinkead, E. R., Geary, D. L. Jr., Sullivan, L. J., and King, J. M. 1975. "Petroleum Hydrocarbon Toxicity Studies. V. Animal and Human Response to Vapors of Mixed Xylenes," *Toxicol. Appl. Pharmacol.* 33:543-538.



- Carpenter, G. P., Weil, C. S., and Smyth, H. F., Jr. 1953. "Chronic Oral Toxicity of di(2ethylhexyl)phthalate for Rats, Guinea Pigs and Dogs," J. Ind. Hyg. Occup. Med. 18:219-226.
- Carter et al. 1995. Sampling and Analysis Plan for a Site Evaluation at the Outfall 012 and 012 Areas, Paducah Gaseous Diffusion Plant, Paducah, Kentucky, Paducah, KY.
- Casarett, A. P. 1968. Radiation Biology, Prentice-Hall, Englewood Cliffs, NJ.
- Cavender, F. 1994. "Ethylbenzene," pp. 1342–1346, in *Patty's Industrial Hygiene and Toxicology*, 4th rev. ed., Vol. II, Part B, Clayton, G. D., and Clayton, F. E., eds., Wiley Interscience, NY.
- Cavender, F. L., Steinhagen, W. H., and Cockrell, B. Y. 1978. "Chronic Toxicity of Aluminum Chlorohydrate," Clin. Toxicol. 12:606.
- Cawein, M. J., and Sydnor, K. L. 1968. "Suppression of Cellular Activity in the Reticuloendothelial System of the Rat by 7,12-Dimethylbenz(a)anthracene," *Cancer Res.* 28:320.
- Cebrian, M. E., Albores, A., Aguilar, M., and Blakely, E. 1983. "Chronic Arsenic Poisoning in the North of Mexico," *Hum. Toxicol.* 2:121-133.
- CEC (Commission of the European Communities) 1986. Organo-Chlorine Solvents: Health Risks to Workers, Royal Society of Chemistry, Rept No. EUR 10531 EN, pp.73-91.
- CH2M Hill 1991a. Results of the Site Investigation, Phase I, at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky, KY/ER-4, Paducah, KY.
- CH2M Hill 1991b. Results of the Public Health and Ecological Assessment, Phase II, at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky, Vol. 6, in Results of the Site Investigation, Phase II, Paducah Gaseous Diffusion Plant, Paducah, Kentucky, KY/SUB/13B-97777C P-03/1991/1.
- CH2M Hill 1992. Results of the Site Investigation, Phase II, at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky, KY/SUB/13B-97777CP-03/1991/1, Paducah, KY.
- Chandra, R. K. 1984. "Excessive Intake of Zinc Impairs Immune Response," J. Amer. Med. Assoc. 252:1443-1446.
- Chang, L. H. 1943. "The Fecal Excretion of Polycyclic Hydrocarbons Following Their Administration to the Rat," J. Biol. Chem. 151:93-99.
- Chang, Z. H. and Young, Z. 1943. "The Metabolism of Acenaphthene in the Rat." J. Biol. Chem. 151:87.
- Chashschin, V. P., Artunina, P. A., and Norseth, T. 1994. "Congenital Defects, Abortion and Other Health Effects in Nickel Refinery Workers," *Science Total Environ*, 148:287–291.
- Chaturapit, S., and Holder, G. M. 1978. "Studies on the Hepatic Microsomal Metabolism of (¹⁴C)phenanthrene," *Biochem. Pharmacol.* 27:1865–1872.
- Cheever, K. L., Cholakis, J. M., el-Hawari, A. M., et al. 1990. "Ethylene Dichloride: The Influence of Disulfiram or Ethanol on Oncogenicity, Metabolism, and DNA Covalent Binding in Rats," Fundam. Appl. Toxicol. 14:243-261.
- Chen, C. J., Chuang, Y. C., Lin, T. M., and Wu, H. Y. 1985. "Malignant Neoplasms Among Residents of a Blackfoot Disease-Endemic Area in Taiwan: High-Arsenic Artesian Well Water and Cancers," *Cancer Res.* 45:5895-5899.

- Chen, C. J., Chuang, Y. C., You, S. L., Lin, T. M., and Wu, H. Y. 1986. "A Retrospective Study on-Malignant Neoplasms of Bladder, Lung, and Liver in a Blackfoot Disease-Endemic Area in Taiwan," Br. J. Cancer 53:399-405.
- Chernoff, N., and Kavlock, R. J. 1982. "An In Vivo Teratology Screen Utilizing Pregnant Mice," J. Toxicol. Environ. Health 10:541-550.
- Chien, L., Robertson, H., and Gerrard, J. W. 1968. "Infantile Gastroenteritis Due to Water with High Sulfate Content," Can. Med. Assoc. J. 99:102-104.
- Chmielewski, J., Tomaszewski, R., Glombiowski, P., et al. 1976. "Clinical Observations of the Occupational Exposure to Tetrachloroethylene," Bull. Inst. Marit. Trop. Med. Gdynia. 27(2):197-205.
- Chobanian, S. J. 1981. "Accidental Ingestion of Liquid Zinc Chloride: Local and Systemic Effects," Ann. Emerg. Med. 10:91-93.
- Cristy, M., and Eckerman, K. F. 1987. Specific Absorbed Fractions of Energy at Various Ages from Internal Photon Sources: I. Methods, ORNL/TM-8381/V1, Oak Ridge National Laboratory, Oak Ridge, TN.
- CIIT (Chemical Industry Institute of Toxicology) 1982. 104-Week Chronic Toxicity Study in Rats: Dinitrotoluene, Final Report, Vols. 1 and 2, CIIT Docket No. 12362, Research Triangle Park, NC.
- Clarke, M. J., and Podbielski, L. 1988. Handbook on Toxicity of Inorganic Compounds, Seiler et al., eds., Marcel Dekker, New York.
- Clausen, J. L., Douthitt, J. W., Davis, K. R., and Phillips, B. E. 1992. Report of the Paducah Gaseous Diffusion Plant Groundwater Investigation, Phase III, Paducah, Kentucky, KY/E-150, Hydrological Services Section, Environmental Restoration Division, Paducah Gaseous Diffusion Plant, Paducah, KY.
- Clayton, G. D., and Clayton, F. E., eds. 1981. Patty's Industrial Hygiene and Toxicology, Vol. 2A, 2B, 3rd rev. ed., John Wiley and Sons, Inc., New York.
- Climie, I. J. G., Hutson, D. H., and Stoydin, G. 1983. "The Metabolism of Ethylbenzene Hydroperoxide in the Rat," Xenobiotica 13:611-618.
- COE (U.S. Army Corps of Engineers) 1994a. Environmental Investigations at the Paducah Gaseous Diffusion Plant and Surrounding Area, McCracken County, Kentucky, Vol. II: Wetland Investigation, Environmental Restoration Division, U.S. Department of Energy, Paducah, KY.
- COE 1994b. Environmental Investigations at the Paducah Gaseous Diffusion Plant and Surrounding Area, McCracken County, Kentucky, Vol. III, Threatened and Endangered Species, Environmental Restoration Division, U.S. Department of Energy, Paducah, Kentucky, KY.
- Cohen, L. S., Friedman, J. M., Jefferson, J. W., et al. 1994. "A Reevaluation of Risk In Utero Exposure to Lithium," J. Am. Med. Assoc. 271:146-50.
- Coler, H. R., and Rossmiller, H. R. 1953. "Tetrachloroethylene Exposure in a Small Industry," Ind. Hyg. Occup. Med. 8:227.
- Constantinidis, K. 1978. "Acute and Chronic Beryllium Disease," Br. J. Clin. Pract. 32:127-136.
- Cooper, D. A., Pendergrass, E. P., Vorwald, A. J., et al. 1968. "Pneumoconiosis Among Workers in an Antimony Industry," Am. J. Roentgenol. Rad. Ther. Nuclear Med. 103:495-508.



- Cornish H. H., and Ryan, R. C. 1965. "Metabolism of Benzene in Nonfasted, Fasted, and Aryl-hydroxylase Inhibited Rats," *Toxicol. Appl. Pharmacol.* 7:767-771.
- Costa, A. K., and Ivanetich, K. M. 1984. "Chlorinated Ethylenes: Their Metabolism and Effect on DNA Repair in Rat Hepatocytes," *Carcinogenesis* 5:1629-1636.
- Crecelius, E. A. 1977. "Changes in the Chemical Speciation of Arsenic Following Ingestion by Man," Environ. Health Perspect. 19:147-150.
- Cronkite, E. P. 1986. "Benzene Hematotoxicity and Leukemogenesis," Blood Cells 12:129-137.
- Cronkite, E. P., Drew, R. T., Inoue, T., and Bullis, J. E. 1985. "Benzene Hematotoxicity and Leukemogenesis," Am. J. Indust. Med. 7:447-456.
- Crowley, J. F., Hamilton, J. G., and Scott, K. J. 1949. "The Metabolism of Carrier-Free Radioberyllium in the Rat," J. Biol. Chem. 177:975-984.
- Daldrup, T., Haarhoff, K., and Szathmary, S. C. 1983. "Toedliche Nickel Sulfate-Intoxikation," Berichte zur Gerichtlichen Medizin 41:141-144.
- Daniel, P. M., Pratt, O. E., and Prichard, M. M. L. 1967. "Metabolism of Labelled Carcinogenic Hydrocarbons in Rats," *Nature* 215:1142-1146.
- Daniels, J. I., ed. 1988. Evaluation of Military Field-Water Quality. Volume 4. Health Criteria and Recommendations for Standards, Part 1, "Chemicals and Properties of Military Concern Associated with Natural and Anthropogenic Sources," AD UCRL-21008.
- Danielsson, G. R. G., Hassoun, E., and Dencker, L. 1982. "Embryotoxicity of Chromium: Distribution in Pregnant Mice and Effects on Embryonic Cells In Vitro," Arch. Toxicol. 51:233-245.
- Datta, D. V. 1976. "Arsenic and Noncirrhotic Portal Hypertension," Lancet 1:433.
- Davies, D. J. A., and Bennett, B. G. 1983. "Summary Exposure Assessments for Copper, Vanadium, and Antimony," in *Exposure Assessments of Environmental Pollutants*, Vol. 30, Monitoring and Assessment Research Centre, Chelsea College, University of London, London.
- Davis, K. 1965. Pathology Report on Mice Fed Aldrin, Dieldrin, Heptachlor and Heptachlor Epoxide for Two Years, Internal FDA memorandum to Dr. A. J. Lehman, July 19.
- Dayhaw-Barker, P., Sambuco, C. P., Forbes, P. D., et al. 1985. "Development of an Animal Model to Study the Phototoxic Effects of Anthracene and UV in Skin and Ocular Tissues," 13th Annual Meeting of the American Society for Photobiology, New Orleans, LA, June 23–27, 1985, Photochem. Photobiol. 41:122S.
- de Groot, G., and Van Heijst, A. N. P. 1988. "Toxicokinetic Aspects of Thallium Poisoning. Methods of Treatment by Toxin Elimination," Sci. Total Environ. 71:411-418.
- Defalque, J. R. 1961. "Pharmacology and Toxicology of Trichloroethylene. A Critical Review of the World Literature," *Clin. Pharmacol. Ther.* 2:665–688.
- Della Porta, G., Terracini, B., and Shubik, P. 1961. "Induction with Carbon Tetrachloride of Liver Cell Carcinomas in Hamsters," J. Natl. Cancer Inst. 26:855-863.

Derobert, L. and Tara, S. 1950. "Mercury Intoxication in Pregnant Women," Ann. Med. Leg. 30:222-225.

- Deutsch-Wenzel, R. P., Brune, H., Grimmer, G., et al. 1983. "Experimental Studies in Rat Lungs on the Carcinogenicity and Dose-Response Relationships of Eight Frequently Occurring Environmental Polycyclic Aromatic Hydrocarbons," J. Natl. Cancer Inst. 71:539-544.
- Dick, R. B, Setzer, J. V., Taylor, B. J., et al. 1989. "Neurobehavioral Effects of Short Duration Exposure to Acetone and Methyl Ethyl Ketone," *Br. J. Ind. Med.* 46:111-121.
- Dietrich, K. N., Succop, P. A., Berger, O. G., Hammond, P. B., and Bornschein, R. L. 1993. "The Developmental Consequences of Low to Moderate Prenatal and Postnatal Lead Exposure: Intellectual Attainment in the Cincinnati Lead Study Cohort Following School Entry," *Neurotoxicol. Teratol.* 15:37-4.
- Dietrich, K. N., Succop, P. A., Berger, O. G., and Keith, R. W. 1992. "Lead Exposure and the Central Auditory Processing Abilities and Cognitive Development of Urban Children: The Cincinnati Lead Study Cohort at Age 5 Years," *Neurotoxicol. Teratol.* 14:51-56.
- Dietrich, K. N., Succop, P. A., Berger, O. G., Hammond, P. B., and Bornschein, R. L. 1991. "Lead Exposure and the Cognitive Development of Urban Preschool Children: The Cincinnati Lead Study Cohort at Age 4 Years," *Neurotoxicol. Teratol.* 13:203-212.
- Dietrich, K. N., Krafft, K. M., Bier, M., et al. 1986. "Early Effects of Fetal Lead Exposure: Neurobehavioral Findings at 6 Months," Inst. J. Biosoc. Res. 8:151-168.
- Dixon, R. L., Sherins, R. J., and Lee, I. P. 1979. "Assessment of Environmental Factors Affecting Male Fertility," *Environ. Health Perspect.* 30:53-68.
- Dobson, R. L., Young, M. R., and Pinto, J. S. 1965. "Palmar Keratoses and Cancer," Arch. Dermatol. 92:553-556.
- DOE (U.S. Department of Energy) 1999. Remedial Investigation Report for Waste Area Grouping 27 at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky, Volume 4 of 4, Baseline Risk Assessment, DOE/OR/07-1777/V4&D2, Paducah, KY.
- DOE 1998a. Work Plan for Waste Area Grouping 28 Remedial Investigation/Feasibility Study and Waste Area Grouping 8 Preliminary Assessment/Site Investigation at the Portsmouth Gaseous Diffusion Plant, Paducah, KY, DOE/OR/07-1592&02, Paducah, KY.
- DOE 1998b. Integrated Remedial Investigation/Feasibility Study Work Plan for the Waste Area Grouping 27 at Paducah Gaseous Diffusion Plant, Paducah, Kentucky, DOE/OR/07-1518&D3, Paducah, KY.
- DOE 1998c. Risk Assessment Information System. Available on the World-Wide Web at <u>http://risk.lsd.ornl.gov/tox/rap hp.shtml.</u> Developed and maintained for the U.S. Department of Energy by the Toxicology and Risk Analysis Section, Oak Ridge National Laboratory, Oak Ridge, TN.
- DOE 1997. Background Levels of Selected Radionuclides and Metals in Soils and Geologic Media at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky, DOE/OR/07-1586&D2, Paducah, KY.
- DOE 1996a. Methods for Conducting Human Health Risk Assessment and Risk Evaluations at the Paducah Gaseous Diffusion Plant, DOE/OR/07-1506&D1, August.
- DOE 1996b. Site Management Plan, Paducah Gaseous Diffusion Plant, Paducah, Kentucky, DOE/OR/07-1207&D2, Paducah, KY

- DOE 1995a. Northeast Plume Preliminary Characterization Summary Report, Paducah Gaseous Diffusion Plant, Paducah, Kentucky, Volumes 1 and 2, DOE/OR/07-1339/V1 & D2, Paducah, KY.
- DOE 1995b. Final Site Evaluation Report for the Outfall 010, 011, and 012 Areas, Paducah Gaseous Diffusion Plant, Paducah, Kentucky, Paducah, KY.
- DOE 1995c. Remedial Inbestigation/Feasibility Study for the Clinch River/Poplar Creek Operable Unit, DOE/OR/01-1393, Oak Ridge, TN.
- DOE 1994. Baseline Risk Assessment and Technical Investigation Report for the Northwest Dissolved Phase Plume, Paducah Gaseous Diffusion Plant, Paducah, Kentucky, DOE/OR/07-1286&D1, Paducah, KY.
- DOE 1993. Human Health Baseline Risk Assessment for the Northwest Plume, Paducah Gaseous Diffusion Plant, Paducah, Kentucky, DOE/OR/07-1202&D1.
- Dolgner, R., Brockhaus, A., Ewers, U., Wiegand, H., Majewski, F., and Soddemann, H. 1983. "Repeated Surveillance of Exposure to Thallium in a Population Living in the Vicinity of a Cement Plant Emitting Dust Containing Thallium," Int. Arch. Occup. Environ. Health 52:79-94.
- Domingo, J. L., Llobet, J. M., and Corbella, J. 1985. "Protection of Mice Against the Lethal Effects of Sodium Metavanadate: A Quantitative Comparison of a Number of Chelating Agents," *Toxicol. Lett.* 26: 95-99.
- Dorfmueller, M. A., Henne, S. P., York, R. G., et al. 1979. "Evaluation of Teratogenicity and Behavioral Toxicity with Inhalation Exposure of Maternal Rats to Trichloroethylene," *Toxicology* 14:153-166.

Dow Chemical Company 1958. MRID No. 00061912, available from EPA, FOI, Washington, DC.

- Dowty, B. J., Laseter, J. L., and Storer, J. 1976. "Transplacental Migration and Accumulation in Blood of Volatile Organic Constituents," *Pediatr. Res.* 10:696-701.
- Drew, R., Bhola, N., Bend, J., and Hook, G. 1974. "Inhalation Studies with a Glycol Complex of Aluminum Chloride Hydroxide," Arc. Environ. Health. 28:321.
- Drew, R. T., and Fouts, J. R. 1974. "The Lack of Effects of Pretreatment with Phenobarbital and Chlorpromazine on the Acute Toxicity of Benzene in Rats," *Toxicol. Appl. Pharmacol.* 27:183-193.
- Drinker, K., and Drinker, P. 1928. "Metal Fume Fever: V. Results of the Inhalation by Animals of Zinc and Magnesium Oxide Fumes," J. Ind. Hyg. 10:56-70.
- Drinker, K. R., Thompson, P. K., and Marsh, M. 1927. "An Investigation of the Effect of Long Continued Ingestion of Zinc, in the Form of Zinc Oxide, by Cats and Dogs, Together With Observations Upon the Excretion and Storage of Zinc," Am. J. Physiol. 80:31-64.
- Druckrey, H., and Schmahl, D. 1955. "Cancerogenic Effect of Anthracene," Die Naturwissenschaften 42:159-160.
- Dunlap, K. L. 1978. "Nitrobenzene and Nitrotoluenes," pp. 930-931, in Kirk-Othmer Encyclopedia of Chemcial Technology, 3nd ed., Vol. 15, Grayson, M., and Eckroth, D., eds., John Wiley & Sons, New York.
- Earl, F. L., and Vish, T. J. 1979. "Teratogenicity of Heavy Metals," pp. 617-668, in Toxicity of Heavy Metals in the Environment, Oehme, F. W., ed. Marcel Dekker, Inc., New York.

- Easton, D. F., Peto, J., Morgan, L. G., et al. 1994. "Respiratory Cancer Mortality in Welsh Nickel Refiners:... Which Nickel Compounds Are Responsible," pp. 603-619, in *Nickel and Human Health*, Nieboer, E., and Nriagu, J. O., eds., John Wiley and Sons, Inc., New York, NY.
- Eckerman, K. F., and Ryman, J. C. 1993. External Exposure to Radionuclides in Air. Water, and Soil, EPA 402-R-93-081, Federal Guidance Report No. 12, Office of Radiation and Indoor Air, U.S. Environmental Protection Agency, Washington, DC.

Ecolajan, S. L. 1965. "The Effect of Molybdenum on the Nervous System," Z. Exp. Klin. Med. 5:70-73.

- Edwards, J., Heston, W. E., and Dalton, A. J. 1942. "Induction of the Carbon Tetrachloride Hepatoma in Strain L Mice," J. Natl. Cancer Inst. 3:297-301.
- Efroymson, R., Sample, B. E., Suter, G. W., II, Beauchamp, J. J., Aplin, M. S., and Will, M. E. 1998. Empirical Models for the Uptake of Inorganic Chemicals from Soil by Plants, Oak Ridge National Laboratory, Oak Ridge, TN.
- Efroymson, R. A., Will, M. E., Suter, G. W., II, and Wooten, A. C. 1997a. Toxicological Benchmarks for Screening Contaminants of Potential Concern for Effects on Terrestrial Plants: 1997 Revision, ES/ER/TM-85/R3, Oak Ridge National Laboratory, Oak Ridge, TN.
- Efroymson, R. A., Will, M. E., and Suter, G. W., II. 1997b. Toxicological Benchmarks for Contaminants of Potential Concern for Effects on Soil and Litter Invertebrates and Heterotrophic Process: 1997 Revision, ES/ER/TM-126/R2, Oak Ridge National Laboratory, Oak Ridge, TN.
- Ehle, A. L., and McKee, D. C. 1990. "Neuropsychological Effect of Lead in Occupationally Exposed Workers: A Critical Review," Crit. Rev. Toxicol. 20:237-255.
- EHRT (Environmental Health Research and Testing, Inc.) 1987. Screening of Priority Chemicals for Reproductive Hazards: Benzethonium Chloride (CAS No. 121-54-0); 3-Ethoxy-1-propanol (CAS No. 111-35-3); Acetone (CAS No. 67-64-1), NTIS PB89-139083, Cincinnati, OH.
- Elinder, C. G. 1986. "Zinc," in Handbook on the Toxicology of Metals, 2nd ed., Vol. II: Specific Metals, Friberg, L., Nordberg, G. F., and Vouk, V. B., eds. Elsevier, NY.
- Ellenhorn, M. J., and Barceloux, D. G. 1988. Medical Toxicology: Diagnosis and Treatment of Human Poisoning, Elsevier, New York.
- Ellis, H. V., Hong, C. B., Lee, C. C., et al. 1985. "Subchronic and Chronic Toxicity Studies of 2,4-Dinitrotoluene. Part I. Beagle Dogs," J. Am. College Toxicol. 4:233-242.
- Ellis, H. V., Hagensen, J. H., Hogdson, J. R., et al. 1979. Mammalian Toxicity of Munitions Compounds. Phase III: Effects of Lifetime Exposure. Part I: 2,4-Dinitrotoluene, Midwest Research Institute, Kansas City, DAMD17-74-C-4073, U.S. Army Medical Bioengineering Research and Development Laboratory, Report AD AO77 692.
- Ellwood, P. C., Davey-Smith, G., Oldham, P. D., and Toothill, C. 1988a. "Two Welsh Surveys of Blood Lead and Blood Pressure," *Environ. Health Perspect.* 78:119-121.
- Ellwood, P. C., Yarnell, J. W. G., Oldham, P. D., et al. 1988b. "Blood Pressure and Blood Lead in Surveys in Wales," Am. J. Epidemiol. 127:942-945.
- Elsisi, A. E., Carter, D. E., and Sipes, I. G. 1989. "Dermal Absorption of Phthalate Diesters in Rats," Fund. Appl. Toxicol. 12(1):70-77.



- Energy Systems (Lockheed Martin Energy Systems) 1996. Chemical-Specific Applicable or Relevant and Appropriate Requirements (ARARs): Federal/Kentucky, Toxicology and Risk Analysis Section, Oak Ridge National Laboratory, Oak Ridge, TN.
- Energy Systems 1990. Paducah Gaseous Diffusion Plant Environmental Report for 1989, ES/ESH-13/V3, Paducah, KY.
- Enterline, P. E., and Marsh, G. M. 1982. "Mortality Among Workers in a Nickel Refinery and Alloy Plant in West Virginia," J. Natl. Cancer Inst. 68:925-933.
- EPA (U.S. Environmental Protection Agency) 1998a. Integrated Risk Information System (IRIS), available on the World-wide Web at <u>http://www.epa.gov/ngispgm3/iris/index.html</u>. Office of Health and Environmental Assessment, National Center for Environmental Assessment, Cincinnati, OH.
- EPA 1998b. Health Effects Assessment Summary Tables (HEAST), FY 1997 Update, EPA-540-R-97-036, Office of Solid Waste and Emergency Response, Office of Emergency and Remedial Response, Washington, DC.
- EPA 1998c. Guidelines for Ecological Risk Assessment, EPA/630/R-95/002F, U.S. Environmental Protection Agency, Washington, DC.
- EPA 1996a. Soil Screening Guidance: Technical Background Document, EPA/540/R95/128, Office of Solid Waste and Emergency Response, Washington, DC.
- EPA 1996b. "Ethylbenzene," Integrated Risk Information System (IRIS), online file, updated May 1, 1992, retrieved September 14, 1995, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1996c. PCBs: Cancer Dose-Response Assessment and Application to Environmental Mixtures, EPA/600/P-96/001A, Washington, DC.
- EPA 1995a. Supplemental Guidance to RAGS: Region 4 Bulletins, Human Health Risk Assessment, Interim Guidance, Office of Health Assessment, Region 4 EPA, Atlanta, GA.
- EPA 1995b. "Health Risk Assessment for Barium," Integrated Risk Information System (IRIS), (Verification date June 21, 1990) Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.
- EPA 1995c. "Health Risk Assessment for Manganese," on line, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, National Center for Environmental Assessment, Cincinnati, OH.
- EPA 1995d. "Nickel," Integrated Risk Information System (IRIS), Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1995e. "2,4-Dinitrotoluene," Integrated Risk Information System (IRIS), Evironmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1995f. "Dinitrotoluene Mixture, 2,4-/2,6-," Integrated Risk Information System (IRIS), Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1995g. "Acetone," Integrated Risk Information System (IRIS), on line file updated August 2, 1993, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1995h. Risk Assessment Issue Paper for: Deprivation of a Professional Subchronic Oral RfD for Ethylbenzene, Superfund Technical Support Center, National Center for Environmental Assessment, Cincinnati, OH. Correspondence dated September 27, 1995.

- EPA 1995i. "Polychlorinated Biphenyls File," Integrated Risk Information System (IRIS), Environmental. Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1995j. "Aroclor 1254," Integrated Risk Information System (IRIS), Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1995j. Ecological Risk Assessment Bulletin No. 3: Endpoint Selection, U.S. Environmental Protection Agency Region 4, Waste Management Division, Atlanta, GA.
- EPA 1994a. Interim Soil Lead Guidance for CERCLA Sites and RCRA Corrective Action Facilities, OSWER Directive 9355.4-12. Office of Solid Waste and Emergency Response, Washington, DC,
- EPA 1994b. "Thallium Acetate," Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, U.S. Environmental Protection Agency, Cincinnati, OH.
- EPA 1994c. "Thallium Carbonate," Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, U.S. Environmental Protection Agency, Cincinnati, OH.
- EPA 1994d. "Thallium Chloride," Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, U.S. Environmental Protection Agency, Cincinnati, OH.
- EPA 1994e. "Thallium Nitrate," Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, U.S. Environmental Protection Agency, Cincinnati, OH.
- EPA 1994f. "Thallium Sulfate," Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, U.S. Environmental Protection Agency, Cincinnati, OH.
- EPA 1994g. "Thallium Selenite," Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, U.S. Environmental Protection Agency, Cincinnati, OH.
- EPA 1994h. "Toxicity Information for 1,2-Dichloroethane for the Ft. Devens Army NPL Site/MA," Joan S. Dollarhide, Superfund Health Risk Technical Support Center, Environmental Criteria and Assessment Office, Cincinnati, OH, memorandum to Ann-Marie Burke, U.S. EPA, Region 1.
- EPA 1994i. "Chrysene," Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, U.S. Environmental Protection Agency, Cincinnati, OH.
- EPA 1993a. *Wildlife Exposure Factors Handbook*, Vol. I, EPA/600/R-93/187a, Office of Research and Development, Washington, DC.
- EPA 1993b. "1,1-Dichloroethane," Integrated Risk Information System (IRIS), online file updated January 22, 1992, retrieved October 1993, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1993c. "Risk Estimate for Carcinogenicity of 1,2-Dichloroethane," Integrated Risk Information System (IRIS), online file updated July 1993, retrieved October 1993, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1993d. EPA Superfund Standard Default Exposure Factors for the Central Tendency and Reasonable Maximum Exposure Preliminary, Review Draft.
- EPA 1992a. Supplemental Guidance to RAGS: Calculating the Concentration Term, OSWER Directive 9285.7-081, Office of Solid Waste and Emergency Response, Washington, DC.
- EPA 1992b. Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual. Supplemental Guidance: Dermal Risk Assessment, Interim Guidance, Office of Emergency and Remedial Response, Washington, DC.

- EPA 1992c. "Health Risk Assessment for Benzo(a)pyrene," Integrated Risk Information System (IRIS), online, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.
- EPA 1992d. "Benzene," Integrated Risk Information System (IRIS), Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH
- EPA 1992e. "Chloroform," Integrated Risk Information System (IRIS), Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1992f. Superfund Health Risk Technical Support Center Fact Sheet for Tetrachloroethylene, December 14, 1992.
- EPA 1992g. "Trichloroethylene," Integrated Risk Information System (IRIS), Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1992h. Superfund Health Risk Technical Support Center, Fact Sheet for Trichloroethylene (TCE) (CASRN 79-01-6), December 14, 1992.
- EPA 1992i. Framework for Ecological Risk Assessment, EPA/630/R-92/001, Risk Assessment Forum, U.S. Environmental Protection Agency, Washington, DC.
- EPA 1991a. "Risk Estimate for Carcinogenicity and Reference Dose for Oral Exposure for Arsenic," Integrated Risk Information System (IRIS), online file retrieved November 1991, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.
- EPA 1991b. "Beryllium," Integrated Risk Information System (IRIS), Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1991c. "Chromium(VI)," Integrated Risk Information System (IRIS), Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1991d. "Copper," Integrated Risk Information System (IRIS), Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1991e. "Vanadium Pentoxide," Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.
- EPA 1991f. "Carcinogenicity Assessment for Zinc and Compounds, and Oral RfD Assessment for Zinc Phosphide," Integrated Risk Information System (IRIS), online file, verification date June 1, 1991, retrieved February 1992, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.
- EPA 1991g. "Dinitrotoluene Mixture, 2,4-/2,6-," Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.
- EPA 1991h. "Anthracene," Health Effects Assessment Summary Tables. Annual FY-1991, NTIS PB91-921199, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC.
- EPA 1991i. "Anthracene," Integrated Risk Information System (IRIS), Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1991j. "Health Risk Assessment for Benz(a)anthracene," Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

- EPA 1991k. Drinking Water Criteria for Polycyclic Aromatic Hydrocarbons (PAHs), ECAO-CIN-D010, Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH, for the Office of Water, Washington, DC.
- EPA 19911. Risk Assessment Guidance for Superfund. Volume I—Human Health Evaluation Manual (Part B, Development of Risk-Based Preliminary Remediation Goals), Appendix D, OSWER Directive 9285.7-01B, Office of Emergency and remedial Response, Washington, DC.
- EPA 1991m. "Naphthalene," Integrated Risk Information System (IRIS), Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1990a. Air Quality Criteria for Lead: Supplement to the 1986 Addendum, EPA-600/8-89/049F, Environmental Criteria and Assessment Office, Research Triangle Park, NC.
- EPA 1990b. Health and Environmental Effects Document for Molybdenum and Selected Molybdenum Compounds, ECAO-CIN-G070, U.S Environmental Protection Agency, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.
- EPA 1990c. National Primary and Secondary Drinking Water Regulations; Synthetic Organic Chemicals and Inorganic Chemicals, *Federal Register*, Vol. 55, No. 143. 30370.
- EPA 1990d. "Tetrachloroethylene," EPA Cover Sheet, Integrated Risk Information System (IRIS), Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.
- EPA 1990e. "Trichloroethylene," Integrated Risk Information System (IRIS), Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1989a. Risk Assessment Guidance for Superfund, Volume 1: Human Health Evaluation Manual (Part A, Baseline Risk Assessment), EPA/540/1-89/002, Office of Emergency and Remedial Response, Washington, DC.
- EPA 1989b. Evaluation of the Potential Carcinogenicity of Lead and Lead Compounds, EPA/600/8-89/045A, Office of Health and Environmental Assessment, Washington, DC.
- EPA 1989c. Review of the National Ambient Air Quality Standards for Lead: Exposure Analysis Methodology and Validation, PB89207914/A5.OAQPS Staff Report, Office of Air Quality Planning and Standards, Research Triangle Park, NC.
- EPA 1989d. Mouse Oral Subchronic Study with Acenaphthene, Hazelton Laboratories, Inc., for the Office of Solid Waste, Washington, DC.
- EPA 1989e. Subchronic Toxicity Study in Mice with Anthracene, HLA Study No. 2399-131, Final Report, Hazleton Laboratories America, Inc., Rockville, MD, for the Office of Solid Waste, U.S. Environmental Protection Agency, Washington, DC.
- EPA 1988a. Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons (PAH), ECAO-CINDO10, Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH, for the Office of Drinking Water, Washington, DC.
- EPA 1988b. 13-Week Mouse Oral Subchronic Toxicity Study, Toxicity Research Laboratories, Ltd., Muskegon, MI, for the Office of Solid Waste and Emergency Response, Washington, DC.



- EPA 1988c. Updated Health Effects Assessment for Tetrachloroethylene, Final draft, ECAO-CIN-H009a, Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Washington, DC.
- EPA 1987a. Special Report on Ingested Arsenic: Skin Cancer; Nutritional Essentiality, EPA/625/3-87/013, Risk Assessment Forum, U.S. Environmental Protection Agency, Washington, DC.
- EPA 1987b. Health Effects Assessment for Beryllium and Compounds, ECAO-CIN-H113, Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Washington, DC.
- EPA 1987c. Health Assessment Document for Beryllium, EPA/600/8-84/026F, Office of Health and Environmental Assessment, Washington, DC.
- EPA 1987d. Drinking Water Criteria Document for Copper, ECAO-CIN-417, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Drinking Water, Washington, DC.
- EPA 1987e. Peer Review Workshop on Mercury Issues. Summary Report, Environmental Criteria and Assessment Office, Cincinnati, OH.
- EPA 1987f. Health Effects Assessment for Vanadium and Compounds, ECAO-CINN-H108, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.
- EPA 1987g. Health Effects Assessment for 1,1-Dichloroehtylene, ECAD-CIN-HO51a, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC.
- EPA 1987h. "Health and Environmental Effects Profile for Anthracene," Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Washington, DC.
- EPA 1987i. Health and Environmental Effects Profile for Benzo(ghi)perylene, ECAO-CIN-P276, Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Washington, DC.
- EPA 1987j. Health Effects Assessment for Selected Phthalic Acid Esters, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Washington, DC.
- EPA 1987k. Health and Environmental Effects Profile for Phthalic Acid Alkyl, Aryl and Alkyl/Aryl Ester, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Washington, DC.
- EPA 19871. Health and Environmental Effects Profile for Phenanthrene, ECAO-CIN-P226, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Washington, DC.
- EPA 1987m. Health and Environmental Effects Profile for Pyrene, ECAO-CIN-P277, Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Washington, DC.

- EPA 1987n. Health Assessment Document for Tetrachloroethylene (Perchloroethylene) Addendum, EPA 600/8-82-005FA, Environmental Criteria and Assessment Office, Office of Solid Waste and Emergency Response, Research Triangle Park, NC.
- EPA 19870. Addendum to the Health Assessment Document for Trichloroethylene. Updated Carcinogenicity Assessment for Trichloroethylene, External Review Draft, EPA/600/8-82/006FA, PB87-228045, Office of Health and Environmental Assessment, Office of Research and Development, Washington, DC.
- EPA 1986a. Drinking Water Criteria Document for Beryllium, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Drinking Water, Washington, DC.
- EPA 1986b. *Health Assessment Document for Beryllium*, External Review Draft, EPA 600-8-84-026B, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC.
- EPA 1986c. 90-Day Oral Toxicity Study of Copper Cyanide (CuCN) in Sprague-Dawley Rats, IIT Research Institute for the Office of Solid Waste, Washington, DC.
- EPA 1986d. Air Quality Criteria for Lead, Vol. I of IV, EPA-600/8-83/028aF, Environmental Criteria and Assessment Office, Research Triangle Park, NC.
- EPA 1986e. Air Quality Criteria for Lead, Vol. III of IV, EPA-600/8-83/028cF, Environmental Criteria and Assessment Office, Research Triangle Park, NC.
- EPA 1986f. Air Quality Criteria for Lead, Vol. II of IV, EPA-600/8-83/028bF, Environmental Criteria and Assessment Office, Research Triangle Park, NC.
- EPA 1986g. Air Quality Criteria for Lead, Vol. IV of IV, EPA-600/8-83/028dF, Environmental Criteria and Assessment Office, Research Triangle Park, NC.
- EPA 1986h. "Lead Effects on Cardiovascular Function, Early Development, and Stature; An Addendum to EPA Air Quality for Lead (1986)," in *Air Quality Criteria for Lead, Vol. I*, EPA-600/8-83/028aF, pp. A1-67, Environmental Criteria and Assessment Office, Research Triangle Park, NC.
- EPA 1986i. Subchronic (90-day) Toxicity of Thallium Sulfate in Sprague-Dawley Rats, Office of Solid Waste, Washington, DC.
- EPA 1986j. Health and Environmental Effects Profile for Dinitrotoluene, ECAO-CIN-P183, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.
- EPA 1986k. Ninety-day Gavage Study in Albino Rats Using Acetone, Office of Solid Waste and Emergency Response, Washington, DC.
- EPA 19861. Carcinogenicity Assessment of Chlordane and Heptachlor/Heptachlor Epoxide, EPA-600/6-87/004, Carcinogen Assessment Group, Office of Health and Environmental Assessment, Washington, DC.
- EPA 1986m. "Health and Environmental Effects Profile for Naphthalene," EPA-CIN-P192, Environmental Criteria and Assessment Office, Office of Health and Environmental Assessment, Cincinnati, OH.
- EPA 1985a. Drinking Water Criteria Document for Silver (Final Draft), ECAO-CIN-026, PB86-118288, Environmental Criteria and Assessment Office, Cincinnati, OH.



- EPA 1985b. Health Assessment Document for Chloroform, Final Report, EPA/600/8-84/004F, NTIS PB86-105004/XAB, Office of Health and Environmental Assessment, Washington, DC.
- EPA 1985c. Drinking Water Criteria Document for Heptachlor, Heptachlor Epoxide and Chlordane (Final Draft), ECAO-CIN-406; EPA-600/X-84-197-1; PB86-117991, Office of Environmental Criteria and Assessment, Cincinnati, OH, for the Office of Drinking Water, Washington, DC.
- EPA 1985d. Health Assessment Document for Tetrachloroethylene (Perchloroethylene), Final Draft, EPA 600/8-82-005F; NTIS PB 84-162882, Environmental Criteria and Assessment Office, Office of Solid Waste and Emergency Response, Research Triangle Park, NC.
- EPA 1985e. Health Assessment Document for Trichloroethylene, Final Report, EPA/600/8-82/006F, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC.
- EPA 1985f. Health and Environmental Effects Profile for Chloroethene, EPA-CIN-P155, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.
- EPA 1984a. Health Assessment Document for Arsenic, EPA 600/8-32-021F, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC.
- EPA 1984b. Health Effects Assessment of Hexavalent Chromium, EPA/540/1-86-019, Office of Research and Development, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.
- EPA 1984c. Health Effects Assessment for Zinc (and Compounds), EPA/540/1-86-048, Office of Research and Development, Washington, DC.
- EPA 1984d. Health Effects Assessment for Polycyclic Aromatic Hydrocarbons, EPA/540/1-86-013, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC.
- EPA 1983. Reportable Quantity for Dichloromethane. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC.
- EPA 1982. Health Assessment Document for Tetrachloroehtylene (Perchloroethylene), Final Report, EPA/600/8-82/005F, Office of Health and Environmental Assessment, Washington, DC.
- EPA 1980a. Ambient Water Quality Criteria Document for Chlorinated Ethanes, EPA-440/5-80-029, PB81-117400, Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Water Regulations and Standards, Washington, DC.
- EPA 1980b. Ambient Water Quality Criteria for Dinitrotoluene, EPA-440/5-80-045, NTIS PB81-117566, Office of Water Regulations and Standards, Washington, D.C.
- EPA 1980c. Ambient Water Quality Criteria for Acenaphthene, EPA-440/5-80-015, Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC.
- EPA 1980d. Water Quality Criteria Document for Polynuclear Aromatic Hydrocarbons, Office of Research and Development, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Water Regulations and Standards, Washington, DC.
- EPA 1980e. Ambient Water Quality Criteria for Vinyl Chloride, EPA 440/5-80-078, Office of Water Regulations and Standards, Criteria and Standards Division, Washington, DC.

Epstein, S. S. 1976. "Carcinogenicity of Heptachlor and Chlordane," Sci. Total Environ. 6:103-154.

- Epstein, S. S., and Ozonoff, D. 1987. "Leukemias and Blood Dyscrasias Following Exposure to Chlordane and Heptachlor," *Teratogen. Carcinog. Mutagen.* 7:527-540.
- Etnier, E. 1987. Water Quality Criteria for 2,4-Dinitrotoluene and 2,6-Dinitrotoluene. Final Report, AD-ORNL-6312, Oak Ridge National Laboratory, Oak Ridge, Tennessee.
- Fairhall, L. T., Dunn, R. C., Sharpless, N. E., and Pritchard, E. A. 1945. "The Toxicity of Molybdenum," U.S. Public Health Bull. No. 293.
- Falkson, G., Klein, B., and Falkson, H. 1985. "Hematological Toxicity: Experience with Anthracyclines and Anthracenes," *Exp. Hematol.* 13:64-71.
- Fawer, R. F., Ribaupierre, Y., Guillemin, M. P., Berook, M., and Lob, M. 1983. "Measurement of Hand Tremor Induced by Industrial Exposure to Metallic Mercury," *Br. J. Indust. Med.* 40:204–208.
- Feldman, R. G., Niles, C. A., Kelly-Heyes, M., Sax, D. S., Dixon, W. J., Thompson, D. J., and Landau, E. 1979. "Peripheral Neuropathy in Arsenic Smelter Workers," *Neurology* 29:939-944.
- Felicetti, S. W., Thomas, R. G., and McClellan, R. O. 1974a. "Retention of Inhaled Antimony-124 in the Beagle Dog as a Function of Temperature of Aerosol Formation," *Health Phys.* 26:525-532.
- Felicetti, S. W., Thomas, R. G., and McClellan, R. O. 1974b. "Metabolism of Two Valence States of Inhaled Antimony in Hamsters," Am. Ind. Hyg. Assoc. J. 355:292-300.
- Fenves, A. Z., Emmett, M., and White, M. G. 1984. "Lithium Intoxication Associated with Acute Renal Failure," South Med. J. 77:1472-1474.
- Ferm, V. H., and Carpenter, S. J. 1968. "Malformations Induced by Sodium Arsenate," J. Reprod. Fert. 17:199-201.
- Ferm, V. H., and Hanlon, D. P. 1974. "Toxicity of Copper Salts in Hamster Embryonic Development," Biol. Reprod. 11:97-101.
- Feron, V. J., Hendriksen, C. F. M., Speek, A. J., et al. 1981. "Lifespan Oral Toxicity Study of Vinyl Chloride in Rats," Food Cosmet. Toxicol. 19:317-333.
- Fishbein, L. 1985. "An Overview of Environmental and Toxicological Aspects of Aromatic Hydrocarbons. IV. Ethylbenzene," Sci. Total Environ. 44:269-287.
- Fitzhugh, O. G., and Buschke, W. H. 1949. "Production of Cataracts in Rats by Beta-Tetralol and Other Derivatives of Naphthalene," Arch. Ophthal. 41:572-582.
- Forbes, P. D., Davies, R. E., and Urbach, F. 1976. 'Phototoxicity and Photocarcinogenesis: Comparative Effects of Anthracene and 8-Methoxypsoralen in the Skin of Mice," Food Cosmet. Toxicol. 14:303-306.
- Ford, E., and Huggins, C. 1963. "Selective Destruction in Testis Induced by 7,12-Dimethylbenz(a)anthracene," J. Exp. Med. 118:27.
- Formigli, E., Scelsi, R., Poggi, P., et al. 1986. "Thallium Induced Testicular Toxicity in the Rat," *Environ.* Res. 40:531.

Fowler, B. A., and Weissberg, J. B. 1974. "Arsine Poisoning," New Eng. J. Med. 291:1171-1174.

- Fowler, B. A., and Woods, J. S. 1979. "The Effects of Prolonged Oral Arsenate Exposure on Liver Mitochondria of Mice: Morphometric and Biochemical Studies," *Toxicol. Appl. Pharmacol.* 50:177-187.
- Fowler, B. A., Woods, J. S., and Schiller, C. M. 1979. "Studies of Hepatic Mitochondria Structure and Function: Morphometric and Biochemical Evaluation of In Vivo Perturbation by Arsenate," Lab. Invest. 42:313-320.
- Fox, M. R. S., and Jacobs, R. M. 1986. "Zinc: Essentiality, Function, Effects of Deficiency, and Requirementsy," pp. 214–248, in *Metal Ions in Biological Systems*, Sigel, H., ed., Marcel Dekker, Inc., New York.
- Frank, S. B., and Cohen, H. J. 1961. "Fixed Drug Eruption Due to Paradichlorobenzene," NY State J. Med. 61:4079.
- Franzblau, A., and Lilis, R. 1989. "Acute Arsenic Intoxication from Environmental Arsenic Exposure," Arch. Environ. Health 44(6):385-390.
- Freiman, D. G., and Hardy, H. L. 1970. "Beryllium Disease: The Relation of Pulmonary Pathology to Clinical Course and Prognosis Based on a Study of 130 Cases from the U.S. Beryllium Case Registry," Hum. Pathol. 1:25-44.
- Freundt, K. J., Liebaldt, G. P. and Lieberwirth, E. 1977. "Toxicity Studies on trans-1,2-Dichloroethene," Toxicology 7:141-153.
- Friberg, L. 1950. "Health Hazards in the Manufacture of Alkaline Accumulators with Special Reference to Chronic Cadmium Poisoning," *Acta Med. Scad.* (Suppl. 240) 138:1-124.
- Friberg, L., and Lener, J. 1986. "Molybenum," pp. 446–461, in Handbook on the Toxicology of Metals, 2nd ed., Friberg, L., Nordberg, G. F., and Vouk, V. B., eds., Elsevier/North-Holland Biomedical Press, New York.
- Friedlander, B. R., Hearne, F. T., and Hall, S. 1978. "Epidemiologic Investigation of Employees Chronically Exposed to Methylene Chloride." J. Occup. Med. 20:657-666.
- Fukuda, K., Takemoto, K., and Tsuruta, H. 1983. "Inhalation Carcinogenicity of Trichloroethylene in Mice and Rats," Ind. Health 21:243-254.
- Furchner, J. E., Richmond, C. R., and London, J. E. 1973. "Comparative Metabolism of Radionuclides in Mammals: VII. Retention of Beryllium in the Mouse, Rat, Monkey and Dog," *Health Phys.* 24:292-300.
- Geddie, J. E., Amin, S., Hui, K. et al. 1987. "Formation and Tumorigenicity of Benzo(b)fluoranthene Metabolites in Mouse Epidermis," *Carcinogenesis* 8:1579-1584.
- Gehring, P. J. 1968. "Hepatotoxic Potency of Various Chlorinated Hydrocarbon Vapors Relative to Their Narcotic and Lethal Potencies in Mice," *Toxicol. Appl. Pharmacol.* 13:287-298.
- Gerber, G. B., Maes, J., and Eykens, B. 1982. "Transfer of Antimony and Arsenic to the Developing Organism," Arch. Toxicol. 49:159-168.
- Ghantous, H., and Danielsson, B. R. G. 1986. "Placental Transfer and Distribution of Toluene, Xylene, and Benzene and Their Metabolites During Gestation in Mice," *Biol. Res. Preg.* 7:98–105.
- Ghetti, G., and Mariani, L. 1956. [Eye Changes due to Naphthalene.] Med. Lav. 47:533-538. (In Italian)

- Gilioli, R., Bulgheroni, C., Terrano, T., Filippini, G., Massetto, G., and Boeri, R. 1978. "Studio... Neurologico Transverwale e Longitudinale di Una Poplazione Operaia Addetta Alla Produzione di Ftalati," Med. Lav. 69(5):620-631.
- Glaser, U., Hochrainer, D., Kloppel, H., and Oldiges, H. 1986. "Carcinogenicity of Sodium Dichromate and Chromium (VI/III) Oxide Aerosols Inhaled by Male Wistar Rats," *Toxicology* 42:219-232.
- Goldschmidt, B. M., Katz, C., and Van Duuren, B. L. 1973. "The Cocarcinogenic Activity of Non-Carcinogenic Aromatic Hydrocarbons" (Abstract No. 334), Proc. Am. Assoc. Cancer Res. 17:84.

Goldwater, L. J. 1972. Mercury: A History of Quicksilver, York Press, Baltimore, MD.

- Goodman, L. S., and Gilman, A. 1985. The Pharmacologic Bases of Therapeutics, 7th ed., MacMillan Publishing Co., New York.
- Gopinath, C., Hall, G. A., and Howell, J. M. 1974. (Title not given), Res. Vet. Sci. 16:57-69.
- Gosselin, R. E., Smith, R. P., Hodge, H. C. et al. 1984. Clinical Toxicology of Commercial Products, 5th ed., The Williams and Wilkins Co., New York.
- Goyer, R. A. 1988. "Lead," pp. 359-382, in *Handbook on Toxicity of Inorganic Compounds*. Seiler, H. G., and Sigel, H., eds., Marcel Dekker, Inc., New York.
- Goyer, R. A. 1991. "Toxic Effects of Metals," pp. 662-663, in Casarett and Doull's Toxicology. The Basic Science of Poisons, 4th ed., Amdur, M. O., Doull, J. D., and Klaassen, C. D., eds., Permagon Press, New York.
- Grandjean, E., Munchinger, R., Turrian, V., et al. 1955. "Investigations into the Effects of Exposure to Trichloroethylene in Mechanical Engineering," Brit. J. Ind. Med. 12:131-142.

Grant, W. M. 1986. Toxicology of the Eye, 3rd ed., p. 1036, Charles C. Thomas, Springfield, IL.

- Gray, T. J. B, Butterworth, K. R., Gaunt, I. F., Grasso, P., and Gangolli, S.D. 1977. "Short-term Toxicity Study of di(2-Ethylhexyl)phthalate in Rats," *Food Cosmet. Toxicol.* 15(5):389-400.
- Green, V. A. 1970. "Effects of Pesticides on Rat and Chick Embryo," pp. 183-209, in *Trace Substance Environmental Health 3*, Proceedings of the 3rd Annual Conference, University of Missouri.
- Greenspan, B. J., Allen, M. D., and Rebar, A. H. 1986. "Inhalation Toxicity of Lithium Combustion Aerosols in Rats," J. Toxicol. Environ. Health 18:627-637.
- Grimmer, G., Brune, H., Dettbarn, G., et al. 1988. "Urinary and Faecal Excretion of Chrysene and Chrysene Metabolites by Rats After Oral, Intraperitoneal, Intratracheal or Intrapulmonary Application," Arch. Toxicol. 62:401-405.
- Gross, P., Harely, R. H., and deTreville, R. T. P. 1973. "Pulmonary Reaction to Metallic Aluminum Powders," Arch. Environ. Health. 26:227-236.

Groth, P. H., Stettler, L. E., Burg, J. R. et al. 1986. "Carcinogenic Effects of Antimony Trioxide and Antimony Ore Concentrate in Rats," J. Toxicol, Environ. Health 18:607-626.

Guja, A. 1973. "Gastric Disorders and the Uropepsin Level in Persons Exposed to the Action of Zinc Oxides," Wiad. Lek. 26:141.

Guthrie, B. E. 1982. "The Nutritional Role of Chromium," pp. 117–148, in *Biological and Environmental* Aspects of Chromium, Langard, S., ed., Elsevier Biomedical Press, Amsterdam.

- Hake, C., and Stewart, R. 1977. "Human Exposure to Tetrachloroethylene: Inhalation and Skin Contact," Environ. Health Perspect. 21:231-238.
- Hall, H. C., Perl, M., and Pfefferbaum, B. 1979. "Lithium Therapy and Toxicity," Am. Fam. Pract. 19:133-139.
- Hall, T. C., Wood, C. H., Stoeckle, J. D., and Tepper, L. B. 1959. "Case Data from the Beryllium Registry," Am. Med. Assoc. Arch. Ind. Health 19:100-103.
- Halme, E. 1961. "On the Carcinogenic Effect of Drinking Water Containing Zinc," (Ger.) Vitalstoffe 6:59-66.
- Hamill, P. V. V., Steinberger, E., Levine, R. J., et al. 1982. "The Epidemiologic Assessment of Male Reproductive Hazard from Occupational Exposure to TDA and DNT," J. Occup. Med. 24:985-993.
- Hamilton, J. W., and Wetterhahn, K. E. 1988. "Chromium," pp. 239-250, in Handbook on Toxicity of Inorganic Compounds, Seiler, H. G., and Sigel, H., eds., Marcel Dekker, Inc., New York.
- Hansen, E. S. 1990. "Shared Risk Factors for Cancer and Artherosclerosis---A Review of the Epidemiological Evidence," *Mutat Res.* 239:163-179.
- Harada, M. 1978. "Congenital Minamata Disease: Intrauterine Methylmercury Poisoning," Teratology 18:285-288.
- Harlan, W. R., Landi, J. R., Shcmouder, R. L., Goldstein, N. G., and Harlan, L. C. 1985. "Blood Lead and Blood Pressure: Relationship in the Adolescent and Adult US Population," J. Am. Med. Assoc. 253:530-534
- Harris, W. R., Friedman, S. B., and Silberman, D. 1984. "Behavior of Vanadate and Vanadyl Ion in Canine Blood," J. Inorg. Biochem. 20:157-169.
- Hatch, G. E., Boykin, E., Graham, J. A., Lewtas, J., Pott, F., Loud, K., and Mumford, J. L. 1985. "Inhalable Particles and Pulmonary Host Defense: In Vivo and In Vitro Effects of Ambient Air and Combustion Particles," Environ. Res. 36:67-80.
- Haun, C. C., Vernot, E. H., Darmer, K. I., et al. 1972. "Continuous Animal Exposure to Low Levels of Dichloromethane," AMRL-TR-130, Paper No. 12, pp. 199–208, Proc. 3rd An. Conf. Env. Toxicol. Aerospace Med. Res. Lab., Wright-Patterson Air Force Base, Ohio.
- Hawkins, D. R., Chasse, L. F., Woodhouse, R. N., and Cresswell, G. D. 1980. "The Distribution, Excretion, and Biotransformation of p-Dichloro[¹⁴C]benzene in Rats After Repeated Inhalation, Oral, and Subcutaneous Doses," *Xenobiotica* 10:81–95.
- Hawley, G. G., ed., 1981. The Condensed Chemical Dictionary, 10th ed., p. 375, Van Nostrand Reinhold Co., New York.
- Hayes, R. B. 1982. "Carcinogenic Effects of Chromium," pp. 221–247, in *Biological and Environmental* Aspects of Chromium, Langard, S., ed., Elsevier Biomedical Press, Amsterdam.
- Hayes, W. J., Jr. 1982. Pesticide Studies in Man, pp. 41-50, Williams and Eilkins, Baltimore, MD.
- Haywood, S. 1985. "Copper Toxicosis and Tolerance in the Rat. I. Changes in Copper Content of the Liver and Kidney," J. Pathol. 145:149-158.
- Haywood, S. 1980. "The Effect of Excess Dietary Copper on the Liver and Kidney of the Male Rat," J. Comp. Pathol. 90:217-232.

Healy, J. W. 1980. "Review of Resuspension Models," pp. 209-235, in *Transuranic Elements in the*_____ Environment, Hanson, W. C., ed., U.S. Department of Energy, Washington, DC.

Healy, T. E. J., Poole, T. R., and Hopper, A. 1982. "Rat Fetal Development and Maternal Exposure to Trichloroethylene at 100 ppm," Br. J. Anaesth. 54:337-341.

Hearne, F. T., Grose, F., Pifer, J. W. et al. 1987. "Methylene Chloride Mortality Study: Dose-Response Characterization and Animal Model Comparison," J. Occup. Med. 29:217-228.

Heldaas, S. S., Langård, S. L., and Andersen, A. 1984. "Incidence of Cancer Among Vinyl Chloride and Polyvinyl Chloride Workers," Br. J. Ind. Med. 41:25-30.

Heller, W. 1950. "Experimental Study on Tumors Produced by Light. 2. Tumors Produced by Light Through Photosensibilisation," *Strahlentherapie* 81:529-548.

Henderson, R. G., Sabourin, P. J., Bechtold, W. E., et al. 1989. "The Effect of Dose, Dose Rate, Route of Administration, and Species on Tissue and Blood Levels of Benzene Metabolites," *Environ. Health Perspect.* 82:9-17.

Henschler, D. 1977. "Metabolism and Mutagenicity of Halogenated Olefins—A Comparison of Structure and Activity," *Environ. Health Perspec.* 21:61-64.

Henschler, D., and Hoos, R. 1982. "Metabolic Activation and Deactivation Mechanisms of Di, Tri, and Tetrachloroethylenes," pp. 659–666, in Snyder, R., et al., eds., *Biological Reactive Intermediates-II: Chemical Mechanisms and Biological Effects*, Plenum Press, New York.

Heppel, L. A., Neal, P. A., Perrin, T. L., et al. 1946. "Toxicology of 1,2-Dichloroethane. V. Effects of Daily Inhalations," J. Ind. Hyg. Toxicol. 28:113-120.

Heppel, L., Neal, P., Perrin, T., et al. 1944. "Toxicology of Dichloromethane (Methylene Chloride). I. Studies on Effects of Daily Inhalation," J. Ind. Hyg. Toxicol. 26:8-16.

Hestbech, J., Hansen, H. E., Amdisen, A., and Olsen, S. 1977. "Chronic Renal Lesions Following Long-Term Treatment with Lithium," Kidney Int. 12:205-213.

Heyman, A., Pfeiffer, J. B., Willett, R. W., and Taylor, H. M. 1956. "Peripheral Neuropathy Caused by Arsenical Intoxication," *New England J. Med.* 254(9):401-409.

Heywood, R., and Sortwell, R. J. 1979. "Arsenic Intoxication in the Rhesus Monkey," Toxicol. Lett. 3:137-144.

Heywood, R., Sortwell, R. J., Noel, P. R. B., et al. 1979. "Safety Evaluation of Toothpaste Containing Chloroform. III. Long-Term Study in Beagle Dogs," J. Environ. Pathol. Toxicol. 2:835-851.

Hindmarsh, J. T., and McCurdy, R. F. 1986. "Clinical and Environmental Aspects of Arsenic Toxicity," CRC Crit. Rev. Clin. Lab. Sci. 23:315-347.

Hindmarsh, J. T., McLetchie, O. R., Heffernan, L. P., Haynie, O. A., Ellenberger, H. A., McCurdy, R. F., and Thiebaux, H. J. 1977. "Electromyographic Abnormalities in Chronic Environmental Arsenicalism," J. Anal. Toxicol. 1:270-276.

Hine, C. H., Pinto, S. S., and Nelson, K.W. 1977. "Medical Problems Associated with Arsenic Exposure," J. Occup. Med. 19(6):391-396.

Hipolito, R. 1980. "Xylene Poisoning in Laboratory Workers: Case Reports and Discussion," Lab. Med. 11:593-595.

- Hodgson, R. M., Seidel, A., Bochnitschek, W., Glatt, H. R., Oesch, F., and Grover, P. L. 1986. "Metabolism of the Bay-Region Diol-epoxide of Chrysene to a Triol-epoxide and the Enzyme-Catalyzed Conjugation of these Epoxides with Glutathione," *Carcinogenesis* 7:2095-2098.
- Hodgson, R. M., Weston, A., and Grover, P. L. 1983. "Metabolic Activation of Chrysene in Mouse Skin: Evidence for the Involvement of Triol-epoxide," *Carcinogenesis* 4:1639-1643.
- Hoffmann, D., and Wynder, E. L. 1966. "Contribution to the Carcinogenic Action of Dibenzopyrenes," Z. Krebsforsch 68:137-149.
- Hoffmann, D., Rathkamp, F., Nesnow, S. and Wynder, E. L. 1972. "Fluoranthenes: Quantitative Determination in Cigarette Smoke, Formation by Pyrolysis, and Tumor-Initiating Activity," J. Natl. Cancer Inst. 49:1165-1175.
- Hollingsworth, R. L., Rowe, V. K., Oyen, R., et al. 1956. "Toxicity of Paradichlorobenzene: Determinations on Experimental Animals and Human Subjects," AMA Arch. Ind. Health 14:138-147.
- Hong, C. B., Winston, J. M., Thornburg, L. P., and Lee, C. C. 1981. "Follow-up Study on the Carcinogenicity of Vinyl Chloride and Vinylidene Chloride in Rats and Mice: Tumor Incidence and Mortality Subsequent to Exposure," J. Toxicol. Environ. Health 7:909-924.
- Hood, R. D., and Bishop, S. L. 1972. "Teratogenic Effects of Sodium Arsenate in Mice," Arch. Environ. Health 24:62-65.
- Hood, R. D., and Ottley, M. S. 1985. "Developmental Effects Associated with Exposure to Xylene: A Review," Drug Chem. Toxicol. 8:281-297.
- Hood, R. D., Thecker, G. T., and Patterson, B. L. 1977. "Effects in the Mouse and Rat of Prenatal Exposure to Arsenic," *Environ. Health Perspect.* 19:219-222.
- Horton, A. W., and Christian, G. M. 1974. "Cocarcinogenic Versus Incomplete Carcinogenic Activity Among Aromatic Hydrocarbons: Contrast Between Chrysene and Benzo(b)triphenylene," J. Natl. Cancer Inst. 49:1017-1020.
- HSDB (Hazardous Substances Database) 1995. Online file, available through the National Library of Medicine's MEDLARS System, National Library of Medicine, Bethesda, MD.
- HSDB 1993. National Library of Medicine, Bethesda, MD.
- Hsieh, G. C., Parker, R. D. R., and Sharma, R. P. 1988. "Subclinical Effects of Groundwater Contaminants: II. Alteration of Regional Brain Monoamine Neurotransmitters by Benzene in CD-1 Mice," Arch. Environ. Contam. Toxicol. 17:799-805.
- Huang, Y. Z., Qian, X. C., Wang, G. Q., Xiao, B. Y., Ren, D. D., Feng, Z. Y., Wu, J. Y., Xu, R. J., and Zhang, F. 1985. "Endemic Chronic Arsenism in Xinjiang," *Chin. Med. J.* (Eng). 98:219–222.
- Huff, J. E., Kaseman, J. K., DeMarini, D. M., et al. 1989. "Multiple Site Carcinogenicity of Benzene in Fischer 344 Rats and B6C3F₁ Mice," *Environ. Health Perspect.* 82:125-163.

Huggins, C., and Yang, N. C. 1962. "Induction and Extinction of Mammary Cancer," Science 37:257-262.

IAEA (International Atomic Energy Agency) 1994. Handbook of Parameter Values for the Prediction of Radionuclide Transfer In Temperate Environment, Tech. Rep. Ser. No. 364, Vienna, Austria.

- IAEA 1992. Effects of Ionizing Radiation on Plants and Animals at Levels Implied by Current Radiation. Protection Standards, IAEA Technical Report Series 332, Vienna, Austria.
- IARC (International Agency for Research on Cancer) 1987. "Overall Evaluations of Carcinogenicity: An Updating of IARC Monographs Volumes 1 to 42," IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Supplement 7, Geneva, Switzerland.
- IARC 1986a. "Vinylidine Chloride," pp. 195-226, in IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol. 39, World Health Organization, Lyon, France.
- IARC 1986b. "Dichloromethane," pp. 43-85, in IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Some Halogenated Hydrocarbons and Pesticide Exposures, Vol. 41, World Health Organization, Lyon, France.
- IARC 1985. "IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Polynuclear Aromatic Compounds, Part 4, Bitumens, Coal-Tars and Derived Products, Shale-Oils and Soots," pp. 88, 91, 164, 225, in *IARC Monographs on the Evaluation of Carcinogenic Risk of the Chemical to Man*, Vol. 35, World Health Organization, Lyon, France.
- IARC 1983a. "Benzo(a)pyrene," pp. 33-224, in IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Polynuclear Aromatic Compounds. Part 1. Chemical, Environmental and Experimental Data, Vol. 32, World Health Organization, Lyon, France.
- IARC 1983b. "Benzo(b)fluoranthene," pp. 33-91, 147-153, 163-170, in IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Polycyclic Aromatic Compounds, Part 1, Chemical, Environmental, and Experimental Data, Vol. 32, World Health Organization, Lyon, France.
- IARC 1983c. "Benzo(g,h,i)perylene," pp. 195-204, in IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals for Humans. Polynuclear Aromatic Compounds, Part 1, Chemical, Environmental, and Experimental Data, Vol. 32, World Health Organization, Lyon, France.
- IARC 1983d. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Polynuclear Aromatic Compounds. Part 1. Chemical, Environmental and Experimental Data, pp. 247–261, Vol. 32, World Health Organization, Lyon, France.
- IARC 1983e. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Polynuclear Aromatic Compounds. Part 1. Chemical, Environmental and Experimental Data, pp. 33-224, Vol. 32, World Health Organization, Lyon, France.
- IARC 1983f. "Fluoranthene," pp. 355-364, in IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Polynuclear Aromatic Compounds. Part 1. Chemical, Environmental and Experimental Data, Vol. 32, World Health Organization, Lyon, France.
- IARC 1983g. "Indeno(1,2,3-cd)pyrene," pp. 373-378, in IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans. Polycyclic Aromatic Compounds, Part 1, Chemical, Environmental and Experimental Data, Vol 32, World Health Organization, Lyon, France.
- IARC 1983h. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Polynuclear Aromatic Compounds. Part 1. Chemical, Environmental and Experimental Data, pp. 419–430, Vol. 32, World Health Organization, Lyon, France.
- IARC 1983i. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Polynuclear Aromatic Compounds. Part 1. Chemical, Environmental and Experimental Data, pp. 430-445, Vol. 32, World Health Organization, Lyon, France.

- IARC 1980. "Lead and Lead Compounds," pp. 325-415, in IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Some Metals and Metallic Compounds, Vol. 23, World Health Organization, Lyon, France.
- IARC 1979a. "1,1,2-Trichloroethane," pp. 533-543, in IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemical to Humans, Some Halogenated Hydrocarbons, Vol. 20. World Health Organization, Lyon, France.
- IARC 1979b. "Chloroform," pp. 401-427 in Some Halogenated Hydrocarbons. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol. 20, World Health Organization, Lyon, France.
- IARC 1979c. "Heptachlor and Heptachlor Epoxide," pp. 129–154, in IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Some Halogenated Hydrocarbons, Vol. 20, World Health Organization, Lyon, France.
- IARC 1979d. "Vinyl Chloride, Polyvinyl Chloride and Vinyl Chloride-Vinyl Acetate Polymers," pp. 377-438, in IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans. Some Monomers, Plastics and Synthetic Elastomers, and Acrolein, Vol. 19, World Health Organization, Lyon, France.
- IARC 1973a. "Benzo(a)pyrene," pp. 91–136, in IARC Monographs on the Evaluation of Carcinogenic Risks of the Chemical to Man. Centain Polycyclic Aromatic Hydrocarbons and Heterocyclic Compounds, Vol. 3, World Health Organization, Lyon, France.
- IARC 1973b. "Dibenz(a,h)anthracene," pp. 178–196, in IARC Monographs on the Evaluation of Carcinogenic Risk of the Chemical to Man. Certain Polycyclic Aromatic Hydrocarbons and Heterocyclic Compounds, Vol. 3, World Health Organization, Lyon, France,.
- IBTL (Industrial Biotest Laboratories) 1972. Acute Toxicity with Antimony Oxide, OTS206223.
- ICRP (International Commission on Radiological Protection) 1960. Report of Committee II on Permissible Dose for Internal Radiation. Recommendations of the International Commission on Radiological Protection, p. 163, ICRP Publ. No. 2, Pergamon Press, Oxford, England.
- Iijima, S., Matsumoto, N., and Lu, C. C. 1983. "Transfer of Chromic Chloride to Embryonic Mice and Changes in the Embryonic Mouse Neuroepithelium," *Toxicology* 26:257-265.
- Infante, P. F., Wagoner, J. K., McMichael, A. J., et al. 1976. "Genetic Risks of Vinyl Chloride;" Lancet (April) 734-735.
- Inoue, T. 1983. "Acetone," pp. 38-39, in *Encyclopaedia of Occupational Health and Safety*, 3rd rev. ed., Parmeggiani, L., ed., International Labor Office, Geneva.
- IRDC (International Research and Development Corporation) 1973. Unpublished report to Velsicol Chemical Corporation, Eighteen Month Oral Carcinogenic Study in Mice, September 26.
- Irvin, T. R., and Martin, J. E. 1987. "In Vitro and In Vivo Embryotoxicity of Fluoranthene, a Major Prenatal Toxic Component of Diesel Soot," *Teratology* 35:65A. (Abstract)
- ITII (International Technical Information Institute) 1988. Toxic and Hazardous Industrial Chemicals Safety Manual for Handling and Disposal, with Toxicity and Hazard Data, pp. 562–564, Toyko, Japan.

Ivankovic, S., and Preussmann, R. 1975. "Absence of Toxic and Carcinogenic Effects After... Administrations of High Doses of Chromic Oxide Pigment in Subacute and Long-term Feeding Experiments in Rats," Fd. Cosmet. Toxicol. 13:347-351.

Ivanov, S. V. 1962. "Toxicity of Ethylbenzene," Tr. Voronezhsk. Gos. Med. Inst. 47:80.

- Jacob, J., Schmoldt, A., and Grimmer, G. 1982. "Formation of Carcinogenic and Inactive Chrysene Metabolites by Rat Liver Microsomes of Various Monooxygenase Activities," Arch. Toxicol. 51:255-265.
- Jacob, J., Schmoldt, A., Hamann, M., Raab, G., and Grimmer, G. 1987. "Monooxygenase Induction by Various Xenobiotics and Its Influence on Rat Liver Microsomal Metabolism Of Chrysene in Comparison to Benz(a)anthracene," Cancer Lett. 34:91-102.
- Jaeger, A., Sauder, P., Kopferschmitt, J., and Jaegle, J. L. 1985. "Toxicokinetics of Lithium Intoxication Treated by Hemodialysis," J. Toxicl. Clin. Toxicol. 23:501-517.
- Jarrell, W. M., Page, A. L., and Elseewi, A. A. 1980. "Molybdenum in the Environment," Residue Rev. 74:1-43.
- Jerina, D. M., Lehr, R., Schaefer-Ridder, M., Yagi, H., Karle, J. M., Thakker, D. R., Wood, A. W., Lu, A. Y. H., Ryan, D., West, S., Levin, W., and Conney, A.H. 1977. "Bay-Region Epoxides of Dihydrodiols: A Concept Explaining the Mutagenic and Carcinogenic Activity of Benzo(a)pyrene and Benzo(a)anthracene," pp. 639-657, in Origins of Human Cancer, Cold Spring Harbor Laboratory.
- Jeter, M., and Davis, G. K. 1954. "The Effect of Dietary Molybdenum Upon Growth, Hemoglobin, Reproduction and Lactation of Rats," J. Nutr. 54:215-220.
- Jorens, P. G., and Schepens. J. C. 1993. "Human Pentachlorophenol Poisoning," Human and Exp. Toxicol. 12:479-495.
- Jorgenson, T. A., Meierhenry, E. F., Rushbrook, C. J., et al. 1985. "Carcinogenicity of Chloroform in Drinking Water to Male Osborne-Mendel Rats and Female B6C3F₁ Mice," Fund. Appl. Toxicol. 5:760-769.
- Kabata-Pendias, A, and Pendias, H. 1984. Trace Elements in Soils and Plants, CRC Press, Inc., Boca Raton, FL.
- Kalf, G. F., Post, G. B., and Snyder, R. 1987. "Solvent Toxicology: Recent Advances in the Toxicology of Benzene, the Glycol Ethers and Carbon Tetrachloride," Ann. Rev. Pharmacol. Toxicol. 27:399-427.
- Kazantzis, G. 1986. "Thallium," pp. 549-567, in *Handbook of the Toxicology of Metals*, 2nd ed., Friberg, L., Nordberg, G. F., and Vouk, V., eds., Elsevier Science Publishers B.V., New York.
- KDEP (Commonwealth of Kentucky, Department for Environmental Protection) 1998. Letter from R. H. Daniell to J. Hodges and J. C. Massey entitled "Re: Methods for Conducting Human Health Risk Assessments and Risk Evaluations at the Paducah Gaseous Diffusion Plant, Paducah, McCracken County, Kentucky, DOE/OR/07-1506&D1. KY8-890-008-982," dated February 18, 1998.
- KDEP 1995. Risk Assessment Guidance, Natural Resources and Environmental Protection Cabinet, Department for Environmental Protection, Frankfort, KY.
- Keller, K. A., and Snyder, C. A. 1988. "Mice Exposed In Utero to 20 ppm Benzene Exhibit Altered Numbers of Recognizable Hematopoietic Cells up to Seven Weeks After Exposure," Fundam. Appl. Toxicol. 10:224-232.



- Kellerova, V. 1985. "Electroencephalographic Findings in Workers Exposed to Benzene," J. Hyg. Epidemiol. Microbiol. Immunol. 29:337-346.
- Kennaway, E. L. 1924a. "On Cancer-Producing Factor in Tar," Br. Med. J. i:564-567.
- Kennaway, E. L. 1924b. "On the Cancer-Producing Tars and Tar-Fractions," J. Ind. Hyg. 5:462-488.
- Ketcheson, M. R., Barron, G. P., and Cox, D. H. 1969. "Relationship of Maternal Dietary Zinc During Gestation and Lactation to Development and Zinc, Iron, and Copper Content of the Postnatal Rat," J. Nutr. 98:303-311.
- Ketkar, M., Reznik, G., Schneider, P., and Mohr, U. 1978. "Investigations on the Carcinogenic Burden by Air Pollution in Man. Intratracheal Instillation Studies with Benzo(a)pyrene in Bovine Albumin in Syrian Hamsters," Cancer Lett. 4:235-239.
- Kiviluoto, M., Pyy, L., and Pakarinen, A. 1981. "Clinical Laboratory Results of Vanadium-Exposed Workers," Arch. Environ. Health 36:109-113.
- Kjellstrand, P., Holmquist, B., Alm, P., et al. 1983. "Trichloroethylene: Further Studies of the Effects on Body and Organ Weights and Plasma Butyryl Cholinesterase Activity in Mice," Acta Pharmacol. Toxicol. 53:275-384.
- Kjellstrand, P., Kanje, M., Manson, L. et al., 1981. "Trichloroethylene: Effects on Body and Organ Weights in Mice, Rats, and Gerbils," *Toxicology* 21:105-115.
- Klaassen, C. D., Amdur, M. O., and Doull, J. (eds.) 1986. Casarett and Doull's Toxicology: The Basic Science of Poisons, 3rd ed., Macmillan Publishing Company, New York.
- Klaunig, J. E., Ruch, R. J., and Pereira, M. A. 1986. "Carcinogenicity of Chlorinated Methane and Ethane Compounds Administered in Drinking Water to Mice," *Environ. Health Perspect.* 69:89-95.
- Klein, M. 1963. "Susceptibility of Strain B6AF₁/J Hybrid Infant Mice to Tumorigenesis with 1,2-Benzanthracene, Deoxycholic Acid, and 3-Methylcholanthrene," *Cancer Res.* 23:1701–1707.
- Kline, R. D., Hays, V. W., and Cromwell, G. L. 1971. "Effects of Copper, Molybdenum and Sulfate on Performance, Hematology and Copper Stores of Pigs and Lambs," J. Animal Sci. 33:771-779.
- Kluwe, W. M., Haseman, J. K., Douglas, J. F., and Huff, J. E. 1982. "The Carcinogenicity of Dietary di(2ethylhexyl)phthalate (DEHP) in Fischer 344 Rats and B6C3F₁ Mice," J. Toxicol. Environ. Health. 10(4-5):797-815.
- Knobloch, K., Szedzikowski, S., and Slusarcyk-Zablobona, A. 1969. "Acute and Subacute Toxicity of Acenaphthene and Acenaphthylene," Med. Pracy 20:210-222. (Polish, Engl. Summary)
- Kochevar, I. E., Armstrong, R. B., Einbinder, J. , et al. 1982. "Coal Tar Phototoxicity: Active Compounds and Action Spectra," *Photochem. Photobiol.* 36:65-69.
- Kocsis, J. H., Shaw, E. D., Stokes, P. E., et al. 1993. "Neuropsychologic Effects of Lithium Discontinuation," J. Clin. Psychopharmacol. 13:268-276. (IPA Abstract)
- Komada, H., Kise, Y., Nakagawa, M., et al. 1990. "Effect of Dietary Molybdenum on Esophageal Carcinogenesis in Rats Induced by N-methyl-N-benzylnitrosamine," Cancer Res. 50:2418-2422.
- Kondakis, X. G., Makris, N., Leotsinidis, M., Prinou, M., and Papapetropoulos, T. 1989. "Possible Health Effects of High Manganese Concentration in Drinking Water," Arch. Environ. Health 44(3):175-178.

- Koppel, C., Arndt, I., Arndt, U., and Koeppe, P. 1985. "Acute Tetrachloroethylene Poisoning: Blood. Elimination Kinetics During Hyperventilation Therapy," J. Toxicol. Clin. Toxicol. 23:103-116.
- Kovalskii, V. V., Yarovaya, G. E., and Shmavonyan, D. M. 1961. "Changes in Purine Metabolism in Man and Animals in Various Molybdenum-Rich Biogeochemical Provinces," Z. Obsc. Biol. 22:179.
- Kozik, I. 1957. "Problems of Occupational Hygiene in the Use of Dichloroethane in the Aviation Industry," Gig. Tr. Prof. Zabol. 1:31-38.
- Krasavage, W. J., O'Donoghue, J. L., and Divincenzo, G. D. 1982. "Acetone," pp. 4720-4727, in *Patty's Industrial Hygiene and Toxicology*, 3rd rev. ed., vol. 2c, Clayton, G. D., and Clayton, F. E., eds., Wiley Interscience, New York.

Krasovskii, G. N., Vasulovich, L. Y., and Charie, O. G. 1979. "Experimental Study of Biological Effects of Lead and Aluminum Following Oral Administration," *Environ. Health Perspect.* 30:47-51.

- Kraup, T. 1970. "Oocyte Survival in the Mouse Ovary After Treatment with 9,10-Dimethyl-1,2benz(a)anthracene," J. Endocrinol. 46:483.
- Kriebel, D., Brain, J. D., Sprince, N. L., and Kazemi, H. 1988. "The Pulmonary Toxicity of Beryllium," Am. Rev. Respir Dis. 137:464-473.
- Kronevi, T., Wahlberg, J., and Holmberg, B. 1977. "Morphological Lesions in Guinea Pigs During Skin Exposure to 1,1,2-Trichloroethane," Acta Pharmacol. Toxicol. 41:298-305.

Kumai, M., Koizumi, A., Saito, K. et al., 1983. "A Nationwide Survey on Organic Solvent Components in Various Solvent Products: Part 2. Heterogeneous Products Such As Paints, Inks, and Adhesives," *Ind. Health* 21:185-197.

Kutegov, K. V., Pavlov, O. N., and Shvedov, V. P. 1968. "Technetium," Advan. Inorganic Chem. Radiochem. 11:1-90.

- Kyle, R. A., and Pease, G. L. 1965. "Hematologic Aspects of Arsenic Intoxication," New Eng. J. Med. 273(1):18-23.
- Kyyronen, P., Taskinen, H., Lindbohm, M. L., Hemminki, K., and Heinonen, O. P. 1989. "Spontaneous Abortions and Congenital Malformations Among Women Exposed to Tetrachloroethylene in Dry Cleaning," J. Epidemiol. Comm. Health 43:346-351.

Lacassagne, A., Buu-Hoi, N. P., Zajdela, F., et al. 1963. "Activite Cancerogene d'Hydrocarbures Aromatiques Polycycliques a Noyau Fluoranthene," Un. Int. Cancer Acta 19:490-496.

Lagerkvist, B. E. A., Linderholm, H., and Nordberg, G. F. 1986. "Vasospastic Tendency and Raynaud's Phenomenon in Smelter Workers Exposed to Arsenic," *Environ. Res.* **39**:465–474.

- Lam, H. F., Conner, M. W., Rogers, A. E., et al. 1985. "Functional and Morphological Changes in the Lungs of Guinea Pigs Exposed to Freshly Generated Ultrafine Zinc Oxide," *Toxicol. Appl. Pharmacol.* 78:29–38.
- Lambert, J., Schepens, P., Janssens, J., et al. 1986. "Skin Lesions as a Sign of Subacute Pentachlorophenol Intoxication," Acta Derm. Venreol. (Stockh) 66:170-172.
- Land, P. D., Owen, E. L., and Linde, H. W. 1981. "Morphological Changes in Mouse Spermatozoa After Exposure to Inhalational Anesthetics During Early Spermatogenesis," *Anesthesiology* 54:53-56.



- Landau, E. D., Thompson, D. J., Feldman, R. G., Goble, G. J., and Dixon, W. J. 1977. Selected Noncarcinogenic Effects of Industrial Exposure to Inorganic Arsenic, EPA 659/6-77-018, U.S. Environmental Protection Agency, Washington, DC.
- Landis, J. R., and Flegal, K. M. 1988. "A Generalized Mantel-Haenszel Analysis of the Regression of Blood Pressure on Blood Lead Using NHANES II Data," *Environ. Health Prospect.* 78:35-41.
- Landrigan, P. J., Stein, G. F., Kominski, J. R., et al. 1987. "Common-Source Community and Industrial Exposure to Trichloroethylene," Arch. Environ. Health 43:327-332.
- Langard, S. 1994. "Nickel-related Cancer in Welders," Science of the Total Environ. 148:303-309.
- Langard, S. 1983. "The Carcinogenicity of Chromium Compounds in Man and Animals," pp. 13-30, in Chromium: Metabolism and Toxicity, Burrows, C., ed., CRC Press, Inc., Boca Raton, FL.
- Langard, S. 1982. "Absorption, Transport and Excretion of Chromium in Animals," pp. 149-169, in Biological and Environmental Aspects of Chromium, Langard, S., ed., Elsevier Biomedical Press, Amsterdam.
- Langauer-Lewowicka, H., Kurzbauer, H., Byczkowska, Z., et al. 1983. "Vinyl Chloride Disease-Neurological Disturbances," Int. Arch. Occup. Environ. Health 52:151-157. (CA 99: 145391)
- Larsen, J. J., Lykkegaard, M., and Ladefoged, O. 1991. "Infertility in Rats Induced by 2,4-Hexanedione in Combination with Acetone," *Pharmacol. Toxicol.* 69:43-46.
- Larsen, R. V. 1976. "The Placental Transfer and Teratology of Pentachlorophenol in Rats" [abstract], Diss. Abstr. Int. B37:1184–1185.
- Larsen, R. V., Born, G. S., Kessler, W. V., et al. 1975. "Placental Transfer and Teratology of Pentachlorophenol in Rats," *Environ. Lett.* 10:121-128.
- LaVoie, E. J., Braley, J., Rice, J. E., and Rivenson, A. 1987. "Tumorigenic Activity for Non-alternant Polynuclear Aromatic Hydrocarbons in Newborn Mice," *Cancer Lett.* 34:15-20.
- LaVoie, E. J., Hecht, S. S., Bedenko, V., and Hoffmann, D. 1982a. "Identification of the Mutagenic Metabolites of Fluoranthene, 2-Methylfluoranthene, and 3-Methylfluoranthene," Carcinogenesis 3:841-846.
- LaVoie, E. J., Amin, S., Hecht, S. S., et al. 1982b. "Tumor Initiating Activity of Dihydrodiols of Benzo(b)fluoranthene, Benzo(j)fluoranthene, and Benzo(k)fluoranthene," Carcinogenesis 3:49-52.
- LaVoie, E. J., Tulley-Freiler, L., Bedenko, V., and Hoffmann, D. 1981. "Mutagenicity, Tumor-Initiating Activity, and Metabolism of Methylphenanthrenes," *Cancer Res.* 41:3441-3447.
- LaVoie, E. J., Hecht, S. S., Amin, S. S., et al. 1980. "Identification of Mutagenic Dihydrodiols as Metabolites of Benzo(j)fluoranthene and Benzo(k)fluoranthene," *Cancer Res.* 40:4528-4532.
- LaVoie, E. J., Bedenko, E. V., Hirota, N., et al. 1979. "A Comparison of the Mutagenicity, Tumor-Initiating Activity and Complete Carcinogenicity of Polynuclear Aromatic Hydrocarbons," pp. 705-721, in *Polynuclear Aromatic Hydrocarbons*, Jones, P.W., and Leber, P., eds., Ann Arbor Science Publishers, Ann Arbor, MI.
- Leber, A. P., and Benya, T. J. 1994. "Halogenated Benzene," in *Patty's Industrial Hygiene and Toxicology*, 4th ed, Vol. II, Part B, Clayton, G. D., and Clayton, F. E., eds.
- Lecyk, M. 1980. "Toxicity of Cupric Sulfate in Mice Embryonic Development," Zool. Pol. 28:101-105.

- Lee, A. M., and Fraumeni, J. F., Jr. 1969. "Arsenic and Respiratory Cancer in Man—An Occupational. Study," J. Natl. Cancer Inst. 42:1045-1052.
- Lee, C. C., Bhandari, J. C., Winston, J. M., et al. 1977. "Inhalation Toxicity of Vinyl Chloride and Vinylidine Chloride," *Environ. Health Perspect.* 21:25-32.
- Lee, C. C., Hong, C. B., Ellis, H. V., et al. 1985. "Subchronic and Chronic Toxicity Studies of 2,4-Dinitrotoluene. Part II. CD Rats," J. Am. Coll. Toxicol. 4:243-256.
- Lee, C. C., Bhandari, J. C., Winston, J. M., et al. 1978a. "Carcinogenicity of Vinyl Chloride and Vinylidine Chloride, J. Toxicol. Environ. Health 4:15-30.
- Lee, C. C., Ellis, H. V., Kowalski, J. J., et al. 1978b. Mammalian Toxicity of Munitions Compounds. Phase II: Effects of Multiple Doses. Part II: 2,4-Dinitrotoluene, DAMD-17-74-C-4073, U.S. Army Medical Bioengineering Research and Development Laboratory, Kansas City, MO.
- Legraverend C., Guenthner, T. M., and Nebert, D. W. 1984. "Importance of the Route of Administration for Genetic Differences in Benzo(a)pyrene-Induced in utero Toxicity and Teratogenicity," *Teratology* 29:35-47.
- Leonard, A., and Lauwerys, R. R. 1980. "Carcinogenicity and Mutagenicity of Chromium," Mutat. Res. 76:227-239.

Leonard, T. B., Graichen, M. E., and Popp, J. A. 1987. "Dinitrotoluene Isomer-Specific Hepatocarcinogenesis in F-344 Rats," J. Nat. Cancer Inst. 79:1313-319.

Levine, R. J., Andjelkovich, D. A., Kersteter, S. L., et al. 1986. "Heart Disease in Workers Exposed to Dinitrotoluene," J. Occup. Med. 28:811-816.

Lewis, C. 1959. "The Biological Effects of Vanadium. II. The Signs and Symptoms of Occupational Vanadium Exposure," A.M.A. Arch. Ind. Health 19:497-503.

- Lide, D.R., ed. 1991. CRC Handbook of Chemistry and Physics, 1991-1992, 72nd ed., pp. 3-96, Boca Raton, FL.
- Liebman, K.C., and Ortiz E. 1977. "Metabolism of Halogenated Ethylenes, *Environ. Health Perspec.* 21:91-97.
- Lindberg, E., and Hedenstierna, G. 1983. "Chrome Plating: Symptoms, Findings in the Upper Airways, and Effects on Lung Function," Arch. Environ. Health 38:367-374.
- Lloyd, T. B. 1984. "Zinc Compounds," pp. 851-863, in Kirk-Othmer Encyclopedia of Chemical Technology, 3rd ed., Mark, H. F., Othmer, D. F., Overberger, C. G., and Seaborg, G. T., eds., John Wiley & Sons, New York.
- Lloyd, M. H., Gauld, S., Copland, L., et al. 1984. "Epidemiological Study of the Lung Function of Workers at a Factory Manufacturing Polyvinyl Chloride." Br. J. Ind. Med. 41:328-333.
- Lloyd, W. E., Hill, H. T., and Meerdink, G. L. 1976. "Observations of a Case of Molybdenosis-Copper Deficiency in a South Dakota Dairy Herd," pp. 85-95, in *Molybdenum in the Environment*, Chappell, W., and Peterson, K. K., eds., Marcel Dekker, Inc., New York.
- Love, A. H. G. 1983. "Chromium-Biological and Analytical Considerations," p. 1, in Chromium: Metabolism and Toxicity, Burrows, D., ed., CRC Press, Inc., Boca Raton, FL.

- Low, L. K., Meeks, J. R., Norris, K. J., Mehlman, M. A, and Mackerer, C. R. 1989. "Pharmacokinetics and Metabolism of Benzene in Zymbal Gland and Other Key Target Tissues after Oral Administration in Rats," *Environ. Health Perspect.* 82:215-222.
- Ludersdorf, R., Fuchs, A., Mayer, P., et al. 1987. "Biological Assessment of Exposure to Antimony and Lead in the Glass-Producing Industry," Int. Arch. Occup. Environ. Health 59:469-474.
- Lundgren, K. D. 1954. "Damage in the Respiratory Organs of Workers at a Smeltery," Nord. Hyg. Tidskr. 3:66-82.
- Luo, X. M., Wei, H. J., and Yang, S. P. 1983. "Inhibitory Effects of Molybdenum on Esophageal and Forestomach, Carcinogenesis in Rats," J. Natl. Cancer Inst. 71:75-80.
- Lynge, E., and Thygesen, L. 1990. "Primary Liver Cancer Among Women in Laundry and Dry-Cleaning Work in Denmark," Scand J. Work Environ. Health 16:108-112.
- Lyster, W. R. 1977. "Arsenic and Sex Ratio in Man," Med. J. Austral. 2:442.
- Mack, R. B. 1989. "A Hard Day's Knight, Zinc Phosphide Poisoning," N.C. Med. J. 50:17-18.
- MacKenzie, K. M., and Angevine, D. M. 1981. "Infertility in Mice Exposed in utero to Benzo(a)pyrene," Biol. Reprod. 24:183-191.
- 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," *Arch. Ind. Health* 18:232-234.
- Mahaffey, K. R., Capar, S. G., Gladen, B. C., and Fowler, B. A. 1981. "Concurrent Exposure to Lead, Cadmium, and Arsenic: Effects on Toxicity and Tissue Metal Concentrations in the Rat," J. Lab. Clin. Med. 98:463-481.
- Maita, K., Hirano, M., Mitsumori, K., et al. 1981. "Subacute Toxicity with Zinc Sulfate in Mice and Rats," J. Pest. Sci. 6:327-336.
- Makarov, I. A. 1984. "Sexual Disorders in Male Workers Occupationally Exposed to Methyl-methacrylate and Vinyl Chloride," *Gig. Tr. Prof. Zabol.* 6:19-23. (Trans. 1-11)
- Makarov, I. A., Solovyeva, M. S., and Gnelitsky, G. I. 1984. "On Sexual Disorders in Women Chronically Exposed to Methyl-methacrylate and Vinyl Chloride," *Gig. Tr. Prof. Zabol.* 3:22-27. (Trans.)
- Maltoni, C. 1977. "Recent Findings on the Carcinogenicity of Chlorinated Olefins," *Environ. Health* Perspect. 21:1-5.
- Maltoni, C., Conti, B., Cotti, G. 1989. "Benzene: A Multi-Potential Carcinogen. Results of Long-Term Bioassays Performed at the Bologna Institute of Oncology," Am. J. Ind. Med. 4:589-630.
- Maltoni, C., Lefemine, G., Cotti, G., et al. 1988. "Long-Term Carcinogenicity Bioassasy on Trichloroethylene Administered by Inhalation to Sprague-Dawley Rats and Swiss and B6C3F₁ Mice," Ann. N.Y. Acad. Sci. 534:316-342.
- Maltoni, C., Lefemine, G., and Cotti, G. 1986. "Experimental Research on Trichloroethylene Carcinogenesis," p. 393, Archives of Industrial Carcinogenesis Series, Vol. V, Maltoni, C., and Mehlman, M.A., eds., Princeton Scientific Publishing Co., Princeton, NJ.
- Maltoni, C., Lefemine, G., Chieco, P., et al. 1985. "Experimental Research on Vinylidine Carcinogenesis," in Archives of Research on Industrial Carcinogenesis, Vol. 3. Maltoni, C., and Mehlman, M., eds., Princeton Scientific Publishers, Princeton, NJ.

- Maltoni, C., Cotti, G., and Chieco, P. 1984. "Chronic Toxicity and Carcinogenicity Bioassays of Vinylidine_ Chloride," Acta Oncol. 5:91-145.
- Maltoni, C., Lefemine, G., Cilibert, A., et al. 1981. "Carcinogenicity Bioassays of Vinyl Chloride Monomer: A Model Risk Assessment on an Experimental Basis," *Environ. Health Perspect.* 41:3–29.
- Maltoni, C., Lefemine, G., Ciliberti, A., et al. 1980. "Vinyl Chloride Carcinogenicity Bioassays (BT project) as an Experimental Model for Risk Identification and Assessment in Environmental and Occupational Carcinogenesis," pp. 11–112, Epidemiol. Anim. Epidemiol. Hum.: Cas Chlorure Vinyle Momomere (Reun. Club Cancerog. Chim.), 20th Meeting, 1979, Publ. Essent, Paris, France.
- Mancuso, T. F. 1980. "Mortality Study of Beryllium Industry Workers' Occupational Lung Cancer," Environ. Res. 21:48-55.
- Mancuso, T. F. 1975. International Conference on Heavy Metals in the Environment, Toronto, Ontario, Canada.
- Mancuso, T. F. 1979. "Occupational Lung Cancer Among Beryllium Workers in Dusts and Disease," in Proceedings of the Conference on Occupational Exposure to Fibrous and Particulate Dust and Their Extension into the Environment, Lemen, R., and Dement, J., eds., Pathrotox Publishers, Inc.
- Mancuso, T. F. 1970. "Relation of Duration of Employment and Prior Illness to Respiratory Cancer Among Beryllium Workers," *Environ. Res.* 3:251-275.
- Mancuso, T. F., and Hueper, W. C. 1951. "Occupational Cancer and Other Health Hazards in a Chromate Plant: A Medical Appraisal. I. Lung Cancer in Chromate Workers," *Ind. Med. Surg.* 20:358-363.
- Manzo, L., Scelsi, R., Moglia, A., et al. 1983. "Long-Term Toxicity of Thallium in the Rat," pp. 401-405, in Proceed. 2nd Int. Conf., Chem. Toxicol. Clin. Chem. Mat.
- Marathe, M. R., and Thomas, G. P. 1986. "Embryotoxicity and Teratogenicity of Lithium Carbonate in Wistar Rats," *Toxicol. Lett.* 34:115-120.

Marcus, S. 1980. "Lithium," Clin. Toxicol. Rev. 2:1-2.

- Marks, T. A., Ledoux, T. A., and Moore, J. A. 1982. "Teratogenicity of a Commercial Xylene Mixture in the Mouse," J. Toxicol. Environ. Health 9:97-106.
- Marmo, E., Matera, M. G., Acampora, R., et al. 1987. "Prenatal and Postnatal Metal Exposure: Effect on Vasomotor Reactivity Development of Pups," Cur. Ther, Res. 42:823-838.
- Marsh, D. O., Clarkson, T. W., Cox, C., Amin-Zaki, L., and Al-Trkirti, S. 1987. "Fetal Methylmercury Poisoning: Relationship Between Concentration in a Single Strand of Maternal Hair and Child Effects," Arch. Neurol. 1017-1022.
- Mast, T. J., Evanoff, J. J., Rommereim, R. L., et al. 1988. Inhalation Developmental Toxicology Studies: Teratology Study of Acetone in Mice and Rats, National Toxicology Program, Contract DE-AC06-76RLO 1830, Pacific Northwest Laboratory, Battelle Memorial Institute.
- Mathesson, D. S., Clarkson, T. W., and Gelfand, E. 1980. "Mercury Toxicity (Acrodynia) Induced by Long-Term Injection of Gamma Globulin," J. Pediatr. 97:153-155.
- Matsumoto, N., Iijima, S., and Katsunuma, H. 1976. "Placental Transfer of Chromic Chloride and Its Teratogenic Potential in Embryonic Mice," J. Toxicol. Sci. 2:1-13.
- Mattison, D. R., and Thorgeirsson, S. S. 1977. "Ovarian Metabolism of Polycyclic Aromatic Hydrocarbons and Associated Ototoxicity in the Mouse," *Gynecol. Invest.* 8:11.

- Matuk, Y., Gosh, M., and McCulloch, C. 1981. "Distribution of Silver in the Eyes and Plasma Proteins of the Albino Rat," Can. J. Ophthalmol. 16:145-150.
- Mazza, V. and Brancaccio, A. 1967. "Characteristics of the Formed Elements of the Blood and Bone Marrow in Experimental Trichloroethylene Intoxication," *Folia Med.* 50:318-324.
- McCall, S. N., Jurgens, P., and Ivanetich, K. M. 1983. "Hepatic Microsomal Metabolism of Dichloroethanes," *Biochem. Pharmacol.* 3:207-213.
- McCauley, P.T., Robinson, M., Condie, L.W., and Parvell, M., n.d. The Effects of Subacute and Subchronic Oral Exposure to cis-1,2-Dichloroethylene in Rats, Health Effects Research Laboratory, EPA, Cincinnati, OH.
- McCormick, D. L., Burns, F. J., and Albert, R. E. 1981. "Inhibition of Benzo(a)pyrene-Induced Mammary Carcinogenesis by Retinyl Acetate," J. Natl. Cancer Inst. 66:559-564.
- McGee, L. C., McCausland, A., Plume, C. A., et al. 1942. "Metabolic Disturbances in Workers Exposed to Dinitrotoluenes," *Am. J. Digest. Diseases* 9:329-331.
- McMichael, A. J., Baghurst, P. A., Wigg, N. R., et al. 1988. "Port Pirie Cohort Study: Environmental Exposure to Lead and Children's Ability at the Age of Four Years," N. Engl. J. Med. 319:468-475.
- Meehan, W. R. and Smyth. L. E. 1967. "Occurrence of Beryllium as a Trace Element in Environmental Materials," *Environ. Sci. Technol.* 1:839-844.
- Menon, J. A. 1958. "Tropical Hazards Associated with the Use of Pentachlorophenol," Br. Med. J. 1:1156-1158.
- Menzie, C. A., Burmaster, D. E., Freeman, J. S., and Callahan, C. A. 1992. "Assessment of Methods for Estimating Ecological Risk in the Terrestrial Component: A Case Study of the Baird and McGuire Superfund Site in Holbrook, Massachusetts," *Environ. Toxicol. Chem.* 11:245-260.
- Mestitzova, M. 1967. "On Reproduction Studies on the Occurrence of Cataracts in Rats After Long-Term Feeding of the Insecticide Heptachlor," *Experientia* 23:42-43.
- Metreveli, D. M., Babayan, E. A., and Melkonyan, A. N. 1985. "On the Effect of Molybdenum on the Health of Female Workers of the Kajaran Integrated Copper-Molybdenum Mill USSR," Soobshch. Akad. Nauk Gruz. SSR 119(2):385-388 (Abst).
- Metreveli, D. M., and Danelizu, G. S. 1984. "Effects of Molybdenum on the Internal Genitals," Soobshch. Adad. Nauk. Gruz. SSR 116:141-144. (Abstract)
- Milby, T. H. 1968. "Chronic Trichloroethylene Intoxication," J. Occup. Med. 10:252-254.
- Milkkov, L. B., Aldjreva, M. V., Popova, T. B., et al. 1973. "Health Status of Workers Exposed to Effect of Phthalate Plasticizers in the Production of Artificial Leather and Films (on the Basis of PVC Resins)," translation of *Gig. Tr. Prof Zabol.* 13:14–17, 1969. (NTIS PB221973-T, 5 pp.)
- Mitoma, C., Tyson, C. A., and Riccio, E. S. 1985a. Investigation of the Species Sensitivity and Mechanism of Carcinogenicity of Halogenated Hydrocarbons, EPA/OTS, Document No. 40-842-8424225, Final Report, EPA Contract No. 68-01-5079.
- Mitoma, C., Steeger, T., Jackson, S. E., et al. 1985b. "Metabolic Disposition Study of Chlorinated Hydrocarbons in Rats and Mice," Drug Chem. Toxicol. 8:183-194.

- Mizuta, N., Mizuta, M., Ita, F., Ito, T., Uchida, H., Watanabe, Y., Akama, H., Murakami, T., Hayashi, F., Nakamura, K., Yamaguchi, T., Mizuia, W., Oishi, S., and Matsumura, H. 1956. "An Outbreak of Acute Arsenic Poisoning Caused by Arsenic-Contaminated Soy Sauce (Shoye). A Clinical Report of 220 Cases," Bull. Yamaguchi Med. Sch. 4:131-150.
- Modica, R., Fiume, M., Guaitani, A., and Bartosek, I. 1983. "Comparative Kinetics of Benz(a)anthracene, Chrysene and Triphenylene in Rats After Oral Administration. I. Study with Single Compounds," *Toxicol. Lett.* 18:103-109.
- Moeller, W. 1973. "Chronic Carbon Tetrachloride Poisoning from an Ophthalmological Viewpoint," Z. Ges. Hyg. 19:127-133.
- Mogilevskaya, O. Y. 1963. "Experimental Studies on the Effect on the Organism of Rare, Dispersed and Other Metals and Their Compounds Used in Industry; Molybdenum," pp. 12–28, in *Toxicology of Rare Metals*, Z. I. Izrael'son. (Translation from *Gosudartsvennoe Isdatel'stvo Meditsinskoi Literatury*, Moscow)
- Monson, R. R., Peters, J. M., and Johnson, M. N. 1974. "Proportional Mortality Among Vinyl-Chloride Workers," Lancet 2:397-398.
- Monster, A. C., Beersman, G., Steenweg, H. 1979. "Kinetics of Tetrachloroethylene in Volunteers: Influence of Exposure Concentration and Work Load," Int. Arch Occup. Environ. Health 53:331-336.
- Moore, E. W. 1952. "Physiological Effects of the Consumption of Saline Drinking Water," in A Progress Report to the 16th Meeting of Subcommittee on Water Supply of the Committee on Sanitary Engineering and Environment, National Academy of Sciences, Washington, DC.
- Moore, R. 1978. "Bleeding Gastric Erosion After Oral Zinc Sulfate," Br. Med. J. 1:754.
- Morgan, A., Black, A., and Belcher, D. R. 1970. "The Excretion in Breath of Some Aliphatic Halogenated Hydrocarbons Following Administration by Inhalation," Ann. Occup. Hyg. 13:219-233.
- Morgott, D. A. 1993. "Acetone," in *Patty's Industrial Hygiene and Toxicology*, 4th ed., vol. 2, Part A, Clayton, G. D., and Clayton, F. E., eds., John Wiley & Sons, Inc., New York.
- Morita, M., and Ohi, G. 1975. "Para-dichlorobenzene in Human Tissue and Atmosphere in Tokyo Metropolitan Area," *Environ. Pollut.* 8:269-274.
- Morita, M., Minur, S., Ohi, G., et al. 1975. "A Systematic Determination of Chlorinated Benzenes in Human Adipose Issues," *Environ. Pollut.* 9:175-179.
- Morris, J. S., Schmid, M., Newman, S., Scheuer, P. J., and Sherlock, S. 1974. "Arsenic and Noncirrhotic Portal Hypertension," *Gastroenterology* 64:86-94.
- Moskalev, Y. I., Buldakov, L. A., Zhuravleva, A. K., et al. 1979. Toxicological and Radiobiology of Neptunium-237, ORNL-tr-4936, Moscow Atomizdat Publishers.
- Mueller-Hoecker, J., Meyer, U., Wiebecke, B., et al. 1988. "Copper Storage Disease of the Liver and Chronic Dietary Copper Intoxication in Two Further German Infants Mimicking Indian Childhood Cirrhosis," Pathol. Red. Pract. 183:39-45.
- Muller, E. 1968. "Carcinogenic Substances in Water and Soils. XX. Studies on the Carcinogenic Properties of 1,12-Benzoperylene," Arch. Hyg. 152:23-26.
- Munson, A. E., Sanders, V. M., Douglas, K. A., et al. 1982a. "In Vivo Assessment of Immunotoxicity," Environ. Health Perspect. 43:41-53.



- Munson, A. E., Sain, L. E., Sanders, V. M., et al. 1982. "Toxicology of Organic Drinking Water Contaminants: Trichloromethane, Bromodichloromethane, Dibromochloromethane, and Tribromomethane," *Environ. Health Perspect.* 46:117–126.
- Murray, F. J., Nitschke, K. D., Rampy, L. W., and Schwetz, B. A. 1979a. "Embryotoxicity and Fetotoxicity of Inhaled or Ingested Vinylidine Chloride in Rats and Rabbits," *Toxicol. Appl. Pharmacol.* 49:189-202.
- Murray, A. E., Schwetz, B. A., McBride, J. G., and Staples, R. E. 1979b. "Toxicity of Inhaled Chloroform in Pregnant Mice and Their Offspring," *Toxicol. Appl. Pharmacol.* 50:515-522.
- Nagymjtenyi, L., Selypes, A., and Berencsi, G. 1985. "Chromosomal Aberrations and Fetotoxic Effects of Atmospheric Arsenic Exposure in Mice," J. Appl. Toxicol. 5:61-63.
- NAS (National Academy of Sciences) 1977. Drinking Water and Health, pp. 425-428.
- NAS 1975. Nickel, pp. 4-5, 17, Washington, DC.
- Nawrot, P. S., and Staples, R.E. 1979. "Embryo-Fetal Toxicity and Teratogenicity of Benzene and Toluene in the Mouse," *Teratology* 19:41A.
- NCA (National Coffee Association) 1983. "24-Month Chronic Toxicity and Oncogenicity Study of Methylene Chloride in Mice," Final Report, prepared by Hazleton Laboratories America, Inc., Vienna, VA. (Unpublished)
- NCI (National Cancer Institute) 1978a. Bioassay of 1,1,2-Trichloroethane for Possible Carcinogenicity, CAS No. 79-00-5. DEWH Publ. No. (NIH) 78-1324, Carcinogenesis Tech. Report Series No. 74, NCI-CG-TR-74, U.S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, Bethesda, MD.
- NCI 1978b. Carcinogenesis Bioassay of 1,1-Dichloroethane, NCI Carcinogenesis Technical Report Series Number 66, NCI-CG-TR-66, DHEW Publication No. (NIH) 78-1316.
- NCI 1978c. Bioassay of 2,4-Dinitrotoluene for Possible Carcinogenicity, CAS No. 121-14-2, Carcinogenesis Tech. Rep. Series No. 54, Publ. No. 78-1360, U.S. Department of Health, Education, and Welfare, Public Health Service, National Institutes of Health, Washington, DC.
- NCI 1977a. Bioassay of Heptachlor for Possible Carcinogenicity, CAS No. 76-44-8, NCI-CG-TR-9, DEWH Publ. No. 77-809.
- NCI 1977b. Bioassay of Tetrachloroethylene for Possible Carcinogenicity, NCI-TR-13, U.S. Department of Health, Education, and Welfare, Bethesda, MD.
- NCI 1976a. Report on Carcinogenesis Bioassay of Chloroform, Carcinogenesis Program, Division of Cancer Cause and Prevention, Bethesda, MD.
- NCI 1976b. Report on Carcinogenesis Bioassay of Trichloroethylene, National Cancer Institute Carcinogenesis Technical Report Series, No. 2, NCI-CG-TR-2.
- NCI 1976c. Report on Carcinogenesis Bioassay of Chloroform, NTIS-PB-264018, National Cancer Institute, Washington, DC.
- NCI 1976d. Carcinogenesis Bioassay of Trichloroethylene, CAS No. 79-01-6, NCI-CG-TR-2, U.S. Department of Health, Education, and Welfare, National Institutes of Public Health, 76-802.

- NCRP (National Council on Radiation Protection and Measurements) 1989. Screening Techniques for Determining Compliance with Environmental Standards: Releases of Radionuclides to the Atmosphere, NCRP Commentary No. 3, Bethesda, MD.
- NCRP 1987. Recommendations on Limits for Exposure to Ionizing Radiation, NCRP Report No. 91, National Council on Radiation Protection and Measurements, Bethesda, MD.
- Neal, J., and Rigdon, R. H. 1967. "Gastric Tumors in Mice Fed Benzo(a)pyrene: A Quantitative Study," Tex. Rep. Biol. Med. 25:553.
- Nelson, B. K., Taylor, B. J., Setzer, J. V., and Hornung, R. W. 1980. "Behavioral Teratology of Perchloroethylene in Rats," J. Environ. Pathol. Toxicol. 3:233-250.

Nemery, B. 1990. "Metal Toxicity and the Respiratory Tract," Eur. Respir. J. 3:202-219.

- Nettesheim, P., Hanna, M. G., Jr., Doherty, D. G., Newell, R. F., and Hellman, A. 1971. "Effect of Calcium Chromate Dust, Influenza Virus, and 100 R Whole-Body X-Radiation on Lung Tumor Incidence in Mice," J. Natl. Cancer Inst. 47:1129-1138.
- NIOSH (National Institute for Occupational Safety and Health) 1976. Criteria for a Recommended Standard. Occupational Exposure to Ethylene Dichloride, HEW Publication No. (NIOSH) 76–139.
- Nishimura, M. 1966. "Clinical and Experimental Studies on Acute Beryllium Disease," Nagoya J. Med. Sci. 18:17-44.
- Nitschke, K. D., Bured, J. D., Bell, T., J., et al. 1988a. "Methylene Chloride: A 2-Year Inhalation Toxicity and Oncogenicity Study in Rats," *Fund. Appl. Toxicol.* 11:48-59.
- Nitschke, K. D., Smith, F. A., Quast, J. F., et al. 1983. "A Three-Generation Rat Reproductive Toxicity Study of Vinylidine Chloride in the Drinking Water," *Fund. Appl. Toxicol.* 3:75-79.
- Nomiyama, K., Nomiyama, H., and Arai, H. 1986. "Reevaluation of Subchronic Toxicity of Trichloroethylene," *Toxicol. Lett.* 31(Suppl.): 225.
- Nordberg, G. F., Kjellstrom, T., and Nordberg, M. 1985. "Kinetics and Metabolism," pp. 103-178, in Cadmium and Health: A Toxicological and Epidemiological Appraisal, Vol. 1., Exposure, Dose, and Metabolism, Friberg, L., Elinder, C. G., Kjellstrom, T., and Nordberg, G. F., eds., CRC Press, Boca Raton, FL.
- Nordquist, M., Thakker, D. R., Vyas, K. P., et al. 1981. "Metabolism of Chrysene and Phenanthrene to Bay-Region Diol Epoxides by Rat Liver Enzymes," *Mol. Pharmacol.* 19:168–178.
- Nordström, S., Beckman, L., and Nordenson, I. 1978a. "Occupational and Environmental Risks in and Around a Smelter in Northern Sweden. I. Variations in Birth Weight," *Hereditas* 88:43-46.
- Nordström, S., Beckman, L., and Nordenson, I. 1978b. "Occupational and Environmental Risks in and Around a Smelter in Northern Sweden. III. Frequencies of Spontaneous Abortions," *Hereditas* 88:51-54.
- Nouchi, T., Miura, H., Kanayama, M., et al. 1984. "Fatal Intoxification by 1,2-Dichloroethane—A Case Report," Int. Arch. Occup. Environ. Health 54:111-113.
- NRC (National Research Council) 1989. Recommended Dietary Allowances, 10th rev. ed., pp. 230-235, Food and Nutrition Board, National Research Council, National Academy Press, Washington, DC.
- NRC 1989. Zinc, University Park Press, Baltimore, MD.

- NRCC (National Research Council of Canada) 1980. Effects of Vanadium in the Canadian Environment, Report No. 18132, National Research Council of Canada, Ottawa.
- NTP (National Toxicology Program) 1995. NTP Management Status Report, 1995, National Toxicology Program, Research Triangle Park, NC.
- NTP 1992. Toxicity Studies of Ethylbenzene in F344/N Rats and B6C3F₁ Mice (Inhalation Studies), NTP TOX 10 (NIH Publ. No. 92-3129), National Toxicology Program, Research Triangle Park, NC.
- NTP 1991. NTP Report on the Toxicity Studies of 1,2-Dichloroethane in F344/N Rats, Sprague Dawley Rats, Osborne-Mendel Rats and B6C3F₁ Mice (Drinking Water and Gavage Studies), U.S. Department of Health and Human Services, Washington, DC.
- NTP 1988. Toxicology and Carcinogenesis Studies of Trichloroethylene (CAS No. 79-01-6) in Four Strains of Rats (ACI, August, Marshall, Osborne-Mendel) (Gavage Studies), NTP TR 273, U.S. Department of Health and Human Services, National Institutes of Health, Bethesda, MD.
- NTP 1987a. Toxicology and Carcinogenesis Studies of 1,4-Dichlorobenzene (CAS No. 106-46-7) in F344/N Rats and B6C3F₁ Mice (Gavage Studies), NTP TR 319, NIH Publication No. 87-2575, Research Triangle Park, NC.
- NTP 1987b. NTP Report on the Toxicology and Carcinogenesis Studies of Bromodichloromethane on F344/N Rats and B6C3F₁ Mice (Gavage Studies), NTP TR 321, U.S. Dept. Health and Human Services, Public Health Service, National Institute of Health.
- NTP 1986a. Toxicology and Carcinogenesis Studies of Dichloromethane (Methylene Chloride) (CAS No. 75-09-2) in F344/N Rats and B6C3F₁ Mice (Inhalation Studies), U.S. Department of Health and Human Services, Research Triangle Park, NC,
- NTP 1986b. NTP Technical Report on the Toxicology and Carcinogenesis Tetrachloroethylene (Perchloroethylene) in F344/N Rats and B6C3F₁ Mice (Inhalation Studies), NTP TR 311, National Toxicology Program, U.S. Dept. of Health and Human Services, Research Triangle Park, NC.
- NTP 1986c. Toxicology and Carcinogenesis Studies of Trichloroethylene (CAS No. 79-01-6) in F344 Rats and B6C3F₁ Mice, NTP TR 243, U.S. Department of Health and Human Services, National Institutes of Health, Bethesda, MD.
- NTP 1986d. Toxicology and Carcinogenesis Studies of Xylenes (Mixed) (60% m-Xylene, 14% p-Xylene, 9% o-Xylene, and 17% Ethylbenzene) (CAS No. 1330-0-7), in F344/N Rats and B6C3F₁ Mice (Gavage Studies), NTP Technical Report Series No. 327.
- NTP 1982a. Carcinogenesis Bioassay of Trichloroethylene in K344 Rats and B6C3F₁ Mice. CAS No. 79-01-6, NIH Publ. 82-1799, U.S. Department of Health and Human Services, National Institutes of Health, Bethesda, MD.
- NTP 1982b. Carcinogenesis Bioassay of Vinylidine Chloride (CAS No. 75-35-4) in F344 Rats and B6C3F₁ Mice (Gavage Study), NTP Tech. Rep. Series No. 228. NTP-80-82; NIH Publ. No. 82-1784.
- NTP 1980a. Unpublished Subchronic Toxicity Study: Naphthalene (C52904), Fischer 344 Rats, prepared by Battelle Columbus Laboratories.
- NTP 1980b. Unpublished Subchronic Toxicity Study: Naphthalene (C52904), B6C3F₁ Mice, prepared by Battelle Columbus Laboratories.
- O'Bryan, T. R., and Ross, R. J. 1988. "Chemical Scoring System for Hazard and Exposure Identification," J. Toxicol. Environ. Health 1:119-134.

Ohtsuki, T., Sato, K., Koizumi, A., Kumai, M., and Ikeda, M. 1983. "Limited Capacity of Humans to. Metabolize Tetrachloroethylene," Int. Arch. Occup. Environ. Health 51:381-390.

- Olcott, C. T. 1948. "Experimental Argyrosis. IV. Morphologic Changes in the Experimental Animal," Am. J. Path. 24:813-833.
- Olcott, C. T. 1950. "Experimental Argyrosis. V. Hypertrophy of the Left Ventricle of the Heart," Archives of Pathol. 49:138-149.
- O'Malley, M. A., Carpenter, A. V., Sweeney, M. H., et al. 1990. "Chloracne Associated with Employment in the Production of Pentachlorophenol," Am. J. Ind. Med. 17:411-421.
- ORNL (Oak Ridge National Laboratory) 1996. Comparison of Trichloroethene (TCE) Concentrations at Area of Concern (AOC) 204 to Human Health Risk-Based Concentrations, Oak Ridge, TN.
- OSHA (Occupational Safety and Health Administration) 1989. "Toxic and Hazardous Substances," Table Z-1, 29 CFR XVII; sect. 1910.1000.
- Ott, M. G., Skory, L. K., Holder, B. B., et al. 1983. "Health Surveillance of Employees Occupationally Exposed to Methylene Chloride. I. Mortality," Scand. J. Work Environ. Health 9 (Suppl. 1):8-16.
- Ott, M. G., Townsend, J. C., Fishbeck, W. A., and Langner, R.A. 1978. "Mortality Among Workers Occupationally Exposed to Benzene," Arch. Environ. Health. 33:3-10.
- Ott, M. G., Fishbeck, W. A., Townsend, J. C., and Schneider, E. J. 1976. "A Health Study of Employees Exposed to Vinylidine Chloride," J. Occup. Med. 18:735-738.
- Ottolenghi, A. D., Haseman, J. K., Payne, W. W., Falk, H. L., and MacFarland, H. N. 1974. "Inhalation Studies of Nickel Sulfide in Pulmonary Carcinogenesis of Rats," J. Natl. Cancer Inst. 54:1165-1172.
- Palmer, A. K., Street, A. E., Roe, F. J. C., et al. 1979. "Safety Evaluation of Toothpaste Containing Chloroform. II. Long Term Studies in Rats," J. Environ. Path. Toxicol. 2:821-833.
- Parker, R. D. R., Sharma, R. P., and Oberg, S. G. 1980. "Distribution and Accumulation of Vanadium in Mice Tissues," Arch. Environ. Contam. Toxicol. 9:393-403.
- Patty, F. A., Yapt, W. P., and Waite, C. P. 1930. "Acute Response of Guinea Pigs to Vapors of Some New Commercial Organic Compounds. V. Vinyl Chloride," *Public Health Rep.* 45:1963-1971.
- Pedersen, N. B. 1982. "The Effects of Chromium on the Skin," pp. 249-275, Biological and Environmental Aspects of Chromium, Langard, S., ed., Elsevier Biomedical Press, Amsterdam.
- Pegg, D. G. 1982. "Disposition of di(2-ethylhexyl)phthalate Following Inhalation and Peroral Exposure in Rats," OTS 8(d) submission, Microfiche No. 0206189.
- Penn, A., and Snyder, C. 1988. "Arteriosclerotic Plaque Development is 'Promoted' by Polynuclear Aromatic Hydrocarbons," *Carcinogenesis* 9:2185–2189.
- Perry, H. M., Kopp, S. J., Erlanger, M. W., and Perry, E. F. 1983. "Cardiovascular Effects of Chronic Barium Ingestion," pp. 155-164, in Proc. Univ. Missouri's 17th Ann. Conf. on Trace Substances in Environmental Health, Hemphill, D.D., ed., University of Missouri Press, Columbia, MO.
- Perry, K., Bowler, R. G., Buckell, H. M., Druett, H. A., and Schilling, R. S. F. 1948. "Studies in the Incidence of Cancer in a Factory Handling Inorganic Compounds of Arsenic - II: Clinical and Environmental Investigations," Br. J. Ind. Med. 5:6-15.

- Pershagen, G., Lind, B., and Bjorklund, N. E. 1982. "Lung Retention and Toxicity of Some Inorganic. Arsenic Compounds," *Environ. Res.* 29:425-434.
- Peterson, N. L. 1951, "Sulfates in Drinking Water," Off. Bull., N.D., Water Sewage Works, 18 (10/11)11.
- Pfeiffer, E. H. 1977. "Oncogenic Interaction of Carcinogenic and Non-Carcinogenic Polycyclic Aromatic Hydrocarbons in Mice," pp. 69-77, in *Air Pollution and Cancer in Man*, Mohr, V., et al., eds., International Agency for Research on Cancer, Lyon, France.
- Pflasterer, M. R., Bradshaw, W. S., Booth, G. M., and Carter, M. W. 1985. "Developmental Toxicity of Nine Selected Compounds Following Prenatal Exposure in the Mouse: Naphthalene, p-nitrophenol, Sodium Selenite, Dimethyl Phthalate, Ethylene Thiourea and Four Glycol Ether Derivatives," J. Toxicol. Environ. Health 15:25-38.
- Philips, F. S., et al. 1973. "In vivo Cytotoxicity of Polycyclic Hydrocarbons," in Pharmacology and the Future of Man, Proc. 5th Intl. Congr. Pharmacol., 1972, San Francisco, 2:75.
- Pinto, S. S., and McGill, C. M. 1953. "Arsenic Trioxide Exposure in Industry," Ind. Med. Surg. 22:281-287.
- Pirkle, J. L., Schwartz, J., Landis, J. R., and Harlan, W. R. 1985. "The Relationship Between Blood Lead Levels and Blood Pressure and Its Cardiovascular Risk Implication," *Am. J. Epidemiol.* 121:246-258.
- Platt, K. L., Pfeiffer, E., Petrovic, P., et al. 1990. "Comparative Tumorigenicity of Picene and Dibenz(a,h)anthracene in the Mouse," *Carcinogenesis* 11:1721-1726.
- Pocock, S. J., Shaper, A. G., Ashby, D., Delves, T., and Whitehead, T. P. 1984. "Blood Lead Concentration, Blood Pressure, and Renal Function," Br. Med. J. 289:872-874.
- Pollack, G. M., Li, R. C. K., Ermer, J. C., and Shen, D. D. 1985. "Effects of Route of Administration and Repetitive Dosing on the Disposition Kinetics of di(2-ethylhexyl)phthalate and Its Mono De-esterified Metabolite in Rats," *Toxicol. Appl. Pharmacol.* 79(2):246-256.
- Pollia, J. A. 1939. "Investigations on the Possible Carcinogenic Effect of Anthracene and Chrysene and Some of Their Compounds. I. The Effect of Skin Painting on the Skin of Mice," J. Ind. Hyg. Toxicol. 23:449-451.
- Popp, J. A., and Leonard, T. B., 1982. "The Use of *in vivo* Hepatic Initiation-Promotion Systems in Understanding the Hepatocarcinogenesis of Technical Grade Dinitrotoluene," *Toxicol. Pathol.* 10:190-196.
- Porter, K. G., McMaster, D., Elmes, M. E., et al. 1977. "Anemias and Low Serum-Copper During Zinc Therapy," Lancet: 744.
- Potkonjak, V., and Pavlovich, M. 1983. "Antimoniosis: A Particular Form of Pneumoconiosis. I. Etiology, Clinical and X-ray Findings," Int. Arch. Occup. Environ. Health 51:199-207.
- Pound, A. W. 1970. "Induced Cell Proliferation and the Initiation of Skin Tumor Formation in Mice by Ultraviolet Light," *Pathology* 2:269-275.
- Prasad, A. S., Schulert, A. R., Sandstead, H. H., et al. 1963. "Zinc, Iron, and Nitrogen Content of Sweat in Normal and Deficient Subjects," J. Lab. Clin. Med. 62:84-89.
- Price, N. H., Yates, W. G., and Allen, S. D. 1979. Toxicity Evaluation for Establishing IDLH Values, PB87-229498, Till, J. E., and Meyer, H. R., eds., National Institute for Occupational Safety and Health, Cincinnati, OH.

Prodan, L., Suciu, I., Pislaru, V., et al. 1975. "Experimental Chronic Poisoning with Vinyl Chloride_____ (Monochloroethene), Ann. NY Acad. Sci. 246:159-163.

- Quast, J. F., Humiston, C. G., Wade, C. E., et al. 1983. "A Chronic Toxicity and Oncogenicity Study in Rats and Subchronic Toxicity Study in Dogs on Ingested Vinylidine Chloride," Fund. Appl. Toxicol. 3:55-62.
- Quast, J. F., McKenna, M. J., Rampy, L. W., and Norris, J. M. 1986. "Chronic Toxicity and Oncogenicity Study on Inhaled Vinylidine Chloride in Rats," *Fund. Appl. Toxicol.* 6:105-144.
- Radike, M. J., Warkany, J., Stemmer, K., and Bingham, E. 1988. "Perinatal Carcinogenesis Induced by Inhaled Vinyl Chloride," *Toxicologist* 8:166.
- Rampy, L. W., Quast, J. F., Humiston, C. G., et al. 1977. "Interim Results of Two-Year Toxicological Studies in Rats of Vinylidine Chloride Incorporated in the Drinking Water or Administered by Repeated Inhalation," *Environ. Health Perspect.* 21:333-343.
- Rana, S. V. S., and Kumar, A. 1980. "Some Biological, Hematological and Histological Observations in Molybdenotic Rats," Curr. Sci. 49:383-386.
- Rana, S. V. S., and Kumar, A. 1978. "Simultaneous Effects of Dietary Molybdenum and Copper on the Accumulation of Copper in the Liver and Kidney of Copper Poisoned Rats. A Histochemical Study," Ind. Health 18:9-17.
- Rao, M. S., and Reddy, J. K., 1991. "An Overview of Peroxisome Proliferator-Induced Hepatocarcinogenesis," *Environ. Health Perspect.* 93:205-209.
- Rao, M. S., Yeldandi, A. V., and Subbarao, V. 1990. "Quantitative Analysis of Hepatocellular Lesions Induced by di(2-ethylhexyl)phthalate in F-344 Rats," J. Toxicol Environ Health. 30(2):85-89.
- Rebar, A. H., Greenspan, B. J., and Allen, M. D. 1986. "Acute Inhalation Toxicopathology of Lithium Combustion Aerosols in Rats," *Fundam. Appl. Toxicol*, 7:58-67.
- Reddy, J. K., and Lalwani, N. D. 1983. "Carcinogenesis by Hepatic Peroxisome Proliferators: Evaluation of the risk of Hypolipidemic Drugs and Industrial Plasticizers to Humans," CRC Crit. Rev. Toxicol. 12:1-58.
- Reeves, A. L. 1965. "The Absorption of Beryllium from the Gastrointestinal Tract," Arch. Environ. Health 11:209-214.
- Reeves, A. L., and Vorwald, A. J. 1967. "Beryllium Carcinogenesis. II. Pulmonary Deposition and Clearance of Inhaled Beryllium Sulfate in the Rat," *Cancer Res.* 27:446–451.
- Reeves, A. L., Deitch, D., and Vorwald, A. J. 1967. "Beryllium Carcinogenesis. I. Inhalation Exposure of Rats to Beryllium Sulfate Aerosol," *Cancer Res.* 27:439-445.
- Reeves, A. L., and Deitch, D. 1969. "Influence of Age on the Carcinogenic Response to Beryllium Inhalation," in *Proc. 16th Internat. Cong. Occup Health, Tokyo, Japan*, Harishima, S., ed., Japan Industrial Safety Association, Tokyo, Japan.

Reitz, R. H., Fox, T. R., Ramsey, J. C., et al. 1982. "Pharmacokinetics and Macromolecular Interactions of Ethylene Dichloride in Rats After Inhalation or Gavage." *Toxicol. Appl. Pharmacol.* 62:190-204.

Rendall, R. E. G., Phillips, J. I., and Renton, K. A. 1994. "Death Following Exposure to Fine Particulate Nickel from a Metal Arc Process," Ann. Occup. Hyg. 38:921-930.

Renes, L. E. 1953. "Antimony Poisoning in Industry," Arch. Ind. Hyg. 7:99-108.

- Reshetyuk, A. L., Talakina, E. I., and En'yakova, P. A. 1970. "Toxicological Evaluation of Acenaphthene and Acenaphthylene," Gig. Tr. Prof. Zabol. 14:46-47.
- Reuber, M. D. 1979. "Carcinogenicity of Chloroform," Environ. Health Perspect. 31:171-182.
- Reymann, F., Moller, R., and Nielsen, A. 1978. "Relationship Between Arsenic Intake and Internal Malignant Neoplasms," Arch. Dermatol. 114:378-381.
- Reznik-Schuller, H., and Mohr, U. 1974. "Investigations on the Carcinogenic Burden by Air Pollution in Man. IX. Early Pathological Alterations of the Bronchial Epithelium in Syrian Golden Hamsters After Intratracheal Instillation of Benzo(a)pyrene," Zbl. Bakt. Hyg., I. Abt. Orig. B. 159:493.
- Rice, J. E., Hosted, T. J., Jr., DeFloria, M. C., et al. 1986. "Tumor-Initiating Activity of Major in vivo Metabolites of Indeno(1,2,3-cd)pyrene on Mouse Skin," *Carcinogenesis* 7:1761-1764.
- Rice, J. E., Coleman, D. T., Hosted, T. J., Jr., et al. 1985. "Identification of Mutagenic Metabolites of Indeno(1,2,3-cd)pyrene Formed *in vitro* with Rat Liver Enzymes." *Cancer Res.* 45:5421-5425.
- Richman, A. V., Masco, H. L., Rifkin, S. I., et al. 1980. "Minimal Change Disease and Nephrotic Syndrome Associated with Lithium Therapy," Ann. Int. Med. 92:70-72.
- Rickert, D. E., Baker, T. S., Bus, J. S., et al. 1979. "Benzene Disposition in the Rat After Exposure by Inhalation," *Toxicol. Appl. Pharmacol.* 49:417-423.
- Rinsky, R. A., Young, R. J., and Smith, A. B. 1981. "Leukemia in Benzene Workers," Am. J. Ind. Med. 2:217-245.
- Roberts, R. S., Julian, J. A., Jadon, N., and Muir, D. C. F. 1994. "Cancer Mortality in Ontario Nickel Workers: 1950-1984," pp. 629-648, in *Nickel and Human Health*, Nieboer, E., and Nriagu, J. O., eds., John Wiley and Sons, Inc., New York.
- Robertson, I. G. C., and Jernström, B. 1986. "The Enzymatic Conjugation of Glutathione with Bay-Region Diol-epoxides of Benzo(a)pyrene, Benz(a)anthracene, and Chrysene," *Carcinogenesis* 7:1633-1636.
- Robinson, J. R., Felton, J. S., Levitt, R. C., Thorgiersson, S. S., and Nebert, D. W. 1975. "Relationship Between 'Aromatic Hydrocarbon Responsiveness' and the Survival Times in Mice Treated with Various Drugs and Environmental Compounds," *Mol. Pharmacol.* 11:850–865.
- Roe, F. J. C. 1962. "Effect of Phenanthrene On Tumor-Initiation by 3,4-Benzopyrene," Br. J. Cancer 16:503-506.
- Roe, F. J. C., and Grant, G. A. 1964. "Tests of Pyrene and Phenanthrene for Incomplete Carcinogenic and Anticarcinogenic Activity," Br. Emp. Cancer Campaign 41:59-60. (Abstract)
- Roe, F. J. C., Palmer, A. A. K., Worden, A. N., et al. 1979. "Safety Evaluation Of Toothpaste Containing Chloroform. I. Long-Term Studies in Mice," J. Environ. Toxicol. 2:799-819.
- Roels, H., Ghyselen, P., Buchet, J. P., Ceulemans, E., and Lauwerys, R. R. 1992. "Assessment of Permissible Exposure Level to Manganese in Workers Exposed to Manganese Dioxide Dust," Br. J. Ind. Med. 49:25-34.
- Roll, R., and Matthiaschk, G. 1981. "Investigations on Embryotoxic Effects of Thallium Chloride and Thallium Acetate in Mice and Rats," *Teratology* 24:46A-47A.
- Rosenman, K. D., Moss, A., and Kon, S. 1979. "Argyria: Clinical Implications of Exposure to Silver Nitrate and Silver Oxide," J. Occup. Med. 21:430-435.

Rosenthal, G. J., and Snyder, C.A. 1985. "Modulation of the Immune Response to Listeria monocytogenes_ by Benzene Inhalation," *Toxicol. Appl. Pharmacol.* 80:502-510.

Roshchin, I. V. 1967. "Toxicity of Vanadium Compounds Used in Modern Industry," Gig. Sanit. 32:26-32.

Roshchin, I. V. 1968. "Vanadium and Its Compounds," Medicina, Moscow.

- Roshchin, I. V., Ordzhonikidze, E. K., and Shalganova, I. V. 1980. "Vanadium Toxicity, Metabolism, Carrier State," J. Hyg. Epidemiol. Microbiol. Immunol. 24:377-383.
- Rossi, F., Acampora, R., Vacca, C., et al. 1987. "Prenatal and Postnatal Antimony Exposure in Rats: Effect on Vasomotor Reactivity Development of Pups," *Teratogen. Carcinogen. Mutagen.* 7:491-496.
- Rozenshstein, I. S. 1970. "Sanitary Toxicological Assessment of Low Concentrations of Arsenic Trioxide in the Atmosphere," *Hyg. Sanit.* 34:16-22.

RTECS (Registry for Toxic Effects of Chemical Substances) 1989. Pentachlorophenol.

RTECS 1983. "Fluoranthene" (computer printout).

- Rungby, J., and Danscher, G. 1984. "Hypoactivity in Silver Exposed Mice," Acta Pharmacol. Toxicol. 55:398-401.
- Russell, H. 1947. "An Unsuccessful Attempt to Induce Gliomata in Rabbits with Cholanthrene," J. Pathol. Bacteriol. 59:481-483.
- Sabourin, P. J., Chen, B. T., Lucier, G., et al. 1987. "Effects of Dose on the Absorption and Excretion of [¹⁴C]Benzene Administered Orally or by Inhalation in Rats and Mice," *Toxicol. Appl. Pharmacol.* 87:325-336.
- Saffiotti, U., et al. 1968. "A Method for the Experimental Induction of Bronchogenic Carcinoma," Cancer Res. 28:104.
- Salaman, M. H., and Roe, F. J. C. 1956. "Further Tests for Tumor-Initiating Activity: N,N-di(2chloroethyl)-p-aminophenylbutyric Acid (CB1348) as an Initiator of Skin Tumor Formation in the Mouse," Br. J. Cancer 10:363-378.
- Salcedo, J. C., Portales, A., Landecho, E., and Diaz, R. 1984. "Transverse Study of a Group of Patients with Vasculopathy from Chronic Arsenic Poisoning in Communities of the Francisco de Madero and San Pedro Districts, Coahuila, Mexico," *Revista de Medicina de Torreor* 12:16.
- Samman, S., and Roberts, D. C. K. 1987. "The Effect of Zinc Supplements on Plasma Zinc and Copper Levels and the Reported Symptoms in Healthy Volunteers," *Med. J. Australia* 146:246-249.
- Sample, B. E., Beauchamp, J. J., Efroymson, R. A., Suter, G. W., II, and Ashwood, T. L. 1998a. Development and Validation of Bioaccumulation Models for Earthworms, ES/ER/TM-220, Oak Ridge National Laboratory, Oak Ridge, TN.
- Sample, B. E., Beauchamp, J. J., Efroymson, R. A., Suter, G. W., II, and Ashwood, T. L. 1998b. Development and Validation of Bioaccumulation Models for Small Mammals, ES/ER/TM-219, Oak Ridge National Laboratory, Oak Ridge, TN.
- Sample, B. E., Opresko, D. M., and Suter, G. W., II 1996. Toxicological Benchmarks for Wildlife: 1996 Revisions, ES/ER/TM-125, Oak Ridge National Laboratory, Oak Ridge, TN.



- Sample, B. E., and Suter, G. W., II 1994. Estimating Exposure of Terrestrial Wildlife to Contaminants, ES/ER/TM-125, Oak Ridge National Laboratory, Oak Ridge, TN.
- Sanders, V. M., White, K. L., Jr., Shopp, G. M., et al. 1985. "Humoral and Cell-Mediated Immune Status of Mice Exposed to 1,1,2-Trichloroethane;" Drug Chem. Toxicol. 8:357-372.
- Sanders, V. M., Tucker, A. N., White, K. L., Jr., et al. 1982. "Humoral and Cell-Mediated Immune Status in Mice Exposed To Trichloroethylene in Drinking Water," *Toxicol. Appl. Pharmacol.* 62:358-368.
- Sandmeyer, E. E. 1981. "Aromatic hydrocarbons," pp. 3291-3300, 3333-3343, 3346, and 3351-3353, in *Patty's Industrial Hygiene and Toxicology*, 3rd. rev. ed., Vol. 2B, Clayton, G. D., and Clayton, F. E., eds., John Wiley & Sons, Inc., New York.
- Sandmeyer, E. E., and Kirwin, C. J., Jr. 1978. "Esters," pp. 2342-2352, in Patty's Industrial Hygiene and Toxicology, Vol. 2A, Clayton, G. D., and Clayton, F. E., eds., John Wiley & Sons, Inc., New York.
- Sansone, M. E. G., and Ziegler, D. K. 1985. "A Review of Neurologic Complications," Clin. Neuropharmacol. 8:242-248.
- SAS[®] (Statistical Analysis System) 1990. SAS[®] Procedures Guide, Version 6, 3rd ed. SAS Institute, Inc., Cary, NC.
- Savolainen, H., Pfaffli, P., Tengen, M., and Vaino, H. 1977. "Biochemical and Behavioural Effects of Inhalation Exposure to Tetrachloroethylene and Dichloromethane," J. Neuropathol. Exp. Neurol. 36:941-949.
- Sax, N. I. 1984. Dangerous Properties of Industrial Materials, 6th ed., pp. 2717-2720, Van Nostrand Reinhold Company, New York.
- Sax, N. I., and Lewis, R. J. 1989. Properties of Industrial Materials, p. 1152, 7th ed., Volume III, Van Nostrand Reinhold Co., New York.
- Schafer, W. B. 1951. "Acute Hemolytic Anemia Related to Naphthalene: Report of a Case in a Newborn Infant," *Pediatrics* 7:172-174.
- Schepers, G. W. H., Durkan, T. M., Delahant, A. B., and Creedon, F. T. 1957. "The Biological Action of Inhaled Beryllium Sulfate: A Preliminary Chronic Toxicity Study on Rats," Am. Med. Assoc Arch. Ind. Health 15:32-38.
- Schlede, E., Kuntzman, R., Haber, S., and Conney, A. H. 1970. "Effect of Enzyme Induction on the Metabolism and Tissue Distribution of Benzo(a)pyrene," *Cancer Res.* 30: 2898-2904.
- Schlicker, S. A., and Cox, D. H. 1968. "Maternal Dietary Zinc and Development; Zinc, Iron, and Copper Content of the Rat Fetus," J. Nutr. 95:287-294.
- Schlicker, S. A., and Cox, D. H. 1967. "Maternal Dietary Zinc in Excess, Fetal Development, and Iron and Copper Metabolism" (abstract), *Fed. Amer. Proc.* 26:520.
- Schmahl, D. 1955. "The Testing of Naphthalene and Anthracene as Carcinogenic Agents in the Rat," Z. Krebsforsch. 60:697-710. (German with English translation)
- Schroeder, H. A., and Mitchener, M. 1975a. "Life-Term Studies in Rats: Effects of Aluminum, Barium, Beryllium, and Tungsten," J. Nutr. 105:421-427.
- Schroeder, H. A., and Mitchener, M. 1975b. "Life-Term Effects of Mercury, Methyl Mercury and Nine Other Trace Metals on Mice," J. Nutr. 105:452-458.

Schroeder, H. A., and Mitchener, M. 1971. "Toxic Effects of Trace Elements on the Reproduction of Mice_ and Rats," Arch. Environ. Health 23:102-106.

Schroeder, H. A., Mitchener, M., and Nason, A. P. 1970. "Zirconium, Niobium, Antimony, Vanadium and Lead in Rats: Life-time Studies," J. Nutr. 100:59-68.

Schroeder, H. A., Balassa, D. D., and Tipton, I. H. 1966. "Essential Trace Metals in Man: Manganese, A Study in Homeostasis," J. Chron. Dis. 19:545-571.

Schroeder, H. A., Balassa, J. J., and Tipton, I. H. 1962. "Abnormal Trace Metals in Man: Chromium," J. Chron. Dis. 15:941.

Schroeder, S. R., and Hawk, B. 1986. "Child-Caregiver Environmental Factors Related to Lead Exposure and IQ," in *Toxic Substances and Mental Retardation: Neurobehavioral Toxicology and Teratology*, Schroeder, S. R., ed., Washington, DC. (AAMD Monograph Series)

Schwartz, J. 1991. "Lead, Blood Pressure, and Cardiovascular Disease in Men and Women." Environ. Health Perspect. 91:71-75.

Schwetz, B. A., Quast, J. F., Keeler, P. A. et al. 1978. "Results of Two-Year Toxicity and Reproduction Studies on Pentachlorophenol," pp. 301-309, in *Pentachlorophenol: Chemistry, Pharmacology and Environmental Toxicology*, Rao, K. R., ed., Plenum Press, New York.

Schwetz, B., Leong, B., and Gehring, P. 1975. "The Effects of Maternally Inhaled Trichloroethylene, Perchloroethylene, Methylchloroform, and Methylene Chloride on Embryonal and Fetal Development in Mice and Rats," *Toxicol. Appl. Pharmacol.*, 32:84–92.

Schwetz, B. A., Leong, B. K. G., and Gehring. P. J. 1974a. "Embryo- and Fetotoxicity of Inhaled Carbon Tetrachloride, 1,1-Dichloroethane, and Methyl Ethyl Ketone in Rats," *Toxicol. Appl. Pharmacol.* 28:452-464.

Schwetz, B. A., Keeler, P. A., and Gehring, P. J. 1974b. "The Effect of Purified and Commercial Grade Pentachlorophenol on Rat Embryonal and Fetal Development," *Toxicol. Appl. Pharmacol.* 28:151-161.

Schwetz, B., Leong, B., and Gehring, P. 1974c. "Embryo- and Fetotoxicity of Inhaled Chloroform in Rats," *Toxicol. Appl. Pharmacol.* 28:442-451.

Scribner, J. D. 1973. "Brief Communication: Tumor Initiation by Apparently Noncarcinogenic Polycyclic Aromatic Hydrocarbons," J. Natl. Cancer Inst. 50:1717-1719.

Sears, J. K., and Touchette, N. W. 1982. "Plasticizers," p. 174, in Grayson, M., and Eckroth, D., eds., Kirk-Othmer Encyclopedia of Chemical Technology, Vol. 18, 3rd ed., John Wiley & Sons, New York.

Sechzer, J. A., Alexander, G. J., and Lieberman, K. W. 1992. "Maternal Neglect and Delayed Development: Effects of Lithium Intake in Rats," Anat. Rec. 232:79A-80A. (Abstract of a paper presented at the 105th meeting of the American Association of Anatomists)

Seidenberg, J. M., Anderson, D. G., and Becker, R. A. 1986. "Validation of an *in vivo* Developmental Toxicity Screen in the Mouse," *Teratogen. Carcinogen. Mutagen.* 6:361-374.

Sellakumar, A., and Shubik, P. 1974. "Carcinogenicity of Different Polycyclic Hydrocarbons in the Respiratory Tract of Hamsters," J. Natl. Cancer Inst. 53:1713-1719.

Sharma, R. P., Oberg, S. G., and Parker, R. D. R. 1980. "Vanadium Retention in Rat Tissues Following Acute Exposures to Different Dose Levels," J. Toxicol. Environ. Health 6:45-54.

- Shear, M. J., and Leiter, J. 1941. "Studies in Carcinogenesis. XVI. Production of Subcutaneous Tumors on Mice by Miscellaneous Polycyclic Compounds," J. Natl. Cancer Inst. 11:241-258.
- Shear, M. J. 1938. "Studies in Carcinogenesis. V. Methyl Derivatives of 1,2-Benzanthracene," Am. J. Cancer 33:449-551.
- Sherwood, R. L., O'Shea, W., Thomas, P. T., et al. 1987. "Effects of Inhalation of Ethylene Dichloride on Pulmonary Defenses of Mice and Rats," *Toxicol. Appl. Pharmacol.* 91:491-496.
- Shiota, K., and Mima, S. 1985. "Assessment of the Teratogenicity of di(2-ethylhexyl)phthalate and mono(2-ethyl hexyl)phthalate in Mice," Arch. Toxicol. 56(4):263-266.
- Shopp, G. M., White, K. L., Jr., Holsapple, M. P., et al. 1984. "Naphthalene Toxicity in CD-1 Mice: General Toxicology and Immunotoxicology," *Fund. Appl. Toxicol.* 4:405-419.
- Short, R. D., Jr., Minor, J. L., Winston, J. M., et al. 1977. Toxicity Studies of Selected Chemicals. Task II. The Developmental Toxicity of Vinylidine Chloride Inhaled by Rats and Mice during Gestation, EPA-560/6-77-022, Midwest Research Institute for the Office of Toxic Substances, U.S. Environmental Protection Agency.
- Shubik, P., Pietra, G., and Della Porta, G. 1960. "Studies of Skin Carcinogenesis in the Syrian Golden Hamster," Cancer Res. 20:100-105.
- Siddiqui, A., and Srivastava, S. P. 1992. "Effect of di(2-ethylhexyl)phthalate Administration on Sperm Count and on Sperm Metabolic Enzymes," *Bull. Environ. Contam. Toxicol.* 48(1):115-119.
- Sims, P. 1970. "Qualitative and Quantitative Studies of the Metabolism of a Series of Aromatic Hydrocarbons by Rat Liver Preparations," *Biochem. Pharmacol.* 19:795-818.
- Sims, P., and Grover, P. L. 1981. "Involvement of Dihydrodiols and Diol-epoxides in the Metabolic Activation of Polycyclic Hydrocarbons Other Than Benzo(a)pyrene," pp. 117-181, in *Polycyclic Hydrocarbons and Cancer.*, Vol. 3., Gelboin, H. V., and Ts'O, P. O. P., eds., Academic Press, New York.
- Sims, P., and Grover, P. L. 1974. "Epoxides in Polycyclic Aromatic Hydrocarbon Metabolism and Carcinogenesis," Adv. Cancer Res. 20:165-274.
- Sittig, M. (ed.) 1985a. Handbook of Toxic and Hazardous Chemicals and Carcinogens, 2nd ed. Noyes Publications, Park Ridge, NJ.
- Sittig, M. 1985b. "Di(2-ethylhexyl)phthalate," pp. 345-346, in Handbook of Toxic and Hazardous Chemicals and Carcinogens, 2nd ed., Noyes Publications, Park Ridge, NJ.
- Sittig, M. 1981. Handbook of Toxic and Hazardous Chemicals, Noyes Publications, Park Ridge, NJ.
- Sjoberg, S. G. 1956. "Vanadium Dust, Chronic Bronchitis and Possible Risk of Emphysema—A Followup Investigation of Workers at a Vanadium Factory," Acta Med. Scand. 154:381–386.
- Sjoberg, S. G. 1955. "Vanadium Bronchitis from Cleaning Oil-Fired Boilers," A.M.A. Arch. Ind. Health 11:505-12.
- Sjoberg, S. G. 1951. "Health Hazards in the Production and Handling of Vanadium Pentoxide," A.M.A. Arch. Ind. Hyg. Occup. Med. 3:631-646.
- Sjoberg, S. G. 1950. "Vanadium Pentoxide Dust A Clinical and Experimental Investigation on Its Effect After Inhalation," Acta Med. Scand. 238(suppl):338.

- Slaga, T. J., Jecker, L., Bracken, W. M., et al. 1979. "The Effects of Weak or Non-carcinogenic Polycyclic Hydrocarbons on 7,12-Dimethylbenz(a)anthracene and Benzo(a)pyrene Skin Tumor-Initiation," Cancer Lett. 7:51-59.
- Smialowicz, R. J., Rogers, R. R., Rowe, D. G., et al. 1987. "The Effects of Nickel on Immune Function in the Rat," *Toxicology* 44:271-281.
- Smialowicz, R. J., Rogers, R. R., Riddle, M. M., et al. 1985. "Immunologic Effects of Nickel: II. Suppression of Natural Killer Cell Activity," *Environ. Res.* 36:56–66.
- Smialowicz, R. J., Rogers, R. R., Riddle, M. M., and Scott, G. A. 1984. "Immunologic Effects of Nickel: I. Suppression of Cellular and Humoral Immunity," *Environ. Res.* 33:413–427.
- Smith, S. P., Peterson, J., and Kwan, K. H. M. 1989. "Chromium Accumulation, Transport and Toxicity in Plants," Toxicol. Environ. Chem. 24:241-251.
- Smyth, H. F., Smyth, H. F., Jr., and Carpenter, C. P. 1936. "The Chronic Toxicity of Carbon Tetrachloride—Animal Exposures and Field Studies," J. Ind. Hyg. Toxicol. 18:277-298.
- Smyth, H. F., Jr., Carpenter, C. P., Weil, C. S. et al. 1962. "Range-Finding Toxicity Data: List VI," Am. Ind. Hyg. Assoc. J. 23:95-107.
- Snell, K. C., and Stewart, H. L. 1962. "Pulmonary Adenomatosis Induced in DBA/2 Mice by Oral Administration of Dibenz(a,h)anthracene," J. Natl. Cancer Inst. 28:1043-1049.
- Snyder, C. A. 1987. "Hydrocarbons," pp. 3–37, in *Ethel Browning's Toxicity and Metabolism of Industrial Solvents*, 2nd ed., Vol. 1, R. Snyder, ed. Elsevier, New York.
- Snyder, R. 1984. "The Benzene Problem in Historical Perspective," Fundam. App. Toxicol. 4:692-699.
- Sokal, J. A., Baranski, B., Majka, J., et al. 1980. "Experimental Studies on the Chronic Effects of Vinyl Chloride in Rats," J. Hyg. Epidemiol. Microbiol. Immunol. 24:285-294.
- Sommers, S. C., and McManus, R. G. 1953. "Multiple Arsenical Cancers of the Skin and Internal Organs," *Cancer* 6:347-359.
- Spencer, H. C., Rowe, V. K., Adams, E. M., et al. 1951. "Vapor Toxicity of Ethylene Dichloride Determined by Experiments on Laboratory Animals," Arch. Ind. Hyg. Occup. Med. 4:482-493.
- Spiegl, C. J., Scott, J. K., Steinhardt, H., et al. 1956. "Acute Inhalation Toxicity of Lithium Hydride," AMA Arch. Ind. Health 14:468–470.
- Spitalny, K. C., Brondum, J., Vogt, R. L., et al. 1984. "Drinking-Water-Induced Copper Intoxication in a Vermont Family," *Pediatrics* 74:1103-1106.
- Spreafico, F., Zuccato, E., Msarcucci, F., et al. 1980. "Pharmacokinetics of Ethylene Dichloride in Rats Treated by Different Routes and Its Long-term Inhalatory Toxicity," pp. 107–133, *Ethylene Dichloride: A Potential Health Risk*, Banbury Report No. 5, Ammes, B. N., Infante, P., and Reitz, R., eds, Cold Spring Harbor, New York.
- Srbova, J., Teisinger, J., and Skramovsky, S. 1950. "Absorption and Elimination of Inhaled Benzene in Man," Arch. Ind. Hyg. Occup. Med. 2:1-8.
- Stanton, M. F., Miller, E., Wrench, C., et al. 1972. "Experimental Induction of Epidermoid Carcinoma in the Lungs of Rats by Cigarette Smoke Condensate," J. Natl. Cancer Inst. 49:867-877.



- Stayner, L. T., Dannenberg, A. L., Bloom, T., and Thun, M. 1993. "Excess Hepatobiliary Cancer Montality Among Munitions Workers Exposed to Dinitrotoluene," J. Occup. Med. 35:291-296.
- Steffee, C. H., and Baetjer, A. M. 1965. "Histopathologic Effects of Chromate Chemicals. Report of Studies in Rabbits, Guinea Pigs, Rats, and Mice," Arch. Environ. Health 11:66-75.
- Steiner, P. E. 1955. "Carcinogenicity of Multiple Chemicals Simultaneously Administered," Cancer Res. 15:632-635.
- Stern, R. M. 1982. "Chromium Compounds: Production and Occupational Exposure," pp. 7-47, in Biological and Environmental Aspects of Chromium, Langard, S., ed. Elsevier, New York.
- Stevenson, C. J. 1965. "Antimony Spots," Trans. St. John's Hospital Dermat. Soc. 51:40-42.
- Stewart, R. 1969. "Acute Tetrachloroethylene Intoxication," J. Am. Med. Assoc. 208:1490-1493.
- Stewart, R. D., Baretta, E. D., Dodd, H. C., and Torkelson, T. R. 1970. "Experimental Human Exposure to Tetrachloroethylene," Arch. Environ. Health 20:224-229.
- Stiefel, T., Schulze, K., Zorn, H., and Tolg, G. 1980. "Toxicokinetic and Toxicodynamic Studies of Beryllium," Arch. Toxicol. 45:81-92.
- Stiles, K. M., and Bellinger, D. C. 1993. "Neuropsychological Correlates of Low-level Lead Exposure in School-age Children: A Prospective Study;" *Neurotoxicol. Teratol.* 15:27-35.
- Stokinger, H. E. 1981a. "Molybdenum," pp. 1807–1820, in *Patty's Industrial Hygiene and Toxicology*, 3rd ed., Vol. 2A., *Toxicology*. Clayton, G. D., and Clayton, F. E., eds. John Wiley & Sons, New York.
- Stokinger, H. E. 1981b. "Silver," pp. 1881–1894, in Patty's Industrial Hygiene and Toxicology, Vol. 2A, Clayton, G. D., and Clayton, F. E., eds. John Wiley & Sons, New York.
- Stokinger, H. E. 1981c. "The Metals," pp. 1749–1769, in *Patty's Industrial Hygiene and Toxicology*, Vol. 2A, Clayton, G. D., and Clayton, F. E., eds. John Wiley & Sons, New York.
- Stokinger, H. E. 1981d. "Zinc," pp. 2033–2049, in *Patty's Industrial Hygiene and Toxicology*, 3rd rev. ed., Vol. 2A. Clayton, G. D., and Clayton, E., eds., John Wiley and Sons, New York.
- Stollery, B. T., Broadbent, D. E., Banks, H. A., and Lee, W. R. 1991. "Short-Term Prospective Study of Cognitive Functioning in Lead Workers," Br. J. Ind. Med. 48:739-749.
- Stoner, G. D., Shimmkin, M. B., Troxell, M. C., et al. 1976. "Test for Carcinogenicity of Metallic Compounds by the Pulmonary Tumor Response in Strain A Mice," *Cancer Res.* 36:1744-1747.
- Storer, R. D., Jackson, N. M., and Conolly, R. B. 1984a. "In Vivo Genotoxicity and Acute Hepatotoxicity of 1,2-Dichloroethane in Mice: Comparison of Oral, Intraperitoneal, and Inhalation Routes of Exposure," Cancer Res. 44:4267-4271.
- Storer, J. S., DeLeon, I., Millikan, L. E., et al. 1984b. "Human Absorption of Crude Coal Tar Products," Arch Dermatol. 120:874-877.
- Story, D. L., Meierhenry, E. F., Tyson, C. A., et al. 1986. "Differences in Rat Liver Enzyme-Altered Foci Produced by Chlorinated Aliphatics and Phenobarbital," *Toxicol. Ind. Health* 2:351-362.
- Stott, W. T., Quast, J. F., and Watanabe, P. G. 1982. "Pharmacokinetics and Macromolecular Interactions of Trichloroethylene in Mice and Rats," *Toxicol. Appl. Pharmacol.* 62:137-151.
- Suciu, I., Prodan, L., Lazar, V., et al. 1981. "Research on Copper Poisoning," Med. Lav. 72:190-197.

- Sun, J. D., Wolff, R. K., and Kanapilly, G. M. 1982. "Deposition, Retention, and Biological Fate of Inhaled Benzo(a)pyrene Adsorbed onto Ultrafine Particles and as a Pure Aerosol," *Toxicol. Appl. Pharmacol.* 65:231-244.
- Sunderman, F. W., Jr., Dingle, B., Hopfer, S. M., and Swift, T. 1988. "Acute Nickel Toxicity in Electroplating Workers Who Accidentally Ingested a Solution of Nickel Sulfate and Nickel Chloride," Am. J. Indust. Med. 14:257-266.
- Sunderman, F. W., and Donnelly, A. J. 1965. "Studies of Nickel Carcinogenesis Metastasizing Pulmonary Tumors in Rats Induced by the the Inhalation of Nickel Carbonyl," *Am. J. Path.* 46:1027.
- Sunderman, F. W., Donnelly, A. J., West, B., and Kincaid, J. F. 1959. "Nickel Poisoning. IX. Carcinogenesis in Rats Exposed to Nickel Carbonyl," Arch. Ind. Health 20:36.
- Suntzeff, V., Croninger, A. B., Wynder, E. L., et al. 1957. "Use of Sebaceous-Gland Test of Primary Cigarettes-Tar Fractions and of Certain Noncarcinogenic Polycyclic Hydrocarbons," *Cancer* 10:250-254.
- Susten, A., Dames, B., Burg, J., and Niemeier, R. 1985. "Percutaneous Penetration of Benzene in Hairless Mice: An Estimate of Dermal Absorption During Tire-Building Operations," Am. J. Ind. Med. 7:323-335.
- Suter, G. W., II, Sample, B. E., Jones, D. S., and Ashwood, T. L. 1995. Approach and Strategy for Performing Ecological Risk Assessments for the U.S. Department of Energy's Oak Ridge Reservation: 1995 Revision, ES/ER/TM-33/R2, Oak Ridge National Laboratory, Oak Ridge, TN.

Suter, G. W., II 1993. Ecological Risk Assessment, Lewis Publishing, Chelsea, MI.

Suter, G. W., II 1989. "Ecological Endpoints," p. 2-1 to 2-28, in Ecological Assessment of Hazardous Waste Sites: A Field and Laboratory Reference Document, EPA/600/3-89/013, Warren-Hicks, W., Parkhurst, B. R., and Baker, S. S., Jr., eds., U.S. Environmental Protection Agency, Washington, DC.

Sutton, W. R., and Nelson, V. E. 1937. "Studies on Zinc," Proc. Soc. Exp. Biol. Med. 36:211-213.

Suzuki, Y. 1980. "Nonneoplastic Effects of Vinyl Chloride in Mouse Lung," Environ. Res. 21:235-253.

- Suzuki, Y. 1978. "Pulmonary Tumors Induced in Mice by Vinyl Chloride Monomer," Environ. Res. 16:285-301.
- Symanski, H. 1939. "Industrial Vanadium Poisoning, Its Origin and Symptomatology," Arch. Gewerbepathol. Gewerbehyg. 9:295-313.

Szabo, K. T. 1970. "Teratogenic Effects of Lithium Carbonate in the Foetal Mouse," Nature 225:73-75.

- Tarasenko, M., Promin, O., and Silayev, A. 1977. "Barium Compounds as Industrial Poisons (An Experimental Study)," J. Hyg. Epidem. Microbiol. Immunol. 21:361-373.
- Tashiro, S., and Matsumara, F. 1978. "Metabolism of *trans*-Nonachlor and Related Chlordane Components in Rat and Man," Arch. Environ. Contam. Toxicol. 7:113-117.
- Tay, C. H., and Seah, C. S. 1975. "Arsenic Poisoning from Anti-Asthmatic Herbal Preparations," Med. J. Aust. 2:424-428.

Tebrock, H. E., and Machle, W. 1968. "Exposure to Europium-Activated Yttrium Orthovanadate—A Cathodoluminescent Phosphor," J. Occup. Med. 10:692-696.

- Teirlynck, O. A., and Belpaire, J. 1985. "Disposition of Orally Administered di(2-ethylhexyl)phthalate and mono(2-ethylhexyl)phthalate in the Rat," Arch. Toxicol. 57(4):226-230.
- Tepper, L. B., Hardy, H. L., and Chamberlin, R. J. 1961. Toxicity of Beryllium Compounds, pp. 146-168, Elsevier, New York.
- Terada, H., Katsuta, K., Sasakawa, T., Saito, T., Shrota, H., Fukuchi, K., Sekiya, E., Yokoyama, Y., Hirokawa, S., Watanabe, G., Hasegawa, K., Shina, T. O., and Sekiguchi, E. 1960. "Clinical Observations of Chronic Toxicosis by Arsenic," *Nihon Rinsho* 18:2394–2403.
- Thienes, C. H., and Haley, T. J. 1972. Clinical Toxicology, 5th ed., Lea and Febiger, Philadelphia.
- Thiess, A. M., Korte, A., and Fleig, H. 1978. "Studies on Morbidity in Workers Exposed to di(2ethylhexyl)phthalate," Moeglichkeiten Grenzen Biol. Monit./Arbietsmed. Probl. Dienstleitungsgewerbes/Arbeitsmed, Kollog., Ber. Jahrestag. 18:137-154.
- Thiess, J., Stoner, G., Shimkin, M., and Weisburger, E. 1977. "Tests for Carcinogenicity of Orgnanic Contaminants of United States Drinking Waters by Pulmonary Response in Strain A Mice," *Cancer* Res. 37:2717-2720.
- Thyssen, J., Althoff, J., Kimmerle, G., and Mohr, U. 1981. "Inhalation Studies with Benzo(a)pyrene in Syrian Golden Hamsters," J. Natl. Cancer Inst. 66:575-577.
- Tierney, D. R., Blackwood, T. R., and Piana, M. R. 1979. Status Assessment of Toxic Chemicals: Vinylidine Chloride, EPA 600/2-79-2100; PB80-146442, Monsanto Research Corporation for Office of Research and Development, U.S. Environmental Protection Agency, Cincinnati, OH.
- Tipton, I. H., Stewart, P. I., and Dickson, J. 1969. "Patterns of Elemental Excretion in Long-Term Balance Studies," *Health Phys.* 16:455.
- Toft, K., Olofsson, T. A., and Berlin, M. 1982. "Toxic Effects on Mouse Bone Marrow Caused by Inhalation of Benzene," Arch. Toxicol. 51:295-302.
- Tomaszewski, D. E., Agarwal, D. K., and Melnick, R. L. 1986. "In vitro Steady-State Levels of Hydrogen Peroxide After Exposure of Male F344 Rats and Female B6C3F₁ Mice To Hepatic Peroxisome Proliferators," Carcinogenesis 7(11):1871-1876.
- Torkelson, T. R., Oyen, F., and Rowe, V. K. 1976. "The Toxicity of Chloroform as Determined by Single and Repeated Exposure of Laboratory Animals," *Am. Ind. Hyg. Assoc.* 37:697-704.
- Torkelson, R. R., Oyen, F., and Rowe, V. K. 1961. "The Toxicity of Vinyl Chloride as Determined by Repeated Exposure of Laboratory Animals," Am. Ind. Hyg. Assoc. J. 22:354-361.
- Torkelson, T. R. 1994. "Halogenated Aliphatic Hydrocarbons," pp. 4128–4132, in Patty's Industrial Hygiene and Toxicology, 4th ed., Volume II, Part E, Clayton, G. D., and Clayton, F. E., eds., John Wiley & Sons, New York.
- Torkelson, T. R., and Rowe, V. K., 1981a. "Tetrachloroethylene," pp. 3560-3568, 3586-3601, in *Patty's Industrial Hygiene and Toxicology*, 3rd ed., Clayton, G. D., and Clayton, F. E., eds., John Wiley & Sons, New York.
- Torkelson, T. R., and Rowe, V. K. 1981. "Halogenated Aliphatic Hydrocarbons," pp. 3537-3545, in Patty's Industrial Hygiene and Toxicology, 3rd. ed., Vol. 2B, Clayton, G. D., and Clayton, F. E., eds., John Wiley and Sons, New York.

- Travis. C. C., and Arms, A. D. 1988. "Bioconcentration of Organics in Beef, Milk, and Vegetation,". Environ Sci. Technol. 22:271-274.
- TRL (Toxicity Research Laboratories) 1989. "13-Week Mouse Oral Subchronic Toxicity Study," TRL Study No. 042-012, Toxicity Research Laboratories, Ltd., Muskegon, MI.
- Truelove, J. F., Tanner, J. R., Langlois, I. A., Stapley, R. A., Arnold, D. L., and Mes, J. C. 1990. "Effect of Polychlorinated Biphenyls on Several Endocrine Reproductive Parameters in the Female Rhesus Monkey," Arch. Environ. Contam. Toxicol. 19:939-943.
- Tseng, W. P., Chu, H. M., How, S. W., Fong, J. M., Lin, C. S., and Yeh, S. 1968. "Prevalence of Skin Cancer in an Endemic Area of Chronic Arsenium in Taiwan," J. Natl. Cancer Inst. 40:453-463.
- Tucker, A. N., Sanders, V. M., Barnes, D. W., et al. 1982. "Toxicology of Trichloroethylene in the Mouse," Toxicol. Appl. Pharmacol. 62:351-357.
- Tyl, R. W., and Neeper-Bradley, T. L. 1989. "Paradichlorobenzene: Two Generation Reproductive Study of Inhaled Paradichlorobenzene in Sprague-Dawley (CD) Rats," Laboratory Project 86-81-90605, Chemical Manufacturers Association, Chlorobenzene Producers Association, Washington, DC.
- Ungvary, G., Hudak, A., Tatrai, E., et al. 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," *Toxicology* 11:44-54.
- Urusova, T. P., 1953. "The Possible Presence of Dichloroethane in Humans with Exposure in Industrial Conditions," Gig. Sanit. 18:36-37.
- USAF (U.S. Air Force) 1990a. "Arsenic," pp. 75-1 through 75-102, in *The Installation Restoration Program Toxicology Guide*, Vol. 5, Harry G. Armstrong Aerospace Medical Research Laboratory, Wright-Patterson Air Force Base, OH.
- USAF 1990b. "Cadmium," in *The Installation Restoration Program Toxicology Guide*, Vol. 5. Harry G. Armstrong Aerospace Medical Research Laboratory, Wright Patterson Air Force Base, OH.
- USAF 1990c. "Chromium," in *The Installation Restoration Program Toxicology Guide*, Vol. 5, Harry G. Armstrong Aerospace Medical Research Laboratory, Wright-Patterson Air Force Base, OH.
- USAF 1990d. "Copper," p. 77 (1-43), in *The Installation Restoration Program Toxicology Guide*, Vol. 5. Harry G. Armstrong Aerospace Medical Research Laboratory, Wright-Patterson Air Force Base, OH.
- USAF 1990e. "Nickel," in *The Installation Restoration Program Toxicology Guide*, Vol. 5, Harry G. Armstrong Aerospace Medical Research Laboratory, Wright Patterson Air Force Base, OH.
- USAF 1989a. "1,1-Dichloroethane," pp. 9-1 through 9-38, in *The Installation Restoration Program Toxicology Guide*, Vol.1. Harry G. Armstrong Aerospace Medical Research Laboratory, Wright-Patterson Air Force Base, OH
- USAF 1989b. "Benzene," pp. 18-1 through 18-42, in The Installation Restoration Program Toxicology Guide, Wright Patterson Air Force Base, OH.
- USAF 1989c. "Pentachlorophenol," The Installation Restoration Toxicology Guide Vol. 3, pp. 39-1 through 39-49, Harry G. Armstrong Aerospace Medical Research Laboratory, Air Force Systems Command, Wright-Patterson Air Force Base, OH.
- USAF 1989d. The Installation Restoration Program Toxicology Guide, pp. 52-1 through 52-68, Vol. 3, Aerospace Medical Division, Air Force Systems Command, Wright-Patterson Air Force Base, OH.

- Valentine, J. L., Campion, D. S., Schluchter, M. D., and Massey, F. J. 1982. "Arsenic Effects on Human Nerve Conduction," pp. 409-412, in *Proceedings of the Fourth International Symposium on Trace Element Metabolism in Man and Animals*, Gawthorne, J. M., Howell, J. M., and White, C. L., eds., Springer-Verlag, New York.
- Valjcuk, N. K., and Sramko, N. P. 1973. "The Effects of Molybdenum on the Organism at Low-Level, Long-Term Exposure," Gig. Sanit. 2:107-108.
- Valli, V. E. O., McCarter, A., McSherry, B. J., et al. 1969. "Hematopoiesis and Epiphyseal Growth Zones in Rabbits with Molybdenosis," Am. J. Vet. Res. 30:435-445.
- van der Gulden, J. W., and Zielhuis, G. A. 1989. "Reproductive Hazards Related to Perchloroethylene. A review," Int. Arch. Occup. Environ. Health 61:235-242.
- Van Duuren, B. L., Goldschmidt, B. M., Lowengart, G., et al. 1979. "Carcinogenicity of Halogenated Olefinic and Aliphatic Hydrocarbons in Mice," J. Natl. Cancer Inst. 63:1433-1439.
- Van Duuren, B. L., Langseth, L., Goldschmidt, B. M., and Orris, L. 1967. "Carcinogenicity of Epoxides, Lactones and Peroxy Compounds. VI. Structure and Carcinogenic Activity," J. Natl. Cancer Inst. 39:1217.
- Van Duuren, B. L., and Goldschmidt, B. M. 1976. "Cocarcinogenic and Tumor-Promoting Agents in Tobacco Carcinogenesis." J. Natl. Cancer Inst. 56:1237-1242.
- Van Duuren, B. L., Sivak, A., Goldschmidt, B. M., et al. 1970. "Initiating Activity of Aromatic Hydrocarbons in Two-Stage Carcinogenesis." J. Natl. Cancer Inst. 44:1167-1173.
- Van Duuren, B. L., Katz, C., and Goldschmidt, B. M. 1973. "Brief Communication: Cocarcinogenic Agents in Tobacco Carcinogenesis," J. Natl. Cancer Inst. 51:703-705.
- Van Gestel, C. A. M. 1992. "The Influence of Soil Characteristics on the Toxicity of Chemicals for Earthworms: A Review," pp.44–45, in *Ecotoxicology of Earthworms*, Grieg-Smith, P. W., et al., eds., Intercept Ltd.
- Van Heyningen, R., and Pirie, A. 1976. "Naphthalene Cataract in Pigmented and Albino Rabbits," Exp. Eye Res. 22:393-394.
- Van Rensburg, S. J., Hall, J. M., and Gathercole, P. S. 1986. "Inhibition of Esophageal Carcinogenesis in Corn-Fed Rats by Riboflavin, Nicotinic Acid, Selenum, Molybdenum, Zinc, and Magnesium," Nutr. Cancer 8:163-170.
- Vanoeteren, C., Cornelis, R., Versieck, J, Hoste, J., and De Roose, J. 1982. "Trace Element Patterns in Human Lung Tissues," J. Radioanal. Chem. 70(1-2):219-238.
- Velsicol Chemical Corporation 1955. MRID No. 00062599, available from EPA, FOI, Washington, DC.
- Venugopal, B., and Luckey, T. D. 1978. "Molybdenum," pp. 253–257, in Metal Toxicity in Mammals. Vol. 2, Chemical Toxicity of Metals and Metalloids, Plenum Press, New York.
- Viallet, A., Guillaume, L., Cote, J., Legare, A., Lavoie, P. 1972. "Presinusoidal Portal Hypertension Following Chronic Arsenic Intoxication," *Gastroenterology* 62:177.
- Vimpani, G. V., Wigg, N. R., Robertson, E. F., et al. 1985. "The Port Pirie Cohort Study: Blood Lead Concentration and Childhood Developmental Assessment," pp. 139–155, in *Lead Environmental Health—The Current Issues*, Goldwater, L. J., Wysocki, L. M., Volpe, R. A., eds., edited proceedings, May, Duke University Press, Durham, NC.

- Vimpani, G., Baghurst, P., McMichael, A. J., et al. 1989. "The Effects of Cumulative Lead Exposure on Pregnancy Outcome and Childhood Development During the First Four Years," presented at Conference on Advances in Lead Research: Implications for Environmental Research, National Institute of Environmental Health Sciences, Research Triangle Park, NC.
- Vintinner, F. J., Vallenas, R., Carlin, C. E., et al. 1955. "Study of the Health of Workers Employed in Mining and Processing Vanadium Ore," A.M.A. Arch. Ind. Health 12(6):635-642.
- Viola, P. L., Bigotti, A., and Caputo, A. 1971. "Oncogenic Response of Rat Skin, Lungs, and Bones to Vinyl Chloride," *Cancer Res.* 31:516-522.
- Volkova, N. I. 1983. "Anthracene and Derivatives," pp. 162–163, in *Encyclopaedia of Occupational* Health and Safety, Vol. 1, Parmeggiani, L., ed., International Labour Office, Geneva.
- von Oettingen, W. R. 1964. The Halogenated Hydrocarbons of Industrial and Toxicological Importance, p. 271–283, Elsevier Press, Amsterdam.
- Vorwald, A. J. 1968. "Biologic Manifestation of Toxic Inhalation in Monkeys," p. 2, in Use of Nonhuman Primates in Drug Evaluation, Vagtborg, H., ed., Univ of Texas Press, Austin, TX.
- Vorwald, A. J. 1962. Progress Report (unpublished), Grant No. F-253-6. Am. Cancer Soc.
- Vorwald, A. J. 1953. "Adenocarcinoma in the Lung of Albino Rats Exposed to Compounds of Beryllium," pp. 103–109, in *Cancer of the Lung - An Evaluation of the Problem*, Proc. of the Scientific Session, Annual Meeting, November. Am. Cancer Soc., New York.
- Vorwald, A. J., Reeves, A. L., and Urban, E. J. 1966. "Experimental Beryllium Toxicology," in Beryllium—Its Industrial Hygiene Aspects, Stokinger, H. E., ed., Academic Press, New York.
- Vorwald, A. J., Pratt, P. C., and Urban, E. J. 1955. "The Production of Pulmonary Cancer in Albino Rats Exposed by Inhalants to an Aerosol of Beryllium Sulfate," Acta. Unio. Int. Cancrum. 11:735.
- Vouk, V. 1979. "Vanadium," pp. 659–674, in *Handbook on the Toxicology of Metals*, Friberg, L., Nordberg, G. F., and Voul, V. B., eds. Elsivie/North-Holland Biomedical Press, New York.
- Vozovaya, M. A. 1977. "The Effect of Dichloroethane on the Sexual Cycle and Embryogenesis of Experimental Animals," Akusk. Ginekol. 2:57-59.
- Wagner, S. L., Durand, L. R., Inman, R. D., Kiigemagi, U., and Deinzer, M. L. 1991. "Residues of Pentachlorophenol and Other Chlorinated Contaminants in Human Tissues: Analysis by Electron Capture Gas Chromatography and Electron Capture Negative Ion Mass Spectrophotometry," Arch. Environ. Contam. Toxicol. 21:596-606.
- Wagoner, J. K., Infante, P. F., and Bayliss, D. L. 1980. "Beryllium: An Etiologic Agent in the Induction of Lung Cancer, Nonneoplastic Respiratory Disease and Heart Disease Among Industrially Exposed Workers," Environ. Res. 21:15-34.
- Wahlberg, J. E. 1984. "Edema-Inducing Effects of Solvents Following Topical Administration," Dermatosen Beruf Umwelt 32:91-94.
- Walravens, P. A., Moure-Eraso, R., Solomons, C. C., et al. 1979. "Biochemical Abnormalities in Workers Exposed to Molybdenum Dust," Arch. Environ. Health 34:302-308.
- Walters, M., and Roe, F. J. C. 1965. "A Study of the Effects of Zinc and Tin Administered Orally to Mice over a Prolonged Period," Food Cosmet. Toxicol. 3:271-276.
- Wang, H. H., and MacMahon, B. 1979a. "Mortality of Pesticide Applicators," J. Occup. Med. 21:741-744.

- Wang, H. H., and MacMahon, B. 1979b. "Mortality of Workers Employed in the Manufacture of Chlordane and Heptachlor," J. Occup. Med. 21:745-748.
- Ward, C. O., Duna, R. A., Snyder, N. K., Alasker, R. D., Coate, W.B., and Craig, P. H. 1985. "Toxicological Studies of Certain Alkylated Benzenes and Benzene," A.M.A. Arch. Ind. Health 14:387-398.
- Warner, J. S. 1979. "Nickel Carbonyl. Prenatal Exposure," Science 203:1194-1195.
- Watanabe, K., Shima, S., Tachikawa, S., et al. 1985. Biotoxicity and Beryllium Distribution in Organs by Oral Administration of Beryllium Compounds for Long Periods. II. Experimental Study.
- Watt, W. D. 1980. Chronic Inhalation Toxicity of Antimony Trioxide: Validation for the TLV, Progress Report-Summary of Results, OTS206195.
- Wattenberg, L.W., and Leong, J. L. 1970. "Inhibition of the Carcinogenic Action of Benzo(a)pyrene by Flavones," *Cancer Res.* **30**:1922–1925.
- Waxweiler, R. J., Falk, H., McMichael, A., et al. 1977. "A Cross-Sectional Epidemiologic Survey of Vinyl-Chloride Workers," NTIS PB274193.
- Waxweiler, R. J., Stringer, W., Wagoner, J. K., et al. 1976. "Neoplastic Risk Among Workers Exposed to Vinyl Chloride," Ann. NY Acad. Sci. 271:40-48.
- Webb, D. R., Wilson, S. E., and Carter, D. E. 1987. "Pulmonary Clearance and Toxicity of Respirable Gallium Arsenide Particulates Intratracheally Instilled into Rats," Am. Ind. Hyg. Assoc. J. 48(7):660-667.
- Webb, D. R., Wilson, S. E., Carter, D. E. 1986. "Comparative Pulmonary Toxicity of Gallium Arsenide, Gallium (III) Oxide, and Arsenic (III) Oxide Intratracheally Instilled into Rats," *Toxicol. Appl. Pharmacol.* 82:405-416.
- Wei, H. J., Luo, X. M., and Yang, X. P. 1987. "Effect of Molybdenum and Tungsten on Mammary Carcinogenesis in Sprague-Dawley (SD) Rats," Chung. Hua. Chung. Tsa. Chih. 9:204-207.
- Weinstein, R. S., and Diamond, S. S., 1972. "Hepatotoxicity of Dichloromethane (Methylene Chloride) with Continuous Inhalation Exposure at a Low Dose Level," pp. 209-220, Proc. 3rd. Ann,. Conf. Env. Toxicol. Aerospace Med. Res. Lab., AMRL-TR-72-130, Wright-Patterson Air Force Base, OH.
- Weinstein, R. S., Boyd, D. D., and Back, K. C. 1972. "Effects of Continuous Inhalation of Dichloromethane in the Mouse-Morphological and Functional Observations," *Toxicol. Appl. Pharmacol.* 23:660.
- Weisburger, E. K. 1977. "Carcinogenicity Studies on Halogenated Hydrocarbons," Environ. Health Perspect. 21:7-16.
- Welch, L. 1987. "Reports of Clinical Disease Secondary to Methylene Chloride Exposure—A Collection of 141 Cases," unpublished study submitted to OPTS/EPA 3/31.
- Wennig, R., and Kirsch, N. 1988. "Molybdenum," pp. 437-447, in Handbook on Toxicity of Inorganic Compounds, Seiler, H. G., and Sigel, H., eds. Marcel Dekker, Inc., New York.
- Weyand, E. H., and Bevan, D. R. 1986. "Benzo(a)pyrene Disposition and Metabolism in Rats Following Intratracheal Instillation," *Cancer Res.* 46:5655-5661.

- White, J., and White, A. 1939. "Inhibition of Growth of the Rat by Oral Administration of Methylcholanthrene, Benzpyrene, or Pyrene and the Effects of Various Dietary Supplements," J. Biol. Chem. 131:149-161.
- White, K. L., Jr., Sanders, V. M., Barnes, D. W., et al. 1985a. "Toxicology of 1,1,2-Trichloroethane in the Mouse," Drug Chem. Toxicol. 8:333-355.
- White, K. L., Lysy, H. H., and Holsapple, M. P. 1985b. "Immunosuppression by Polycyclic Aromatic Hydrocarbons. A Structure-Activity Relationship in B6C3F₁ and DBA/2 Mice," *Immunopharmacology* 9:155–164.
- WHO (World Health Organization) 1984a. Guidelines for Drinking Water Quality, Vol. 2, Health Criteria and Other Supporting Information, WHO, Geneva, Switzerland.
- WHO 1984b. Guidelines for Drinking Water Quality. Vol. 1. Recommendations, WHO, Geneva, Switzerland.
- WHO 1973. Trace Elements in Human Nutrition: Manganese. Report of a WHO Expert Committee, pp. 34-36, Technical Report Service, 532, WHO, Geneva, Switzerland.
- Wide, M. 1984. "Effect of Short-Term Exposure to Five Industrial Metals on the Embryonic and Fetal Development of the Mouse," *Environ. Res.* 33:47-53.
- Wiegmann, T. B., Day, H. D., and Patak, R. V. 1982. "Intestinal Absorption and Secretion of Radioactive Vanadium (⁴⁸VO₃) in Rats and Effect of Aluminum Hydroxide," J. Toxicol. Environ. Health 10(2):233-245.
- Wigg, N. R., Vimpani, G. V., McMichael, A. J., et al. 1988. "Port Pirie Cohort Study: Childhood Blood Lead and Neuropsychological Development at Age Two Years," J. Epidemiol. Commun. Health. 42:213-219.
- Williams, P. L. 1982. "Pentachlorophenol, An Assessment of the Occupational Hazard," J. Am. Ind. Hyg. Assoc. 43:799-810.
- Williams, W. J., and Williams, W. R. 1983. "Value of Beryllium Lymphocyte Transformation Tests in Chronic Beryllium Disease and in Potentially Exposed Workers," *Thorax* 38:41-42.
- Wilson, J. T. 1954. "Influence of the Offspring of Altered Physiologic States During Pregnancy in the Rat," Ann. NY Acad. Sci. 57:517-525.
- Winek, A. L., and Collom, W. D. 1971. "Benzene and Toluene Fatalities," J. Occup. Med. 13:259-261.
- Wingren, G., and Axelson, O. 1985. "Mortality Pattern in a Glass Producing Area in S.E. Sweden." British J. Indust. Med. 42:411-414.
- Wintrobe, M. M, Lee, G. R., Boggs, D. R. et al. 1974. Clinical Hematology, 7th ed., Lea and Febiger, Philadelphia, PA.
- Withey, J. R., and Karpinski, K. 1985. "The Fetal Distribution of Some Aliphatic Chlorinated Hydrocarbons in the Rat After Vapor Phase Exposure," *Biol. Res. Pregnancy Perinatol.* 6:79-88.
- Withey, R. J., and Hall, J. W. 1975. "The Joint Toxic Action of Perchloroethylene with Benzene or Toluene in Rats," *Toxic*. 4:5-15.
- Wolkowski-Tyl, R., Jones-Price, C., and Marr, M. C. 1984a. "Teratologic Evaluation of Diethylhexyl Phthalate (CAS No. 117-81-7) in Fischer 344 Rats," Gov. Rep. Announce. Index. 85(2):70.



- Wolkowski-Tyl, R., Jones-Price, C., Marr, M. C., and Kimmel, C. A. 1984b. "Teratologic Evaluation of Diethylhexyl Phthalate (CAS No. 117-81-7) in CD-1 Mice," Gov. Rep. Announce. Index. 85(2):70.
- 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.* 85:1–13.
- Wong, L. C. K., Winston, J. M., Hagensen, J., et al. 1979. Study of Carcinogenicity and Toxicity of Inhaled Antimony Trioxide, Antimony Ore Concentrate and Thallic Oxide in Rats, NIOSH/U.S. Dept. of Health OTS0511065.
- Wood, A. W., Levin, W., Chang, R. L., et al. 1980. "Mutagenicity and Tumor-Initiating Activity of Cyclopenta(c,d)pyrene and Structurally Related Compounds," *Cancer Res.* 40:642-649.
- Wood, A. W., Levin, W., Chang, R. L., et al. 1979. "Mutagenicity and Tumorigenicity of Phenanthrene and Chrysene Epoxides and Diol Epoxides," *Cancer Res.* 39:4069–4077.
- Wood, A. W., Levin, W., Ryan, D., et al. 1977. "High Mutagenicity of Metabolically Activated Chrysene 1,2-Dihydrodiol: Evidence for Bay Region Activation of Chrysene," *Biochem. Biophys. Res. Commun.* 78:847-854.
- Woods, J. S., and Fowler, B. A. 1978. "Altered Regulation of Mammalian Hepatic Heme Biosynthesis and Urinary Porphyrin Excretion During Prolonged Exposure to Sodium Arsenate," *Toxicol. Appl. Pharmacol.* 43:361–371.
- Woods, J. S., and Fowler, B. A. 1977. "Effects of Chronic Arsenic Exposure on Hematopoietic Function in Adult Mammalian Liver," *Environ. Health Perspect.* 19:209-213.
- Wright, W. H., and Schaffer, J. M. 1932. "Critical Anthelmintic Tests of Chlorinated Alkyl Hydrocarbons and a Correlation Between the Anthelmintic Efficacy, Chemical Structure and Physical Properties," Am. J. Hyg. 16:325-426.
- Wu, M. M., Kuo, T. L., Hwang, Y. H., and Chen, C. J. 1989. "Dose-Responsive Relation Between Arsenic Concentration in Well Water and Mortality from Cancers and Vascular Diseases," Am. J. Epidemiol. 130:1123-1132.
- Wynder, E. L., and Hoffman, D. 1959a. "A Study of Tobacco Carcinogenesis. VII. The Role of Higher Polycyclic Aromatic Hydrocarbons," *Cancer* 12:1079-1086.
- Wynder, E. L., and Hoffman, D. 1959b. "The Carcinogenicity of Benzofluoranthene," Cancer 12:1194.
- Yant, W. P., Schrenk, H. H., Waite, C. P., and Patty, F. A. 1930. Acute Response of Guinea Pigs to Vapors of Some New Commercial Organic Compounds. II. Ethylbenzene, Public Health Reports 45:1241-1250.
- Yin, S. -N., Li, G. -L., Tain, F. -D., et al. 1987. "Leukaemia in Benzene Workers: A Retrospective Cohort Study," Br. J. Ind. Med. 44:124-128.
- Yoshikawa, T., Ruhr, L. P., Flory, W., et al. 1985. "Toxicity of Polycyclic Aromatic Hydrocarbons. 1. Effect of Phenanthrene, Pyrene and Their Ozonized Products on Blood Chemistry in Rats," *Toxicol. Appl. Pharmacol.* 79:218-226.
- Zaldivar, R. 1974. "Arsenic Contamination of Drinking Water and Foodstuffs Causing Endemic Chronic Poisoning," Beitr. Path. 151:384-400.
- Zaporowska, H., and Wasilewski, W. 1991. "Significance of Reduced Food and Water Consumption in Rats Intoxicated with Vanadium," Comp. Biochem. Physiol. 99C (3):349-352.

- Zenz, C., and Berg, B. A. 1967. "Human Responses to Controlled Vanadium Pentoxide Exposure," Arch. --Environ. Health 14(5):709-712.
- Zinkham, W. J., and Childs, B. 1958. "A Defect of Glutathione Metabolism in Erythrocytes from Patients with a Naphthalene-Induced Hemolytic Anemia," *Pediatrics* 22:461-471.

Zorn, H., Stiefel, T., and Porcher, H. 1988. "Clinical and Analytical Follow-Up of 25 Persons Exposed Accidentally to Beryllium," pp. 164-171, in *Toxicological and Environmental Chemistry*, Vol. 12, Nos. 3 and 4.

APPENDIX A

TABLES

	Table 1.1.	Assignment of sampling stations by	y location
		LOCATION=AOC 204	
		Sampling	
		station	
	•	204-01	
· .		204-02	
•		204-028	
	·	204-03: 204-030	
		204-031	
		204-04	
		204-13 204-15	
	.•.	204-15	
<u>.</u> .		204-17	
		204-18 204-19	
· ·		204-19	
		204-22	•
			•
		LOCATION=SWMU 193A	
·			
		Sampling	
1		station	
		099-038	
		193-026	
_		193-028	
		193-029 193-030	
	:	193-031	
	,	193-032	
		193-041 .193-049	•
:		MW108	·
		P4-H5	
		P4-H6 P2109	
•		·P2110	
		PZ114	
•		PZ115	
	,	P2117 P2118	
		LOCATION-SWMU 193B	
		Sampling	
		Sampling station	
·		099-037	
	<u>.</u>	099-037 193-022	
	,	193-023	
		193-025	
		P4-G5	•
		LOCATION-SWMU 193C	
		Complete -	
	,	Sampling station	
		193-033	.•
		193-034 193-036	
		T22-036	

A-3

		LOCAT	anntinuedi					
			continued)					
			Sampling					
			station					
			BLALION					
			in in a					
		.	193-038					
			193-039					
			193-1					
			193-10					
			193-11					
			193-12					
		•	193-13					
			193-14					
			193-15					
			193-16					
			193-17		•			
		•	193-18					
			193-19					
			193-2					
		•	193-20	•				
	•	,	193-21	•				
	•		193-3					
			193-4					
			193-5				•	
			193-6					
· ·			TA1-0					
			193-7					
			193-8					
	*		193-9					
			MW121					
		Loca	TION-SWMU 194 Sampling					
		Loca	TION-SWMU 194			a a 16 i - u - i		
		Loca	TION-SWMU 194 Sampling station	•				
		Loca	TION-SWMU 194 Sampling station 194-008					
		Loca	TION-SWMU 194 Sampling station 194-008 194-009					
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010					
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011					
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1					
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2		. [.]			
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2 194-3					
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2 194-3 194-4					
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2 194-3 194-4 194-5		. : . :			
•		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2 194-3 194-4 194-5 194-5 194-6		· · · · · · · · · · · · · · · · · · ·			
•		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2 194-3 194-4 194-5					
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2 194-3 194-4 194-5 194-5 194-6					
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2 194-2 194-3 194-4 194-5 194-6 194-7		· · · · · · · · · · · · · · · · · · · ·			
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2 194-3 194-3 194-5 194-5 194-6 194-7 TION-SWMU 99A					
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2 194-3 194-3 194-5 194-5 194-6 194-7 TION-SWMU 99A					
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2 194-2 194-3 194-4 194-5 194-6 194-7					
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2 194-3 194-4 194-5 194-6 194-6 194-7 TION-SWMU 99A Sampling station		· • • • • • • • • • • • • • • • • • • •			
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-2 194-3 194-4 194-5 194-5 194-6 194-7 TION-SWMU 99A Sampling station 082-014		· • • • • • • • • • • • • • • • • • • •			
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2 194-3 194-4 194-5 194-6 194-6 194-7 TION-SWMU 99A Sampling station 082-014 082-015					
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2 194-3 194-4 194-5 194-6 194-5 194-6 194-7 TION-SWMU 99A Sampling station 082-014 082-015 099-001					
		LOCA	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-2 194-2 194-3 194-4 194-5 194-5 194-6 194-7 TION-SWMU 99A Sampling station 082-014 082-015 099-001 099-003					
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-2 194-3 194-4 194-5 194-6 194-7 TION-SWMU 99A Sampling station 082-014 082-015 099-001 099-003	-	· 			
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-2 194-3 194-3 194-4 194-5 194-6 194-6 194-7 TION-SWMU 99A Sampling station 082-014 082-015 099-001 099-005		· • • • • • • • • • • • • • • • • • • •			
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-2 194-3 194-3 194-4 194-5 194-6 194-6 194-7 TION-SWMU 99A Sampling station 082-014 082-015 099-001 099-005		· • • • • • • • • • • • • • • • • • • •			
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2 194-3 194-4 194-5 194-6 194-6 194-7 TION-SWMU 99A Sampling station 082-014 082-015 099-001 099-003 099-005 099-006		· · · ·			
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-3 194-3 194-5 194-6 194-7 TION-SWMU 99A Sampling station 082-014 082-015 099-001 099-003 099-004 099-005 099-008					
		Loca	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-2 194-3 194-4 194-5 194-6 194-7 TION-SWMU 99A Sampling station 082-014 082-015 099-001 099-003 099-004 099-005 099-008 099-009					
		LOCA	TION-SWMU 194 Sampling station 194-008 194-009 194-010 194-011 194-1 194-3 194-3 194-5 194-6 194-7 TION-SWMU 99A Sampling station 082-014 082-015 099-001 099-003 099-004 099-005 099-008					

Table 1.1. Assignment of sampling stations by location

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A-4

	(continued)		
	Sampling		
	station		
•	099-014		
	099-015		
	099-016		
	099-030		
	099-031	•	
	099-033		
	099-034		
	099-035		
	H217		
. •	H218		
·	MW163		
	MW256		
	P4-E6		
	P4-E7		
	LOCATION-SWMU 99B	 	

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099-019 099-022 099-025 099-029 MW258

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A-5

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Analyte	Subsurface soil background value ⁿ	Surface soil background value [#]	RGA groundwater background value ^b	McNairy groundwater background value ^b
Inorganic chemical (I	ng/kg or mg/L)			
Aluminum	12,000	13,000	2.19	0.687
Antimony	0.21	0.21	0.06	0.06
Arsenic	7.9	12	0.005	0.005
Barium	170	200	0.235	0.296
Beryllium	0.69	0.67	0.004	0.017
Cadmium	0.21	0.21	0.01	0.01
Calcium	6100	200,000	41.2	38.9
Chloride		•	91.0	19.7
Chromium			0.144	0.06
Chromium (III)	43	16		
Chromium (VI)				
Cobalt	13	14	0.045	0.096
Copper	25	19	0.036	0.057
Cyanide (CN-)				
Fluoride	·.	•	0.27	0.33
Iron	28,000	28,000	5.03	18.4
Lead	23	36	0.129	0.05
Magnesium	2100	7700	16.3	13.4
Manganese	820	1500	0.119	0.941
Mercury	0.13	0.2	0.0002	0.0002
Molybdenum			0.05	0.05
Nickel	22	21	0.682	0,109
Nitrate as N				
Nitrate as Nitrogen			15.56	.47
Potassium	950	1300	5.2	55.75
Selenium	0.7	0.8	0.005	0.005
Silica			26.4	26.0
Silver	2.7	2.3	0.011	0.05
Sodium	340	320	59.45	29.2
Sulfate			19:95	28.9
Sulfide				
Thallium	0.34	0.21	0.056	0.644
Tin.				
Uranium	4.6	4.9	0:002	0.001
Vanadium	37	38:	0.134	0.126
Zinc	60	65	0.054	0.142

Table 1.2. Background concentrations of inorganic compounds and radionuclides by media

A-6

Analyte	Subsurface soil background value [*]	Surface soil background value [#]	RGA groundwater background value ^b	McNairy groundwate background value ^b
Radionuclides (pCi/	g or pCi/L)			
Cesium-137	0.28	0.49		
Neptunium-237		0,1	0.8	0:5
Plutonium-238		0.073		
Plutonium-239		0.025	0.1	0.2
Potassium-40	16	16		
Radium-226	1.5	1.5	0.6	1.2
Radon-222			626.0	295.0
Total radium			1.3	0.7
Strontium-90		4.7		
Technetium-99	2.8	2.5	22.3	20.6
Thorium-228	1.6	1.6		
Thorium-230	1.4	1.5	1.1	1.5
Thorium-232	1.5	1.5	•	
Uranium-234	2.4	2.5	0.7	0.3
Uranium-235	0.14	0.14	0.3	0.2
Uranium-238	1.2	1.2	0.7	0.3

Table 1.2. (Continued)

Note: Blank cells indicate data not available or appropriate.

^a Subsurface and surface soil values are from Background Levels of Selected Radionuclides and Metals in Soils and Geologic Media at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky (DOE 1997a).

^b Groundwater values are from *Background Concentrations of Naturally-Occurring Inorganic Chemicals and Selected Radionuclides in the Regional Gravel Aquifer and McNairy Formation* (Bonczek 1999). These values are not used in the development of the list of chemicals of potential concern in the data evaluation portion of the baseline risk assessment because they are being revised. However, the values are used as reference values in the uncertainty analysis.

Table 1.3. Data summary for all analytes by location and medium

----- LOCATION=AOC 204 MEDIA=RGA Groundwater

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	Frequency	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
1,1,1-Trichloroethane	11/11	1.00E-02 - 1.80E-02		N	1.13E-02	mg/L
1,1-Dichloroethane	1/1	5.00E+00 - 5.00E+00		NT	5.00E+00	mg/L
1,1-Dichloroethene	12/15	1.00E-04 - 4.00E-02	1.00E-03 - 1.00E-03	L X	3.25E-02	mg/L
PCB-1254	11/11	2.50E-02 - 2.50E-02	• •	N	2.50E-02	mg/L
PCB-1260	11/11	2.50E-02 - 2.50E-02		N	2.50E-02	mg/L
Polychlorinated biphenyl	1/1	1.70B-01 - 1.70E-01		NT	1.70E-01	mg/L
Tetrachloroethene	11/11	5.00E-03 - 5.00E+00		N	4.59E-01	mg/L
Trichloroethene	15/15	5.00B-03 - 7.70B-01		L	1.41E-01	mg/L
Vinyl Chloride	1/4	1.00E-04 - 1.00E-04	1.00E-03 - 1.00E-03	N	7.75E-04	mg/L
cis-1,2-Dichloroethene	3/4	9.00E-04 - 6.00E-03	1.00E-03 - 1.00E-03	N	3.48E-03	mg/L
trans-1,2-Dichloroethene	2/4	1.00E-04 - 1.00E-04	1.00E=03 = 1.00E-03	N	5.50E-04	mg/L
Alpha activity	2/4	2.40E+00 - 6.80E+00	7.90E-01 - 1.70E+00	N	2.92E+00	pCi/L
Beta activity	2/4	3.40E+00 - 5.20E+00	1.90E+00 - 2.60E+00	N	3.28E+00	pCi/L
Technetium-99	0/4		=4.50E+00 = 7.00E+00	NT	1.58E+00	pCi/L

A-8

----- LOCATION=AOC 204 MEDIA=Subsurface Soil ------

Analyte	Frequency of Detection	Detected Range	Nondetected Ränge	Distribution*	Arithmetic Mean	Units
1,1,1=Trichloroethane	10/16	1.00E-02 ~ 1.00E+00	1.00E-02 - 1.00E-02	L	4.64E-02	mg/kg
1,1,2,2-Tetrachloroethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,1,2-Trichloroethane	0/6		1.00E = 02 = 1.00E = 02	NT	5.00E-03	mg/kg
1,1-Dichloroethane	8/14	1.00E+00 - 1.00E+00	1.00E-02 - 1.00E-02	N	5.74E-01	mg/kg
1,1-Dichloroethene	11/17	4.00E-02 - 4.00E-02	3.36E-01 - 4.27E-01	n Nt	9.35E-02	mg/kg
1,2-Dichloroethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,2-Dichloropropane	0/6		1.00E = 02 = 1.00E = 02	NT	5.00E-03	mg/kg
1,2-Dimethylbenzene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
2-Butanone	0/6	•	1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
2-Hexanone	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
4-Methyl-2-pentanone	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Acetone	0/6		1.00E-02 = 1.00E-02	NT	5.00E-03	mg/kg
Benzene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Bromodichloromethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Bromoform	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Bromomethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Carbon Disulfide	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Carbon Tetrachloride	Ű/Ő		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chlorobenzene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg

*L=Lognormal, N=Normal, NT=Not tested

Table 1.3. Data summary for all analytes by location and medium

CONTINNANC 204 MEDIA-Subsurface Soil ------ (continued)

	Frequency of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Chloroethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E~03	mg/kg
Chloroform	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chloromethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Dibromochloromethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Ethylbenzene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Methylene Chloride	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
PCB-1254	11/11	2.50E-02 - 2.50E-02		N	2.50E-02	mg/kg
PCB-1260	11/11	2.50E-02 - 2.50E-02		N	2.50E-02	mg/kg
Polychlorinated biphenyl	8/8	1.00E-01 - 1.00E-01		N	1.00E-01	mg/kg
Styrene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Tetrachloroethene	11/17	5.00E-03 - 1.00E+00	1.00E-02 - 1.00E-02	N	4.73E-01	mg/kg
Toluene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Trichloroethene	11/17	5.00E-03 - 1.00E+00	3.36E-01 - 4.27E-01	L	1.67E-01	mg/kg
Vinyl Chloride	0/6		3.65E-01 - 1.00E+01	NT	1.99E+00	mg/kg
cis-1,2-Dichloroethene	0/6		3.36E-01 - 4.27E-01	NT	3.83E-01	mg/kg
cis-1,3-Dichloropropene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
m,p-Xylene	0/6		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/kg
trans-1,2-Dichloroethene	0/6		3.36E-01 - 4.27E-01	NT	3.83E-01	mg/kg
trans-1,3-Dichloropropene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Alpha activity	6/6	9.50E+00 - 1.96E+01		ท	1.53E+01	pCi/g
Americium-241	0/6		4.50E+00 - 7.70E+00	NT	6.40E+00	pCi/g
Beta activity	6/6	1.71E+01 - 2.91E+01		N	2.27E+01	pCi/g
Cesium-137	0/6		7.00E-01 - 2.90E+00	NT	1.44E+00	pCi/g
Cobalt-60	0/6		9.50E-01 - 1.30E+00	NT	1.13E+00	pCi/g pCi/g
Protactinium-234m	0/5		1.30E+02 - 4.50E+02	NT	2.00E+02	pCi/g
Technetium-99	0/6		0.00E+00 - 5.10E-01	NT	1.52E-01	
Thorium-234	0/6		9.80E+00 - 1.80E+01	NT	1.43E+01	pCi/g
Uranium-235	0/6		2.10E+00 - 7.70E+00	NT	4.90E+00	pCi/g pCi/g

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----- LOCATION=SWMU 193A MEDIA=McNairy Groundwater -----

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
Calcium	4/4	1.30E+01 - 8.21E+01		N	3.53E+01	mg/L
Chloride	4/4	8.00E+00 - 1.60E+01		N	1.25E+01	mg/L
Iron	4/4	2.89E+00 - 3.14E+02		N	8.36E+01	mg/L
Magnesium	4/4	5.33E+00 = 4.02E+01		Ń	1.46E+01	mg/L

Table 1.3. Data summary for all analytes by location and medium

----- LOCATION=SWMU 193A MEDIA=McNairy Groundwater -----(continued)

	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Potassium	4/4	4.07E+00 - 2.15E+01		N	9.02E+00	.mg/L
Sođium	4/4	1.64E+01 - 8.08E+01		N	4.13E+01	mg/L
Tetraoxo-sulfate(1-)	4/4	2.10E+01 - 8.40E+01		N	5.05E+01	mg/L
1,1,1-Trichloroethane	0/5		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
1,1,2,2-Tetrachloroethane	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
1,1,2-Trichloroethane	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
1,1-Dichloroethane	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
1,1-Dichloroethene	0/11		1.00E-03 - 1.00E-02	NT	2.32E-03	mg/L
1,2,4-Trichlorobenzene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
1,2-Dichlorobenzene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
1,2-Dichloroethane	0/5		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
1,2-Dichloropropane	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
1,2-Dimethylbenzene	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
1,3-Dichlorobenzene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
1,4-Dichlorobenzene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
2,4,5-Trichlorophenol	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
2,4,6-Trichlorophenol	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
2,4-Dichlorophenol	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
2,4-Dimethylphenol	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
2,4-Dinitrophenol	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
2,4-Dinitrotoluene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
2,6-Dinitrotoluene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
2-Butanone	0/1		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/L
2-Chloronaphthalene	0/6	· ·	1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
2-Chlorophenol	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
2-Hexanone	0/1		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/L
2-Methyl-4,6-dinitrophenol	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
2-Methylnaphthalene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
2-Methylphenol	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
2-Nitroaniline	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
2-Nitrophenol	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
3,3'-Dichlorobenzidine	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
3-Nitroaniline	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
4-Bromophenyl phenyl ether	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
4-Chloro-3-methylphenol	0/6		1.00E-02 - 4.00E-02.	NT	8.42E-03	
4-Chloroaniline	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
4-Chlorophenyl phenyl ether	0/6		1.00E-02 - 4.00E-02	NT		mg/L
4-Methyl-2-pentanone	0/1		1.00E-02 - 1.00E-02	NT	8.42E-03	mg/L
4-Methylphenol	0/6		1.00E-02 - 4.00E-02	NT	5.00E-03	mg/L
4-Nitroaniline	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
				IN I	8.42E-03	mg/L

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-	LOCATION=SWMU	193A	MEDIA=McNairy	Groundwater	
		(c	ontinued)		

	Frequency	Detected			.	
Analyte	Detection	Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
4-Nitrophenol	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Acenaphthene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Acenaphthylene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Acetone	1/1	1.40E-02 - 1.40E-02		NT	1.40E-02	mg/L
Anthracene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-02	mg/L
Benz (a) anthracene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Benzene	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Benzo (a) pyrene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Benzo (b) fluoranthene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Benzo (ghi) perylene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Benzo(k)fluoranthene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Bromodichloromethane	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Bromoform	0/1	·	5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Butyl benzyl phthalate	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Carbazole	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Carbon Disulfide	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Carbon Tetrachloride	0/5		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Chlorobenzene	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Chloroethane	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Chloroform	0/1		5.00E~03 - 5.00E-03	NT	2.50E-03	mg/L
Chloromethane	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Chrysene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Di-n-butylphthalate	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Di-n-octylphthalate	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Dibenz (a, h) anthracene	0/6		1.00E - 02 - 4.00E - 02	NT	8.42E-03	mg/L
Dibenzofuran	0/6		1.00E-02 - 4.00E-02	NT	8.42Ë-03	mg/L
Dibromochloromethane	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Diethylphthalate	1/6	1.90E-02 - 1.90E-02	1.00E-02 - 4.00E-02	Ň	1.04E-02	mg/L
Dimethylphthalate	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Ethylbenzene	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Fluoranthene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Fluorene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Hexachlorobenzene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	
Hexachlorobutadiene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Hexachlorocyclopentadiene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Hexachloroethane	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Indeno (1,2,3-cd) pyrene	0/6		1.00E-02 - 4.00E-02	NT		mg/L
Isophorone	0/6		1.00E-02 = 4.00E-02	NT	8.42E-03 8.42E-03	mg/L
Methylene Chloride	0/1		1.00E-02 - 1.00E-02	NT	8.42E-03 5.00E-03	mg/L
N-Nitroso-di-n-propylamine	0/6		1.00E-02 - 4.00E-02	NT		mg/L
	-, -		1.000 02 - 4.000-02	NI	8.42E-03	mg/L

*L=Lognormal, N=Normal, NT=Not tested

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 LOCATION=SWMU	193A	MEDIA=McNairy	Groundwater	÷
	(c	ontinued)		

	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
N-Nitrosodiphenylamine	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Naphthalene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Nitrobenzene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Pentachlorophenol	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Phenanthrene	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
Phenol	0/6		1.00B-02 - 4.00B-02	NT	8.42E-03	mg/L
Pyrene	0/6		1.00E-02 - 4.00E-02	NT	8.42E=03	mg/L
Pyridine	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Styrene	0/1		5,00E-03 - 5,00E-03	NT	2.50E-03	mg/L
Tetrachloroethene	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Toluene	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Trichloroethene	8/13	2.00E-04 - 1.10E-02	1.00E=03 = 1.00E=03	L	1.70E-03	mg/L
Vinyl Chloride	0/7		1.00E-03 - 5.00E-03	NT	1.57E-03	mg/L
bis (2-Chloroethoxy) methane	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
bis(2-Chloroethyl)ether	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
bis(2-Chloroisopropyl)ether	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
bis(2-Ethylhexyl)phthalate	0/6		1.00E-02 - 4.00E-02	NT	8.42E-03	mg/L
cis-1,2-Dichloroethene	1/11	1.70E-01 - 1.70E-01	1.00E-03 - 2.00E+00	L	6.85E+00	mg/L
cis-1,3-Dichloropropene	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
m,p-Xylene	0/1		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/L
trans-1,2-Dichloroethene	0/11		1.00E-03 - 2.00E+00	NT	7.28E-01	mg/L
trans=1,3-Dichloropropene	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Alpha activity	6/10	2.40E+00 - 4.00E+01	2.90E-01 - 1.40E+00	L	4.44E+00	pCi/L
Beta activity	8/10	5.10E+00 - 6.46E+01	1.00E+00 - 4.40E+00	Ŀ	2.13E+01	pCi/L
Neptunium-237	0/1		-2.00E-022.00E-02	NT	-2.00E-02	pCi/L
Plutonium-239	0/1		-6.00E-026.00E-02	NT	-6.00E-02	pCi/L
Technetium-99	5/10	1.00E+01 = 1.45E+02	4.00E+00 - 8.50E+00	Ĺ	1.38E+01	pCi/L
Thorium-234	1/1	8.40E-01 - 8.40E-01		NT	8.40Ē-01	pCi/L
Uranium-234	1/1	8.10E-01 - 8.10E-01		NT	8.10E-01	pCi/L
Uranium-238	1/1	1.32E+00 - 1.32E+00		NT	1.32E+00	pCi/L

LOCATION-SWMU 193A MEDIA-RGA Groundwater

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmétic Mean	Units
Aluminum	4/4	1.29E-01 - 1.78E-01		N	1.43E-01	mg/L
Ammonia	1/1	3.00E-01 - 3.00E-01		NT	3.00E-01	mg/L

*L=Lognormal, N=Normal, NT=Not tested

----- LOCATION-SWMU 193A MEDIA-RGA Groundwater ------ (continued)

Analyte	of	Detected	No			
Applato		Derected	Nondetected		Arithmetic	
Allalyce	Detection	Range	Range	Distribution*	Mean	Units
Calcium	5/5	2.62E+01 - 1.34E+02		N	5.96E+01	mg/L
Chloride	5/5	1.30E+01 - 6.40E+01		Ń	3.10E+01	mg/L
Chromium	0/4		5.00E-02 - 5.00E-02	NT	2.50E-02	mg/L
Copper	1/4	1.80E-02 - 1.80E-02	1.00E-02 - 1.00E-02	N	8.25E-03	mg/L
Fluoride	1/1	4.20E-01 - 4.20E-01	-	NT	4.20E-01	mg/L
Iron	7/9	2.00E-02 - 3.66E+01	1.00E-02 - 1.00E-02	L	2.93E+00	mg/L
Magnesium	5/5	3.91E+00 - 1.85E+01		N	1.05E+01	mg/L
Ničkel	0/4		5.00E-02 - 5.00E-02	NT	2.50E-02	mg/L
Potassium	5/5	2.66E+00 - 2.65E+02		N	6.10E+01	mg/L
Silica	1/1	1.90B+01 - 1.90E+01		NT	1.90E+01	mg/L
Sodium	5/5	3.50E+01 - 1.34E+02		N	7.80E+01	mg/L
Sulfide	0/1		1.00E+00 - 1.00E+00	NT	5.00E-01	mg/L
Tetraoxo-sulfate(1-)	5/5	2.10E+01 - 2.62E+02		N .	1.12E+02	mg/L
Uranium	0/4		1.00E-03 - 1.00E-03	NT	1.00E-03	mg/L
Zinc	4/4	7.205-02 - 2.125-01		N	1.21E-01	mg/L
1,1,1-Trichloroethane	0/16		5.00E-03 - 5.00E+00	NT	1.90E-01	mg/L
1,1,2,2-Tetrachloroethane	0/3		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
1,1,2-Trichloroethane	0/10		5.00E-03 - 2.50E-01	NT	5.25E-02	mg/L
1,1-Dichloroethane	0/10		5.00E-03 - 2.50E-01	NT	5.25E-02	mg/L
1,1-Dichloroethene	2/43	1.00E-04 - 2.00E-04	1.00E-03 - 5.00E+00	L	1.68E-04	mg/L
1,2,4-Trichlorobenzene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
1,2-Dichlorobenzene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
1,2-Dichloroethane	0/16		5.00E-03 - 5.00E+00	NT	1.90E-01	mg/L
1,2-Dichloropropane	0/3		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
1,2-Dimethylbenzene	0/3		5.00E-03 - 5.00 <u>B</u> -03	NT	2.50E-03	mg/L
1,3-Dichlorobenzene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
1,4-Dichlorobenzene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
2,4,5-Trichlorophenol	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
2,4,6-Trichlorophenol	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
2,4-Dichlorophenol	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
2,4-Dimethylphenol	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
2,4-Dinitrophenol	0/6		1.00E-02 - 2.00E-02	NT	6.75E-03	mg/L
2,4-Dinitrotoluene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
2,6-Dinitrotoluene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
2-Butanone	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/L
2-Chloronaphthalene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
2-Chlorophenol	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
2-Hexanone	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/L
2-Methyl-4,6-dinitrophenol	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
2-Methylnaphthalene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mġ/L

 LOCATION=SWMU	193A MEDI	A=RGA G	Groundwater	
	(continue	ed)		

	Frequency	.				
Prove 2 rest	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
2-Methylphenol	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
2-Nitroaniline	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
2-Nitrophenol	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
3,3'-Dichlorobenzidine	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
3-Nitroaniline	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
4-Bromophenyl phenyl ether	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
4-Chloro-3-methylphenol	0/25		1.00E-02 - 4.00E-02	NT	7.508-03	mg/L
4-Chloroaniline	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
4-Chlorophenyl phenyl ether	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
4-Methyl-2-pentanone	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/L
4-Methylphenol	0/25		1.00E-02 ~ 4.00E-02	NT	7.50E-03	mg/L
4-Nitroaniline	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
4-Nitrophenol	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Acenaphthene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Acenaphthylene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Acetone	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/L
Anthracene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Benz (a) anthracene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Benzene	0/10		5.00E-03 ~ 2.50E-01	NT	5.25E-02	mg/L
Benzo (a) pyrene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Benzo(b)fluoranthene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Benzo (ghi) perylene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Benzo(k)fluoranthene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Bromodichloromethane	0/10		5.00E-03 - 2.50B-01	NT	5.25E-02	mg/L
Bromoform	0/3		5.00E-03 - 5.00E-03	NT ·	2.50E-03	mg/L
Butyl benzyl phthalate	0/6		1.00E-02 - 2.00E-02	NT	6.75E-03	mg/L
Carbazole	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Carbon Disulfide	0/3		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Carbon Tetrachloride	0/16		5.00B-03 - 5.00E+00	NT	1.90E-01	mg/L
Chlorobenzene	0/3		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Chloroethane	0/4		5.00E-03 - 2.50E-01	NT	3.31E-02	mg/L
Chloroform	0/12		5.00E-03 - 5.00E+00	NT	2.52E-01	mg/L
Chloromethane	0/3		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Chrysene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Di-n-butylphthalate	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Di-n-octylphthalate	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Dibenz(a,h)anthracene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Dibenzofuran	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Dibromochloromethane	0/3		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Diethylphthalate	3/25	9.00E-03 - 1.50E-02	1.00E-02 ~ 4.00E-02	L	9.17E-03	
	•			-	2.1/2-03	mg/L

*L=Lognormal, N=Normal, NT=Not tested

-- LOCATION=SWMU 193A MEDIA=RGA Groundwater ------(continued)

	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Dimethylphthalate	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Ethane	0/1		3.00E=02 - 3.00E-02	NT	1.50E-02	mg/L
Ethylbenzene	0/10		5.00E-03 - 2.50E-01	NT	5.25E-02	mg/L
Ethylene	0/1		3.00E-02 - 3.00E-02	NT	1.50E-02	mg/L
Fluoranthene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Fluorene	0/25	•	1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Hexachlorobenzene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Hexachlorobutadiene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Hexachlorocyclopentadiene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Hexachloroethane	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Indeno(1,2,3-cd)pyrene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Isophorone	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Methylene Chloride	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/L
N-Nitroso-di-n-propylamine	0/25		1.00E-02 - 4.00E-02	NT	7.508-03	mg/L
N-Nitrosodiphenylamine	0/25	•	1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Naphthalene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Nitrobenzene	0/25		1.00E-02 - 4.00E-02	NT	7.502-03	mg/L
Pentachlorophenol	1/25	1.20E-02 - 1.20E-02	1.00E-02 - 4.00E-02	L	7.74E-03	mg/L
Phenanthrene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
Phenol	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	
Pyrene	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L mg/L
Pyridine	0/2		5.00E-03 - 1.00E-02	NT	3.75E-03	
Styrene	0/3		5.00B-03 - 5.00E-03	NT	2.50E-03	mġ∕L
Tetrachloroethene	0/10		5.00E-03 - 2.50E-01	NT	5.25E-02	mg/L
Toluene	0/10		5.00E-03 - 2.50E-01	NT	5.25E-02	mg/L
Trichloroethene	44/51	2.00E-04 - 6.70E+00	1.00E-03 - 1.00E-03	L	7.76E-02	mg/L
Vinyl Chloride	0/39		1.00E-03 - 1.00E+01	NT	3.04E-01	mg/L
Xylene	0/7		1.00E-02 - 5.00E-01	NT	1.30E-01	mg/L
bis (2-Chloroethoxy) methane	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
bis(2-Chloroethyl)ether	0/25		1.00E-02 - 4.00E-02	NT	7.50E-03	mg/L
bis(2-Chloroisopropyl)ether	0/25		1.00E-02 - 4.00E-02	NT		mg/L
bis(2-Ethylhexyl)phthalate	3/25	1.30E-02 - 2.20E-02	1.00E-02 - 4.00E-02	L	7,50E-03	mg/L
cis-1,2-Dichloroethene	17/42	1.00E-04 - 8.40E-02	1.00E-03 - 5.00E+00	L	7.94E-03	mg/L
cis-1,3-Dichloropropene	0/3	1.001 04 - 0.405-02	5.00E-03 - 5.00E-03	NT	1.51E-03	mg/L
m,p-Xylene	0/3		1.00E-02 - 1.00E-02	NT	2.50E-03	mg/L
trans-1,2-Dichloroethene	7/43	1.00E-04 - 7.00E-04	1.00E-02 - 1.00E-02 1.00E-03 - 5.00E+00		5.00E-03	mg/L
trans-1, 3-Dichloropropene	0/3	1.001-04 - 7.001-04		L	3.07E-04	mg/L
Alpha activity	19/34	1.50E+00 - 1.76E+01	5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Beta activity	34/34	2.90E+00 - 8.80E+02	-4.10E-01 - 4.00E+00	N	3.64E+00	pCi/L
Neptunium-237	0/8	2.90E+00 - 8.80E+02	-1 COE:01 - 2 025	L	8.53E+01	pCi/L
are proverie dim # # 2 /	0/8		-1.60E+01 - 2.92E+01	NT	1.96E+00	pCi/L

*L=Lognormal, N=Normal, NT=Not tested

 LOCATION=SWMU	193A MEDIA=RGA (continued)	Groundwater	
	(continued)		

	Frequency	Detected	Nondetected		Arithmétic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Plutonium-239	0/4		2.10E-011.00E-02	NT	-1.10E-01	pCi/L
Plutonium-239/240	0/4		-4.00E-02 - 8.00E-02	NT	1.00E-02	pCi/L
Technetium-99	26/39	8.00E+00 - 1.39E+03	-7.60E+00 = 1.70E+01	Ň	1.21E+02	pCi/L
Thorium-234	1/8	5.40E-01 - 5.40E-01	-1.65E+02 - 5.58E+01	N	-2.51E+01	pCi/L
Uranium-234	0/2		1.77E+01 - 2.99E+01	NT	2.38E+01	pCi/L
Uranium-238	0/2		0.00E+00 - 0.00E+00	NT	0.00E+00	pCi/L

----- LOCATION-SWMU 193A MEDIA-Subsurface Soil ------

A-	Analyte	Frequency of Detection	Detected Range	Nondëtected Range	Distribution*	Arithmetic Mean	Units
16	Aluminum	8/8	3.01E+03 - 1.40E+04		N	9.71E+03	mg/kg
	Antimony	0/8		2.00E+01 = 2.00E+01	NT	1.00E+01	mg/kg
	Arsenic	0/8		5.00E+00 - 5.00E+00	NT	2.50E+00	mg/kg
	Barium	8/8	2.16E+01 - 8.73E+01		N	5.66E+01	mg/kg
	Beryllium	5/8	5.20E-01 - 7.00E-01	5.00E-01 - 5.00E-01	N	4.60E-01	mg/kg
	Boron	0/8		1.00E+02 - 1.00E+02	NT	5.00E+01	mg/kg
	Cadmium	0/8		2.00E+00 - 2.00E+00	NT	1.00Ë+00	mg/kg
	Calcium	4/4	1.06E+03 - 2.73E+05		N	9.07E+04	mg/kg
	Chromium	8/8	4.31E+00 - 2.77E+01		N	1.65E+01	mg/kg
	Cobalt	8/8	1.47E+00 - 8.66E+00		Ĺ	4.13E+00	mg/kg
	Copper	8/8	2.45B+00 = 7.31E+00		N	4.97E+00	mg/kg
	Cyanide	0/8		1.00E+00 - 1.00E+00	NT	5.00E-01	mg/kg
	Iron	8/8	3.74E+03 - 1.54E+04	• 1	N	1.11E+04	mg/kg
	Lead	0/8		2.00E+01 - 2.00E+01	NT	1.00E+01	mg/kg
	Lithium	8/8	3.78E+00 - 1.12E+01		N	7.14E+00	mg/kg
	Magnesium	8/8	1.16E+03 - 1.70E+04		L	3.92E+03	mg/kg
	Manganese	8/8	4.86E+01 - 5.64E+02		Ĺ	2.30E+02	mg/kg
	Mercury	0/8		2.00E-01 - 2.00E-01	NT	1.00E-01	mg/kg
	Nickel	5/8	5.50E+00 - 9.16E+00	5.00E+00 - 5.00E+00	N	5.42E+00	mg/kg
	Potassium	8/8	2.89E+02 - 1.44E+03		Ĺ	5.27E+02	mg/kg
	Selenium	0/8		1.00E+00 - 5.00E+00	NT	1.50E+00	mg/kg
	Silver	1/8	4.00E+00 - 4.00E+00	4.00E+00 - 4.00E+00	N	2.25E+00	mg/kg
	Sodium	5/8	2.13E+02 - 3.13E+02	2.00E+02 - 2.00E+02	N	1.94E+02	mg/kg
	Strontium	8/8	6.36E+00 - 2.53E+02		L	7.47E+01	mg/kg
	Thallium	0/B		1.50E+01 = 1.50E+01	NT	7.50E+00	mg/kg
	Vanadium	8/8	5.67E+00 - 3.15E+01		Ń	2.18E+01	mg/kg

*L=Lognormal, N=Normal, NT-Not tested

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..... LOCATION-SWMU 193A MEDIA-Subsurface Soil ----- (continued)

	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Zinc	8/8	1.84E+01 - 5.54E+01		L	3.45E+01	mg/kg
1,1,1-Trichloroethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,1,2,2-Tetrachloroethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,1,2-Trichloroethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,1-Dichloroethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,1-Dichloroethene	0/8		1.68E-01 - 4.40E-01	NT	1.52E-01	mg/kg
1,2,4-Trichlorobenzene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
1,2-Dichlorobenzene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
1,2-Dichloroethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,2-Dichloropropane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,2-Dimethylbenzene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,3-Dichlorobenzene	0/8		5.00E-01 - 5,00E-01	NT	2.50E-01	mg/kg
1,4-Dichlorobenzene	. 0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4,5-Trichlorophenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4,6-Trichlorophenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4-Dichlorophenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4-Dimethylphenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4-Dinitrophenol	0/6		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4-Dinitrotoluene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,6-Dinitrotoluene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Butanone	0/1		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
2-Chloronaphthalene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Chlorophenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Hexanone	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
2-Methyl-4,6-dinitrophenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Methylnaphthalene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Methylphenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Nitroaniline	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Nitrophenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
3,3'-Dichlorobenzidine	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
3-Nitroaniline	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kq
4-Bromophenyl phenyl ether	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Chloro-3-methylphenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Chloroaniline	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Chlorophenyl phenyl ether	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Methyl-2-pentanone	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
4-Methylphenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Nitroaniline	0/8	Ŷ	5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Nitrophenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Acenaphthene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
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--- LOCATION-SWMU 193A MEDIA-Subsurface Soil ----- (continued)

	Frequency of Detection	Detected	Nondetected Range	Distribution*	Arithmetic Mean	Units
Analyte	Detection	Range	Range	DISCIDUCION	Mean	UNILS
Acenaphthylene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Acetone	1/2	1.10E-02 - 1.10E-02	1.00E-02 - 1.00E-02	N	8.00E-03	mg/kg
Anthracene	1/8	1.16E-01 - 1.16E-01	5.00E-01 - 5.00E-01	N	2.33E-01	mg/kg
Benz(a) anthracene	2/8	1.60E-01 - 1.80E-01	5.00E-01 - 5.00E-01	L	1.75E-01	mg/kg
Benzene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Benzo (a) pyrene	2/8	2.40E-01 - 2.50E-01	5.00E-01 - 5.00E-01	. N	2.49E-01	mg/kg
Benzo (b) fluoranthene	2/8	3.90E-02 - 5.10E-02	5.00E-01 - 5.00E-01	L	4.77E-02	mg/kg
Benzo (ghi) perylene	2/8	1.66E-01 - 1.70E-01	5.00E-01 - 5.00E-01	L	1.69E-01	mg/kg
Benzo(k) fluoranthene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Bromodichloromethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Bromoform	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Bromomethane	0/6		1.00E-02 ~ 1.00E-02	NT	5.00E-03	mg/kg
Butyl benzyl phthalate	0/6		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Carbazole	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Carbon Disulfide	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Carbon Tetrachloride	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chlorobenzene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chloroethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chloroform	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chloromethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chrysene	2/8	1.70E-01 - 1.70E-01	5.00E-01 - 5.00E-01	N	2.30E-01	mg/kg
Di-n-buty1phthalate	1/8	7.70E-02 - 7.70E-02	5.00E-01 - 6.60E-01	N	2.38E-01	mg/kg
Di-n-octylphthalate	1/8	1.20E-01 - 1.20E-01	5.00E-01 - 5.00E-01	N	2.34E-01	mg/kg
Dibenz(a, h) anthracene	1/8	1.30E-01 - 1.30E-01	5.00E-01 - 5.00E-01	N	2.35E-01	mg/kg
Dibenzofuran	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Dibromochloromethane	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Diethylphthalate	1/8	4.00B-01 - 4.00E-01	5.00E-01 - 5.00E-01	N	2.69E-01	mg/kg
Dimethylphthalate	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Ethylbenzene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Fluoranthene	2/8	2.30E-01 - 3.10E-01	5.00E-01 - 5.00E-01	L	2.88E-01	mg/kg
Fluorene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorobenzene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorobutadiene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorocyclopentadiene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachloroethane	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Indeno(1,2,3-cd)pyrene	2/8	1.38E-01 - 1.60E-01	5.00E-01 - 5.00E-01	L	1.54E-01	mg/kg
Isophorone	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Methylene Chloride	0/6		1.00E-02 - 1.20E-02	NT	5.17E-03	mg/kg
N-Nitroso-di-n-propylamine	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
N-Nitrosodiphenylamine	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg

*L=Lognormal, N=Normal, NT=Not tested

(continued)

	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Naphthalene	0/8		5.00E-01 - 5.00E-01	NT	2.508-01	mg/kg
Nitrobenzene	0/8		5.00E-01 - 5.00E-01	NT	2.505-01	mg/kg
PCB-1016	0/8		1.01E-01 - 1.24E-01	NT	1.12E-01	mg/kg
PCB-1221	0/8		1.01E-01 - 1.24E-01	NT	1.12E-01	mg/kg
PCB-1232	0/8		1.01E-01 - 1.24E-01	NT	1.12E-01	mg/kg
PCB-1242	0/8		1.01E-01 - 1.24E-01	NT	1.12E-01	mg/kg
PCB-1248	0/8		1.01E-01 - 1.24E-01	NT	1.12E-01	mg/kg
PCB-1254	0/8		1.01E-01 - 1.24E-01	NT	1.12E-01	mg/kg
PCB-1260	0/8		1.01E-01 - 1.24E-01	NT	1.12E-01	mg/kg
PCB-1268	0/2		1.00E-01 - 1.00E-01	NT	1.00E-01	mg/kg
Pentachlorophenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Phenanthrene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Phenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Polychlorinated biphenyl	0/2		1.00E-01 - 1.00E-01	NT	1.00E-01	mg/kg
Pyrene	2/8	2.40E-02 - 2.95E-01	5.00E-01 - 5.00E-01	N	2.27E-01	mg/kg
Pyridine	0/1		3.80E-01 - 3.80E-01	NT	1.90E-01	mg/kg
Styrene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Tetrachloroethene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Toluene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Trichloroethene	0/8		1.68E-01 - 4.40E-01	NT	3.04E-01	mg/kg
Vinyl Chloride	0/8		1.68E-01 - 1.00E+02	NT	1.28E+01	mg/kg
bis (2-Chloroethoxy) methane	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
bis(2-Chloroethyl)ether	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	
bis (2-Chloroisopropyl) ether	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
bis (2-Ethylhexyl) phthalate	2/8	8.10E-02 - 1.70E-01	5.00E-01 - 5.00E-01	N	2.19E-01	mg/kg
cis-1,2-Dichloroethene	0/8		1.68E-01 - 5.00E-01	NT	3.28E-01	mg/kg
cis-1,3-Dichloropropene	0/6		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
m,p-Xylene	0/6		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/kg
trans-1,2-Dichloroethene	0/8		1.68E-01 - 5.00E-01	NT	3.28E-01	mg/kg
trans-1,3-Dichloropropene	0/6		1.00E-02 - 1.00E-02	NT		mg/kg
Alpha activity	8/8	9.60E+00 - 2.60E+01	1.000-02 - 1.000-02	L	5.00E-03	mg/kg
Americium-241	0/8	21002100 21002101	1.80E+00 - 9.40E+00	NT	1.62E+01	pCi/g
Beta activity	8/8	1.41E+01 - 2.37E+01	1.802+00 - 9.402+00		5.91E+00	pCi/g
Cesium-137	0/8	1.410+01 * 2.576+01	6.10E-01 - 1.10E+00	L NT	1.90E+01	pCi/g
Cobalt-60	0/8		8.30E-01 - 4.00E+00		8.40E-01	pCi/g
Protactinium-234m	0/8		1.10E+02 - 5.80E+02	NT	1.51E+00	pCi/g
Technetium-99	0/8			NT	2.04E+02	pCi/g
Thorium-234	0/8		0.00E+00 - 1.37E+00	NT	5.64E-01	pCi/g
Uranium-235	0/8		5.20E+00 - 2.10E+01	NT	1.33E+01	pCi/g
	0,0		2.00E+00 - 8.20E+00	NT	5.14E+00	pCi/g

----- LOCATION-SWMU 193A MEDIA-Surface Soil -----

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	Frequency of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Aluminum	4/4	3.01E+03 - 1.09E+04		N	7.24E+03	mg/kg
Antimony	0/4		2.00E+01 - 2.00E+01	NT	1.00E+01	mg/kg
Arsenic	0/4		5.00E+00 - 5.00E+00	NT	2.50E+00	mg/kg
Barium	4/4	2.16E+01 - 8.40E+01		N	5.34E+01	mg/kg
Beryllium	1/4	6.40E-01 - 6.40E-01	5.00E-01 - 5.00E-01	N	3.48E-01	mg/kg
Boron	0/4		1.00E+02 - 1.00E+02	NT	5.00E+01	mg/kg
Cadmium	0/4		2.00E+00 - 2.00E+00	NT	1.00E+00	mg/kg
Calcium	2/2	8.76E+04 - 2.73E+05		N	1.80E+05	mg/kg
Chromium	4/4	4.31E+00 - 2.65E+01		N	1.29E+01	mg/kg
Cobalt	4/4	1.47E+00 - 5.70E+00		N	3.36E+00	mg/kg
Copper	4/4	2.45E+00 - 7.31E+00		N	5.32E+00	mg/kg
Cyanide	0/4	2.100+00 7.010+00	1.00E+00 - 1.00E+00	NT	5.00E-01	mg/kg
Iron	4/4	3.74E+03 - 1.54E+04	2.002.00 2.00400	N	9.39E+03	mg/kg
	0/4	J./48+03 - 1.J48+04	2.00E+01 - 2.00E+01	NT	1.00E+01	mg/kg
Lead Lithium	4/4	3.78E+00 - 1.12E+01	2.006+01 - 2.006+01	N	6.84E+00	mg/kg
Magnesium	4/4	1.66E+03 - 1.70E+04		N	6.91E+03	mg/kg
Maganèse	4/4	1.35E+02 - 3.98E+02		N	2.14E+02	mg/kg
-	0/4	1.552402 - 5.588402	2.00E-01 - 2.00E-01	NT	1.00E-01	mg/kg
Mercury Nickel	2/4	7.27E+00 - 7.50E+00	5.00B+00 - 5.00E+00	N	4.94E+00	mg/kg
Potassium	4/4	2.89E+02 - 1.44E+03	3.008+00 - 3.008+00	N	6.78E+02	mg/kg
Selenium	0/4	2.096402 - 1.346403	1.00E+00 - 5.00E+00	NT	1.50E+00	
Silver	0/4		4.00E+00 - 4.00E+00	NT	2.00E+00	mg/kg
Sodium	1/4	2.13E+02 - 2.13E+02	2.00E+02 - 2.00E+02	N	1.28E+02	mg/kg
Strontium	4/4	1.21E+01 - 2.53E+02	2.008+02 - 2.008+02	N		mg/kg
	0/4	1.216+01 - 2.536+02	1.50E+01 - 1.50E+01	NT	1.22E+02	mg/kg
Thallium	•	E 678.00 3 158.01	1.506+01 - 1.506+01	N	7.50E+00	mg/kg
Vanadium	4/4	5.67E+00 - 3.15E+01		_	1.76E+01	mg/kg
Zinc	4/4	3.34E+01 - 5.54E+01		N	4.64E+01	mg/kg
1,1,1-Trichloroethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,1,2,2-Tetrachloroethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,1,2-Trichloroethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,1-Dichloroethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,1-Dichloroethene	0/4		1.68E-01 - 3.07E-01	NT	1.27E-01	mg/kg
1,2,4-Trichlorobenzene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
1,2-Dichlorobenzene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
1,2-Dichloroethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kc
1,2-Dichloropropane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,2-Dimethylbenzene	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,3-Dichlorobenzene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
1,4-Dichlorobenzene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4,5-Trichlorophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4,6-Trichlorophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg

*L=Lognormal, N=Normal, NT=Not tested

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 LOCATION=SWMU	1 93A	MEDIA=Surface	Soil	
	cont	inued)		

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	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
2,4-Dichlorophenol	0/4		5.00E+01 - 5.00E-01	NT	2.50Ĕ-01	mg/kg
2,4-Dimethylphenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4-Dinitrophenol	0/3		5.00E-01 - 5.00E-01	ŇT	2.50E-01	mg/kg
2,4-Dinitrotoluene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,6-Dinitrotoluene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Chloronaphthalene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Chlorophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Hexanone	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
2-Methyl-4,6-dinitrophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Methylnaphthalene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Methylphenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Nitroaniline	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Nitrophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
3,3'-Dichlorobenžidine	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
3-Nitroaniline	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Bromophenyl phenyl ether	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Chloro-3-methylphenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Chloroaniline	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Chlorophenyl phenyl ether	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Methyl-2-pentanone	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
4-Methylphenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Nitroaniline	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Nitrophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Acenaphthene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Acenaphthylene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Anthracene	1/4	1.16E-01 - 1.16E-01	5.00E-01 - 5.00E-01	N	2.17E-01	mg/kg
Benz (a) anthracene	2/4	1.60E-01 - 1.80E-01	5.00E-01 - 5.00E-01	Ň	2.10E-01	mg/kg
Benzene	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Benzo (a) pyrene	2/4	2.40E-01 - 2.50E-01	5.00E-01 - 5.00E-01	N	2.48E-01	. mg/kg
Benzo (b) fluoranthene	2/4	3.90E-02 - 5.10E-02	5.00E-01 - 5.00E-01	Ň	1.48E-01	mg/kg
Benzo (ghi) përylene	2/4	1.66E-01 - 1.70E-01	5.00E-01 - 5.00E-01	N	2.09E-01	mg/kg
Benzo(k)fluoranthene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Bromodichloromethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Bromoform	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Bromomethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Butyl benzyl phthalate	0/3		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Carbazole	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	
Carbon Disulfide	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Carbon Tetrachloride	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chlorobenzene	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
				14.1	3.005-03	mg∕kg

*L=Lognormal, N=Normal, NT=Not tested

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---- LOCATION=SWMU 193A MEDIA=Surface Soil ----- (continued)

	Frequency of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Chloroethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chloroform	0/3		1.00E-02 ~ 1.00E-02	NT	5.00E-03	mg/kg
Chloromethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chrysene	2/4	1.70E-01 - 1.70E-01	5.00E-01 - 5.00E-01	N	2.10E-01	mg/kg
Di-n-butylphthalate	1/4	7.70E-02 - 7.70E-02	5.00E-01 - 6.60E-01	N	2.27E-01	mg/kg
Di-n-octylphthalate	1/4	1.20E-01 - 1.20E-01	5.00E-01 - 5.00E-01	N	2.18E-01	mg/kg
Dibenz (a, h) anthracene	1/4	1.30E-01 - 1.30E-01	5.00E-01 - 5.00E-01	N	2.20E-01	mg/kg
Dibenzofuran	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Dibromochloromethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Diethylphthalate	1/4	4.00E-01 - 4.00E-01	5.00E-01 - 5.00E-01	N	2.88E-01	mg/kg
Dimethylphthalate	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Ethylbenzene	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Fluoranthene	2/4	2.30E-01 - 3.10E-01	5.00E-01 - 5.00E-01	N	2.60E-01	mg/kg
Fluorene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorobenzene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorobutadiene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorocyclopentadiene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachloroethane	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Indeno (1,2,3-cd) pyrene	2/4	1.38E-01 - 1.60E-01	5.00E-01 - 5.00E-01	N	2.00E-01	mg/kg
Isophorone	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Methylene Chloride	0/3		1.00E-02 - 1.20E-02	NT	5.33E-03	mg/kg
N-Nitroso-di-n-propylamine	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
N-Nitrosodiphenylamine	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Naphthalene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Nitrobenzene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
PCB-1016	0/4		1.01E-01 - 1.14E-01	NT	1.08E-01	mg/kg
PCB-1221	0/4		1.01E-01 - 1.14E-01	NT	1.08E-01	mg/kg
PCB-1232	0/4		1.01E-01 - 1.14E-01	NT	1.08E-01	mg/kg
PCB-1242	0/4		1.01E-01 - 1.14E-01	NT	1.08E-01	mg/kg
PCB-1248	0/4		1.01E-01 - 1.14E-01	NT	1.08E-01	mg/kg
PCB-1254	0/4		1.01E-01 - 1.14E-01	NT	1.08E-01	mg/kg
PCB-1260	0/4		1.01E-01 - 1.14E-01	NT	1.08E-01	mg/kg
PCB-1268	0/1		1.00E-01 - 1.00E-01	NT	1.00E-01	mg/kg
Pentachlorophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Phenanthrene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Phenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Polychlorinated biphenyl	0/1		1.00E-01 - 1.00E-01	NT	1.00E-01	mg/kg
Pyrene	2/4	2.40E-02 - 2.95E-01	5.00E-01 - 5.00E-01	N	2.05E-01	mg/kg
Pyridine	0/1		3.80E-01 - 3.80E-01	NT	1.90E-01	mg/kg
Styrene	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg

----- LOCATION=SWMU 193A MEDIA=Surface Soil ----- (continued)

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	Frequency					
	öf	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Tetrachloroethene	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Toluene	0/3		1.00E-02 ~ 1.00E-02	NT	5.00E-03	mg/kg
Trichloroethene	0/4		1.68E-01 - 3.07E-01	NT	2.54E-01	mg/kg
Vinyl Chloride	0/4		1.68E-01 - 3.07E-01	NT	2.54E-01	mg/kg
bis(2-Chloroethoxy)methane	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
bis(2-Chloroethyl)ether	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
bis (2-Chloroisopropyl) ether	0/4		5.00B-01 - 5.00B-01	NT	2.50E-01	mg/kg
bis(2-Ethylhexyl)phthalate	2/4	8.10E-02 - 1.70E-01	5.00E-01 - 5.00E-01	N	1.88E-01	mg/kg
cis-1,2-Dichloroethene	0/4		1.68E-01 - 3.07E-01	NT	2.54E-01	mg/kg
cis-1,3-Dichloropropene	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
m,p-Xylene	0/3		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/kg
trans-1,2-Dichloroethene	0/4		1.68E-01 - 3.07E-01	NT	2.54E-01	mg/kg
trans-1,3-Dichloropropene	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Alpha activity	4/4	9.60E+00 - 1.70E+01		N	1.32E+01	pCi/g
Americium-241	0/4		1.80E+00 - 7.20E+00	NT	5.28E+00	pCi/g
Beta activity	4/4	1.41E+01 ~ 2.37E+01		N	2.10E+01	pCi/g
Cesium-137	0/4		6.10E=01 - 9.00E-01	NT	7.40E-01	pCi/g
Cobalt-60	0/4		8.30E-01 - 4.00E+00	NT	1.74E+00	pCi/g
Protactinium-234m	0/4		1.10E+02 - 5.80E+02	ŇT	2.38E+02	pCi/g
Technetium-99	0/4		0.00E+00 - 1.09E+00	NT	3.58E-01	pCi/g
Thorium-234	0/4		1.10E+01 - 1.80E+01	NT	1.45E+01	pCi/g
Uranium-235	0/4		2.00E+00 - 6.30E+00	NT	4.50E+00	pCi/g pCi/g

----- LOCATION-SWMU 193B MEDIA=McNairy Groundwater -----

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
1,1,1-Trichloroethane	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
1,1-Dichloroethene	Ö/2		1.00E-03 - 1.00E-02	NT	2.75E-03	mg/L
1,2,4-Trichlorobenzene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
1,2-Dichlorobenzene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
1,2-Dichloroethane	0/1		2.00E+00 - 2.00E+00	NT	1.00E+00	mg/L
1,3-Dichlorobenzene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
1,4-Dichlorobenzene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
2,4,5-Trichlorophenol	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
2,4,6-Trichlorophenol	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
2,4-Dichlorophenol	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L

*L=Lognormal, N=Normal, NT=Not tested

 LOCATION-SWMU	193B	MEDIA=McNairy	Groundwater	
		ontinued)		

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
2,4-Dimethylphenol	0/1	-	- 1.00E-01 - 1.00E-01	NT		
2,4-Dinitrotoluene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
2,6-Dinitrotoluene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
2-Chloronaphthalene	0/1		1.00E-01 - 1.00E-01 1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
2-Chlorophenol	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
2-Methyl-4,6-dinitrophenol	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
2-Methylnaphthalene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
2-Methylphenol	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
2-Nitroaniline	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
2-Nitrophenol	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02 5.00E-02	mg/L
3,3'-Dichlorobenzidine	0/1		1.00E-01 - 1.00E-01	NT	-	mg/L
3-Nitroaniline	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02 5.00E-02	mg/L
4-Bromophenyl phenyl ether	0/1		1.00E-01 - 1.00E-01	NT		mg/L
4-Chloro-3-methylphenol	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02 5.00E-02	mg/L
4-Chloroaniline	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
4-Chlorophenyl phenyl ether	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
4-Methylphenol	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
4-Nitroaniline	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L mg/L
4-Nitrophenol	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	.
Acenaphthene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L mg/L
Acenaphthylene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Anthracene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Benz (a) anthracene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Benzo (a) pyrene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Benzo(b)fluoranthene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Benzo (ghi) perylene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Benzo(k) fluoranthene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Carbazole	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Carbon Tetrachloride	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Chloroform	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	
Chrysene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Di-n-butylphthalate	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Di-n-octylphthalate	0/1		1.00E-01 - 1.00E-01	NT		mg/L
Dibenz (a, h) anthracene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Dibenzofuran	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Diethylphthalate	0/1		1.00E-01 - 1.00E-01 1.00E-01 - 1.00E-01	NI NT	5.00E-02	mg/L
Dimethylphthalate	0/1		1.00E-01 - 1.00E-01	NI NT	5.00E-02	mg/L
Fluoranthene	0/1		1.00E-01 - 1.00E-01 1.00E-01 - 1.00E-01		5.00E-02	mg/L
Fluorene	0/1		1.00E-01 - 1.00E-01 1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Hexachlorobenzene	0/1		1.00E-01 - 1.00E-01 1.00E-01 - 1.00E-01	NT NT	5.00E-02	mg/L
	-, <u>-</u>		1.00E-01 - 1.00E-01	NI	5.00E-02	mg/L

*L=Lognormal, N=Normal, NT=Not tested

----- LOCATION=SWMU 193B MEDIA=McNairy Groundwater -------(continued)

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Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Méan	Units
Hexachlorobutadiene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Hexachlorocyclopentadiene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Hexachloroethane	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Indeno (1, 2, 3-cd) pyrene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Isophorone	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
N-Nitroso-di-n-propylamine	0/1		1.00E-01 - 1.00E-01	ŇT	5.00E-02	mg/L
N-Nitrosodiphenylamine	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Naphthalene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Nitrobenzene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Pentachlorophenol	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Phenanthrene	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Phenol	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
Pyrene Trichloroethene	1/2	1.30E-02 - 1.30E-02	1.00E-03 - 1.00E-03	N	7.00E-03	mg/L
	0/2	1.504 1.504 04	1.00E-03 - 1.00E-02	NT	5.50E-03	mg/L
Vinyl Chloride	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
bis (2-Chloroethoxy) methane			1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
bis (2-Chloroethyl) ether	0/1 0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
bis (2-Chloroisopropyl) ether	0/1		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L
bis (2-Ethylhexyl) phthalate		2.30E-02 - 2.30E-02	1.00E-03 - 1.00E-03	N	1.20E-02	mg/L
cis-1,2-Dichloroethene	1/2	2.308-02 - 2.308-02	1.00E-03 - 2.00E+00	NT	1.00E+00	1119715 m=/T
trans-1,2-Dichloroethene	0/2	1 007 00 1 007 00				mg/L
Alpha activity	1/2	1.29E+00 - 1.29E+00	1.20E+00 - 1.20E+00	N	1.25E+00	pCi/L
Beta activity	2/2	3.15E+00 - 4.80E+00		N	3.98E+00	pCi/L
Technetium-99	0/2		1.40E+00 - 5.70E+00	NT	3.55E+00	pCi/L

----- LOCATION=SWMU 193B MEDIA=RGA Groundwater ------

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
1,1,1-Trichloroethane	0/5		5.00E-03 - 5.00E-02	NT	1.60E-02	mg/L
1,1,2,2-Tetrachloroethane	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
1,1,2-Trichloroethane	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
1.1-Dichloroethane	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
1,1-Dichloroethene	3/17	2.30E-04 - 2.00E-02	1.90E-04 - 5.00E-02	L	3.36E-04	mg/L
1,2,4-Trichlorobenzene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
1.2-Dichlorobenzene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
1.2-Dichloroethane	0/5		5.00E-03 - 2.00E+00	NT	6.01E-01	mg/L

---- LOCATION=SWMU 193B MEDIA=RGA Groundwater ----(continued)

	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
1,2-Dichloropropane	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
1,2-Dimethylbenzene	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
1,3-Dichlorobenzene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
1,4-Dichlorobenzene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2,4,5-Trichlorophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2,4,6-Trichlorophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2,4-Dichlorophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2,4-Dimethylphenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2,4-Dinitrophenol	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
2,4-Dinitrotoluene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2,6-Dinitrotoluene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2-Butanone	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/L
2-Chloronaphthalene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2-Chlorophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2-Hexanone	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/L
2-Methyl-4,6-dinitrophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2-Methylnaphthalene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2-Methylphenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2-Nitroaniline	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2-Nitrophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
3,3'-Dichlorobenzidine	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
3-Nitroaniline	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
4-Bromophenyl phenyl ether	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
4-Chloro-3-methylphenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
4-Chloroaniline	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
4-Chlorophenyl phenyl ether	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
4-Methyl-2-pentanone	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/L
4-Methylphenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
4-Nitroaniline	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
4-Nitrophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Acenaphthene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Acenaphthylene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Acetone	1/2	3.30E-02 - 3.30E-02	1.00E-02 - 1.00E-02	N	1.90E-02	mg/L
Anthracene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Benz (a) anthracene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Benzene	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Benzo(a)pyrene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Benzo(b) fluoranthene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Benzo (ghi) perylene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Benzo(k)fluoranthene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
					2.002 02	

*L=Lognormal, N=Normal, NT=Not tested

----- LOCATION-SWMU 193B MEDIA-RGA Groundwater

	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Bromodichloromethane	0/2		5.008÷03 - 5.00E-03	NT	2.50E-03	mg/L
Bromoform	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Butyl benzyl phthalate	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Carbazole	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Carbon Disulfide	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Carbon Tetrachloride	1/5	5.50E-03 - 5.50E-03	5.00E-03 - 5.00E-02	N	1.21E-02	mg/L
Chlorobenzene	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Chloroethane	0/2		5.00E-03 ~ 5.00E-03	NT	2.50E-03	mg/L
Chloroform	0/5		5.00E-03 - 5.00E-02	NT	1.60E-02	mg/L
Chloromethane	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Chrysene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Di-n-butylphthalate	2/10	1.30E-02 - 1.30E-02	2.00E-02 - 2.00E-02	N	1.06E-02	mg/L
Di-n-octylphthalate	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Dibenz (a, h) anthracene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Dibenzofuran	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Dibromochloromethane	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Diethylphthalate	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Dimethylphthalate	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Ethylbenzene	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Fluoranthene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Fluorene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Hexachlorobenzene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Hexachlorobutadiene	0/10		2.00E-02 - 2.00E-02	ŃŤ	1.00E÷02	mg/L
Hexachlorocyclopentadiene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Hexachloroethane	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Indeno(1,2,3-cd)pyrene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Isophorone	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Methylene Chloride	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/L
N-Nitroso-di-n-propylamine	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
N-Nitrosodiphenylamine	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Naphthalene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Nitrobenzene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	- ·
Pentachlorophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Phenanthrene	0/10		2.00E-02 - 2.00E-02	NT		mg/L
Phenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02 1.00E-02	mg/L
Pyrene	0/10		2.00E-02 - 2.00E-02	NT		mg/L
Pyridine	0/1		5.00E-03 - 5.00E-03	NT	1.00E-02	mg/L
Styrene	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Tetrachloroethene	0/2		5.00E-03 - 5.00E-03 5.00E-03 - 5.00E-03	NT NT	2.50E-03	mg/L
Toluene	0/2			-·-	2.50E-03	mg/L
			5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L

LOCATION=SWMU 193B MEDIA=RGA Groundwater - (continued)

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Analyte	of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
Trichloroethene	17/17	1.00E=04 = 5.00E=01		L	8.01E-01	mg/L
Vinvl Chloride	0/17		1.00E-03 - 1.00E-01	NT	2.05E-02	mg/L
bis (2-Chloroethoxy) methane	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
bis (2-Chloroethyl) ether	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
bis (2-Chloroisopropyl) ether	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
bis (2-Ethylhexyl) phthalate	1/10	1.80E-02 - 1.80E-02	2.00E-02 - 2.00E-02	Ň	1.08E-02	mg/L
cis-1,2-Dichloroethene	12/17	1.90E-04 - 9.87E-02	2.20E-04 - 5.00E-03	L	2.92E-03	mg/L
cis-1,3-Dichloropropene	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
m,p-Xylene	0/2		1.00Ē-02 - 1.00Ē-02	NT	5.00E-03	mg/L
trans-1,2-Dichloroethene	8/17	1.00E-04 - 8.10E-04	4.90E-04 - 2.00E+00	L	3.29E-04	mg/L
trans-1, 3-Dichloropropene	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Alpha activity	12/17	1.00E+00 - 6.60E+02	1.10E+00 - 2.80E+00	Ŀ	6.09E+00	pCi/L
Beta activity	16/17	2.70E+00 - 5.85E+02	5.12E+00 - 5.12E+00	L	2.39E+01	pCi/L
Neptunium-237	0/1		-2.34E-032.34E-03	NT	-2.34E-03	pCi/L
Technetium-99	8/17	1.45E+01 - 6.10E+01	-5.00E-01 - 1.20E+01	N	1.89E+01	pCi/L

Frequency Nondetected of Detected Arithmetic Detection Range Range Distribution* Analyte Mean Units 7.43E+03 - 1.12E+04 Aluminum 4/4 N 9.98E+03 mg/kg 0/4 2.00E+01 - 2.00E+01 NT Antimony 1.00E+01 mg/kg Arsenic 0/4 5.00E+00 - 5.00E+00 NT 2.50E+00 mg/kg 4/4 3.80E+01 - 8.42E+01 Barium N 5.36E+01 mg/kg Beryllium 2/4 5.90E-01 - 1.57E+00 5.00E-01 - 5.00E-01 N mg/kg 6.65E-01 0/4 1.00E+02 = 1.00E+02NT Boron 5.00E+01 mg/kg 0/4 2.00E+00 - 2.00E+00 Cadmium NT 1.00E+00 mg/kg 4/4 1.04E+01 - 8.87E+01 Chromium N 3.24E+01 mg/kg 4/4 3.18E+00 - 7.76E+00 N Cobalt 4.99E+00 mg/kg 4/4 4.18E+00 - 7.43E+00 N Copper 6.21E+00 mg/kg 0/2 1.00E+00 - 1.00E+00 NT Cyanide 5.00E-01 mg/kg 4/4 Iron 9.73E+03 - 2.43E+04 N 1.45E+04 mg/kg 0/4 2.00E+01 - 2.00E+01 NT Lead 1.00E+01 mg/kg 4/4 Lithium 3.44E+00 - 7.72E+00 Ň 5.82E+00 mg/kg 4/4 7.74E+02 - 4.31E+03 N Magnesium 1.84E+03 mg/kg 4/4 1.05E+02 - 2.22E+02N Manganese 1.54E+02 mg/kg 0/4 2.00E-01 - 2.00E-01 NT Mercury 1.00E-01 mg/kg

*L=Lognormal, N=Normal, NT=Not tested

----- LOCATION=SWMU 193B MEDIA=Subsurface soil -----(continued)

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	Frequency					
	of	Detected	Nondetectéd		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Nickel	2/4	7.82E+00 - 2.06E+01	5.00E+00 - 5.00E+00	N	8.36E+00	mg/kg
Potassium	4/4	2.37E+02 - 6.86E+02		Ň	3.91E+02	mg/kg
Selenium	0/4		5.00E+00 - 5.00E+00	NT	2.50E+00	mg/kg
Silver	0/4		4.00E+00 - 4.00E+00	NT	2.00E+00	mg/kg
Sodium	4/4	2.44E+02 - 4.48E+02		N	3.12E+02	mg/kg
Strontium	4/4	8.11E+00 - 9.39E+01		N	3.13E+01	mg/kg
Thallium	0/4		1.50E+01 - 1.50E+01	NT	7.50E+00	mg/kg
Vanadium	4/4	1.75E+01 - 6.50E+01		N	3.10E+01	mg/kg
Zinc	4/4	1.75E+01 - 5.57E+01		N	3.09E+01	mg/kg
1,1,1-Trichloroethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,1,2,2-Tetrachloroethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,1,2-Trichloroethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,1-Dichloroethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,1-Dichloroethene	0/4		1.08E-01 - 4.09E-01	NT	1.23E-01	mg/kg
1,2,4-Trichlorobenzene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mğ/kg
1,2-Dichlorobenzene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
1,2-Dichloroethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,2-Dichloropropane	0/3		$1.00E-02 \div 1.00E-02$	NT	5.00 <u>E</u> -03	mg/kg
1,2-Dimethylbenzene	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,3-Dichlorobenzene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
1,4-Dichlorobenzene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4,5-Trichlorophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4,6-Trichlorophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg∕kg
2,4-Dichlorophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4-Dimethylphenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4-Dinitrophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4-Dinitrotoluene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,6-Dinitrotoluene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Chloronaphthalene	0/4		5.00E-01 - 5.00 <u>E</u> -01	NT	2.50E-01	mg/kg
2-Chlorophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Hexanone	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
2-Methyl-4,6-dinitrophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50Ē-01	mg/kg
2-Methylnaphthalene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Methylphenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Nitroaniline	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Nitrophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
3,3'-Dichlorobenzidine	0/4		5.00E-01 - 5.00E-01	NŢ	2.50E-01	mg/kg
3-Nitroaniline	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Bromophenyl phenyl ether	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Chloro-3-methylphenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg

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------ LOCATION=SWMU 193B MEDIA=Subsurface Soil ------(continued)

	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
4-Chloroaniline	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Chlorophenyl phenyl ether	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Methyl-2-pentanone	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
4-Methylphenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Nitroaniline	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Nitrophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Acenaphthene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Acenaphthylene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Acetone	1/1	8.00E-02 - 8.00E-02		NT	8.00E-02	mg/kg
Anthracene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Benz (a) anthracene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Benzene	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Benzo (a) pyrene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Benzo (b) fluoranthene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Benzo (ghi) perylene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Benzo(k)fluoranthene	0/4		5.00B-01 - 5.00E-01	NT	2.50E-01	mg/kg
Bromodichloromethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Bromoform	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Bromomethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Butyl benzyl phthalate	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Carbazole	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Carbon Disulfide	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Carbon Tetrachloride	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chlorobenzene	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chloroethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chloroform	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chloromethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chrysene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Di-n-butylphthalate	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Di-n-octylphthalate	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Dibenz (a, h) anthracene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Dibenzofuran	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Dibromochloromethane	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Diethylphthalate	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Dimethylphthalate	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Ethylbenzene	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Fluoranthene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Fluorene	0/4		5.00E-01 ~ 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorobenzene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorobutadiene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
					2.JUN-U1	

*L=Lognormal, N=Normal, NT=Not tested

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Continued)

Analyte	of					
ADALVER		Detected	Nondetected		Arithmetic	
	Detection	Range	Range	Distribution*	Mean	Units
Hexachlorocyclopentadiene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachloroethane	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Indeno (1,2,3-cd) pyrene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Isophorone	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Methylene Chloride	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
N-Nitroso-di-n-propylamine	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
N-Nitrosodiphenylamine	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Naphthalene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Nitrobenzene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
PCB-1016	0/4		1.07E-01 - 1.20E-01	NT	1.13E-01	mg/kg
PCB-1221	0/4		1.07E-01 - 1.20E-01	NT	1.13E-01	mg/kg
PCB-1232	0/4		1.07E-01 - 1.20E-01	NT	1.13E-01	mg/kg
PCB-1242	0/4		1.07E-01 - 1.20E-01	NT	1.13E-01	mg/kg
PCB-1248	0/4		1.07E-01 - 1.20E-01	NT	1.13E-01	mg/kg
PCB-1254	0/4		1.07E-01 - 1.20E-01	NT	1.13E-01	mg/kg
PCB-1260	0/4		1.07E-01 - 1.20E-01	NT	1.13E-01	mg/kg
Pentachlorophenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Phenanthrene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Phenol	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Pyrene	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Styrene	0/3		1.00E-02 - 1.00E-02	ŇT	5.00E-03	mg/kg
Tetrachloroethene	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Toluene	1/3	1.00E-02 - 1.00E-02	1.00E-02 - 1.00E-02	N	6.67E-03	mg/kg
Trichloroethene	0/4	-	1.08E-01 - 4.09E-01	NT	2.46E-01	mg/kg
Vinyl Chloride	0/4		1.08E-01 - 1.00E+02	NT	2.52E+01	mg/kg
bis(2-Chloroethoxy)methane	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
bis(2-Chloroethyl)ether	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
bis (2-Chloroisopropyl) ether	0/4		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
bis(2-Ethylhexyl)phthalate	0/4		5.00E-01 - 5.00E-01	ŇŤ	2.50E-01	mg/kg
cis-1,2-Dichloroethene	0/4		1.08E-01 - 5.00E-01	NT	3.32E-01	mg/kg
cis-1,3-Dichloropropene	0/3		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
m,p-Xylene	0/3		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/kg
trans-1,2-Dichloroethene	0/4		1.08E-01 - 5.00E-01	NT	3.32E-01	
trans-1,3-Dichloropropene	0/3		1.00E-02 - 1.00E-02	NT		mg/kg
Alpha activity	4/4	2.14E+00 - 1.86E+01	2.002 02 1.002-02	N	5.00E-03	mg/kg
Americium-241	0/4	1.00<u>.</u>101	2.20E+00 - 1.00E+01	NT	1.19Ê+01	pCi/g
Beta activity	4/4	9.10E+00 - 2.29E+01	1.10B+00 - 1.00B+01	N	6.00E+00	pCi/g
Cesium-137	0/4		7.60E-01 - 2.70E+00	N NT	1.52E+01	pCi/g
Cobalt-60	0/4		1.00E+00 - 1.30E+00	NT	1.74E+00	pCi/g
Protactinium-234m	0/4		1.50E+02 - 6.10E+02	NT	1.15E+00 3.53E+02	pCi/g pCi/g

.----- LOCATION=SWMU 193B MEDIA=Subsurface Soil ------ LOCATION=SWMU 193B MEDIA=Subsurface Soil ------

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	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Technetium-99	0/4		5.60E-01 - 2.51E+00	NT	1.51E+00	pCi/g
Thorium-234	0/4		5.10E+00 ~ 1.60E+01	NT	1.15E+01	pCi/g
Uranium-235	0/4		2.00E+00 - 6.30E+00	NT	3.23E+00	pCi/g
		- LOCATION-SWMU 193B MED	Desurface Soil			
	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Analyte	200001011			• • • • • • • • • • •		
Aluminum	2/2	7.43E+03 - 1.08E+04		N	9.12E+03	mg/kg
Antimony	0/2		2.00E+01 - 2.00E+01	NT	1.00E+01	mg/kg
Arsenic	0/2		5.00E+00 - 5.00E+00	NT	2.50E+00	mg/kg
Barium	2/2	3.80E+01 - 8.42E+01		N	6.11E+01	mg/kg
Beryllium	1/2	1.57E+00 - 1.57E+00	5.00E-01 - 5.00E-01	N	9.105-01	mg/kg
Boron	0/2		1.00E+02 - 1.00E+02	NT	5.00E+01	mg/kg
Cadmium	0/2		2.00E+00 - 2.00E+00	NT	1.00E+00	mg/kg
Chromium	2/2	1.04E+01 ~ 8.87E+01		N	4.96E+01	mg/kg
Cobalt	2/2	3.82E+00 - 7.76E+00		N	5.79E+00	mg/kg
Copper	2/2	7.07E+00 - 7.43E+00		N	7.25E+00	mg/kg
Cyanide	0/1 ·		1.00E+00 - 1.00E+00	NT	5.00E-01	mg/kg
Iron	2/2	1.16E+04 - 2.43E+04		N	1.80E+04	mg/kg
Lead	0/2		2.00E+01 - 2.00E+01	NT	1.00E+01	mg/kg
Lithium	2/2	3.44E+00 - 7.72E+00		N	5.58E+00	mg/kg
Magnesium	2/2	7.74E+02 - 4.31E+03		N	2.54E+03	mg/kg
Manganese	2/2	1.13E+02 - 2.22E+02		N	1.68E+02	mg/kg
Mercury	0/2		2.00E-01 - 2.00E-01	NT	1.00E-01	mg/kg
Nickel	1/2	2.06E+01 - 2.06E+01	5.00E+00 - 5.00E+00	N	1.16E+01	mg/kg
Potassium	2/2	2.37E+02 - 6.86E+02		N	4.62E+02	mg/kg
Selenium	0/2		5.00E+00 - 5.00E+00	NT	2.50E+00	mg/kg
Silver	0/2		4.00E+00 - 4.00E+00	NT	2.00E+00	mg/kg
Sodium	2/2	2.44E+02 - 2.49E+02		N	2.47E+02	mg/kg
Strontium	2/2	1.42E+01 - 9.39E+01		N	5.41E+01	mg/kg
Thallium	0/2		1.50E+01 - 1.50E+01	NT	7.50E+00	mg/kg
Vanadium	2/2	1.75E+01 - 6.50E+01		N	4.13E+01	mg/kg
Zinc	2/2	3.21E+01 - 5.57E+01		N	4.39E+01	mg/kg
1,1,1-Trichloroethane	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
1,1,2,2-Tetrachloroethane	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	
1,1,2-Trichloroethane	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg

*L=Lognormal, N=Normal, NT=Not tested

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1,1-Dichloroethane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,1-Dichloroethane 0/2 1.55E-01 - 3.12E-01 NT 1.17E-01 mg/ 1,2,4-Trichlorobenzene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg/ 1,2-Dichlorobenzene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg/ 1,2-Dichlorobenzene 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dichloroperpane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dichloroperpane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dichloroperpane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dimethylbenzene 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/	
1,1-Dichloroethane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,1-Dichloroethane 0/2 1.55E-01 - 3.12E-01 NT 1.17E-01 mg/ 1,2,4-Trichlorobenzene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg/ 1,2-Dichlorobenzene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg/ 1,2-Dichlorobenzene 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dichloroperpane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dichloroperpane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dichloroperpane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dimethylbenzene 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/	
1,1-Dichloroethene 0/2 1.55E-01 - 3.12E-01 NT 1.17E-01 mg/ 1,2,4-Trichlorobenzene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg/ 1,2-Dichlorobenzene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg/ 1,2-Dichlorobenzene 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dichloropropane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dichloropropane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dimethylbenzene 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/	nits
1,2,4-Trichlorobenzene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg/ 1,2-Dichlorobenzene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg/ 1,2-Dichlorobenzene 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dichloropropane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dimethylbenzene 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/	j/kg
1,2-Dichlorobenzene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg/ 1,2-Dichloroethane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dichloropropane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dichloropropane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dimethylbenzene 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/	/kg
1,2-Dichloroethane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dichloropropane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dimethylbenzene 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/	j/kg
1,2-Dichloropropane 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/ 1,2-Dimethylbenzene 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg/	/kg
1,2-Dimethylbenzene 0/2 1.00E-02 NT 5.00E-03 mg/	r/kg
	j/kġ
1,3-Dichlorobenzene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 max	j/kg
	/kg
	r/kg
	J/kg
2,4,6-Trichlorophenol 0/2 5.00E-01 NT 2.50E-01 mg/	j/kg
	r/kg
	J/kg
2,4-Dinitrophenol 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	J/kg
2,4-Dinitrotoluene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	J/kg
2,6-Dinitrotoluene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	j/kg
	j/kg
2-Chlorophenol 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	g∕kg
2-Hexanone 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg	j/kg
2-Methyl-4,6-dinitrophenol 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	g/kg
2-Methylnaphthalene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	j/kg
2-Methylphenol 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	j/kg
2-Nitroaniline 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	j/kg
2-Nitrophenol 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	i/kg
3,3'-Dichlorobenzidine 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	r/kg
3-Nitroaniline 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	j/kg
4-Bromophenyl phenyl ether 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	/kg
4-Chloro-3-methylphenol 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	/kg
4-Chloroaniline 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	/kg
4-Chlorophenyl phenyl ether 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg/	i/kg
4-Methyl-2-pentanone 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg	i/kg
4-Methylphenol 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	i/kg
4-Nitroaniline 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	/kg
4-Nitrophenol 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	i/kg
Acenaphthene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	j/kg
	j/kg
Anthracene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	i/kg
Benz (a) anthracene 0/2 5.00E-01 - 5.00E-01 NT 2.50E-01 mg	i/kg
Benzene 0/2 1.00E-02 - 1.00E-02 NT 5.00E-03 mg	i/kg
	j/kg

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-- LOCATION=SWMU 193B MEDIA=Surface Soil -----(continued)

	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Benzo(b)fluoranthene	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Benzo (ghi) perylene	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Benzo(k)fluoranthene	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Bromodichloromethane	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Bromoform	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mq/kq
Bromomethane	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Butyl benzyl phthalate	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Carbazole	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Carbon Disulfide	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Carbon Tetrachloride	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chlorobenzene	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chloroethane	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chloroform	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chloromethane	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Chrysene	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Di-n-butylphthalate	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Di-n-octylphthalate	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Dibenz (a, h) anthracene	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Dibenzofuran	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Dibromochloromethane	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Diethylphthalate	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Dimethylphthalate	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Ethylbenzene	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Fluoranthene	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Fluorene	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorobenzene	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorobutadiene	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorocyclopentadiene	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachloroethane	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Indeno(1,2,3-cd)pyrene	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Isophorone	0/2		5.00E-01 - 5.00E-01	NT ·	2.50E-01	mg/kg
Methylene Chloride	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
N-Nitroso-di-n-propylamine	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
N-Nitrosodiphenylamine	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Naphthalene	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Nitrobenzene	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
PCB-1016	0/2		1.08E-01 - 1.20E-01	NT	1.14E-01	mg/kg
PCB-1221	0/2		1.08E-01 - 1.20E-01	NT	1.14E-01	mg/kg
PCB-1232	0/2		1.08E-01 - 1.20E-01	NT	1.14E-01	mg/kg
PCB-1242	0/2		1.08E-01 - 1.20E-01	NT	1.14E-01	mg/kg

*L=Lognormal, N=Normal, NT=Not tested

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----- LOCATION-SWMU 193B MEDIA-Surface Soil ------ (continued)

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
PCB-1248	0/2		1.08E+01 - 1.20E-01	NT	1.14E-01	mg/kg
PCB-1254	0/2		1.08E-01 - 1.20E-01	NT	1.14E-01	mg/kg
PCB-1260	0/2		1.08E-01 - 1.20E-01	NT	1.14E-01	mg/kg
Pentachlorophenol	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Phenanthrene	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Phenol	0/2		5.00E÷01 - 5.00B-01	NT	2.50E-01	mg/kg
Pyrene	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Styrene	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Tetrachloroethene	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Toluene	1/2	1.00E-02 - 1.00E-02	1.00E-02 - 1.00E-02	N	7.50E-03	mg/kg
Trichloroethene	0/2		1.55E-01 - 3.12E-01	NT	2.34E-01	mg/kg
Vinyl Chloride	0/2		3.12E-01 - 1.00E+02	NT	5.02E+01	mg/kg
bis(2-Chloroethoxy)methane	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
bis(2-Chloroethyl)ether	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
bis (2-Chloroisopropy1) ether	0/2		5.00Ë-01 - 5.00E-01	NT	2.50E-01	mg/kg
bis(2-Ethylhexyl)phthalate	0/2		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
cis-1,2-Dichloroethene	0/2		3.12E-01 - 5.00E-01	NT	4.06E-01	mg/kg
cis-1,3-Dichloropropene	0/2	•	1.00E-02 - 1.00E-02	ŇT	5.00E-03	mg/kg
m,p-Xylene	0/2		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/kg
trans-1,2-Dichloroethene	0/2		3.12E-01 - 5.00E-01	NT	4.06E-01	mg/kg
trans-1,3-Dichloropropene	0/2		1.00E-02 - 1.00E-02	NT	5.00E-03	mg/kg
Alpha activity	2/2	1.63E+01 - 1.86E+01		N	1.75E+01	pCi/g
Americium-241	0/2		2.20E+00 - 1.00E+01	NT	6.10E+00	pCi/g
Beta activity	2/2	1.66E+01 - 2.29E+01		N	1.98E+01	pCi/g
Cesium-137	0/2		7.60E-01 - 2.60E+00	NT	1.68E+00	pCi/g
Cobalt-60	0/2		1.00E+00 - 1.30E+00	NT	1.15E+00	pCi/g
Protactinium-234m	0/2		4.90E+02 - 6.10E+02	NT	5.50E+02	pCi/g
Technetium-99	0/2		1.94E+00 - 2.51E+00	NT	2.23E+00	pCi/g
Thorium-234	0/2		1.30E+01 - 1.60E+01	NT	1.45E+01	
Uranium-235	0/2		2.00E+00 - 6.30E+00	NT	4.15E+00	pCi/g pCi/g

----- LOCATION-SWMU 193C MEDIA-MCNairy Groundwater -----

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units	
Aluminum Antimony	4/4 5/5	7.50E-01 - 9.04E+01 6.00E-02 - 2.50E-01		N N	2.52E+01 1.48E-01	mg/L mg/L	

*L=Lognormal, N=Normal, NT=Not tested

 LOCATION-SWMU	193C MEDIA-McNairy	Groundwater	
	(continued)		

	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Arsenic	5/5	5.00E-03 - 3.60E-02		N	1.12E-02	mg/L
Barium	5/5	9.20E-02 - 6.70E-01		N	2.65E-01	mg/L
Beryllium	5/5	5.00E-03 - 2.50E-02		N	1.54E-02	mg/L
Cadmium	5/5	1.00E-02 - 1.00E-01		N	3.62E-02	mg/L
Calcium	5/5	6.48E+00 - 4.10E+01		N	2.19E+01	mg/L
Chloride	5/5	1.45E+01 - 1.68E+01		N	1.56E+01	mg/L
Chromium	3/3	5.00E-02 - 2.32E-01		N	1.14E-01	mg/L
Cobalt	5/5	4.50E-02 - 1.21E-01		N	7.22E-02	mg/L
Copper	5/5	1.30E-02 - 1.63E-01		N	6.52E-02	mg/L
Fluoride	4/4	2.00E-01 - 2.80E-01		N	2.38E-01	mg/L
Iron	5/5	5.04E+00 - 1.79E+02		N	4.75E+01	mg/L
Lead	1/1	2.50E-01 - 2.50E-01		NT	2.50E-01	mg/L
Magnesium	5/5	2.14E+00 - 2.16E+01		N	7.47E+00	mg/L
Manganese	5/5	3.57E-01 - 3.91E+00		N	1.34E+00	mg/L
Mercury	1/1	2.00E-04 - 2.00E-04		NT	2.00E-04	mg/L
Molybdenum	4/4	5.00E-02 - 1.00E-01		N	6.38E-02	mg/L
Nickel	5/5	1.00E-01 - 1.09E-01		N	1.03E-01	mg/L
Nitrate as Nitrogen	5/5	1.00E+00 - 1.00E+00		N	1.00E+00	mg/L
Potassium	5/5	3.73E+01 ~ 1.01E+02		N	6.92E+01	mg/L
Selenium	3/3	5.00E-03 ~ 5.00E-03		N	5.00E-03	mg/L
Silica	5/5	1.10B+01 - 1.80B+01		N	1.32E+01	mg/L
Silver	3/3	5.00E-02 - 6.00E-02		N	5.67E-02	mg/L
Sodium	5/5	1.90E+01 - 2.63E+01		N	2.23E+01	mg/L
Tetraoxo-sulfate(1-)	5/5	9.60E+00 - 1.30E+01		N	1.19E+01	mg/L
Thallium	2/2	6.00E-02 - 1.23E-01		N	9.15E-02	mg/L
Uranium	9/9	1.00E-03 - 1.80E-02		N	2.89E-03	mg/L
Vanadium	2/2	5.70E-02 - 8.36E-01		N	4.47E-01	mg/L
Zinc	5/5	2.60E-02 - 5.64E-01		N	1.89E-01	mg/L
1,1,1-Trichloroethane	4/4	5.00E-03 - 5.00B-03		N	5.00E-03	mg/L
1,1,2-Trichloroethane	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
1,1-Dichloroethane	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
1,1-Dichloroethene	4/4	5.00B-03 - 5.00E-03		N	5.00E-03	mg/L
1,2-Dichloroethane	4/4	5.00E-03 ~ 5.00E-03		N	5.00E-03	mg/L
Benzene	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
Bromodichloromethane	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
Carbon Tetrachloride	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
Chloroform	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	
Ethylbenzene	4/4	5.00E-03 - 5.00E-03		N		mg/L
Polychlorinated biphenyl	1/1	1.00E-04 - 1.00E-04		NT	5.00E-03	mg/L
Tetrachloroethene	4/4	5.00E-03 - 5.00E-03	•	N	1.00E-04	mg/L
	• -			14	5.00E-03	mg/L

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*L=Lognormal, N=Normal, NT=Not tested

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	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Toluene	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
Trichloroethene	12/12	1.00E-03 - 2.00E-03		N	1.08E-03	mq/L
Vinyl Chloride	4/4	5.00E-03 - 1.00E-02		Ň	6.25E-03	mg/L
Xvlene	4/4	5.00E-03 - 1.00E-02		Ň	7.50E-03	mg/L
cis-1,2-Dichloroethene	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
trans-1,2-Dichloroethene	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
Alpha activity	12/12	-1.80E+01 - 1.07E+02		N	7.47E+00	pCi/L
Beta activity	12/12	5.50E+01 - 2.36E+02		L	1.29E+02	pCi/L
Radon-222	2/2	1.43E+02 - 1.57E+02		Ň	1.50E+02	pCi/L
Technetium-99	13/13	-7.00E+00 - 2.70E+01		N	6.18E+00	pCi/L
1901119011011-23					0.102700	PC1/2
		LOCATION-SWMU 193C MEDIA	A=RGA Groundwater			·
	Frequency					
	of	Detected	Nondetected	,	Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
ranaly ce		3 -				0
1,1,1-Trichloroethane	0/2		1.00E-03 - 1.00E-03	NT	5.00E-04	mg/L
1.2-Dichloroethene	1/2	5.62E-01 - 5.62E-01	5.00E-03 - 5.00E-03	Ň	2.82E-01	mg/L
Benzene	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Ethylbenzene	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Toluene	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Trichloroethene	1/2	1.62E-01 - 1.62E-01	1.00E-03 - 1.00E-03	N	8.15E-02	mg/L
Xylene	0/1		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Ayrene	0, 1		5.002 05 5.002 05	AV <u>A</u>	2.301-03	11197 H
		LOCATION-SWMU 193C MEDIA	A=Subsurface Soil			
	Frequency	- · · · ·	·			
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Aluminum	20/20	3.14E+02 - 1.37E+04		N	8.52E+03	mg/kg
Antimony	0/20	•	2.00E+01 - 2.00E+01	NT	1.00E+01	mg/kg
Arsenic	5/20	5.01E+00 - 6.57E+00	5.00E+00 - 5.00E+00	N	3.33E+00	mg/kg
Barium	1/1	1.42E+02 - 1.42E+02		NT	1.42E+02	mg/kg
Beryllium	10/20	5.00E-01 - 9.80E-01	5.00E-01 - 5.00E-01	N	4.61E-01	mg/kg
Boron	1/20	1.00E+02 - 1.00E+02	1.00E+02 - 1.00E+02	N	5.25E+01	mq/kq
Cadmium	3/59	2.41E-01 - 5.00E+00	2.10E-01 - 5.00E+00	N	1.92E+00	mg/kg
				4 7	1.720700	à. v.A

*L=Lognormal, N=Normal, NT=Not tested

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------ LOCATION=SWMU 193C MEDIA=Subsurface Soil ------ LOCATION=SWMU 193C MEDIA=Subsurface Soil -----

	Frequency	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Calcium	18/20	2.238+02 - 4.008+05	5.00E+00 - 5.00E+00	L	2.60E+04	mg/kg
Chromium	59/61	3.76E+00 - 8.30E+01	2.00E+00 - 2.00E+00	L	1.89E+01	mg/kg
Chromium, hexavalent	0/20	•	5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Cobalt	17/20	1.22E+00 - 8.61E+01	1.00E+00 - 1.00E+00	L	6.46E+00	mg/kg
Copper	16/20	2.15E+00 - 2.82E+01	2.00E+00 - 2.00E+00	L	7.56E+00	mg/kg
Iron	6/6	1.20E+04 - 3.00E+04		N	2.225+04	mg/kg
Lead	42/61	5.10E+00 ~ 6.77E+01	5.00E+00 - 2.00E+01	L	1.20E+01	mg/kg
Lithium	17/20	2.50E+00 - 1.25E+01	2.00E+00 - 2.00E+00	N	7.17E+00	mg/kg
Magnesium	20/20	8.52E+02 - 1.45E+04		L	3.41E+03	mg/kg
Manganese	20/20	1.63E+01 - 2.27E+03		L	3.99E+02	mg/kg
Mercury	0/20		2.00E-01 - 2.00E-01	NT	1.00E-01	mg/kg
Nickel	13/20	5.99E+00 - 2.15E+01	5.00E+00 - 5.00E+00	L	9.76E+00	mg/kg
Potassium	20/20	1.43E+02 - 1.57E+03		L	5.33E+02	mg/kg
Selenium	0/20		5.00E+00 - 5.00E+00	NT	2.50E+00	mg/kg
Silver	0/20		4.00E+00 - 4.00E+00	NT	2.00E+00	mg/kg
Sodium	15/20	2.02E+02 - 4.44E+02	2.00E+02 - 2.00E+02	N	2.62E+02	mg/kg
Strontium	20/20	7.25E+00 - 3.91E+02		L	1.06E+02	mg/kg
Thallium	0/20		1.50E+01 - 1.50E+01	NT	7.50E+00	mg/kg
Vanadium	20/20	2.12E+00 - 4.42E+01		N	2.05E+01	mg/kg
Zinc	19/20	1.63E+01 - 9.25E+01	1.50E+01 - 1.50E+01	N	4.14E+01	mg/kg
1,1,1-Trichloroethane	0/38		1.00E-03 - 5.00E-03	NT	8.68E-04	mg/kg
1,2-Dichloroethene	0/55		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/kg
Benzene	0/20	•	5.00E-03 - 5.00E-03	NT	2.50E-03	mg/kg
Ethylbenzene	0/20		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/kg
Polychlorinated biphenyl	0/12		1.00E-01 - 1.00E-01	NT	1.00E-01	mg/kg
Toluene	0/20		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/kg
Trichloroethene	0/55		1.00E-03 - 5.00E-03	. NT	2.24E-03	mg/kg
Xylene	1/20	1.00E-02 - 1.00E-02	5.00E-03 - 5.00E-03	N	2.88E-03	mg/kg
Alpha activity	53/53	1.50E-03 - 4.00E+00		N	1.54E+00	pCi/g
Beta activity	53/53	5.00E-03 - 1.00E+01		N	4.45E+00	pCi/g

----- LOCATION=SWMU 193C MEDIA=Surface Soil ------

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
Aluminum Antimony	5/5 0/5	3.14E+02 - 3.36E+03	2.00E+01 - 2.00E+01	N NT	1.90E+03 1.00E+01	mg/kg mg/kg

*L=Lognormal, N=Normal, NT=Not tested

(continued)

		Frequency	Detected	Nondetected		Arithmetic	
	Analyte	Detection	Range	Range	Distribution*	Mean	Units
•	Arsenic	0/5		5.00E+00 - 5.00E+00	NT	2.50E+00	mg/kg
	Beryllium	0/5		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
	Boron	1/5	1.00E+02 - 1.00E+02	1.00E+02 - 1.00E+02	N	6.00E+01	mg/kg
	Cadmium	0/5		2.00E+00 - 2.00E+00	NT	1.00E+00	mg/kg
	Calcium	5/5	2.53E+05 - 4.00E+05		N	3.37E+05	mg/kg
	Chromium	3/5	4.49E+00 - 1.20E+01	2.00E+00 - 2.00E+00	N	5.90E+00	mg/kg
	Chromium, hexavalent	0/5		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
	Cobalt	2/5	1.22B+00 - 2.14E+00	1.00E+00 - 1.00E+00	N	9.72E-01	mg/kg
	Copper	2/5	3.37E+00 - 2.82E+01	2.00E+00 - 2.00E+00	N	6.91E+00	mg/kg
	Lead	1/5	6.77E+01 - 6.77E+01	2.00E+01 - 2.00E+01	N	2.15E+01	mg/kg
	Lithium	3/5	5.43E+00 - 1.25E+01	2.00E+00 - 2.00E+00	N	6.03E+00	mg/kg
	Magnesium	5/5	3.19E+03 - 1.45E+04		N	8.76E+03	mg/kg
	Manganese	5/5	3.55E+01 - 1.98E+02		N	7.90E+01	mg/kg
	Mercury	0/5		2.00E-01 - 2.00E-01	NT	1.00E-01	mg/kg
	Nickel	1/5	6.43E+00 - 6.43E+00	5.00E+00 - 5.00E+00	N	3.29E+00	mg/kg
	Potassium	5/5	1.43E+02 - 1.57E+03		N	7.01E+02	mg/kg
	Selenium	0/5	11010101	5.00E+00 ~ 5.00E+00	NT	2.50E+00	mg/kg
	Silver	0/5		4.00E+00 - 4.00E+00	NT	2.00E+00	mg/kg
	Sodium	4/5	2.29E+02 - 3.10E+02	2.00E+02 - 2.00E+02	N	2.37E+02	mg/kg
	Strontium	5/5	1.96E+02 - 3.91E+02	2.002+02 * 2.002+02	N	2.92E+02	mg/kg
	Thallium	0/5	1.900,02 9.910,02	1.50E+01 - 1.50E+01	NT	7.50E+00	
	Vanadium	5/5	2.12E+00 - 6.70E+00	1.504+01 • 1.504+01	N	4.00E+00	mg/kg
	Zinc	5/5	4.59E+01 - 9.25E+01	•	N	4.00E+00 6.59E+01	mg/kg
				÷		0.552401	mg/kg
		Frequency	LOCATION=SWMU 194 MEDIA	=Subsurface Soil	·····		
		of	Detected	Nondetected		Arithmetic	
	Analyte	Detection	Range	Range	Distribution*	Mean	Units
	Malyce	Decection	Kange	Kange	DIBCT TOUL TOUL	Mean	onits
	Aluminum	12/12	5.38E+03 - 1.45E+04	•	L	9.47E+03	mg/kg
	Antimony	0/12		2.00E+01 ~ 2.00E+01	NT	1.00E+01	mg/kg
	Arsenic	1/12	6.73E+00 - 6.73E+00	5.00E+00 - 5.00E+00	N	2.85E+00	mg/kg
	Barium	12/12	2.05E+01 - 1.39E+02		N	7.51E+01	mg/kg
	Beryllium	6/12	5.40E-01 - 4.80E+00	5.00E-01 - 5.00E-01	L	7.48E-01	mg/kg
	Boron	0/12		1.00E+02 - 1.00E+02	NT	5.00E+01	mg/kg
	Cadmium	1/35	8.55E+00 - 8.55E+00	2.00E+00 - 5.00E+00	L	2.15E+00	mg/kg
	Calcium	12/12	5.68E+02 - 6.81E+03		L	1.67E+03	mg/kg
	Chromium	35/35	8.24E+00 - 1.03E+02		L	1.84E+01	mg/kg

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----- LOCATION=SWMU 194 MEDIA=Subsurface Soil ------(continued)

	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Chromium, hexavalent	0/12		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Cobalt	12/12	2.52E+00 - 9.46E+00		L	5.05E+00	mg/kg
Copper	12/12	2.41E+00 - 1.67E+01		L	7.49E+00	mg/kg
Cyanide	0/12		1.00E+00 - 1.00E+00	NT	5.00E-01	mg/kg
Iron	12/12	6.41E+03 - 2.00E+04		L	1.24E+04	mg/kg
Lead	20/35	5.03E+00 - 3.60E+02	5.00E+00 - 2.00E+01	L	1.19E+01	mg/kg
Lithium	12/12	2.41E+00 - 9.00E+00		Ň	6.45E+00	mg/kg
Magnesium	12/12	4.15E+02 ~ 2.34E+03		L	1.32E+03	mg/kg
Manganese	12/12	3.49E+01 - 4.67E+02		L	1.60E+02	mg/kg
Mercury	0/12		2.00E-01 - 2.00E-01	NT	1.00E-01	mg/kg
Nickel	8/12	5.74E+00 - 1.37E+01	5.00E+00 - 5.00E+00	N	7.22E+00	mg/kg
Potassium	12/12	1.53E+02 - 6.32E+02		L	3.80E+02	mg/kg
Selenium	0/12		1.00E+00 - 1.00E+00	NT	5.00Ê-01	mg/kg
Silver	0/12		4.00E+00 - 4.00E+00	NT	2.00E+00	mg/kg
Sodium	8/12	2.10E+02 - 3.69E+02	2.00E+02 - 2.00E+02	N L	2.37E+02	mg/kg
Strontium	12/12	3.92E+00 - 2.60E+01		L	1.29E+01	mg/kg
Thallium	0/12		1.50E+01 - 1.50E+01	NT	7.50E+00	mg/kg
Vanadium	12/12	1.50E+01 - 2.58E+01		Ĺ	1.99E+01	mg/kg
Zinc	11/12	1.57E+01 - 6.76E+01	1.50E+01 - 1.50E+01	L	3.82E+01	mg/kg
1,1,1-Trichloroethane	0/21		1.00E-03 - 1.00E-03	NT	5.00E-04	mg/kg
1,2-Dichloroethene	0/22		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/kg
Benzene	0/19		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/kg
Ethylbenzene	1/19	1.50E-02 - 1.50E-02	5.00E-03 - 5.00E-03	N	3.16E-03	mg/kg
Polychlorinated biphenyl	0/6		1.00E-01 - 1.00E-01	NT	1.00E-01	mg/kg
Toluene	0/19		5.00E-03 = 5.00E=03	NT	2.50E-03	mg/kg
Trichloroethene	0/22	•	1.00E-03 - 1.00E-03	NT	1.00E-03	mg/kg
Xylene	0/19		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/kg
Alpha activity	23/23	1.20E+00 - 2.50E+00	•.	L	1.85E+00	pCi/g
Beta activity	23/23	3.00E+00 - 7.00E+00		N	4.83E+00	pCi/g

---- LOCATION=SWMU 99A MEDIA=McNairy Groundwater -----

Analyte	of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
1,1,1-Trichloroethane 1,1-Dichloroethene 1,2-Dichloroethane	1/4 1/4 0/4	1.20E-03 - 1.20E-03 2.29E-02 - 2.29E-02	5.00E-03 - 5.00E-03 1.00E-02 - 1.00E-02 5.00E-03 - 2.00E+00	n N NT	2.18E-03 9.48E-03 5.01E-01	mg/L mg/L mg/L

*L=Lognormal, N=Normal, NT=Not tested

------ LOCATION=SWMU 99A MEDIA=McNairy Groundwater ------(continued)

	Frequency of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Carbon Tetrachloride	1/4	2.80E-03 - 2.80E-03	5.00E-03 - 5.00E-03	N	2.58E-03	mg/L
Chloroform	0/1	•	5.00E-02 - 5.00E-02	NT	2.50E-02	mg/L
Trichloroethene	3/4	3.00E-04 - 5.19E-01	5.00E-03 - 5.00E-03	Ń	1.31E-01	mg/L
Vinyl Chloride	0/1		1.00E-01 - 1.00E-01	NT	1.00E-01	mg/L
cis-1,2-Dichloroethene	2/4	2.80E-02 - 1.15E-01	2.00E+00 - 2.00E+00	Ň	1.04E+00	mg/L
trans-1,2-Dichloroethene	0/4		2.00E+00 - 2.00E+00	NT	2.00E+00	mg/L
Alpha activity	2/2	2.60E+00 - 2.90E+00		N	2.75E+00	pCi/L
Beta activity	2/2	2.30E+01 - 3.50E+01	•	Ň	2.90E+01	pCi/L
Technetium-99	2/2	1.00E+01 - 1.90E+01		N	1.45E+01	pCi/L

- LOCATION=SWMU 99A MEDIA=RGA Groundwater ----

	Frequency	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Aluminum	16/35	2.00E-01 - 6.59E+02	2.00E-01 - 1.00E+00	L	2.17E+00	mg/L
Antimony	0/19		6.00E-02 - 2.50E-01	NT	9.50E-02	mg/L
Arsenic	4/27	5.00E-03 - 1.00E-02	5.00E-03 - 5.00E-03	L	3.56E-03	mg/L
Barium	39/39	1.30E-01 - 3.30E+00		L	5.41E-01	mg/L
Beryllium	8/35	8.00E-03 - 1.00E-01	5.00E-03 - 2.50E-02	L	3.87E-03	mg/L
Boron	0/10		2.00E+00 - 2.00E+00	NT	1.00E+00	mg/L
Cadmium	0/19		5.00E-03 - 1.00E-01	NT	1.24E-02	mg/L
Calcium	39/39	2.10E+01 - 1.20E+02		L	4.14E+01	mg/L
Chloride	9/9	5.68E+01 - 1.20E+02		L	6.83E+01	mg/L
Chromium	11/39	6.00E-02 - 1.78E+00	5.00E-02 - 6.00E-02	L	4.39E-02	mg/L
Cobalt	20/37	1.00E-02 - 5.70E-01	1.00E-02 - 1.00E-01	L	5.64E-02	mg/L
Copper	9/35	7.00E-02 - 6.40E-01	1.00E-02 - 1.00E-01	L	3.18E-02	mg/L
Cyanide	0/30		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Fluoride	8/8	1.70B-01 - 2.00E-01		N	1.84E-01	mg/L
Iron	31/39	2.10E-01 - 1.20E+03	2.00E-01 - 3.55E-01	L	9.19E+00	mg/L
Lead	6/29	5.00E-02 - 4.10E-01	5.00E-02 - 2.50E-01	L	4.03E-02	mg/L
Lithium	6/23	5.00E-02 - 1.70E-01	5.00E-02 - 5.00E-02	L	4.45E-02	mg/L
Magnesium	39/39	8.38E+00 - 4.97E+01		L L	1.64E+01	mg/L
Manganese	37/39	4.30E-02 - 4.60E+00	1.00E-01 - 1.00E-01	` L	1.13E+00	mg/L
Mercury	5/25	2.00E-04 - 2.00E-02	2.00E-04 - 2.00E-04	L	6.33E-05	mg/L
Molybdenum	0/5		5.00E-02 - 1.00E-01	NT	3.50E-02	mg/L
Nickel	16/39	5.00E-02 - 9.10E-01	5.00E-02 - 1.00E-01	L	9.25E-02	mg/L
Nitrate as Nitrogen	7/9	1.00E+00 - 2.10E+00	1.00E+00 - 1.00E+00	N	1.12E+00	mg/L

----- LOCATION=SWMU 99A MEDIA=RGA Groundwater ----- LOCATION=SWMU 99A MEDIA=RGA Groundwater -----

	Frequency of	Detected	Nondetected		Arithmetic	
Apolyto	Detection	Range	Range	Distribution*	Mean	Units
Analyte	Decection	Kange	Range	DISCI IDUCION~	Mean	onics
Potassium	24/39	2.08E+00 - 2.17E+01	2.00E+00 - 1.05E+01	L	4.17E+00	mg/L
Selenium	0/17		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Silica	9/9	1.50E+01 - 2.50E+01		L	1.87E+01	mg/L
Silver	0/17		5.00E-02 - 6.00E-02	NT	2.62E-02	mg/L
Sodium	39/39	1.50E+01 - 7.24E+01		N	5.34E+01	mg/L
Strontium	30/30	1.20E-01 - 4.70E-01		N	2.49E-01	mg/L
Sulfate	2/2	1.75E+01 - 1.92E+01		N	1.84E+01	mg/L
Tetraoxo-sulfate(1-)	7/7	1.10E+01 - 2.20E+01		N	1.67E+01	mg/L
Thallium	0/12		6.00E-02 - 2.00E-01	NT	8.83E-02	mg/L
Total Phosphate as Phosphorus	0/3		2.00E+00 - 2.00E+00	NT	1.00E+00	mg/L
Uranium	0/11		1.00E-03 - 1.00E-03	NŤ	1.00E-03	mg/L
Vanadium	10/28	8.50E-02 - 2.15E+00	1.00E-01 - 1.00E-01	L	1.49E-01	mg/L
Zinc	10/35	1.10E-02 - 2.55E+00	3.00E-02 - 2.50E-01	L	8.96E-02	mg/L
1,1,1-Trichloroethane	0/25		5.00E-03 - 1.00E-01	NT	1.98E-02	mg/L
1,1,2,2-Tetrachloroethane	0/2		5.00E-02 - 1.00E-01	NT	3.75E-02	mg/L
1,1,2-Trichloroethane	0/19		5.00E-03 - 1.00E-01	NT	2.51E-02	mg/L
1,1-Dichloroethane	0/19		5.00E-03 - 1.00E-01	NT	2.51E-02	mg/L
1,1-Dichloroethene	7/33	8.00E-03 - 6.50E-02	1.00E-03 - 5.00E-02	N	1.70E-02	mg/L
1,2,4-Trichlorobenzene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
1,2-Dichlorobenzene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
1,2-Dichloroethane	0/25		5.00E-03 - 2.00E+00	NT	1.39E-01	mg/L
1,2-Dichloropropane	0/2	·	5.00E-02 - 1.00E-01	NT	3.75E-02	mg/L
1,2-Dimethylbenzene	0/2		5.00E-02 - 1.00E-01	NT	3.75E-02	mg/L
1,3-Dichlorobenzene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
1,4-Dichlorobenzene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2,4,5-Trichlorophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2,4,6-Trichlorophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2,4-Dichlorophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2,4-Dimethylphenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2,4-Dinitrophenol	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
2,4-Dinitrotoluene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2,6-Dinitrotoluene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2-Butanone	0/2		1.00E-01 - 2.00E-01	NT	7.50E-02	mg/L
2-Chloronaphthalene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2-Chlorophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2-Hexanone	0/2		1.00E-01 - 2.00E-01	NT	7.50E-02	mg/L
2-Methyl-4,6-dinitrophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2-Methylnaphthalene	0/10	•	2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2-Methylphenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
2-Nitroaniline	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
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*L=Lognormal, N=Normal, NT=Not tested

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(continued)

	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
2-Nitrophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
3,3'-Dichlorobenzidine	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
3-Nitroaniline	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
4-Bromophenyl phenyl ether	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
4-Chloro-3-methylphenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
4-Chloroaniline	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
4-Chlorophenyl phenyl ether	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
4-Methyl-2-pentanone	0/2		1.00E-01 - 2.00E-01	NT	7.50E-02	mg/L
4-Methylphenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
4-Nitroaniline	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
4-Nitrophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Acenaphthene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Acenaphthylene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Acetone	0/2		1.00E-01 - 2.00E-01	NT	7.50E-02	mg/L
Anthracene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Benz (a) anthracene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Benzene	0/19		5.00E-03 - 1.00E-01	NT	2.51E-02	mg/L
Benzo (a) pyrene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Benzo(b)fluoranthene	0/10		2.00E-02 - 2.00E-02	NT	1.002-02	mg/L
Benzo(ghi)perylene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Benzo(k) fluoranthene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Bromodichloromethane	0/19		5.00E-03 - 1.00E-01	NT	2.51E-02	mg/L
Bromoform	0/2		5.00E-02 - 1.00E-01	NT	3.75E-02	mg/L
Butyl benzyl phthalate	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Carbazole	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Carbon Disulfide	0/2		5.00E-02 - 1.00E-01	NT	3.75E-02	mg/L
Carbon Tetrachloride	0/25		5.00B-03 - 1.00E-01	NT	1.98E-02	mg/L
Chlorobenzene	0/2		5.00E-02 - 1.00E-01	NT	3.75E-02	mg/L
Chloroethane	0/2		5.00E-02 - 1.00E-01	NT	3.75E-02	mg/L
Chloroform	0/20		5.00E-03 - 1.00E-01	NT	2.41E-02	mg/L
Chloromethane	0/2		5.00E-02 - 1.00E-01	NT	3.75E-02	mg/L
Chrysene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Di-n-butylphthalate	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Di-n-octylphthalate	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Dibenz (a, h) anthracene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Dibenzofuran	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	
Dibromochloromethane	0/2		5.00E-02 - 1.00E-01	NT	3.75E-02	mg/L
Diethylphthalate	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Dimethylphthalate	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Ethylbenzene	0/19		5.00E-03 - 1.00E-01	NT	2.51E-02	mg/L
•			1.000 01		2.515-02	mg/L

*L=Lognormal, N=Normal, NT=Not tested

(continued)

	Frequency of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Fluoranthene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Fluorene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Hexachlorobenzene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Hexachlorobutadiene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Hexachlorocyclopentadiene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Hexachloroethane	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Indeno (1,2,3-cd) pyrene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Isophorone	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Methylene Chloride	0/2		1.00E-01 - 2.00E-01	NT	7.50E-02	mg/L
N-Nitroso-di-n-propylamine	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
N-Nitrosodiphenylamine	0/10		2.00E-02 - 2.00E-02	NT	1,00E-02	mg/L
Naphthalene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Nitrobenzene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Pentachlorophenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Phenanthrene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Phenol	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Polychlorinated biphenyl	0/2		1.70E-04 - 1.70E-04	NT	1.70E-04	mg/L
Pyrene	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
Pyridine	0/2		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L
Styrene	0/2		5.00E-02 - 1.00E-01	NT	3.75E-02	mg/L
Tetrachloroethene	0/19		5.00E-03 - 1.00E-01	NT	2.51E-02	mg/L
Toluene	0/19		5.00E-03 - 1.00E-01	NT	2.51E-02	mg/L
Trichloroethene	41/43	2.00E-04 - 2.37E+00	1.00E-03 - 5.00E-03	L	3.47E-01	mg/L
Vinyl Chloride	0/28		1.00E-03 - 1.00E-01	NT	4.94E-02	mg/L
Xylene	0/17		5.00E-03 - 1.00E-01	NT	3.69E-02	mg/L
bis (2-Chloroethoxy) methane	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
bis(2-Chloroethyl)ether	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
bis(2-Chloroisopropyl)ether	0/10		2.00E-02 - 2.00E-02	NT	1.00E-02	mg/L
bis (2-Ethylhexyl) phthalate	5/10	6.00E-03 - 1.60E-02	2.00E-02 - 2.00E-02	L	1.31E-02	mg/L
cis-1,2-Dichloroethene	10/33	3.00E-04 - 3.48E-02	1.00E-03 - 2.00E+00	L	3.70E-03	mg/L
cis-1,3-Dichloropropene	0/2		5.00E-02 - 1.00E-01	NT	3.75E-02	mg/L
m,p-Xylene	0/2		1.00E-01 - 2.00E-01	NT	7.50E-02	mg/L
trans-1,2-Dichloroethene	3/33	3.00E-04 - 6.00E-04	1.00E-03 - 2.00E+00	L	5.49E-04	mg/L
trans-1, 3-Dichloropropene	0/2		5.00E-02 - 1.00E-01	NT	3.75E-02	mg/L
Alpha activity	33/39	-2.50E+00 - 5.38E+01	-2.20E+00 - 2.60E+00	N	4.56E+00	pCi/L
Beta activity	39/39	3.00E+00 - 1.37E+02		Ľ	3.16E+01	pCi/L
Radon-222	4/4	2.86E+02 - 6.75E+02		N	4.75E+02	pCi/L
Technetium-99	34/40	3.00E+00 - 1.39E+02	4.10E+00 - 1.70E+01	L	3.48E+01	pC1/L
Temmertum 23	2 - ,	2,204,00 2,320,00		-	JIIUATUI	PCT\ T

*L=Lognormal, N=Normal, NT=Not tested

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----- LOCATION-SWMU 99A MEDIA-Subsurface Soil

Off Detection Name Name Range Distribution* Antimetic Aluminum 22/22 1.808+03 = 1.418+04 N 7.558±03 mg/kg Antimony 5/22 1.708+00 = 2.908+00 2.008+01 = 2.008+01 L 2.168+00 mg/kg Arsenic 11/22 2.038+01 = 2.478+03 5.008+00 = 5.008+00 N 4.228+00 mg/kg Barlum 22/22 2.038+01 = 2.478+03 5.008+01 = 5.008+02 N 5.008+02 mg/kg Barlum 22/22 7.008+01 = 6.3008-01 5.008+00 N 6.338+02 mg/kg Cadaium 5/22 7.008+02 2.008+00 L 6.368+00 N 6.308+00 1.008+00 N 6.308+00 mg/kg Cobalt .20/22 1.668+00 1.008+00 1.008+00 N 7.318+00 mg/kg Copper 21/22 1.668+00 1.008+00 1.008+00 N 7.318+00 mg/kg Itead 0.721 1.6688+00 1.008+00 1.008+00		Frequency					
Analyte Detection Range Range Distribution* Mean Units Aluminum 22/22 1.080+03 = 1.418+04 N 7.555+03 mg/kg Antimony 5/22 1.080+00 = 2.905+00 2.002+01 = 2.002+00 N 4.222+00 mg/kg Arsenic 11/22 2.032+01 = 2.4075+00 5.002+00 = 5.002+00 N 4.222+00 mg/kg Berrin 22/22 2.032+01 = 8.902-01 5.002+00 = 2.002+00 L 1.532+02 mg/kg Berrin 21/22 7.002+01 = 8.302-01 2.002+00 = 2.002+00 L 8.192-01 mg/kg Cadadum 5/22 7.002+00 = 4.578+01 L 1.662+03 mg/kg Cabalt 20/22 7.002+00 = 4.578+01 L 1.442+01 mg/kg Cabalt 20/22 1.482+01 = 3.202+01 1.002+00 = 1.002+00 N 7.932+01 mg/kg Cabalt 20/22 1.442+01 = mg/kg 1.002+00 1.002+00 N 1.932+01 mg/kg Cabalt 20/22 1.442			Detected	Nondetected		Arithmetic	
Artenic 5/22 1.70E+00 2.90E+00 2.00E+01 L 2.16E+00 mg/kg Barjum 22/22 2.03E+01 2.47E+03 L 1.53E+02 mg/kg Baryllum 11/22 2.02E+01 8.90E+01 5.00E+00 N.0E+02 N.0E+02 N.0E+02 N.0E+02 N.0E+02 N.0E+02 N.0E+02 N.0E+02 N.0E+02 N.0E+02 N.0E+02 N.0E+02 N.0E+02 N.0E+02 N.0E+02 N.0E+02 N.0E+01 Mg/kg Cadnium 20/20 1.14E+03 2.0FF+01 2.0DE+00 L 8.19E+01 mg/kg Chromium 20/22 1.0EE+00 -1.12E+01 1.0E+00 1.0E+00 N 7.33E+00 mg/kg Copper 21/22 3.0E+00 -1.6E+01 2.0DE+00 N 4.99E+01 mg/kg Lithium 17/17 2.0E2+00 -1.29E+01 N 1.4EE+00 mg/kg Ladd 6/22 3.03E+00 -2.00E+01 2.00E+01 L 1.3EE+01 mg/kg	Analyte				Distribution*		Units
Artenic 5/22 1.70E+00 2.90E+00 2.00E+01 L 2.16E+00 mg/kg Barjum 22/22 2.03E+01 2.47E+03 L 1.53E+02 mg/kg Baryllum 11/22 2.02F+01 8.90E+01 5.00E+00 N.53E+02 mg/kg Boron 0/17 2.02F+01 8.90E+01 5.00E+02 NT 5.00E+01 mg/kg Cadnium 5/22 7.50E+01 -8.30E+01 2.00E+00 L 8.19E+01 mg/kg Chromium 20/20 1.14E+03 2.07E+01 L 1.00E+00 1.07E+00 L 1.44E+01 mg/kg Cobalt 20/22 1.68E+00 1.12E+01 1.00E+00 1.00E+00 L 4.97E+00 mg/kg Copper 21/22 3.60E+00 1.46E+01 2.00E+00 N 4.97E+01 mg/kg Lithium 17/17 2.02E+01 1.29E+01 L 1.36E+01 mg/kg Lithium 17/17 2.20E+01 1.29E+01 L		22/22					(1
Arrenic 11/22 2.408+00 e.558+00 5.008+00 N 4.228+00 mg/kg Barjum 22/22 2.038+00 2.478+03 L 1.538+02 mg/kg Boron 0/17 1.008+00 1.008+00 N 4.328=01 mg/kg Cadnium 5/21 7.508-01 8.308-01 2.008+00 2.008+00 L 8.198=01 mg/kg Calcium 20/20 1.148403 2.078+05 L 1.668+05 mg/kg Cobalt 20/22 1.688+00 1.158+01 1.008+00 1.088+00 N 4.398+01 mg/kg Copper 21/22 1.688+01 1.108+00 1.008+00 N 4.398+01 mg/kg Iron 22/22 1.458+03 2.338+04 1.008+00 N 1.988+04 mg/kg Lithium 17/17 2.828+00 1.238+01 2.008+01 2.008+01 1.088+00 mg/kg Margasium 22/22 3.978+02 2.738+01 2.008+01 2.008+01 1.308+01 mg/kg Margasium 22/22 3.978+02				2 000 01 2 000 01	Ň.		
Barylium 22/22 2.032+01 2.472F403 L 1.532+02 mg/kg Berynlium 11/22 2.002+01 6.902-01 5.003+01 5.003+01 N 4.322-01 mg/kg Boron 0/17 1.002+02 NT 5.002+01 K.902+01					Ϋ́,		
Berynlium 11/22 2.202-01 5.002-01 N 4.328-01 mg/kg Boron 1.008+02 1.008+02 NT 5.008-01 Mg/kg Cadnium 5/22 7.502-01 8.302-01 2.008+02 NT 5.008-01 mg/kg Calcium 22/22 7.502-01 4.578+01 1.008+00 1.008+00 1.4848-01 Mg/kg Cobalt 22/22 1.688+00 1.198+01 1.008+00 1.008+00 N 7.318+00 mg/kg Copper 21/22 1.688+00 -1.389+01 1.008+00 N 1.388+01 mg/kg Iron 22/22 1.458+03 -2.338+01 N 1.388+01 mg/kg Magnesium 22/22 3.978+02 2.378+04 2.008+01 -2.008+01 N 9.958-02 mg/kg Magnesium 22/22 3.978+02 2.008+01 -2.008+01 N 9.958-02 mg/kg Magnesium 22/22 2.828+00 1.128+03 L 6.498+03				5.008+00 - 5.008+00			
Dermin 0/17 1.008+02 NT 5.00E+01 mg/kg Cadnum 20/20 1.348+03 2.07E+01 2.00E+00 L 8.19E-01 mg/kg Chronium 20/20 1.348+03 2.07E+01 L 1.66E+05 mg/kg Cobalt 20/22 1.66E+06 1.19E+01 1.00E+00 L 4.97E+00 mg/kg Coper 21/22 1.66E+00 1.19E+01 1.00E+00 1.00E+00 N 7.31E+00 mg/kg Cymide 21/22 1.65E+03 2.33E+04 N 1.85E+04 mg/kg Lithium 17/7 2.22E+00 1.29E+01 2.00E+01 2.00E+01 L 7.85E+00 mg/kg Marganese 22/22 3.97E+02 2.73E+00 N 9.95E+02 mg/kg Marganese 22/22 3.97E+02 2.73E+00 N 9.95E+02 mg/kg Mercury 5/22 6.00E+02 1.20E+01 2.00E+01 N 9.95E+02 mg/kg					Ц. Х.		
Calcium 5/22 7.502-01 - 8.302-01 2.002+00 L 8.192-01 mg/kg Calcium 22/22 7.002+00 - 4.57E+01 L 1.66E+05 mg/kg Cobalt 20/22 7.002+00 - 4.57E+01 L 1.44E+01 mg/kg Copper 21/22 3.80E+00 - 1.19E+01 1.00E+00 1.00E+00 N 7.31E+00 mg/kg Cyanide 21/22 1.45E+03 - 2.33E+04 N 1.98E+01 mg/kg Lithium 22/22 1.45E+03 - 2.33E+04 N 1.36E+01 mg/kg Magnesime 22/22 1.45E+03 - 2.33E+04 L 7.36E+04 N 1.36E+01 mg/kg Lithium 17/17 2.622+02 1.23E+01 1.00E+01 2.00E+01 2.00E+01 N 9.55E-02 mg/kg Margnesime 22/22 3.97E+02 r.7.3E+04 L 3.02E+02 mg/kg Margnesime 22/22 3.97E+02 r.92E+01 S.00E+00	-		2.20E-01 - 8.90E-01				
Clinim 20/20 1.148+03 - 2.078+05 L 1.668+05 mg/kg Chromium 22/22 1.08400 - 1.198+01 1.002+00 1.04400 1.448+01 3.002+00 N 4.972+00 mg/kg Copper 21/22 3.602+00 - 1.198+01 2.002+00 N 4.972+00 mg/kg Cyanide 21/22 3.602+00 - 1.092+00 N 4.972+00 mg/kg Iron 22/22 1.458+03 - 2.338+04 N 1.188+04 mg/kg Lead 6/22 7.008+00 - 4.738+01 2.002+01 2.002+01 1.3658+01 mg/kg Marganese 22/22 3.938+01 - 1.485+03 - 6.4958+03 mg/kg Marganese 22/22 3.938+01 - 1.465403 - 5.002+00 1.002+00 L 5.022+02 mg/kg Nickel 17/22 2.028+02 1.202+01 2.002+01 2.028+00 1.002+00 L 5.1202+02 mg/kg Sclenium							
Chromium 22/22 7.008+00 - 4.578+01 L 1.448+01 wg/kg Coppir 21/22 3.688+00 - 1.198+01 1.008+00 - 1.008+00 N 7.318+00 mg/kg Cymride 21/22 3.688+00 - 1.648+01 2.008+00 - 1.008+00 N 7.318+00 mg/kg Cymride 21/22 1.458+03 - 2.338+01 2.008+01 - 2.008+01 N 1.988+04 mg/kg Lichium 17.07 2.028+00 - 1.228+01 2.008+01 - 2.008+01 L 1.368+03 mg/kg Magnaesim 22/22 3.978+02 - 2.738+04 L 6.498+03 mg/kg Marganese 22/22 3.978+02 - 2.738+04 L 0.082+01 9.088+03 mg/kg Marganese 22/22 3.978+02 - 2.588+01 5.008+00 - 5.008+00 J 9.988+02 mg/kg Marganese 22/22 2.088+01 - 3.208+01 5.008+00 J 9.988+02 mg/kg Marganese 22/22 2.288+02 1.208+01 1.008+00 J 9.988+02 mg/kg Stortium				2.00E+00 - 2.00E+00	L		
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Selenium 5/20 2.90E-01 - 3.20E-01 1.00E+00 - 1.00E+00 L 3.11E-01 mg/kg Silver 5/22 6.40E-01 - 7.10E-01 4.00E+00 - 4.00E+00 L 6.99E-01 mg/kg Sodium 14/22 6.63E+01 - 3.93E+02 2.00E+02 - 2.51E+02 N 2.14E+02 mg/kg Strontium 17/17 8.88E+00 - 5.14E+02 L 2.25E+02 mg/kg Thallium 5/22 5.30E-01 - 5.90E-01 1.50E+01 - 1.50E+01 L 5.75E-01 mg/kg Vanadium 22/22 4.48E+00 + 3.55E+01 N 1.79E+01 mg/kg 1,1,1-Trichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 N 5.63E-03 mg/kg 1,1,2-Trichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 N 5.63E-03 mg/kg 1,2-Trichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 N 5.63E-03 mg/kg 1,2-Dichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 N 5.63E-03 mg/kg 1,2-Dichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 N 5.63E-0	Nickel	17/22	2.50E+00 - 2.58E+01	5.00E+00 - 5.00E+00	Ľ,	9.72E+00	mg/kg
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Sodium 14/22 6.63E+01 - 3.93E+02 2.00E+02 - 2.51E+02 N 2.14E+02 mg/kg Strontium 17/17 8.88E+00 - 5.14E+02 L 2.25E+02 mg/kg Thallium 5/22 5.30E-01 - 5.90E-01 1.50E+01 - 1.50E+01 L S.75E-01 mg/kg Vanadium 22/22 4.48E+00 - 3.55E+01 N 1.79E+01 mg/kg Zinc 21/22 1.16E+01 - 1.63E+02 4.76E+01 - 4.76E+01 L 6.96E+01 mg/kg 1,1,2.7=Trichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 - 1.00E-02 N 5.63E-03 mg/kg 1,1.2=Trichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 - 1.00E-02 N 5.63E-03 mg/kg 1,1-Dichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 - 1.00E-02 N 5.63E-03 mg/kg 1,1-Dichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 N 5.63E-03 mg/kg 1,2-Dichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 N 5.63E-03 mg/kg 1,2-Dichloroethane 5/22 3.60E-01 - 4.10E-01 5.00E-01	Selenium	5/20	2.90E-01 - 3.20E-01	1.00E+00 - 1.00E+00	· L	3.11E-01	
Sodium 14/22 6.63E+01 - 3.93E+02 2.00E+02 - 2.51E+02 N 2.14E+02 mg/kg Strontium 17/17 8.88E+00 - 5.14E+02 L 2.25E+02 mg/kg Thallium 5/22 5.30E-01 - 5.90E-01 1.50E+01 - 1.50E+01 L S.75E-01 mg/kg Vanadium 22/22 4.48E+00 - 3.55E+01 N 1.79E+01 mg/kg Zinc 21/22 1.16E+01 - 1.63E+02 4.76E+01 - 4.76E+01 L 6.96E+01 mg/kg 1,1,2.7=Trichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 - 1.00E-02 N 5.63E-03 mg/kg 1,1.2=Trichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 - 1.00E-02 N 5.63E-03 mg/kg 1,1-Dichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 - 1.00E-02 N 5.63E-03 mg/kg 1,1-Dichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 N 5.63E-03 mg/kg 1,2-Dichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 N 5.63E-03 mg/kg 1,2-Dichloroethane 5/22 3.60E-01 - 4.10E-01 5.00E-01	Silver	5/22	6.40E-01 - 7.10E-01	4.00E+00 - 4.00E+00	L	6.99E-01	
Strontium 17/17 8.88E+00 = 5.14E+02 L 2.25E+02 mg/kg Thallium 5/22 5.30E-01 = 5.90E-01 1.50E+01 = 1.50E+01 L 5.75E-01 mg/kg Vanadium 22/22 4.48E+00 = 3.55E+01 N 1.79E+01 M 5.75E-01 mg/kg Zinc 21/22 1.16E+01 = 1.63E+02 4.76E+01 = 4.76E+01 L 6.96E+01 mg/kg 1,1,2-Trichloroethane 5/8 6.00E-03 = 6.00E-03 1.00E-02 = 1.00E-02 N 5.63E-03 mg/kg 1,1,2-Trichloroethane 5/8 6.00E-03 = 6.00E-03 1.00E-02 = 1.00E-02 N 5.63E-03 mg/kg 1,1-Dichloroethane 5/8 6.00E-03 = 6.00E-03 1.00E-02 = 1.00E-02 N 5.63E-03 mg/kg 1,1-Dichloroethane 5/8 6.00E-03 = 6.00E-03 1.00E-02 = 1.00E-02 N 5.63E-03 mg/kg 1,2-Arrichloroethane 5/10 6.00E-03 = 6.00E-03 1.98E-01 = 5.27E-01 N 8.91E-02 mg/kg 1,2-Dichloroethane 5/22 3.60E-01 = 4.10E-01 5.00E-01 = 5.00E-01 N 2.83E-01 mg/kg 1,2-Dichloroethane	Sodium	14/22	6.63E+01 - 3.93E+02	2.00E+02 - 2.51E+02			
Thallium 5/22 5.30È-01 5.90È-01 1.50È+01 1 5.75È-01 mg/kg Vanadium 22/22 4.48E+00 3.55È+01 N 1.79È+01 mg/kg Zinc 21/22 1.16E+01 = 1.63E+02 4.76E+01 - 4.76E+01 L 6.96E+01 mg/kg 1,1,1-Trichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 - 1.00E-02 N 5.63E-03 mg/kg 1,1,2-Trichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 - 1.00E-02 N 5.63E-03 mg/kg 1,1,2-Trichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 - 1.00E-02 N 5.63E-03 mg/kg 1,1-Dichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 - 1.00E-02 N 5.63E-03 mg/kg 1,1-Dichloroethane 5/8 6.00E-03 - 6.00E-03 1.00E-02 - 1.00E-02 N 5.63E-03 mg/kg 1,2,2-Trichlorobenzene 5/22 3.60E-01 - 4.10E-01 5.00E-01 - 5.00E-01 N 2.83E-01 mg/kg 1,2-Dichlorobenzene 5/22 3.60E-01 - 4.10E-01 5.00E-01 - 5.00E-01 N 2.83E-01 mg/kg 1,2-Dich	Strontium	17/17	8.88E+00 - 5.14E+02		· L		
Vanadium 22/22 4.48E+00 ÷ 3.55E+01 N 1.79E+01 mg/kg Zinc 21/22 1.16E+01 ÷ 1.63E+02 4.76E+01 – 4.76E+01 L 6.96E+01 mg/kg 1,1,1-Trichloroethane 5/8 6.00E-03 ÷ 6.00E-03 1.00E-02 ÷ 1.00E-02 N 5.63E-03 mg/kg 1,1,2-Trichloroethane 5/8 6.00E-03 ÷ 6.00E-03 1.00E-02 ÷ 1.00E-02 N 5.63E-03 mg/kg 1,1,2-Trichloroethane 5/8 6.00E-03 ÷ 6.00E-03 1.00E-02 ÷ 1.00E-02 N 5.63E-03 mg/kg 1,1-Dichloroethane 5/8 6.00E-03 ÷ 6.00E-03 1.00E-02 ÷ 1.00E-02 N 5.63E-03 mg/kg 1,1-Dichloroethane 5/8 6.00E-03 ÷ 6.00E-03 1.00E-02 ÷ 1.00E-02 N 5.63E-03 mg/kg 1,2,4-Trichloroethane 5/8 6.00E-03 ÷ 6.00E-03 1.00E-02 × 1.00E-02 N 8.91E-02 mg/kg 1,2,2-Dichloroethane 5/22 3.60E-01 + 4.10E-01 5.00E-01 × 5.00E-01 N 2.83E-01 mg/kg 1,2-Dichloroethane 5/2 3.60E-03 × 6.00E-03 1.00E-02 × 1.00E-02 N 5.63E-03 mg/kg 1,2-Dichloroeth	Thallium		5.308-01 - 5.908-01	1.50E+01 - 1.50E+01	Ľ		
Zinc21/221.16E+01 = 1.63E+024.76E+01 - 4.76E+01L6.96E+01mg/kg1,1,1-Trichloroethane5/86.00E-03 - 6.00E-031.00E-02 - 1.00E-02N5.63E-03mg/kg1,1,2-Trichloroethane5/86.00E-03 - 6.00E-031.00E-02 - 1.00E-02N5.63E-03mg/kg1,1,2-Trichloroethane5/86.00E-03 - 6.00E-031.00E-02 - 1.00E-02N5.63E-03mg/kg1,1-Dichloroethane5/86.00E-03 - 6.00E-031.00E-02 - 1.00E-02N5.63E-03mg/kg1,1-Dichloroethane5/86.00E-03 - 6.00E-031.00E-02 - 1.00E-02N5.63E-03mg/kg1,1-Dichloroethane5/86.00E-03 - 6.00E-031.98E-01 - 5.27E-01N8.91E-02mg/kg1,2,4-Trichloroethane5/223.60E-01 - 4.10E-015.00E-01 - 5.00E-01N2.83E-01mg/kg1,2-Dichloroethane5/86.00E-03 - 6.00E-031.00E-02 - 1.00E-02N5.63E-03mg/kg1,2-Dichloroethane5/86.00E-03 - 6.00E-031.90E-01 - 5.00E-01N2.83E-01mg/kg1,2-Dichloroethane5/86.00E-03 - 6.00E-031.00E-02 - 1.00E-02N5.63E-03mg/kg1,2-Dichloroethane5/86.00E-03 - 6.00E-031.00E-02 - 1.00E-02N5.63E-03mg/kg1,2-Dichloroethane5/86.00E-03 - 6.00E-031.00E-02 - 1.00E-02N5.63E-03mg/kg1,2-Dichloroethane5/86.00E-03 - 6.00E-031.00E-02 - 1.00E-02N5.63E-03mg/kg <td< td=""><td></td><td></td><td>4.48E+00 - 3.55E+01</td><td></td><td></td><td></td><td></td></td<>			4.48E+00 - 3.55E+01				
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1,4-Dichlorobenzene 5/22 3.60E-01 - 4.10E-01 5.00E-01 - 5.00E-01 N 2.83E-01 mg/kg							
2,4,5-Trichlorophenol 5/22 1.80E+00 - 2.10E+00 5.00E-01 - 5.00E-01 N 6.43E-01 mg/kg							
	2,4,5-Trichlorophenol	5/22	1.80E+00 - 2.10E+00	5.00E-01 - 5.00E-01	N	6.43E-01	mg/kg

*L=Lognormal, N=Normal, NT=Not tested

----- LOCATION=SWMU 99A MEDIA=Subsurface Soil ----- (continued)

	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
	5/22		5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
2,4,6-Trichlorophenol	5/22	3.60E-01 - 4.10E-01 3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01 2.83E-01	mg/kg
2,4-Dichlorophenol			5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
2,4-Dimethylphenol	5/22	3.60E-01 - 4.10E-01		N	1.69E+00	mg/kg
2,4-Dinitrophenol	5/6	1.80E+00 - 2.10E+00	4.80E-01 - 4.80E-01	NN		
2,4-Dinitrotoluene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01		2.83E-01	mg/kg
2,6-Dinitrotoluene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
2-Butanone	5/8	6.00E-03 - 1.20E-02	1.00E-02 - 2.50E-01	L	1.15E-02	mg/kg
2-Chloronaphthalene	5/22	3.60E-01 - 4.10E-01	5.DOE-01 - 5.00E-01	N	2.83E-01	mg/kg
2-Chlorophenol	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
2-Hexanone	5/8	1.10E-02 - 1.20E-02	1.00E-02 - 1.00E-02	N	9.25E-03	mg/kg
2-Methyl-4,6-dinitrophenol	5/22	1.80E+00 - 2.10E+00	5.00E-01 - 5.00E-01	N	6.43E-01	mg/kg
2-Methylnaphthalene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
2-Methylphenol	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
2-Nitroaniline	5/22	1.80E+00 - 2.10E+00	5.00E-01 - 5.00E-01	Ν.	6.43E-01	mg/kg
2-Nitrophenol	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
3,3'-Dichlorobenzidine	5/22	7.20E-01 - 8.20E-01	5.00E-01 - 5.00E-01	N	3.73E-01	mg/kg
3-Nitroaniline	5/22	1.80E+00 - 2.10E+00	5.00E-01 - 5.00E-01	N	6.43E-01	mg/kg
4,4'-DDD	2/2	2.00E-02 - 3.50E-02		N	2.75E-02	mg/kg
4,4'-DDE	2/2	2.00E-02 - 3.50E-02		N	2.75E-02	mg/kg
4,4'-DDT	2/2	2.00E-02 - 3.50E-02		N	2.75E-02	mg/kg
4-Bromophenyl phenyl ether	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
4-Chloro-3-methylphenol	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
4-Chloroaniline	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
4-Chlorophenyl phenyl ether	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
4-Methyl-2-pentanone	5/8	1.10E-02 - 1.20E-02	1.00E-02 - 2.50E-01	L	1.19E-02	mg/kg
4-Methylphenol	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
4-Nitroaniline	5/22	1.80E+00 - 2.10E+00	5.00E-01 - 5.00E-01	N	6.43E-01	mg/kg
4-Nitrophenol	5/22	1.80E+00 - 2.10E+00	5.00E-01 - 5.00E-01	N	6.43E-01	mg/kg
Acenaphthene	7/22	3.00E-01 - 4.10E-01	5.00E-01 - 5.00E-01	L	3.89E-01	mg/kg
Acenaphthylene	6/22	3.60E-01 - 6.10E-01	5.00E-01 - 5.00E-01	L	4.32E-01	mg/kg
Acetone	5/8	1.20E-02 - 5.30E-02	1.00E-02 - 2.50E-01	L	2.47E-02	mg/kg
Aldrin	2/2	9.80E-03 - 1.70E-02		N	1.34E-02	mg/kg
Anthracene	7/22	3.60E-01 - 7.50E-01	5.00E-01 - 5.00E-01	L	4.42E-01	mg/kg
Benz (a) anthracene	8/22	2.20E-01 - 1.70E+00	5.00E-01 - 5.00E-01	L	4.40E-01	mg/kg
Benzene	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	Ñ	5.63E-03	mg/kg
Benzo (a) pyrene	7/22	3.60E-01 - 2.10E+00	5.00E-01 - 5.00E-01	L	4.38E-01	mg/kg
Benzo (b) fluoranthene	11/22	1.70E-01 - 5.70E+00	5.00E-01 - 5.00E-01	Ľ	5.03E-01	mg/kg
Benzo (d) fiuoranchene Benzo (dhi) perylene	7/22	3.60E-01 - 1.18E+00	5.00E-01 - 5.00E-01	L	4.40E-01	mg/kg
Benzo (ghi) peryiene Benzo (k) fluoranthene	8/22	3.60E-01 - 7.90E-01	5.00E-01 - 5.00E-01	L	4.52E-01	mg/kg
Benzoic Acid	5/5	1.80E+00 - 2.10E+00	5.002-01 5.002-01	N	1.98E+00	
Benzolc Acia	5/5	1.808400 - 2.108400		14	1.305+00	mg/kg

*L=Lognormal, N=Normal, NT=Not tested

-- LOCATION-SWMU 99A MEDIA-Subsurface Soil -----(continued)

· · · ·	Frequency	8 - 1 - 1 - 1 - 1 - 1 - 1 - 1 - 1 - 1 -		•		
-	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Benzyl Alcohol	5/5	3.60E-01 - 4.10E-01		N	3.94E-01	mg/kg
Bromodichloromethane	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Bromoform	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Bromomethane	5/8	1.10E-02 - 1.20E-02	1.00E-02 - 2.00E-02	N	1.05E-02	mg/kg
Butyl benzyl phthalate	5/6	3.60E-01 - 4.10E-01	4.80E-01 - 4.80E-01	N ·	3.68E-01	mg/kg
Carbazole	0/17		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Carbon Disulfide	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Carbon Tetrachloride	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Chlorobenzene	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	. N	5.63E-03	mg/kg
Chloroethane	5/8	1.10E-02 - 1.20E-02	1.00E-02 - 2.00E-02	N	1.05E-02	mg/kg
Chloroform	. 5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Chloromethane	5/8	1.10E-02 - 1.20E-02	1.00E-02 - 2.00E-02	N	1.05E-02	mg/kg
Chrysene	7/22	3.60E-01 - 2.10E+00	5.00E-01 - 5.00E-01	L	4.38E-01	mg/kg
Di-n-butylphthalate	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Di-n-octylphthalate	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Dibenz (a, h) anthracene	6/22	3.60E-01 - 4.80E-01	5.00B-01 - 5.00E-01	N	2.93E-01	mg/kg
Dibenzofuran	6/22	1.23E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.77E-01	mg/kg
Dibromochloromethane	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Dichlorodifluoromethane	0/2	• •.	2.00E-02 - 2.00E-02	NT	1.00E-02	mg/kg
Dieldrin	2/2	2.00E-02 - 3.50E-02		N	2.75E-02	mg/kg
Diethylphthalate	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	ng/kg
Dimethylphthalate	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Endosulfan I	2/2	9.80E-03 - 1.70E-02		N	1.34E-02	mg/kg
Endosulfan II	2/2	2.00E-02 - 3.50E-02		Ň	2.75E-02	mg/kg
Endosulfan Sulfate	2/2	2.00E-02 - 3.50E-02		N	2.75E-02	mg/kg
Endrin	2/2	2.00E-02 - 3.50E-02	•	N	2.75E-02	mg/kg
Endrin Ketone	2/2	2.00E-02 - 3.50E-02		N	2.75E-02	mg/kg
Ethylbenzene	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Fluoranthene	9/22	1.40E-01 - 2.66E+00	5.00E-01 - 5.00E-01	Ĺ	4.23E-01	mg/kg
Fluorene	6/22	2.19E-01 - 4.10E-01	5.00E-01 - 5.00E-01	Ĩ	3.80E-01	mg/kg
Heptachlor	2/2	9.80E-03 - 1.70E=02		N	1.34E-02	mg/kg
Heptachlor Epoxide	2/2	9.80E-03 - 1.70E-02		N	1.34E-02	mg/kg
Hexachlorobenzene	5/22	3.60B-01 - 4.10E-01	5.00E-01 - 5.00E-01	Ň	2.83E-01	mg/kg
Hexachlorobutadiene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Hexachlorocyclopentadiene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Hexachloroethane	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	Ň	2.83E-01	mg/kg
Indeno (1,2,3-cd) pyrene	7/22	3.60E-01 - 1.05E+00	5.00E-01 - 5.00E-01	· L	4.46E-01	mg/kg
Isophorone	5/22	3.60E-01 - 4.10E-01	5.00E-01 = 5.00E-01	Ň	2.83E-01	mg/kg
Methoxychlor	2/2	9.80E-02 - 1.70E-01		N	1.34E-01	mg/kg
Methylene Chloride	5/8	2.00E-03 - 8.00E-03	1.00E-02 - 1.00E-02	L	5.20E-03	mg/kg
HECHATENE CUTOTICE	5/0	2.000-03 - 0.000-03	T. 000 02 - T. 000 02	<u>ب</u>	3.205-03	mg/kg

*L=Lognormal, N=Normal, NT=Not tested

----- LOCATION=SWMU 99A MEDIA=Subsurface Soil ----- LOCATION=SWMU 99A MEDIA=Subsurface Soil -----

	Frequency of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
N-Nitroso-di-n-propylamine	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
N-Nitrosodiphenylamine	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Naphthalene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Nitrobenzene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
PCB-1016	3/23	9.80E-02 - 1.87E+00	1.02E-01 - 1.28E-01	L	2.54E-02	mg/kg
PCB-1221	2/23	9.80E-02 - 1.70E-01	1.02 E- 01 - 5.45E-01	L	7.82E-02	mg/kg
PCB-1232	2/23	9.80E-02 - 1.70E-01	1.02E-01 - 5.45E-01	L	7.82E-02	mg/kg
PCB-1242	2/23	9.80E-02 - 1.70E-01	1.02E-01 - 5.45E-01	L	7.82E-02	mg/kg
PCB-1248	2/23	9.80E-02 - 1.70E-01	1.02E-01 - 5.45E-01	L	7.82E-02	mg/kg
PCB-1254	3/23	9.60E-02 - 3.50E-01	1.02E-01 - 5.45E-01	L	5.92E-02	mg/kg
PCB-1260	7/23	6.00E-02 - 6.31E-01	1.02E-01 - 5.45E-01	L	1.04E-01	mg/kg
PCB-1268	0/1		1.00E-01 - 1.00E-01	NT	1.00E-01	mg/kg
Pentachlorophenol	5/22	1.80E+00 - 2.10E+00	5.00E-01 - 5.00E-01	N	6.43E-01	mg/kg
Phenanthrene	7/22	3.60E-01 - 1.63E+00	5.00E-01 - 5.00E-01	L	4.41E-01	mg/kg
Phenol	5/22	3.60E-01 ~ 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Polychlorinated biphenyl	0/1		1.00E-01 - 1.00E-01	NT	1.00E-01	mg/kg
Pyrene	8/22	1.30E-01 - 2.70E+00	5.00E-01 - 5.00E-01	L	4.25E-01	mg/kg
Pyridine	0/1		4.80E-01 - 4.80E-01	NT	2.40E-01	mg/kg
Styrene	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Tetrachloroethene	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Toluene	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03.	mg/kg
Toxaphene	2/2	2.00E-01 - 3.50E-01		N	2.75E-01	mg/kg
Trichloroethene	6/10	4.80E-03 - 6.00E-03	1.98E-01 - 5.27E-01	N	1.36E-01	mg/kg
Vinyl Acetate	5/5	1.10E-02 - 1.20E-02		N	1.18E-02	mg/kg
Vinyl Chloride	5/10	1.10E-02 - 1.20E-02	1.98E-01 - 1.00E+01	L	1.20E-02	mg/kg
Xylene	5/5	4.00E-03 - 6.00E-03		N	5.60E-03	mg/kg
alpha-BHC	2/2	9.80E-03 - 1.70E-02		N	1.34E-02	mg/kg
alpha-Chlordane	2/2	9.80E-02 - 1.70E-01		N	1.34E-01	mg/kg
beta-BHC	2/2	9.80E-03 - 1.70E-02		N	. 1.34E-02	mg/kg
bis(2-Chloroethoxy)methane	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
bis (2-Chloroethyl) ether	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
bis (2-Chloroisopropyl) ether	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
bis(2-Ethylhexyl)phthalate	5/22	7.90E-02 - 3.60E-01	5.00E-01 - 5.00E-01	N	2.37E-01	mg/kg
cis-1,2-Dichloroethene	0/5		1.98E-01 - 5.27E-01	NT	3.44E-01	mg/kg
cis-1,3-Dichloropropene	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
delta-BHC	2/2	9.80E-03 - 1.70E-02		N	1.34E-02	mg/kg
gamma-BHC (Lindane)	2/2	9.80E-03 - 1.70E-02		N	1.34E-02	mg/kg
gamma-Chlordane	2/2	9.80E-02 - 1.70E-01		N	1.34E-01	mg/kg
m,p-Xylene	0/3		1.00E-02 - 2.00E-02	NT	6.67E-03	mg/kg
trans-1,2-Dichloroethene	0/5		1.98E-01 - 5.27E-01	NT	3.44E-01	mg/kg

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 LOCATION-SWMU	99A MEDIA-Subsurface	Soil
	(continued)	

ection Ra /8 6.00E-03 0/21 9.70E+00 /21	0 - 1.42E + 02	Nondetected Range 1.00E-02 - 1.000 3.10E+00 - 3.100		Arithmetic Mean 5.63E-03	Units
0/21 9.70E+00 /21	0 - 1.42E + 02			5.63E-03	ma/ka
/21		3.10E+00 = 3.10I	F. 66		
			E+00 L	2.33E+01	pCi/g
1	· · ·	1.10E+00 - 1.30	E+01 NT	5.70E+00	pCi/g
L/21 6.70B+00	0 - 2.73E+03		L	6.15E+01	pCi/g
/21 1.10E+00	0 - 1.90E+00	3.80E-01 - 3.50	E+00 L	3.77E-01	pCi/g
/21 -		5.20E-01 - 4.60	E+00 NT	1.43E+00	pCi/g
4 -2.00E-03	3 - 1.28E+01		N	3.20E+00	pCi/q
/3 -5.00E-03	3 - 6.00E-03	÷ . · · .	N	1.00E-03	pCi/q
/1		5.70E-01 - 5.70	E-01 NT	5.70E-01	pCi/g
21		4.10E+01 - 5.00		1.69E+02	pCi/g
	J - 2.65E+03	0.00E+00 - 3.731	E+00 N	1.19E+02	pCi/q
/3 5.80E-01	1 - 6.70E-01	1	N	6.30E-01	pCi/g
/21 5.308+01	1 - 5.30E + 01	5.30E+00 - 2.20	Ê+01 L	1.58E+01	pCi/g
/4 1.80E-0:	1 - 1.64E+01		N	4.39E+00	pCi/q
	3 - 4.10E-02	1.30E+00 - 9.90	E+00 N	4.25E+00	pC1/q
		•	N	1.37E+01	pCi/g
	/4 -2.00E-03 /3 -5.00E-03 /1 /21 /23 -1.30E+00 /3 5.80E-01 /21 5.30E+00 /21 5.30E+00 /21 7.20E-0	$ \begin{array}{rrrrrrrrrrrrrrrrrrrrrrrrrrrrrrrrrrrr$	$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	$ \begin{array}{cccccccccccccccccccccccccccccccccccc$	$ \begin{array}{cccccccccccccccccccccccccccccccccccc$

- LOCATION=SWMU 99A MEDIA=Surface Soil --

Frequency of Nondetected Detected Arithmetic Analyte Detection Range Range Distribution* Mean Units 13/13 1.80E+03 - 1.29E+04 Aluminum Ľ 6:19E+03 mg/kg Antimony 0/13 2.00E+01 - 2.00E+01 NT 1.00E+01 mg/kg Arsenic 6/13 5.55E+00 - 8.55E+00 5.00E+00 - 5.00E+00 N 4.47E+00 mg/kg 13/13 2.08E+01 - 2.47E+03 Ĺ Barium 2.11E+02 mg/kg 5/13 5.20E-01 - 8.90E-01 5.00E-01 - 5.00E-01 Ъ Beryllium 5.38E-01 mg/kg 0/13 1.00E+02 - 1.00E+02 NT Boron 5.00E+01 mg/kg Cadmium 0/13 2.00E+00 - 2.00E+00NT 1.00E+00 mg/kg 11/11 6.10E+03 - 2.87E+05 Calcium L 2.44E+05 mg/kg Chromium 13/13 7.00E+00 - 4.57E+01 Ľ 1.47E+01 mg/kg 11/13 1.68E+00 - 9.67E+00 Cobalt 1.00E+00 - 1.00E+00 L 3.70E+00 mg/kg 12/13 4.37E+00 - 1.22E+01 2.00E+00 - 2.00E+00 N Copper 6.66E+00 mg/kg 0/11 1.00E+00 - 1.00E+00NT Cyanide 5.00Ë-01 mg/kg Iron 13/13 1.45E+03 - 2.33E+04L 1.09E+04 mg/kg 0/13 Léad 2.00E+01 - 2.00E+01 NT 1.00E+01 mg/kg 13/13 2.82E+00 - 1.29E+01Lithium Ľ 7.58E+00 mg/kg 13/13 1.35E+03 - 2.73E+04 Magnesium L 1.09E+04 mg/kg

*L=Lognormal, N=Normal, NT=Not tested

----- LOCATION=SWMU 99A MEDIA=Surface Soil ----- (continued)

	Frequency of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Manganese	13/13	3.93E+01 - 3.87E+02		N	1.91E+02	mg/kg
Mercury	0/13		2.00E-01 - 2.00E-01	NT	1.00E-01	mg/kg
Nickel	8/13	5.47E+00 - 2.16E+01	5.00E+00 - 5.00E+00	L	8.52E+00	mg/kg
Potassium	13/13	2.91E+02 - 1.12E+03		L	5.47E+02	mg/kg
Selenium	0/11		1.00E+00 - 1.00E+00	NT	5.00E-01	mg/kg
Silver	0/13		4.00E+00 - 4.00E+00	NT	2.00E+00	mg/kg
Sodium	6/13	2.17E+02 - 3.66E+02	2.00E+02 - 2.51E+02	N	1.95E+02	mg/kg
Strontium	13/13	1.46E+01 - 5.14E+02		L	2.71E+02	mg/kg
Thallium	0/13		1.50E+01 - 1.50E+01	NT	7.50E+00	mg/kg
Vanadium	13/13	4.48E+00 - 3.55E+01		L	1.54E+01	mg/kg
Zinc	12/13	4.71E+01 - 1.63E+02	4.76E+01 - 4.76E+01	N	8.24E+01	mg/kg
1,1-Dichloroethene	0/2		1.98E-01 - 2.64E-01	NT	1.16E-01	mg/kg
1,2,4-Trichlorobenzene	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
1,2-Dichlorobenzene	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
1,3-Dichlorobenzene	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
1,4-Dichlorobenzene	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4,5-Trichlorophenol	0/13		5.00 E -01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4,6-Trichlorophenol	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4-Dichlorophenol	0/13		5.00E-01 - 5.00 E-01	NT	2.50E-01	mg/kg
2,4-Dimethylphenol	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,4-Dinitrophenol	0/1		4.80E-01 - 4.80E-01	NT	2.40E-01	mg/kg
2,4-Dinitrotoluene	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2,6-Dinitrotoluene	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Chloronaphthalene	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Chlorophenol	0/13		5.00B-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Methyl-4,6-dinitrophenol	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Methylnaphthalene	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Methylphenol	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Nitroaniline	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
2-Nitrophenol	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
3,3'-Dichlorobenzidine	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
3-Nitroaniline	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Bromophenyl phenyl ether	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Chloro-3-methylphenol	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Chloroaniline	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Chlorophenyl phenyl ether	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Methylphenol	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Nitroaniline	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
4-Nitrophenol	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Acenaphthene	2/13	3.00E-01 - 3.30E-01	5.00E-01 - 5.00E-01	L	3.22E-01	mg/kg
Acenaphtnene	61 13	3.00B-01 - 3.30E-01	5.00E-01 - 5.00E-01	Ц	3.226-01	mg/kg

*L=Lognormal, N=Normal, NT=Not tested

----- LOCATION-SWMU 99A MEDIA-Surface Soil ----- LOCATION-SWMU 99A MEDIA-Surface Soil -----

	Frequency	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Analyce	Derection	Kange	Nange	DIBCLIDUCIO	neal	UIILES
Acenaphthylene	1/13	6.10E-01 - 6.10E-01	5.00E-01 - 5.00E-01	N	2.78E-01	mg/kg
Anthracene	2/13	4.91E-01 - 7.50E-01	5.00E-01 - 5.00E-01	L	4.03E-01	mg/kg
Benz (a) anthracene	3/13	2.20É-01 - 1.70E+00	5,00E-01 - 5.00E-01	L	3.38E-01	mg/kĝ
Benzo (a) pyrene	2/13	1.70E+00 ~ 2.10E+00	5.00E-01 - 5.00E-01	N	5.04E-01	mg/kg
Benzo(b) fluoranthene	6/13	1.70E-01 - 5.70E+00	5.00E-01 - 5.00E-01	Ĺ	5.31E-01	mg/kg
Benzo(ghi) perylene	2/13	5.50B-01 - 1.18E+00	5.00E-01 - 5.00E-01	L	3.14E-01	mg/kg
Benzo(k)fluoranthene	3/13	4.66E-01 - 7.90E-01	5.00E-01 - 5.00E-01	L	4.53E~01	mg/kg
Butyl benzyl phthalate	0/1		4.80E-01 - 4.80E-01	NT	2.40E-01	mg/kgï
Carbazole	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Chrysene	2/13	1.36E+00 - 2.10E+00	5.00E-01 - 5.00E-01	N	4.78E-01	mg/kg
Di-n-butylphthalate	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Di-n-octylphthalate	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Dibenz (a, h) anthracene	1/13	4.80E-01 - 4.90E-01	5.00E-01 - 5.00E-01	N	2.68E-01	mg∕kg
Dibenzofuran	1/13	1.23E-01 - 1.23E-01	5.00E-01 - 5.00E-01	N	2.40E-01	mg/kg
Diethylphthalate	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mq/kg
Dimethylphthalate	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Fluoranthene	4/13	1.40E-01 - 2.66E+00	5.00E-01 - 5.00E-01	L	3.38E-01	mg/kg
Fluorene	1/13	2.19E-01 - 2.19E-01	5.00E-01 - 5.00E-01	N	2.48E-01	mg/kg
Hexachlorobenzene	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorobutadiene	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorocyclopentadiene	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachloroethane	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Indeno (1,2,3-cd) pyrene	2/13	7.80E-01 - 1.05E+00	5.00E-01 - 5.00E-01	N	3.52E-01	mg/kg
Isophorone	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
N-Nitroso-di-n-propylamine	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
N-Nitrosodiphenylamine	0/13		5.00E-01 - 5.00E±01	NT	2.50E-01	mg/kg
Naphthalene	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Nitrobenzene	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
PCB-1016	1/16	1.87E+00 - 1.87E+00	1.02E-01 - 1.28E-01	L	1.72E-01	mg/kg
PCB-1221	0/16		1.02E-01 - 5.45E-01	NT	1.40E-01	mg/kg
PCB-1232	0/16		1.02E-01 - 5.45E-01	NT	1.40E-01	mg/kg
PCB-1242	0/16		1.02E-01 = 5.45E-01	NT	1.40E-01	mg/kg
PCB-1248	0/16		1.02E-01 - 5.45E-01	NT	1.40E-01	mg/kg
PCB-1254	1/16	9.60E-02 - 9.60E-02	1.02E-01 - 5.45E-01	Ľ	1.34E-01	mg/kg
PCB-1260	5/16	6.00E-02 - 6.31E-01	1.02E-01 - 5.45E-01	Ľ	1.01E-01	mg/kg
PCB-1268	0/1		1.00E-01 - 1.00E-01	NT	1.00E-01	mg/kg
Pentachlorophenol	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Phenanthrene	2/13	8.50E-01 - 1.63E+00	5.00E-01 - 5.00E-01	L	2.45E-01	mg/kg
Phenol	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Polychlorinated biphenyl	0/1		1.00E-01 - 1.00E-01	NT	1.00E-01	mg/kg

*L=Lognormal, N=Normal, NT=Not tested

----- LOCATION-SWMU 99A MEDIA-Surface soil -----(continued)

Frequency

Analyte	of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
Pyrene	3/13	1.30E-01 - 2.70E+00	5.00E-01 - 5.00E-01	L	2.74E-01	mg/kg
Pyridine	0/1		4.80E-01 - 4.80E-01	ÑT	2.40E-01	mg/kg
Trichloroethene	0/2		1.98E-01 - 2.64E-01	NT	2.31E-01	mg/kg
Vinyl Chloride	0/2		1.98E-01 - 2.64E-01	NT	2.31E-01	mg/kg
bis (2-Chloroethoxy) methane	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
bis(2-Chloroethyl)ether	0/13		5.00B+01 - 5.00B-01	NT	2.50E-01	mg/kg
bis (2-Chloroisopropyl) ether	0/13		5.00E=01 - 5.00E-01	NT	2.50E-01	mg/kg
bis (2-Ethylhexyl) phthalate	0/13		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
cis-1,2-Dichloroethene	0/2		1.98E-01 - 2.64E-01	NT	2.31E-01	mg/kg
trans-1,2-Dichloroethene	0/2		1.98E-01 - 2.64E-01	NT	2.31E-01	mg/kg
Alpha activity	15/16	9.708+00 - 1.42E+02	3.10E+00 - 3.10E+00	L	2.53E+01	pCi/g
Americium-241	0/16		1.10E+00 - 1.30E+01		5.58E+00	pCi/q
Beta activity	16/16	6.70E+00 - 2.73E+03		NT L	8.58E+01	pCi/g
Cesium-137	3/16	1.10E+00 - 1.90E+00	3.80E-01 - 3.50E+00	L	4.50E-01	pCi/g
Cobalt-60	0/16		5.20E-01 - 4.60E+00	NT	1.53E+00	pCi/g
Neptunium-237	1/1	1.28E+01 - 1.28E+01		NT	1.28E+01	pCi/g
Plutonium-239/240	0/1		5.70E-01 - 5.70E-01	NT	5.70E-01	pCi/g
Protactinium-234m	0/16		4.10E+01 - 5.00E+02	NT	1.77E+02	pCi/g
Technetium-99	3/16	1.66E+01 = 2.65E+03	0.00E+00 - 3.73E+00	N	1.71E+02	pCi/g
Thorium-234	1/16	5.30E+01 - 5.30E+01	5.30E+00 = 2.20E+01	L	1.67E+01	pCi/g
Uranium-234	1/1	1.64E+01 - 1.64E+01		NT	1.64E+01	pCi/g
Uranium-235	0/16		1.30E+00 - 9.90E+00	NT	5.03E+00	pCi/g
Uranium-238	1/1	5.17E+01 - 5.17E+01		NT	5.17E+01	pCi/g

-- LOCATION-SWMU 99B MEDIA-RGA Groundwater -----

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
Aluminum	0/3		7.50E-01 - 1.00E+00	NT	4.58E-01	mg/L
Antimony	0/7		1.85E-01 - 2.50E-01	NT	1.06E-01	mg/L
Arsenic	Ó/7		5.00E±03 ÷ 5.00E±03	NT	2.50E-03	mg/L
Barium	7/7	2.00E-01 - 2.70E+00		L	4.78E-01	mg/L
Beryllium	0/7		1.50E-02 - 2.50E-02	NT	9.29E-03	mg/L
Cadmium	0/7		2.50E-02 - 1.00E-01	NT	2.86E-02	mg/L
Calcium	7/7	2.84E+01 - 3.27E+01		L	3.04E+01	mg/L
Chloride	7/7	8.29E+01 - 1.08E+02		N	9.59E+01	mg/L
Chromium	1/7	2.60E-01 - 2.60E-01	5.00E-02 - 6.00E-02	L	5.62E-02	mg/L

*L=Lognormal, N=Normal, NT=Not tested

(continued)

	Frequency								
	of	Detected	Nondetected		Arithmetic				
Analyte	Detection	Range	Range	Distribution*	Mean	Units			
Cobalt	0/7		4.50E-02 - 1.00E-01	NT	2.71E-02	mg/L			
Copper	1/7	4.00E-02 - 4.00E-02	2.50E-02 - 1.00E-01	N	3.25E-02	mg/L			
Fluoride	7/7	1.60E-01 - 2.10E-01		L	1.77E-01	mg/L			
Iron	3/7	2.94E-01 - 3.34E+00	3.00E-01 - 3.60E-01	ī	6.08E-01	mg/L			
Lead	0/7		2.50E-01 - 2.50E-01	NT	1.25E-01	mg/L			
Magnesium	7/7	1.15E+01 - 1.31E+01		L	1.23E+01	mg/L			
Manganese	5/7	6.00E-02 - 2.90E-01	1.00E-01 - 1.00E-01	L	1.65E-01	mg/L			
Mercury	0/7	······································	2.00E-04 - 2.00E-04	NT	1.00E-04	mg/L			
Molybdenum	0/3		5.00E-02 - 1.00E-01	NT	4.17E-02	mg/L			
Nickel	0/7		1.00E-01 - 1.00E-01	NT	5.00E-02	mg/L			
Nitrate as Nitrogen	7/7	1.70E+00 - 2.10E+00		L	1.84E+00	mg/L			
Potassium	0/6		5.00E+00 - 1.05E+01	NT	3.88E+00	mg/L			
Selenium	0/7		5.00E-03 - 5.00E-03	NT	2.50E-03	mg/L			
Silica	7/7	1.50E+01 - 2.00E+01		L	1.72E+01	mg/L			
Silver	0/7		5.00E-02 - 6.00E-02	NT	2.79E-02	mg/L			
sodium	7/7	6.32E+01 - 7.86E+01		L	6.99E+01	mg/L			
Sulfate	2/2	1.75E+01 - 2.67E+01		N	2.21E+01	mg/L			
Tetraoxo-sulfate(1-)	5/5	1.92E+01 - 2.90E+01		Ń	2.46E+01	mg/L			
Total Phosphate as Phosphorus	0/3		2.00E+00 - 2.00E+00	NT	1.00E+00	mg/L			
Uranium	0/7		1.00E-03 - 1.00E-03	NT	1.00E-03	mg/L			
Zinc	2/7	3.00E-02 - 6.00E-02	3.00E-02 - 2.50E-01	L	3.72E-02	mg/L			
1,1,1-Trichloroethane	0/15		2.50E-01 - 5.00E-01	NT	1.67E-01	mg/L			
1,1,2-Trichloroethane	0/15		2.50E-01 - 5.00E-01	NT	1.67E-01	mg/L			
1,1-Dichloroethane	0/15		2.50E-01 ~ 5.00E-01	NT	1.67E-01	mg/L			
1,1-Dichloroethene	0/15		2.50E-01 - 5.00E-01	NT	1.67E-01	mg/L			
1,2-Dichloroethane	0/15		2.50E-01 - 5.00E-01	NT	1.67E-01	mg/L			
Benzene	0/15		2.50E-01 - 5.00E-01	NT	1.67E-01	mg/L			
Bromodichloromethane	0/15		2.50E-01 - 5.00E-01	NT	1.67E-01	mg/L			
Carbon Tetrachloride	0/15		2.50E-01 - 5.00E-01	NT	1.67E-01	mg/L			
Chloroform	0/15		2.50E-01 - 5.00E-01	NT	1.67E-01	mg/L			
Ethylbenzene	0/15		2.50E-01 - 5.00E-01	NT	1.67E-01	mg/L			
Polychlorinated biphenyl	0/1		1.70E-04 - 1.70E-04	NT	1.70E-04	mg/L			
Tetrachloroethene	0/15		2.50E-01 - 5.00E-01	ŇT	1.67E-01	mg/L			
Toluene	0/15		2.50E-01 - 5.00E-01	NT	1.67E-01	mg/L			
Trichloroethene	16/16	1.30E+00 - 2.30E+00		N	1.94E+00	mg/L			
Vinyl Chloride	0/15		2.50E-01 - 1.00E+00	NT	5.00E-01	mg/L			
Xylene	0/15		2.50E-01 - 1.00E+00	NT	2.67E-01	mg/L			
cis-1,2-Dichloroethene	0/15		2.50E-01 - 5.00E-01	NT	3.33E-01	mg/L mg/L			
trans-1,2-Dichloroethene	0/15		2.50E-01 - 5.00E-01	NT	3.33E-01	mg/L mg/L			
Alpha activity	12/16	-4.20E+00 - 4.20E+00	-2.03E+00 - 4.60E+00	N	7.42E-01	pCi/L			
					/. 326-UI	pc1/L			

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
Beta activity	16/16	3.00E+00 - 4.50E+01		L	1.20E+01	pCi/L
Radon-222	4/4	2.57E+02 - 4.12E+02		N	3.66E+02	pCi/L
Technetium-99	12/17	-2.00E+00 - 1.90E+01	-3.00E+00 - 1.17E+01	N	5.51E+00	pCi/L

------ LOCATION-SWMU 99B MEDIA-Subsurface Soil -------

	Frequency	B-44-1	March 3 - 6 6 - 3		•	
	of	Detected	Nondetected	- 1	Arithmetic	• •
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Aluminum	8/8	9.31E+03 - 1.70E+04		L	1.25E+04	mg/kg
Antimony	0/8		2.00E+01 - 2.00E+01	NT	1.00E+01	mg/kg
Arsenic	2/6	6.89E+00 - 8.05E+00	5.00E+00 - 5.00E+00	N	4.16E+00	mg/kg
Barium	8/8	6.50E+01 - 1.55E+02		L	9.63E+01	mg/kg
Beryllium	6/8	5.70E-01 - 1.00E+00	5.00E-01 - 5.00E-01	N	5.66E-01	mg/kg
Boron	0/8		1.00E+02 - 1.00E+02	NT	5.00E+01	mg/kg
Cadmium	0/8 ·		2.00E+00 - 2.00E+00	NT	1.00E+00	mg/kg
Calcium	6/6	5.03E+02 - 7.17E+03		N	2.51E+03	mg/kg
Chromium	8/8	1.18E+01 - 2.61E+01		L	1.79E+01	mg/kg
Cobalt	8/8	1.91E+00 - 6.94E+00		L	4.17E+00	mg/kg
Copper	· 8/8	5.25E+00 - 1.30E+01		N	8.66E+00	mg/kg
Cyanide	0/7		1.00E+00 - 1.00E+00	NT	5.00E-01	mg/kg
Iron	8/8	9.66E+03 - 1.81E+04		N	1.48E+04	mg/kg
Lead	0/8		2.00E+01 - 2.00E+01	NT	1.00E+01	mg/kg
Lithium	8/8	6.50E+00 - 1.14E+01		L	8.62E+00	mg/kg
Magnesium	8/8	1.10E+03 - 2.53E+03		N	1.76E+03	mg/kg
Manganese	8/8	6.32E+01 - 5.24E+02		L	2.43E+02	mg/kg
Mercury	0/8		2.00E-01 - 2.00E-01	NT	1.00E-01	mg/kg
Nickel	5/8	7.27E+00 - 2.51E+01	5.00E+00 - 5.00E+00	L	1.05E+01	mg/kg
Potassium	8/8	3.37E+02 - 1.04E+03		L	6.44E+02	mg/kg
Selenium	0/4		1.00E+00 - 1.00E+00	NT	5.00E-01	mg/kg
Silver	0/8		4.00E+00 - 4.00E+00	NT	2.00E+00	mg/kg
Sodium	3/8	2.11E+02 - 3.09E+02	2.00E+02 - 2.00E+02	N	1.58E+02	mg/kg
Strontium	8/8	9.46E+00 - 2.22E+01		L	1.61E+01	mg/kg
Thallium	0/8		1.50E+01 - 1.50E+01	NT	7.50E+00	mg/kg
Vanadium	8/8	1.97E+01 - 3.44E+01		L	2.46E+01	mg/kg
Zinc	8/8	1.96E+01 - 5.22E+01		N	3.74E+01	mg/kg
1,1,1-Trichloroethane	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
1,1,2,2-Tetrachloroethane	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg

*L=Lognormal, N=Normal, NT=Not tested

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LOCATION-SWMU 99B MEDIA-Subsurface Soil -----(continued)

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	Frequency		-	-	•	ithmetic Mean Units .00E-01 mg/kg .00E-01 mg/kg .50E-01 mg/kg .50E-01 mg/kg .00E-01 mg/kg .00E-01 mg/kg .50E-01 mg/kg .50E-01 mg/kg .50E-01 mg/kg .50E-01 mg/kg .50E-01 mg/kg			
-	of	Detected	Nondetected	• •	Arithmetic				
Analyte	Detection	Range	Range	Distribution*		Units			
1,1,2-Trichloroethane	0/7		1.20E+00 - 1.20E+00	NT	6 008-01				
1,1-Dichloroethane	0/7	. ·	1.20E+00 - 1.20E+00	NT					
1,1-Dichloroethene	0/7		1.20E+00 - 1.20E+00	NT					
1,2,4-Trichlorobenzene	0/8		5.00E-01 - 5.00E-01	NT					
1,2-Dichlorobenzene	0/8		5.00E=01 = 5.00E-01	NT					
1,2-Dichloroethane	0/7		1.20E+00 - 1.20E+00	NT					
1,2-Dichloropropane	0/7		1.20B+00 - 1.20B+00	NT					
1,2-Dimethylbenzene	0/7		1.20E+00 - 1.20E+00	NT					
1,3-Dichlorobenzene	0/B	• .	5.00E-01 - 5.00E-01	NT	1 I I I I I I I I I I I I I I I I I I I				
1,4-Dichlorobenzene	_ 0/8		5.00E-01 - 5.00E-01	NT	2.50E-01				
2,4,5-Trichlorophenol	0/8	· · ·	5.00E-01 - 5.00E-01	NT	the second second second				
2,4,6-Trichlorophenol	0/8		5.00E-01 - 5.00E-01	NT					
2,4-Dichlorophenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
2,4-Dimethylphenol	0/8	-	5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
2,4-Dinitrotoluene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
2,6-Dinitrotoluene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
2-Butanone	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg			
2-Chloronaphthalene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
2-Chlorophenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
2-Hexanone	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg			
2-Methyl-4,6-dinitrophenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
2-Methylnaphthalene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
2-Methylphenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
2-Nitroaniline	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
2-Nitrophenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
3,3'-Dichlorobenzidine	. 0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
3-Nitroaniline	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
4-Bromophenyl phenyl ether	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
4-Chloro-3-methylphenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
4-Chloroaniline	·0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
4-Chlorophenyl phenyl ether	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
4-Methyl=2-pentanone	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg			
4-Methylphenol	0/8	ь · ·	5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
4-Nitroaniline	Ó/B		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
4-Nitrophenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
Acenaphthene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
Acenaphthylene	0/8		5.00E-01 - 5.00B-01	NT	2.50E-01	mg/kg			
Acetone	1/7	5.50E-01 - 5.50E-01	1.20E+00 - 1.20E+00	N	5.93E-01	mg/kg			
Anthracene	0/8	· .	5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			
Benz (a) anthracene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg			

A-55

continued

	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Benzene	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Benzo (a) pyrene	0/8		5.00E-01 - 5.00B-01	NT	2.50E-01	mg/kg
Benzo(b)fluoranthene	0/8		5.00E-01 - 5.00E-01	· NT	2.50E-01	mg/kg
Benzo(ghi)perylene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Benzo(k)fluoranthene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Bromodichloromethane	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Bromoform	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Bromomethane	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Carbazole	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Carbon Disulfide	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Carbon Tetrachloride	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Chlorobenzene	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Chloroethane	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Chloroform	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Chloromethane	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Chrysene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Di-n-butylphthalate	0/8		5.00E-01 ~ 5.00E-01	NT	2.50E-01	mg/kg
Di-n-octylphthalate	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Dibenz (a, h) anthracene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Dibenzofuran	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Dibromochloromethane	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Diethylphthalate	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Dimethylphthalate	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Ethylbenzene	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Fluoranthene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Fluorene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorobenzene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorobutadiene	0/8	·	5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachlorocyclopentadiene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Hexachloroethane	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Indeno(1,2,3-cd)pyrene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Isophorone	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Methylene Chloride	3/7	1.20E+00 - 1.20E+00	1.20E+00 - 1.20E+00	N	8.57E-01	mg/kg
N-Nitroso-di-n-propylamine	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
N-Nitrosodiphenylamine	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Naphthalene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Nitrobenzene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
PCB-1016	0/6		1.17E-01 - 1.25E-01	NT	1.20E-01	mg/kg
PCB-1221	0/6		1.17E-01 - 1.25E-01	NT	1.20E-01	mg/kg
PCB-1232	0/6		1.17E-01 - 1.25E-01	NT	1.20E-01	mg/kg
					1.200 VI	

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Table 1.3. Data summary for all analytes by location and medium

(continued)

	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
PCB-1242	0/6		1.178-01 - 1.258-01	NT	1.20E-01	mg/kg
PCB-1248	0/6		1.17E-01 - 1.25E-01	NT	1.20E-01	mg/kg
PCB-1254	0/6		1.17E-01 - 1.25E-01	NT	1.20E-01	mg/kg
PCB-1260	0/6		1.17E-01 - 1.25E-01	NT	1.20E-01	mg/kg
Pentachloropheno1	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Phenanthrene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Phenol	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Pyrene	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
Styrene	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Tetrachloroethene	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Toluene	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Trichloroethene	0/7		1.20E+00 - 1.20E+00	NT	1.20E+00	mg/kg
Vinyl Chloride	0/7		1.20E+00 - 1.00E+02	NT	1.53E+01	mg/kg
bis (2-Chloroethoxy) methane	0/8		5.00E-01 - 5.00E-01	NT	2.508-01	mg/kg
bis(2-Chloroethyl)ether	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
bis(2-Chloroisopropyl)ether	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
bis (2-Ethylhexyl) phthalate	0/8		5.00E-01 - 5.00E-01	NT	2.50E-01	mg/kg
cis-1,2-Dichloroethene	0/7		1.20E+00 - 1.20E+00	NT	1.20E+00	mg/kg
cis-1,3-Dichloropropene	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
m,p-Xylene	0/7		2.40E+00 - 2.50E+00	NT	1.22E+00	mg/kg
trans-1,2-Dichloroethene	0/7		1.20E+00 - 1.20E+00	NT	1.20E+00	mg/kg
trans-1,3-Dichloropropene	0/7		1.20E+00 - 1.20E+00	NT	6.00E-01	mg/kg
Alpha activity	8/8	1.33E+01 - 2.14E+01		N	1.73E+01	pCi/g
Americium-241	0/8	· · ·	2.20E+00 - 1.30E+01	ŇT	6.90E+00	pCi/g
Beta activity	8/8	1.48E+01 - 2.26E+01		L	1.86E+01	pCi/g
Cesium-137	0/8		7.60E-01 - 4.00E+00	NT	2.19E+00	pci/g
Cobalt-60	0/8		1.00E+00 - 1.70E+00	NT	1.38E+00	pCi/g
Protactinium-234m	0/8		1.40E+02 - 7.70E+02	NT	3.54E+02	pCi/g
Technetium-99	0/8		0.00E+00 - 9.40E-01	NT	· · · · ·	pCi/g
Thorium-234	0/8		6.30E+00 - 2.50E+01	NT	1.18E-01	pCi/g
Uranium-235	0/8		2.00E+00 - 9.90E+00	NT	1.89E+01	pCi/g
	•,•		2.002+00 - 9.902+00	IN I	5.94E+00	pCi/g

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----- LOCATION=AOC 204 MEDIA=RGA Groundwater -

	Frequency of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
1,1,1-Trichloroethane	11/11	1.00E-02 - 1.80E-02		N	1.13E-02	mg/L
1,1-Dichloroethane	1/1	5.00E+00 - 5.00E+00		NT	5.00E+00	mg/L
1,1-Dichloroethene	12/15	1.00E-04 - 4.00E-02	1.00E-03 - 1.00E-03	L	3.25E-02	mg/L
PCB-1254	11/11	2.50E-02 - 2.50E-02		N	2.50E-02	mg/L
PCB-1260	11/11	2.50E-02 - 2.50E-02		N	2.50E-02	mg/L
Polychlorinated biphenyl	1/1	1.70E-01 - 1.70E-01		NT	1.70E-01	mg/L
Tetrachloroethene	11/11	5.00E-03 - 5.00E+00		N	4.59E-01	mg/L
Trichloroethene	15/15	5.00E-03 - 7.70E-01		L	1.41E-01	mg/L
Viny1 Chloride	1/4	1.00E-04 - 1.00E-04	1.00E-03 - 1.00E-03	N	7.75E-04	mg/L
cis-1.2-Dichloroethene	3/4	9.00E-04 - 6.00E-03	1.00E-03 - 1.00E-03	N	3.48E-03	mg/L
trans-1,2-Dichloroethene	2/4	1.00E-04 - 1.00E-04	1.00E-03 - 1.00E-03	N	5.50E-04	mg/L
Alpha activity	2/4	2.40E+00 - 6.80E+00	7.90E-01 - 1.70E+00	. N	2.92E+00	pCi/L
Beta activity	2/4	3.40E+00 - 5.20E+00	1.90E+00 - 2.60E+00	N	3.28E+00	pCi/L

------ LOCATION=AOC 204 MEDIA=Subsurface Soil ------

A-58

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
1,1,1-Trichloroethane	10/16	1.00E-02 - 1.00E+00	1.00E-02 - 1.00E-02	L	4.64E-02	mg/kg
1,1-Dichloroethane	8/14	1.00E+00 - 1.00E+00	1.00E-02 - 1.00E-02	N	5.74E-01	mg/kg
1,1-Dichloroethene	11/17	4.00E-02 - 4.00E-02	3.36E-01 - 4.27E-01	N	9.35E-02	mg/kg
PCB-1254	11/11	2.50E-02 - 2.50E-02		N	2.50E-02	mg/kg
PCB-1260	11/11	2.50E-02 - 2.50E-02		N	2.50E-02	mg/kg
Polychlorinated biphenyl	8/8	1.00E-01 - 1.00E-01		N	1.00E-01	mg/kg
Tetrachloroethene	11/17	5.00E-03 - 1.00E+00	1.00E-02 - 1.00E-02	N	4.73E-01	mg/kg
Trichloroethene	11/17	5.00E-03 - 1.00E+00	3.36E-01 - 4.27E-01	L	1.67E-01	mg/kg
Alpha activity	6/6	9.50E+00 - 1.96E+01		N	1.53E+01	pCi/g
Beta activity	6/6	1.71E+01 - 2.91E+01		N	2.27E+01	pCi/g

-- LOCATION=SWMU 193A MEDIA=McNairy Groundwater

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
Calcium	4/4	1.30E+01 - 8.21E+01		N	3.53E+01	mg/L
Chloride	4/4	8.00E+00 - 1.60E+01		N	1.25E+01	mg/L

 LOCATION=SWMU	193A	MEDIA=McNairy	Groundwater
	· (c	ontinued)	

	Frequency	Detected	Nondetected		Arithmetic	1
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Iron	4/4	2.89E+00 - 3.14E+02		'n.	8.36E+01	mg/L
Magnesium	4/4	5.33E+00 - 4.02E+01		N	1.46E+01	mg/L
Potassium	4/4	4.07E+00 - 2.15E+01		N	9.02E+00	mg/L
Sodium	4/4	1.64E+01 - 8.08E+01		N	4.13E+01	mg/L
Tetraoxo-sulfate(1-)	4/4	2.10E+01 - 8.40E+01		N	5.05E+01	mg/L
Acetone	1/1	1.40E-02 - 1.40E-02		NT	1.40E-02	mg/L
Diethylphthalate	1/6	1.90E-02 - 1.90E-02	1.00E-02 - 4.00E-02	N	1.04E-02	mg/L
Trichloroethene	8/13	2.00E-04 - 1.10E-02	1.00E-03 - 1.00E-03	L	1.70E=03	mg/L
cis-1,2-Dichloroethene	1/11	1.70E-01 - 1.70E-01	1.00E-03 - 2.00E+00	L	6.85E+00	mg/L
Alpha activity	6/10	2.40E+00 - 4.00E+01	2.90E-01 - 1.40E+00	Ľ	4.44E+00	pCi/I
Beta activity	8/10	5.10E+00 - 6.46E+01	1.00E+00 - 4.40E+00	L .	2.13E+01	pCi/I
Technetium-99	5/10	1.00E+01 - 1.45E+02	4.00E+00 - 8.50E+00	Ĺ.	1.38E+01	pCi/1
Thorium-234	1/1	8.40E-01 - 8.40E-01	-	NT	8.40E-01	pCi/I
Uranium-234	1/1	8.10E-01 - 8.10E-01	· · ·	NT	8.10E-01	pCi/I
Uranium-238	1/1	1.32E+00 - 1.32E+00		NT	1.32E+00	pCi/l

LOCATION-SWMU 193A MEDIA-RGA Groundwater

A-59

Frequency Arithmetic ō£ Nondetected Detected Detection Range Range Distribution* Mean Units Analyte 4/4 1.29E-01 - 1.78E-01 N 1.43E-01 mg/L Aluminum NT Ammonia 1/1 3.00E-01 - 3.00E-01 3.00E-01 mġ/L 5/5 2.62E+01 - 1.34E+02 N 5.96E+01 mq/L Calcium Chloride 5/5 1.30E+01 - 6.40E+01 N 3.10E+01 mg/L 1/4 1.80E-02 - 1.80E-02 Ń 8.25E-03 mg/L Copper 1.00E-02 - 1.00E-02 1/1 4.20E-01 - 4.20E-01 NT 4.20E-01 mg/L Fluoride 7/9 2.00E-02 - 3.66E+01 1.00E-02 - 1.00E-02 Ŀ 2.93E+00 mg/L Iron 3.91E+00 - 1.85E+01 N 1.05E+01 mq/L 5/5 Magnesium 5/5 N Potassium 2.66E+00 - 2.65E+02 6.10E+01 mg/L 1/1NT 1.90E+01 mg/L 1.90E+01 - 1.90E+01Silica 5/5 N 7.80E+01 Sodium 3.50E+01 - 1.34E+02 mg/L 5/5 2.10E+01 - 2.62E+02 N 1.12E+02 mg/L Tetraoxo-sulfate(1-) 7.20E-02 - 2.12E-01 N 1.21E-01 mg/L 4/4 Zinc mg/L 2/43 1.00E-04 - 2.00E-04 1.00E+03 - 5.00E+00 L 1.68E-04 1,1-Dichloroethene 3/25 9.00E-03 - 1.50E-02 1.00E-02 - 4.00E-02 L 9.17E-03 mg/L Diethylphthalate 1.00E-02 - 4.00E-02 Ŀ 1/25 1.20E-02 - 1.20E-02 7.74E-03 mg/L Pentachlorophenol 44/51 2.00E-04 - 6.70E+00 1.00E-03 - 1.00E-03 Г 7.76E-02 mg/L Trichloroethene

---- LOCATION=SWMU 193A MEDIA=RGA Groundwater ---(continued)

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
bis(2-Ethylhexyl)phthalate	3/25	1.30E-02 - 2.20E-02	1.00E-02 - 4.00E-02	L	7.94E-03	mg/L
cis-1,2-Dichloroethene	17/42	1.00E-04 - 8.40E-02	1.00E-03 - 5.00E+00	L	1.51E-03	mg/L
trans-1,2-Dichloroethene	7/43	1.00E-04 - 7.00E-04	1.00E-03 - 5.00E+00	L	3.07E-04	mg/L
Alpha activity	19/34	1.50E+00 - 1.76E+01	-4.10E-01 - 4.00E+00	N	3.64E+00	pCi/L
Beta activity	34/34	2.90E+00 - 8.80E+02		L	8.53E+01	pCi/L
Technetium-99	26/39	8.00E+00 - 1.39E+03	-7.60E+00 - 1.70E+01	N	1.21E+02	pCi/L
Thorium-234	1/8	5.40E-01 - 5.40E-01	-1.65E+02 - 5.58E+01	N	-2.51E+01	pCi/L

----- LOCATION=SWMU 193A MEDIA=Subsurface Soil -------

	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Aluminum	8/8	3.01E+03 - 1.40E+04		N	9.71E+03	mg/kg
Barium	8/8	2.16E+01 - 8.73E+01		N	5.66E+01	mg/kg
Beryllium	5/8	5.20E-01 - 7.00E-01	5.00E-01 - 5.00E-01	N	4.60E-01	mg/kg
Calcium	4/4	1.06E+03 - 2.73E+05		N	9.07E+04	mg/kg
Chromium	8/8	4.31E+00 - 2.77E+01		N	1.65E+01	mg/kg
Cobalt	8/8	1.47E+00 - 8.66E+00		L	4.13E+00	mg/kg
Copper	8/8	2.45E+00 - 7.31E+00		N	4.97E+00	mg/kg
Iron	8/8	3.74E+03 - 1.54E+04		N	1.11E+04	mg/kg
Lithium	8/8	3.78E+00 - 1.12E+01		N	7.14E+00	mg/kg
Magnesium	8/8	1.16E+03 - 1.70E+04		L	3.92E+03	mg/kg
Manganese	8/8	4.86E+01 - 5.64E+02		L	2.30E+02	mg/kg
Nickel	5/8	5.50E+00 - 9.16E+00	5.00E+00 - 5.00E+00	N	5.42E+00	mg/kg
Potassium	8/8	2.89E+02 - 1.44E+03		L	5.27E+02	mg/kg
Silver	1/8	4.00E+00 - 4.00E+00	4.00E+00 - 4.00E+00	N	2.25E+00	mg/kg
Sodium	5/8	2.13E+02 ~ 3.13E+02	2.00E+02 - 2.00E+02	N	1.94E+02	mg/kg
Strontium	8/8	6.36E+00 - 2.53E+02		L	7.47E+01	mg/kg
Vanadium	8/8	5.67E+00 - 3.15E+01		N	2.18E+01	mg/kg
Zinc	8/8	1.84E+01 - 5.54E+01		L	3.45E+01	mg/kg
Acetone	1/2	1.10E - 02 - 1.10E - 02	1.00E-02 - 1.00E-02	N	8.00E-03	mg/kg
Anthracene	1/8	1.16E-01 - 1.16E-01	5.00E-01 - 5.00E-01	N	2.33E-01	mg/kg
Benz (a) anthracene	2/8	1.60E-01 - 1.80E-01	5.00E-01 - 5.00E-01	L	1.75E-01	mg/kg
Benzo(a) pyrene	2/8	2.40E-01 - 2.50E-01	5.00E-01 - 5.00E-01	N	2.49E-01	mg/kg
Benzo (b) fluoranthene	2/8	3.90E-02 - 5.10E-02	5.00E-01 - 5.00E-01	L	4.77E-02	mg/kg
Benzo (ghi) perylene	2/8	1.66E-01 - 1.70E-01	5.00E-01 - 5.00E-01	Ľ	1.69E-01	mg/kg
Chrysene	2/8	1.70E - 01 - 1.70E - 01	5.00E-01 - 5.00E-01	N	2.30E-01	mg/kg
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*L=Lognormal, N=Normal, NT=Not tested

A-60

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
Di-n-butylphthalate	1/8	7.70E-02 - 7.70E-02	5.00E-01 - 6.60E-01	N	2.38E-01	mg/kg
Di-n-octylphthalate	1/8	1.20E-01 - 1.20E-01	5.00E-01 - 5.00E-01	N	2.34E-01	mg/kg
Dibenz (a, h) anthracene	1/8	1.30E-01 - 1.30E-01	5.00E-01 - 5.00E-01	N	2.35E-01	mg/kg
Diethylphthalate	1/8	4.00E-01 - 4.00E-01	5.00E-01 - 5.00E-01	N	2.69E-01	mg/kg
Fluoranthene	2/8	2.30E-01 - 3.10E-01	5.00E-01 - 5.00E-01	L	2.88E-01	mg/kg
Indeno (1,2,3-cd) pyrene	2/8	1.38E-01 - 1.60E-01	5.00E-01 - 5.00E-01	· ' L	1.54E-01	mg/kg
Pyrene	2/8	2.40E-02 - 2.95E-01	5.00E-01 - 5.00E-01	N	2.27E-01	mg/kg
bis (2-Ethylhexyl) phthalate	2/8	8.10E-02 - 1.70E-01	5.00E-01 - 5.00E-01	N	2.19E-01	mg/kg
Alpha activity	8/8	9.60E+00 - 2.60E+01		L	1.62E+01	pCi/g
Beta activity	8/8	1.41E+01 - 2.37E+01		Ĺ	1.90E+01	pCi/g

	Frequency	-				
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Aluminum	4/4	3.01Ē+03 - 1.09E+04		N	7.24E+03	mg/kg
Barium	4/4	2.16E+01 - 8.40E+01		N	5.34E+01	mg/kg
Beryllium	1/4	6.40E-01 - 6.40E-01	5.00E-01 - 5.00E-01	N	3.48E-01	mg/kg
Calcium	2/2	8.76E+04 - 2.73E+05		N	1.80E+05	mg/kg
Chromium	4/4	4.31E+00 - 2.65E+01		N	1.29E+01	mg/kg
Cobalt	4/4	1.47E+00 - 5.70E+00		N	3.36E+00	mg/kg
Copper	4/4	2.45E+00 - 7.31E+00		N	5.32E+00	mg/kg
Iron	4/4	3.74E+03 - 1.54E+04	•	N	9.39E+03	mg/kg
Lithium	4/4	3.78E+00 - 1.12E+01		N	6.84E+00	mg/kg
Magnesium	4/4	1.66E+03 - 1.70E+04		N	6.91E+03	mg/kg
Manganese	4/4	1.35E+02 - 3.98E+02		N	2.14E+02	mg/kg
Nickel	2/4	7.27E+00 - 7.50E+00	5.00E+00 - 5.00E+00	Ń	4.94E+00	mg/kg
Potassium	4/4	2.89E+02 - 1.44E+03		Ň	6.78E+02	mg/kg
Sodium	1/4	2.13E+02 - 2.13E+02	2.00E+02 - 2.00E+02	N	1.28E+02	mg/kg
Strontium	4/4	1.21E+01 - 2.53E+02		N	1.22E+02	mg/kg
Vanadium	4/4	5.67E+00 - 3.15E+01		Ń	1.76E+01	mg/kg
Zinc	4/4	3.34E+01 - 5.54E+01		Ň	4.64E+01	mg/kg
Anthracene	1/4	1.16E-01 - 1.16E-01	5.00E-01 - 5.00E-01	N	2.17E-01	mg/kg
Benz (a) anthracene	2/4	1.60E-01 - 1.80E-01	5.00E-01 - 5.00E-01	N	2.10E-01	mg/kg
Benzo (a) pyrene	2/4	2.40E-01 - 2.50E-01	5.00E-01 - 5.00E-01	N	2.48E-01	mg/kg
		3.90E-02 - 5.10E-02	5.00E-01 - 5.00E-01	N	1.48E-01	
Benzo (b) fluoranthene	2/4 2/4	1.66E-01 - 1.70E-01	5.00E-01 - 5.00E-01	N	2.09E-01	mg/kg
Benzo (ghi) perylene	4/ 1	1.002-01 - 1./02-01	3.008-01 - 3,00E-01	14	2.036-01	mg/kg

----- LOCATION=SWMU 193A MEDIA=Surface Soil -----(continued)

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	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Chrysene	2/4	1.70E-01 - 1.70E-01	5.00E-01 - 5.00E-01	N	2.10E-01	mg/kg
Di-n-butylphthalate	1/4	7.70E-02 - 7.70E-02	5.00E-01 - 6.60E-01	N	2.27E-01	mq/kq
Di-n-octylphthalate	1/4	1.20E-01 - 1.20E-01	5.00E-01 - 5.00E-01	N	2.18E-01	mg/kg
Dibenz (a, h) anthracene	1/4	1.30E-01 - 1.30E-01	5.00E-01 - 5.00E-01	N	2.20E-01	mg/kg
Diethylphthalate	1/4	4.00E-01 - 4.00E-01	5.00E-01 - 5.00E-01	N	2.88E-01	mg/kg
Fluoranthene	2/4	2.30E-01 - 3.10E-01	5.00E-01 - 5.00E-01	N	2.60E-01	mg/kg
Indeno (1,2,3-cd) pyrene	2/4	1.38E-01 - 1.60E-01	5.00E-01 - 5.00E-01	N	2.00E-01	mg/kg
Pyrene	2/4	2.40E-02 - 2.95E-01	5.00E-01 - 5.00E-01	N	2.05E-01	mg/kg
bis (2-Ethylhexyl) phthalate	2/4	8.10E-02 - 1.70E-01	5.00E-01 - 5.00E-01	N	1.88E-01	mg/kg
Alpha activity	4/4	9.60E+00 - 1.70E+01		N	1.32E+01	pCi/g
Beta activity	4/4	1.41E+01 - 2.37E+01		N	2.10E+01	pCi/g
		OCATTON COMPLETA				
		LOCATION-SWMU 193B MEDIA	=MCNairy Groundwater			
	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Trichloroethene	1/2	1.30E-02 - 1.30E-02	1.00E-03 - 1.00E-03	N	7.00E-03	mg/L
Trichloroethene cis-1,2-Dichloroethene	1/2 1/2	1.30E-02 - 1.30E-02 2.30E-02 - 2.30E-02	1.00E-03 - 1.00E-03 1.00E-03 - 1.00E-03	n N	7.00E-03 1.20E-02	mg/L mg/L
cis-1,2-Dichloroethene Alpha activity	•				1.20E-02	mg/L
cis-1,2-Dichloroethene	1/2	2.30E-02 - 2.30E-02	1.00E-03 - 1.00E-03	N	-	
cis-1,2-Dichloroethene Alpha activity	1/2 1/2 2/2	2.30E-02 - 2.30E-02 1.29E+00 - 1.29E+00	1.00E-03 - 1.00E-03 1.20E+00 - 1.20E+00	N N N	1.20E-02 1.25E+00 3.98E+00	mg/L pCi/L pCi/L
cis-1,2-Dichloroethene Alpha activity	1/2 1/2 2/2	2.30E-02 - 2.30E-02 1.29E+00 - 1.29E+00 3.15E+00 - 4.80E+00	1.00E-03 - 1.00E-03 1.20E+00 - 1.20E+00	N N N	1.20E-02 1.25E+00 3.98E+00	mg/L pCi/L pCi/L
cis-1,2-Dichloroethene Alpha activity	1/2 1/2 2/2	2.30E-02 - 2.30E-02 1.29E+00 - 1.29E+00 3.15E+00 - 4.80E+00	1.00E-03 - 1.00E-03 1.20E+00 - 1.20E+00	N N N	1.20E-02 1.25E+00 3.98E+00	mg/L pCi/L pCi/L
cis-1,2-Dichloroethene Alpha activity	1/2 1/2 2/2 Frequency	2.30E-02 - 2.30E-02 1.29E+00 - 1.29E+00 3.15E+00 - 4.80E+00 - LOCATION=SWMU 193B MED	1.00E-03 - 1.00E-03 1.20E+00 - 1.20E+00 IA=RGA Groundwater	N N N	1.20E-02 1.25E+00 3.98E+00	mg/L pCi/L pCi/L
cis-1,2-Dichloroethene Alpha activity Beta activity Analyte	1/2 1/2 2/2 Frequency of Detection	2.30E-02 - 2.30E-02 1.29E+00 - 1.29E+00 3.15E+00 - 4.80E+00 - LOCATION=SWMU 193B MED Detected Range	1.00E-03 - 1.00E-03 1.20E+00 - 1.20E+00 IA=RGA Groundwater Nondetected Range	N N N Distribution*	1.20E-02 1.25E+00 3.98E+00 Arithmetic	mg/L pCi/L pCi/L
cis-1,2-Dichloroethene Alpha activity Beta activity Analyte 1,1-Dichloroethene	1/2 1/2 2/2 Frequency of Detection 3/17	2.30E-02 - 2.30E-02 1.29E+00 - 1.29E+00 3.15E+00 - 4.80E+00 - LOCATION=SWMU 193B MED Detected Range 2.30E-04 - 2.00E-02	1.00E-03 - 1.00E-03 1.20E+00 - 1.20E+00 IA=RGA Groundwater Nondetected Range 1.90E-04 - 5.00E-02	N N N Distribution* L	1.20E-02 1.25E+00 3.98E+00 Arithmetic	mg/L pCi/L pCi/L
cis-1,2-Dichloroethene Alpha activity Beta activity Analyte 1,1-Dichloroethene Acetone	1/2 1/2 2/2 Frequency of Detection 3/17 1/2	2.30E-02 - 2.30E-02 1.29E+00 - 1.29E+00 3.15E+00 - 4.80E+00 - LOCATION=SWMU 193B MED Detected Range 2.30E-04 - 2.00E-02 3.30E-02 - 3.30E-02	1.00E-03 - 1.00E-03 1.20E+00 - 1.20E+00 IA=RGA Groundwater Nondetected Range 1.90E-04 - 5.00E-02 1.00E-02 - 1.00E-02	N N N Distribution*	1.20E-02 1.25E+00 3.98E+00 Arithmetic Mean	mg/L pCi/L pCi/L Units
cis-1,2-Dichloroethene Alpha activity Beta activity Analyte 1,1-Dichloroethene Acetone Carbon Tetrachloride	1/2 1/2 2/2 Frequency of Detection 3/17 1/2 1/5	2.30E-02 - 2.30E-02 1.29E+00 - 1.29E+00 3.15E+00 - 4.80E+00 - LOCATION=SWMU 193B MED Detected Range 2.30E-04 - 2.00E-02 3.30E-02 - 3.30E-02 5.50E-03 - 5.50E-03	1.00E-03 - 1.00E-03 1.20E+00 - 1.20E+00 IA=RGA Groundwater Nondetected Range 1.90E-04 - 5.00E-02 1.00E-02 - 1.00E-02 5.00E-03 - 5.00E-02	N N N Distribution* L	1.20E-02 1.25E+00 3.98E+00 Arithmetic Mean 3.36E-04	mg/L pCi/L pCi/L Units mg/L
cis-1,2-Dichloroethene Alpha activity Beta activity Analyte 1,1-Dichloroethene Acetone Carbon Tetrachloride Di-n-butylphthalate	1/2 1/2 2/2 Frequency of Detection 3/17 1/2 1/5 2/10	2.30E-02 - 2.30E-02 1.29E+00 - 1.29E+00 3.15E+00 - 4.80E+00 - LOCATION=SWMU 193B MED Detected Range 2.30E-04 - 2.00E-02 3.30E-02 - 3.30E-02	1.00E-03 - 1.00E-03 1.20E+00 - 1.20E+00 IA=RGA Groundwater Nondetected Range 1.90E-04 - 5.00E-02 1.00E-02 - 1.00E-02	N N N Distribution* L N	1.20E-02 1.25E+00 3.98E+00 Arithmetic Mean 3.36E-04 1.90E-02	mg/L pCi/L pCi/L Units mg/L mg/L mg/L
cis-1,2-Dichloroethene Alpha activity Beta activity Analyte 1,1-Dichloroethene Acetone Carbon Tetrachloride	1/2 1/2 2/2 Frequency of Detection 3/17 1/2 1/5	2.30E-02 - 2.30E-02 1.29E+00 - 1.29E+00 3.15E+00 - 4.80E+00 - LOCATION=SWMU 193B MED Detected Range 2.30E-04 - 2.00E-02 3.30E-02 - 3.30E-02 5.50E-03 - 5.50E-03	1.00E-03 - 1.00E-03 1.20E+00 - 1.20E+00 IA=RGA Groundwater Nondetected Range 1.90E-04 - 5.00E-02 1.00E-02 - 1.00E-02 5.00E-03 - 5.00E-02	N N N Distribution* L N N	1.20E-02 1.25E+00 3.98E+00 Arithmetic Mean 3.36E-04 1.90E-02 1.21E-02	mg/L pCi/L pCi/L Units mg/L mg/L mg/L mg/L
cis-1,2-Dichloroethene Alpha activity Beta activity Analyte 1,1-Dichloroethene Acetone Carbon Tetrachloride Di-n-butylphthalate	1/2 1/2 2/2 Frequency of Detection 3/17 1/2 1/5 2/10	2.30E-02 - 2.30E-02 1.29E+00 - 1.29E+00 3.15E+00 - 4.80E+00 - LOCATION=SWMU 193B MED Detected Range 2.30E-04 - 2.00E-02 3.30E-02 - 3.30E-02 5.50E-03 - 5.50E-03 1.30E-02 - 1.30E-02	1.00E-03 - 1.00E-03 1.20E+00 - 1.20E+00 IA=RGA Groundwater Nondetected Range 1.90E-04 - 5.00E-02 1.00E-02 - 1.00E-02 5.00E-03 - 5.00E-02	N N N Distribution* L N N N	1.20E-02 1.25E+00 3.98E+00 Arithmetic Mean 3.36E-04 1.90E-02 1.21E-02 1.06E-02 8.01E-01	mg/L pCi/L pCi/L Units mg/L mg/L mg/L mg/L mg/L
cis-1,2-Dichloroethene Alpha activity Beta activity Analyte 1,1-Dichloroethene Acetone Carbon Tetrachloride Di-n-butylphthalate Trichloroethene	1/2 1/2 2/2 Frequency of Detection 3/17 1/2 1/5 2/10 17/17	2.30E-02 - 2.30E-02 1.29E+00 - 1.29E+00 3.15E+00 - 4.80E+00 - LOCATION=SWMU 193B MED Detected Range 2.30E-04 - 2.00E-02 3.30E-02 - 3.30E-02 5.50E-03 - 5.50E-03 1.30E-02 - 1.30E-02 1.00E-04 - 5.00E-01	1.00E-03 - 1.00E-03 1.20E+00 - 1.20E+00 IA=RGA Groundwater Nondetected Range 1.90E-04 - 5.00E-02 1.00E-02 - 1.00E-02 5.00E-03 - 5.00E-02 2.00E-02 - 2.00E-02	N N N Distribution* L N N N L	1.20E-02 1.25E+00 3.98E+00 Arithmetic Mean 3.36E-04 1.90E-02 1.21E-02 1.06E-02 8.01E-01 1.08E-02	mg/L pCi/L pCi/L Units mg/L mg/L mg/L mg/L mg/L mg/L
cis-1,2-Dichloroethene Alpha activity Beta activity Analyte 1,1-Dichloroethene Acetone Carbon Tetrachloride Di-n-butylphthalate Trichloroethene bis (2-Ethylhexyl)phthalate	1/2 1/2 2/2 Frequency of Detection 3/17 1/2 1/5 2/10 17/17 1/10	2.30E-02 - 2.30E-02 1.29E+00 - 1.29E+00 3.15E+00 - 4.80E+00 - LOCATION=SWMU 193B MED Detected Range 2.30E-04 - 2.00E-02 3.30E-02 - 3.30E-02 5.50E-03 - 5.50E-03 1.30E-02 - 1.30E-02 1.00E-04 - 5.00E-01 1.80E-02 - 1.80E-02	1.00E-03 - 1.00E-03 1.20E+00 - 1.20E+00 IA=RGA Groundwater Nondetected Range 1.90E-04 - 5.00E-02 1.00E-02 - 1.00E-02 5.00E-02 - 2.00E-02 2.00E-02 - 2.00E-02	N N Distribution* L N N L N L	1.20E-02 1.25E+00 3.98E+00 Arithmetic Mean 3.36E-04 1.90E-02 1.21E-02 1.06E-02 8.01E-01 1.08E-02 2.92E-03	mg/L pCi/L pCi/L Units mg/L mg/L mg/L mg/L mg/L mg/L mg/L
cis-1,2-Dichloroethene Alpha activity Beta activity Analyte 1,1-Dichloroethene Acetone Carbon Tetrachloride Di-n-butylphthalate Trichloroethene bis(2-Ethylhexyl)phthalate cis-1,2-Dichloroethene	1/2 1/2 2/2 Frequency of Detection 3/17 1/2 1/5 2/10 17/17 1/10 12/17	2.30E-02 - 2.30E-02 1.29E+00 - 1.29E+00 3.15E+00 - 4.80E+00 - LOCATION=SWMU 193B MED Detected Range 2.30E-04 - 2.00E-02 3.30E-02 - 3.30E-02 5.50E-03 - 5.50E-03 1.30E-02 - 1.30E-02 1.00E-04 - 5.00E-01 1.80E-02 - 1.80E-02 1.90E-04 - 9.87E-02	1.00E-03 - 1.00E-03 1.20E+00 - 1.20E+00 IA=RGA Groundwater Nondetected Range 1.90E-04 - 5.00E-02 1.00E-02 - 1.00E-02 5.00E-03 - 5.00E-02 2.00E-02 - 2.00E-02 2.20E-04 - 5.00E-03	N N N Distribution* L N N N L N	1.20E-02 1.25E+00 3.98E+00 Arithmetic Mean 3.36E-04 1.90E-02 1.21E-02 1.06E-02 8.01E-01 1.08E-02	mg/L pCi/L pCi/L Units mg/L mg/L mg/L mg/L mg/L mg/L

*L=Lognormal, N=Normal, NT=Not tested

	,	(continu	ed)			
	Frequency					
Analyte	of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
Beta activity	16/17	2.70E+00 - 5.85E+02	5.12E+00 - 5.12E+00	L	2.39E+01	pCi/L
Technetium-99	8/17		-5.00E-01 - 1.20E+01	N	1.89E+01	pCi/L
	:					
		LOCATION=SWMU 193B MET	IA-Subsurface Soil			
	Frequency	Determine 1	and a start for a			
Analyte	of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
Aluminum	4/4	7.43E+03 - 1.12E+04		N	9.98E+03	mg/kg
Barium	4/4	3.80E+01 - 8.42E+01		N	5.36E+01	mg/kġ
Beryllium	2/4	5.90E-01 - 1.57E+00	5.00E-01 - 5.00E-01	N	6.65E-01	mg/kg
Chromium	4/4	1.04E+01 - 8.87E+01		N	3.24E+01	mg/kg
Cobalt	4/4	3.18E+00 - 7.76E+00		N	4.99E+00	mg/kg
Copper	4/4	4.18E+00 - 7.43E+00		N	6.21E+00	mg/kg
Iron	4/4	9.73E+03 - 2.43E+04		N	1.45E+04	mg/kg
Lithium	4/4	3.44E+00 - 7.72E+00		N	5.82E+00	mg/kg
Magnesium	4/4	7.74E+02 - 4.31E+03		N	1.84E+03	
Manganese	4/4	1.05E+02 - 2.22E+02		N		mg/kg
Nickel	2/4	7.82E+00 - 2.06E+01	5.00E+00 - 5.00E+00	N	1.54E+02	mg/kg
Potassium	4/4	2.37E+02 - 6.86E+02	5.002+00 - 5.002+00	N	8.36E+00	mg/kg
Sodium	4/4	2.44E+02 - 4.48E+02			3.91E+02	mg/kg
	4/4			N	3.12E+02	mg/kg
Strontium		8.11E+00 - 9.39E+01		N	3.13E+01	mg/kg
Vanadium	4/4	1.75E+01 - 6.50E+01		N	3.10E+01	mg/kg
Zinc	4/4	1.75E+01 - 5.57E+01		N	3.09E+01	mg/kg
Acetone	1/1	8.00E-02 - 8.00E-02		NT	8.00E-02	mg/kg
Toluene	1/3	1.00E-02 - 1.00E-02	1.00E-02 - 1.00E-02	N	6.67E-03	mg/kg
Alpha activity	4/4	2.14E+00 - 1.86E+01		N	1.19E+01	pCi/g
Beta activity	4/4	9.10E+00 - 2.29E+01		N	1.52E+01	pCi/g
÷		- LOCATION=SWMU 193B M	DIA=Surface Soil	*****		
	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Aluminum	2/2	7.43E+03 - 1.08E+04		N	9.12E+03	mg/kg
Barium	2/2	3.80E+01 - 8.42E+01		N	6.11E+01	mg/kg

*L=Lognormal, N=Normal, NT=Not tested

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Continued)

	Frequency of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Beryllium	1/2	1.57E+00 - 1.57E+00	5.00E-01 - 5.00E-01	N	9.10E-01	mg/kg
Chromium	2/2	1.04E+01 - 8.87E+01		N	4.96E+01	mg/kg
Cobalt	2/2	3.82E+00 - 7.76E+00		Ň	5.79E+00	mg/kg
Copper	2/2	7.07E+00 - 7.43E+00		N	7.25E+00	mg/kg
Iron	2/2	1.16E+04 - 2.43E+04		N	1.80E+04	mg/kg
Lithium	2/2	3.44E+00 - 7.72E+00		N	5.58E+00	mg/kg
Magnesium	2/2	7.74E+02 - 4.31E+03		N	2.54E+03	mg/kg
Manganese	2/2	1.13E+02 - 2.22E+02		N	1.68E+02	mg/kg
Nickel	1/2	2.06E+01 = 2.06E+01	5.00E+00 - 5.00E+00	N	1.16E+01	mg/kg
Potassium	2/2	2.37E+02 - 6.86E+02		N	4.62E+02	mg/kg
Sodium	2/2	2.44E+02 - 2.49E+02		N	2.47E+02	mg/kg
Strontium	2/2	1.42E+01 - 9.39E+01		N N	5.41E+01	mg/kg
Vanadium	2/2	1.75E+01 - 6.50E+01		N	4.13E+01	mg/kg
Zinc	2/2	3.21E+01 - 5.57E+01		N	4.39E+01	mg/kg
Toluene	1/2	1.00E-02 - 1.00E-02	1.00E-02 - 1.00E-02	N	7.50E-03	mg/kg
Alpha activity	2/2	1.63E+01 - 1.86E+01		N	1.75E+01	pCi/g
Beta activity	2/2	1.66E+01 - 2.29E+01		N	1.98E+01	pCi/g

---- LOCATION-SWMU 193C MEDIA-McNairy Groundwater -----

Analyte	Frequency of Detection	Detected Ränge	Nondetected Range	Distribution*	Arithmetic Mean	Units
Aluminum	4/4	7.50E-01 - 9.04E+01		N	2.52E+01	mg/L
Antimony	5/5	6.00E-02 - 2.50E-01		N	1.48E-01	mg/L
Arsenic	5/5	5.00E-03 = 3.60E=02		Ň	1.12E-02	mg/L
Barium	5/5	9.20E-02 - 6.70E-01		N	2.65E-01	mg/L
Beryllium	5/5	5.00E-03 - 2.50E-02		N	1.54E-02	mg/L
Cadmium	5/5	1.00E-02 - 1.00E-01		N	3.62E-02	mg/L
Calcium	5/5	6.48E+00 = 4.10E+01		N	2.19E+01	mg/L
Chloride	5/5	1.45E+01 = 1.68E+01		N	1.56E+01	mg/L
Chromium	3/3	5.00E-02 - 2.32E-01		N	1.14E-01	mg/L
Cobalt	5/5	4.50E-02 - 1.21E-01		N	7.22E-02	mg/L
Copper	5/5	1.30E-02 - 1.63E-01		N	6.52E-02	mg/L
Fluoride	4/4	2.00E-01 - 2.80E-01		Ń	2.38E-01	mg/L
Iron	5/5	5.04E+00 - 1.79E+02		N	4.75E+01	mg/L
Lead	1/1	2.50E-01 - 2.50E-01		NT	2.50E-01	mg/L
Magnesium	5/5	2.14E+00 - 2.16E+01		N	7.47E+00	mg/L

*L=Lognormal, N=No-1, NT=Not tested

----- LOCATION=SWMU 193C MEDIA=McNairy Groundwater ------(continued)

	Frequency					
	of	Detected	Nondetected	•	Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Manganese	5/5	3.57E-01 - 3.91E+00		N	1.34E+00	mg/L
Mercury	1/1	2.00E-04 - 2.00E-04		NT	2.00E-04	mg/L
Molybdenum	4/4	5.00E-02 - 1.00E-01		N	6.38E-02	mg/L
Nickel	5/5	1.00E-01 - 1.09E-01		N	1.03E-01	mg/L
Nitrate as Nitrogen	5/5	1.00E+00 - 1.00E+00		N	1.00E+00	mg/L
Potassium	5/5	3.73E+01 - 1.01E+02		N	6.92E+01	mg/L
Selenium	3/3	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
Silica	5/5	1.10E+01 - 1.80E+01		N	1.32E+01	mg/L
Silver	3/3	5.00E-02 - 6.00E-02		N	5.67E-02	mg/L
Sodium	5/5	1.90E+01 - 2.63E+01		Ň	2.23E+01	mg/L
Tetraoxo-sulfate(1-)	5/5	9.60E+00 - 1.30E+01		N	1.19E+01	mg/L
Thallium	2/2	6.00E-02 - 1.23E-01		Ń	9.15E-02	mg/L
Uranium	9/9	1.00E-03 - 1.80E-02		N	2.89E-03	mg/L
Vanadium	2/2	5.70E-02 - 8.36E-01		N	4.47E-01	mg/L
Zinc	5/5	2.60E-02 - 5.64E-01		N	1.89E-01	mg/L
1,1,1-Trichloroethane	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
1,1,2-Trichloroethane	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
1,1-Dichloroethane	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
1,1-Dichloroethene	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
1,2-Dichloroethane	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/Ľ
Benzene	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
Bromodichloromethane	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
Carbon Tetrachloride	4/4	5.00E = 03 = 5.00E = 03		N	5.00E-03	mg/L
Chloroform	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
Ethylbenzene	4/4	5.00E-03 - 5.00E-03		Ň	5.00E-03	mg/L
Polychlorinated biphenyl	1/1	1.00E-04 - 1.00E-04	•	NT	1.00E-04	mg/L
Tetrachloroethene	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
Toluene	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
Trichloroethene	12/12	1.00E-03 - 2.00E-03		N	1.08E-03	mg/L
Vinyl Chloride	4/4	5.00E-03 - 1.00E-02		N	6.25E-03	mg/L
Xylene	4/4	5.00E-03 - 1.00E-02		N	7.50E-03	mg/L
cis-1,2-Dichloroethene	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
trans-1,2-Dichloroethene	4/4	5.00E-03 - 5.00E-03		N	5.00E-03	mg/L
Alpha activity	12/12	-1.80E+01 - 1.07E+02		N	7.47E+00	pCi/L
Beta activity	12/12	5.50E+01 - 2.36E+02		L	1.29E+02	pCi/L
Radon-222	2/2	1.43E+02 - 1.57E+02		N	1.50E+02	pCi/L
Technetium-99	13/13	-7.00E+00 - 2.70E+01		N	6.18E+00	pCi/L

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
1,2-Dichloroethene Trichloroethene	1/2 1/2	5.62E-01 - 5.62E-01 1.62E-01 - 1.62E-01		N N	2.82E-01	mg/L
Iffchiloroethene	1/2	1.026-01 - 1.026-01	1.002-03 - 1.002-03	И	8.15E-02	mg/L
	· · · · · · · · · · · · · · · · · · ·	- LOCATION=SWMU 193C MED	IA=Subsurface Soil			
	Frequency					
Analyte	of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
-	22/22	2 148.00 1 278.04		27	0.000.00	
Aluminum	20/20	3.14E+02 - 1.37E+04		N	8.52E+03	mg/kg
rsenic	5/20	5.01E+00 - 6.57E+00	5.00E+00 - 5.00E+00	N	3.33E+00	mg/kg
arium	1/1 10/20	1.42E+02 - 1.42E+02		NT	1.42E+02	mg/kg
Beryllium	1/20	5.00E-01 - 9.80E-01 1.00E+02 - 1.00E+02	5.00E-01 - 5.00E-01 1.00E+02 - 1.00E+02	N N	4.61E-01	mg/kg
Boron	3/59	2.41E-01 - 5.00E+02			5.25E+01	mg/kg
Cadmium Calcium	18/20	2.41E-01 - 5.00E+00 2.23E+02 - 4.00E+05	2.10E-01 - 5.00E+00 5.00E+00 - 5.00E+00	N	1.92E+00	mg/kg
Chromium	59/61	3.76E+00 - 8.30E+01	2.00E+00 - 2.00E+00	L L	2.60E+04	mg/kg
	17/20	1.22E+00 - 8.61E+01	1.00E+00 - 1.00E+00	L	1.89E+01	mg/kg
Cobalt	16/20	2.15E+00 - 2.82E+01	2.00E+00 - 2.00E+00		6.46E+00	mg/kg
Copper Iron	5/6	1.20E+04 - 3.00E+04	2.000+00 - 2.000+00	L	7.56E+00	mg/kg
Lead	42/61	5.10E+00 - 6.77E+01	5.00E+00 - 2.00E+01	N	2.22E+04	mg/kg
Lithium	17/20	2.50E+00 - 1.25E+01	2.00E+00 - 2.00E+01	L N	1.20E+01	mg/kg
Maqnesium	20/20	8.52E+02 - 1.45E+04	2.000+00 ~ 2.000+00	L	7.17E+00	mg/kg
Manganese	20/20	1.63E+01 - 2.27E+03		L	3.41E+03	mg/kg
Nickel	13/20	5.99E+00 - 2.15E+01	5.00E+00 - 5.00E+00	L	3.99E+02	mg/kg
Potassium	20/20	1.43E+02 - 1.57E+03	5.00E+00 - 5.00E+00		9.76E+00	mg/kg
Sodium	15/20	1.43E+02 = 1.57E+03 2.02E+02 = 4.44E+02		L	5.33E+02	mg/kg
Socium Strontium	20/20	7.25E+02 - 4.44E+02 7.25E+00 - 3.91E+02	2.00E+02 - 2.00E+02	N	2.62E+02	mg/kg
				L	1.06E+02	mg/kg
Vanadium	20/20	2.12E+00 - 4.42E+01		N	2.05E+01	mg/kg
Zinc	19/20	1.63E+01 - 9.25E+01	1.50E+01 - 1.50E+01	N	4.14E+01	mg/kg
Xylene	1/20	1.00E-02 - 1.00E-02	5.00E-03 - 5.00E-03	N	2.88E-03	mg/kg
Alpha activity	53/53	1.50B-03 - 4.00E+00		N	1.54E+00	pCi/g
Beta activity	53/53	5.00E-03 - 1.00E+01		N	4.45E+00	pCi/g

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-- LOCATION=SWMU 193C MEDIA=Surface Soil -----

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Analyte		Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
Aluminum		5/5	3.14B+02 - 3.36B+03		Ň	1.90E+03	mg/kg
Boron		1/5	1.00E+02 - 1.00E+02	1.00E+02 - 1.00E+02	N	6.00E+01	mg/kg
Calcium	an 19	5/5	2.53E+05 - 4.00E+05	• •	N	3.37E+05	mg/kg
Chromium		3/5	4.49E+00 - 1.20E+01	2.00E+00 - 2.00E+00	N	5.90E+00	mg/kg
Cobalt		2/5	1.22E+00 - 2.14E+00	1.00E+00 - 1.00E+00	N	9.72E-01	mg/kg
Copper		2/5	3.37E+00 - 2.82E+01	2.00E+00 - 2.00E+00	N	6,91E+00	mg/kg
Lead		1/5	6.77E+01 - 6.77E+01	2.00E+01 - 2.00E+01	N	2.15E+01	mg/kg
Lithium		3/5	5.43E+00 - 1.25E+01	2.00E+00 - 2.00E+00	N	6.03E+00	mg/kg
Magnesium		5/5	3.19E+03 - 1.45E+04	<u>.</u>	N	8.76E+03	mg/kg
Manganese		5/5	3.55E+01 - 1.98E+02		N	7.90E+01	mg/kg
Nickel		1/5	6.43E+00 - 6.43E+00	5.00E+00 - 5.00E+00	N	3.29E+00	mg/kg
Potassium		5/5	1.43E+02 - 1.57E+03		N .	7.01E+02	mg/kg
Sodium		4/5	2.29E+02 - 3.10E+02	2.00E+02 - 2.00E+02	N	2.37E+02	mg/kg
Strontium	· · ·	5/5	1.96E+02 - 3.91E+02	•	N	2.92E+02	mg/kg
Vanadium		5/5	2.12E+00 - 6.70E+00		N	4.00E+00	mg/kg
Zinc		5/5	4.59E+01 - 9.25E+01		N	6.59E+01	mg/kg

LOCATION-SWMU 194 MEDIA-Subsurface Soil -----

	Frequency of	Detected	Nondetected	· · ·	Ařithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Aluminum	12/12	5.38E+03 = 1.45E+04		L	9.47E+03	mg/kg
Arsenic	1/12	6.73E+00 - 6.73E+00	5.00E+00 - 5.00E+00	N	2.85E+00	mg/kg
Barium	12/12	2.05E+01 - 1.39E+02		N	7.51E+01	mg/kg
Beryllium	6/12	5.40E-01 - 4.80E+00	5.00E-01 - 5.00E-01	L	7.48E-01	mg/kg
Cadmium	1/35	8.55E+00 - 8.55E+00	2.00E+00 - 5.00E+00	Ľ	2.15E+00	mg/kg
Calcium	12/12	5.68E+02 - 6.81E+03		L	1.67E+03	mg/kg
Chromium	35/35	B.24E+00 - 1.03E+02		L	1.84E+01	mg/kg
Cobalt	12/12	2.52E+00 - 9.46E+00		Ĺ	5.05E+00	mg/kg
Copper	12/12	2.41E+00 - 1.67E+01		L	7.49Ë+00	mg/kg
Iron	12/12	6.41E+03 - 2.00E+04		L	1.24E+04	mg/kg
Lead	20/35	5.03E+00 - 3.60E+02	5.00E+00 - 2.00E+01	Г	1.19E+01	mg/kg
Lithium	12/12	2.41E+00 - 9.00E+00		N	6.45E+00	mg/kg
Magnesium	12/12	4.15E+02 - 2.34E+03		Ĺ	1.32E+03	mg/kg
Manganese	12/12	3.49E+01 - 4.67E+02		Ĺ	1.60E+02	mg/kg
Nickel	8/12	5.74E+00 - 1.37E+01	5.00E+00 - 5.00E+00	Ń	7.22E+00	mg/kg
Potassium	12/12	1.53E+02 - 6.32E+02		· L	3.80E+02	mg/kg
Sodium	8/12	2.10E+02 - 3.69E+02	2.00E+02 - 2.00E+02	N	2.37E+02	mg/kg

*L=Lognormal, N=Normal, NT=Not tested

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..... LOCATION-SWMU 194 MEDIA-Subsurface Soil ----- LOCATION-SWMU 194 MEDIA-Subsurface Soil -----

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
Střontium Vanadium Žinc	12/12 12/12 11/12	3.92E+00 - 2.60E+01 1.50E+01 - 2.58E+01 1.57E+01 - 6.76E+01	1.508+01 - 1.508+01	L L L	1.29E+01 1.99E+01 3.82E+01	mg/kg mg/kg mg/kg
Ethylbenzene Alpha activity Beta activity	1/19 23/23 23/23	1.50E-02 - 1.50E-02 1.20E+00 - 2.50E+00 3.00E+00 - 7.00E+00	5.00E-03 - 5.00E-03	N L N	3.16E=03 1.85E+00 4.83E+00	mg/kg pCi/g pCi/g
		OCATION=SWMU 99A MEDIA=	AcNairy Groundwater	-		PC1/9
	Frequency		-			
Analyte	of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units

Analyte	Detection	kange	Range	Distribution*	Mean	Units
1,1,1-Trichloroethane	1/4	1.20E-03 - 1.20E-03	5.00E-03 - 5.00E-03	N	2.18Ē-03	mg/L
1,1-Dichloroethene	1/4	2.29E-02 - 2.29E-02	1.00E-02 - 1.00E-02	N	9.48E-03	mg/L
Carbon Tetrachloride	1/4	2.80E-03 - 2.80E-03	5.00E-03 - 5.00E-03	N	2.58E-03	mg/L
Trichloroethene	3/4	3.00E-04 - 5.19E-01	5.00E-03 - 5.00E-03	N	1.31E-01	mg/L
cis-1,2-Dichloroethene	2/4	2.80E-02 - 1.15E-01	2.00E+00 = 2.00E+00	Ň	1.04E+00	mq/L
Alpha activity	2/2	2.60E+00 - 2.90E+00		Ň	2.75E+00	pCi/L
Beta activity	2/2	2.30E+01 - 3.50E+01		N	2.90E+01	pCi/L
Technetium-99	2/2	1.00E+01 - 1.90E+01		N	1.45E+01	pCi/L

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----- LOCATION-SWMU 99A MEDIA-RGA Groundwater -----

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
Aluminum	16/35	2.00E-01 - 6.59E+02	2.00E-01 - 1.00E+00	L	2.17E+00	mg/L
Arsenic	4/27	5.00E-03 - 1.00E-02	5.00B-03 - 5.00B-03	L	3.56E-03	mg/L
Barium	39/39	1.30E-01 - 3.30E+00		Ľ	5.41E-01	mg/L
Beryllium	8/35	8.00E-03 - 1.00E-01	5.00E-03 - 2.50E-02	L	3.87E-03	mg/L
Calcium	39/39	2.10E+01 - 1.20E+02		L	4.14E+01	mg/L
Chloride	9/9	5.68E+01 - 1.20E+02		Ĺ	6.83E+01	mg/L
Chromium	11/39	6.00E-02 - 1.78E+00	5.00E-02 - 6.00E-02	L	4.39E-02	mg/L
Cobalt	20/37	1.00E-02 - 5.70E-01	1.00E-02 - 1.00E-01	Ĺ	5.64E-02	mg/L
Copper	9/35	7.00E=02 - 6.40E-01	1.00Ē-02 - 1.00Ē-01	L	3.18E-02	mg/L
Fluoride	8/8	1.70E=01 = 2.00E=01		N	1.84E-01	mg/L

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----- LOCATION-SWMU 99A MEDIA-RGA Groundwater ------ LOCATION-SWMU 99A MEDIA-RGA Groundwater ------

	Frequency of	Detected	Nondetected	- 1 - 1 - 1 - 1 - 1	Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Iron	31/39	2.10E-01 - 1.20E+03	2.00E-01 - 3.55E-01	L	9.19E+00	mg/L
Lead	6/29	5.00E-02 - 4.10E-01	5.00E-02 - 2.50E-01	L	4.03E-02	mg/L
Lithium	6/23	5.00E-02 - 1.70E-01	5.00E-02 - 5.00E-02	Ŀ	4.45E-02	mģr/L
Magnesium	39/39	8.38E+00 - 4.97E+01		L	1.64E+01	mg/L
Manganese	37/39	4.30E-02 - 4.60E+00	1.00E-01 - 1.00E-01	L	1.13E+00	mg/L
Mercury	5/25	2.00E-04 - 2.00E-02	2.00E-04 - 2.00E-04	L	6.33E-05	mg/L
Nickel	16/39	5.00E-02 - 9.10E-01	5.00E-02 - 1.00E-01	L	9.25E-02	mg/L
Nitrate as Nitrogen	7/9	1.00E+00 - 2.10E+00	1.00E+00 - 1.00E+00	N	1.12E+00	mg/L
Potassium	24/39	2.08E+00 - 2.17E+01	2.00E+00 - 1.05E+01	L	4.17E+00	mg/L
Silica	9/9	1.50E+01 - 2.50E+01	•	L	1.87E+01	mg/L
Sodium	39/39	1.50E+01 - 7.24E+01	· .	N	5.34E+01	mg/L
Strontium	30/30	1.20E-01 - 4.70E-01		N	2.49E-01	mg/L
Sulfate	2/2	1.75E+01 - 1.92E+01		N	1.84E+01	mg/L
Tetraoxo-sulfate(1-)	7/7	1.10E+01 - 2.20E+01		N	1.67E+01	mg/L
Vanadium	10/28	8.50E-02 - 2.15E+00	1.00E-01 - 1.00E-01	L	1.49E-01	mg/L
Zinc	10/35	1.10E-02 - 2.55E+00	3.00E-02 - 2.50E-01	L	8.96E-02	mg/L
1,1-Dichloroethene	7/33	8.00E-03 - 6.50E-02	$1.00E-03 \div 5.00E-02$	N	1.70E-02	mg/L
Trichloroethene	41/43	2.00E-04 - 2.37E+00	1.00E-03 - 5.00E-03	\mathbf{L}	3.47E-01	mg/L
bis(2-Ethylhexyl)phthalate	5/10	6.00E-03 - 1.60E-02	2.00E-02 - 2.00E-02	L	1.31E-02	mg/L
cis-1,2-Dichloroethene	10/33	3.00E-04 - 3.48E-02	1.00E-03 - 2.00E+00	L	3.70E-03	mg/L
trans-1,2-Dichloroethene	3/33	3.00E-04 - 6.00E-04	1.00E-03 - 2.00E+00	L	5.49E-04	mg/L
Alpha activity	33/39	-2.50E+00 - 5.38E+01	-2.20E+00 - 2.60E+00	N	4.56E+00	pCi/L
Beta activity	39/39	3.00E+00 - 1.37E+02		L	3.16E+01	pCi/L
Radon-222	4/4	2.86E+02 - 6.75E+02		N	4.75E+02	pCi/L
Technetium-99	34/40	3.00E+00 - 1.39E+02	4.10E+00 - 1.70E+01	Ĺ	3.48E+01	pCi/L
		- LOCATION-SWMU 99A MEDI	A=Subsurface Soil			
	Frequency	Detected			-	
_ .	of	Detected	Nondetected		Arithmetic	· ·
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Aluminum	22/22	1.80E+03 - 1.41E+04		Ņ	7.55E+03	mg/kg
Antimony	5/22	1.70E+00 - 2.90E+00	2.00E+01 - 2.00E+01	L	2.16E+00	mg/kg
Arsenic	11/22	2.40E+00 - 8.55E+00	5.00E+00 - 5.00E+00	Ń	4.22E+00	mg/kg
Barium	22/22	2.03E+01 - 2.47E+03		L	1.53E+02	mg/kg
Beryllium	11/22	2.20E-01 - 8.90E-01	5.00E-01 - 5.00E-01	N	4.32E-01	mg/kg
Cadmium	5/22	7.50E-01 - 8.30E-01	2.00E+00 - 2.00E+00	L	8.19E-01	mg/kg
Calcium	20/20	1.14E+03 - 2.87E+05		L	1.66E+05	mg/kg

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----- LOCATION=SWMU 99A MEDIA=Subsurface Soil ----- LOCATION=SWMU 99A MEDIA=Subsurface Soil -----

	Frequency of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Chromium	22/22	7.00E+00 - 4.57E+01		L	1.44E+01	mg/kg
Cobalt	20/22	1.68 5 +00 - 1.19E+01	1.00E+00 - 1.00E+00	L	4.97E+00	mg/kg
Copper	21/22	3.80E+00 - 1.64E+01	2.00E+00 - 2.00E+00	N	7.31E+00	mg/kg
Cyanide	2/16	4.40B-01 - 5.40B-01	1.00E+00 - 1.00E+00	N	4.99E-01	mg/kg
Iron	22/22	1.45E+03 - 2.33E+04		N	1.18E+04	mg/kg
Lead	6/22	7.00E+00 - 4.73E+01	2.00E+01 - 2.00E+01	L	1.36E+01	mg/kg
Lithium	17/17	2.82E+00 - 1.29E+01		L	7.38E+00	mg/kg
Magnesium	22/22	3.97E+02 - 2.73E+04		L	6.49E+03	mg/kg
Manganese	22/22	3.93E+01 - 1.46E+03		L	3.02E+02	mg/kg
Mercury	5/22	8.00E-02 - 1.20E-01	2.00E-01 - 2.00E-01	N	9.95E-02	mg/kg
Nickel	17/22	2.50E+00 - 2.58E+01	5.00E+00 - 5.00E+00	L	9.72E+00	ng/kg
Potassium	22/22	2.28E+02 - 1.12E+03		L	5.27E+02	mg/kg
Selenium	5/20	2.90E-01 - 3.20E-01	1.00E+00 - 1.00E+00	L	3.11E-01	mg/kg
Silver	5/22	5.40E-01 - 7.10E-01	4.00E+00 - 4.00E+00	L	6.99E-01	mg/kg
Sodium	14/22	6.63E+01 - 3.93E+02	2.00E+02 - 2.51E+02	N	2.14E+02	mg/kg
Strontium	17/17	8.88E+00 - 5.14E+02		L	2.25E+02	mg/kg
Thallium	5/22	5.30E-01 - 5.90E-01	1.50E+01 - 1.50E+01	L	5.75E-01	mg/kg
Vanadium	22/22	4.48E+00 - 3.55E+01		N	1.79E+01	mg/kg
Zinc	21/22	1.16E+01 - 1.63E+02	4.76E+01 - 4.76E+01	L	6.96E+01	mg/kg
1,1,1-Trichloroethane	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
1,1,2,2-Tetrachloroethane	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
1,1,2-Trichloroethane	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
1,1-Dichloroethane	. 5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
1,1-Dichloroethene	5/10	6.00E-03 - 6.00E-03	1.98E-01 - 5.27E-01	N	8.91E-02	mg/kg
1,2,4-Trichlorobenzene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
1,2-Dichlorobenzene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
1,2-Dichloroethane	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
1,2-Dichloroethene	5/5	6.00E-03 - 6.00E-03		N	6.00E-03	mg/kg
1,2-Dichloropropane	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
1,3-Dichlorobenzene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
1,4-Dichlorobenzene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
2,4,5-Trichlorophenol	5/22	1.80E+00 - 2.10E+00	5.00E-01 - 5.00E-01	N	6.43E-01	mg/kg
2,4,6-Trichlorophenol	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
2,4-Dichlorophenol	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
2,4-Dimethylphenol	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
2,4-Dinitrophenol	5/6	1.80E+00 - 2.10E+00	4.80E-01 - 4.80E-01	N	1.69E+00	mg/kg
2,4-Dinitrotoluene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
2,6-Dinitrotoluene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
2-Butanone	5/8	6.00E-03 - 1.20E-02	1.00E-02 - 2.50E-01	L	1.15E-02	mg/kg
2-Chloronaphthalene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	
VL VIM Per VIM L VIN	-,		STOP OF STOPPOT	23	7.03E-01	mg/kg

A-70

----- LOCATION=SWMU 99A MEDIA=Subsurface Soil ------(continued)

	Frequency	•				
-	of	Detected	Nondetected		Arithmetic	• •
Analyte	Detection	Range	Range	Distribution*	Mean	Units
2-Chlorophenol	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
2-Hexanone	5/8	1.10E-02 - 1.20E-02	1.00E-02 - 1.00E-02	N	9.25E-03	mg/kg
2-Methyl-4,6-dinitrophenol	5/22	1.80E+00 - 2.10E+00	5.00B-01 - 5.00E-01	N	6.43E-01	mg/kg
2-Methylnaphthalene	5/22	3.608-01 - 4.10E-01	5.008-01 - 5.00E-01	N	2.83E-01	mg/kg
2-Methylphenol	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
2-Nitroaniline	5/22	1.80E+00 - 2.10E+00	5.00E-01 - 5.00E-01	N	6.43É-01	mg/kg
2-Nitrophenol	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
3,3'-Dichlorobenzidine	5/22	7.20E-01 - 8.20E-01	5.00E-01 - 5.00E-01	N	3.73E-01	mg/kg
3-Nitroaniline	5/22	1.80E+00 - 2.10E+00	5.00E-01 - 5.00E-01	N	6.43E-01	mg/kg
4,4'-DDD	2/2	2.00E-02 - 3.50E-02		N	2.75E-02	mg/kg
4, 4' - DDE	2/2	2.00E-02 - 3.50E-02		N	2.75E-02	mÿ/kġ
4,4'-DDT	2/2	2.00E-02 - 3.50E-02		Ň	2.75E-02	mg/kg
4-Bromophenyl phenyl ether	5/22	3.60B-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
4-Chloro-3-methylphenol	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
4-Chloroaniline	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
4-Chlorophenyl phenyl ether	5/22	3.60E-01 - 4.10B-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
4-Methyl-2-pentanone	5/8	1.10E-02 - 1.20E-02	1.00E-02 - 2.50E-01	L ·	1.19E-02	mg/kg
4-Methylphenol	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
4-Nitroaniline	5/22	1.80E+00 - 2.10E+00	5.00E-01 - 5.00E-01	N	6.43E-01	mg/kg
4-Nitrophenol	5/22	1.80E+00 - 2.10E+00	5.00E-01 - 5.00E-01	N	6.43E-01	mg/kg
Acenaphthene	7/22	3.00E-01 - 4.10E-01	5.00E-01 - 5.00E-01	L	3.89E-01	mg/kg
Acenaphthylene	6/22	3.60E-01 - 6.10E-01	5.00E-01 - 5.00E-01	L	4.32E-01	mg/kg
Acetone	5/8	1.20E-02 - 5.30E-02	1.00E-02 - 2.50E-01	Ĺ	2.47E-02	mg/kg
Aldrin	2/2	9.80E-03 - 1.70E-02		N	1.34E-02	mg/kg
Anthracene	7/22	3.60E-01 - 7.50E-01	5.00E-01 - 5.00E-01	L	4.42E-01	mg/kg
Benz (a) anthracene	8/22	2.20E-01 - 1.70E+00	5.00E-01 - 5.00E-01	Ľ	4.40E-01	mg/kg
Benzene	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Benzo (a) pyrene	7/22	3.60E-01 - 2.10E+00	5.00E-01 - 5.00E-01	L	4.38E-01	mg/kg
Benzo (b) fluoranthene	11/22	1.70E-01 - 5.70E+00	5.00E-01 - 5.00E-01	L	5.03E-01	mg/kg
Benzo (ghi) perylene	7/22	3.60E-01 - 1.18E+00	5.00E-01 - 5.00E-01	L	4.40E-01	mg/kg
Benzo(k) fluoranthene	8/22	3.60E-01 - 7.90E-01	5.00E-01 - 5.00E-01	L	4.52E-01	mg/kg
Benzoic Acid	5/5	1.80E+00 - 2.10E+00		N	1.98E+00	mg/kg
Benzyl Alcohol	5/5	3.60E-01 - 4.10E-01		N	3.94E-01	mg/kg
Bromodichloromethane	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Bromoform	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Bromomethane	5/8	1.10E-02 - 1.20E-02	1.00E-02 - 2.00E-02	N	1.05E-02	mg/kg
Butyl benzyl phthalate	5/6	3.60E-01 - 4.10E-01	4.80E-01 - 4.80E-01	N	3.68E-01	mg/kg
Carbon Disulfide	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Carbon Tetrachloride	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Chlorobenzene	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
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----- LOCATION-SWMU 99A MEDIA-Subsurface Soil ------ (continued)

	Frequency of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Chloroethane	5/8	1.10E-02 - 1.20E-02	1.00E-02 - 2.00E-02	N	1.05E-02	mg/kg
Chloroform	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Chloromethane	5/8	1.10E-02 - 1.20E-02	1.00E-02 - 2.00E-02	N	1.05E-02	mg/kg
Chrysene	7/22	3.60E-01 - 2.10E+00	5.00E-01 - 5.00E-01	L	4.38E-01	mg/kg
Di-n-butylphthalate	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Di-n-octylphthalate	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Dibenz (a, h) anthracene	6/22	3.60E-01 - 4.80E-01	5.00E-01 - 5.00E-01	N	2.93E-01	mg/kg
Dibenzofuran	6/22	1.23E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.77E-01	mg/kg
Dibromochloromethane	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Dieldrin	2/2	2.00E-02 - 3.50E-02		N	2.75E-02	mg/kg
Diethylphthalate	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Dimethylphthalate	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Endosulfan I	2/2	9.80E-03 - 1.70E-02		N	1.34E-02	mg/kg
Endosulfan II	2/2	2.00E-02 - 3.50E-02		N	2.75E-02	mg/kg
Endosulfan Sulfate	2/2	2.00E-02 - 3.50E-02		N	2.75E-02	mg/kg
Endrin	2/2	2.00E-02 - 3.50E-02		N	2.75E-02	mg/kg
Endrin Ketone	2/2	2.00E-02 - 3.50E-02		N	2.75E-02	mg/kg
Ethylbenzene	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Fluoranthene	9/22	1.40E-01 - 2.66E+00	5.00E-01 - 5.00E-01	L	4.23E-01	mg/kg
Fluorene	6/22	2.19E-01 - 4.10E-01	5.00E-01 - 5.00E-01	L	3.80E-01	mg/kg
Heptachlor	2/2	9.80E-03 - 1.70E-02		N	1.34E-02	mg/kg
Heptachlor Epoxide	2/2	9.80E-03 - 1.70E-02		N	1.34E-02	mg/kg
Hexachlorobenzene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Hexachlorobutadiene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Hexachlorocyclopentadiene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Hexachloroethane	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Indeno (1,2,3-cd) pyrene	7/22	3.60E-01 - 1.05E+00	5.00E-01 - 5.00E-01	L	4.46E-01	mg/kg
Isophorone	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Methoxychlor	2/2	9.80E-02 - 1.70E-01		N	1.34E-01	mg/kg
Methylene Chloride	5/8	2.00E-03 - 8.00E-03	1.00E-02 - 1.00E-02	L	5.20E-03	mg/kg
N-Nitroso-di-n-propylamine	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mq/kg
N-Nitrosodiphenylamine	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Naphthalene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Nitrobenzene	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kq
PCB-1016	3/23	9.80E-02 - 1.87E+00	1.02E-01 - 1.28E-01	L	2.54E-02	mg/kg
PCB-1221	2/23	9.80E-02 - 1.70E-01	1.02E-01 - 5.45E-01	L	7.82E-02	mg/kg
PCB-1232	2/23	9.80E-02 - 1.70E-01	1.02E-01 - 5.45E-01	L	7.82E-02	mg/kg
PCB-1242	2/23	9.80E-02 - 1.70E-01	1.02E-01 - 5.45E-01	Ľ	7.82E-02	mg/kg
PCB-1248	2/23	9.80E-02 - 1.70E-01	1.02E-01 - 5.45E-01	г	7.82E-02	mg/kg
PCB-1254	3/23	9,60E-02 - 3.50E-01	1.02E-01 - 5.45E-01	L	5.92E-02	mg/kg
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*L=Lognormal, N=Normal, NT=Not tested

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----- LOCATION=SWMU 99A MEDIA=Subsurface Soil ----- (continued)

•	Frequency					
	of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
PCB-1260	7/23	6.00E-02 - 6.31E-01	1.02E-01 - 5.45E-01	L	1.04E-01	mg/kg
Pentachlorophenol	5/22	1.80E+00 - 2.10E+00	5.00E-01 - 5.00E-01	N	6.43E-01	mg/kg
Phenanthrene	7/22	3.60E-01 - 1.63E+00	5.00E-01 - 5.00E-01	L	4.41E-01	mg/kg
Phenol	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
Pyrene	8/22	1.30E-01 - 2.70E+00	5.00E-01 - 5.00E-01	L	4.25E-01	mg/kg
Styrene	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Tetrachloroethene	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Toluene	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Toxaphene	2/2	2.00E-01 - 3.50E-01		Ň	2.75E-01	mg/kg
Trichloroethene	6/10	4.80E-03 - 6.00E-03	1.98E-01 - 5.27E-01	N	1.36E-01	mg/kg
Vinyl Acetate	5/5	1.10E-02 - 1.20E-02		N	1.18E-02	mg/kg
Vinyl Chloride	5/10	1.10E-02 - 1.20E-02	1.98E-01 - 1.00E+01	L	1.20E-02	mg/kg
Xylene	5/5	4.00E-03 - 6.00E-03		N	5.60E-03	mig/kg
alpha-BHC	2/2	9.80E-03 - 1.70E-02	-	N	1.34E-02	mg/kg
alpha-Chlordane	2/2	9.80E-02 - 1.70E-01		N	1.34E-01	mg/kg
beta-BHC	2/2	9.80E-03 - 1.70E-02		Ň	1.34E-02	mg/kg
bis (2-Chloroethoxy) methane	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	mg/kg
bis(2-Chloroethyl)ether	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	N	2.83E-01	πġ/kġ
bis (2-Chloroisopropyl) ether	5/22	3.60E-01 - 4.10E-01	5.00E-01 - 5.00E-01	Ņ	2.83E-01	mg/kg
bis (2-Ethylhexyl) phthalate	5/22	7.90E-02 - 3.60E-01	5.00E-01 - 5.00E-01	N	2.37E-01	mg/kg
cis-1,3-Dichloropropene	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
delta-BHC	2/2	9.80E-03 - 1.70E-02		N	1.34E-02	mg/kg
gauma-BHC (Lindane)	2/2	9.80E-03 - 1.70E-02		N	1.34E-02	mg/kg
gamma-Chlordane	2/2	9.80E-02 - 1.70E-01		N	1.34E-01	mg/kg
trans-1,3-Dichloropropene	5/8	6.00E-03 - 6.00E-03	1.00E-02 - 1.00E-02	N	5.63E-03	mg/kg
Alpha activity	20/21	9.70E+00 - 1.42E+02	3.10E+00 - 3.10E+00	L	2.33E+01	pCi/g
Beta activity	21/21	6.70E+00 - 2.73E+03		L	6.15E+01	pCi/g
Cesium-137	3/21	1.10E+00 - 1.90E+00	3.80E-01 - 3.50E+00	L	3.77E-01	pCi/g
Neptunium-237	4/4	-2.00E-03 - 1.28E+01		N	3.20E+00	pCi/g
Plutonium-239	3/3			Ň	1.00E-03	pCi/g
Technetium-99	6/23	-1.30E+00 - 2.65E+03	0.00E+00 - 3.73E+00	N	1.19E+02	pCi/g
Thorium-230	3/3	5.80E-01 - 6.70E-01		N	6.30E=01	pCi/g
Thorium-234	1/21	5.30E+01 - 5.30E+01	5.30E+00 - 2.20E+01	L	1.58E+01	pCi/g
Uranium-234	4/4	1.80E-01 - 1.64E+01		N	4.39E+00	pCi/g
Uranium-235	3/24	7.20E-03 - 4.10E-02	1.30E+00 - 9.90E+00	N	4.25E+00	pCi/g
Uranium-238	4/4	2.30B-01 - 5.17E+01	21202100 21202100	N	1.37E+01	pCi/g
01 ant am- 230	•/ •	2,502 01 511,2401			1.3/5+01	ber/a

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	Frequency of	Detected	Nondetected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Aluminum	13/13	1.80E+03 - 1.29E+04		L	6.19E+03	mg/kg
Arsenic	6/13	5.55E+00 - 8.55E+00	5.00E+00 - 5.00E+00	N	4.47E+00	mg/kg
Barium	13/13	2.08E+01 - 2.47E+03		L	2.11E+02	mg/kg
Beryllium	5/13	5.20E-01 - 8.90E-01	5.00B-01 - 5.00E-01	L	5.38E-01	mg/kg
Calcium	11/11	6.10E+03 - 2.87E+05		L	2.44E+05	mg/kg
Chromium	13/13	7.00E+00 - 4.57E+01		L	1.47E+01	mg/kg
Cobalt	11/13	1.68E+00 - 9.67E+00	1.00E+00 - 1.00E+00	L	3.70E+00	mg/kg
Copper	12/13	4.37E+00 - 1.22E+01	2.00E+00 - 2.00E+00	N	6.66E+00	mg/kg
Iron	13/13	1.45E+03 - 2.33E+04		L	1.09E+04	mg/kg
Lithium	13/13	2.82E+00 - 1.29E+01		L	7.58E+00	mg/kg
Magnesium	13/13	1.35E+03 - 2.73E+04		L	1.09E+04	mg/kg
Manganese	13/13	3.93E+01 - 3.87E+02		N	1.91E+02	mg/kg
Nickel	8/13	5.47E+00 - 2.16E+01	5.00E+00 - 5.00E+00	L	8.52E+00	mg/kg
Potassium	13/13	2.918+02 - 1.12E+03		L	5.47E+02	mg/kg
Sodium	6/13	2.17E+02 - 3.66E+02	2.00E+02 - 2.51E+02	N	1.95E+02	mg/kg
Strontium	13/13	1.46E+01 - 5.14E+02		L	2.71E+02	mg/kg
Vanadium	13/13	4.48E+00 - 3.55E+01		L	1.54E+01	mg/kg
Zinc	12/13	4.71E+01 - 1.63E+02	4.76E+01 - 4.76E+01	N	8.24E+01	mg/kg
Acenaphthene	2/13	3.00E-01 - 3.30E-01	5.00E-01 - 5.00E-01	L	3.22E-01	mg/kg
Acenaphthylene	1/13	6.10E-01 - 6.10E-01	5.00E-01 - 5.00E-01	N	2.78E-01	mg/kg
Anthracene	2/13	4.91E-01 - 7.50E-01	5.00E-01 - 5.00E-01	L	4.03E-01	mg/kg
Benz (a) anthracene	3/13	2.20E-01 - 1.70E+00	5.00E-01 - 5.00E-01	L	3.38E-01	mg/kg
Benzo (a) pyrene	2/13	1.70E+00 - 2.10E+00	5.00E-01 - 5.00E-01	N	5.04E-01	mg/kg
Benzo(b)fluoranthene	6/13	1.70E-01 - 5.70E+00	5.00E-01 - 5.00E-01	L	5.31E-01	mg/kg
Benzo (ghi) perylene	2/13	5.50E-01 - 1.18E+00	5.00E-01 - 5.00E-01	L	3.14E-01	mg/kg
Benzo(k)fluoranthene	3/13	4.66E-01 - 7.90E-01	5.00E-01 - 5.00E-01	L	4.53E-01	mg/kg
Chrysene	2/13	1.36E+00 - 2.10E+00	5.00E-01 - 5.00E-01	N	4.78E-01	mg/kg
Dibenz (a, h) anthracene	1/13	4.80E-01 - 4.80E-01	5.00E-01 - 5.00E-01	N	2.68E-01	mg/kg
Dibenzofur an	1/13	1.23E-01 - 1.23E-01	5.00E-01 - 5.00E-01	N	2.40E-01	mg/kg
Fluoranthene	4/13	1.40E-01 - 2.66E+00	5.00E-01 - 5.00E-01	L	3.38E-01	mg/kg
Fluorene	1/13	2.19E-01 - 2.19E-01	5.00E-01 - 5.00E-01	N	2.48E-01	mg/kg
Indeno (1,2,3-cd) pyrene	2/13	7.80E-01 - 1.05E+00	5.00E-01 - 5.00E-01	N	3.52E-01	mg/kg
PCB-1016	1/16	1.87E+00 - 1.87E+00	1.02E-01 - 1.28E-01	L	1.72E-01	mg/kg
PCB-1254	1/16	9.60E-02 - 9.60E-02	1.02E-01 - 5.45E-01	Ŀ	1.34E-01	mg/kg
PCB-1260	5/16	6.00E-02 - 6.31E-01	1.02E-01 - 5.45E-01	L	1.01E-01	mg/kg
Phenanthrene	2/13	8.50E-01 - 1.63E+00	5.00E-01 - 5.00E-01	L	2.45E-01	mg/kg
Pyrene	3/13	1.30E-01 - 2.70E+00	5.00E-01 - 5.00E-01	L	2.74E-01	mg/kg
Alpha activity	15/16	9.70E+00 - 1.42E+02	3.10E+00 - 3.10E+00	L	2.53E+01	pCi/g
Beta activity	16/16	6.70E+00 - 2.73E+03		L	8.58E+01	pCi/g
Cesium-137	3/16	1.10E+00 - 1.90E+00	3.80E-01 - 3.50E+00	L	4.50E-01	pCi/g
Neptunium-237	1/1	1.28E+01 - 1.28E+01		NT	1.28E+01	pCi/g

*L=Lognormal, N=Normal, NT=Not tested

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Analyte	Frequency of Detection	Detected. Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
Technetium-99	3/16	1.66E+01 - 2.65E+03	0.00E+00 - 3.73E+00	N	1.71E+02	pCi/g
Thorium-234	1/16	5.30E+01 - 5.30E+01	5.30E+00 - 2.20E+01	Ĺ	1.67E+01	pCi/g
Uranium-234	1/1	1.64E+01 - 1.64E+01		NT	1.64E+01	pCi/g
Úranium-238	1/1	5.17E+01 - 5.17E+01		NT	5.17E+01	pCi/g

----- LOCATION=SWMU 99B MEDIA=RGA Groundwater

Analyte	Frequency of Detection	Detected Range	Nondetected Range	Distribution*	Arithmetic Mean	Units
Barium	7/7	2.00E-01 - 2.70E+00		L	4.78Ė-01	mġ/L
Calcium	7/7	2.84E+01 - 3.27E+01		L	3.04E+01	mg/L
Chloride	7/7	8.29E+01 - 1.08E+02		N	9.59E+01	mg/L
Chromium	1/7	2.60E-01 - 2.60E-01	5.00E-02 - 6.00E-02	L	5.62E-02	mg/L
Copper	1/7	4.00E-02 - 4.00E-02	2.50E-02 - 1.00E-01	N	3.25E-02	mg/L
Fluoride	7/7	1.60E-01 - 2.10E-01		L	1.77E-01	mg/L
Iron	3/7	2.94E-01 - 3.34E+00	3.00E-01 - 3.60E-01	L	6.08E-01	mg/L
Magnesium	7/7	1.15E+01 - 1.31E+01		L	1.23E+01	mg/L
Manganese	5/7	6.00E-02 - 2.90E-01	1.00E-01 - 1.00E-01	L	1.65E-01	mg/L
Nitrate as Nitrogen	7/7	1.70E+00 - 2.10E+00		L .	1.84E+00	mg/L
Silica	7/7	1.50E+01 - 2.00E+01		L	1.72E+01	mg/L
Sodium	7/7	6.32E+01 - 7.86E+01		L	6.99E+01	mg/L
Sulfate	2/2	1.75E+01 = 2.67E+01		N	2.21E+01	mg/L
Tetraoxo-sulfate(1-)	5/5	1.92E+01 - 2.90E+01		N	2.46E+01	mg/L
Zinc	2/7	3.00E-02 - 6.00E-02	3.00E-02 - 2.50E-01	L	3.72E-02	mg/L
Trichloroethene	16/16	1.30E+00 - 2.30E+00		N	1.94E+00	mg/L
Alpha activity	12/16	-4.20E+00 - 4.20E+00	-2.03E+00 - 4.60E+00	N	7.42E-01	pCi/L
Beta activity	16/16	3.00E+00 - 4.50E+01		L	1.20E+01	pCi/L
Radon-222	4/4	2.57E+02 - 4.12E+02		N	3.66E+02	pCi/L
Technetium-99	12/17	-2.00E+00 - 1.90E+01	-3.00E+00 - 1.17E+01	N	5.51E+00	pCi/L

*L=Lognormal, N=Normal, NT=Not tested

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----- LOCATION-SWMU 99B MET

UM	99B	MEDIA-Subsurface	Sőil	
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2	Frequency	Detected	Nondétected		Arithmetic	
Analyte	Detection	Range	Range	Distribution*	Mean	Units
Aluminum	8/8	9.31E+03 - 1.70E+04		L	1.25E+04	mg/kg
Arsenic	2/6	6.89E+00 - 8.05E+00	5.00E+00 - 5.00E+00	L N L	4.16E+00	mg/kg
Barium	8/8	6.50E+01 - 1.55E+02		Ĩ	9.63E+01	mg/kg
Beryllium	6/8	5.70E-01 - 1.00E+00	5.00E-01 - 5.00E-01	N	5.66E-01	mg/kg
Calcium	6/6	5.03E+02 - 7.17E+03		Ň	2.51E+03	mg/kg
Chromium	8/8	1.18E+01 - 2.61E+01		L	1.79E+01	mg/kg
Cobalt	8/8	1.91E+00 - 6.94E+00		Ľ.	4.17E+00	mg/kg
Copper	8/8	5.25E+00 - 1.30E+01		Ň	8.66E+00	mg/kg
Iron	8/8	9.66E+03 - 1.81E+04		N	1.48E+04	mg/kg
Lithium	8/8	6.50E+00 - 1.14E+01		L	8.62E+00	mg/kg
Magnesium	B/8	1.10B+03 = 2.53B+03		Ń	1,76E+03	mg/kg
Manganese	8/8	6.32E+01 - 5.24E+02		L	2.43E+02	mg∕kg
Nickel	5/8	7.275+00 - 2.515+01	5.00E+00 - 5.00E+00	Ē	1.05E+01	mg/kg
Potassium	8/8	3.37E+02 - 1.04E+03		L	6.44E+02	mg/kg
Sodium	3/8	2.11E+02 - 3.09E+02	2.00E+02 - 2.00E+02	Ň	1.58E+02	mg/kg
Strontium	8/8	9.46E+00 - 2.22E+01		Ĩ.	1.61E+01	mg/kg
Vanadium	8/8	1.97E+01 - 3.44E+01		Ē	2.46E+01	mg/kg
Zinc	8/8	1.96E+01 - 5.22E+01		- N	3.74E+01	mg/kg
Acetone	1/7	5.50E=01 - 5.50E-01	1.20E+00 - 1.20E+00	N	5.93E-01	mg/kg
Methylene Chloride	3/7	1.20E+00 = 1.20E+00	1.20E+00 = 1.20E+00	N	8.57E-01	mg/kg
Alpha activity	8/8	1.33E+01 - 2.14E+01		N	1.73E+01	pCi/g
Beta activity	8/8	1.48E+01 - 2.26E+01		Ĺ	1.86E+01	pCi/g

	Frequency of	Maximum detected			Exceed	Exceed	•
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
1,1,1-Trichloroethane	11/11	1.80E-02	5.4E-02	•	No		mg/L
1,1-Dichloroethane	1/1	5.00E+00	2.7E-02		Yes		mg/L
1,1-Dichloroethene	12/15	4.00E-02	1.8E-03	9.3E-07	Yes	Yes	mg/L
PCB-1254	11/11	2.50E-02	1.9E-05	8.0E-06	Yes	Yes	mg/L
CB-1260	11/11	2.50E-02		4.4E-06		Yes	mg/L
Polychlorinated biphenyl	1/1	1.70B-01		8.0E-06		Yes	mg/L
Tetrachloroethene	11/11	5.00E+00	7.9E-03	5.7E-05	Yes	Yes	mg/L
richloroethene	15/15	7.70E-01	1.2E-03	1.4E-04	Yes	Yes	mģr∕L
inyl Chloride	1/4	1.00E-04		1.7E-06		Yes	mg/L
is-1,2-Dichloroethene	3/4	6.00E-03	2.0E-03		Yes		mg/L
rans-1,2-Dichloroethene	2/4	1.00E-04	4.0E-03		No		mg/L
lpha activity	2/4	6.80E+00		•			pCi/L
Beta activity	2/4	5.20E+00					pCi/L
	LOCATI	ON=AOC 204 MEDIA=	Subsurface So	oil			
	Frequency	Maximum					
- .	of	detected			Exceed	Exceed	
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
1,1,1-Trichloroethane	10/16	1.00E+00	1.2E+02		No		mg/kg
1,1-Dichloroethane	8/14	1.002+00	6.7E+01		No		mg/kg
1,1-Dichloroethene	11/17	4.00E-02	3.5E+00	3.9E-03	No	Yes	mg/kg
PCB-1254	11/11	2.50E-02	6.6E-02	9.9E-03	No	Yes	mg/kg
PCB-1260	11/11	2.50B-02		9.8E-03		Veŝ	ma/ka

1,1,1-Trichloroethane	10/16	1.00E+00	1.2E+02		No		mg/kg
1,1-Dichloroethane	8/14	1.00E+00	6.7E+01		No		mg/kg
1,1-Dichloroethene	11/17	4.00E-02	3.5E+00	3.9E-03	No	Yes	mg/kg
PCB-1254	11/11	2.50E-02	6.6E-02	9.9E-03	No	Yes	mg/kg
PCB-1260	11/11	2.50E-02		9.8E-03		Yes	mg/kg
Polychlorinated biphenyl	8/8	1.00E-01		1.0E-02		Yes	mg/kg
Tetrachloroethene	11/17	1.00E+00	1.2E+01	1.3E-01	No	Yes	mg/kg
Trichloroethene	11/17	1.00E+00	1.25+00	9.1E-02	No	Yes	mg/kg
Alpha activity	6/6	1.96E+01	•				pCi/g
Beta activity	6/6	2.91E+01			•		pCi/g
	·						
	LOCATION=	SWMU 193A MEDIA-	McNairy Groun	dwater			
			-	• • •			

		Frequency	Maximum						
Analyt	e	of Detection	detected concentration	HI	ELCR	Exceed HI?	Exceed ELCR?	Units	-
Calciu	m	4/4	8.21E+01					mg/L	

	Frequency	Maximum			Ëxceed	Exceed	
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Unite
Thloride	4/4	1.60E+01					mg/L
fron	4/4	3.14E+02	4.5E-01		Yes		mg/L
Magnesium	4/4	4.02E+01					mg/L
Potassium	4/4	2.15E+01					mg/L
Sodium	4/4	8.08E+01					mg/L
etraoxo-sulfate(1-)	4/4	8.40E+01					mg/L
cetone	1/1	1.40E-02	2.0E-02		No		mg/L
liethylphthalate	1/5	1.90E=02	1.2E+00		No		mg/L
richloroethene	8/13	1,10E-02	1.2E-03	1.4E-04	Yes	Yes	mg/L
cis-1,2-Dichloroethene	1/11	1.70E-01	2.0E-03		Yes		mg/L
lpha activity	6/10	4.00E+01					pCi/l
eta activity	8/10	6.46E+01				• .	pCi/I
echnetium-99	5/10	1.45E+02		2.8E+01		Yes	pCi/I
Chorium-234	1/1	8.40E-01		2.0E+00		No	pCi/I
Uranium-234	1/1	8.10E-01	•	8.7E-01		No	pCi/I
Uranium-238	1/1	1.32E+00		6.2E-01		Yes	pCi/I

--- LOCATION-SWMU 193A MEDIA-RGA Groundwater -----

	Frequency of	Maximum detected			Exceed	Exceed	
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
Aluminum	4/4	1.78E-01	1.5E+00	•	No		₩ġ/L
Ammonia	1/1	3.00E-01					mg/L
Calcium	5/5	1.34E+02					mg/L
Chloride	5/5	6.40E+01					mg/L
Copper	1/4	1.80E-02	6.0E-02		No		mg/L
Fluoride	1/1	4.20E-01	9.1E-02		Yes		mg/L
Iron	. 7/9	3.66E+01	4.5E-01		Yes		mg/L
Magnesium	.5/5	1.85E+01					mg/L
Potassium	5/5	2.65E+02		•			mg/L
Silica	1/1	1.90E+01					mg/L
Sodium	5/5	1.34E+02					mg/L
Tetraoxo-sulfate(1-)	5/5	2.62E+02					mg/L
Zinc	4/4	2.12E-01	4.5E-01		No		mg/L
1,1-Dichloroethene	2/43	2.00E-04	1.8E-03	9.3E-07	No	Yes	mg/L
Diethylphthalate	3/25	1.50E-02	1.2E+00		No		mg/L

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(continued)								
Analyte	Frequency of Detection	Maximum detected concentration	HI	ELCR	Exceed HI?	Exceed ELCR?	Ünits	
Pentach1oropheno1	1/25	1.20E-02	2.3E-02	2.1E-05	No	Yes	mg/L	
Trichloroethene	44/51	6.70B+00	1.2E-03	1,4Ë-04	Yes	Yes	mg/L	
bis(2-Ethylhexyl)phthalate	3/25	2.202-02	2.6E-02	3.1E-04	No	Yes	mg/L	
cis-1,2-Dichloroethene	17/42	8.40E-02	2.0E-03		Yes		mg/L	
trans-1,2-Dichloroethene	7/43	7.00E-04	4.0E-03		No		mg/L	
Alpha activity	19/34	1.76E+01					pCi/L	
Beta activity	34/34	8.80B+02			-		pCi/L	
Technetium-99	26/39	1.39E+03		2.8E+01		Yes	pCi/L	
Thorium-234	1/8	5.40E-01		2.0E+00		No	pCi/L	

LOCATION-SWMU 193A MEDIA-Subsurface Soil --

	Frequency of	Maximum detected			Exceed	Freed	
Analyte	Detection	concentration	HI	ELCR	HI?	Exceed ELCR?	Units
Aluminum	8/8	1.40E+04	7.3Ë+02		Yes		mg/kg
Barium	8/8	8.73E+01	3.7E+01		Yes		mg/kg
Beryllium	5/8	7.00Å-01	1.6E-01	1.0E-04	Yes	Yes	mg/kg
Calcium	4/4	2.73E+05					mg/kg
Chromium	9/8	2.77E+01	4.8E-01	4.9E+02	Yes	No	mg/kg
Cobalt	8/8	8.66E+00	2.1E+02		No		mg/kg
Copper	8/8	7.31E+00	7.4E+01		No		mg/kg
Iron	8/8	1.548+04	3.1E+02		Yes		mg/kg
Lithium	8/8	1.12E+01	7.0E+01		No		mg/kg
Magnesium	8/8	1.70E+04					mg/kg
Manganese	8/8	5.64E+02	1.42+01		Yes		mg/kg
Nickel	5/8	9,16E+00	3.4E+01		No		mg/kg
Potassium	8/8	1.44E+03					mg/kg
Silver	1/8	4.00E+00	6.1E+00		No		mg/kg
Sodium	5/8	3.13E+02	•	•			mg/kg
Strontium	8/8	2.53E+02	8.0E+02		No		mg/kg
Vanadium	8/8	3.15E+01	5.6E-01		Yes		mg/kg
Zinc	8/8	5.54B+01	4.0E+02		No		mg/kg
Acetone	1/2	1.10E-02	9.2E+01		No		mg/kg
Anthracene	1/8	1.16E-01	6.5E+02	· ·	No ·		mg/kg
Benz (a) anthracene	2/8	1.80E-01		8.5E-03		Yes	mg/kg
Benzo (a) pyrene	2/8	2.50E-01		8.5E-04		Yes	mg/kg
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		(continued)	1				
	Frequency	Maximum					
	of	detected			Exceed	Exceed	
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
Benzo(b)fluoranthene	2/8	5.108-02		8.5E-03		Yes	mg/kg
Benzo(ghi)perylene	2/8	1.70E-01					mg/kg
Chrysene	2/8	1.70E-01		8.5E-01		No	mg/kg
Di-n-butylphthalate	1/8	. 7.70E-02	2.6E+02		No		mg/kg
Di-n-octylphthalate	1/8	1.20E-01	4.9E+01		No		mg/kg
Dibenz (a, h) anthracene	1/8	1.308-01		8.5E-04		Yes	mg/kg
Diethylphthalate	1/8	4.00E-01	2.0E+03		No		mg/kg
Fluoranthene	2/8	3.10E-01	4.3E+01		No		mg/kg
Indeno(1,2,3-cd)pyrene	2/8	1.60E-01		8.5E-03		Yes	mg/kg
Pyrene	2/8	2.95E-01	3.2E+01		No		mg/kg
bis (2-Ethylhexyl) phthalate	2/8	1.70E-01	1.4E+01	2.8E-01	No	No	mg/kg
Alpha activity	8/8	2.60E+01					pCi/g
Beta activity	8/8	2.37E+01				•	pCi/g
	Frequency	Maximum			·		
	of	detected		PT (79	Exceed	Exceed	Thite
Analyte	of Detection		Ш	ELCR	Exceed HI?	Exceed ELCR?	Units
Aluminum	of Detection 4/4	detected concentration 1.09B+04	7.3E+02	ELCR	HI? Yes		mg/kg
Aluminum Barium	of Detection 4/4 4/4	detected concentration 1.09E+04 8.40E+01	7.3E+02 3.7E+01		HI?	ELCR?	mg/kg mg/kg
Aluminum Barium Beryllium	of Detection 4/4 4/4 1/4	detected concentration 1.09E+04 8.40E+01 6.40E-01	7.3E+02	ELCR 1.05-04	HI? Yes		mg/kg mg/kg mg/kg
Aluminum Barium Beryllium Calcium	of Detection 4/4 4/4 1/4 2/2	detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05	7.3E+02 3.7E+01 1.6B-01	1.05-04	HI? Yes Yes Yes	ELCR? Yes	mg/kg mg/kg mg/kg mg/kg
Aluminum Barium Beryllium Calcium Chromium	of Detection 4/4 4/4 1/4 2/2 4/4	detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01	7.3E+02 3.7E+01 1.6E-01 4.8E-01		HI? Yes Yes Yes Yes	ELCR?	mg/kg mg/kg mg/kg mg/kg mg/kg
Aluminum Barium Beryllium Calcium Chromium Cobalt	of Detection 4/4 4/4 1/4 2/2 4/4 4/4	detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00	7.3E+02 3.7E+01 1.6E-01 4.8E-01 2.1E+02	1.05-04	HI? Yes Yes Yes No	ELCR? Yes	mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg
Aluminum Barium Beryllium Calcium Chromium Cobalt Copper	of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4	detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00	7.3E+02 3.7E+01 1.6E-01 4.8E-01 2.1E+02 7.4E+01	1.05-04	HI? Yes Yes Yes No No	ELCR? Yes	mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg
Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron	of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4	detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04	7.3E+02 3.7E+01 1.6E-01 4.8E-01 2.1E+02 7.4E+01 3.1E+02	1.05-04	HI? Yes Yes Yes No No Yes	ELCR? Yes	mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg
Aluminum Barium Beryllium Calcium Chromium Cobalt Copper . Iron Lithium	of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4	detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00	7.3E+02 3.7E+01 1.6E-01 4.8E-01 2.1E+02 7.4E+01	1.05-04	HI? Yes Yes Yes No No	ELCR? Yes	mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg
Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron	of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4	detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01 1.70E+04	7.3E+02 3.7E+01 1.6E-01 4.8E-01 2.1E+02 7.4E+01 3.1E+02 7.0E+01	1.05-04	HI? Yes Yes Yes No No Yes	ELCR? Yes	mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg
Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium Magnesium Manganese	of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4	detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01	7.3E+02 3.7E+01 1.6E-01 4.8E-01 2.1E+02 7.4E+01 3.1E+02 7.0E+01 1.4E+01	1.05-04	HI? Yes Yes Yes No No Yes	ELCR? Yes	mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg
Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium Magnesium	of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4	detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01 1.70E+04	7.3E+02 3.7E+01 1.6E-01 4.8E-01 2.1E+02 7.4E+01 3.1E+02 7.0E+01	1.05-04	HI? Yes Yes Yes No No Yes No	ELCR? Yes	mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg
Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium Magnesium Manganese	of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4 2/4 4/4	detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01 1.70E+04 3.98E+02 7.50E+00 1.44E+03	7.3E+02 3.7E+01 1.6E-01 4.8E-01 2.1E+02 7.4E+01 3.1E+02 7.0E+01 1.4E+01	1.05-04	HI? Yes Yes Yes No No Yes No Yes	ELCR? Yes	mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg
Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium Magnesium Manganese Nickel	of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4	detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01 1.70E+04 3.98E+02 7.50E+00	7.3E+02 3.7E+01 1.6E-01 4.8E-01 2.1E+02 7.4E+01 3.1E+02 7.0E+01 1.4E+01	1.05-04	HI? Yes Yes Yes No No Yes No Yes	ELCR? Yes	mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg
Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium Magnesium Magnesium Manganese Nickel Potassium Sodium Strontium	of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4	detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01 1.70E+04 3.98E+02 7.50E+00 1.44E+03	7.3E+02 3.7E+01 1.6B-01 4.8E-01 2.1E+02 7.4E+01 3.1E+02 7.0E+01 1.4E+01 3.4E+01 8.0E+02	1.05-04	HI? Yes Yes No No Yes No Yes No	ELCR? Yes	mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg
Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium Magnesium Manganese Nickel Potassium Sodium	of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4	detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01 1.70E+04 3.98E+02 7.50E+00 1.44E+03 2.13E+02 2.53E+02 3.15E+01	7.3E+02 3.7E+01 1.6B-01 4.8E-01 2.1E+02 7.4E+01 3.1E+02 7.0E+01 1.4E+01 3.4E+01 8.0E+02 5.6E-01	1.05-04	HI? Yes Yes Yes No Yes No Yes No	ELCR? Yes	mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg
Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium Magnesium Magnesium Manganese Nickel Potassium Sodium Strontium	of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4	detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01 1.70E+04 3.98E+02 7.50E+00 1.44E+03 2.13E+02 2.53E+02	7.3E+02 3.7E+01 1.6B-01 4.8E-01 2.1E+02 7.4E+01 3.1E+02 7.0E+01 1.4E+01 3.4E+01 8.0E+02	1.05-04	HI? Yes Yes No No Yes No Yes No	ELCR? Yes	mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg

<u>.</u> . .

---- LOCATION-SWMU 193A MEDIA-Surface Soil -----(continued)

	Frequency of	Maximum detected			Exceed	Exceed	
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
Benz (a) anthracene	2/4	1.80E-01	•	8.5E-03		Yes	mg/kg
Benzo (a) pyrene	2/4	2.50E-01		8.5E-04		Yes	mg/kg
Benzo (b) fluoranthene	2/4	5.10E-02		8.5E-03		Yes	mg/kg
Benzo (ghi) perylene	2/4	1.70E-01					mg/kg
Chrysene	2/4	1.70E-01		8.5E-01		No	mg/kg
Di-n-butylphthalate	1/4	7.70E-02	2.6E+02		No		mg/kg
Di-n-octylphthalate	1/4	1.20E-01	4.9E+01		No		mg/kg
Dibenz (a, h) anthracene	1/4	1.30B-01		8.5E-04		Yes	mg/kg
Diethylphthalate	1/4 ·	4.00E-01	2.0E+03		No		ng/kg
Fluoranthene	2/4	3.10E-01	4.3E+01	•	No		mg/kg
Indeno (1, 2, 3-cd) pyrene	2/4	1.60E-01		8.5B-03		Yes	mg/kg
Pyrene	2/4	2.95E-01	3.2E+01		No		mg/kg
bis(2-Ethylhexyl)phthalate	- 1.	- 1.70E-01	1.4E+01	2.8E-01	No	No	mg/kg
Alpha activity	4/4	1.70E+01					pCi/g
Beta activity	4/4	2.37E+01	. •				pCi/g

- LOCATION-SWMU 193B MEDIA-McNairy Groundwater ------

Analyte	Frequency of Detection	Maximum detected concentration	HI	ELCR	Exceed HI?	Exceed ELCR?	Units
Trichloroethene cis-1,2-Dichloroethene Alpha activity Beta activity	1/2 1/2 1/2 2/2	1.30E-02 2.30E-02 1.29E+00 4.80E+00	1.2E-03 2.0E-03	1.4E-04	Yes Yes	Ŷes	mg/L mg/L pCi/L pCi/L

LOCATION-SWMU 193B MEDIA-RGA Groundwater

Analyte	Frequency of Detection	Maximum detected concentration	HI	ELCR	Exceed HI?	Exceed ELCR?	Units
1,1-Dichloroethene	3/17	2.00E-02	1.8E-03	9.3E-07	Yes	Yes	mg/L
Acetone	1/2	3.30E-02	2.0E-02		Yes		mg/L
Carbon Tetrachloride	1/5	5.50E-03	1.2E-04	1.5E-05	Yes	Yes	mg/L
Di-n-butylphthalate	2/10	1.30E-02	1.3E-01		No		mg/L

----- LOCATION=SWMU 193B MEDIA=RGA Groundwater ------(continued)

Analyte	Frequency of Detection	Maximum detected concentration	HI	ELCR	Exceed HI?	Exceed ELCR?	Units
Trichloroethene bis(2-Ethylhexyl)phthalate cis-1,2-Dichloroethene trans-1,2-Dichloroethene Alpha activity	17/17 1/10 12/17 8/17 12/17	5.00E-01 1.80E-02 9.87E-02 8.10E-04 6.60E+02	1.28-03 2.68-02 2.08-03 4.08-03	1.42-04 3.12-04	Yeb No Yes No	Yes Yes	mg/L mg/L mg/L mg/L
Beta activity Technetium-99	16/17 8/17	5.85E+02 6.10E+01		2.8E+01		Yes	pCi/L pCi/L pCi/L

- LOCATION-SWMU 1938 MEDIA-Subsurface Soil ------

Frequency	Maximum detected			Exceed	Exceed	
	centration	HI	ELCR	HI?	ELCR?	Units
Aluminum 4/4	1.122+04	7.3E+02		Yes		mg/kg
Barium 4/4	B.42E+01	3.7E+01		Yes		mg/kg
Beryllium 2/4	1.57E+00	1.6E-01	1.0E-04	Yes	Yes	mg/kg
Chromium 4/4	8.87E+01	4.8E-01	4.9E+02	Yes	No	mg/kg
Cobalt 4/4	7.76B+00	2.1E+02		No		mg/kg
Copper 4/4	7.43E+00	7.4E+01		No		mg/kg
Iron 4/4	2.43E+Ó4	3.1E+02		Yes		mg/kg
Lithium 4/4	7.728+00	7.0E+01		No		mg/kg
Magnesium 4/4	4.31E+03					mg/kg
Manganese 4/4	2.22E+02	1.4E+01		Yes		mg/kg
Nickel 2/4	2.06E+01	3.4E+01		No		mg/kg
Potassium 4/4	6.86E+02					mg/kg
Sodium 4/4	4.48E+02					mg/kg
Strontium 4/4	9.39E+01	8.0E+02		No		mg/kg
Vanadium 4/4	6.50E+01	5.6E-01		Yes		mg/kg
Zinc 4/4	5.57E+01	4,0E+02		No		mg/kg
Acetone 1/1	8.00E-02	9.2B+01		No		mg/kg
Toluene 1/3	1.00E-02	9.8E+01		No		mg/kg
Alpha activity 4/4	1.86E+01					pCi/g
Beta activity 4/4	2.29E+01					pCi/g



-- LOCATION-SWMU 193B MEDIA-Surface Soil ------

. Frequency Maximum detected of Exceed Exceed Analyte Detection concentration ΗI ELCR HI? ELCR? Units Aluminum 2/2 1.08E+04 7.3E+02 Yes mg/kg Barium 2/2 8.42E+01 3.7E+01 Yes mg/kg Beryllium 1/2 1.57E+00 1.6E-01 1.0B-04 Yes Yes mg/kg Chromium 2/2 8.87E+01 4.8E-01 4.9E+02 Yes No mg/kg 2/2 Cobalt 2.1E+02 7.76E+00 No mg/kg Copper 2/2 7.43E+00 7.4E+01 No mg/kg Iron 2/2 2.43E+04 3.1E+02 Yes mg/kg Lithium 2/2 7.72E+00 7.0E+01 No mg/kg 2/2 Magnesium 4.31E+03 mg/kg Manganese 2/2 2.22E+02 Yes 1.4E+01 mg/kg Nickel 1/2 2.06E+01 3.4E+01 No mg/kg Potassium 2/2 6.86E+02 mg/kg Sodium 2/2 2.49E+02 mg/kg Strontium 2/2 9.39E+01 8.0E+02 No mg/kg Vanadium 2/2 6.50E+01 5.6E-01 Yes mg/kg Zinc 2/2 5.57E+01 4.0E+02 No mg/kg 1/2 Toluene 1.00E-02 9.8E+01 No mg/kg Alpha activity 2/2 1.86E+01 pCi/g Beta activity 2/2 2.29E+01 pCi/g

-- LOCATION=SWMU 193C MEDIA=McNairy Groundwater ------

		Frequency	Maximum detected			Exceed	Exceed	
	Analyte	Detection	concentration	. HI	ELCR	HI?	ELCR?	Units
	Aluminum	4/4	9.04Ē+01	1.5E+00		Yes		mg/L
•	Antimony	5/5	2.50E-01	5.6E-04		Yes		mg/L
	Arsenic	5/5	3.60E-02	4.5E-04	3.5E-06	Yes	Yes	mg/L
	Barium	5/5	6.70E-01	1.0E-01		Yes		mg/L
	Beryllium	5/5	2.50E-02	2.6E-03	1.0E-06	Yes	Yes	mg/L
	Cadmium	5/5	1.00E-01	6.6E-04		Yes		mg/L
	Calcium	5/5	4.10E+01					mg/L
	Chloride	5/5	1.68E+01					mg/L
	Chromium	3/3	2.328-01	4.2E-03		Yes		mg/L
	Cobalt	5/5	1.21E-01	9.1E-02		Yes		mg/L
	Copper	5/5	1.63E-01	6.0E-02		Yes		mg/L
	Fluoride	4/4	2.80E-01	9.1E-02		Yes		mg/L
	Iron	5/5	1.79E+02	4.5E-01		Yes		mg/L

	LOCATION=	SWMU 193C MEDIA=M (continued)		iwater ,			
	Frequency	Maximum					
	of	detected			Exceed	Exceed	
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
Lead	1/1	2.50E-01	1.5E-07		Yes		mg/L
Magnesium	5/5	2.16E+01					mg/L
Manganese	5/5	3.91E+00	6.7E-02		Yes		mg/L
Mercury	1/1	2.00E-04	4.4E-04		No		mg/L
Molybdenum	4/4	1.00E-01	7.5E-03		Yes		mg/L
Nickel	5/5	1.09E-01	3.0E-02		Yes		mg/L
Nitrate as Nitrogen	5/5	1.00E+00	2.4E+00		No		mg/L
Potassium	5/5	1.01E+02					mg/L
Selenium	3/3	5.00E-03	7.5E-03		No		mg/L
Silica	5/5	1.80E+01					mg/L
Silver	3/3	6.00E-02	7.5E-03		Yes		mg/L
Sodium	5/5	2.63E+01					mg/L
Tetraoxo-sulfate(1-)	5/5	1.30E+01					mg/L
Thallium	2/2	1.23E-01					mg/L
Uranium	9/9	1.80E-02	4.5E-03		Yes		mg/L
Vanadium	2/2	8.36E-01	9.3E-03		Yes	•	mg/L
Zinc	5/5	5.64E-01	4.5E-01		Yes		mg/L
1,1,1-Trichloroethane	4/4	5.00E-03	5.4E-02		No		mg/L
1,1,2-Trichloroethane	4/4	5.00B-03	8.1E-04	1.88-05	Yes	Yes	mg/L
1,1-Dichloroethane	4/4	5.00E-03	2.7E-02		No		mg/L
1,1-Dichloroethene	4/4	5.00E-03	1.8E-03	9.3E-07	Yes	Yes	mg/L
1,2-Dichloroethane	4/4	5.00E-03	6.7E-04	1.1E-05	Yes	Yes	mg/L
Benzene	4/4	5.00E-03	4.0E-04	3.5E-05	Yes	Yes	mg/L
Bromodichloromethane	4/4	5.00E-03	4.0E-03	8.4E-05	Yes	Yes	mg/L
Carbon Tetrachloride	4/4	5.00E-03	1.2E-04	1.5E-05	Yes	Yes	mg/L
Chloroform	4/4	5.00E-03	2.0E-03	1.5E-05	Yes	Yes	mg/L
Ethylbenzene	4/4	5.00E-03	4.5E-02		No		mg/L
Polychlorinated biphenyl	1/1	1.00E-04		8.0E-06		Yes	mg/L
Tetrachloroethene	4/4	5.00E-03	7.9E-03	5.7E-05	No	Yes	mg/L
Toluene	4/4	5.008-03	2.4E-02		No		mg/L
Trichloroethene	12/12	2.00E-03	1.2E-03	1.4E-04	Yes	Yes	mg/L
Vinyl Chloride	4/4	1.00E-02		1.7E-06		Yes	mg/L
Xylene	4/4	1.00E-02	4.0E-01	1./1 00	No	100	mg/L
cis-1,2-Dichloroethene	4/4	5.00E-03	2.0E-03		Yes		mg/L
trans-1, 2-Dichloroethene	4/4	5.00E-03	4.0E-03		Yes		mg/L
Alpha activity	12/12	1.07E+02					pCi/L
Beta activity	12/12	2.36E+02					pCi/L
Radon-222	2/2	1.57E+02		1.4E+00		Yes	pCi/L pCi/L
Technetium-99	13/13	2.70E+01		2.8E+01		No	pCi/L pCi/L
rechnetrum-33	13/13	4./USTU1		2.05+01		TNO	PCI/L

Analyte	Frequency of Detection	Maximum detected concentration	HI	ĒĹCR	Exceed HI?	Exceed ELCR?	Units
1,2-Dichloroethene Trichloroethene	1/2 1/2	5.628-01 1.628-01	1.8E-03 1.2E-03	1.4E-04	Yes Yes	Yes	mg/L mg/L

LOCATION-SWMU 193C MEDIA-RGA Groundwater ------

--- LOCATION-SWMU 193C MEDIA-Subsurface Soil --

	Frequency	Maximm detected		•	Exceed	Exceed	
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
Aluminum	20/20	1.37E+04	7.3E+02		Yes		mg/kg
Arsenic	5/20	6.57E+00	6.9E-01	9.2E-03	Yes	Yes	mg/kg
Barium	1/1	1.42E+02	3.7E+01		Yes		mg/kg
Beryllium	10/20	9.80E-01	1.6E-01	1,0E-04	Yes	Yes	mg/kg
Boron	1/20	1.00E+02	3.3E+02		No		mg/kg
Cadmium	3/59	5.00E+00	3.8E-01	3.3E+03	Yes	No	mg/kg
Calcium	18/20	4.00E+05					mg/kg
Chromium	59/61	8,30E+01	4.8E-01	4.9E+02	Yes	No	mg/kg
Cobalt	17/20	8.61E+01	2.1E+02		No		mg/kg
Copper	16/20	2.822+01	7.4E+01	•	No		mg/kg
Iron	6/6	3.00E+04	3.1E+02		Yes	•	mg/kg
Lead	42/61	6.77E+01	1.0E-04		Yes		mg/kg
Lithium	17/20	1.25E+01	7.0E+01		No		mg/kg
Magnesium	20/20	1.45E+04					mg/kg
Manganese	20/20	2.27E+03	1.4E+01		Yes		mg/kg
Nickel	13/20	2.15E+01	3.4Ê+01		No		mg/kg
Potassium	20/20	1.57E+03					mg/kg
Sodium	15/20	4.44E+02					mg/kg
Strontium	20/20	3.91E+02	8.0E+02		No		mg/kg
Vanadium	20/20	4.42E+01	5.6Ë-01		Yes		mg/kg
Zinc	19/20	9.25E+01	4.0E+02		No		mg/kg
Xylene	1/20	1.00E-02	1.7E+03	· ·	No		mg/kg
Alpha activity	53/53	4.00E+00					pCi/g
Beta activity	53/53	1.00E+01					pCi/g

	LOCATI	ON-SWMU 193C MEDI	A=Surface So	11				
	Frequency	Maximum						
	of	detected			Exceed	Exceed		
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units	
Aluminum	5/5	3.36E+03	7.3E+02		Yes		mg/kg	
Boron	1/5	1.00E+02	3.3E+02		No		mg/kg	
Calcium	5/5	4.00E+05					mg/kg	
Chromium	3/5	1.202+01	4.8E-01	4.9E+02	Yes	No	mg/kg	
Cobalt	2/5	2.14E+00	2.1E+02		No		mg/kg	
Copper	2/5	2.82E+01	7.4E+01		No		mg/kg	
Lead	1/5	6.77E+01	1.0E-04		Yes		mg/kg	
Lithium	3/5	1.25B+01	7.0E+01	•.	No		mg/kg	
Magnesium	5/5	1.45E+04			10	•	mg/kg	
Manganese	5/5	1.988+02	1.4E+01		Yes		mg/kg	
Nickel	1/5	6.438+00	3.4E+01		NO			
Potassium	5/5	1.57E+03	3.45701		NO		mg/kg	
Sodium	4/5	3.10E+02					mg/kg	
Strontium	5/5	3.918+02	8.0E+02		No		mg/kg	
Vanadium	5/5	6.70E+00	5.6E-01		Yes		mg/kg	
Zinč	5/5	9.25E+01					mg/kg	
21nc	3/3	9.456+01	4.0E+02		No		mg/kg	
	LOCATIO	N-SWMU 194 MEDIA-	Subsurface So	óil				
2	Frequency	Maximum						
•	õ£	detected			Exceed	Exceed		
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units	
Aluminum	12/12	1.45E+04	7.3E+02		Yes		mg/kg	
Arsenic	1/12	6.73E+00	6.9E-01	9.28-03	Yes	Yes	mg/kg	
Barium	12/12	1.39E+02	3.7E+01		Yes		mg/kg	
Beryllium	6/12	4.805+00	1.6E-01	1.0E-04	Yes	Yes	mg/kg	
Cadmium	1/35	8.552+00	3.8E-01	3.3E+03	Yes	No	mg/kg	
2 . .								

4.85-01

2.1E+02

7.4E+01

3.1E+02

1.0E-04

7.0E+01

1.4E+01

3.4E+01

4.9E+02

Yes

No

No

Yes

Yes

Yes

No

No

No ·

mg/kg

mg/kg

mg/kg

mg/kg mg/kg

mg/kg

mg/kg

mg/kg mg/kg

mg/kg

mg/kg

6.81E+03

1.03E+02

9.46E+00

1.67E+01

2.00E+04

3.60E+02

9.00E+00

2.34E+03

4.678+02

1.37E+01

6.32E+02

12/12

35/35

12/12

12/12

12/12

20/35

12/12

12/12

12/12

12/12

8/12

Table 1.5. Comparison of maximum detected concentrations and activities to human health risk-based screening criteria by location and medium

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Calcium

Cobalt

Copper

Lithium

Nickel

Magnesium

Manganese

Potassium

Iron

Lead

Chromium

		(continued)					
	Frequency	Maximum		•			
	of	detected	•		Exceed	Exceed	
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
	- <u>,</u>						
Sodium	8/12	3.69E+02					mg/kg
Strontium	12/12	2.60E+01	8,0E+02		No		mg/kg
Vanadium	12/12	2.58E+01	5.6E-01		Yes		mg/kg
Zinc	11/12	6.76E+01	4.0E+02		No		mg/kg
Ethylbenzene	1/19	1,50E-02	1.1E+02		No		mg/kg
Alpha activity	23/23	2.50E+00					pCi/g
Beta activity	23/23	7.00E+00					pCi/g
	LOCATION=	SWMU 99A MEDIA-MCI	Nairy Ground	water			
	Frequency	Maximum					
	of	' detected			Exceed	Exceed	
Analyte	Detection	concentration	HI	ELCR	HI7	ELCR?	Units
Analyce	Detection	CONCENTRATION		· Elica	111 I	<u>EUCK</u>	UNICS
1,1,1=Trichloroethane	1/4	1.20E-03	5.4E-02		No		mg/L
1,1-Dichloroethene	1/4	2.29E-02	1.8E-03	9.3E-07	Yes	Yes	mg/L
Carbon Tetrachloride	1/4	2.80E-03	1.2E-04	1.5E-05	Yes	Yes	mg/L
Trichloroethene	3/4	5.19E-01	1.2E-03	1.4E-04	Yes	Yes	mg/L
cis-1,2-Dichloroethene	2/4	1.15E-01	2.0E-03		Yes		mg/L
Alpha activity	2/2	2.90E+00					pCi/L
Beta activity	2/2	3.50E+01					pCi/L
Technetium-99	2/2	1.90E+01		2.8E+01		No	pCi/L
Termerium-32	2 /2	1.904401		2.06+01		NO	per/L
	LOCATIC	N-SWMU 99A MEDIA-	RGA Groundwa	ter			
	Frequency	Maximum					
	of	detected			Exceed	Exceed	
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
Analyte	Delection	concentration .	μī	ELCK	at:	ELLCR. F	UNICS
Aluminum	16/35	6.59E+02	1.5E+00		Yes		mg/L
Arsenic	4/27	1.00E-02	4.58-04	3.5E-06	Yes	Yes	mg/L
Barium	39/39	3.30E+00	1.0E-01	•	Yes		mg/L
Beryllium	8/35	1.00E-01	2.6E-03	1.0E-06	Yes	Yes	mg/L
Calcium	39/39	1.20E+02					mg/L
	9/9	1.205+02					mg/L
Chromium	11/39	1.78E+00	4.2E-03		Yes		mg/L
Cobalt	20/37	5.70E-01	9.1E-02		Yes		mg/L mg/L
LODALE	20/37	5.705=01	9.16-02		Yes		mm / T.

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	Frequency of	Maximum detected			Exceed	Exceed	
nalyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
Copper	9/35	6.40E-01	6.0E-02		Yes		mg/L
luoride	8/8	2.00E-01	9.1E-02		Yes		mg/L
Iron	31/39	1.20E+03	4.5E-01		Yes		mg/L
ead	6/29	4.10E-01	1.5E-07		Yes		mg/L
Lithium	6/23	1.708-01	3.0E-02		Yes		mg/L
lagnesium	39/39	4.97E+01					mg/L
langanese	37/39	4.60E+00	6.7E-02		Yes		mg/L
fercury	5/25	2.005-02	4.4E-04		Yes		mg/L
Nickel	16/39	9.10E-01	3.0E-02		Yes		mg/L
Nitrate as Nitrogen	7/9	2.10E+00	2.4E+00		No		mg/L
Potassium	24/39	2.17E+01					mg/L
Bilica	9/9	2.50B+01					mg/L
Jodium	39/39	7.24E+01					mg/L
Strontium	30/30	4.70E-01	9.0E-01		No		mg/L
Sulfate	2/2	1.92E+01	•				mg/L
Tetraoxo-sulfate(1-)	7/7	2.20E+01					mg/L
Vanadium	10/28	2.15E+00	9.3E-03		Yes		mg/L
Zinc	10/35	2.55E+00	4.5E-01		Yes		mg/L
1,1-Dichloroethene	7/33	6.50E-02	1.8E-03	9.3B-07	Yes	Yes	mg/L
Trichloroethene	41/43	2.37E+00	1.2E-03	1.4B-04	Yes	Yes	mg/L
bis(2-Ethylhexyl)phthalate	5/10	1.60E-02	2.6E-02	3.1E-04	No	Yes	mg/L
cis-1,2-Dichloroethene	10/33	3.48E-02	2.0E-03		Yes		mg/L
trans-1,2-Dichloroethene	3/33	6.00E-04	4.0E-03		No		mg/L
Alpha activity	33/39	5.38E+01	•		-		pCi/L
Beta activity	39/39	1.37E+02			•		pCi/L
Radon-222	4/4	6.75E+02		1.4E+00		Yes	pCi/L
Technetium-99	34/40	1.39E+02	•	2.8E+01		Yes	pCi/L

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Analyte	Prequency of Detection	Maximum detected concentration	HI	ELCR	Exceed HI?	Exceed ELCR?	Units
Aluminum	22/22	1.41E+04	7.3E+02		Yes		mg/kg
Antimony	5/22	2.90E+00	6.4B-02		Yes		mg/kg
Arsenic	11/22	8.55E+00	6.9B-01	9.2E-03	Yes	Yes	mg/kg
Barium	22/22	2.47E+03	3.7E+01		Yes		mg/kg

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----- LOCATION-SWMU 99A MEDIA-Subsurface Soil -----

	Bochiat	(continued					
	Frequency	Maximum					
_	of	detected			Exceed	Exceed	
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
Beryllium	11/22	8.90E-01	1.6E-01	1.0E-04	Yes	Yes	mg/kg
Cadmium	5/22	8.308-01	3.8E-01	3.3E+03	Yes	No	mg/kg
Calcium	20/20	2.87E+05					mg/kg
Chromium	22/22	4.57E+01	4.8E-01	4.9E+02	Yes	No	mg/kg
Cobalt	20/22	1.192+01	2.1E+02		No		mg/kg
Copper	21/22	1.64E+01	7.4E+01		No		mg/kg
Cyanide	2/16	5.408-01	2.3E+01		No		mg/kg
Iron	22/22	2.33E+04	3.1E+02		Yes		mg/kg
Lead	6/22	4.73E+01	1.0E-04		Yes		mg/kg
Lithium	17/17	1.29E+01	7.0E+01		No		mg/kg
Magnesium	22/22	2.73E+04	•				mg/kg
Manganese	22/22	1.46E+03	1.4E+01		Yes		mg/kg
Mercury	5/22	1.20E-01	1.4E-01		No		mg/kg
Nickel	17/22	2.58E+01	3.4E+01		No		mg/kg
Potassium	22/22	1.12E+03					mg/kg
Selenium	· 5/20	3.20E-01	1.2E+01		No		mg/kg
Silver	5/22	7.10E-01	6.1E+00		No		mg/kg
Sodium	14/22	3.93E+02					mg/kg
Strontium	17/17	5.14E+02	8.0E+02		No		mg/kg
Thallium	5/22	5.90B-01			•		mg/kg
Vanadium	22/22	3.55B+01	5.6B-01		Yes		mg/kg
Zinc	21/22	1.63E+02	4.0E+02		No		mg/kg
1,1,1-Trichloroethane	5/8	6.00B-03	1.2E+02		No	•	mg/kg
1,1,2,2-Tetrachloroethane	5/8	6.00B-03		1.8E-02		No	mg/kg
1,1,2-Trichloroethane	5/8	6.00B-03	3.1E+00	4.6E-02	No	No	mg/kg
1,1-Dichloroethane	5/8	6.00B-03	6.7E+01		No		mg/kg
1,1-Dichloroethene	5/10	6.00E-03	3.5E+00	3.9B-03	No	Yes	mg/kg
1,2,4-Trichlorobenzene	5/22	4.10E-01	2.5E+01		No		mg/kg
1,2-Dichlorobenzene	5/22	4.10E-01	7.6E+01		No		mg/kg
1,2-Dichloroethane	5/8	6.00E-03	4.3E+00	2.2E-02	No	No	mg/kg
1,2-Dichloroethene	5/5	6.00E-03	1.0E+01		No		mg/kg
1,2-Dichloropropane	5/8	6.00E-03	1.6E+00	8.7E-02	No	No	mg/kg
1,3-Dichlorobenzene	5/22	4.10E-01	3.3E+01		No		mg/kg
1,4-Dichlorobenzene	5/22	4.108-01	1.1E+03	2.9E-01	No	Yes	mg/kg
2,4,5-Trichlorophenol	5/22	2.10E+00	1.6E+02		No		mg/kg
2,4,6-Trichlorophenol	5/22	4.10E-01		8.1E-01		No	mg/kg
2,4-Dichlorophenol	5/22	4.10E-01	6.8E+00	J	No	10	mg/kg
2,4-Dimethylphenol	5/22	4.10E-01	3.1E+01		No		mg/kg
2,4-Dinitrophenol	5/6	2.10E+00	5.0E+00		No		mg/kg
-/·	0,0	2.104700	3.04+00		TIC .		mg/kg

		(continued))	•		-	
Ànalyte	Frequency of Detection	Maximum detected concentration	HI	ELCR	Exceed HI?	Exceed ELCR?	UNITS
Anaryce	Decection	CONCENTIALION	пі	EIKK	ni r	ELCRI	UNILS
2,4-Dinitrotoluene	5/22	4.10E-01	4.7E+00	2.1E-02	NO	Yes	mg/kg
2,6-Dinitrotoluene	5/22	4.10E-01	2.3E+00	2.1E-02	No	Yes	mg/kg
2-Butanone	5/8	1.202-02	3.9E+02		No		mg/kg
2-Chloronaphthalene	5/22	4.108-01	1.1E+02		No		mg/kg
2-Chlorophenol	5/22	4.10E-01	7.0E+00		No		mg/kg
2-Hexanone	5/8	1.20E-02					mg/kg
2-Methyl-4,6-dinitrophenol	5/22	2.10E+00					mg/kg
2-Methylnaphthalene	5/22	4.10B-01					mg/kg
2-Methylphenol	5/22	4.10E-01	7.8E+01		No		mg/kg
2-Nitroaniline	5/22	2.10E+00	7.0E-02		Yes		mg/kg
2-Nitrophenol	5/22	4.10E-01					mg/kg
3,3'-Dichlorobenzidine	5/22	8.208-01	,	2.1E-02		Yes	mg/kg
3-Nitroaniline	5/22	2.10E+00					mg/kg
4,4'-DDD	2/2	3.50E-02		5.1E-02		No	mg/kg
4,4'-DDE	2/2	3.50E-02		3.6E-02		No	mg/kg
4, 4' - DDT	2/2	3.50E-02	1.0E+00	3.6E-02	No	No	mg/kg
4-Bromophenyl phenyl ether	5/22	4.108-01				•	mg/kg
4-Chloro-3-methylphenol	5/22	4.10E-01			· ·		mg/kg
4-Chloroaniline	5/22	4.10E-01	6.3E+00	•	No		mg/kg
4-Chlorophenyl phenyl ether	5/22	4.10E-01					mg/kg
4-Methyl-2-pentanone	5/8	1.20E-02	3.3E+01		No		mg/kg
4-Methylphenol	5/22	4.10E-01	9.6E+00		No		mg/kg
4-Nitroaniline	5/22	2.10E+00			•		mg/kg
4-Nitrophenol	5/22	2.10E+00	1.6E+02		No		mg/kg
Acenaphthene	7/22	4.10E-01	6.4E+01		No		mg/kg
Acenaphthylene	6/22	6.10B-01					mg/kg
Acetone -	5/8	5.30E-02	9.2E+01		No		mg/kg
Aldrin	2/2	1.70E-02	4.8E-02	5.5E-04	No	Yes	mg/kg
Anthracene	7/22	7.50E-01	6.5E+02		No		mg/kg
Benz (a) anthracene	8/22	1.70E+00		8.5E-03		Yes	mg/kg
Benzene	5/8	6.00E-03	1.8E+00	5.1B-02	No	No	mg/kg
Benzo (a) pyrene	7/22	2.10E+00		8.5E-04		Yes	mg/kg
Benzo (b) fluoranthene	11/22	5.708+00		8.5E-03		Yes	mg/kg
Benzo (ghi) perylene	7/22	1.18E+00					mg/kg
Benzo(k)fluoranthene	B/22	7.90E-01		8.5E-02		Yes	mg/kg
Benzoic Acid	5/5	2.10E+00	9.8E+03		NO		mg/kg
Benzyl Alcohol	5/5	4.10E-01	5.8E+02		No		mg/kg
Bromodichloromethane	5/8	6.00E-03	1.9E+01	1.2E-01	No	No	mg/kg
Bromoform	5/8	6.00E-03	1.6E+01	5.4E-01	No	No	
	-, -	0.000 00	TIONLAT		110	AU	mg/kg

LOCATION-SWMU 99A MEDIA-Subsurface Soil

Table 1.5. Comparison	of	maximum detected	concentrations and	activities	to human heal	th risk-based
		screening cr	iteria by location	and medium		

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	•	(continued)	•				
	Frequency	Maximum					
	of	detected			Exceed	Exceed	
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
Bromomethane	5/8	1.20E-02	6.2E-01		No		mg/kg
Butyl benzyl phthalate	5/6	4.10E-01	3.75+02		NO		mg/kg
Carbon Disulfide	5/8	6.00E-03	4.6E+01		No		mg/kg
Carbon Tetrachloride	5/8	6.00E-03	2.7E-01	1.6E-02	No	No	mg/kg
Chlorobenzene	5/8	6.00E-03	5.6E+00		No		mg/kg
Chloroethane	5/8	1.208-02	2.8E+02		No		mg/kg
Chloroform	5/8	6.00E-03	2.4B+00	2.1E-02	No	No	mg/kg
Chloromethane	5/B	1.20E-02		1.3E-01		No	mg/kg
Chrysene	7/22	2.10E+00		8.5E-01		Yes	mg/kg
Di-n-butylphthalate	5/22	4.10E-01	2.6E+02		NO	•	mg/kg
Di-n-octylphthalate	5/22	4.10E-01	4.9E+01		No		mg/kg
Dibenz (a, h) anthracene	6/22	4.80E-01		8.5E-04		Yes	mg/kg
Dibenzofuran	6/22	4.10E-01	6.3E+00		No	•	mg/kg
Dibromochloromethane	5/8	6.00E-03	1.5E+01	5.9E-02	No	No	mg/kg
Dieldrin	2/2	3.50E-02	8.0E-02	5.8E-04	No	Yes	mg/kg
Diethylphthalate	5/22	4.10E-01	2.0E+03		No		mg/kg
Dimethylphthalate	5/22	4.10E-01	2.4E+04		No		mg/kg
Endosulfan I	2/2	1.70E-02	•				mg/kg
Endosulfan II	2/2	3.50E-02					mg/kc
Endosulfan Sulfate	2/2	3.50E-02					mg/kc
Endrin	2/2	3.50E-02	2.4E-02		Yes		mg/kg
Endrin Ketone	2/2	3.50E-02					mg/kg
Ethylbenzene	5/8	6.00E-03	1.1E+02		No		mg/kg
Fluoranthene	9/22	2.66E+00	4.3E+01		NO		mg/kg
Fluorene	6/22	4.105-01	6.3E+01		No		mg/kg
Heptachlor	2/2	1.70E-02	1.1E+00	2.8E-03	No	Yes	mg/kg
Heptachlor Epoxide	2/2	1.70E-02	2.7E-02	1.4E-03	No	Yes	mg/kg
Hexachlorobenzene	5/22	4.10E-01	1.3E+00	5.4E-03	No	Yes	mg/kg
Hexachlorobutadiene	5/22	4.10E-01	3.0E-01	1.0E-01	Yes	Yes	mg/kg
Hexachlorocyclopentadiene	5/22	4.10E÷01	1.0E+00	1102 01	No		mg/kg
Hexachloroethane	5/22	4.10E-01	1.5E+00	5.8E-01	No	No	mg/kg
Indeno (1,2,3-cd) pyrene	7/22	1.05E+00	1.02+00	8.5E-03	ЦU	Yes	mg/kg
Isophorone	5/22	4.10E-01	3.0E+02	9.9Ē+00	No	No	mg/kg
Methoxychlor	2/2	1.70E-01	8.0E+02	3.35400	Ňo	NO	
Methylene Chloride	5/8	1.70E-01 8.00E-03	7.0E+01	5.0E-01	NO	No	mg/kg
	5/22	4.10E-01	/.05+01	7.3E-04	NO		mg/kg
N-Nitroso-di-n-propylämine						Yes	mg/kĝ
N-Nitrosodiphenylamine	5/22	4.105-01		1.0E+00		No	mg/kg
Naphthalene	5/22	4.10E-01	1.3E+01		No		mg/kg
Nitrobenzene	5/22	4.10E-01	6.1E-01		No		mg/kg

	LOCATIC	N=SWMU 99A MEDIA=8 (continued)		oil			
	Frequency	Maximum	•				
	of	detected			Exceed	Exceed	
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
PCB-1016	3/23	1.87E+00	2.3E-01	9.9E-03	Yes	Yes	mg/kg
PCB-1221	2/23	1.70E-01		1.1E-02		Yes	mg/kg
PCB-1232	2/23	1.70E-01		1.1E-02		Yes	mg/kg
PCB-1242	2/23	1.70E-01		9.7E-03		Yes	mg/kg
PCB-1248	2/23	1.70B-01		1.1B-02		Yes	mg/kg
PCB-1254	× 3/23	3.50E-01	6.6E-02	9.9E-03	Yes	Yes	mg/kg
PCB-1260	7/23	6.31E-01		9.8E-03		Yes	mg/kg
Pentachlorophenol	5/22	2.10E+00	7.9E+01	1.3E-01	NO	Yes	mg/kg
Phenanthrene	7/22	1.63E+00					mg/kg
Phenol	5/22	4.10E-01	1.4E+03		No		mg/kg
Pyrene	B/22	2.70E+00	3.2E+01		No		mg/kg
Styrene	5/8	6.00E-03	1.9E+02		No		mg/kg
Tetrachloroethene	5/8	6.00E-03	1.2E+01	1.3B-01	NO	No	mg/kg
Toluene	5/8	6.00E-03	9.8E+01		No		mg/kg
Toxaphene	2/2	3.50E-01	•	8.5E-03		Yes	mg/kg
Trichloroethene	6/10	6.00E-03	1.2E+00	9.1E-02	No	No	mg/kg
Vinyl Acetate	5/5	1.20E-02	9.5E+01	•	No		mg/kg
Vinyl Chloride	5/10	1.20E-02		1.5E-03		Yes	mg/kg
Xylene	5/5	6.00E-03	1.7E+03		No	•	mg/kg
alpha-BHC	2/2	1.70E-02		2.4E-03		Yes	mg/kg
alpha-Chlordane	2/2	1.70E-01					mg/kg
beta-BHC	2/2	1.70E-02		8.2E-03		Yes	mg/kg
bis (2-Chloroethoxy) methane	5/22	4.10E-01					mg/kg
bis(2-Chloroethyl)ether	5/22	4.10B-01		5.9E-03		Yes	mg/kg
bis (2-Chloroisopropyl) ether	5/22	4.10E-01		1.1E-01		Yes	mg/kg
bis (2-Ethylhexyl) phthalate	5/22	3.60E-01	1.4E+01	2.8E-01	No	Yes	mg/kg
cis-1,3-Dichloropropene	5/8	6.00B-03					mg/kg
delta-BHC	2/2	1.70E-02		•			mg/kg
gamma-BHC (Lindane)	2/2	1.70E-02	7.75-01	1.2E-02	No	Yes	mg/kg
gamma-Chlordane	2/2	1.70E-01				100	mg/kg
trans-1,3-Dichloropropene	5/8	6.00E-03					mg/kg
Alpha activity	20/21	1.42E+02					pCi/g
Beta activity	21/21	2.73E+03					
Cesium-137	3/21	1.90E+00		1.6E-02		Yes	pCi/g
Neptunium=237	4/4	1.28E+01		6.8E-02		Yes	pCi/g
							pCi/g
Plutonium-239	3/3	6.00E-03		2.0E+00		No	pCi/g
Technetium-99	6/23	2.65E+03		4.4E+02	•	Yes	pCi/g
Thorium-230	3/3	6.70E-01		1.6E+01		NO	pCi/g
Thorium-234	1/21	5.30E+01		7.2E+00		Yes	pCi/g

	LOCATIC	.1				́		
Analyte	Frequency of Detection	Maximum detected concentration	ĤI	ELCR	Exceed HI?	Exceed ELCR?	Units	
Uranium-234 Uranium-235 Uranium-238	4/4 3/24 4/4	1.64E+01 4.10E-02 5.17E+01		1.4E+01 1.2E-01 4.7E-01		Yes No Yes	pCi/g pCi/g pCi/g	

---- LOCATION-SWMU 99A MEDIA=Surface soil -----

Analyte	Frequency of Detection	Maximum detected concentration	HI	ELCR	Exceed HI?	Exceed ELCR?	Units
-							
Aluminum	13/13	1.29E+04	7.3E+02		Yes		mg/kg
Arsenic	6/13	8.558+00	6.9E-01	9.2E-03	Yes	Yes	mg/kg
Barium	13/13	2.47E+03	3.7E+01		Yes		mg/kg
Beryllium	5/13	8.90E-01	1.6E-01	1.0E-04	Yes	Yes	mg/kg
Calcium	11/11	2.87E+05					mg/kg
Chromium	13/13	4.57E+01	4.8E-01	4.9E+02	Yes	No	mg/kg
Cobalt	11/13	9.67E+00	2.1E+02		No		mg/kg
Copper	12/13	1.22E+01	7.4E+01		No		mg/kg
Iron	13/13	2.33E+04	3.1E+02		Yes		mg/kg
Lithium	13/13	1.29E+01	7.0 <u>E</u> +01		No		mg/kg
Magnesium	13/13	2.73E+04					mg/kg
Manganese	13/13	3.875+02	1.4E+01		Yes		mg/kg
Nickel	8/13	2.16E+01	3.4E+01		No		mg/kg
Potassium	. 13/13	1.12E+03	•		•		mg/kg
Sodium	6/13	3.66E+02					mg/kg
Strontium	13/13	5.14E+02	8.0E+02		No		mg/kg
Vanadium	13/13	3.55E+01	5.6E-01		Yes		mg/kg
Zinc	12/13	1.63E+02	4.0E+02		No .		mg/kg
Acenaphthene	2/13	3.30E-01	6.4E+01		No		mg/kg
Acenaphthylene	1/13	6.10E-01					mg/kg
Anthracene	2/13	7.502-01	6.5E+02		No	•	mg/kg
Benz (a) anthracene	3/13	1.70E+00	•	8.5E-03		Yes	mg/kg
Benzo (a) pyrene	2/13	2.10E+00		8.5E-04		Ŷes	mg/kg
Benzo (b) fluoranthene	6/13	5.70E+00		8.5E-03		Yes	mg/kg
Benzo (ghi) perylene	2/13	1.18E+00					mg/kg
Benzo (k) fluoranthene	3/13	7.90E-01		8.5E-02		Yes	mg/kg
Chrysene	2/13	2.10E+00		8.5E-01		Yes	mg/kg
Dibenz (a, h) anthracene	1/13	4.80E-01		8.5E-04		Yes	mg/kg

Table 1.5.	Comparison	of ma	uximum d	letected	concentra	tions ar	id act	tivities	to	human	health	risk-base	đ
	_		BCTE	ening ci	citeria by	locatio	n and	d medium					

LOCATION-SWMU 99A MEDIA-Surface Soil ------(continued)

	Frequency of	Maximum detected			Exceed	Exceed	
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
Dibenzofuran	1/13	1.23B-01	6.3B+00		No		mg/kg
Fluoranthene	4/13	2.66E+00	4.3E+01		No		mg/kg
Fluorene	1/13	2.19E-01	6.3E+01		No		mg/kg
Indeno (1, 2, 3-cd) pyrene	2/13	1.05B+00		8.5E-03	•	Yes	mg/kg
PCB-1016	1/16	1.87E+00	2.3E-01	9.9E-03	Yes	Yes	mg/kg
PCB-1254	1/16	9.60B-02	6.6E-02	9.9E-03	Yes	Yes	mg/kg
PCB-1260	5/16	6.31E-01		9.8E-03		Yes	mg/kg
Phenanthrene	2/13 ·	1.63E+00					mg/kg
Pyrene	3/13	2.708+00	3.2E+01		No		mg/kg
Alpha activity	15/16	1.422+02					pCi/g
Beta activity	16/16	2.73E+03					pCi/g
Cesium-137	3/16	1.90E+00		1.6E-02		Yes	pCi/g
Neptunium-237	1/1	1.28E+01		6.8E-02		Yes	pCi/g
Technetium-99	3/16	2.65E+03	•	4.4E+02		Yes	pCi/g
Thorium-234	1/16	5.30E+01	•	7.2E+00		Yes	pCi/g
Uranium-234	1/1	1.64B+01		1.4E+01		Yes	pCi/g
Uranium-238	1/1	5.17E+01		4.7E-01	•	Yes	pCi/g

-- LOCATION=SWMU 998 MEDIA=RGA Groundwater --

	Frequency • of	Maximum detected			Exceed	Exceed	
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
Barium	7/7	2.70E+00	1.0E-01		Yes		mg/L
Calcium	7/7	3.27E+01	•				mg/L
Chloride	7/7	1.08E+02					mg/L
Chromium	1/7	2.60E-01	4.2E-03		Yes		mg/L
Copper	1/7	4.00E-02	6.0E-02		No		mg/L
Fluoride	7/7	2.10B-01	9.1E-02		Yes		mg/L
Iron	. 3/7	3.34E+00	4.5E-01		Yes		mg/L
Magnesium	7/7	1.31E+01					mg/L
Manganese	5/7	2.90E-01	6.7E-02		Yes		mg/L
Nitrate as Nitrogen	7/7	2.10E+00	2.4E+00		No		mg/L
Silica	7/7	2.00E+01					mg/L
Sodium	7/7	7.86E+01					mg/L
Sulfate	2/2	2.67E+01			•		mg/L
Tetraoxo-sulfate(1-)	5/5	2.90E+01					mg/L

· · · · · · · · · · · · · · · · · · ·
ed Exceed ELCR? Units
mg/L
Yes mg/L
pCi/L
pCi/L
Yes pCi/L
NO pCi/L
- · ·

----- LOCATION-SWMU 99B MEDIA-Subsurface Soil ------

	Frequency	Maximum detected			Exceed	Exceed	·
Analyte	Detection	concentration	HI	ELCR	HI?	ELCR?	Units
Aluminum	8/8	1.70E+04	7.3E+02		Yes		mg/kg
Arsenic	2/6	8.05E+00	6.9E-01	9.2E-03	Yes	Yes	mg/kg
Barium	8/8	1.55E+02	3.7E+01		Yes		mg/kg
Beryllium	6/8	1.00E+00	1.6E-01	1.0E-04	Yes	Yes	mg/kg
Calcium	6/6	7.17E+03					mg/kg
Chromium	8/8	2.61B+01	4.8E-01	4.9E+02	Yes	No	mg/kg
Cobalt	8/8	6.94E+00	2.1E+02		No	•	mg/kg
Copper	8/8	1.30E+01	7.4B+01		No		mg/kg
Iron	8/8	1.81E+04	3.1E+02		Yes		mg/kg
Lithium	8/8	1.14E+01	7.0E+01		No		mg/kg
Magnesium	8/8	2.53E+03					mg/kg
Manganese	8/8	5.24E+02	1.4E+01		Yes		mg/kg
Nickel	5/8	2.51E+01	3.4E+01		No		mg/kg
Potassium	8/8	1.04E+03	•				mg/kg
Sodium	3/8	3.09E+02					mg/kg
Strontium	8/8	2.22E+01	8.0E+02		No		mg/kg
Vanadium	8/8	3.44E+01	5.6E-01		Yes		mg/kg
Zinc	8/8	5.222+01	4.0E+02		No		mg/kg
Acetone	1/7	5.50E-01	9.2E+01		No		mg/kg
Methylene Chloride	3/7	1.20E+00	7.0E+01	5.0E-01	No	Yes	mg/kg
Alpha activity	8/8	2.14E+01					pCi/g
Beta activity	8/8	2.26E+01					pCi/g

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------ LOCATION=AOC 204 MEDIA=RGA Groundwater ------

Frequency Maximum detected of Background Exceed Analyte concentration Detection concentration Background? Units 1,1,1-Trichloroethane 11/11 1.80E-02 mg/L 1,1-Dichloroethane 5.00E+00 1/1 mg/L 1,1-Dichloroethene 12/15 4.00E-02 mg/L 11/11 2.50E-02 PCB-1254 mg/L PCB-1260 11/11 2,50E-02 mg/L Polychlorinated biphenyl 1/1 1.70E-01 mg/L Tetrachloroethene 11/11 5.00E+00 mg/L Trichloroethene 15/15 7.70E-01 mg/L 1.00E-04 Vinyl Chloride 1/4 mg/L cis-1,2-Dichloroethene 3/4 6.00E-03 mg/L 1.00E-04 trans-1,2-Dichloroethene 2/4 mg/L 6.80E+00 pCi/L Alpha activity 2/4 Beta activity 2/4 5.20E+00 pCi/L

------ LOCATION=AOC 204 MEDIA=Subsurface Soil ------

Analyte	Frequency of Detection	Maximum detected concentration	Background	Exceed Background?	Units
1,1,1-Trichloroethane	10/16	1.00E+00			mg/kg
1,1-Dichloroethane	8/14	1.00E+00			mg/kg
1,1-Dichloroethene	11/17	4.00E-02			mg/kg 🛡
PCB-1254	11/11	2.50E-02			mg/kg
PCB-1260	11/11	2.50E-02			mg/kg
Polychlorinated biphenyl	8/8	1.00E-01			mg/kg
Tetrachloroethene	11/17	1.00E+00			mg/kg
Trichloroethene	11/17	1.00B+00			mg/kg
Alpha activity	6/6	1.96E+01	·.		pCi/g
Beta activity	6/6	2.91E+01			pCi/g

----- LOCATION-SWMU 193A MEDIA-McNairy Groundwater

	Frequency	Maximum			
	of	detected	Background	Exceed	
Analyte	Detection	concentration	concentration .	Background?	Units
Calcium	4/4	8.218+01			mg/L
Chloride	4/4	1.605+01			mg/L
Iron	4/4	3.14E+02			mg/L
Magnesium	4/4	4.028+01			mg/L
Potassium	4/4	2.15E+01			mg/L
Sodium	4/4	8.08E+01			mg/L
Tetraoxo-sulfate(1-)	4/4	8.40E+01			mg/L
Acetone	1/1	1.40E-02			mg/L
Diethylphthalate	1/6	1.90B-02			mg/L
Trichloroethene	8/13	1.10E-02			mg/L
cis-1,2-Dichloroethene	1/11	1.70E-01			mg/L
Alpha activity	6/10	4.00E+01			pCi/L
Beta activity	8/10	6.46E+01			pCi/L
Technetium-99	5/10	1.45E+02	·		pCi/L
Thorium-234	1/1	8.40E-01			pCi/I
Uranium-234	1/1	8.10E-01			pCi/L
Uranium-238	1/1	1.32E+00		•	pCi/I

	_				
	Frequency	Maximum			
· · · · · · · · · · · · · · · · · · ·	of	detected	Background	Exceed	
Analyte	Detection	concentration	concentration	Background?	Onits
Aluminum	4/4	1.78E-01			mg/L
Ammonia	1/1	3.00E-01		•	mg/L
Calcium	5/5	1.34E+02			mg/L
Chloride	5/5	6.40E+01			mg/L
Copper	1/4	1.80E-02			mg/L
Fluoride	1/1	4.20E-01	. •		mg/L
Iron	7/9	3.66E+01			mg/L
Magnesium	5/5	1.85E+01			mg/L
Potassium ·	5/5 [.]	2.65E+02	•		mg/L
Silica .	1/1	1.90E+01			mg/L
Sodium	5/5	1.34E+02		:	mg/L
Tetraoxo-sulfate(1-)	5/5	2.62E+02			mg/L
Zinc	4/4	2.12E-01			mg/L
1,1-Dichloroethene	2/43	2.00E-04			mg/L
Diethylphthalate	3/25	1.50E-02			mg/L
Pentachlorophenol	1/25	1.20E-02			mg/L
Trichloroethene	44/51	6.70E+00			mg/L
bis(2-Ethylhexyl)phthalate	3/25	2.20E-02			mg/L
cis-1,2-Dichloroethene	17/42	8.40E-02			mg/L
trans-1,2-Dichloroethene	7/43	7.00E-04			mg/L
Alpha activity	19/34	1.76E+01			pCi/L
Beta activity	34/34	8.80E+02			pCi/L
Technetium-99	26/39	1.39E+03			pCi/L
Thorium-234	1/8	5.40E-01			pCi/L

-- LOCATION=SWMU 193A MEDIA=Subsurface Soil ------

Analyte	Frequency of Detection	Maximum detected concentration	Background concentration	Exceed Background?	Units
Analyte	Decector		001100110201	Padagaodina.	044.00
Aluminum	8/8	1.40E+04	12000.00	Yes	mg/kg
Barium	8/8	8.73E+01	170.00	No	mg/kg
Beryllium	5/8	7.00E-01	0.69	Yes	mg/kg
Calcium	4/4	2.73E+05	6100.00	Yes	mg/kg
Chromium	8/8	2.778+01		•	mg/kg
Cobalt	8/8	8.66E+00	13.00	No	mg/kg
Copper	8:/8	7.31E+00	25.00	No	mg/kg
Iron	8/8	1.54E+04	28000.00	No	mg/kg
Lithium	8/8	1.125+01			mg/kg
Magnesium	8/8	1.705+04	2100.00	Yes	mg/kg
Manganese	8/8	5.645+02	820.00	No	mg/kg
Nickel	5/8	9.16E+00			mg/kg
Potassium	8/8	1.44E+03	950.00	Yes	mg/kg
Silver	1/8	4.00E+00			mg/kg
Sodium	5/8	3.13E+02	340.00	No	mg/kg
Strontium	8/8	2.53E+02			mg/kg
Vanadium	8/8	3.15E+01	37.00	No	mg/kg
Zinc	8/8	5.548+01	60.00	No	mg/kg
Acetone	1/2	1.10E~02			mg/kg
Anthracene	1/8	1.16E-01			mg/kg
Benz (a) anthracene	2/8	1.80E-01			mg/kg
Benzo (a) pyrene	2/8	2,50E-01			mg/kg
Benzo (b) fluoranthene	2/8	5.10E-02			mg/kg
Benzo (ghi) perylene	2/8	1.70E-01			mg/kg
Chrysene	2/8	1.70E-01			mg/kg
Di-n-butylphthalate	1/8	7.70E-02			mg/kg
Di-n-octylphthalate	1/8	1.20E-01			mg/kg
Dibenz (a, h) anthracene	1/8	1.30E-01			mg/kg





----- LOCATION=SWMU 193A MEDIA=Subsurface Soil ------

		(continued)			
Analyte	Frequency of Detection	Maximum detected concentration	Background	Exceed Background?	Onits
Diethylphthalate	1/8	4.00E-01			mg/kg
Fluoranthene	2/8	3.10E-01			mg/kg
Indeno (1, 2, 3-cd) pyrene	2/8	1.60E-01			mg/kg
Pyrene	2/8	2.95E-01			mg/kg
bis (2-Ethylhexyl) phthalate	2/8	1.70E-01			mg/kg
Alpha activity	8/8	2.60E+01			pCi/g
Beta activity	8/8	2.37E+01			pCi/g

----- LOCATION=SWMU 193A MEDIA=Surface Soil ------

	Frequency of	Maximum detected	Background	Exceed	÷
Analyte	Detection	concentration	concentration	Background?	Units
Aluminum	4/4	1.09E+04	13000.00	No	mg/kg
Barium	4/4	8.405+01	200.00	No	mg/kg
Beryllium	1/4	6.40E-01	0.67	No	mg/kg
Calcium	2/2	2.73E+05	200000.00	Yes	mg/kg
Chromium	4/4	2.65E+01			mg/kg
Cobalt	4/4	5.70E+00	14.00	No	mg/kg
Copper	4/4	7.31E+00	19.00	No	mg/kg
Iron	4/4	1.54E+04	28000.00	No	mg/kg
Lithium	4/4	1.12E+01			mg/kg
Magnesium	4/4	1.70E+04	7700.00	Yes	mg/kg
Manganese	4/4	3.98E+02	1500.00	No	mg/kg
Nickel	2/4	7.50E+00	•		mg/kg
Potassium	4/4	1.44E+03	1300.00	Yes	mg/kg
Sodium	1/4	2.13E+02	320.00	No	mg/kg
Strontium	4/4	2.53E+02	· .		mg/kg
Vanadium	4/4	3.15E+01	38.00	No	mg/kg
Zinc	4/4	5.54E+01	65.00	No	mg/kg
Anthracene	1/4	1.16E-01			mg/kg
Benz (a) anthracene	2/4	1.805-01			mg/kg
Benzo (a) pyrene	2/4	2.50E-01			mg/kg
Benzo(b)fluoranthene	2/4	5.10E-02			mg/kg
Benzo (ghi) perylene	2/4	1.708-01			mg/kg
Chrysene	2/4	1.708-01			mg/kg
Di-n-butylphthalate	1/4	7.70E-02			mg/kg
Di-n-octylphthalate	1/4	1.20E-01			mg/kg
Dibenz (a, h) anthracene	1/4	1.305-01			mg/kg
Diethylphthalate	1/4	4.00E-01		,	mg/kg
Fluoranthene	2/4	3.10 5 -01			mg/kg
Indeno (1,2,3-cd) pyrene	2/4	1.60B-01			mg/kg
Pyrene	2/4	2.958-01			mg/kg
bis (2-Ethylhexyl) phthalate	2/4	1.705-01			mg/kg
Alpha activity	4/4	1.705+01			pCi/g
Beta activity	4/4	2.378+01			pCi/g

----- LOCATION-SWMU 193B MEDIA-McNairy Groundwater

Analyte	Frequency of Detection	Maximum detected concentration	Background	Exceed Background?	Units	
Trichloroethene cis-1,2-Dichloroethene Alpha activity	1/2 1/2 1/2	1.30E-02 2.30E-02 1.29E+00			mg/L mg/L pCi/L	

••••••••••••••••••••••••••••••••••••••	 LOCATION=SWMU	193B MEDIA=McNairy (continued)	Groundwatér		
Analyte	Frequency of Detection	Maximum detected concentration	Background concentration	Exceed Background?	Units
Beta activity	 2/2	4.80E+00			pCi/L

----- LOCATION=SWMU 193B MEDIA=RGA Groundwater -----

	,						
ı	Analyte	Frequency of Detection	Maximum detected concentration	Background concentration	Exceed Background?	Units	
	1,1-Dichloroethene	3/17	2.00E-02			mg/L	
	Acetone	1/2	3.30E-02	· .		mg/L	
	Carbon Tetrachloride	1/5	5.50E-03			mg/L	
	Di-n-butylphthalate	2/10	1.30E-02			mg/L	
	. Trichloroethene	17/17	5.00E-01			mg/L	
	bis (2-Ethylhexyl) phthalate	1/10	1.80E-02			mg/L	
	cis-1,2-Dichloroethene	12/17	9.87E-02			mg/L	
	trans-1,2-Dichloroethene	8/17	8.10E-04			mg/L	
	Alpha activity	12/17	6.60E+02			pC1/L	
	Beta activity	16/17	5.85R+02		'	pC1/L	
	Technetium-99	8/17	6.10E+01			pCi/L	

----- LOCATION-SWMU 193B MEDIA-Subsurface Soil -----

· · · · · · · · · · · · · · · · · · ·	Frequency of	Maximum detected	Background	Exceed	
Analyte	Detection	concentration	concentration	Background?	Onita
Aluminum	4/4	1.12E+04	12000.00	No	¤g/kg
Barium	4/4	8.42E+01	170.00	No	mg/kg
Beryllium	2/4	1.57E+00	0.69	Yes	mg/kg
Chromium	4/4	8.87E+01	14 C		mg/kg
Cobalt	4/4	7.76E+00	13.00	No	mg/kg
Copper	4/4	7.432+00	25.00	No	mg/kg
Iron	4/4	2.43E+04	28000.00	No	mg/kg
Lithium	4/4	7.72E+00			mg/kg
Magnesium	4/4	4.31E+03	2100.00	Yes	mg/kg
Manganese	4/4	2.22E+02	820.00	No	mg/kg
Nickel	2/4	2.06E+01			mg/kg
Potassium	4/4	6.86E+02	950.00	No	mg/kg
Sodium	4/4	4.48E+02	340.00	Yes	mg/kg
Strontium	4/4	9.39E+01			mg/kg
Vanadium	4/4	6.50E+01	37.00	Yes	mg/kg
Zinc	4/4	5.57E+01	60.00	No	mg/kg
Acetone	1/1	8.00E-02		14 - E	mg/kg
Toluene	1/3	1,00E-02	•		mg/kg
Alpha activity	4/4	1.86E+01		·.	pC1/g
Beta activity	4/4	2.295+01		·	pCi/g

----- LOCATION-SWMU 193B MEDIA-Surface Soil -----

Analyte	Frequency of Detection	Maximum detected concentration	Background concentration	Exceed Background? Un	lits
Aluminum Barium Beryllium	2/2 2/2 1/2	1.08 E+04 8.42E+01 1.57E+00	13000.00 200.00 0.67	No mg	J/kg J/kg J/kg

Table 1.6. Comparison	of maximum	detected	concentrations	and activities	to	background concentrations
			y location and a			-

LOCATION=SWMU 193B MEDIA=Surface Soil ----

		(continued)				
Analyte	Frequency of Detection	Maximum detected concentration	Background	Exceed Background?	. Units	
Chromium	2/2	8.878+01	· · ·		mg/kg	
Cobalt	2/2	7.76E+00	14.00	No	mg/kg	
Copper	2/2	7.43E+00	19.00	No	mg/kg	
Iron	2/2	2.43E+04	28000.00	No	mg/kg	
Lithium	2/2	7.728+00			mg/kg	
Magnesium	2/2	4.31E+03	7700.00	No	mg/kg	
Manganese	2/2	2.228+02	1500.00	No	mg/kg	
Nickel	1/2	2.06E+01			mg/kg	
Potassium	2/2	6.86E+02	1300.00	No	mg/kg	
Sodium	2/2	2.49E+02	320.00	No	mg/kg	
Strontium	2/2	9.39E+01			mg/kg	
Vanadium	2/2	6.50E+01	38.00	Yes	mg/kg	
Zinc	2/2	5.57E+01	65.00	No	mg/kg	
Toluene	1/2	1.00E-02			mg/kg	
Alpha activity	2/2	1.86E+01			pC1/g	
Beta activity	2/2	2.29E+01			pC1/g	

LOCATION-SWMU 193C MEDIA-McNairy Groundwater -----

Analyte	Frequency of Detection	Maximum detected concentration	Background concentration	Exceed Background?	Units
Aluminum	4/4	9.04E+01			mg/L
Antimony	5/5	2.50E-01			mg/L
Arsenic	5/5	3.605-02			mg/L
Barium	5/5	6.70E-01			mg/L
Beryllium	5/5	2.50E-02			mg/L
Cadmium	5/5	1.00E-01			mg/L
Calcium	5/5	4.10E+01		•	mg/L
Chloride	5/5	1.68E+01			mg/L
Chromium	3/3	2.328-01			mg/L
Cobalt	5/5	1.21E-01			mg/L
Copper	5/5	1.63E-01			mg/L
Fluoride	4/4	2.805-01			mg/L
Iron	5/5	1.798+02			mg/L
Lead	1/1	2.50E-01		,	mg/L
Magnesium	5/5	2.16E+01			mg/L
Manganese	5/5	3.91E+00	•		mg/L
Mercury	1/1	2.005-04			mg/L
Molybdenum	4/4	1.005-01			mg/L
Nickel	5/5	1.095-01			mg/L
Nitrate as Nitrogen	5/5	1.00E+00			mg/L
Potassium	5/5	1.016+02			mg/L
Selenium	3/3	5.00E-03			mg/L
Silica	5/5	1.805+01			mg/L
Silver	3/3	6.00E-02			mg/L
Sodium	5/5	2.63E+01	· .		mg/L
Tetraoxo-sulfate (1-)	5/5	1.30 B +01			mg/L
Thallium	2/2	1.238-01			mg/L
Oranium	9/9	1.80E-02			mg/L
Vanadium	2/2	8.36E-01			mg/L
Zinc	5/5	5.64B-01			mg/L
1,1,1-Trichloroethane	4/4	5.00E-03			mg/L
1,1,2-Trichloroethane	4/4	5.00E-03			mg/L
1,1-Dichloroethane	4/4	5.00E-03			mg/L
1,1-Dichloroethene	4/4	5.00E-03			mg/L
1,2-Dichloroethane	4/4	5.00E-03			mg/L

----- LOCATION-SWMU 193C MEDIA-McNairy Groundwater

(continued) . Frequency Maximum Background of detected Exceed Analyte Detection concentration concentration Background? Units 4/4 5.00E-03 Benzene mg/L Bromodichloromethane 4/4 5.00E-03 mg/L Carbon Tetrachloride 4/4 5.00E-03 mg/L Chloroform 4/4 5.00E-03 mg/L 4/4 Ethylbenzene 5.00E-03 mg/L Polychlorinated biphenyl 1/1 1.00E-04 mg/L Tetrachloroethene 4/4 5.00E-03 mg/L 5.00E-03 4/4 Toluene mg/L Trichloroethene 12/12 2.00E-03 mg/L Vinyl Chloride 4/4 1.00E-02 mg/L 1.00E-02 Xylene 4/4 mg/L cis-1,2-Dichloroethene 4/4 5.00E-03 mg/L 4/4 trans-1,2-Dichloroethene 5.00E-03 mg/L Alpha activity 12/12 1.07E+02 pCi/L pCi/L Beta activity 12/12 2.36E+02 Radon-222 2/2 1.57E+02 pCi/L Technetium-99 13/13 2.70E+01 pCi/L

----- LOCATION=SWMU 193C MEDIA=RGA Groundwater --

Analyte		Frequency of Detection	Maximum detected concentration	Background	Exceed Background?	Units
1,2-Dichloroethene Trichloroethene	•	1/2 1/2	5.62E-01 1.62E-01	· · · · · · · · · · · · · · · · · · ·	•	mg/L mg/L

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----- LOCATION-SWMU 193C MEDIA-Subsurface Soil ----

	Frequency of	Maximum detected	Background	Exceed	
Analyte	Detection	concentration	concentration	Background?	Unit
Aluminum	20/20	1.37E+04	12000.00	Yes	mg/k
Arsenic	5/20	6.578+00	7.90	No	mg/k
Barium	1/1	1.425+02	170.00	No	mg/k
Beryllium	10/20	9.80E-01	0.69	Yes .	mg/k
Boron	1/20	1.00E+02	•		mg/k
Cadmium	3/59	5.00E+00	0.21	Yes	mg/k
Calcium	18/20	4.00E+05	6100.00	Yes	mg/k
Chromium	59/61	8.30E+01	· · · · · · · · · · · · · · · · · · ·	10 A 10 A 10 A 10 A 10 A 10 A 10 A 10 A	mg/k
Cobalt	17/20	8.61E+01	13.00	Yes	mg/k
Copper	16/20	2.82E+01	25.00	Yes	mg/k
Iron	6/6	3,00E+04	28000.00	Yes	mg/k
Lead	42/61	6.77E+01	23.00	Yes	ang/k
Lithium	17/20	1.25E+01			mg/k
Magnesium	20/20	1.458+04	2100.00	Yes	mg/k
Manganese	20/20	2.27E+03	820.00	Yes	mg/k
Nickel	13/20	2.15E+01			mg/k
Potassium	20/20	1.57E+03	950.00	Yes	mg/k
Sodium	15/20	4.44E+02	340.00	Yes	mg/k
Strontium	20/20	3.91E+02		.,	mg/k
Vanadium	20/20	4.42E+01	37.00	Yes	mg/k
Zinc	19/20	9.25E+01	60.00	Yes	mg/k
Kylene	1/20	1.00E-02		•	mg/k
Alpha activity	53/53	4,00E+00			pCi/
Beta activity	53/53	1.00E+01			pCi/



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------ LOCATION-SWMU 193C MEDIA-Surface Soil -----

	Frequency	Maximum			
	of	detected	Background	Exceed	
Analyte	Detection	concentration	concentration	Background?	Units
Aluminum	5/5	3.36E+03	13000.00	No	mg/kg
Boron	1/5	1.00E+02			mg/kg
Calcium	5/5	4.00E+05	200000.00	Yes	mg/kg
Chromium	3/5	1.20E+01			mg/kg
Cobalt	2/5	2.14E+00	14.00	No	mg/kg
Copper	2/5	2.82E+01	19.00	Yes	mg/kg
Lead	1/5	6.77E+01	36.00	Yes	mg/kg
Lithium	3/5	1.25E+01			mg/kg
Magnesium	5/5	1.45E+04	7700.00	Yes	mg/kg
Manganese	5/5	1.98E+02	1500.00	No	mg/kg
Nickel	1/5	6.43E+00			mg/kg
Potassium	5/5	1.57E+03	1300.00	Yes	mg/kg
Sodium	4/5	3.10E+02	320.00	No	mg/kg
Strontium	5/5	3.91E+02			mg/kg
Vanadium	5/5	6.70E+00	38.00	No	mg/kg
Zinc	5/5	9.255+01	65.00	Yes	ng/kg

----- LOCATION-SWMU 194 MEDIA-Subsurface Soil -----

	Frequency of	Maximum detected	Background	Exceed	
Analyte	Detection	concentration	concentration	Background?	Units
Aluminum	12/12	1.45E+04	12000.00	Yes	mg/kg
Arsenic	1/12	6.73E+00	7.90	No	mg/kc
Barium	12/12	1.39E+02	170.00	No	mg/kg
Beryllium	6/12	4.80E+00	0.69	Yes	mg/kg
Cadmium	1/35	8.558+00	0.21	Yes	mg/kg
Calcium	12/12	6.81E+03	6100.00	Yes	mg/kc
Chromium	35/35	1.03E+02			mg/ka
Cobalt	12/12	9.46B+00	13.00	No	mg/kg
Copper	12/12	1.67E+01	25.00	No	mg/kg
Iron	12/12	2.00E+04	28000.00	No	mg/kg
Lead	20/35	3.60E+02	23.00	Yes	mg/kg
Lithium	12/12	9.00E+00			mg/kg
Magnesium	12/12	2.34E+03	2100.00	Yes	mg/kg
Manganese	12/12	4.67E+02	820.00	No	mg/kg
Nickel	8/12	1.37E+01			mg/kg
Potassium	12/12	6.32E+02	950.00	No	mg/kg
Sodium	8/12	3.69E+02	340.00	Yes	mg/kg
Strontium	12/12	2.60E+01			mg/kg
Vanadium	12/12	2.58E+01	37.00	No	mg/kg
Zinc	11/12	6.76E+01	60.00	Yes	mg/kg
Ethylbenzene	1/19	1.50E-02			mg/kg
Alpha activity	23/23	2.50E+00			pCi/g
Beta activity	23/23	7.00E+00			pCi/g

----- LOCATION-SWMU 99A MEDIA-MCNairy Groundwater -----

Analyte	Frequency of Detection	Maximum detected concentration	Background	Exceed Background?	Units
1,1,1-Trichloroethane	1/4	1.20E-03			mg/L
1,1-Dichloroethene	1/4	2.29E-02			mg/L
Carbon Tetrachloride	1/4	2.80E-03			mg/L
Trichloroethene	3/4	5.195-01			mg/L
cis-1,2-Dichloroethene	2/4	1.15E-01			mg/L

			LOCATION-SWMU	99A MEDIA=McNairy (continued)	Groundwater		
•	Analyte		Frequency of Detection	Maximum detected concentration	Background concentration	Exceed Background?	Units
	Alpha activity Beta activity Technetium-99	• • •	2/2 2/2 2/2	2.90E+00 3.50E+01 1.90E+01		•	pCi/L pCi/L pCi/L

-- LOCATION=SWMU 99A MEDIA=RGA Groundwater

Analyte	Frequency of Detection	Maximum detected concentration	Background concentration	Exceed Background?	Units
Aluminum	16/35	6.59E+02	•		mg/L
Arsenic	4/27	1.00E-02			mg/L mg/L
Barium	39/39	3.30E+00			mg/L
Beryllium	8/35	1.00E-01		1	mg/L
Calcium	39/39	1.20E+02		1	mg/L
Chloride	9/9	1.20E+02	·		mg/L
Chromium	11/39	1.78E+00			mg/L
Cobalt	20/37	5.70E-01			mg/L
Copper	9/35	6.40E-01			mg/L
Fluoride	8/B	2.00E-01	• ,		mg/L
Iron	31/39	1.20E+03	•	•	mg/L
Lead	6/29	4.10E-01	· · ·	and the second second second	mg/L
Lithium	6/23	1.70B-01	· · · · · · · · · · · · · · · · · · ·		mg/L
Magnesium	39/39	4.97E+01		• • •	mg/L
Manganese	37/39	4.60E+00	•		mg/L
Mercury	5/25	2.00E-02	• •		mg/L
Nickel	16/39	9.10E-01		· .	mg/L
Nitrate as Nitrogen	7/9	2.10E+00	• • •		mg/L
Potassium	24/39	2.17E+01			mg/L
Silica	9/9	2.50E+01		• •	mg/L
Sodium	39/39	7.24E+01			mg/L
Strontium	30/30	4.70E-01			mg/L
Sulfate	2/2	1.92E+01		1. · · ·	mg/L
Tetraoxo-sulfate(1-)	7/7	2.20E+01		•	mg/L
Vanadium	10/28	2.15E+00			mg/L
Zinc	10/35	2.55E+00		,	mg/L
1,1-Dichloroethene	7/33	6.50E-02			mg/L
Trichloroethene	41/43	2.37E+00	1. 1. Contract of the second second second second second second second second second second second second second		mg/L
bis (2-Ethylhexyl) phthalate	5/10	1.60B-02			mg/L
cis-1,2-Dichloroethene	10/33	3.48E-02			mg/L
trans-1,2-Dichloroethene	3/33	6.00E-04		1	mg/L
Alpha activity	33/39	5.38E+01		r e statu	pCi/L
Beta activity	39/39	1.37E+02		and the second second second second second second second second second second second second second second second	pCi/L
Radon-222	4/4	6.75E+02		•	pCi/L
Technetium-99	34/40	1.39E+02	• • •		pCi/L

-- LOCATION=SWMU 99A MEDIA=Subsurface Soil -----

۴.	Analyte	Frequency of Detection	Maximum detected concentration	Background concentration	Exceed Background?	Units
	Aluminum	22/22	1.41E+04	12000.00	Yes	mg/kg
	Antimony	5/22	2.90E+00	0.21	Yes	mg/kg
	Arsenic	11/22	8.55E+Q0	7.90	Yes	mg/kg
	Barium	22/22	2.47E+03	170.00	Yes	mg/kg
•;	Beryllium	11/22	8.90E-01	0.69	Yes	mg/kg



		(continued)			
	Frequency	Maximum			
•	of	detected	Background	Exceed	
Analyte	Detection	concentration	concentration	Background?	Units
Cadmium	5/22	8.30E-01	0.21	Voe	
	20/20	2.87E+05	6100.00	Yes Yes	mg/kg
Thromium	22/22	4.57E+01	0100.00	Ies	mg/kg
Cobalt	20/22	1.19E+01	13.00	Ne	mg/kg
Copper	21/22	1.64E+01	25.00	No	mg/kg
	2/16	5.40E-01	25.00	No	mg/kg
Cyanide	22/22	2.33E+04	28000.00	17-	mg/kg
lron Lead				NO	mg/kg
	6/22	4.73E+01	23.00	Yes	mg/kg
Lithium	17/17	1.29E+01			mg/kg
Magnesium	22/22	2.73E+04	2100.00	Yes	mg/kg
Anganese	22/22	1.46E+03	820.00	Yes	mg/kg
Mercury	5/22	1.20E-01	0.13	No	mg/kg
Nickel	17/22	2.58E+01	· ··		mg/kg
Potassium	22/22	1.12E+03	950.00	Yes	mg/kg
Selenium	5/20	3.20E-01			mg/kg
Silver	5/22	7.10E-01			mg/kg
Sodium	14/22	3.93E+02	340.00	Yes	mg/kg
Strontium	17/17	5.14E+02			mg/kg
Thallium	5/22	5,90E-01	0.34	Yes	mg/kg
Vanadium	22/22	3.55E+01	37.00	No	mg/kg
Zinc	21/22	1.63E+02	60.00	Yes	mg/kg
1,1,1-Trichloroethane	5/8	6.00E-03		XCD.	mg/kg
1,1,2,2-Tetrachloroethane	5/8	6.00E-03			mg/kg
1,1,2-Trichloroethane	5/8	6.00E-03			mg/kg
1,1-Dichloroethane					
	5/8	6.00E-03			mg/kg
1,1-Dichloroethene	5/10	6.00E-03			mg/kg
1,2,4-Trichlorobenzene	5/22	4.10E-01	·		mg/kg
1,2-Dichlorobenzene	5/22	4.10E-01		•	mg/kg
1,2-Dichloroethane	5/8	6.00E-03			mg/kg
1,2-Dichloroethene	5/5	6.00E-03			mg/kg
1,2-Dichloropropane	5/8	6.00E-03	<i>x</i>		mg/kg
1,3-Dichlorobenzene	5/22	4.10E-01			mg/kg
1,4-Dichlorobenzene	5/22	4.10E-01			mg/kg
2,4,5-Trichlorophenol	5/22	2.10E+00			mg/kg
2,4,6-Trichlorophenol	5/22	4.10E-01			mg/kg
2,4-Dichlorophenol	5/22	4.10E-01			mg/kg
2,4-Dimethylphenol	5/22	4.10E-01			mg/kg
2,4-Dinitrophenol	5/6	2.10E+00			mg/kg
2,4-Dinitrotoluene	5/22	4.10E-01			mg/kg
2,6-Dinitrotoluene	5/22	4.10E-01			mg/kg
2-Butanone	5/8	1.20E-02			mg/kg
2-Sulanone 2-Chloronaphthalene	5/22	4.10E-01			mg/kg
	5/22	4.10E-01			mg/kg
2-Chlorophenol	5/22	1.20E-02			mg/kg
2-Hexanone		-			mg/kg
2-Methyl-4,6-dinitrophenol	5/22	2.10E+00			mg/kg
2-Methylnaphthalene	5/22	4.10E-01			mg/kg
2-Methylphenol	5/22	4.10E-01		• •	mg/kg
2-Nitroaniline	5/22	2.10E+00			mg/kg
2-Nitrophenol	5/22	4.10E-01			
3,3'-Dichlorobenzidine	5/22	8.20E-01			mg/kg
3-Nitroaniline	5/22	2.10E+00			mg/kg
4,4'-DDD	2/2	3.50E-02			mg/kg
4,4"-DDE	2/2	3.50E-02			mg/kg
4,4'-DDT	2/2	3.50E-02			mg/kg
4-Bromophenyl phenyl ether	5/22	4.10E-01			mg/kg
4-Chloro-3-methylphenol	5/22	4.10E-01			mg/kg
4-Chloroaniline	5/22	4.10E-01			mg/kg
		4.10E-01			mg/kg
4-Chlorophenyl phenyl ether	5/22 5/8	1.20E-02			mg/kg

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	Frequency of	detected	Background	Exceed	
halyte	Detection	concentration	concentration	Background?	Units
		1.1.1	· · · · · · · · · · · · · · · · · · ·	Damiground,	0112 03
Methylphenol	5/22	4.10E-01	. •		mg/kg
-Nitroaniline	5/22	2.10E+00			mg/kg
-Nitrophenol	5/22	2.10E+00			mg/kg
cenaphthene	7/22	4.10E-01			mg/kg
cenaphthylene	6/22	6.10E-01	· .		mg/kg
cetone	5/8	5.30E-02			mg/kg
ldrin	2/2	1.70E-02			mg/kg
nthracene	7/22	7.50E-01			mg/kg
anz (a) anthracene	8/22	1.70E+00		•	mg/kg
anzene	5/8	6.00E-03			mg/kg
enzo(a) pyrene	7/22	2.10E+00			mg/kg
anzo (b) fluoranthene	11/22 7/22	5.70E+00 1.18E+00			mg/kg
enzo (ghi) perylene	8/22	7.90E-01			mg/kg
enzo(k)fluoranthene enzoic Acid	8/22 5/5	2.10E+00	· ·		mg/kg
	5/5	4.10E-01		•	ng/kg ng/kg
enzyl Alcohol romodichloromethane	5/8	6.00E-03			mg/kg
romodichioromethane	5/8	6.00E-03			mg/kg
romotorm	5/8	1.20E-02			mg/kg
utyl benzyl phthalate	5/6	4.10E-01	•		mg/kg
arbon Disulfide	5/8	6.00E-03	•		mg/kg
arbon Disdifide arbon Tetrachloride	5/8	6.00E-03			mg/kg
hlorobenzene	5/8	6.00E-03			mg/kg
hloroethane	5/8	1.20E-02			mg/kg
hloroform	5/8	6.00E-02			mg/kg
hloromethane	5/8	1.20E-02	;		mg/kg
hrysene	7/22	2.10E+00			mg/kg
i-n-butylphthalate	5/22	4.10E-01		1	mg/kg
i-n-octylphthalate	5/22	4.10E-01			mg/kg
ibenz(a, h) anthracene	6/22	4.80E-01			mg/kg
ibenzofuran	6/22	4.10E-01			mg/kg
ibromochloromethane	5/8	6.00E-03			ng/kg
ieldrin	2/2	3.50E-02			mg/kg
iethylphthalate	5/22	4.10E-01			mg/kg
imethylphthalate	5/22	4.10E-01			mg/kg
ndosulfan I	2/2	1.70E-02	•		mg/kg
ndosulfan II	2/2	3.50E-02			mg/kg
ndosulfan Sulfate	2/2	3.50E-02	1. A.		mg/kg
ndrin	2/2	3.50E-02			mg/kg
ndrin Ketone	2/2	3.50E-02			mg/kg
thylbenzene	5/8	6.00E-03		1	mg/kg
luoranthene	9/22	2.66E+00			mg/kg
luorene	6/22	4.10E-01			mg/kg
eptachlor	2/2	1.70E-02			mg/kg
eptachlor Epoxide	2/2	1.705-02	• 1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1.		mg/kg
exachlorobenzene	5/22	4.10E-01			mg/kg
exachlorobutadiene	5/22	4.10E-01			mg/kg
exachlorocyclopentadiene	5/22	4.10E-01			mg/kg
rachloroethane	5/22	4.10E-01		: ·	mg/kg
ndeno (1,2,3-cd) pyrene	7/22	1.05E+00			mg/kg
ophorone	5/22	4.10E-01			mg/kg
thoxychlor	2/2	1.70E-01			mg/kg
thylene Chloride	5/8	8.00E-03		-	mg/kg
-Nitroso-di-n-propylamine	5/22	4.10E-01	•		mg/kg
-Nitrosodiphenylamine	5/22	4.10E-01		•	mg/kg
aphthalene	5/22	4.10E-01			mg/kg
itrobenzene	5/22	4.10E-01			mg/kg
CB-1016	3/23	1.87E+00			mg/kg
	2/23	1.70E-01			mg/kg



	LOCATION-SWMU	(continued)	rface Soil		·
	Frequency	Maximum	Background	Exceed	
Analyte	Detection	concentration	concentration	Background?	Units
PCB-1232	2/23	1.70E-01			mg/kg
PCB-1242	2/23	1.70E-01			mg/kg
PCB-1248	2/23	1.70E-01			mg/kg
PCB-1254	3/23	3.50E-01			mg/kg
PCB-1260	7/23	6.31E-01			mg/kg
Pentachlorophenol	5/22	2.10E+00			mg/kg
Phenanthrene	7/22	1.63E+00			mg/kg
Phenol	5/22	4.102-01			mg/kg
Pyrene	8/22	2.70E+00			mg/kg
Styrene	5/8	6.00E-03			mg/kg
Tetrachloroethene	5/8	6.00E-03			mg/kg
Toluene	5/8	6.00E-03			mg/kg
Toxaphene	2/2	3.508-01			mg/kg
Trichloroethene	6/10	6.00E-03			mg/kg
Vinyl Acetate	5/5	1.20E-02			mg/kg
Vinyl Chloride	5/10	1.20B-02			mg/kg
Xylene	5/5	6.00B-03		•	mg/kg
alpha-BHC	2/2	1.70B-02			mg/kg
alpha-Chlordane	2/2	1.70B-01			mg/kg
beta-BHC	2/2	1.70E-02			mg/kg
bis(2-Chloroethoxy)methane	5/22	4.10E-01			mg/kg
bis(2-Chloroethyl)ether	5/22	4.108-01		•	mg/kg
bis (2-Chloroisopropyl) ether	5/22	4.108-01			mg/kg
bis(2-Ethylhexyl)phthalate	5/22	3.60B-01			mg/kg
cis-1,3-Dichloropropene	5/8	6.00B-03			mg/kg
delta-BHC	2/2	1.70E-02			mg/kg
gamma-BHC (Lindane)	2/2	1.70E-02			mg/kg
gamma-Chlordane	2/2	1.70B-01			mg/kg
trans-1,3-Dichloropropene	5/8	6.00E-03			mg/kg
Alpha activity	20/21	1.42E+02			pCi/g pCi/g
Beta activity	21/21	2.73E+03	0.28	Yes	pCi/g
Cesium-137	3/21	1.90E+00	0.28	Ies	pCi/g
Neptunium-237	4/4	1.28E+01		•	pCi/g
Plutonium-239	3/3	6.00E-03	2.80	Yes	pCi/g
Technetium-99	6/23	2.65E+03	2.80	168	pCi/g
Thorium-230	3/3	6.70B-01			pCi/g
Thorium-234	1/21	5.30B+01	2.40	Yes	pCi/g
Uranium-234	4/4	1.64B+01	0.14	No	pCi/g
Uranium-235	3/24	4.10E-02 5.17E+01	1.20	Yes	pCi/g
Uranium-238	4/4	D.T.VATOT	1.40		Perio a

--- LOCATION-SWMU 99A MEDIA-Surface Soil --

Analyte	Frequency of Detection	Maximum detected concentration	Background concentration	Exceed Background?	Units
Aluminum Arsenic Barium Beryllium Calcium Chromium Cobalt Copper Iron	13/13 6/13 13/13 5/13 11/11 13/13 11/13 12/13 13/13	1.298+04 8.558+00 2.478+03 8.908-01 2.878+05 4.578+01 9.678+00 1.228+01 2.338+04	13000.00 12.00 200.00 0.67 200000.00 14.00 19.00 28000.00	No No Yes Yes No No No	mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg
Lithium Magnesium	13/13 13/13	1.29E+01 2.73E+04	7700.00	Yes	mg/kg mg/kg

----- LOCATION=SWMU 99A MEDIA=Surface Soil ----- (continued)

	Frequency of	Maximum detected	Background		•
Analyte	Detection	concentration	concentration	Exceed Background?	Units
·	Deperture			Dackgroundr	OUTES
Manganese	13/13	3.87E+02	1500.00	NO	mg/kg
Nickel	8/13	2.16E+01			mg/kg
Potassium	13/13	1.12E+03	1300.00	No	mg/kg
Sodium	6/13	3.66E+02	320.00	Yes	mg/kg
Strontium	13/13	5.145+02		•	mg/kg
Vanadium	.13/13	3.55 E +01	38.00	NO	mg/kg
Zinc	12/13	1.63E+02	65.00	Yes	mg/kg
Acenaphthene	2/13	3.30E-01			mg/kg
Acenaphthylene	1/13	6.10E-01			mg/kg
Anthracene	2/13	7.50E-01			mg/kg
Benz (a) anthracene	3/13	1.70E+00		•	mg/kg
Benzo (a) pyrene	2/13	2.10E+00			mg/kg
Benzo(b) fluoranthene	6/13	5.70E+00			mg/kg
Benzo (ghi) perylene	2/13	1.18E+00			mg/kg
Benzo(k) fluoranthene	3/13	7.90E-01			mg/kg
Chrysene	2/13	2.10E+00			mg/kg
Dibenz (a, h) anthracene	1/13	4.80E-01			mg/kg
Dibenzofuran	1/13	1.23E-01	•		mg/kg
Fluoranthene	4/13	2.66E+00			mg/kg
Fluorene	1/13	2.19E-01			mg/kg
Indeno(1,2,3-cd)pyrene	2/13	1.05E+00			mg/kg
PCB-1016	1/16	1.87E+00			mg/kg
PCB-1254	1/16	9.60E-02			mg/kg
PCB-1260	5/16	6.31E-01			mg/kg
Phenanthrene	2/13	1.63E+00			mg/kg
Pyrene	3/13	2.70E+00			mg/kg
Alpha activity	15/16	1.42E+02	•		pCi/g
Beta activity	16/16	2.73E+03			pCi/g
Cesium-137	3/16	· 1.90E+00	· 0.49	Yes	pCi/g
Neptunium-237	1/1	1.28E+01	0.10	Yes	pCi/g
Technetium-99	3/16	2.65E+03	2.50	Yes	pCi/g
Thorium-234	1/16	5.30E+01			pCi/g
Uranium-234	1/1	1.64E+01	2.50	Yes	pC1/g
Uranium-238	1/1	5.17E+01	1.20	Yes	pCi/g

----- LOCATION=SWMU 99B MEDIA=RGA Groundwater

Analyte	Frequency of Detection	Maximum detected concentration	Background concentration	Exceed Background?	Units
Maryca	Decederon				
Barium	7/7	2.70E+00			mg/L
Calcium	7/7	3.27E+01			mg/L
Chloride	7/7	1.08E+02			mg/L
Chromium	1/7	2.60E-01			mg/L
Copper	1/7	4.00E-02			mg/L
Fluoride	7/7	2.10E-01			mg/L
Iron	3/7	3.34E+00			mg/L
Magnesium	7/7	1.31E+01			mg/L
Manganese	5/7	2.90E-01			mg/L
Nitrate as Nitrogen	7/7	2.10E+00			mg/L
Silica	7/7	2.00E+01			mg/L
Sodium	7/7	7.86E+01			mg/L
Sulfate	2/2	2.67E+01			mg/L
Tetraoxo-sulfate(1-)	5/5	2,90E+01			mg/L
Zinc	2/7	6.00E-02			mg/L
Trichloroethene	16/16	2.30E+00			mg/L
Alpha activity	12/16	4.20E+00			pCi/L

 	LOCATION=SWMU	99B MEDIA-RGA (continued)	Groundwater -	 * 	
Analyte	Frequency of Detection	Maximum detected concentration	Backgrou Concentrat	 eed round? (Jnits
 Beta activity Radon-222 Technetium-99	16/16 4/4 12/17	4.50E+01 4.12E+02 1.90E+01		F	0C1/L 0C1/L 0C1/L

-- LOCATION-SWMU 998 MEDIA-Subsurface Soil -----

Analyte	Frequency of Detection	Maximum detected concentration	Background concentration	Exceed Background?	Units
Aluminum	8/8	1.70E+04	12000.00	Yes	mg/kg
Arsenic	2/6	8.05E+00	7.90	Yes	mg/kg
Barium	8/8	1.558+02	170.00	No	mg/kg
Beryllium	6/8	1.00E+00	0.69	Yes	mg/kg
Calcium	6/6	7.17E+03	6100.00	Yes	mg/kg
Chromium	8/8	2.61E+01			mg/kg
Cobalt	8/8	6.94E+00	13.00	No	mg/kg
Copper	8/8	1.308+01	25.00	No	mg/kg
Iron	8/8	1.81E+04	28000.00	No	mg/kg
Lithium	8/8	1.14E+01		•,	mg/kg
Magnesium	8/8	2.53E+03	2100.00	Yes	mg/kg
Manganese	8/8	5.24E+02	820.00	No	mg/kg
Nickel	5/8	2.51E+01			mg/kg
Potassium	8/8	1.04E+03	950.00	Yes	mg/kg
Sodium	3/8	3.09E+02	340.00	No	mg/kg
Strontium	8/8	2.22E+01	•		mg/kg
Vanadium	8/8	3.44E+01	37:.00	No	mg/kg
Zinc	8/8	5.228+01	60.00	No	mg/kg
Acetone	1/7	5.50E-01	•		mg/kg
Methylene Chloride	3/7	1.20E+00			mg/kg
Alpha activity	8/8	2.14E+01			pCi/g
Beta activity	8/8	2.26E+01			pCi/g

Analyte	Recommended dietary allowance ^a (mg/d)
Calcium	800
Chloride	600 ^b
Copper	1.0-2.0
Fluoride	1.5-2.5
lodine	0.12
Iron	10
Magnesium	170
Molybdenum	0.05-0.15
Phosphorus	800
Potassium	1600 ^b
Selenium	0.03
Sodium	400 ^b

Table 1.7. Recommended dietary allowances of essential human nutrients

^a Taken from National Research Council (NRC), 1989. *Recommended Dietary Allowances*, 10th Ed. RDAs listed are those for children ages 7–10.

^bEstimated minimum requirements of healthy persons ages 6–9.

Table 1.8. Comp	parison of	f maximum dete			ntial nutrient	ts to recommended	dietary allowances	
			f	or children				
 			- TOCATTON-30C 2	04 MEDTA-DOA	Groundwater			

Analyte	Frequency of Detection	Maximum detected concentration	Units	Daily dose for child	RDA for child	1/5 RDA	Exceed RDA?
1,1,1-Trichloroethane	~ 11/11	1.80E-02	mg/L	1.80E-02	•		
1,1-Dichloroethane	1/1	5.00E+00	mg/L	5.00E+00			•
1,1-Dichloroethene	12/15	4.00E-02	mg/L	4.00E-02			
PCB-1254	11/11	2.50E-02	mg/L	2.50E-02			
PCB-1260	11/11	2.50E-02	mg/L	2.50E-02			
Polychlorinated biphenyl	1/1	1.70E-01	mg/L	1.70E-01			
Tetrachloroethene	11/11	5.00B+00	mg/L	5.00E+00	•		
Trichloroethene	15/15	7.70E-01	mg/L	7.70E-01			
Vinyl Chloride	. 1/4	1.00E-04	mg/L	1.00E-04			
cis-1,2-Dichloroethene	3/4	6.00E-03	mg/L	6.00E-03	,		
trans-1,2-Dichloroethene	2/4	1.00E-04	mg/L	1.00E-04			
Alpha activity	2/4	6.80E+00	pCi/L			•	
Beta activity	2/4	5.20E+00	pCi/L				
	Frequency	ATION=AOC 204 MEI	IA-Subsur		RDA		
Analyte			DIA-Subsur Units	face Soil Daily dose for child	RDA for child	1/5 RDA	Exceed RDA?
-	Frequency of	Maximum detected		Daily dose	for	1/5 RDA	
1,1,1-Trichloroethane 1,1-Dichloroethane	Frequency of Detection	Maximum detected concentration	Units mg/kg mg/kg	Daily dose for child	for	1/5 RDA	
1,1,1-Trichloroethane 1,1-Dichloroethane 1,1-Dichloroethene	Frequency of Detection 10/16 8/14 11/17	Maximum detected concentration 1.00E+00 1.00E+00 4.00E-02	Units mg/kg mg/kg mg/kg	Daily dose for child 2.00E-04	for	1/5 RDA	
1,1,1-Trichloroethane 1,1-Dichloroethane 1,1-Dichloroethene PCB-1254	Frequency of Detection 10/16 8/14 11/17 11/11	Maximum detected concentration 1.00E+00 1.00E+00 4.00E-02 2.50E-02	Units mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.00E-04 2.00E-04 8.00E-06 5.00E-06	for	1/5 RDA	
1,1,1-Trichloroethane 1,1-Dichloroethane 1,1-Dichloroethene PCB-1254 PCB-1260	Frequency of Detection 10/16 8/14 11/17 11/11 11/11	Maximum detected concentration 1.00E+00 1.00E+00 4.00E-02 2.50E-02 2.50E-02	Units mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.00E-04 2.00E-04 8.00E-06 5.00E-06 5.00E-06	for	1/5 RDA	
1,1,1-Trichloroethane 1,1-Dichloroethane 1,1-Dichloroethane PCB-1254 PCB-1260 Polychlorinated biphenyl	Frequency of Detection 10/16 8/14 11/17 11/11 11/11 8/8	Maximum detected concentration 1.00E+00 4.00E-02 2.50E-02 2.50E-02 1.00E-01	Units mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.00E-04 2.00E-04 8.00E-06 5.00E-06 5.00E-06 2.00E-05	for	1/5 RDA	
1,1,1-Trichloroethane 1,1-Dichloroethane 1,1-Dichloroethene PCB-1254 PCB-1260 Polychlorinated biphenyl Tetrachloroethene	Frequency of Detection 10/16 8/14 11/17 11/11 11/11 8/8 11/17	Maximm detected concentration 1.00E+00 4.00E-02 2.50E-02 2.50E-02 1.00E-01 1.00E+00	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.00E-04 2.00E-04 8.00E-06 5.00E-06 5.00E-06 2.00E-05 2.00E-04	for	1/5 RDA	
1,1,1-Trichloroethane 1,1-Dichloroethane 1,1-Dichloroethene PCB-1254 PCB-1260 Polychlorinated biphenyl Tetrachloroethene Trichloroethene	Frequency of Detection 10/16 8/14 11/17 11/11 11/11 8/8 11/17 11/17	Maximm detected concentration 1.00E+00 1.00E+00 4.00E-02 2.50E-02 2.50E-02 1.00E-01 1.00E+00 1.00E+00	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.00E-04 2.00E-04 8.00E-06 5.00E-06 5.00E-06 2.00E-05	for	1/5 RDA	
1,1,1-Trichloroethane 1,1-Dichloroethane 1,1-Dichloroethene PCB-1254 PCB-1260 Polychlorinated biphenyl Tetrachloroethene	Frequency of Detection 10/16 8/14 11/17 11/11 11/11 8/8 11/17 11/17 6/6	Maximm detected concentration 1.00E+00 1.00E+00 4.00E-02 2.50E-02 2.50E-02 1.00E-01 1.00E+00 1.00E+00 1.96E+01	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.00E-04 2.00E-04 8.00E-06 5.00E-06 5.00E-06 2.00E-05 2.00E-04	for	1/5 RDA	
1,1,1-Trichloroethane 1,1-Dichloroethane 1,1-Dichloroethene PCB-1254 PCB-1260 Polychlorinated biphenyl Tetrachloroethene Trichloroethene	Frequency of Detection 10/16 8/14 11/17 11/11 11/11 8/8 11/17 11/17	Maximm detected concentration 1.00E+00 1.00E+00 4.00E-02 2.50E-02 2.50E-02 1.00E-01 1.00E+00 1.00E+00	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.00E-04 2.00E-04 8.00E-06 5.00E-06 5.00E-06 2.00E-05 2.00E-04	for	1/5 RDA	
1,1,1-Trichloroethane 1,1-Dichloroethane 1,1-Dichloroethane PCB-1254 PCB-1260 Polychlorinated biphenyl Tetrachloroethane Trichloroethane Alpha activity	Frequency of Detection 10/16 8/14 11/17 11/11 11/11 8/8 11/17 11/17 6/6	Maximm detected concentration 1.00E+00 1.00E+00 4.00E-02 2.50E-02 2.50E-02 1.00E-01 1.00E+00 1.00E+00 1.96E+01	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg pCi/g	Daily dose for child 2.00E-04 2.00E-04 8.00E-06 5.00E-06 5.00E-06 2.00E-05 2.00E-04	for	1/5 RDA	
1,1,1-Trichloroethane 1,1-Dichloroethane 1,1-Dichloroethane PCB-1254 PCB-1260 Polychlorinated biphenyl Tetrachloroethane Trichloroethane Alpha activity	Frequency of Detection 10/16 8/14 11/17 11/11 11/11 8/8 11/17 11/17 6/6 6/6	Maximm detected concentration 1.00E+00 1.00E+00 4.00E-02 2.50E-02 2.50E-02 1.00E-01 1.00E+00 1.00E+00 1.96E+01	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg pCi/g pCi/g	Daily dose for child 2.00E-04 2.00E-04 8.00E-06 5.00E-06 5.00E-06 2.00E-05 2.00E-04 2.00E-04	for child	1/5 RDA	
1,1,1-Trichloroethane 1,1-Dichloroethane 1,1-Dichloroethane PCB-1254 PCB-1260 Polychlorinated biphenyl Tetrachloroethane Trichloroethane Alpha activity	Frequency of Detection 10/16 8/14 11/17 11/11 11/11 8/8 11/17 11/17 6/6 6/6	Maximum detected concentration 1.00E+00 1.00E+00 4.00E-02 2.50E-02 2.50E-02 1.00E-01 1.00E+00 1.00E+00 1.96E+01 2.91E+01	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg pCi/g pCi/g	Daily dose for child 2.00E-04 2.00E-04 8.00E-06 5.00E-06 5.00E-06 2.00E-05 2.00E-04 2.00E-04	for child	1/5 RDA	
1,1,1-Trichloroethane 1,1-Dichloroethane 1,1-Dichloroethane PCB-1254 PCB-1260 Polychlorinated biphenyl Tetrachloroethane Trichloroethane Alpha activity	Frequency of Detection 10/16 8/14 11/17 11/11 11/11 8/8 11/17 11/17 6/6 6/6	Maximum detected concentration 1.00E+00 1.00E+00 4.00E-02 2.50E-02 2.50E-02 1.00E-01 1.00E+00 1.00E+00 1.96E+01 2.91E+01 2.91E+01	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg pCi/g pCi/g	Daily dose for child 2.00E-04 2.00E-04 8.00E-06 5.00E-06 5.00E-06 2.00E-05 2.00E-04 2.00E-04	for child	1/5 RDA	

mg/L

8.21E+01

8.00E+02

1.60E+02

No

4/4

8.21E+01

A-110

Calcium

	Frequency	Maximum			RDA		
Bana Triable	of Detection	detected concentration	Units	Daily dose for child	for child	1/5 RDA	Exceed RDA?
Analyte	Derection	concenciacion	UIILLA	tor child	GHLIG .	1/5 KDA	KDAT
Chloride	4/4	1.60E+01	mg/L	1.60E+01	6.00E+02	1.20E+02	NO
Iron	4/4	3.14E+02	mg/L	3.14E+02	1.00E+01	2.00E+00	Yes
Magnesium	4/4	4.02B+01	mg/L	4.02E+01	1.70E+02	3.40E+01	Yes
Potassium	4/4	2.15B+01	mg/L	2.15E+01	1.60E+03	3.20E+02	NO
Sodium	4/4	8.08E+01	mg/L	8.08E+01		• .	
Tetraoxo-sulfate (1-)	4/4	8.40E+01	mg/L	8.40E+01 ~~			•
Acetone	1/1	1.40E-02	mg/L	1.40E-02	· • • ·		• •
Diethylphthalate	1/6	1.90E-02	mg/L	1.90E-02	N		
Trichloroethene	8/13	1.10E-02	mg/L	1.10E-02	· _		
cis-1,2-Dichloroethene	1/11	1.70E-01	mg/L	1.70E-01			
Alpha activity	6/10	4.00E+01	pCi/L	•		· · ·	
Beta activity	8/10	6.46E+01	pCi/L	•			
Technetium-99	5/10	1.45E+02	pC1/L			-	
Thorium-234	1/1	8.40E-01	pCi/L			1	
Uranium-234	<u>1/1</u>	8.10E-01	pC1/L	• ·		,	
Uranium-238	1/1	1,32B+00	pCi/L				

LOCATION-SWMU 193A MEDIA-RGA Groundwater -

		•		·. ·			
Analyte	Frequency of Detection	Maximum detected concentration	Unitë	Daily dose for child	RDA for child	1/5 RDA	Exceed RDA?
Aluminum	4/4	1.78E-01	mg/L	1.78E-01		· -	•
Ammonia	1/1	3.00E-01	mg/L	3.00E-01			
Calcium	5/5	1.34E+02	mg/L	1.34E+02	8.00E+02	1.60E+02	No
Chloride	5/5	6.40E+01	mg/L	6.40E+01	6.00E+02	1.20E+02	No
Copper	1/4	1.80E-02	mg/L	1.80E-02	1.00E+00	2.00E-01	No
Fluoride	1/1	4.20E-01	mg/L	4.20E-01	1.50E+00	3.00E-01	Yes
Iron	7/9	3.66E+01	mg/L	3.66E+01	1.00E+01	2.00E+00	Yes
Magnesium	5/5	1.85E+01	mg/L	1.85E+01	1.70E+02	3.40E+01	No
Potassium	5/5	2.65E+02	mg/L	2.65E+02	1.60E+03	3.20E+02	No
Silica	1/1	1.90E+01	mg/L	1.90E+01			
Sodium	5/5	1.34E+02	mg/L	1.34E+02	•		
Tetraoxo-sulfate(1-)	5/5	2.62E+02	mg/L	2.62E+02			
Zinc	4/4	2.12E-01	mg/L	2.12E-01			
1,1-Dichloroethene	2/43	2.00E-04	mg/L	2.00E-04			
Diethylphthalate	- 3/25	1.50E-02	mg/L	1.50E-02	·		

	_						
	Frequency	Maximum			RDA		
- - -	of	detected		Daily dose	for		Exceed
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?
Pentachlorophenol	1/25	1.20E-02	mg/L	1.20E-02	•••		
Trichloroethene	44/51	6.70E+00	mg/L	6.70E+00			
bis(2-Ethylhexyl)phthalate	3/25	2.20E-02	mg/L	2.20E-02			
cis-1,2-Dichloroethene	17/42	8.40E-02	mg/L	8.40E-02			
trans-1,2-Dichloroethene	7/43	7.00E-04	mg/L	7.00E-04			
Alpha activity	19/34	1.76E+01	pCi/L				
Beta activity	34/34	8.80E+02	pCi/L				
Technetium-99	26/39	1.39E+03	pCi/L				
Thorium-234	1/8	5.40E-01	pCi/L				
						· · ·	
	LOCA	TION-SWMU 193A ME	DIA=Subsu	rface Soil			
	Frequency	Maximum			RDA		
	of	detected		Daily dose	for	•	Exceed
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?
Aluminum	8/8	1.40E+04	mg/kg	2.80E+00			
Barium	8/8	8.73E+01	wg/kg	1.75E-02			
Beryllium	5/8	7.00E-01	tug/kg	1.40E-04			
Calcium	4/4	2.73E+05	mg/kg	5.46B+01	8.00E+02	1.60E+02	No
Chromium	8/8	2.77E+01	mg/kg	5.54E-03			
Cobalt	8/8	8.66E+00	mg/kg	1.73E-03			
Copper	8/8	7.31E+00	mg/kg	1.46E-03			
Iron	8/8	1.54E+04	mg/kg	3.08E+00			
Lithium	8/8	1.12E+01	mg/kg	2.24E-03			
Magnesium	8/8	1.70E+04	mg/kg	3.40E+00	1.50E+02	3.00E+01	No
Manganese	8/8	5.64E+02	ng/kg	1.13E-01			
Nickel	5/8	9.16E+00	mg/kg	1.83E-03			
Potassium	8/8	1.44E+03	mg/kg	2.88E-01	1.60E+03	3.20E+02	No
Silver	1/8	4.00E+00	mg/kg	8.00E-04			
Sodium	5/8	3.13E+02	mg/kg	6.26E-02			
Strontium	8/8	2.53E+02	mg/kg	5.06E-02			
Vanadium	8/8	3.15E+01	mg/kg	6.30E-03			
Zinc	8/8	5.54E+01	mg/kg	1.11E-02			
Acetone	1/2	1.10E-02	mg/kg	2.20E-06			
Anthracene	1/8	1.16E-01	mg/kg	2.32E-05	• •		
Benz (a) anthracene	2/8	1.80E-01	mg/kg	3.60B-05			
Benzo (a) pyrene	2/8	2.50E-01	tug/kg	5.00E-05			

		(contin	ued)					
	Frequency	Maximum			RDA			
	of			· Pad Tan Jama				
		detected		Daily dose	for		Exceed	
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?	
Benzo (b) fluoranthene	2/8	5.10E-02	mg/kg	1.028-05				
Benzo (ghi) perylene	2/8	1.70E-01	mg/kg	3.40E-05				
Chrysene	2/8	1.70E-01	mg/kg	3.40E-05	•			
Di-n-butylphthalate	1/8	7.70E-02	mg/kg	1.548-05				
Di-n-octylphthalate	1/8	1.208-01	mg/kg	2.40E-05				
Dibenz (a, h) anthracene	1/8	1.30E-01	mg/kg	2.60E-05				
Diethylphthalate	1/8	4.00B-01	mg/kg	8.00E-05				
Fluoranthene	2/8	3.10E-01	mg/kg	6.20E-05				
Indeno (1,2,3-cd) pyrane	2/8	1.60E-01	mg/kg	3.20E-05				
Pyrene	2/8	2.95E-01	mg/kg	5.90E-05				
bis(2-Ethylhexyl)phthalate	2/8	1.70E-01	mg/kg	3.40E-D5				
Alpha activity	8/8	2.508+01	pCi/g	5.402-05				
Beta activity	8/8	2.37E+01	pCi/g					
Bela activity	8/8	2.3/8+01	Pct/d					
	Frequency	ATION-SWMU 193A M Maximum	EDIA=Surf	ace Soil	RDA		<u>-</u>	
Analyte			EDIĂ=Surf Units	ace Soil Daily dose for child		1/5 RDA	Exceed RDA?	
	Frequency	Maximum detected concentration	Units	Daily đóše for child	RDA for	1/5 RDA		
Analyte	Frequency of Detection	Maximum detected	Units mg/kg	Daily dose	RDA for	1/5 RDA		
Analyte Aluminum Barium	Frequency of Detection 4/4	Maximum detected concentration 1.09E+04	Units	Daily dose for child 2.18E+00	RDA for	1/5 RDA		
Analyte Aluminum	Frequency of Detection 4/4 4/4	Maximum detected concentration 1.09E+04 8.40E+01	Units mg/kg mg/kg mg/kg	Daily dose for child 2.18E+00 1.68E-02	RDA for		RDA?	
Analyte Aluminum Barium Beryllium	Frequency of Detection 4/4 4/4 1/4	Maximum detected concentration 1.09E+04 8.40E+01 6.40E-01	Units mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.18E+00 1.68E-02 1.28E-04	RDA for child	1/5 RĎA 1.60E+02		
Analyte Aluminum Barium Beryllium Calcium	Frequency of Detection 4/4 4/4 1/4 2/2	Maximum detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05	Units mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.18E+00 1.68E-02 1.28E-04 5.46E+01	RDA for child		RDA?	
Analyte Aluminum Barium Beryllium Calcium Chromium Cobalt	Frequency of Detection 4/4 4/4 1/4 2/2 4/4	Maximum detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01	Units mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.18E+00 1.68E-02 1.28E-04 5.46E+01 5.30E-03	RDA for child		RDA?	
Analyte Aluminum Barium Beryllium Calcium Chromium Cobalt Copper	Frequency of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4	Maximum detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.18E+00 1.68E-02 1.28E-04 5.46E+01 5.30E-03 1.14E-03 1.46E-03	RDA for child		RDA?	
Analyte Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron	Frequency of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4	Maximum detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.18E+00 1.68E-02 1.28E-04 5.46E+01 5.30E-03 1.14E-03 1.46E-03 3.08E+00	RDA for child		RDA?	
Analyte Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium	Frequency of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4	Maximum detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.18E+00 1.68E-02 1.28E-04 5.46E+01 5.30E-03 1.14E-03 1.46E-03 3.08E+00 2.24E-03	RDA for child 8.00E+02	1.60E+02	RDA? No	
Analyte Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium Magnesium	Frequency of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4	Maximum detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01 1.70E+04	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.18E+00 1.68E-02 1.28E-04 5.46E+01 5.30E-03 1.14E-03 1.46E-03 3.08E+00 2.24E-03 3.40E+00	RDA for child		RDA?	
Analyte Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium Magnesium Manganese	Frequency of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4	Maximum detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01 1.70E+04 3.98E+02	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.18E+00 1.68E-02 1.28E-04 5.46E+01 5.30E-03 1.14E-03 1.46E-03 3.08E+00 2.24E-03 3.40E+00 7.96E-02	RDA for child 8.00E+02	1.60E+02	RDA? No	
Analyte Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium Magnesium Manganese Nickel	Frequency of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4	Maximum detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01 1.70E+04 3.98E+02 7.50E+00	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.18E+00 1.68E-02 1.28E-04 5.46E+01 5.30E-03 1.14E-03 1.46E-03 3.08E+00 2.24E-03 3.40E+00 7.96E-02 1.50E-03	RDA for child 8.00E+02 1.50E+02	1.60E+02 3.00E+01	RDA? No	
Analyte Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium Magnesium Manganese Nickel Potassium	Frequency of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4	Maximum detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01 1.70E+04 3.98E+02 7.50E+00 1.44E+03	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.18E+00 1.68E-02 1.28E-04 5.46E+01 5.30E-03 1.14E-03 1.46E-03 3.08E+00 2.24E-03 3.40E+00 7.96E-02 1.50E-03 2.88E-01	RDA for child 8.00E+02	1.60E+02	RDA? No	
Analyte Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium Magnesium Manganese Nickel Potassium Sodium	Frequency of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4	Maximum detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01 1.70E+04 3.98E+02 7.50E+00 1.44E+03 2.13E+02	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.18E+00 1.68E-02 1.28E-04 5.46E+01 5.30E-03 1.14E-03 1.46E-03 3.08E+00 2.24E-03 3.40E+00 7.96E-02 1.50E-03 2.88E-01 4.26E-02	RDA for child 8.00E+02 1.50E+02	1.60E+02 3.00E+01	RDA? No	
Analyte Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium Magnesium Manganese Nickel Potassium Sodium Strontium	Frequency of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4	Maximum detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01 1.70E+04 3.98E+02 7.50E+00 1.44E+03 2.13E+02 2.53E+02	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.18E+00 1.68E-02 1.28E-04 5.46E+01 5.30E-03 1.14E-03 1.46E-03 3.08E+00 2.24E-03 3.40E+00 7.96E-02 1.50E-03 2.88E-01 4.26E-02 5.06E-02	RDA for child 8.00E+02 1.50E+02	1.60E+02 3.00E+01	RDA? No	
Analyte Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium Magnesium Manganese Nickel Potassium Sodium Strontium Vanadium	Frequency of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4	Maximum detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01 1.70E+04 3.98E+02 7.50E+00 1.44E+03 2.13E+02 2.53E+02 3.15E+01	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.18E+00 1.68E-02 1.28E-04 5.46E+01 5.30E-03 1.14E-03 1.46E-03 3.08E+00 2.24E-03 3.40E+00 7.96E-02 1.50E-03 2.88E-01 4.26E-02 5.06E+02 6.30E-03	RDA for child 8.00E+02 1.50E+02	1.60E+02 3.00E+01	RDA? No	
Analyte Aluminum Barium Beryllium Calcium Chromium Cobalt Copper Iron Lithium Magnesium Manganese Nickel Potassium Sodium Strontium	Frequency of Detection 4/4 4/4 1/4 2/2 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4	Maximum detected concentration 1.09E+04 8.40E+01 6.40E-01 2.73E+05 2.65E+01 5.70E+00 7.31E+00 1.54E+04 1.12E+01 1.70E+04 3.98E+02 7.50E+00 1.44E+03 2.13E+02 2.53E+02	Units mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg mg/kg	Daily dose for child 2.18E+00 1.68E-02 1.28E-04 5.46E+01 5.30E-03 1.14E-03 1.46E-03 3.08E+00 2.24E-03 3.40E+00 7.96E-02 1.50E-03 2.88E-01 4.26E-02 5.06E-02	RDA for child 8.00E+02 1.50E+02	1.60E+02 3.00E+01	RDA? No	

Table 1.8. Comparison of maximum detected	concentrations of essential r	nutrients to	recommended	dietary	allowances
	for children				

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	Frequency of	Maximum detected		Daily dose	RDA for		Exceed
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?
Benz (a) anthracene	2/4	1,80E-01	mg/kg	3.60B-05			
Benzo (a) pyrene	2/4	2.50E-01	mg/kg	5.00B-05			
Benzo (b) fluoranthene	2/4	5.10E-02	mg/kg	1.02B-05			
Benzo (ghi) perylene	2/4	1.70B-01	mg/kg	3.402-05			
Chrysene	2/4	1.70B-01	mg/kg	3.40B-05			
Di-n-butylphthalate	1/4	7.70B-02	mg/kg	1.54E-05			
Di-n-octylphthalate	1/4	1.20E-01	mg/kg	2.40E-05			
Dibenz (a, h) anthracene	1/4	1.30B-01	mg/kg	2.60B-05		•	
Diethylphthalate	1/4	4.00E-01	mg/kg	8.00B-05			
Fluoranthene	2/4	3.10E-01	mg/kg	6.20E-05			
Indeno (1, 2, 3-cd) pyrene	2/4	1.60B-01	mg/kg	3.20B-05			
Pyrene	2/4 2/4	2.95B-01	mg/kg	5.90E-05			
bis (2-Ethylhexyl) phthalate	2/4	1.70E-01	mg/kg	3.40E-05			
Alpha activity	4/4	1.70E+01	pCi/g				
Beta activity	4/4	2.37E+01	pCi/g				

Analyte	Frequency of Detection	Maximum detected concentration	Units	Daily dose for child	~	RDA for child	1/5 RDA	Exceed RDA?
Trichloroethene	1/2	1.30E-02	mg/L	1.30B-02				
cis-1,2-Dichloroethene	1/2	2-30B-02	mg/L	2.30E-02				
Alpha activity	1/2	1.298+00	pCi/L					
Beta activity	2/2	4.80B+00	pC1/L					

LOC

CATION=SWMU 193B MEDIA=RGA Groundwater	CATION	SWMU	193B	MEDIA=RGA	Groundwater	
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Analyte	Frequency of Detection	Maximum detected concentration	Units	Daily dose for child	RDA for child	1/5 RDA	Exceed RDA?	
1.1-Dichloroethene	3/17	2.00E-02	mg/L	2.00E-02				
Acetone	1/2	3.30B-02	mg/L	3.30E-02				
Carbon Tetrachloride	1/5	5.50E-03	mg/L	5.50E-03				
Di-n-butylphthalate	2/10	1.30E-02	mg/L	1.30Ê-02				

		LOCA	TION-SWMU 193B ME (contin		roundwater			
	Analyte	Frequency of Detection	Maximum detected concentration	Units	Daily dose for child	RDA for child	1/5 RDA	Exceed RDA?
	Trichloroethene bis (2-Ethylhexyl)phthalate cis+1,2-Dichloroethene trans-1,2-Dichloroethene Alpha activity Beta activity Technetium-99	17/17 1/10 12/17 8/17 12/17 16/17 8/17	5.00E-01 1.80E-02 9.87E-02 8.10E-04 6.60E+02 5.85E+02 6.10E+01	ng/L ng/L ng/L pCi/L pCi/L pCi/L	5.00E~01 1.80E-02 9.87E-02 8.10E-04	•		
		Loca	TION=SWMU 193B ME	DIA-Subsu	rface Soil			
A-115	Analyte	Frequency of Detection	Maximum detected concentration	Units	Daily dose for child	RDA for child	1/5 RDA	Exceed RDA?
	Aluminum Barium Beryllium Chromium Cobalt Copper Iron Lithium Magnesium Manganese Nickel Potassium Sodium Strontium Vanadium Zinc Acetone Toluene Alpha activity Beta activity	4/4 2/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4 4/4	1.12E+04 8.42E+01 1.57E+00 8.87E+01 7.76E+00 7.43E+00 2.43E+04 7.72E+00 4.31E+03 2.22E+02 2.06E+01 6.86E+02 4.48E+02 9.39E+01 6.50E+01 5.57E+01 8.00E-02 1.00E-02 1.86E+01 2.29E+01	ng ng ng ng ng ng ng ng ng ng ng ng ng n	2.24E+00 1.68E-02 3.14E-04 1.77E-02 1.55E-03 1.49E-03 4.86E+00 1.54E-03 8.62E-01 4.44E-02 4.12E-03 1.37E-01 8.96E-02 1.88E-02 1.30E-02 1.30E-02 1.11E-02 1.60E-05 2.00E-06	1.50E+02	3.00 2 +01	No

	Frequency	Maximum			RDA		
	of	detected		Daily dose	for		Exceed
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?
Aluminum	2/2	1.08E+04	mg/kg	2.16E+00		·	
Barium	2/2	8.42E+01	mg/kg	1.68E-02			
Beryllium	1/2	1.57E+00	mg/kg	3.148-04			•
Chromium	2/2	8.87E+01	mg/kg	1.77E-02			
Cobalt	2/2	7.76E+00	mg/kg	1.55E-03			
Copper	2/2	7.43E+00	mg/kg	1.49E-03			
Iron	2/2	2.43B+04	mg/kg	4.86E+00	•		
Lithium	2/2	7.72E+00	mg/kg	1.548-03			
Magnesium	2/2	4.31E+03	mg/kg	8.62B-01			
Manganese	2/2	2.22E+02	mg/kg	4.448-02		•	
Nickel	1/2	2.06E+01	mg/kg	4.12B-03			
Potassium	2/2	6.86E+02	mg/kg	1.37E-01			
Sodium	2/2 .	2.49B+02	mg/kg	4.988-02			
Strontium	2/2	9.39E+01	mg/kg	1.88E-02			
Vanadium	2/2	6.50B+01	mg/kg	1.30B-02	• "		
Zinc	2/2	5.57E+01	mg/kg	1.11E-02			
Toluene	1/2	1.00E-02	mg/kg	2.00E-06			
Alpha activity	2/2	1.86E+01	pCi/g				
Beta activity	2/2	2.29E+01	pCi/g				

----- LOCATION=SWMU 193C MEDIA=McNairy Groundwater -----

Analyte	Frequency of Detection	Maximum detected concentration	Units	Daily dose for child	RDA for child	1/5 RDA	Exceed RDA?
Aluminum	4/4	9.04E+01	mg/L	9.04E+01			
Antimony	5/5	2.50E-01	mg/L	2.50E-01			
Arsenic	5/5	3.60E-02	mg/L	3.60E-02		•	
Barium	5/5	6.70E-01	mg/L	6.70E-01			
Beryllium	5/5	2.50E-02	mg/L	2.50E-02			
Cadmium	5/5	1.00E-01	mg/L	1.00E-01			
Calcium	5/5	4.10E+01	mg/L	4.10E+01	8.00E+02	1.60E+02	No
Chloride	5/5	1.68E+01	mg/L	1.68E+01	6.00E+02	1.20E+02	No
Chromium	3/3	2.328-01	mg/L	2.32E-01		_ ·	
Cobalt	5/5	1.21B-01	mg/L	1.21E-01			
Copper	5/5	1.63E-01	mg/L	1.63E-01	1.00E+00	2.00E-01	Nö
Fluoride	4/4	2.80E-01	mg/L	2.808-01	1.50E+00	3.00E-01	No
Iron	5/5	1.79E+02	mg/L	1.79E+02	1.00E+01	2.00E+00	Yes

A-1:16

•	Frequency	Maximum	•		RDA		
· · ·	of	detected	•	Daily dose	for	•	Exceed
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?
Lead	1/1	2.50E-01	mg/L	2.50E-01		•	
Magnesium	5/5	2.16E+01	mg/L	2.16E+01	1.70E+02	3.40E+01	No
Manganese	5/5	3.91B+00	mg/L	3.91 E +00			
Mercury	1/1 '	2.00E-04	mg/L	2.00E-04			•
Molybdenum	4/4	1.00E-01	mg/L	1.00E-01	5.00B-02	1.00E-02	Yes
Nickel	5/5	1.09B-01	mg/L	1.09E-01			
Nitrate as Nitrogen	5/5	1.00E+00	mg/L	1.00E+00			
Potassium	5/5	1.01B+02	mg/L	1.01B+02	1.60E+03	3.20E+02	No
Selenium	3/3	5.00E-03	mg/L	5.00E-03			
Silica	5/5	1.80E+01	mg/L	1.80E+01			
Silver	3/3	6.00E-02	mg/L	6.00E-02			
Sodium	5/5	2.63E+01	mg/L	2.63E+01			
Tetraoxo-sulfate(1-)	5/5	1.30E+01	mg/L	1.30E+01		•	
Thallium	2/2	1.23E-01	mĝ/L	1.23E-01		,	
Uranium	9/9	1.80E-02	mg/L	1.80E-02			
Vanadium	2/2	8.36E-01	mg/L	8.36E-01			
Zinc	5/5	5.64E-01	mg/L	5.64E-01			
1,1,1-Trichloroethane	4/4	5.00E-03	mq/L	5.00E-03			
1,1,2-Trichloroethane	4/4	5.00E-03	mg/L	5.00E-03			
1,1-Dichloroethane	• 4/4	5.00B-03	mg/L	5.00E-03			
1,1-Dichloroethene	4/4	5.00E-03	mg/L	5.00B-03			
1,2-Dichloroethane	4/4	5.00E-03	mg/L	5.00E-03			
Benzene	4/4	5.00E-03	mg/L	5.00E-03			
Bromodichloromethane	4/4	5.00E+03	mg/L	5.00E-03			
Carbon Tetrachloride	4/4	5.00B-03	mq/L	5.00E-03			
Chloroform	4/4	5.00E-03	mg/L	5.00E-03			
Ethylbenzene	4/4	5.00E-03	mg/L	5.00E-03			
Polychlorinated biphenyl	1/1	1.00E-04	mg/L	1.00E-04			
Tetrachloroethene	4/4	5.00B-03	mg/L	5.00Ē-03			
Toluene	4/4	5.00E-03	mg/L	5.00B-03			
Trichloroethene	12/12	2.00E-03	mg/L	2.00B-03			
Vinyl Chloride	4/4	. 1.00E-02	mg/L	1.00B-02			
Xylene	4/4	1.00B-02	mg/L	1.00B-02			
cis-1,2-Dichloroethene	4/4	5.00E-03	mg/L	5.00E-03			
trans-1,2-Dichloroethene	4/4	5.00E-03	mg/L	5.00E-03			
Alpha activity	12/12	1.07E+02	pCi/L	3.002-03			
	12/12	2.36E+02					
Beta activity	2/2	1.57E+02	pCi/L				
Radon-222			pCi/L	•			
Technetium-99	13/13	2.70B+01	pCi/L				

	Frequency	Maximum detected		Daily dose	RDA for		Exceed
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?
1,2-Dichloroethene Trichloroethene	1/2 1/2	5.62E-01 1.62E-01	mg/L mg/L	5.628-01 1.628-01			
	LOCA	TION-SWMU 193C ME	DIA=Subsu	rface Soil			
	Frequency	Maximum			RDA		
	of	detected	•	Daily dose	for		Exceed
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?
Aluminum	20/20	1.37E+04	mg/kg	2.74E+00	_		
Arsenic	5/20	6.57E+00	mg/kg	1.31E-03			
Barium	1/1	1.425+02	mg/kg	2.84E-02			
Beryllium	10/20	9.80E-01	mg/kg	1.96E-04			
Boron	1/20	1.00B+02	mg/kg	2.00E-02			
Cadmium	3/59	5.00E+00	mg/kg	1.00E-03			
Calcium	18/20	4.00E+05.	mg/kg	8.00E+01	8.00E+02	1.60E+02	No
Chromium	59/61	8.30E+01	mg/kg	1.66E-02			
Cobalt	17/20	8.61E+01	mg/kg	1.72E-02			
Copper	16/20	2.82E+01	mg/kg	5.64E-03	1.00E+00	2.00E-01	No
Iron	6/6	3.00E+04	mg/kg	6.00E+00	1.00E+01	2.00E+00	Yes
Lead	42/61	6.77E+01	mg/kg	1.35E-02			
Lithium	17/20	1.25E+01	mg/kg	2.50E-03			
Magnesium	20/20	1.458+04	mg/kg	2.90E+00	1.50E+02	3.00E+01	No
Manganese	20/20	2.27E+03	mg/kg	4.54E-01			
Nickel	13/20	2.15E+01	mg/kg	4.30E-03			
Potassium	20/20	1.57E+03	mg/kg	3.14E-01	1.60E+03	3.20E+02	No
Sodium	15/20	4.448+02	mg/kg	8.88E-02			
Strontium	20/20	3.91E+02	mg/kg	7.82E-02			
Vanadium	20/20	4.42E+01	mg/kg	8.84E-03			
Zinc	19/20	9.25E+01	mg/kg	1.85E-02			
Xylene	1/20	1.00E-02	mg/kg	2.00E-06			
Alpha activity	53/53	4.00E+00	pCi/g				
Beta activity	53/53	1.00E+01	pCi/g				

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·	Frequency	Maximum			RDA		
	of	detected		Daily dose	for		Exceed
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?
Aluminum	5/5	3.36E+03	mg/kg	6.72E-01			
Boron	1/5	1.00E+02	mg/kg	2.00E-02			
Calcium	. 5/5	4.00E+05	mg/kg	8.00E+01	8.00E+02	1.60E+02	No
Chromium	3/5	1.20E+01	mg/kg	2.40E-03			
Cobalt	2/5	2.14E+00	mg/kg	4.28E-04		· .	
Copper	2/5	2.82E+01	mg/kg	5.64E-03	1.00E+00	2.00E-01	No
Lead	1/5	6.77E+01	mg/kg	1.35E-02		•	
Lithium	3/5	1.25B+01	mg/kg	2.50E-03			
lagnesium	5/5	1.45E+04	mg/kg	2.90E+00	1.50E+02	3.00E+01	No
langanese	5/5	1.98E+02	mg/kg	3,96E-02			
Nickel	• 1/5	6.43E+00	mg/kg	1.29E-03			
Potassium	5/5	1.57E+03	mg/kg	3.14B-01	1.60E+03	3.20E+02	No
Sodium	4/5	3.10E+02	mg/kg	6.20B-02			
Strontium	5/5	3.91E+02	mg/kg	7.82E-02			
Vanadium	5/5	6.70E+00	mg/kg	1.34E-03			
linc	5/5	9.25E+01	mg/kg	1.85E-02			
	LOCA	TION=SWMU 194 MEI	IA=Subsu	face Soil			
	Frequency	Maximum			RDA		
	of	detected		Daily dose	for		Excee
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?
Aluminum	12/12	1.45E+04	ng/kg	2.90E+00			•
Arsenic	1/12	6.73E+00	mg/kg	1.35E-03			
Tourism	/	1 200.00		0.000.00			

Barium 12/12 1.39E+02 2.78E-02 mg/kg Beryllium 6/12 4.805+00 mg/kg 9.60E-04 Cadmium 1/35 8.55E+00 mg/kg 1.71E-03 Calcium 12/12 6.81E+03 mg/kg 1.36E+00 8.00E+02 1.60E+02 No Chromium 35/35 1.03E+02 mg/kg 2.06B-02 Cobalt 12/12 mg/kg 1.89E-03 9.46E+00 Copper 12/12 1.67E+01 mg/kg 3.34E-03 12/12 mg/kg Iron 2.00E+04 4.00E+00 20/35 Lead 3.60E+02 mg/kg 7.208-02 Lithium 12/12 1.80E-03 9.00E+00 mg/kg Magnesium 12/12 2.34E+03 mg/kg 4.688-01 1.50E+02 3.00E+01 No Manganese 12/12 4.67E+02 mg/kg 9.34E-02 mg/kg Nickel 8/12 1.37E+01 2.74E-03

6.32E+02

mg/kg

1.26E-01

12/12

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Potassium

		(contin	/				
	Frequency	Maximum			RDA		
-	of	detected		Daily dose	for		Exceed
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?
Sodium	8/12	3.69E+02	mg/kg	7.38E-02			
Strontium	12/12	2.60E+01	mg/kg	5.20E-03			-
Vanadium	12/12	2.582+01	mg/kg	5.16E-03			
Zinc	11/12	6.76B+01	mg/kg	1.35E-02			
Ethylbenzene	1/19	1.50E-02	mg/kg	3.00E-06			
Alpha activity	23/23	2.502+00	pCi/g				
Beta activity	23/23	7.002+00	pC1/g				• •
	LOCATI	ON-SWMU 99A MEDIA	-McNairy	Groundwater			
	Frequency	Maximum			RDA		
	of	detected		Daily dose	for		Exceed
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?
1,1,1-Trichloroethane	1/4	1.20E-03	mg/L	1.20E-03			
1,1-Dichloroethene	1/4	2.298-02	mg/L	2.29E-02			
Carbon Tetrachloride	1/4	2.80E-03	mg/L	2.80E-03 ·			
Trichloroethene	3/4	5.198-01	mg/L	5.19B-01			
cis-1,2-Dichloroethene	2/4	1.158-01	mg/L	1.15E-01			
Alpha activity	2/2	2.90E+00	pCi/L				
Beta activity	2/2	3.50E+01	pCi/L .	•			
Technetium-99	2/2	1.908+01	pCi/L				
	LOCA	tion-swmu 99a mei	DIA=RGA Gr	oundwater			
	Frequency	Maximm			RDA		
	of	detected		Daily dose	for		Exceed
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?
Aluminum	16/35	6.59E+02	mg/L	6.59E+02			
Arsenic	4/27	1.008-02	mg/L	1.002-02			· · · · · · · · · · · · · · · · · · ·
Barium	39/39	3.30E+00	mg/L	3.30E+00			
Beryllium	8/35	1.00B-01	mg/L	1.00E-01			
	39/39	1.20E+02	mg/L	1.20E+02	8.00E+02	1.60E+02	No
Calcium						210001102	***
Calcium Chloride		1.20E+02	mg/L	1.20E+02	6.00R+02	1.208+02	Vec
	9/9 11/39	1.20E+02 1.78E+00	mg/L mg/L	1.20E+02 1.78E+00	6.00B+02	1.20E+02	Үев

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	Frequency	Maximum			RDA		
	of	detected		Daily dose	for		Exceed
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?
Copper	9/35	6.40E-01	mg/L	6.40E-01	1.00E+00	2.00E-01	Yes
Fluoride	8/8	2.00E-01	mg/L	2.00E-01	1.50E+00	3.00E-01	NO
Iron	31/39	1.20E+03	mg/L	1.20E+03	1.00E+01	2.00E+00	Yes
Lead	6/29	4.10E-01	mg/L	4.10E-01		,	
Lithium	6/23	1.70E-01	mg/L	1.70E-01			•
Magnesium	39/39	4.97E+01	mg/L	4.97E+01	1.70E+02	3.40E+01	Yes
Manganese	37/39	4.60E+00	mg/L	4.60E+00			
Mercury	5/25	2.00E-02	mg/L	2.00E-02			•
Nickel	16/39	9.10 <u>8-01</u>	mg/L	9.10E-01			
Nitrate as Nitrogen	7/9	2.10E+00	mg/L	2.10E+00			
Potassium	24/39	2.17E+01	mg/L	2.17E+01	1.60E+03	3.20E+02	NÓ
Silica	9/9	2.50E+01	mg/L	2.50E+01			
Sodium	39/39	7.24E+01	mg/L	7.24E+01			
Strontium	30/30	4.708-01	mg/L	4.70E-01		•	
Sulfate	2/2	1.928+01	mg/L	1.92E+01			
Tetraoxo-sulfate(1-)	7/7	2.20E+01	mg/L	2.20E+01		• •	
Vanadium	10/28	2.158+00	mg/L	2.15E+00			
Zinc	10/35	2.55B+00	mg/L	2.55E+00		1	
1,1-Dichloroethene	7/33	6.50B-02	mg/L	6.50B-02			
Trichloroethene	41/43	2.37E+00	mg/L	2.37E+00	•		
bis (2-Ethylhexyl) phthalate	5/10	1.608-02	mg/L	1.60E-02	•		
cis-1,2-Dichloroethene	10/33	3.48E-02	mg/L	3.48E-02			
trans-1,2-Dichloroethene	3/33	6.00E-04	mg/L	6.00E-04	1		
Alpha activity	33/39	5.38E+01	pCi/L				
Beta activity	39/39	1.37E+02	pCi/L	-			
Radon-222	4/4	6.75E+02	pCi/L				
Technetium-99	34/40	1.39E+02	pCi/L				

- LOCATION-SWMU 99A MEDIA-Subsurface Soil -----

Analyte	Frequency of Detection	Maximum detected concentration	Units	Daily dose for child	RDA for child	1/5 RDA	Exceed RDA?
Aluminum Antimony Arsenic Barium	22/22 5/22 11/22 22/22	1.41E+04 2.90E+00 8.55E+00 2.47E+03	mg/kg mg/kg mg/kg mg/kg	2.828+00 5.808-04 1.71E-03 4.94E-01	•		

-- LOCATION=SWMU 99A MEDIA=Subsurface Soil ----- (continued)

	Frequency	Maximum detected		Daily dose	RDA for		Exceed
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?
Beryllium	11/22	8.90E-01	mg/kg	1.78E-04			
Cadmium	5/22	8.30E-01	mg/kg	1.66E-04			
Calcium	20/20	2.87E+05	mg/kg	5.74E+01	8.00E+02	1.60E+02	NÖ
Chromium	22/22	4.57E+01	mg/kg	9.14E-03			
Cobalt	20/22	1.19E+01	mg/kg	2.38E-03			
Copper	21/22	1.64E+01	mg/kg	3.28E-03			
Cyanide	2/16	5.408-01	mg/kg	1.08E-04			
Iron	22/22	2.33E+04	ng/kg	4.66E+00			
Lead	6/22	4.73E+01	mg/kg	9.46E-03			
Lithium	17/17	1.29E+01	mg/kg	2.58E-03			
Magnesium	22/22	2.73E+04	mg/kg	5.46E+00	1.508+02	3.00E+01	No
Manganese	22/22	1.46E+03	mg/kg	2.92E-01			
Mercury	5/22	1.20E-01	mg/kg	2.40E-05			
Nickel	17/22	2.58E+01	mg/kg	5.16E-03			
Potassium	22/22	1.12Ë+03	mg/kg	2.24B-01	1.60E+03	3.20E+02	No
Selenium	5/20	3.20E-01	mg/kg	6.40E-05			
Silver	5/22	7.10E-01	mg/kg	1.42E-04			
Sodium	14/22	3,936+02	mg/kg	7.86E-02	:		
Strontium	17/17	5.14E+02	mg/kg	1.03E-01			
Thallium	5/22	5.90E-01	mg/kg	1.18E-04			
Vanadium	22/22	3.55E+01	πg/kg	7.10E-03			
Zinc	21/22	1.63E+02	mg/kg	3.26E-02			
1,1,1-Trichloroethane	5/8	6.00E-03	mg/kg	1.20E-06			
1,1,2,2-Tetrachloroethane	5/8	6.00E-03	mg/kg	1.20E-06			
1,1,2-Trichloroethane	5/8	6.00E+03	mg/kg	1.20E-06			
1,1-Dichloroethane	5/8	6.00E-03	mg/kg	1.20E-06			
1,1-Dichloroethene	5/10	6.00Ē-03	mg/kg	1.20E-06			
1,2,4-Trichlorobenzene	5/22	4.10E-01	mg/kg	8.20E-05			
1,2-Dichlorobenzene	5/22	4.10E-01	mg/kg	8.20E-05			
1,2-Dichloroethane	5/8	6,00E-03	mg/kg	1.20E-06			
1,2-Dichloroethene	5/5	6.00E-03	mg/kg	1.20E-06			
1,2-Dichloropropane	5/8	6.00E-03	mg/kg	1.20E-06			
1,3-Dichlorobenzene	5/22	4.10E-01	mg/kg	8.20E-05			
1,4-Dichlorobenzene	5/22	4.10E-01	mg/kg	8.20E-05			
2,4,5-Trichlorophenol	. 5/22	2.10E+00	mg/kg	4.20E-04			
2,4,6-Trichlorophenol	5/22	4.10E-01	ng/kg	8.20E-05			
2,4-Dichlorophenol	5/22	4.10E-01	mg/kg	8.20E-05			
2,4-Dimethylphenol	5/22	4.10E-01	mg/kg	8.20E-05			
2,4=Dinitrophenol	5/6	2.10E+00	mg/kg	4.20E-04			
s's-nutcrobuenor	2/6	3.102+00	mg/kg	4.∡UE-U4			

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		TION-SWMU 99A MEL (contin					
	Frequency	Maximum detected		Daily dose	RDA		Direct
Analytë ,	Detection	concentration	Units	for child	child	1/5 RDA	Exceed RDA?
2,4-Dinitrotoluene	5/22	4.10E-01	mğ/kg	8.208-05			
2,6-Dinitrotoluene	5/22	4.10E-01	mg/kg	8.208-05			
2-Butanone	5/8	1.20E-02	mg/kg	2.40E-06			
2-Chloronaphthalene	5/22	4.10E-01	mg/kg	8.20E-05			
2-Chlorophenol	5/22	4.10B-01	mg/kg	8.20E-05			
2-Hexanone	5/8	1.20E-02	mg/kg	2.40E-06			
2-Methyl-4,6-dinitrophenol	5/22	2.108+00	mg/kg	4.20E-04			
2-Methylnaphthalene	5/22	4.10E-01	mg/kg	8.20E-05			
2-Methylphenol	5/22	4.10E-01	mg/kg	8.20E-05			
2-Nitroaniline	5/22	2.10E+00	mg/kg	4.20B-04			
2-Nitrophenol	5/22	4.10B-01	mg/kg	8.20E-05			
3,3'-Dichlorobenzidine	5/22	8.20E-01	mg/kg	1.64E-04			
3-Nitroaniline	5/22	2.10E+00	mg/kg	4.20E-04			
4,4'-DDD	2/2	3.50E-02	mg/kg	7.00B-06			
4, 4' - DDE	2/2	3.50E-02	mg/kg	7.00B-06			
4,4'-DDT	2/2	3.50E-02	mg/kg	7.00E-06			
4-Bromophenyl phenyl ether	5/22	4.10E-01	mg/kg	8.20E-05			
4-Chloro-3-methylphenol	5/22	4.10B-01	mg/kg	8.20E-05			
4-Chloroaniline	5/22	4.10E-01	mg/kg	8.20E-05			•
4-Chlorophenyl phenyl ether	5/22	4.10E-01	mg/kg	8.20B-05			
4-Methyl-2-pentanone	5/8	1.20E-02	mg/kg	2.40E-06			
4-Methylphenol	5/22	4.108-01	mg/kg	8.20E-05			
4-Nitroaniline	5/22	2.10E+00	mg/kg	4.208-04			
4-Nitrophenol	5/22	2.10E+00	mg/kg	4.20E-04			
Acenaphthene	7/22	4.10E-01	mg/kg	8.208-05			
Acenaphthylene	6/22	6.10E-01	mg/kg	1.22E-04	•	•	
Acetone	5/8	5.30E-02	mg/kg	1.06E-05			
Aldrin	2/2	1.70E-02	mg/kg	3.40E-06			
Anthracene	7/22	7.50B-01	mg/kg	1.50E-04			
Benz (a) anthracene	8/22	1.70E+00	mg/kg	3.40E-04			
Benzene	5/8	6.00E-03	mg/kg	1.208-06			
Benzo (a) pyrene	7/22	2.10E+00	mg/kg	4.20E-04			
Benzo (b) fluoranthene	11/22	5.70E+00	mg/kg	1.14E-03			
Benzo (ghi) perylene	7/22	1.18E+00	mg/kg	2.36E-04			
Benzo (k) fluoranthene	8/22	7.90E-01	mg/kg	1.58E-04			
Benzoic Acid	5/5	2.10E+00 .	mg/kg	4.20E-04			
Benzyl Alcohol	5/5	4.10E-01	mg/kg	8.20E-05			
Bromodichloromethane	5/8	4.10E-01 6.00E-03	mg/kg				
	5/8	6.00E-03		1.20E-06			
Bromoform	5/8	6.008-03	mg/kg	1.20E-06			

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---- LOCATION-SWMU 99A MEDIA-Subsurface Soil ---(continued)

Analyte	Frequency of Detection	Maximum detected concentration	Units	Daily dose for child	RDA for child	1/5 RDA	Exceed RDA?
Bromomethane	5/8	1.20E-02	mg/kg	2.40E-06			
Butyl benzyl phthalate	5/6	4.10E-01	mg/kg	8.20E-05			
Carbon Disulfide	5/8	6.00E-03	mg/kg	1.20E-06			
Carbon Tetrachloride	5/8	6.00E-03	ng/kg	1.20E-06			
Chlorobenzene	5/8	6.00B-03	mg/kg	1.20E-06			
Chloroethane	5/8	1.20B-02	mg/kg	2.40E-06			
Chloroform	5/8	6.00E-03	mg/kg	1.20E-06			
Chloromethane	5/8	1.20E-02	ng/kg	2.40E-06	•		
Chrysene	7/22	2.10E+00	mg/kg	4.208-04			
Di-n-butylphthalate	5/22	4.10E-01	mg/kg	8.202-05			•
Di-n-octylphthalate	5/22	4.10E-01	mg/kg	8.208-05			.1
Dibenz (a, h) anthracene	6/22	4.80E-01	mg/kg	9.608-05			
Dibenzofuran	6/22	4.10E-01	mg/kg	8.208-05			
Dibromochloromethane	5/8	6.00E-03	mg/kg	1.208-06			
Dieldrin	2/2	3.50E-02	mg/kg	7.00E-06			
Diethylphthäläte	5/22	4.10B-01	mg/kg	8.20E-05			
Dimethylphthalate	5/22	4.10E-01	mg/kg	8.20E-05		1 ·	•
Endosulfan I	2/2	1.70E-02	mg/kg	3.40E-06			
Endosulfan II	2/2	3.50E-02	mg/kg	7.00B-06			
Endosulfan Sulfate	2/2	3.50E-02	mg/kg	7.00E-06			
Endrin	2/2	3.50E-02	mg/kg	7.00E-06			
Endrin Ketone	2/2	3.50E-02	mg/kg	7.00E-06			
Ethylbenzene	5/8	6.00E-03	mg/kg	1.20E-06			
Fluoranthene	9/22	2.66E+00	mg/kg	5.32E-04			
Fluorene	6/22	4.10E-01	mg/kg	8.20E-05			
Heptachlor	2/2	1.702-02	mg/kg	3.40E-06			
Heptachlor Epoxide	2/2	1.70E-02	mg/kg	3.40E-06			
Hexachlorobenzene	5/22	4.10E-01	mg/kg	8.20E-05			·
Hexachlorobutadiene	5/22	4.102-01	mg/kg	8.20E-05			
Hexachlorocyclopentadiene	5/22	4.102-01	mg/kg	8.20E-05			
Hexachloroethane	5/22	4.10E-01	mg/kg	8.20E-05			
Indeno (1, 2, 3-cd) pyrene	7/22	1.05E+00	mg/kg	2.10E-04			
Isophorone	5/22 ·	4.10E-01	mg/kg	8.20E-05			
Methoxychlor	2/2	1.70E-01	mg/kg	3.40E-05			
Methylene Chloride	5/8	8.00E-03	mg/kg	1.60E-06			
N-Nitroso=di-n-propylamine	5/22	4.10E-01	mg/kg	8.20E-05			
N-Nitrosodiphenylamine	5/22	4.10B-01	mg/kg	8.20E-05			
Naphthalene	5/22	4.10E-01	mg/kg	8.20E-05			
Nitrobenzene	5/22	4.10E-01	mg/kg	8.20E-05			

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	Frequency	Maximum		÷	RDA		
	of	detected	÷	Daily dose	for		Exceed
Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?
PCB-1016	3/23	1.87E+00	mg/kg	3.74Ē-04			
PCB-1221	2/23	1.70E-01	mg/kg	3.40E-05			
PCB-1232	2/23	1.70E-01	mg/kg	3.408-05		•	
PCB-1242	2/23	1.70E-01	mg/kg	3.408-05			
PCB-1248	2/23	1.70E-01	mg/kg	3.40E-05			
PCB-1254	3/23	3.50E-01	mg/kg	7.00E-05			
PCB-1260	7/23	6.31E-01	mg/kg	1.26E-04			
Pentachlorophenol	5/22	2.10E+00	mg/kg	4.20E-04			
Phenanthrene	7/22	1.63E+00	mg/kg	3.26E-04			
Phenol	5/22	4.10E-01	mg/kg	8.208-05			
Pyrene	8/22	2.70E+00	mg/kg	5.40E-04			
Styrene	5/8	6.00E-03	mg/kg	1.20E-06			
Tetrachloroethene	5/8	6.00E-03	mg/kg	1.208-06			•
Toluene	5/8	6.00E-03	mg/kg	1.208-06			
Toxaphene	2/2	3.50E-01	mg/kg	-7.00E-05			
Trichloroethene	6/10	6.00E-03	mg/kg	1.208-06			
Vinyl Acetate	5/5	1.20E-02	mg/kg	2.408-06			
Vinyl Chloride	5/10	1.20E-02					
Xylene	5/5	6.00E-03	mg/kg	2.40E-06		•	
alpha-BHC	2/2	1.70E-02	mg/kg	1.20E-06			
alpha-Chlordane			mg/kg	3.40E-06		•	
beta-BHC	2/2	1.70E-01	mg/kg	3.40E-05			
	2/2	1.70E-02	mg/kg	3.40E-06			
bis (2-Chloroethoxy) methane	5/22	4.10E-01	mg/kg	8.20E-05			
bis (2-Chloroethyl) ether	5/22	4.10E-01	mg/kg	8.20E-05			
bis (2-Chloroisopropyl) ether	5/22	4.10E-01	mg/kg	8.20E-05			
bis (2-Ethylhexyl) phthalate	5/22	3.60E-01	mg/kg	7.208-05			
cis-1,3-Dichloropropene	5/8	6.00E-03	mg/kg	1.20E-06			
delta-BHC	2/2	1.70E-02	mg/kg	3.40E-06			
gamma-BHC(Lindane)	2/2	1.70E-02	mg/kg	3.40E-06			
gamma-Chlordane	2/2	1.70E-01	mg/kg	3.40E-05			
trans-1,3-Dichloropropene	5/8	6.00E-03	mg/kg	1.20E-06			
Alpha activity	20/21	1.42E+02	pCi/g				
Beta activity	21/21	2.73E+03	pCi/g				
Cesium-137	3/21	1.90E+00	pCi/g			1	
Neptunium-237	4/4	1.28E+01	pCi/g			•	
Plutonium-239	3/3	6.00E-03	pCi/g				
Technetium-99	6/23	2.65E+03	pCi/g				
Thorium-230	3/3	6.70E-01	pCi/g				
Thorium-234	1/21	5.30E+01	pCi/g				

Prequency of ot ocneantration Maximum detected for soften child RDA for child Exceed for child Exceed for child Exceed for child Exceed for child Exceed for child Exceed for for child Exceed for for child Exceed for for child Exceed for for for for for for for for for for		LOCA	TION=SWMU 99A MED (contin		face Soil				
J. T. M. J. C. Marting Join 1.648-01 pC1/g Dranium-235 3/24 4.108-02 pC1/g Dranium-235 4/4 5.17E+01 pC1/g Dranium-238 4/4 5.17E+01 pC1/g Dranium-238 4/4 5.17E+01 pC1/g Dranium-238 4/4 5.17E+01 pC1/g Dranium-238 4/4 5.17E+01 pC1/g Dranium-238 4/4 5.17E+01 pC1/g Dranium-238 4/4 5.17E+01 pC1/g Dranium-238 4/4 5.17E+01 pC1/g Aluminum 13/13 1.29E+04 mg/kg 1.60E+02 Arsenic 6/13 8.55E+00 mg/kg 1.71E=03 Barium 13/13 2.47E+03 mg/kg 1.60E+02 No Chronium 13/13 2.47E+03 mg/kg 1.60E+02 No Copper 12/13 1.22E+01 mg/kg 2.46E+03 1.50E+02 3.00E+01 No Magnasium <td< th=""><th></th><th>of</th><th>detected</th><th>Trátta</th><th></th><th>for</th><th>1/5 PDA</th><th></th><th>·.</th></td<>		of	detected	Trátta		for	1/5 PDA		·.
Diranium-235 j/24 4.108-02 pCi/g Uranium-238 4/4 5.178+01 pCi/g Prequency Maximum Daily dose for Exceed Analyte Detection condetected Daily dose for Exceed Analyte Detection concurration Units for for Exceed Aluminum 13/13 1.292+04 mg/kg 2.58E+00 Adds-01 child 1/5 RDA RDA? Aluminum 13/13 2.478+03 mg/kg 1.712-03 Barium 13/13 2.478+03 8.00E+02 1.60E+02 No Calcium 11/11 2.678+00 mg/kg 1.48E+03 Coper 1.60E+02 No Coper 12/13 1.22E+01 mg/kg 2.58E+03 1.50E+02 No Itrium 13/13 1.328+04 mg/kg 1.50E+02 3.00E+01 No Marganesium 13/13 1.238+04 mg/kg 1.46E+03 1.50E+02 3.00E+01	Analyte	Detection	CONCENTIATION	UNICS	IOI CHIIG	CHILL'S	I/J IOR		
Dirarium-235 j/24 4.10E-02 pCi/g Uranium-238 4/4 5.17E+01 pCi/g Prequency Maximum Daily dose for of detected Daily dose for Exceed Analyte Detection contentration Units for child thild 1/5 RDA RDA? Aluminum 13/13 1.29E+04 mg/kg 2.58E+00 Adds-01 child 1/5 RDA RDA? Aluminum 13/13 1.29E+04 mg/kg 1.71E-03 Barium 13/13 2.47E+03 B.00E+01 8.00E+02 No Calcium 11/11 2.07E+05 mg/kg 1.44E+03 Coper 1.60E+02 No Cobalt 11/13 9.67E+00 mg/kg 2.44E+03 8.00E+02 No S.00E+01 No Lithium 13/13 1.29E+01 mg/kg 2.46E+00 1.50E+02 3.00E+01 No Magnaesum 13/13 1.29E+01 mg/kg 2.42E+03	thrandum-224	A/A	1.64B+01	pCi/g					
Uranium-238 4/4 5.17E+01 pCl/g LOCATION-SWMU 99A MEDIA-Surface Soil Description Of detected Daily dose for Exceed Analyte Detection concentration Units for child child 1/5 RDA RDA Armenic 6/13 1.29E+04 mg/kg 2.58E+00 Armenic 6/13 8.55E+00 mg/kg 1.71E+03 Barium 13/13 2.47E+03 mg/kg 1.71E+03 8.00E+02 1.60E+02 No Calcium 13/13 2.47E+05 mg/kg 5.14E+01 8.00E+02 1.60E+02 No Cobalt 13/13 2.57E+05 mg/kg 1.32E-03 Coper 12/13 1.22E+01 mg/kg 1.50E+02 No Coperer 12/13 1.22E+01 mg/kg 7.4EE+03 1.50E+02 3.00E+01 No Magganese 13/13 2.73E+04 mg/kg 7.4EE+03 1.50E+02 3.00E+01 No Magganese 13/									
Interpretation Interpr									
Frequency of Maximum detected concentration Daily dose for for child RDA for child Exceed l/5 RDA Aluminum 13/13 1.29E404 mg/kg 2.58E+00 child 1/5 RDA RDA? Aumainum 13/13 1.29E404 mg/kg 2.58E+00 child 1/5 RDA RDA? Arsenic 6/13 6.55E+00 mg/kg 1.71E-03 RDA? Barium 13/13 2.47E+03 mg/kg 1.74E-03		LO	CATION-SWMU 99A M	EDIA-Surf	ace Soil				
of detected pairy dose for Exceed Analyte Petection concentration Units for child child 1/5 RDA RDA? Aluminum 13/13 1.292+04 mg/kg 2.56E+00 child 1/5 RDA RDA? Arsenic 6/13 8.55E+00 mg/kg 1.71E-03 state									
Analyte Detection concentration Units for child child 1/5 RDA RDA? Aluminum 13/13 1.292+04 mg/kg 2.582+00 RDA? Armenic 6/13 8.552+00 mg/kg 1.712-03 RDA? Barium 13/13 2.472+03 mg/kg 4.942-01 RDA? Calcium 11/11 2.672+03 mg/kg 1.782-04 8.002+02 1.602+02 No Chromium 13/13 4.572+01 mg/kg 9.142+03 8.002+02 1.602+02 No Cobalt 11/13 9.672+00 mg/kg 1.932+03 8.002+02 1.602+02 No Cobalt 11/13 9.672+00 mg/kg 2.442+03 1.932+03 1.502+02 3.002+01 No Ithium 13/13 1.222+01 mg/kg 2.452+03 1.502+02 3.002+01 No Margarese 13/13 3.122+01 mg/kg 5.462+00 1.502+02 3.002+01 <td></td> <td></td> <td></td> <td></td> <td>Daily dose</td> <td></td> <td></td> <td>Exceed</td> <td></td>					Daily dose			Exceed	
Aluminum 13/13 1.29E+04 mg/kg 2.58E+00 Arsenic 6/13 B.55E+00 mg/kg 1.71E-03 Barium 13/13 2.47E+03 mg/kg 1.71E-03 Barium 13/13 2.47E+03 mg/kg 1.78E-04 Calcium 11/11 2.87E+05 mg/kg 5.74E+01 8.00E+02 1.60E+02 No Chromium 13/13 4.57E+01 mg/kg 5.14E+03 8.00E+02 1.60E+02 No Cobalt 11/13 9.67E+00 mg/kg 5.44E+03 8.00E+02 1.60E+02 No Copper 12/13 1.22E+01 mg/kg 2.44E-03 1.50E+02 3.00E+01 No Magnesium 13/13 2.33E+04 mg/kg 5.46E+00 1.50E+02 3.00E+01 No Manganese 13/13 3.23E+04 mg/kg 5.46E+00 1.50E+02 3.00E+01 No Manganese 13/13 3.67E+02 mg/kg 7.42E+02 3.00E+01 No Sodium 6/13 3.66E+00 1.50E+04 1.50E+04 Sodium </td <td>Analvře</td> <td></td> <td></td> <td>Units</td> <td></td> <td></td> <td>1/5 RDA</td> <td></td> <td></td>	Analvře			Units			1/5 RDA		
Arsenic 6/13 8.552+00 mg/kg 1.712-03 Barium 13/13 2.472+03 mg/kg 4.942-01 Beryllium 5/13 8.902-01 mg/kg 1.785-04 Calcium 11/11 2.872+05 mg/kg 5.742+01 8.002+02 1.602+02 No Calcium 11/11 2.872+05 mg/kg 5.742+01 8.002+02 1.602+02 No Cobalt 11/13 9.672+00 mg/kg 1.938-03 COPPer 1.2/13 1.222+01 mg/kg 2.442-03 Iron 13/13 2.338+04 mg/kg 4.652+00 1.502+02 3.002+01 No Marganesium 13/13 1.222+01 mg/kg 5.462+00 1.502+02 3.002+01 No Marganese 13/13 2.732+04 mg/kg 4.322+03 1.502+02 3.002+01 No Marganese 13/13 1.122+03 mg/kg 4.322+03 1.502+02 3.002+01 No Sodium 6/13 5.162+02 mg/kg 1.032+01 No 2.248+01 Sodium Sodium </td <td>1</td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td> <td></td>	1								
Barium 13/13 2.478+03 mg/kg 4.948-01 Beryllium 5/13 8.908-01 mg/kg 1.788-04 Calcium 11/11 2.878+05 mg/kg 5.148-03 Cobalt 11/13 9.678+00 mg/kg 1.938-03 Cobper 12/13 1.228+01 mg/kg 2.448-03 Tron 13/13 2.338+04 mg/kg 2.668+00 Lithium 13/13 2.7328+04 mg/kg 5.468+00 Magnesium 13/13 2.7328+04 mg/kg 5.468+00 1.508+02 3.008+01 No Magnesium 13/13 2.7328+04 mg/kg 4.328-03 No No Magnesium 13/13 2.168+01 mg/kg 4.328-03 No No Nickel 8/13 2.168+02 mg/kg 7.748-02 No No No Strontium 13/13 1.128+03 mg/kg 1.038-01 No No Strontium 13/13 3.658+02 mg/kg 1.038-01 No Acenaphthene 2/13 1.638+02	Aluminum								
Beryllium 5/13 8.908-01 mg/kg 1.78E-04 Calcium 11/11 2.07E+05 mg/kg 5.74E+01 8.00E+02 1.60E+02 No Chromium 13/13 4.57E+00 mg/kg 9.14E-03 Cobalt 1.60E+02 No Cobalt 11/13 9.67E+00 mg/kg 1.93E-03 Copper 12/13 1.22E+01 mg/kg 2.44E-03 Iron 13/13 1.32E+01 mg/kg 2.6E+00 1.50E+02 3.00E+01 No Magnesium 13/13 1.22E+01 mg/kg 5.46E+00 1.50E+02 3.00E+01 No Magnesium 13/13 2.73E+02 mg/kg 7.74E-02 3.00E+01 No Mangahese 13/13 3.87E+02 mg/kg 7.32E-02 3.00E+01 No Nickel 8/13 2.16E+01 mg/kg 1.32E+03 mg/kg 1.50E+02 3.00E+01 No Sodium 6/13 3.66E+02 mg/kg 7.32E-02 3.00E+01 No No Kangahese 13/13 1.51E+03 mg/kg									
Calcium 11/11 2.87E+05 mg/kg 5.74E+01 8.00E+02 1.60E+02 No Chromium 13/13 4.57E+01 mg/kg 9.14E-03		13/13							
Chromium 13/13 4.57E+01 mg/kg 9.14E-03 Cobalt 11/13 9.67E+00 mg/kg 1.93E-03 Copper 12/13 1.22E+01 mg/kg 2.44E-03 Iron 13/13 2.33E+04 mg/kg 4.66E+00 Lithhum 13/13 2.33E+04 mg/kg 4.66E+00 Magnesium 13/13 2.73E+04 mg/kg 5.46E+00 Manganese 13/13 2.73E+04 mg/kg 5.46E+00 Nickel 8/13 2.16E+01 mg/kg 4.32E-03 Potassium 13/13 1.12E+03 mg/kg 2.24E-01 Sodium 6/13 3.66E+02 mg/kg 7.32E-02 Strontium 13/13 5.14E+02 mg/kg 1.03E-01 Vanadium 13/13 3.55E+01 mg/kg 3.26E-02 Accenaphthene 2/13 1.63E+02 mg/kg 1.22E-04 Acenaphthylene 1/13 6.10E-01 mg/kg 1.22E-04 Anthracene 2/13 7.00E+01 mg/kg 4.20E-04 Benzo (a) pyrene	Beryllium		8.90E-01						
Cobalt 11/13 9.67E+00 mg/kg 1.93E-03 Copper 12/13 1.22E+01 mg/kg 2.44E-03 Lithium 13/13 2.33E+04 mg/kg 2.66E+00 Lithium 13/13 1.29E+01 mg/kg 2.58E-03 Manganesium 13/13 2.73E+04 mg/kg 5.46E+00 Manganese 13/13 3.87E+02 mg/kg 7.4E-02 Maidenese 13/13 1.22E+01 mg/kg 2.58E-03 Manganese 13/13 3.87E+02 mg/kg 7.4E-02 Nickel 8/13 2.16E+01 mg/kg 4.32E-03 Potassium 13/13 1.12E+03 mg/kg 2.24E-01 Sodium 6/13 3.66E+02 mg/kg 1.03E-01 Vanadium 13/13 5.14E+02 mg/kg 7.0E-03 Zinc 12/13 1.63E+02 mg/kg 1.03E-01 Vanadium 13/13 3.30E-01 mg/kg 1.62E-02 Acenaphthene 2/13 3.30E-01 mg/kg 1.22E-04 Anthracene 2/1	Calcium		2.87E+05			8.00E+02	1.60E+02	No	
Copper 12/13 1.22E+01 mg/kg 2.44E-03 Iron 13/13 2.33E+04 mg/kg 4.66E+00 Lithium 13/13 1.29E+01 mg/kg 2.58E+03 Magnesium 13/13 2.73E+04 mg/kg 5.46E+00 1.50E+02 3.00E+01 No Manganese 13/13 3.87E+02 mg/kg 7.74E-02 3.00E+01 No Manganese 13/13 3.87E+02 mg/kg 7.74E-02 3.00E+01 No Manganese 13/13 3.87E+02 mg/kg 7.74E-02 3.00E+01 No Manganese 13/13 3.10E+01 mg/kg 4.32E-03 3.00E+01 No Potassium 13/13 1.12E+03 mg/kg 7.32E-02 Strontium 13/13 5.14E+02 mg/kg 7.32E-02 Strontium 13/13 5.14E+02 mg/kg 3.26E-02 Strontium 13/13 3.05E+01 mg/kg 3.26E-02 Acenaphthene 2/13 3.03E-01 mg/kg 3.22E-04 Acenaphthylene 1/13 6.10E-01 mg/kg 1.50E-04<	Chromium								
Iron 13/13 2.33E+04 mg/kg 4.66E+00 Lithium 13/13 1.29E+01 mg/kg 2.5BE-03 Magnesium 13/13 2.73E+04 mg/kg 5.46E+00 1.50E+02 3.00E+01 No Manganese 13/13 3.87E+02 mg/kg 7.44E+02 1.50E+02 3.00E+01 No Manganese 13/13 3.87E+02 mg/kg 4.32E+03 1.50E+02 3.00E+01 No Maiganese 8/13 2.16E+01 mg/kg 4.32E+03 1.50E+02 3.00E+01 No Sodium 6/13 3.66E+02 mg/kg 7.32E+02 1.50E+02 3.00E+01 No Strontium 13/13 1.12E+03 mg/kg 7.32E+02 1.50E+01 No Vanadium 13/13 5.14E+02 mg/kg 7.10E+03 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.50E+04 1.40E+03	Cobalt						•		
Lithium 13/13 1.29E+01 mg/kg 2.58E-03 Magnesium 13/13 2.73E+04 mg/kg 5.46E+00 1.50E+02 3.00E+01 No Manganese 13/13 3.87E+02 mg/kg 7.74E-02 3.00E+01 No Manganese 13/13 3.87E+02 mg/kg 7.74E-02 3.00E+01 No Nickel 8/13 2.16E+01 mg/kg 4.32E-03 3.00E+01 No Potassium 13/13 1.12E+03 mg/kg 2.24E-01 Sodium 6/13 3.66E+02 mg/kg 7.32E-02 Strontium 13/13 3.55E+01 mg/kg 7.10E-03 3.26E-02 3.26E-02 Acenaphthene 2/13 1.63E+02 mg/kg 6.60E-05 3.26E-02 Acenaphthylene 1/13 6.10E-01 mg/kg 1.22E-04 3.40E-04 Anthracene 2/13 7.50E-01 mg/kg 3.40E-04 3.40E-04 Benzo (a) pyrene 2/13 2.10E+00 mg/kg 4.20E-04 3.40E-04 Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg<	Copper								
Magnesium 13/13 2.73±+04 mg/kg 5.46E+00 1.50E+02 3.00E+01 No Manganese 13/13 3.87E+02 mg/kg 7.74E-02 3.00E+01 No Nickel 8/13 2.16E+01 mg/kg 4.32E-03 3.00E+01 No Potassium 13/13 1.12E+03 mg/kg 2.24E-01 3.00E+01 No Sodium 6/13 3.66E+02 mg/kg 7.32E-02 3.00E No Strontium 13/13 5.14E+02 mg/kg 7.0E-03 3.0E No Vanadium 13/13 3.55E+01 mg/kg 6.60E-05 Acenaphthylene 2/13 3.30E-01 mg/kg 1.22E-04 Anthracene 2/13 7.50E-01 mg/kg 1.50E-04 Benzo (a) pyrene 2/13 2.10E+00 mg/kg 3.40E-04 Benzo (a) pyrene 2/13 2.10E+00 mg/kg 4.20E-04 3.40E-04 Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg 4.20E-04 3.40E-03 Benzo (k) fluoranthene 2/13 1.18E+00 mg/kg 1.14E-0	Iron								
Manganese 13/13 3.87E+02 mg/kg 7.74E-02 Nickel 8/13 2.16E+01 mg/kg 4.32E-03 Potassium 13/13 1.12E+03 mg/kg 2.24E-01 Sodium 6/13 3.66E+02 mg/kg 7.32E-02 Strontium 13/13 5.14E+02 mg/kg 7.10E-03 Vanadium 13/13 3.55E+01 mg/kg 7.10E-03 Zinc 12/13 1.63E+02 mg/kg 1.22E-02 Acenaphthene 2/13 3.30E-01 mg/kg 6.60E-05 Acenaphthylene 1/13 6.10E-01 mg/kg 1.22E-04 Anthracene 2/13 7.50E-01 mg/kg 3.40E-04 Benz (a) anthracene 3/13 1.70E+00 mg/kg 3.40E-04 Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg 1.14E-03 Benzo (k) fluoranthene 2/13 1.10E+00 mg/kg 2.36E-04 Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg 1.4E-03 Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg	Lithium								
Manganese 13/13 3.87E+02 mg/kg 7.74E-02 Nickel 8/13 2.16E+01 mg/kg 4.32E-03 Potassium 13/13 1.12E+03 mg/kg 2.24E-01 Sodium 6/13 3.66E+02 mg/kg 7.32E-02 Strontium 13/13 5.14E+02 mg/kg 7.32E-02 Strontium 13/13 5.14E+02 mg/kg 7.10E-03 Vanadium 13/13 3.55E+01 mg/kg 3.26E-02 Acenaphthene 2/13 1.63E+02 mg/kg 3.26E-03 Acenaphthylene 1/13 6.10E-01 mg/kg 1.22E-04 Anthracene 2/13 7.50E-01 mg/kg 1.50E-04 Benz (a) anthracene 3/13 1.70E+00 mg/kg 4.20E-04 Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg 4.20E-04 Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg 2.36E-04 Benzo (b) fluoranthene 3/13 7.90E-01 mg/kg 1.45E-03 Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg	Magnesium	13/13		mg/kg		1.50E+02	3.00E+01	No	
potassium 13/13 1.12E+03 mg/kg 2.24E-01 Sodium 6/13 3.66E+02 mg/kg 7.32E-02 Strontium 13/13 5.14E+02 mg/kg 1.03E-01 Vanadium 13/13 3.55E+01 mg/kg 7.10E-03 Zinc 12/13 1.63E+02 mg/kg 3.26E-02 Acenaphthene 2/13 3.30E-01 mg/kg 6.60E-05 Acenaphthylene 1/13 6.10E-01 mg/kg 1.50E-04 Anthracene 2/13 7.50E-01 mg/kg 1.50E-04 Benz (a) anthracene 3/13 1.70E+00 mg/kg 4.20E-04 Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg 1.14E-03 Benzo (chi) perylene 2/13 1.18E+00 mg/kg 1.58E-04 Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg 1.58E-04		13/13	3.87E+02	mg/kg	7.74E-02				·
Sodium 6/13 3.66E+02 mg/kg 7.32E-02 Strontium 13/13 5.14E+02 mg/kg 1.03E-01 Vanadium 13/13 3.55E+01 mg/kg 7.10E-03 Zinc 12/13 1.63E+02 mg/kg 3.26E-02 Acenaphthene 2/13 3.30E-01 mg/kg 1.22E-04 Acenaphthylene 1/13 6.10E-01 mg/kg 1.50E-04 Anthracene 2/13 7.50E-01 mg/kg 3.40E-04 Benzo (a) anthracene 3/13 1.70E+00 mg/kg 4.20E-04 Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg 1.14E-03 Benzo (chi) perylene 2/13 1.18E+00 mg/kg 1.35E-04 Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg 1.36E-04	Nickel	8/13	2.16B+01	mg/kg	4.32E-03				
Strontium 13/13 5.14E+02 mg/kg 1.03E-01 Vanadium 13/13 3.55E+01 mg/kg 7.10E-03 Zinc 12/13 1.63E+02 mg/kg 3.26E-02 Acenaphthene 2/13 3.30E-01 mg/kg 6.60E-05 Acenaphthylene 1/13 6.10E-01 mg/kg 1.22E-04 Anthracene 2/13 7.50E-01 mg/kg 1.50E-04 Benz (a) anthracene 3/13 1.70E+00 mg/kg 4.20E-04 Benzo (a) pyrene 2/13 2.10E+00 mg/kg 4.20E-04 Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg 1.14E-03 Benzo (ghi) perylene 2/13 1.18E+00 mg/kg 1.35E-04 Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg 1.36E-04	Potassium	13/13	1.12E+03	mg/kg	2.24E-01				
Strontium 13/13 5.14E+02 mg/kg 1.03E-01 Vanadium 13/13 3.55E+01 mg/kg 7.10E-03 Zinc 12/13 1.63E+02 mg/kg 3.26E-02 Acenaphthene 2/13 3.30E-01 mg/kg 6.60E-05 Acenaphthylene 1/13 6.10E-01 mg/kg 1.22E-04 Anthracene 2/13 7.50E-01 mg/kg 1.50E-04 Benz (a) anthracene 3/13 1.70E+00 mg/kg 4.20E-04 Benzo (a) pyrene 2/13 2.10E+00 mg/kg 4.20E-04 Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg 1.14E-03 Benzo (ghi) perylene 2/13 1.18E+00 mg/kg 1.35E-04 Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg 1.36E-04	Sodium	6/13	3.66B+02	mg/kg	7.32E-02				
Vanadium13/133.55E+01mg/kg7.10E-03Zinc12/131.63E+02mg/kg3.26E-02Acenaphthene2/133.30E-01mg/kg6.60E-05Acenaphthylene1/136.10E-01mg/kg1.22E-04Anthracene2/137.50E-01mg/kg1.50E-04Benzo (a) anthracene3/131.70E+00mg/kg4.20E-04Benzo (b) fluoranthene6/135.70E+00mg/kg1.14E-03Benzo (c) jerylene2/131.18E+00mg/kg1.58E-04Benzo (k) fluoranthene3/137.90E-01mg/kg1.58E-04					1.03E-01				
Zinc 12/13 1.63E+02 mg/kg 3.26E-02 Acenaphthene 2/13 3.30E-01 mg/kg 6.60E-05 Acenaphthylene 1/13 6.10E-01 mg/kg 1.22E-04 Anthracene 2/13 7.50E-01 mg/kg 1.50E-04 Benzo (a) anthracene 3/13 1.70E+00 mg/kg 3.40E-04 Benzo (a) pyrene 2/13 2.10E+00 mg/kg 1.14E-03 Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg 2.36E-04 Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg 1.58E-04						•			
Acenaphthene 2/13 3.30E-01 mg/kg 6.60E-05 Acenaphthylene 1/13 6.10E-01 mg/kg 1.22E-04 Anthracene 2/13 7.50E-01 mg/kg 1.50E-04 Benz (a) anthracene 3/13 1.70E+00 mg/kg 3.40E-04 Benzo (a) pyrene 2/13 2.10E+00 mg/kg 4.20E-04 Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg 1.14E-03 Benzo (gh1) perylene 2/13 1.18E+00 mg/kg 1.58E-04 Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg 1.58E-04			· · · · = · · · · · · · =						
Acenaphthylene 1/13 6.10E-01 mg/kg 1.22E-04 Anthracene 2/13 7.50E-01 mg/kg 1.50E-04 Benz (a) anthracene 3/13 1.70E+00 mg/kg 3.40E-04 Benzo (a) pyrene 2/13 2.10E+00 mg/kg 4.20E-04 Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg 1.14E-03 Benzo (gh1) perylene 2/13 1.18E+00 mg/kg 2.36E-04 Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg 1.58E-04									
Anthracene 2/13 7.50E-01 mg/kg 1.50E-04 Benz (a) anthracene 3/13 1.70E+00 mg/kg 3.40E-04 Benzo (a) pyrene 2/13 2.10E+00 mg/kg 4.20E-04 Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg 1.14E-03 Benzo (gh1) perylene 2/13 1.18E+00 mg/kg 2.36E-04 Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg 1.58E-04									
Benz (a) anthracene 3/13 1.70E+00 mg/kg 3.40E-04 Benzo (a) pyrene 2/13 2.10E+00 mg/kg 4.20E-04 Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg 1.14E-03 Benzo (ghi) perylene 2/13 1.18E+00 mg/kg 2.36E-04 Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg 1.58E-04									
Benzo (a) pyrene 2/13 2.10E+00 mg/kg 4.20E-04 Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg 1.14E-03 Benzo (ghi) perylene 2/13 1.18E+00 mg/kg 2.36E-04 Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg 1.58E-04									
Benzo (b) fluoranthene 6/13 5.70E+00 mg/kg 1.14E-03 Benzo (ghi) perylene 2/13 1.18E+00 mg/kg 2.36E-04 Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg 1.58E-04									
Benzo (ghi) perylene 2/13 1.18E+00 mg/kg 2.36E-04 Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg 1.58E-04									
Benzo (k) fluoranthene 3/13 7.90E-01 mg/kg 1.58E-04									
Chrysene 2/13 2.10E+00 mg/kg 4.20E-04					the second second second second second second second second second second second second second second second se		•		

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		(contin	med)					
Analyte	Frequency of Detection	Maximum detected concentration	Units	Daily dose for child	RDA for child	1/5	RDA	Exceed RDA?
Dibenzofuran	1/13	1.23E-01	mg/kg	2.46E-05				·
Fluoranthene	4/13	2.66E+00	mg/kg	5.32E-04				
Fluorene	1/13	2.19E-01	mg/kg	4.388-05				
Indeno (1,2,3-cd) pyrene	2/13	1.05E+00	mg/kg	2.10E-04		•		
PCB-1016	. 1/16	1.87E+00	mg/kg	3.74E-04				
PCB-1254	1/16	9.60E-02	mg/kg	1.92E-05				
PCB-1260	5/16	6.31E-01	mg/kg	1.26E-04				
Phenanthrene	2/13	1.63E+00	mg/kg	3.26E-04				
Pyrene	3/13	2.70E+00	mg/kg	5.40E-04				
Alpha activity	15/16	1.428+02	pCi/g					
Beta activity	16/16	2.73E+03	pCi/g					
Cesium-137	3/16	1.90E+00	pCi/g					
Neptunium-237	- 1/1	1.28E+01	pCi/g					
Technetium-99	3/16	2.65E+03	pC1/g					
Thorium-234	1/16	5.30E+01	pCi/g					
Uranium-234	1/1	1.64B+01	pCi/g					
Uranium-238	1/1	5.17E+01	pCi/g			•		

-- LOCATION=SWMU 99B MEDIA=RGA Groundwater -----

Analyte	Frequency of Detection	Maximum detected concentration	Units	Daily dose for child	RDA for child	1/5 RDA	Exceed RDA?
Barium	7/7	2.70E+00	mg/L	2.70E+00			
Calcium	7/7	3.27E+01	mg/L	3.27E+01	8.00E+02	1.60E+02	No
Chloride	7/7	1.08E+02	mg/L	1.08E+02	6.00B+02	1.20E+02	Nö
Chromium	1/7 ·	2.60E-01	mg/L	2.60B-01		-	
Copper	1/7	4.00E-02	mg/L	4.00E-02	1.00E+00	2.00E-01	No
Fluoride	7/7	2.10E-01	mg/L	2.10E-01	1.50E+00	3.00B-01	No
Iron	3/7	3.34E+00	mg/L	3.34E+00	1.00E+01	2.00E+00	Yes
Magnesium	7/7	1.31E+01	mg/L	1.31E+01	1.70E+02	3.40E+01	No
Manganese	5/7	2.90E-01	mg/L	2.90E-01	•		
Nitrate as Nitrogen	7/7	2.10E+00	mg/L	2.10E+00			
Silica	7/7	2.00E+01	mg/L	2.00E+01			
Sodium	7/7	7.86E+01	mg/L	7.86E+01	•		
Sulfate	2/2	2.67E+01	mg/L	2.67E+01			
Tetraoxo-sulfate(1-)	5/5	2.90E+01	mg/L	2.90E+01			

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			for chi	ldren			•		
÷		LOCA	TION-SWMU 998 MED		oundwater				
			(contin	ued)					
		Frequency	Maximum			RDA	•		
		of	detected		Daily dose	for		Exceed	
	Analyte	Detection	concentration	Units	for child	child	1/5 RDA	RDA?	•
	Analyce	Decection	CONCENCIACION	UILLEB	tor chirte				
	Zinc	2/7	6.00E-02	mg/L	6.00E-02				
	Trichloroethene	16/16	2.30E+00	mg/L	2,30E+00 .				
	Alpha activity	12/16	4.20E+00	pCi/L				,	
	Beta activity	16/16	4.50E+01	pCi/L					
	Radon-222	4/4	4.12E+02	pCi/L					
	Technetium-99	. 12/17	1.90E+01	pCi/L					
		LOCA	TION-SWMU 99B ME	IA-Subsur	face Soil	***********			
			Maximum			RDA			
		Frequency of	detected		Daily dose	for		Exceed	
<u> </u>)	Detection	concentration	Units	for child	child	1/5 RDA	RDA?	
22	Analyte	Defection	concentration	0111 04	LOI CHILLG	outil	I/J KUR		
æ	Aluminum	8/8	1.70E+04	mg/kg	3.40E+00		i.		
	Arsenic	2/6	8.05E+00	mg/kg	1.61E-03		•		
	Barium	8/8	1.55E+02	mg/kg	3.108-02				
	Beryllium	6/8	1.005+00	mg/kg	2.00E-04				
	Calcium	6/6	7.17E+03	mg/kg	1.43E+00	8.00E+02	1.60E+02	No	
	Chromium	8/8	2.61E+01	mg/kg	5.22E-03			1.0	
	Cobalt	8/8	6.94E+00	mg/kg	1.39E-03				
	Copper	8/8	1.30E+01	mg/kg	2.60E-03				
	Iron	8/8	1.81E+04	mg/kg	3.62E+00				
	Lithium	8/8	1.14E+01	mg/kg	2.28E-03				
	Magnesium	8/8	2.53E+03	mg/kg	5.06E-01	1,50E+02	3.00E+01	Nó	
	Manganese	8/8	5.24E+02	mg/kg	1.05E-01				
	Nickel	5/8	2.51E+01	mg/kg	5.028-03				
	Potassium	8/8	1.04E+03	mg/kg	2.08E-01	1.60E+03	3.20E+02	No	
	Sodium	3/8	3.09E+02	mg/kg	6.18E-02				
	Strontium	8/8	2.228+01	mg/kg	4.44B=03	•			
	Vanadium	8/8	3.44E+01	mg/kg	6,88E+03				
	Zinc	8/8	5.22E+01	mg/kg	1.04E-02	• •			
	Acetone	1/7	5.50E-01	mg/kg	1.10E-04				
	Methylene Chloride	3/7	1.20E+00	mg/kg	2.408-04				
	Alpha activity	6/6	2.148+01	pCi/g					
	Beta activity	8/8	2.26E+01	pCi/g					
	Dela avertily	9/ 9	A . A CATUL	F~-1.3					

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- LOCATION=AOC 204 MED_NAME=RGA Groundwater

	frequency
Analyte	Detection
1,1-Dichloroethane	1/1
1,1-Dichloroethene	12/15
PCB-1254	11/11
PCB-1260	11/11
Polychlorinated biphenyl	1/1
Tetrachloroethene	11/11
Trichloroethene	15/15
Vinyl Chloride	1/4
cis-1,2-Dichloroethene	3/4

LOCATION-AOC 204 MED NAME-Subsurface Soil

Analyte	of Detection
1,1-Dichloroethene	11/17
PCB-1254	11/11
PCB-1260	11/11
Polychlorinated biphenyl	8/8
Tetrachloroethene	11/17
Trichloroethene	11/17

-- LOCATION-SWMU 193A MED_NAME-McNairy Groundwater -----

Analyte	Frequency of Detection
Iron	4/4
Tetraoxo-sulfate (1-) *	4/4
Trichloroethene	8/13
cis-1,2-Dichloroethene	1/11
Technetium-99	5/10
Uranium-238	1/1

- LOCATION-SNMU 193A MED_NAME-RGA Groundwater -

of
Detection
1/1
1/1
7/9
1/1
5/5
4/4
2/43
1/25
44/51
3/25
17/42
26/39

LOCATION=SWMU 193A MED_NAME=Subsurface Soil

	Frequency
Analyte	Detection
Aluminum	8/8
Beryllium	5/8
Chromium	8/8
Anthracene	1/8
Benz (a) anthracene	2/8
Benzo (a) pyrene	2/8
Benzo (b) fluoranthene	2/8
Benzo (ghi) perylene*	2/8
Chrysene	2/8
Di-n-butylphthalate	1/8
Di-n-octylphthalate	1/8
Dibenz (a, h) anthracene	1/8
Fluoranthene	2/8
Indeno (1, 2, 3-cd) pyrene	2/8
Pyrene	2/8
bis (2-Ethylhexyl) phthalate	2/8

- LOCATION-SWMU 193A MED_NAME-Surface Soil

Analyte	Frequency of Detection
Chromium	4/4
Anthracene	1/4
Benz(a) anthracene	2/4
Benzo (a) pyrene	2/4
Benzo (b) fluoranthene	2/4
Benzo (ghi) perylene*	2/4
Chrysene	2/4
Di-n-butylphthalate	1/4
Di-n-octylphthalate	1/4
Dibenz (a, h) anthracene	1/4
Fluoranthene	2/4
Indeno(1,2,3-cd)pyrene	2/4
Pyrene	2/4
bis (2-Ethylhexyl) phthalate	2/4

OCATION-SWMU 193B MED NAME-McNairy Groundwater -

Analyte	Frequency of Detection
Trichloroethene	1/2
cis-1,2-Dichloroethene	1/2

- LOCATION-SWMU 1938 MED_NAME=RGA Groundwater --

	of
Analyte	Detection
1,1-Dichloroethene	3/17
Acetone	1/2
Carbon Tetrachloride	1/5
Di-n-butylphthalate	2/10
Trichloroethene	17/17

 LOCATION-SWMU 193B MED_NAME-RGA (continued)	Groundwater	
Analyte	Frequency of Detection	
bis(2-Ethylhexyl)phthalate cis-1,2-Dichloroethene Technetium-99	1/10 12/17 8/17	
 LOCATION-SWMU 193B MED_NAME-Subs	urface Soil	
 Analyte	Frequency of Detection	
Beryllium Chromium Vanadium	2/4 4/4 4/4	

- LOCATION-SWMU 193B MED_NAME-Surface Soil --

Analyte	• •	Frequency of Detection
Beryllium Chromium Vanadium		1/2 2/2 2/2

- LOCATION-SWMU 193C MED_NAME-MCNairy Groundwater -----

	Frequency
Analyte	Detection
Aluminum	4/4
Antimony	5/5
Arsenic	5/5
Barium	5/5
Beryllium	5/5
Cadmium	5/5
Chromium	3/3
Cobalt	5/5
Iron	5/5
Lead	1/1
Manganese	5/5
Mercury	1/1
Molybdenum	4/4
Nickel	5/5
Silica*	5/5
Silver	3/3
Tetraoxo-sulfate(1-)*	5/5
Thallium*	2/2
Oranium	9/9
Vanadium	2/2
Zinc	5/5
1,1,2-Trichloroethane	4/4
1,1-Dichloroethene	4/4
1,2-Dichloroethane	4/4
Benzene	4/4
Bromodichloromethane	4/4
Carbon Tetrachloride	4/4

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- LOCATION-SWMU 193C MED_NAME-McNairy Groundwater ----(continued)

	Frequency of
Analyte	Detection
Chloroform	4/4
Ethylbenzene	4/4
Polychlorinated biphenyl	1/1
Tetrachloroethene	4/4
Trichloroethene	12/12
Vinyl Chloride	4/4
Xylene	4/4
cis-1,2-Dichloroethene	4/4
trans-1,2-Dichloroethene	4/4
Radon-222	2/2

----- LOCATION-SWMU 193C MED_NAME=RGA Groundwater ---

Analyte	of Detection
1,2-Dichloroethene Trichloroethene	1/2 1/2

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LOCATION-SWMU 193C MED_NAME-Subsurface Soil -----

	Frequency
Analyte	Detection
Aluminum	20/20
Beryllium	10/20
Cadmium	3/59
Chromium	59/61
Cobalt	17/20
Iron	6/6
Lead	42/61
Manganese	20/20
Vanadium	20/20
Zinc	19/20
Xylene	1/20

LOCATION-SWMU 193C MED_NAME-Surface Soil

Analyte		Frequency of Detection
Chromium Lead Zinc	•	3/5 1/5 5/5

LOCATION-SWMU 194 MED_NAME-Subsurface Soil -----

Analyte Detection Aluminum 12/12 Beryllium 6/12

- LOCATION=SWMU 194 MED_NAME=Subsurface Soil -(continued)

Analyte	. Frequency of
	Detection
Cadmium	1/35
Chromium	35/35
Lead	20/35
Zinc	11/12
Ethylbenzene	1/19

-- LOCATION-SWMU 99A MED_NAME-MCNairy Groundwater -----

Analyte	Frequency of Detection
1,1-Dichloroethene	1/4
Carbon Tetrachloride	1/4
Trichloroethene	3/4
cis-1,2-Dichloroethene	2/4

LOCATION-SWMU 99A MED_NAME-RGA Groundwater

	Frequency of
Analyte	Detection
Aluminum	16/35
Arsenic	4/27
Barium	39/39
Beryllium	8/35
Chromium	11/39
Cobalt	20/37
Copper	9/35
Iron	31/39
Lead	6/29
Lithium	6/23
Manganese	37/39
Mercury	5/25
Nickel	16/39
Silica*	9/9
Sulfate*	2/2
Tetraoxo-sulfate(1-) *	7/7
Vanadium	10/28
Zinc	10/35
1,1-Dichloroethene	7/33
Trichloroethene	41/43
bis (2-Ethylhexyl) phthalate	5/10
cis-1,2-Dichloroethene	10/33
Radon-222	4/4
Technetium-99	34/40

LOCATION-SWMU 99A MED_NAME-Subsurface Soil

Analyte	of Detection
Aluminum	22/22
Antimony	5/22
Arsenic	11/22

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LOCATION=SWMU 99A MED_NAME=Subsurface Soil -(continued)

	Frequency
· · · · · · · · · · · · · · · · · · ·	of
Analyte	Detection
Barium	22/22
Beryllium	11/22
Cadmium	5/22
Chromium	22/22
Lead	6/22
Manganese	22/22
Thallium*	5/22
Zinc	21/22
1,1-Dichloroethene	5/10
1,2,4-Trichlorobenzene 1,2-Dichlorobenzene	5/22 5/22
1,3-Dichlorobenzene	5/22
1,4-Dichlorobenzene	5/22
2,4,5-Trichlorophenol	5/22
2,4,6-Trichlorophenol	5/22
2,4-Dinitrotoluene	5/22
2,6-Dinitrotoluene	5/22
2-Chloronaphthalene	5/22
2-Hexanone* 2-Methyl-4,6-dinitrophenol*	5/8 5/22
2-Methylnaphthalena*	5/22
2-Nitroaniline	5/22
2-Nitrophenol*	5/22
3,3'-Dichlorobenzidine	5/22
3-Nitroaniline*	5/22
4,4'-DDD	2/2
4,4'-DDE	
4,4'-DDT 4-Bromophenyl phenyl ether*	2/2 5/22
4-Chloro-3-methylphenol*	5/22
4-Chlorophenyl phenyl ether*	5/22
4-Nitroaniline*	5/22
Acenaphthene	7/22
Acenaphthylene*	6/22
Aldrin	2/2
Anthracene	7/22
Benz (a) anthracene Benzo (a) pyrene	8/22 7/22
Benzo (b) fluoranthene	11/22
Benzo (ghi) perylene*	7/22
Benzo (k) fluoranthene	8/22
Butyl benzyl phthalate	5/6
Chrysene	7/22
Di-n-butylphthalate	5/22
Di-n-octylphthalate Dibenz(a,h) anthracene	5/22 6/22
Dibenzofuran	6/22
Dieldrin	2/2
Endosulfan I*	2/2
Endosulfan II*	2/2
Endosulfan Sulfate*	2/2
Endrin	2/2
Endrin Ketone*	2/2 5/8
Ethylbenzene Fluoranthene	9/22
Fluorene	6/22
Heptachlor	2/2
Heptachlor Epoxide	2/2
Hexachlorobenzene	5/22
Hexachlorobutadiene	5/22

---- LOCATION=SWMU 99A MED_NAME=Subsurface Soil -(continued)

	Frequency of
Analyte	Detection
Hexachlorocyclopentadiene	5/22
Hexachloroethane	5/22
Indeno (1, 2, 3-cd) pyrene	7/22
Methoxychlor	2/2
N-Nitroso-di-n-propylamine	5/22
N-Nitrosodiphenylamine	5/22
Naphthalene	5/22
PCB-1016	3/23
PCB-1221	2/23
PCB-1232	2/23
PCB-1242	2/23
PCB-1248	2/23
PCB-1254	3/23
PCB-1260	7/23
Pentachlorophenol	5/22
Phenanthrene*	7/22
Pyrene	8/22
Toxaphene	2/2
Vinyl Chloride	5/10
Xylene	5/5
alpha-BHC	2/2
alpha-Chlordane*	2/2
beta-BHC	2/2
bis(2-Chloroethoxy)methane*	5/22
bis (2-Chloroethyl) ether	5/22
bis(2-Chloroisopropyl)ether	5/22
bis(2-Ethylhexyl)phthalate	5/22
cis-1, 3-Dichloropropene*	5/8
delta-BHC*	2/2
gamma-BHC (Lindane)	2/2
gamma-Chlordane*	2/2
trans-1,3-Dichloropropene*	5/8
Cesium-137	3/21
Neptunium-237	4/4
Technetium-99	6/23
Thorium-234	1/21
Dranium-234	4/4
Dranium-238	4/.4

----- LOCATION-SWMU 99A MED_NAME-Surface Soil ------

	Frequency of Detection
Analyte	Detection
Barium	13/13
Beryllium	5/13
Chromium	13/13
Zinc	12/13
Acenaphthene	2/13
Acenaphthylene*	1/13
Anthracene	2/13
Benz(a) anthracene	3/13
Benzo (a) pyrene	2/13
Benzo (b) fluoranthene	6/13
Benzo (ghi) perylene*	2/13
Benzo (k) fluoranthene	3/13
Chrysene	2/13
Dibenz(a,h)anthracene	1/13

-- LOCATION-SWMU 99A MED_NAME-Surface Soil -(continued)

	Frequency of
Analyte	Detection
Dibenzofuran	1/13
Fluoranthene	4/13
Fluorene	1/13
Indeno (1,2,3-cd) pyrene	2/13
PCB-1016	1/16
PCB-1254	1/16
PCB-1260	5/16
Phenanthrene*	2/13
Pyrene	3/13
Cesium-137	3/16
Neptunium-237	1/1
Technetium-99	3/16
Thorium-234	1/16
Uranium-234	1/1
Dranium-238	1/1

- LOCATION-SWMU 998 MED_NAME-RGA Groundwater

	Frequency of
Analyte	Detection
Barium	7/7
Chromium	1/7
Iron	3/7
Manganese	5/7
Silica*	7/7
Sulfate*	2/2
Tetraoxo-sulfate(1-) *	5/5
Zinc	2/7
Trichloroethene	16/16
Radon-222	4/4

-- LOCATION-SWMU 99B MED_NAME-Subsurface Soil -

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Analyte	Frequency of Detection
Aluminum	8/8
Arsenic	2/6
Beryllium	6/8
Chromium	8/8
Methylene Chloride	3/7

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,	Frequency of	Nondetected	Detected	Arithmetic			···· _	COPC/
Analyte	Detection	Range	Range	Mean	HI	ELCR	Units	Basis*
1,1,1-Trichloroethane	11/11		1.008-02 - 1.80E-02	1.13E-02	5.48-02		mq/L	No
1,1-Dichloroethane	1/1		5.00E+00 - 5.00E+00	5.008+00	2.78-02		mq/L	Yes/P
1,1-Dichloroethene	12/15	1.00E-03 - 1.00E-03	1.00B-04 - 4.00E-02	3.25E-02	1.8E-03	9.3E-07	mg/L	Yës/P
PCB-1254	11/11		2.50E-02 - 2.50E-02	2.50E-02	1.98-05	8.08-06	mg/L	Yes/P
PCB-1260	11/11		2.50B-02 - 2.50E-02	2.50E-02		4.48-06	mg/L	Yes/P
Polychlorinated biphenyl	1/1		1.70B-01 - 1.70B-01	1.70B-01		8.0B-06	mg/L	Yes/P
Tetrachloroethene	11/11		5.00E-03 - 5.00E+00	4.59E-01	7.98-03	5.78-05	mg/L	Yes/P
Trichloroethere	15/15		5.00E-03 - 7.70E-01	1.41B-01	1.2E-03	1.4E-04	mg/L	Yes/P
Vinyl Chloride	1/4	1.00B-03 - 1.00B-03	1.00E-04 - 1.00E-04	7.75E-04		1.7E-06	mg/L	Yes/P
cis-1,2-Dichloroethene	3/4	1.00E-03 - 1.00E-03	9.00B-04 - 6.00B-03	3.488-03	2.0E-03		ng/L	Yes/P
trans-1,2-Dichloroethene	2/4	1.008-03 - 1.008-03	1.00E-04 - 1.00E-04	5.50E-04	4.0E-03		mg/L	No
Alpha activity	2/4	7.908-01 - 1.708+00	2.40E+00 - 6.80E+00	2.928+00			pC1/L	Yes/Qual
Beta activity	2/4	1.908+00 - 2.608+00	3.408+00 - 5.208+00	3.28E+00			pCi/L	Yes/Qual
- - .	of	Nondetected	Detected	Arithmetic	<u>`</u> -			COPC/
Analyte	Detection	Range	Range	Mean	HI	ELCR	Units	Basis*
1,1,1-Trichloroethane	10/16	1.00E-02 - 1.00E-02	1.00E-02 - 1.00E+00	4.642-02	1.2E+02		mg/kg	No
1,1-Dichloroethane	8/14	1.00E-02 - 1.00E-02	1.00E+00 - 1.00E+00	5.74E-01	6.7B+01		mg/kg	No
1,1-Dichloroethene	11/17	3.36B-01 - 4.27E-01	4.00B-02 - 4.00E-02	9.35B-02	3.5E+00	3.9B-03	mg/kg	Yes/P
PCB-1254	11/11		2.508-02 - 2.508-02	2.50E-02	6.6E-02	9.9E-03	mg/kg	Yes/P
	11/11		2.50E-02 - 2.50E-02	2.50E-02		9.8E-03	mg/kg	Yes/P
PCB-1260	TT/T							
	8/8		1.00E-01 - 1.00B-01	1.00B-01		1.0E-02	ma/ka	Yes/P
Polychlorinated biphényl		1.00 2-02 - 1.00 5-0 2		1.00E-01	1.2E+01	1.0E-02 1.3E-01	ng/kg ng/kg	Yes/P Yes/P
Polychlorinated biphenyl Tetrachloroethene	8/8	1.008-02 - 1.008-02 3.36E-01 - 4.278-01	1.00E-01 - 1.00E-01	1.008-01 4.738-01	1.2E+01 1.2E+00	1.3E-01	mg/kg	Yes/P
Polychlorinated biphényl Tetrachloroethene Trichloroethene	8/8 11/17 11/17		1.00E-01 - 1.00E-01 5.00E-03 - 1.00E+00 5.00E-03 - 1.00E+00	1.008-01 4.738-01 1.678-01	1.2E+01 1.2E+00		mg/kg mg/kg	Yes/P Yes/P
Polychlorinated biphényl Tetrachloroethene Trichlöroethene Alpha activity	8/8 11/17		1.00E-01 - 1.00E-01 5.00E-03 - 1.00E+00	1.008-01 4.738-01 1.678-01 1.538+01		1.3E-01	mg/kg mg/kg pC1/g	Yes/P Yes/P Yes/Qua
Tetrachloroethene Trichloroethene Alpha activity Beta activity	8/8 11/17 11/17 6/6 6/6		1.00E-01 - 1.00E-01 5.00E-03 - 1.00E+00 5.00E-03 - 1.00E+00 9.50E+00 - 1.96E+01 1.71E+01 - 2.91E+01	1.002-01 4.738-01 1.678-01 1.538+01 2.278+01	1.2B+00	1.3E-01 9.1E-02	mg/kg mg/kg pCi/g pCi/g	Yes/P Yes/P Yes/Qual Yes/Qual
Polychlorinated biphényl Tetrachloroethene Trichlöroethene Alpha activity Beta activity	8/8 11/17 11/17 6/6 6/6 Frequency	3.36E-01 - 4.27E-01	1.00E-01 - 1.00E-01 5.00E-03 - 1.00E+00 5.00E-03 - 1.00E+00 9.50E+00 - 1.96E+01 1.71E+01 - 2.91E+01 193A MEDIA=McNairy Groun	1.008-01 4.73E-01 1.67E-01 1.53E+01 2.27E+01	1.2B+00	1.3E-01 9.1E-02	mg/kg mg/kg pCi/g pCi/g	Yes/P Yes/P Yes/Qual Yes/Qual
Polychlorinated biphenyl Tetrachloroethene Trichlöroethene Alpha activity Beta activity	8/8 11/17 11/17 6/6 6/6 Frequency of	3.36E-01 - 4.27E-01	1.00E-01 - 1.00E-01 5.00E-03 - 1.00E+00 5.00E-03 - 1.00E+00 9.50E+00 - 1.96E+01 1.71E+01 - 2.91E+01 193A MEDIA=McNairy Groun Detected	1.002-01 4.73E-01 1.67E-01 1.53E+01 2.27E+01 Avater	1.2 8 +00	1.3E-01 9.1E-02	mg/kg mg/kg pC1/g pC1/g	Yes/P Yes/P Yes/Qual Yes/Qual
Polychlorinated biphenyl Tetrachloroethene Trichlöroethene Alpha activity Beta activity	8/8 11/17 11/17 6/6 6/6 Frequency	3.36E-01 - 4.27E-01	1.00E-01 - 1.00E-01 5.00E-03 - 1.00E+00 5.00E-03 - 1.00E+00 9.50E+00 - 1.96E+01 1.71E+01 - 2.91E+01 193A MEDIA=McNairy Groun	1.008-01 4.73E-01 1.67E-01 1.53E+01 2.27E+01	1.2B+00	1.3E-01 9.1E-02	mg/kg mg/kg pCi/g pCi/g	Yes/P Yes/P Yes/Qual Yes/Qual
Polychlorinated biphényl Tetrachloroethene Trichlöroethene Alpha activity Beta activity	8/8 11/17 11/17 6/6 6/6 Frequency of	3.36E-01 - 4.27E-01	1.00E-01 - 1.00E-01 5.00E-03 - 1.00E+00 5.00E-03 - 1.00E+00 9.50E+00 - 1.96E+01 1.71E+01 - 2.91E+01 193A MEDIA=McNairy Groun Detected	1.002-01 4.73E-01 1.67E-01 1.53E+01 2.27E+01 Avater	1.2 8 +00	1.3E-01 9.1E-02	mg/kg mg/kg pC1/g pC1/g	Yes/P Yes/P Yes/Qual Yes/Qual

*P= > PRG, B= > Background, E= > Essential Nutrient, Bio-Bioaccumulates, Qual-Qualitative analyte

Tetraco-sulfate[1-] 4/4 2.108-01 5.058-01 mg/L Yes/Qua Acetone 1/1 1.408-02 -1.408-02 1.408-02 2.02-02 mg/L No Digthylphthalate 1/6 1.008-02 -4.008-02 1.308-02 1.408-02 1.428-00 mg/L No TrichLorosethane 6/13 1.008-03 -2.008+00 1.708-02 1.308-02 1.428-03 1.42-04 mg/L Yes/P Alpha activity 6/10 2.308-01 1.408+00 2.408+00 4.448+00 2.02-03 mg/L Yes/P Beta activity 6/10 1.008+00 -4.408+00 5.108+01 2.138+01 2.188+01 2.08-01 pC1/L Yes/P Thorimu-234 1/1 8.008+00 -4.408+00 5.008+01 1.328+01 2.138+01 2.08-01 pC1/L Yes/P Thorimu-234 1/1 8.008+01 -8.408+00 1.308+01 1.458+02 1.388+01 2.08+00 pC1/L Yes/P Thorimu-238 1/1 1.228+00 1.328+00 6.28-01 pC1/L Yes/P Thorimu-238 1/1 1.228+00 1.328+00 6.28-01 pC1/L No Dranium-238 1/1 1.228+00 1.328+00 6.28-01 pC1/L No Dranium-238 1/1 1.228+00 1.328+00 1.328+00 6.28-01 pC1/L No COC/ Analyte Detection Range Negative Range Nega				(continued)					
Numlyte Dataction Range Range Maan HI RLCR Units Basis from 4/4 2.897×00 - 3.142×02 8.362×01 4.55×01 mg/L Yes/PE Regnessim 4/4 5.332×00 - 4.022×01 1.665×01 mg/L Yes/PE Sodium 4/4 5.332×00 - 4.022×01 1.65×01 mg/L Yes/PE Sodium 4/4 1.64×01 - 8.025×01 5.022×00 mg/L Yes/PE Sodium 4/4 1.602×02 - 1.602×01 1.602×02 </th <th></th> <th></th> <th></th> <th></th> <th></th> <th></th> <th></th> <th></th> <th></th>									
Tron 4/4 2.898+00 - 3.148+02 8.368+01 4.55-01 mg/L Yes/Ye Magnesium 4/4 5.338+00 1.468+01 9.628+00 mg/L Yes/Ye Sodium 4/4 4.028+01 1.468+01 9.628+00 mg/L Yes/Yes Sodium 4/4 1.648+01 5.038+01 4.038+01 5.088+01 mg/L Yes/Yes Sodium 4/4 1.648+01 5.038+01 5.088+01 5.088+01 mg/L Yes/Yes Acetone 1/1 1.008-02 - 4.008-02 1.080-02 <t< th=""><th></th><th></th><th></th><th></th><th></th><th></th><th></th><th></th><th></th></t<>									
Segmestim 4/4 5.338-00 4.628-01 mg/L Yes/s Sodium 4/4 5.338-00 2.028-00 mg/L No Sodium 4/4 1.648-01 5.038-01 mg/L No Sodium 4/4 1.648-01 5.038-01 mg/L No Sodium 4/4 1.648-01 1.487-01 mg/L Yes/cu Sodium 1/1 1.008-02 1.008-02 1.008-02 1.008-02 1.008-03	Analyte	Detection	Range	Range	Mean	HI	BLCR	Units	Basis*
Profession 4/4 4.078+00 2.158+01 9.022+00 mg/L No Sodiu 4/4 1.668+01 8.068+01 8.138+01 mg/L Yes/Ous Tetraxo-sulfate(1-) 4/4 2.108+01 8.068+01 5.058+01 mg/L Yes/Ous Vettors 1/1 1.008-02 1.408-02 1.048-02 1.28+00 mg/L Yes/Ous Sigth/phthalate 1/6 1.008-02 2.008-04 1.008-02 1.28-01 1.28-03 mg/L Yes/Ous Sigth/phthalate 1/1 1.008-03 2.008+00 1.008-02 1.048-02 1.28-03 mg/L Yes/Yes/Ous Sigth/sig	Iron	-, -				4.5B-01			
Dedium 4/4 1.648-01 8.088-01 4.138+01 mg/L Yes/Outs/ Ves/Outs/ Net/L Tetraxor.sulfate[1-] 4/4 2.108+01 8.088+01 5.058+01 mg/L Yes/Outs/ Ves/Outs/ No Vestone 1/1 1.008-02 1.008-02 1.008-02 1.008-02 1.008-02 1.008-02 1.008-03 1.028-02 1.008-03 1.28+03 1.28+03 1.48-04 mg/L Yes/Outs/ Yes/Dus/ Sight, 2.008+00 1.008-03 2.008+00 1.008-03 4.008+03 1.008-03	lagnesium							mg/L	Yes/B
Tetraxor-sulfate[1-) 4/4 2.1028-02 = 6.4028-01 5.0528-01 mg/L 7es/Dua Vestone 1/1 1.4028-02 - 1.4028-02 1.4028-02 1.4028-02 1.4028-02 1.4028-02 1.228-00 mg/L No Tichiolorostheme 3/11 1.0028-03 - 2.0028+00 1.7028-02 1.3028-02 1.2028-03 1.228-03 mg/L Yes/F Sis-1,2-Dichiorostheme 1/11 1.0028-03 - 2.0028+00 1.7028-01 6.4528+00 2.028-03 mg/L Yes/F Sis-1,2-Dichiorostheme 1/11 1.0028-03 - 2.0028+00 1.7028-01 6.4528+00 2.028-03 mg/L Yes/F Sis-1,2-Dichiorostheme 1/11 1.0028-03 - 2.0028+00 1.0028-01 2.1328+01 2.028-03 mg/L Yes/F Sis-1,2-Dichiorostheme 1/11 1.0028-03 - 2.0028+00 5.0028+00 5.0028+00 2.1328+01 2.028-03 mg/L Yes/F Sis-1,2-Dichiorostheme 1/11 1.0028-00 - 4.4028+00 5.0028+00 5.4628+01 2.1328+01 2.028-03 mg/L Yes/F Sis-1,2-Dichiorostheme 1/11 0.028-00 - 4.4028+00 5.0028+00 5.4628+01 2.1328+01 2.028-03 mg/L Yes/F Sis-1,2-Dichiorostheme 1/11 0.028-00 - 4.4028+00 5.0028+00 5.4028+01 2.1328+01 2.028-03 mg/L Yes/F Sis-1,2-Dichiorostheme 1/11 0.028-00 - 4.4028+00 5.0028+01 1.3228+01 2.028-01 PC1/L Yes/F Sis-1,2-Dichiorostheme 1/1 0.028-00 - 1.3228+00 - 1.3228+00 6.228-01 PC1/L No Diranium-238 1/1 1.028-00 - 1.3228+00 - 1.3228+00 6.228-01 PC1/L No Sis-200 PC	Potassium								No
Display 1/1 1.40E-02 1.40E-02 1.40E-02 2.0E-02 mg/L No Disthylphthalats 1/6 1.00E-03 1.00E-03 1.00E-03 1.00E-03 1.00E-03 1.2E+00 mg/L No Trichloroethane 8/13 1.00E-03 2.00E+00 1.70E-03 1.2E+03 1.4E-04 mg/L Yes/P Lipha activity 6/10 2.90E-01 1.40E+00 2.0E+01 0.68E+00 2.0E+03 mg/L Yes/P Seta activity 6/10 1.00E+00 4.40E+00 5.10E+00 2.0E+01 DC1/L Yes/P Thorium-314 1/1 0.00E+00 8.50E+00 1.03E+01 8.40E-01 8.00E-01 8.0E+01 2.0E+00 DC1/L Yes/P Tranum-238 1/1 1.32E+00 1.32E+00 1.32E+00 1.32E+00 5.2E-01 pC1/L Yes/P Analyte Detection Eange Range Mean HI ELCCR Units Basis* Aluminum 4/4 1.29E-01								mg/L	Yes/Qual
Distriphthalates 1/6 1.00E-02 4.00E-02 1.00E-02 1.00E-02 1.02E-02 1.2E+00 mg/L No Trichloroethane 8/13 1.00E-03 1.20E-03 1.20E-03 1.2E-03 1.4E-04 mg/L Yes/P tipha 1.00E-03 2.00E+00 1.00E-03 2.00E-04 4.00E+00 4.00E+00 4.00E+00 4.00E+00 4.00E+01 4.00E+01 4.00E+01 4.00E+01 4.00E+01 4.00E+01 4.00E+01 1.30E+01 2.0E+01 pc1/L Yes/P Thorium-34 1/1 0.00E+00 4.00E+01 1.40E+00 1.30E+01 2.0E+01 pc1/L Yes/P Thorium-234 1/1 0.00E+01 1.32E+00 1.32E+01 2.0E+01 pc1/L Yes/P Corranum-238 1/1 1.32E+00 1.32E+00 1.32E+00 6.2E-01 pc1/L Yes/P Auminum 4/4 1.29E-01 1.78E-01 1.43E-01 1.5E+00 mg/L No Aumonia 1/1 3.00E-01 3.00E-01	fetraoxo-sulfate (1-)								Yes/Qual
File S/13 1.00E-03 -1.00E-03 1.10E-02 1.70E-03 1.2E-03 1.4E-04 mg/L Yes/P lipha activity 6/10 2.00E+00 1.70E-01 1.70E-02 1.70E-03 1.4E-04 mg/L Yes/P lipha activity 6/10 2.00E+00 1.70E-01 1.45E+00 2.00E+00 4.44E+00 2.00E+01 4.44E+00 2.0E-03 pCi/L Yes/P Pachactivity 6/10 1.00E+00 4.00E+00 5.10E+00 1.30E+01 2.18E+01 pCi/L Yes/P Pachactivity 5/10 4.00E+00 8.50E+00 1.30E+01 8.40E+01 2.8E+01 pCi/L Yes/P Dranium-234 1/1 8.10E+01 8.10E+01 8.10E+01 8.7E+01 pCi/L Yes/P	cetone	1/1		1.40E-02 - 1.40E-02	1.40E-02	2.0E-02		mg/L	No
rrichiorcethene 8/13 1.008-03 -1.008-03 -1.008-02 1.708-03 1.28-03 1.48-04 mg/L Yes/P lipha activity 6/10 2.908-01 -1.008-00 -1.008-01 6.858+00 2.08-03 pCi/L Yes/P lipha activity 6/10 1.008-00 -4.08+00 5.408+00 -4.08+01 2.138+01 PCi/L Yes/P Pcincertaine-39 5/10 4.008+00 -8.008+01 -1.452+02 1.388+01 2.88+01 PCi/L Yes/P Dranium-234 1/1 8.008-01 -8.408-01 8.108-01 8.78-01 PCi/L Yes/P	Diethylphthalate	1/6	1.00B-02 - 4.00B-02	1.90E-02 - 1.90E-02	1.04E-02	1.2B+00		mg/L	No
Alpha activity 6/10 2.908-01 - 1.408+00 2.408+00 - 4.008+01 4.448+00 pC3/L Yes/ous Beta activity 8/10 1.008+00 - 4.608+00 5.108+01 2.88+01 pC3/L Yes/ous Technetium-99 5/10 4.008+00 - 6.508+00 1.408+01 - 1.458+02 1.388+01 2.08+00 pC3/L Yes/pu Technetium-234 1/1 8.108-01 - 8.408-01 8.408-01 8.408-01 8.78-01 pC3/L Yes/pu Oranium-236 1/1 1.328+00 - 1.328+00 1.328+00 6.28-01 pC3/L Yes/pu LOCATION-SWHU 193A MEDIA-RDA Groundwater LOCATION-SWHU 193A MEDIA-RDA Groundwater LOCATION-SWHU 193A MEDIA-RDA Groundwater LOCATION-SWHU 193A MEDIA-RDA Groundwater Analyte Detection Range Mean HI ELCE Units Basis* Aluminum 4/4 1.298-01 - 1.788-01 1.438-01 1.58+00 mg/L No Calcium 5/5 2.628/01 1.348+01 mg/L		8/13	1.00E-03 - 1.00E-03	2.00E-04 - 1.10E-02	1.70E-03	1.2E-03	1.4E-04	mg/L	Yes/P
Bein activity 8/10 1.00E+00 - 4.40E+00 5.10E+00 - 6.46E+01 2.13E+01 pCi/L Yes/Cua Thorium-334 1/1 4.00E+00 - 8.50E+00 1.00E+01 - 1.45E+02 1.38E+01 2.8E+01 2.0E+01 pCi/L Yes/Cua Thorium-334 1/1 8.40E-01 - 8.40E-01 8.40E-01 2.0E+00 pCi/L No Dranium-238 1/1 8.40E-01 - 8.40E-01 <	is-1,2-Dichloroethene	1/11	1.00E-03 - 2.00E+00	1.70E-01 - 1.70E-01	6.85E+00	2.0E-03		mg/L	Yes/P
Bein activity 8/10 1.00E+00 - 4.40E+00 5.10E+00 - 6.46E+01 2.13E+01 pCi/L Yes/Cua Thorium-334 1/1 4.00E+00 - 8.50E+00 1.00E+01 - 1.45E+02 1.38E+01 2.8E+01 2.0E+01 pCi/L Yes/Cua Thorium-334 1/1 8.40E-01 - 8.40E-01 8.40E-01 2.0E+00 pCi/L No Dranium-238 1/1 8.40E-01 - 8.40E-01 <	Alpha activity	6/10	2.90E-01 - 1.40E+00	2.40E+00 - 4.00E+01	4.44E+00			pCi/L	Yes/Qual
Technetium-99 5/10 4.008+00 8.508+00 1.008+01 1.458+02 1.388+01 2.88+01 pC1/L No Dranium-234 1/1 8.408-01		8/10	1.00E+00 - 4.40E+00	5.10B+00 - 6.46B+01	2.138+01		•		Yes/Qual
Thorium-234 1/1 8.408-01 8.408-01 8.408-01 8.108-01 8.108-01 8.7E-01 pCi/L No Dranium-238 1/1 8.108-01		5/10	4.00B+00 - 8.50B+00	1.00B+01 - 1.45B+02	1.38E+01		2.8E+01		
Dramium-234 1/1 8.108-01 8.108-01 8.108-01 8.78-01 pCi/L No Oranium-238 1/1 1.32E+00 1.32E+00 1.32E+00 6.2E-01 pCi/L Yes/P		1/1		8.40E-01 - 8.40E-01	8.40B-01		2.08+00		
Dramium-238 1/1 1.32E+00 - 1.32E+00 1.32E+00 6.2E-01 pC1/L Yes/P LOCATION-SWMU 193A MEDIA-RGA Groundwater Prequency of Analyte Nondetected Detection Detected Range Arithmetic Mean COPC/ Mean UI ELCR Units Basis* Aluminum 4/4 1.29E-01 - 1.78E-01 1.43E-01 1.5E+00 mg/L No Ammonia 1/1 3.00E-01 - 3.00E-01 3.00E-01 3.00E-01 mg/L No Chloride 5/5 2.62E+01 - 1.34E+02 5.95E+01 mg/L No Copper 1/4 1.00E-02 - 1.00E-02 1.80E-02 8.25E-03 6.0E-02 mg/L No Pluoride 1/1 0.00E-02 - 1.00E-02 1.80E-02 8.25E-03 6.0E-02 mg/L No Potassium 5/5 2.66E+00 2.93E+00 4.58-01 mg/L No Stlica 1/1 1.90E+01 1.90E+01 mg/L No Mg/L No Stlica 1/1 1.90E+01 1.90E+01 mg/L Mg/L No Stlica 1/				8.10E-01 - 8.10E-01	8.102-01		8.7E-01		
Interpretation Interpr					1.32E+00				
of Nanalyte Nondetected Detection Nondetected Range Detected Name Arithmetic Nean Fit COPC/ BLCR COPC/ Display Aluminum 4/4 1.29E-01 - 1.78E-01 1.48E-01 1.5E+00 mg/L No Aumonia 1/1 3.00E-01 - 3.00E-01 3.00E-01 1.5E+00 mg/L No Calcium 5/5 2.62E+01 - 1.34E+02 5.95E+01 mg/L No Chloride 5/5 1.30E+01 - 6.40E+01 3.10E+01 9.1E+02 mg/L No Copper 1/4 1.00E-02 - 1.00E-02 1.80E-02 - 3.66E+01 2.93E+00 4.5E-01 mg/L No Fluoride 1/1 4.20E-01 4.20E-01 4.20E-01 9.1E-02 mg/L No Iron 7/9 1.00E-02 - 1.00E-02 2.00E-02 - 3.66E+01 2.93E+00 4.5E-01 mg/L Yes/PE Magnesium 5/5 3.91E+00 1.85E+01 mg/L No Solium 5/5 2.10E+01 1.90E+01 1.90E+01 mg/L Yes/Qua </th <th></th> <th></th> <th> hackiton-sm</th> <th>U 193A MEDIA-RIA GIOUNO</th> <th></th> <th></th> <th></th> <th></th> <th></th>			hackiton-sm	U 193A MEDIA-RIA GIOUNO					
Analyte Detection Range Range Mean HI ELCR Units Basis* Aluminum 4/4 1.29E-01 - 1.78E-01 1.43E-01 1.5E+00 mg/L No Ammonia 1/1 3.00E-01 - 3.00E-01 3.00E-01 3.00E-01 mg/L Yes/Qua Calcium 5/5 2.62E+01 - 1.34E+02 5.96E+01 mg/L No Copper 1/4 1.00E-02 - 1.00E-02 1.80E-02 - 1.80E-02 8.25E-03 6.0E-02 mg/L No Fluoride 1/1 4.20E-01 - 4.20E-01 4.20E-01 9.1E-02 mg/L Yes/PE Iron 7/9 1.00E-02 - 1.00E-02 2.00E-02 - 3.66E+01 2.93E+00 4.5E-01 mg/L Yes/PE Iron 7/9 1.00E-02 - 1.00E-02 2.00E+02 - 6.10E+01 mg/L Yes/PE Magnesium 5/5 3.91E+00 1.85E+01 mg/L No Silica 1/1 1.90E+01 - 1.90E+01 1.90E+01 mg/L Yes/Qua Sodium 5/5 2.									
Auminium 4/4 1.29E-01 - 1.78E-01 1.43E-01 1.5E+00 mg/L No Ammonia 1/1 3.00E-01 - 3.00E-01 3.00R-01 mg/L No Calcium 5/5 2.62E+01 - 1.34E+02 5.96E+01 mg/L No Chloride 5/5 2.62E+01 - 1.34E+02 5.96E+01 mg/L No Copper 1/4 1.00E-02 - 1.008-02 1.80E-02 8.25E-03 6.0E-02 mg/L No Fluoride 1/1 4.20E-01 - 4.20E-01 4.20E-01 9.1E-02 mg/L Yes/PE Iron 7/9 1.00E-02 - 1.00E-02 2.00E-02 - 3.66E+01 2.93E+00 4.5E-01 mg/L Yes/PE Iron 7/9 1.00E-02 - 1.00E-02 2.06E+02 - 3.66E+01 2.93E+00 4.5E-01 mg/L Yes/PE Magnesium 5/5 3.91E+00 - 1.85E+01 1.05E+01 mg/L No Silica 1/1 1.90E+01 - 1.90E+01 1.90E+01 mg/L Yes/Qua Sodium 5/5 3.50E+01 - 2.62E+02 1.12E+01 mg/L Yes/Qua Sodium 5/5 3.50E+01			Nondatastad	Detected	3 mi thmot i a				
Ammonia 1/1 3.00E-01 - 3.00E-01 3.00E-01 mg/L Yes/Qua Calcium 5/5 2.62E+01 - 1.34E+02 5.96E+01 mg/L No Chloride 5/5 1:30E+01 - 6.40E+01 3.10E+01 mg/L No Copper 1/4 1.00E-02 - 1.00E-02 1.80E-02 - 8.25E-03 6.0E-02 mg/L No Fluoride 1/1 4.20E-01 - 4.20E-01 4.20E-01 9.1E-02 mg/L Yes/PE Iron 7/9 1.00E-02 - 1.00E-02 2.00E-02 - 3.66E+01 2.93E+00 4.5E-01 mg/L Yes/PE Magnesium 5/5 3.91E+00 - 1.85E+01 1.05E+01 mg/L No Silica 1/1 1.90E+01 - 1.90E+01 1.90E+01 mg/L Yes/PE Sodium 5/5 3.50E+01 - 1.34E+02 7.80E+01 mg/L Yes/Qua Tetraoxo-sulfate(1-) 5/5 2.10E+01 - 2.62E+02 1.12E+02 mg/L Yes/Qua Zinc 4/4 7.20E-02 - 2.12E+01 1.21E-01 4.5E-01 mg/L Yes/PE Diethylphthalate 3/25 1.00E-03 - 5.00E+00 1.00E-04	Analyte					HI	ELCR	Units .	
Calcium 5/5 2.62E+01 - 1.34E+02 5.96E+01 mg/L No Chloride 5/5 1.30E+01 - 6.40E+01 3.10E+01 mg/L No Copper 1/4 1.00E-02 - 1.00E-02 1.80E-02 - 1.80E-02 8.25E-03 6.0E-02 mg/L No Fluoride 1/1 4.20E-01 4.20E-01 9.1E-02 mg/L No Iron 7/9 1.00E-02 - 1.00E-02 2.00E-02 - 3.66E+01 2.93E+00 4.5E-01 mg/L Yes/PE Magnesium 5/5 3.91E+00 - 1.85E+01 1.05E+01 mg/L No Silica 1/1 1.90E+01 - 1.90E+01 1.90E+01 mg/L No Sodium 5/5 2.66E+00 - 2.65E+02 6.10E+01 mg/L Yes/Qua Sodium 5/5 2.10E+01 - 1.90E+01 1.90E+01 mg/L Yes/Qua Zinc 4/4 7.20E-02 - 2.12E+01 1.12E+02 mg/L Yes/Bio 1, 1-Dichloroethene 2/43 1.00E-03 - 5.00E+00 1.00E-04 - 2.00E-04 1.68E-04 1.8E-03 9.3E-07 mg/L Yes/Bio 1, 1-Dichloroethene 3/	-	Detection		Range .	Mean		ELCR		Basis*
Chloride 5/5 1.30E+01 - 6.40E+01 3.10E+01 mg/L No Copper 1/4 1.00E-02 - 1.00E-02 1.80E-02 - 1.80E-02 8.25E-03 6.0E-02 mg/L No Fluoride 1/1 4.20E-01 - 4.20E-01 4.20E-01 9.1E-02 mg/L Yes/PE Iron 7/9 1.00E-02 - 1.00E-02 2.00E-02 - 3.66E+01 2.93E+00 4.5E-01 mg/L Yes/PE Magnesium 5/5 3.91E+00 - 1.85E+01 1.05E+01 mg/L No Solium 5/5 3.91E+00 - 1.90E+01 1.90E+01 mg/L No solium 5/5 3.50E+01 - 1.90E+01 1.90E+01 mg/L Yes/Qua sodium 5/5 2.10E+01 - 2.62E+02 1.12E+02 mg/L Yes/Qua Zinc 4/4 7.20E-02 - 2.12E+01 1.21E+01 4.5E-01 mg/L Yes/Bio 1,1-Dichloroethene 2/43 1.00E-03 - 5.00E+00 1.00E-04 - 2.00E-04 1.68E-03 9.3E-07 mg/L Yes/Pio 1,21E-01chlorophenol 1/25 1.00E-02 - 4.00E-02 9.00E-03 - 1.50E-02 9.17E-03 1.2E+00 mg/L <td>Aluminum</td> <td>Detection 4/4</td> <td></td> <td>Range . 1.29B-01 - 1.78E-01</td> <td>Mean 1.43B-01</td> <td></td> <td>BLCR</td> <td>mg/L</td> <td>Basis* No</td>	Aluminum	Detection 4/4		Range . 1.29B-01 - 1.78E-01	Mean 1.43B-01		BLCR	mg/L	Basis* No
Copper 1/4 1.00E-02 - 1.00E-02 1.80E-02 - 1.80E-02 8.25E-03 6.0E-02 mg/L No Fluoride 1/1 4.20E-01 - 4.20E-01 4.20E-01 9.1E-02 mg/L Yes/PE Iron 7/9 1.00E-02 - 1.00E-02 2.00E-02 - 3.66E+01 2.93E+00 4.5E-01 mg/L Yes/PE Magnesium 5/5 3.91E+00 - 1.85E+01 1.05E+01 mg/L No Potassium 5/5 2.66E+00 - 2.65E+02 6.10E+01 mg/L No Solium 5/5 2.66E+00 - 2.65E+02 6.10E+01 mg/L No Solium 5/5 2.66E+00 - 2.65E+02 6.10E+01 mg/L Yes/Qua Tetraoxo-sulfate(1-) 5/5 2.10E+01 - 1.90E+01 1.90E+01 mg/L Yes/Qua Zinc 4/4 7.20E-02 - 2.12E+02 1.12E+02 mg/L Yes/Bid 1,1-Dichloroethene 2/43 1.00E-03 - 5.00E+00 1.00E-04 - 2.00E-04 1.68E-04 1.8E-03 9.3E-07 mg/L Yes/Pe Diethylphthalate 3/25 1.00E-02 - 4.00E-02 9.00E-03 - 1.50E-02 9.17E-03 1.2E+00 <t< td=""><td>- Aluminum Ammonia</td><td>Detection 4/4 1/1</td><td></td><td>Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01</td><td>Mean 1.43B-01 3.00B-01</td><td></td><td>BLCR</td><td>mg/L mg/L</td><td>Basis* No Yes/Qual</td></t<>	- Aluminum Ammonia	Detection 4/4 1/1		Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01	Mean 1.43B-01 3.00B-01		BLCR	mg/L mg/L	Basis* No Yes/Qual
Fluoride 1/1 4.20E-01 4.20E-01 4.20E-01 9.1E-02 mg/L Yes/PE Iron 7/9 1.00E-02 1.00E-02 2.00E-02 3.66E+01 2.93E+00 4.5E-01 mg/L Yes/PE Magnesium 5/5 3.91E+00 1.85E+01 1.05E+01 mg/L Yes/PE Magnesium 5/5 2.66E+00 2.65E+02 6.10E+01 mg/L No Potassium 5/5 2.66E+00 2.65E+02 6.10E+01 mg/L Yes/Qua Sodium 5/5 2.10E+01 1.90E+01 1.90E+01 mg/L Yes/Qua Tetraoxo-sulfate(1-) 5/5 2.10E+01 2.62E+02 1.12E+02 mg/L Yes/Qua Zinc 4/4 7.20E-02 2.12E+01 1.21E+01 4.5E-01 mg/L Yes/Bio 1,1-Dichloroethene 2/43 1.00E-03 5.00E+00 9.00E-03 1.50E-02 9.1E+03 9.3E+07 mg/L Yes/PE Diethylphthalate 3/25 1.00E-02 4.00E-02 9.00E-03 1.50E-02 9.17E-03 1.2E+00 mg/L No <	Aluminum Ammonia Calcium	Detection 4/4 1/1 5/5		Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01 2.62E+01 - 1.34E+02	Mean 1.43E-01 3.00E-01 5.96E+01		ELCR	mg/L mg/L mg/L	Basis* No Yes/Qual No
Iron 7/9 1.00E-02 - 1.00E-02 2.00E-02 - 3.66E+01 2.93E+00 4.5E-01 mg/L Yes/PE Magnesium 5/5 3.91E+00 - 1.85E+01 1.05E+01 mg/L No Potassium 5/5 2.66E+00 - 2.65E+02 6.10E+01 mg/L No Silica 1/1 1.90E+01 - 1.90E+01 1.90E+01 mg/L Yes/Qua Sodium 5/5 2.10E+01 - 2.62E+02 1.12E+01 mg/L Yes/Qua Tetraoxo-sulfate(1-) 5/5 2.10E+01 - 2.62E+02 1.12E+02 mg/L Yes/Qua Zinc 4/4 7.20E-02 - 2.12E-01 1.21E-01 4.5E-01 mg/L Yes/Bio 1, 1-Dichloroethene 2/43 1.00E-03 - 5.00E+00 1.00E-04 - 2.00E-02 9.17E-03 9.3E-07 mg/L Yes/PE Pentachlorophenol 1/25 1.00E-02 - 4.00E-02 1.50E-02 9.17E-03 2.3E-02 2.1E-05 mg/L Yes/PE	Aluminum Ammonia Calcium Chloride	Detection 4/4 1/1 5/5 5/5	Range	Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01 2.62E+01 - 1.34E+02 1.30E+01 - 6.40E+01	Mean 1.43E-01 3.00E-01 5.96E+01 3.10E+01	1.58+00	ELCR	mg/L mg/L mg/L mg/L	Basis* No Yes/Qual No No
Magnesium 5/5 3.91E+00 - 1.85E+01 1.05E+01 mg/L No Potassium 5/5 2.66E+00 - 2.65E+02 6.10E+01 mg/L No Silica 1/1 1.90E+01 - 1.90E+01 1.90E+01 mg/L Yes/Qua Sodium 5/5 2.10E+01 - 1.34E+02 7.80E+01 mg/L Yes/Qua Sodium 5/5 2.10E+01 - 2.62E+02 1.12E+02 mg/L Yes/Qua Zinc 4/4 7.20E-02 - 2.12E-01 1.21E+01 4.5E-01 mg/L Yes/Qua 1, 1-Dichloroethene 2/43 1.00E-03 - 5.00E+00 1.00E-04 - 2.00E-04 1.68E-03 9.3E-07 mg/L Yes/Bio Diethylphthalate 3/25 1.00E-02 - 4.00E-02 9.00E-03 - 1.50E-02 9.17E-03 1.2E+00 mg/L No Pentachlorophenol 1/25 1.00E-02 - 4.00E-02 1.20E-02 7.74E-03 2.3E-02 2.1E-05 mg/L Yes/P	Aluminum Aumonia Calcium Chloride Copper	Detection 4/4 1/1 5/5 5/5 1/4	Range	Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01 2.62E+01 - 1.34E+02 1.30E+01 - 6.40E+01 1.80E-02 - 1.80E-02	Mean 1.43B-01 3.00B-01 5.96E+01 3.10E+01 8.25E-03	1.5E+00 6.0E-02	ELCR	mg/L mg/L mg/L mg/L mg/L	Basis* No Yes/Qual No No No
Potassium 5/5 2.66E+00 - 2.65E+02 6.10E+01 mg/L No Silica 1/1 1.90E+01 - 1.90E+01 1.90E+01 1.90E+01 mg/L Yes/Qua Sodium 5/5 3.50E+01 - 1.34E+02 7.80E+01 mg/L Yes/Qua Tetraoxo-sulfate(1-) 5/5 2.10E+01 - 2.62E+02 1.12E+02 mg/L Yes/Qua Zinc 4/4 7.20E-02 - 2.12E-01 1.21E+01 4.5E-01 mg/L Yes/Qua 1,1-Dichloroethene 2/43 1.00E-03 - 5.00E+00 1.00E-04 2.60E-04 1.68E-03 9.3E-07 mg/L Yes/P Diethylphthalate 3/25 1.00E-02 - 4.00E-02 9.0E-03 1.50E-02 9.17E-03 1.2E+00 mg/L No Pentachlorophenol 1/25 1.00E-02 - 4.00E-02 1.20E-02 7.74E-03 2.3E-02 2.1E-05 mg/L Yes/P	Aluminum Aumonia Calcium Chloride Copper	Detection 4/4 1/1 5/5 5/5 1/4 1/1	Range	Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01 2.62E+01 - 1.34E+02 1.30E+01 - 6.40E+01 1.80E-02 - 1.80E-02	Mean 1.43B-01 3.00B-01 5.96E+01 3.10E+01 8.25E-03 4.20E-01	1.5E+00 6.0E-02	ELCR	mg/L mg/L mg/L mg/L mg/L	Basis* No Yes/Qual No No No
Silica 1/1 1.90E+01 - 1.90E+01 1.90E+01 mg/L Yes/Qua Sodium 5/5 3.50E+01 - 1.34E+02 7.80E+01 mg/L Yes/Qua Tetraoxo-sulfate(1-) 5/5 2.10E+01 - 2.62E+02 1.12E+02 mg/L Yes/Qua Zinc 4/4 7.20E-02 - 2.12E-01 1.21E-01 4.5E-01 mg/L Yes/Bic 1,1-Dichloroethene 2/43 1.00E-03 - 5.00E+00 1.00E-04 - 2.00E-04 1.68E-04 1.8E-03 9.3E-07 mg/L Yes/Bic Diethylphthalate 3/25 1.00E-02 - 4.00E-02 9.00E-03 - 1.50E-02 9.17E-03 1.2E+00 mg/L No Pentachlorophenol 1/25 1.00E-02 - 4.00E-02 1.20E-02 - 7.74E-03 2.3E-02 2.1E-05 mg/L Yes/P	Aluminum Ammonia Calcium Chloride Copper Fluoride	Detection 4/4 1/1 5/5 5/5 1/4 1/1 7/9	Range 1.00E-02 - 1.00E-02	Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01 2.62E+01 - 1.34E+02 1.30E+01 - 6.40E+01 1.80E-02 - 1.80E-02 4.20E-01 - 4.20E-01	Mean 1.43B-01 3.00B-01 5.96E+01 3.10E+01 8.25E-03 4.20E-01	1.5E+00 6.0E-02 9.1E-02	ELCR	mg/L mg/L mg/L mg/L mg/L mg/L	Basis* No Yes/Qual No No Yes/PE
Silica 1/1 1.90E+01 - 1.90E+01 1.90E+01 mg/L Yes/Qua Sodium 5/5 3.50E+01 - 1.34E+02 7.80E+01 mg/L Yes/Qua Tetraoxo-sulfate(1-) 5/5 2.10E+01 - 2.62E+02 1.12E+02 mg/L Yes/Qua Zinc 4/4 7.20E-02 - 2.12E-01 1.21E+01 4.5E-01 mg/L Yes/Bio 1,1-Dichloroethene 2/43 1.00E-03 - 5.00E+00 1.00E-04 - 2.00E-04 1.68E-04 1.8E-03 9.3E-07 mg/L Yes/Bio Diethylphthalate 3/25 1.00E-02 - 4.00E-02 9.0E-03 - 1.50E-02 9.17E-03 1.2E+00 mg/L No Pentachlorophenol 1/25 1.00E-02 - 4.00E-02 1.20E-02 - 7.74E-03 2.3E-02 2.1E-05 mg/L Yes/P	Aluminum Ammonia Calcium Chloride Copper Fluoride Iron	Detection 4/4 1/1 5/5 5/5 1/4 1/1 7/9	Range 1.00E-02 - 1.00E-02	Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01 2.62E+01 - 1.34E+02 1.30E+01 - 6.40E+01 1.80E-02 - 1.80E-02 4.20E-01 - 4.20E-01 2.00E-02 - 3.66E+01	Mean 1.43B-01 3.00E-01 5.96E+01 3.10E+01 8.25E-03 4.20E-01 2.93E+00	1.5E+00 6.0E-02 9.1E-02	ELCR	mg/L mg/L mg/L mg/L mg/L mg/L mg/L	Basis* No Yes/Qual No No Yes/PE Yes/PE
Sodium 5/5 3.50E+01 - 1.34E+02 7.80E+01 mg/L Yes/Qua Tetraoxo-sulfate(1-) 5/5 2.10E+01 - 2.62E+02 1.12E+02 mg/L Yes/Qua Zinc 4/4 7.20E-02 - 2.12E-01 1.21E-01 4.5E-01 mg/L Yes/Qua 1,1-Dichloroethene 2/43 1.00E-03 - 5.00E+00 1.00E-04 - 2.00E-04 1.68E-04 1.8E-03 9.3E-07 mg/L Yes/P Diethylphthalate 3/25 1.00E-02 - 4.00E-02 9.00E-03 - 1.50E-02 9.17E-03 1.2E+00 mg/L No Pentachlorophenol 1/25 1.00E-02 - 4.00E-02 1.20E-02 - 7.74E-03 2.3E-02 2.1E-05 mg/L Yes/P	Aluminum Ammonia Calcium Chloride Copper Fluoride Iron Magnesium	Detection 4/4 1/1 5/5 5/5 1/4 1/1 7/9 5/5	Range 1.00E-02 - 1.00E-02	Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01 2.62E+01 - 1.34E+02 1.30E+01 - 6.40E+01 1.80E-02 - 1.80E-02 4.20E-01 - 4.20E-01 2.00E-02 - 3.66E+01 3.91E+00 - 1.85E+01	Mean 1.43E-01 3.00E-01 5.96E+01 3.10E+01 8.25E-03 4.20E-01 2.93E+00 1.05E+01	1.5E+00 6.0E-02 9.1E-02	ELCR	mg/L mg/L mg/L mg/L mg/L mg/L mg/L	Basis* No Yes/Qual No No Yes/PE Yes/PE No
Tetraoxo-sulfate(1-) 5/5 2.10E+01 - 2.62E+02 1.12E+02 mg/L Yes/Qua Zinc 4/4 7.20E-02 - 2.12E-01 1.21E-01 4.5E-01 mg/L Yes/Gua 1,1-Dichloroethene 2/43 1.00E-03 - 5.00E+00 1.00E-04 - 2.00E-04 1.68E-04 1.8E-03 9.3E-07 mg/L Yes/Bio Diethylphthalate 3/25 1.00E-02 - 4.00E-02 9.00E-03 - 1.50E-02 9.17E-03 1.2E+00 mg/L No Pentachlorophenol 1/25 1.00E-02 - 4.00E-02 1.20E-02 - 7.74E-03 2.3E-02 2.1E-05 mg/L Yes/P	Aluminum Ammonia Calcium Chloride Copper Fluoride Iron Magnesium Potassium	Detection 4/4 1/1 5/5 5/5 1/4 1/1 7/3 5/5 5/5	Range 1.00E-02 - 1.00E-02	Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01 2.62E+01 - 1.34E+02 1.30E+01 - 6.40E+01 1.80E-02 - 1.80E-02 4.20E-01 - 4.20E-01 2.00E-02 - 3.66E+01 3.91E+00 - 1.85E+01 2.66E+00 - 2.65E+02	Mean 1.43E-01 3.00E-01 5.96E+01 3.10E+01 8.25E-03 4.20E-01 2.93E+00 1.05E+01 6.10E+01	1.5E+00 6.0E-02 9.1E-02	ELCR	mg/L mg/L mg/L mg/L mg/L mg/L mg/L mg/L	Basis* No Yes/Qual No No Yes/PE Yes/PE No No
Zinc 4/4 7.20E-02 - 2.12E-01 1.21E-01 4.5E-01 mg/L Yes/Bic 1,1-Dichloroethene 2/43 1.00E-03 - 5.00E+00 1.00E-04 - 2.00E-04 1.68E-04 1.8E-03 9.3E-07 mg/L Yes/P Diethylphthalate 3/25 1.00E-02 - 4.00E-02 9.00E-03 - 1.50E-02 9.17E-03 1.2E+00 mg/L No Pentachlorophenol 1/25 1.00E-02 - 4.00E-02 1.20E-02 - 7.74E-03 2.3E-02 2.1E-05 mg/L Yes/P	Aluminum Ammonia Calcium Chloride Copper Fluoride Iron Magnesium Potassium Silica	Detection 4/4 1/1 5/5 5/5 1/4 1/1 7/9 5/5 5/5 1/1	Range 1.00E-02 - 1.00E-02	Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01 2.62E+01 - 1.34E+02 1.30E+01 - 6.40E+01 1.80E-02 - 1.80E-02 4.20E-01 - 4.20E-01 2.00E-02 - 3.66E+01 3.91E+00 - 1.85E+01 2.65E+00 - 2.65E+02 1.90E+01 - 1.90E+01	Mean 1.43E-01 3.00E-01 5.96E+01 3.10E+01 8.25E-03 4.20E-01 2.93E+00 1.05E+01 6.10E+01 1.90E+01	1.5E+00 6.0E-02 9.1E-02	ELCR	mg/L mg/L mg/L mg/L mg/L mg/L mg/L mg/L	Basis* No Yes/Qual No No Yes/PE Yes/PE No Yes/Qual
1,1-Dichloroethene 2/43 1.00E-03 - 5.00E+00 1.00E-04 - 2.00E-04 1.68E-04 1.8E-03 9.3E-07 mg/L Yes/P Diethylphthalate 3/25 1.00E-02 - 4.00E-02 9.00E-03 - 1.50E-02 9.17E-03 1.2E+00 mg/L No Pentachlorophenol 1/25 1.00E-02 - 4.00E-02 1.20E-02 - 1.20E-02 7.74E-03 2.3E-02 2.1E-05 mg/L Yes/P	Aluminum Ammonia Calcium Chloride Copper Fluoride Iron Magnesium Potassium Silica Sodium	Detection 4/4 1/1 5/5 5/5 1/4 1/1 7/9 5/5 5/5 1/1 5/5	Range 1.00E-02 - 1.00E-02	Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01 2.62E+01 - 1.34E+02 1.30E+01 - 6.40E+01 1.80E-02 - 1.80E-02 4.20E-01 - 4.20E-01 2.00E-02 - 3.66E+01 3.91E+00 - 1.85E+01 2.66E+00 - 2.65E+02 1.90E+01 - 1.90E+01 3.50E+01 - 1.34E+02	Mean 1.43E-01 3.00E-01 5.96E+01 3.10E+01 8.25E-03 4.20E-01 2.93E+00 1.05E+01 6.10E+01 1.90E+01 7.80E+01	1.5E+00 6.0E-02 9.1E-02	ELCR	mg/L mg/L mg/L mg/L mg/L mg/L mg/L mg/L	Basis* No Yes/Qual No No Yes/PE Yes/PE No No Yes/Qual Yes/Qual
Diethylphthalate 3/25 1.00E-02 - 4.00E-02 9.00E-03 - 1.50E-02 9.17E-03 1.2E+00 mg/L No Pentachlorophenol 1/25 1.00E-02 - 4.00E-02 1.20E-02 - 1.20E-02 7.74E-03 2.3E-02 2.1E-05 mg/L Yes/P	Aluminum Ammonia Calcium Chloride Copper Fluoride Iron Magnesium Potassium Silica Sodium Tetraomo-sulfate(1-)	Detection 4/4 1/1 5/5 5/5 1/4 1/1 7/9 5/5 5/5 1/1 5/5 5/5	Range 1.00E-02 - 1.00E-02	Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01 2.62E+01 - 1.34E+02 1.30E+01 - 6.40E+01 1.80E-02 - 1.80E-02 4.20E-01 - 4.20E-01 2.00E-02 - 3.66E+01 3.91E+00 - 1.85E+01 2.66E+00 - 2.65E+02 1.90E+01 - 1.90E+01 3.50E+01 - 1.34E+02 2.10E+01 - 2.62E+02	Mean 1.43B-01 3.00E-01 5.96E+01 3.10E+01 8.25E-03 4.20B-01 2.93E+00 1.05E+01 6.10E+01 1.90E+01 7.80E+01 1.12E+02	1.5E+00 6.0E-02 9.1E-02 4.5E-01	ELCR	mg/L mg/L mg/L mg/L mg/L mg/L mg/L mg/L	Basis* No Yes/Qual No No Yes/PE Yes/PE No No Yes/Qual Yes/Qual Yes/Qual
Pentachlorophenol 1/25 1.00E-02 - 4.00E-02 1.20E-02 - 1.20E-02 7.74E-03 2.3E-02 2.1E-05 mg/L Yes/P	Aluminum Ammonia Calcium Chloride Copper Fluoride Iron Magnesium Potassium Silica Sodium Tetraoxo-sulfate (1-) Zinc	Detection 4/4 1/1 5/5 5/5 1/4 1/1 7/9 5/5 5/5 1/1 5/5 5/5 4/4	Range 1.00E-02 - 1.00E-02 1.00E-02 - 1.00E-02	Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01 2.62E+01 - 1.34E+02 1.30E+01 - 6.40E+01 1.80E-02 - 1.80E-02 4.20E-01 - 4.20E-01 2.00E+02 - 3.66E+01 3.91E+00 - 2.65E+02 1.90E+01 - 1.90E+01 3.50E+01 - 1.34E+02 2.10E+01 - 2.62E+02 7.20E-02 - 2.12E-01	Mean 1.43B-01 3.00E-01 5.96E+01 3.10E+01 8.25E-03 4.20B-01 2.93E+00 1.05E+01 6.10E+01 1.90E+01 1.12E+02 1.21E-01	1.5E+00 6.0E-02 9.1E-02 4.5E-01		ng/L ng/L ng/L ng/L ng/L ng/L ng/L ng/L	Basis* No Yes/Qual No No No Yes/PE Yes/PE No No Yes/Qual Yes/Qual Yes/Qual Yes/Bio
	Aluminum Ammonia Calcium Chloride Copper Fluoride Iron Magnesium Potassium Silica Sodium Tetraoxo-sulfate(1-) Zinc 1,1-Dichloroethene	Detection 4/4 1/1 5/5 5/5 1/4 1/1 7/9 5/5 5/5 1/1 5/5 5/5 4/4 2/43	Range 1.00E-02 - 1.00E-02 1.00E-02 - 1.00E-02 1.00E-03 - 5.00E+00	Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01 2.62E+01 - 1.34E+02 1.30E+01 - 6.40E+01 1.80E-02 - 1.80E-02 4.20E-01 - 4.20E-01 2.00E-02 - 3.66E+01 3.91E+00 - 1.85E+01 2.66E+00 - 2.65E+02 1.90E+01 - 1.90E+01 3.50E+01 - 1.34E+02 2.10E+01 - 2.62E+02 1.00E-04 - 2.00E-04	Mean 1.43E-01 3.00E-01 5.96E+01 3.10E+01 8.25E-03 4.20E-01 2.93E+00 1.05E+01 1.90E+01 1.90E+01 1.12E+02 1.21E-01 1.68E-04	6.0E-02 9.1E-02 4.5E-01 4.5E-01 1.8E-03		ng/L ng/L ng/L ng/L ng/L ng/L ng/L ng/L	Basis* No Yes/Qual No No Yes/PE Yes/PE No No Yes/Qual Yes/Qual Yes/Qual Yes/Bio Yes/P
Trichloroethene 44/51 1.00E-03 - 1.00E-03 2.00E-04 - 6.70E+00 7.76E-02 1.2E-03 1.4E-04 mg/L Yes/P	Aluminum Ammonia Calcium Chloride Copper Fluoride Iron Magnesium Potassium Silica Sodium Tetraoxo-sulfate(1-) Zinc 1,1-Dichloroethene Diethylphthalate	Detection 4/4 1/1 5/5 5/5 1/4 1/1 7/9 5/5 5/5 1/1 5/5 5/5 4/4 2/43 3/25	Range 1.00E-02 - 1.00E-02 1.00E-02 - 1.00E-02 1.00E-03 - 5.00E+00 1.00E-02 - 4.00E-02	Range 1.29E-01 - 1.78E-01 3.00E-01 - 3.00E-01 2.62E+01 - 1.34E+02 1.30E+01 - 6.40E+01 1.80E-02 - 1.80E-02 4.20E-01 - 4.20E-01 2.00E-02 - 3.66E+01 3.91E+00 - 1.85E+01 2.66E+00 - 2.65E+02 1.90E+01 - 1.90E+01 3.50E+01 - 1.34E+02 2.10E+01 - 2.62E+02 7.20E-02 - 2.12E-01 1.00E-04 - 2.00E-04 9.00E-03 - 1.50E-02	Mean 1.43E-01 3.00E-01 5.96E+01 3.10E+01 8.25E-03 4.20E-01 2.93E+00 1.05E+01 1.90E+01 1.90E+01 1.12E+02 1.21E-01 1.66E-04 9.17E-03	1.5E+00 6.0E-02 9.1E-02 4.5E-01 4.5E-01 1.8E-03 1.2E+00	9.3E-07	mg/L mg/L mg/L mg/L mg/L mg/L mg/L mg/L	Basis* No Yes/Qual No No Yes/PE Yes/PE No Yes/Qual Yes/Qual Yes/Qual Yes/Bio Yes/P No

*P= > PRG, B= > Background, E= > Essential Nutrient, Bio=Bioaccumulates, Qual=Qualitative analyte

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LOCATION=SWMU 193A MEDIA=RGA Groundwater (continued)								
Analyte	Frequency of Detection	Nondetected Range	Detected Range	Arithmetic Mean	HI	ELCR	Units	COPC/ Basis*
bis (2-Ethylhexyl) phthalate cis-1,2-Dichloroethene trans-1,2-Dichloroethene Alpha activity Beta activity Technetium-99 Thorium-234	3/25 17/42 7/43 19/34 34/34 26/39 1/8	1.00E-02 - 4.00E-02 1.00E-03 - 5.00E+00 1.00E-03 - 5.00E+00 -4.10E-01 - 4.00E+00 -7.60E+00 - 1.70E+01 -1.65E+02 - 5.58E+01	1.30E-02 - 2.20E-02 1.00E-04 - 8.40E-02 1.00E-04 - 7.00E-04 1.50E+00 - 1.76E+01 2.90E+00 - 8.80E+02 8.00E+00 - 1.39E+03 5.40E-01 - 5.40E-01	7.94E-03 1.51E-03 3.07E-04 3.64E+00 8.53E+01 1.21E+02 -2.51E+01	2.6E-02 2.0E-03 4.0E-03	3.1E-04 2.8E+01 2.0E+00	mg/L mg/L pCi/L pCi/L pCi/L pCi/L	Yes/P Yes/P No Yes/Qual Yes/Qual Yes/P No

----- LOCATION=8WMU 193A MEDIA=Subsurface soil ------

Analyte	Frequency of Detection	Nondetected Range	Detected Range	Arithmetic Mean	HI	ELCR	Units	COPC/ Basis*
Aluminum	8/8		3.01E+03 - 1.40E+04	9.71E+03	7.3E+02		mg/kg	Yes/PB
Barium	8/8		2.16E+01 - 8.73E+01	5.66E+01	3.7E+01		tng/kg	No
Beryllium	5/8	5.00E-01 - 5.00E-01	5.20E-01 - 7.00E-01	4.60E-01	1.6E-01	1.0E-04	mg/kg	Yes/PB
Calcium	4/4		1.06E+03 - 2.73E+05	9.07E+04		•	ng/kg	No
Chromium	8/8		4.318+00 - 2.77E+01	1.65E+01	4.8E-01	4.9E+02	tag/kg	Yes/P
Cobalt	8/8		1.47E+00 - 8.66E+00	4.13E+00	2.1E+02		mg/kg	No
Copper	8/8		2.45E+00 - 7.31E+00	4.97E+00	7.4E+01		mg/kg	No
Iron	8/8		3.74E+03 - 1.54E+04	1.11E+04	3.1E+02		mg/kg	No
Lithium	8/8		3.78E+00 - 1.12E+01	7.14E+00	7.0E+01		mg/kg	No
Magnesium	8/8		1.16E+03 - 1.70E+04	3.92E+03			mg/kg	No
Manganese	8/8	•	4.86E+01 - 5.64E+02	2.30E+02	1.4E+01		ng/kg	No
Nickel	5/8	5.00E+00 - 5.00E+00	5.50E+00 - 9.16E+00	5.42E+00	3.4E+01		tag/kg	No
Potassium	8/8		2.898+02 - 1.448+03	5.27E+02			mg/kg	No
Silver	1/8	4.00E+00 - 4.00E+00	4.00E+00 - 4.00E+00	2.25E+00	6.1E+00		mg/kg	No
Sodium	5/8	2.00E+02 - 2.00E+02	2.13E+02 - 3.13E+02	1.94E+02			mg/kg	No
Strontium	8/8		6.36E+00 - 2.53E+02	7.47E+01	8.0E+02		mg/kg	No
Vanadium	8/8		5.67E+00 - 3.15E+01	2.18E+01	5.6B-01		mg/kg	No
Zinc	8/8		1.84E+01 - 5.54E+01	3.45E+01	4.0E+02		ng/kg	No
Acetone	1/2	1.00E-02 - 1.00E-02	1.10E-02 - 1.10E-02	8.00E-03	9.2E+01		mg/kg	No
Anthracene	1/8	5.00E-01 - 5.00E-01	1.16E-01 - 1.16E-01	2.33E-01	6.5E+02		mg/kg	Yes/Bio
Benz (a) anthracene	2/8	5.00B-01 - 5.00B-01	1.60E-01 - 1.80E-01	1.75B-01		8.5E-03	mg/kg	Yes/P
Benzo (a) pyrene	2/8	5.00E-01 - 5.00E-01	2.40E-01 - 2.50E-01	2.49E-01		8.5E-04	mg/kg	Yes/P
Benzo (b) fluoranthene	2/8	5.00B-01 - 5.00B-01	3.90E-02 - 5.10E-02	4.77E-02		8.5E-03	mg/kg	Yes/P
Benzo (ghi) perylene	2/8	5.00E-01 - 5.00E-01	1.66E-01 - 1.70E-01	1.69E-01			mg/kg	Yes/Qual
Chrysene	2/8	5.00B-01 - 5.00B-01	1.70E-01 - 1.70E-01	2.30E-01	•	8.5B-01	mg/kg	Yes/Bio

*P. > PRG, B. > Background, E. > Essential Nutrient, Bio-Bioaccumulates, Qual-Qualitative analyte

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		Location=SWM	(continued)	B011				
	Frequency		Debash J	Arithmetic				
Analyte	of Detection	Nondetected Range	Detected Range	Mean	HI	ELCR	Units	COPC/ Basis*
Di-n-butylphthalate	· 1/8	5.00E-01 - 6.60E-01	7.70B-02 - 7.70B-02	2.38E-01	2.6B+02		mg/kg	Yes/Bio
Di-n-octylphthalate	1/8	5.00E-01 - 5.00E-01	1.20E-01 - 1.20E-01	2.34B-01	4.9E+01		mg/kg	Yes/Bio
Dibenz (a, h) anthracene	1/8	5.00E-01 - 5.00B-01	1.30E-01 - 1.30E-01	2.35B-01		8.5E-04	mg/kg	Yes/P
Diethylphthalate	1/8	5.00E-01 - 5.00E-01	4.00E-01 - 4.00E-01	2.69E-01	2.0E+03		mg/kg	No
Fluoranthene	2/8	5.00E-01 - 5.00E-01	2.30E-01 - 3.10E-01	2.88E-01	4.3E+01		mg/kg	Yes/Bio
Indeno (1, 2, 3-cd) pyrene	2/8	5.00E-01 - 5.00E-01	1.38E-01 - 1.60E-01	1.54E-01		8.5B-03	mg/kg	Yes/P
Pyrene	2/8	5.00B-01 - 5.00E-01	2.40E-02 - 2.95E-01	2.27B-01	3.2E+01		mg/kg	Yes/Bic
bis(2-Sthylhexyl)phthalate	2/8	5.00E-01 - 5.00E-01	8.10E-02 - 1.70E-01	2.19E-01	1,4E+01	2.8E-01	mg/kg	Yes/Bio
Alpha activity	8/8		9.60E+00 - 2.60E+01	1.62E+01			pCi/g	Yes/Qua
Beta activity	8/8		1.41E+01 - 2.37E+01	1.908+01			pCi/g	Yes/Qua
·		LOCATION-SW	MU 193A MEDIA-Surface So	oil				
	Frequency							
	of	Nondetected	Detected	Arithmetic				COPC/
Analyte	Detection	Range	Range	Mean	HI	ELCR	Units	Basis*
Aluminum	4/4	•	3.01E+03 - 1.09E+04	7.24E+03	7.38+02		mg/kg	No
Barium	4/4		2.16E+01 - 8.40E+01	5.34E+01	3.7E+01		mg/kg	No
Beryllium	1/4	5.00E-01 - 5.00E-01	6.40E-01 - 6.40E-01	3.48E-01	1.6E-01	1.0E-04	mg/kg	No
Calcium	2/2		8.76E+04 - 2.73E+05	1.80E+05			ng/kg	No
Chromium	4/4		4.31E+00 - 2.65E+01	1.29B+01	4.8E-01	4.9E+02	mg/kg	Yes/P
Cobalt	4/4		1.47E+00 - 5.70E+00	3.36E+00	2.1E+02		ng/kg	No
Copper	4/4		2.45E+00 - 7.31E+00	5.32E+00	7.4B+01		ng/kg	No
Iron	4/4		3.74 8 +03 - 1.54 8 +04	9.39E+03	3.1E+02		mg/kg	No
Lithium	4/4		3.785+00 - 1.125+01	6.94E+00	7.0E+01		mg/kg	No
Magnesium	4/4		1.66E+03 - 1.70E+04	6.91E+03			ng/kg	No
Manganese	4/4		1.35E+02 - 3.98E+02	2.14E+02	1.4E+01		mg/kg	No
Nickel	2/4	5.00E+00 - 5.00E+00	7.27E+00 - 7.50E+00	4.94E+00	3.4E+01		mq/kq	No
Potassium	4/4		2.89E+02 - 1.44E+03	6.78E+02			mg/kg	No
Sodium	1/4	2.00E+02 - 2.00E+02	2.13E+02 - 2.13E+02	1.28E+02		-	mg/kg	No
Strontium	4/4		1.21E+01 - 2.53E+02	1.22E+02	8.0E+02		mg/kg	No
Vanadium	4/4		5.67B+00 - 3.15B+01	1.76B+01	5.6B-01		mg/kg	No
Zinc	4/4		3.34E+01 - 5.54E+01	4.64E+01	4.0E+02		mg/kg	No
Anthracene	1/4	5.00B-01 - 5.00B-01	1.16E-01 - 1.16E-01	2.17E-01	6.5E+02		mg/kg	Yes/Bic
Benz (a) anthracene	2/4	5.00E-01 - 5.00E-01	1.60E-01 - 1.80E-01	2.10B-01		8.5E-03	mg/kg	Yes/P
Benzo (a) pyrene	2/4	5.00E-01 - 5.00E-01	2.40E-01 - 2.50E-01	2.48E-01		8.5E-04	mg/kg	Yes/P
Benzo (b) fluoranthene	2/4	5.00E-01 - 5.00E-01	3.908-02 - 5.108-02	1.488-01		8.5E-03	ng/kg	Yes/P
Benzo (ghi) perylene	2/4	5.00E-01 - 5.00E-01	1.66E-01 - 1.70E-01	2.09E-01		0.00-03	ug/kg	Yes/Qua

*P= > FRG, B= > Background, E= > Essential Nutrient, Bio=Bioaccumulates, Qual=Qualitative analyte

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Frequency

----- LOCATION-SWMU 193A MEDIA-Surface Soil ------(continued)

	of	Nondetected	Detected	Arithmetic				COPC/
Analyte	Detection	Range	Range	Mean	HI	ELCR	Units	Basis*
Chrysene	2/4	5.008-01 - 5.002-01	1.70E-01 - 1.70E-01	2.10E-01		8.5R-01	mg/kg	Yes/Bio
Di-n-butylphthalate	1/4	5.00B-01 - 6.60B-01	7.70B-02 - 7.70B-02	2.27E-01	2.62+02		ng/kg	Yes/Bio
Di-n-octylphthalate	1/4	5.00B-01 - 5.00E-01	1.20E-01 - 1.20E-01	2.18E-01	4.9B+01		mg/kg	Yes/Bio
Dibenz (a, h) anthracene	1/4	5.00B-01 - 5.00E-01	1.30E-01 - 1.30E-01	2.20E-01		8.5E-04	ng/kg	Yes/P
Diethylphthalate	1/4	5.00B-01 - 5.00B-01	4.00E-01 - 4.00E-01	2.08E-01	2.0E+03		mg/kg	No
Fluoranthene	2/4	5.00E-01 - 5.00E-01	2.308-01 - 3.108-01	2.60E-01	4.3E+01	•	mg/kg	Yes/Bio
Indeno (1, 2, 3-cd) pyrene	2/4	5.00B-01 - 5.00B-01	1.38E-01 - 1.60E-01	2.00B-01		8.5 <u>8</u> -03	mg/kg	Yes/P
Pyrene	2/4	5.00B-01 - 5.00B-01	2,40E-02 - 2,95E-01	2.05B-01	3.2E+01		mg/kg	Yes/Bio
bis (2-Ethylhexyl) phthalate	2/4	5.00B-01 - 5.00E-01	8.10E-02 - 1.70E-01	1.88E-01	1.4B+01	2.8E-01	mg/kg	Yes/Bio
Alpha activity	<u>4/4</u>		9.60E+00 - 1.70E+01	1.32B+01			pCi/g	Yes/Qual
Beta activity	4/4		1.41B+01 - 2.37B+01	2.10E+01			pCi/g	Yes/Qual
		LOCATION-SWMU	1938 MEDIA-McNairy Groun	dwater				
	Frequency of	Nondetected	Detected	Arithmetic				COPC/
Analyte	Detection	Range	Range	Mean	HI	ELCR	Units	Basis*
Trichloroethene	1/2	1.00E-03 - 1.00E-03	1.30B-02 - 1.30B-02	7.00B-03	1.2E-03	1.4B-04	mg/L	Yes/P
cis-1,2-Dichloroethene	1/2	1.00E-03 - 1.00E-03	2.30B-02 - 2.30B-02	1.20E-02	2.0E-03		mg/L	Yes/P
Alpha activity	1/2	1.20E+00 - 1.20E+00	1.29E+00 - 1.29E+00	1.25E+00			pČi/L	Yes/Qual
Beta activity	2/2		3.15E+00 - 4.80E+00	3.98B+00			pCi/L	Yes/Qual
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------ LOCATION-SMMU 193B MEDIA-RGA Groundwater -----

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Analyte	Frequency of Detection	Nondetected Range	Detected Range	Arithmetic Mean	HI	ELCR	Units	COPC/ Basis*
1,1-Dichloroethene	3/17	1.908-04 - 5.00E-02	2.30B-04 - 2.00E-02	3.36E-04	1.8E-03	9.3E-07	mg/L	Yes/P
Acetone	1/2	1.00B-02 - 1.00B-02	3.30E-02 - 3.30E-02	1.90Ê-02	2.0E-02		mg/L	Yes/P
Carbon Tetrachloride	1/5	5.00E-03 - 5.00E-02	5.50B-03 - 5.50B-03	1.21E-02	1.2E-04	1.5E-05	mg/L	Yes/P
Di-n-butylphthalate	2/10	2.00E-02 - 2.00E-02	1.30E-02 - 1.30E-02	1.06E-02	1.3E-01		mg/L	Yes/Bio
Trichloroethene	17/17	•	1.00B-04 - 5.00B-01	8.01E-01	1.2B-03	1.4E-04	mg/L	Yes/P
bis (2-Ethylhexyl) phthalate	1/10	2.00E-02 - 2.00E-02	1.80E-02 - 1.80E-02	1.08E-02	2.6E-02	3.1E-04	ung/L	Yes/P
cis-1,2-Dichloroethene	12/17	2.20E-04 - 5.00E-03	1.90B-04 - 9.87E-02	2.92E-03	2.0E-03		mg/L	Yes/P
trans-1,2-Dichloroethene	8/17	4.90E-04 - 2.00E+00	1.00B-04 - 8.10B-04	3.29B-04	4.0E-03		ng/L	No
Alpha activity	12/17	1.10E+00 - 2.80E+00	1.00E+00 - 6.60E+02	6.09E+00			pCi/L	Yes/Qual

*P= > PRG, B= > Background, E= > Essential Nutrient, Bio-Bioaccumulates, Qual=Qualitative analyte

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		LOCATION-SWM	U 193B MEDIA-RGA Groundw (continued)	ater				
			(cont muse)					
	Frequency							
	of	Nondetected	Detected	Arithmetic				COPC/
Analyte	Detection	Range	Range	Mean	HI	BLCR	Units	Basis*
Beta activity	16/17	5.128+00 - 5.12E+00	2.70E+00 - 5.85E+02	2.39E+01	•		pCi/L	Yes/Qual
Technetium-99	8/17	-5.00E-01 - 1.20E+01	1.45E+01 - 6.10E+01	1.89E+01		2.8E+01	pCi/L	Yes/P
		LOCATION-SWM	U 193B MEDIA-Subsurface	Soil			•••	
•	Frequency							
	of	Nondetected	Detected	Arithmetic				COPC/
Analyte	Detection	Range	Range	Mean	HI	ELCR	Units	Basis*
Aluminum	4/4		7.43E+03 - 1.12E+04	9.98E+03	7.3B+02	•	mg/kg	No
Barium	4/4		3.80E+01 - 8.42E+01	5.36B+01	3.78+01		mg/kg	No
Beryllium	2/4	5.00B-01 - 5.00B-01	5.90E-01 - 1.57E+00	6.65B-01	1.6B-01	1.0E-04	mg/kg	Yes/PB
Chromium	4/4		1.04E+01 - 8.87E+01	3.24E+01	4.85-01	4.95+02	mg/kg	Yes/P
Cobalt	4/4		3.18B+00 - 7.76B+00	4.99E+00	2.1E+02		mg/kg	No
Copper	4/4		4.18E+00 - 7.43E+00	6.21B+00	7.4B+01		mg/kg	No
Iron	4/4		9.73E+03 - 2.43E+04	1.458+04	3.1B+02		mg/kg	No
Lithium	4/4		3.44E+00 - 7.72E+00	5.82B+00	7.0E+01		mg/kg	No
Magnesiun	4/4		7.74E+02 - 4.31E+03	1.84E+03			mg/kg	No
Manganese	4/4		1.05E+02 - 2.22E+02	1.54B+02	1.4B+01		mg/kg	No
Nickel	2/4	5.00E+00 - 5.00E+00	7.828+00 - 2.068+01	8.36E+00	3.4E+01		mg/kg	No
Potassium	4/4	5.002,00 5.002,00	2.378+02 - 6.868+02	3.91B+02	3.123702		mg/kg	No
Sodium	4/4		2.44B+02 - 4.48E+02	3.12B+02			mg/kg	Yes/B
Strontium	4/4		8.11E+00 - 9.39E+01	3.13B+01	8.0E+02		mg/kg	No
	4/4		1.75E+01 - 6.50E+01	3.10B+01	5.6B-01			-
Vanadium			1.75E+01 - 5.57E+01	3.09E+01	4.0E+02		mg/kg	Yes/PB
Zinc	4/4						mg/kg	No
Acetone	1/1		6.00B-02 - 8.00E-02	8.00B-02	9.2B+01		mg/kg	No
Toluene	1/3	1.00E-02 - 1.00E-02	1.00E-02 - 1.00E-02	6.67B-03	9.6E+01		mg/kg	No
Alpha activity	4/4		2.14E+00 - 1.86E+01	1.19E+01			pCi/g	Yes/Qual
Beta activity	4/4		9.10E+00 - 2.29E+01	1.528+01			pCi/g	Yes/Qual
		LOCATION=SW	MU 193B MEDIA-Surface So	oil				
	Frequency							
	of	Nondetected	Detected	Arithmetic				COPC/
Analyte	Detection	Range	Range	Mean	HI	ELCR	Units	Basis*
Aluminum	2/2		7.43E+03 - 1.08E+04	9.12E+03	7.3E+02		mg/kg	No
Barium	2/2		3.80E+01 - 8.42E+01	6.11E+01	3.7E+01		mg/kg	No

*P= > FRG, B= > Background, E= > Essential Nutrient, Bio=Bioaccumulates, Qual=Qualitative analyte

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----- LOCATION-SWMU 1938 MEDIA-Surface Soil -----

· .		· · · ·	(continued)				-	
Analyte	Frequency of Detection	Nondetected Range	Detected Range	Arithmetic Mean	ĦI	ELCR	Units	COPC/ Basis=
Beryllium	1/2	5.008-01 - 5.008-01	1.57E+00 - 1.57E+00	9.10E-01	1.6B-01	1.0E-04	mg/kg	Yes/PB
Chromium	2/2		1.04E+01 - 8.87E+01	4.96E+01	4.8E-01	4.9E+02	ing/kg	Yes/P
Cobalt	2/2		3.82E+00 - 7.76E+00	5.79E+00	2.1B+02	· -	mg/kg	No
Copper	2/2		7.078+00 - 7.438+00	7.25E+00	7.4E+01		mg/kg	No
Iron	2/2		1.15E+04 - 2.43E+04	1.80E+04	3.18+02		mg/kg	No
Lithium	2/2		3.44E+00 - 7.72E+00	5.58E+00	7.0E+01		mg/kg	No
Magnesium	2/2		7.74E+02 - 4.31E+03	2.54B+03			mg/kg	No
Manganese	2/2		1.13E+02 - 2.22E+02	1.685+02	1.4E+01		ng/kg	No
Nickel	1/2	5.00E+00 - 5.00E+00	2.06E+01 - 2.06E+01	1.16E+01	3.4E+01		mg/kg	No
Potassium	2/2		2.37E+02 - 6.86E+02	4.628+02			ng/kg	No
Sodium	2/2		2.44E+02 - 2.49E+02	2.47E+02			mg/kg	No
Strontium	2/2		1.42E+01 - 9.39E+01	5.41E+01	8.0E+02		ng/kg	No
Vanadium	2/2		1.75E+01 - 6.50E+01	4.13B+01	5.6B-01		. mg/kg	Yes/PB
Zinc	2/2	· · · · · · · · · · ·	3.21E+01 - 5:57E+01	4.39E+01	4.0E+02		mg/kg	No
Toluene	1/2	1.00E-02 - 1.00E-02	1.00E-02 - 1.00E-02	7.508-03	9.8B+01		mg/kg	No
Alpha activity	2/2	·	1.63B+01 - 1.86E+01	1.758+01			pCi/g	Yes/Qua
Beta activity	2/2		1.66E+01 - 2.29E+01	1.98E+01			pCi/g	Yes/Qua

LOCATION-SWMU 193C MEDIA-MCNairy Group	dwater	
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Analyte	Frequency of Detection	Nondetected Range	Detected Range	Arithmetic Mean	HI	ELCR	Units	COPC/ Basis*
Aluminum	4/4		7.50B-01 - 9.04E+01	2.52E+01	1.5E+00		mg/L	Yes/P
Antimony	5/5		6.00E-02 - 2.50E-01	1.48E-01	5.6B-04		mg/L	Yes/P
Arsenic	5/5		5.00E-03 - 3.60E-02	1.12E-02	4.5E-04	3.5E-06	mg/L	Yes/P
Barium	5/5		9.20E-02 - 6.70E-01	2.65B-01	1.0E-01	•	mg/L	Yes/P
Beryllium	5/5		5.00E-03 - 2.50E-02	1.54E-02	2.6E-03	1.0E-06	mg/L	Yes/P
Cadmium	5/5	•	1.00E-02 - 1.00E-01	3.62B-02	6.6E-04		mg/L	Yes/P
Calcium	5/5		6.48E+00 - 4.10E+01	2.19E+01			mg/L	No
Chloride	5/5		1.45E+01 - 1.68E+01	1.56E+01		•	mg/L	No
Chromium	3/3		5.00E-02 - 2.32E-01	1.14E-01	4.2B-03		mg/L	Yes/P
Cobalt	5/5		4.508-02 - 1.21E-01	7.22E-02	9.1E-02		mg/L	Yes/P
Copper	5/5		1.308-02 - 1.63E-01	6.52E-02	6.0E-02		mg/L	No
Fluoride	4/4		2.00B-01 - 2.80B-01	2.38E-01	9.1E-02		mg/L	No
Iron	5/5		5.04E+00 - 1.79E+02	4.75E+01	4.5E-01		ng/L	Yes/PE
Lead	1/1		2.508-01 - 2.50E-01	2.50B-01	1.5B-07		mg/L	Yes/P
Magnesium	5/5		2.14B+00 - 2.16E+01	7.472+00			mg/L	No

*Pa > PRG, Ba > Background, Ba > Essential Nutrient, Bio-Bioaccumulates, Qual-Qualitative analyte

Table 1.	.10. Summar	y of dat	a evaluat:	ion
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 LOCATION-SWMU	193C MEDIA-McNairy	y Groundwater	
	(continued)		

	Frequency								
	of	Nondetected	Detected	Arithmetic				COPC/	
Analyte	Detection	Range	Range	Mean	HI	ELCR	Units	Basis*	
Manganese	5/5		3.57E-01 - 3.91E+00	1.34E+00	5.7E-02		mg/L	Yes/P	
Mercury	1/1		2.00E-04 - 2.00E-04	2.00E-04	4.4E-04		mg/L	Yes/Bio	
Molybdenum	4/4		5.00E-02 - 1.00E-01	6.38E-02	7.5E-03		mg/L	Yes/PE	
Nickel	5/5		1.00E-01 - 1.09E-01	1.03E-01	3.0E-02		mg/L	Yes/P	
Nitrate as Nitrogen	5/5		1.00E+00 - 1.00E+00	1.00E+00	2.4E+00		mg/L	No	
Potassium	5/5		3.73E+01 - 1.01E+02	6.92E+01			mg/L	No	
Selenium	3/3		5.00E-03 - 5.00E-03	5.00E-03	7.5E-03		mg/L	No	
Silica	5/5		1.10E+01 - 1.80E+01	1.32E+01			mg/L	Yes/Qual	
Silver	3/3		5.00B-02 - 6.00B-02	5.67E-02	7.5E-03		trg/L	Yes/P	
Sodium	5/5		1.90E+01 - 2.63E+01	2.23E+01			mg/L	Yes/Qual	
Tetraoxo-sulfate(1-)	5/5		9.60E+00 - 1.30E+01	1.19E+01			mg/L	Yes/Qual	
Thallium	2/2		6.00B-02 - 1.23E-01	9.15E-02			mg/L	Yes/Qual	
Uranium	9/9		1.00E-03 - 1.80E-02	2.898-03	4.58-03		mg/L	Yes/P	
Vanadium	2/2		5.708-02 - 8.368-01	4.47E-01	9.3E-03		mg/L	Yes/P	
Zinc	5/5		2.60E-02 - 5.64E-01	1.89E-01	4.5E-01		mg/L	Yes/P	
1,1,1-Trichloroethane	4/4		5.00E-03 - 5.0CE-03	5.00E-03	5.4E-02		mg/L	NO	
1,1,2-Trichloroethane	. 4/4		5.00E-03 - 5.00E-03	5.00E-03	8.18-04	1.8B-05	mg/L	Yes/P	
1,1-Dichloroethane	4/4		5.00B-03 - 5.00B-03	5.00E-03	2.7B-02		mg/L	No	
1,1-Dichloroethene	4/4		5.00E-03 - 5.00E-03	5.00E-03	1.8E-03	9.3E-07	mg/L	Yes/P	
1,2-Dichloroethane	4/4		5.00E-03 - 5.00E-03	5.00B-03	6.7E-04	1.1E-05	mg/L	Yes/P	
Benzene	4/4	•	5.00E-03 - 5.00E-03	5.00E-03	4.0E-04	3.5E-05	mg/L	Yes/P	
Bromodichloromethane	4/4		5.00E-03 - 5.00E-03	5.00B-03	4.0E-03	8.4E-05	mg/L	Yes/P	
Carbon Tetrachloride	4/4		5.00E-03 - 5.00E-03	5.00E-03	1.2E-04	1.5E-05	mg/L	Yes/P	
Chloroform	4/4		5.00E-03 - 5.00E-03	5.00B-03	2.0E-03	1.5E-05	mg/L	Yes/P	
Ethylbenzene	4/4		5.00E-03 - 5.00E-03	5.00E-03	4.5E-02		mg/L	Yes/Bio	
Polychlorinated biphenyl	1/1		1.00E-04 - 1.00E-04	1.00E-04		8.0E-06	mg/L	Yes/P	
Tetrachloroethene	4/4		5.00E-03 - 5.00E-03	5.00E-03	7.9E-03	5.78-05	mg/L	Yes/P	
Toluene	4/4		5.00E-03 - 5.00E-03	5.00E-03	2.4E-02		ng/L	No	
Trichloroethene	12/12		1.00E-03 - 2.00E-03	1.082-03	1.2E-03	1.4E-04	mg/L	Yes/P	
Vinyl Chloride	4/4		5.00E-03 - 1.00E-02	6.25E-03		1.7E-06	mg/L	Yes/P	
Xylene	4/4		5.00E-03 - 1.00E-02	7.50B-03	4.0B-01		mg/L	Yes/Bio	
cis-1,2-Dichloroethene	4/4		5.00E-03 - 5.00E-03	5.00E-03	2.0E-03		mg/L	Yes/P	
trans-1,2-Dichloroethene	4/4		5.00B-03 - 5.00B-03	5.00E-03	4.0E-03		mg/L	Yes/P	
Alpha activity	12/12		-1.80E+01 ~ 1.07E+02	7.47E+00			pCi/L	Yes/Qual	
Beta activity	12/12		5,50E+01 - 2.36E+02	1.29E+02			pCi/L	Yes/Qual	
Radon-222	2/2		1.43E+02 - 1.57E+02	1.50E+02		1.4E+00	pCi/L	Yes/P	
Technetium-99	13/13		-7.00E+00 - 2.70E+01	6.18E+00 :		2.8E+01	pCi/L	No	

*P= > PRG, B= > Background, E= > Essential Nutrient, Bio=Bioaccumulates, Qual=Qualitative analyte

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· · · · ·		Table 1.10.	summary of data evaluat	101			2	
		LOCATION-SWM	J 193C MEDIA-RGA Groundw	ater	• • • • • • • • • • • • • • • • • • •			
Analyté	Prequency of Detection	Nondetected Range	Detected Range	Arithmetic Mean	ĦI	ELCR	Units	COPC/ Basis*
, 2-Dichloroethene richloroethene	1/2 1/2	5.008-03 ÷ 5.008-03 1.008-03 ÷ 1.008-03	5.62B-01 - 5.62B-01 1.62B-01 - 1.62B-01	2.828-01 8.158-02	1.8E-03 1.2E-03	1.4E-04	mg/L mg/L	Yes/P Yes/P
		LOCATION-SWM	U 193C MEDIA=Subsurface	Soil				
	Frequency	1 . <u>.</u>	• 			s =		
Analyte	of Detection	Nondetected Range	Detected Range	Arithmetic Mean	ĦI	ELCR	Units	COPC/ Basis
Aluminum	20/20		3.148+02 = 1.378+04	8.52E+03	7.38+02		mg/kg	Yes/P
rsenic	5/20	5.00E+00 - 5.00E+00	5.018+00 - 6.578+00	3.33E+00	6.98-01	9.2E-03	mg∕kg	No
Jarium	1/1		1.42E+02 - 1.42E+02	1.428+02	3.7E+01		ng/kg	No
Beryllium	10/20	5.00B-01 - 5.00B-01	5.00B-01 - 9.80B-01	4.61E-01	1.6E-01	1.05-04	mg/kg	Yes/P
Boron	1/20	1.00E+02 - 1.00E+02	1.00B+02 - 1.00E+02	5.25B+01	3.3E+02		mg/kg	No
Cadmium	3/59	2.10B-01 - 5.00B+00	2.41B-01 - 5.00B+00	1.92E+00	3.8R-01	3.3E+03	mg/kg	Yes/P
Calcium	18/20	5.008+00 - 5.008+00	2.23E+02 - 4.00E+05	2.60E+04			mg/kg	No
Chromium	59/61	2.008+00 - 2.008+00	3.76B+00 - 8.30B+01	1.898+01	4.8B-01	4.9E+02	mg/kg	Yes/P
Cobalt	17/20	1.00E+00 - 1.00E+00	1.22E+00 - 8.61E+01	5.46E+00	2.1E+02		mg/kg	Yes/B
Copper	16/20	2.00B+00 - 2.00B+00	2.15E+00 - 2.82E+01	7.56E+00	7.4E+01		mg/kg	No
Iron	6/6	• ••	1.20E+04 - 3.00E+04	2.22E+04	3.1E+02		mg/kg	Yes/P
Lead	42/61	5.00E+00 - 2.00E+01	5.10E+00 - 6.77E+01	1.202+01	1.0E-04		mg/kg	Yes/P
Lithium	17/20	2.00E+00 - 2.00E+00	2.50B+00 = 1.25B+01	7.17E+00	7.0E+01		mg/kg	No
Magnesium	20/20	•	8.52B+02 - 1.45B+04	3.41E+03		·. •	mg/kg	No
Manganese	20/20	· ·	1.63E+01 - 2.27E+03	3.99E+02	1.4E+01		mg/kg	Yes/P
Nickel	13/20	5.00B+00 - 5.00B+00	5.998+00 - 2.158+01	9.76E+00	3:4E+01		mg/kg	No
Potassium	20/20	•	1.43E+02 - 1.57E+03	5.338+02			mg/kg	No
Sodium	15/20	2.00B+02 - 2.00E+02	2.02B+02 - 4.44B+02	2.62E+02			mg/kg	Yes/B
Strontium	20/20		7.25B+00 - 3.91E+02	1.06E+02	8.0E+02		mg/kg	No
Vanadium	20/20		2.128+00 - 4.428+01	2.05E+01	5.6E-01		mq/kq	Yes/P
Zinc	19/20	1.50E+01 - 1.50E+01	1.63E+01 - 9.25E+01	4.14E+01	4.0E+02		mg/kg	Yes/B
Xvlene	1/20	5.008-03 - 5.008-03	1.00E-02 - 1.00E-02	2.88E-03	1.78+03	•	mg/kg	Yes/B
Alpha activity	53/53		1.508-03 - 4.00E+00	1.54E+00			pCi/g	Yes/Q
Beta activity	53/53	. •	5.00E-03 - 1.00E+01	4.45E+00			pCi/g	Yes/Q

*P= > PRG, B= > Background, E= > Essential Nutrient, Bio-Bioaccumulates, Qual=Qualitative analyte

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	Frequency							
	of	Nondetected	Detected	Arithmetic				COPC/
nalyte	Detection	Range	Range	Mean	HI	BLCR	Units	Basis*
luminum	5/5		3.14E+02 - 3.36E+03	1.90E+03	7.3E+02		mg/kg	No
oron	1/5	1.00E+02 - 1.00E+02	1.00E+02 - 1.00E+02	6.00E+01	3.3E+02		mg/kg	No
alcium	5/5		2.53E+05 - 4.00E+05	3.37E+05			mg/kg	No
hromium	3/5	2.00E+00 - 2.00E+00	4.49E+00 - 1.20E+01	5.90B+00	4.8E-01	4.9E+02	mg/kg	Yes/P
obalt	2/5	1.00E+00 - 1.00E+00	1.22E+00 - 2.14E+00	9.72B-01	2.1B+02		mg/kg	No
opper	2/5	2.00E+00 - 2.00E+00	3.37E+00 - 2.82E+01	6.91E+00	7.4E+01		mg/kg	No
ead	1/5	2.00E+01 - 2.00E+01	6.77E+01 - 6.77E+01	2.15E+01	1.02-04		mg/kg	Yes/PE
ithium	3/5	2.00E+00 - 2.00E+00	5.43E+00 - 1.25E+01	6.03E+00	7.0Ė+01		mg/kg	No
agnesium	5/5		3.19E+03 - 1.45E+04	8.76E+03			mg/kg	No
langanese	5/5		3.55E+01 - 1.98E+02	7.90E+01	1.4B+01		mg/kg	No
ickel	1/5	5.002+00 - 5.002+00	6.43E+00 - 6.43E+00	3.29E+00	3.4E+01		mg/kg	No
otassium	5/5		1.43E+02 - 1.57E+03	7.01E+02			mg/kg	No
odium	4/5	2.00B+02 - 2.00B+02	2.29E+02 - 3.10E+02	2.37E+02			mg/kg	No
trontium	5/5		1.96E+02 - 3.91E+02	2.928+02	8.0E+02		mg/kg	No
anadium	5/5		2.12B+00 - 6.70E+00	4.00E+00	5.6E-01		mg/kg	No
linc	5/5		4.59E+01 - 9.25E+01	6.598+01	4.08+02	,	mg/kg	Yes/B
		Location=Swm	U 194 MEDIA=Subsurface &	8011			•••••	
	Frequency	Nondetected	Detected	Arithmetic				COPC/
nalyte	Detection	Range	Range	Mean	HI	ELCR	Units	Basis
luminum	12/12		5.38E+03 - 1.45E+04	9.47E+03	7.3B+02		nag/kg	Yes/P
rsenic	1/12	5.00E+00 - 5.00E+00	6.73E+00 - 6.73E+00	2.85E+00	6.9E-01	9.2E-03	mg/kg	No
larium	12/12		2.05E+01 - 1.39E+02	7.51E+01	3.7E+01		mg/kg	No
eryllium	6/12	5.00B-01 - 5.00B-01	5.40E-01 - 4.80E+00	7.48E-01	1.6E-01	1.0E-04	mg/kg	Yes/P
admium	1/35	2.002+00 - 5.00E+00	8.55E+00 - 8.55E+00	2.15E+00	3.8E-01	3.3E+03	mg/kg	Yes/P
Calcium	12/12		5.68E+02 - 6.81E+03	1.67E+03			mg/kg	No
	35/35		8.24E+00 - 1.03E+02	1.84E+01	4.8E-01	4.9E+02	mg/kg	Yes/P
Chromium	33/33		0.446TVV - 1.VJ6TV2	T' 04711	18.05-VI	9.35404		IE8/P

35/35 8.24**6**+00 1.036+02 1.84E+01 4.8E-01 4.9E+02 mg/kg Yes/P 12/12 2.528+00 - 9.468+00 2.1E+02 mg/kg 5.05B+00 Cobalt No 12/12 2.41E+00 - 1.67E+01 7.49E+00 7.4E+01 mg/kg No mg/kg mg/kg 12/12 6.41E+03 - 2.00E+04 1.24E+04 3.1E+02 No 20/35 5.00E+00 - 2.00E+01 5.03E+00 - 3.60E+02 1.19E+01 1.0E-04 Yes/PB Lithium 12/12 2.41E+00 - 9.00E+00 6.45E+00 7.0E+01 mg/kg No mg/kg 12/12 4.15E+02 - 2.34E+03 1.328+03 No Magnesium mg/kg mg/kg 12/12 3.49E+01 - 4.67E+02 1.602+02 1.4E+01 Manganese No 8/12 5.00E+00 - 5.00E+00 5.74E+00 - .1.37E+01 7.22E+00 3.4E+01 Nickel No Potassium 12/12 1.53E+02 - 6.32E+023.80E+02 mg/kg No Sodium 8/12 2.00E+02 - 2.00E+022.10E+02 - 3.69E+02 2.37E+02 Yes/B mg/kg

*P= > PRG, B= > Background, E= > Essential Nutrient, Bio=Bioaccumulates, Qual=Qualitative analyte .

Copper

Iron

Lead

		LOCATION-SWM	U 194 MEDIA-Subsurface S	Boil				
			(continued)					
	Frequency	· ·						
	of	Nondetected	Detected	Arithmetic				COPC/
Analyte	Detection	Range	Range	Mean	HI	ELCR	Units	Basis*
Strontium	12/12	•	3.92E+00 - 2.60E+01	1.29E+01	8.0E+02	·	mg/kg	No
Vanadium	12/12		1.50E+01 - 2.58E+01	1.99E+01	5.6E-01		mg/kg	No
Zinc	11/12	1.50E+01 - 1.50E+01	1.57E+01 - 6.76E+01	3.82E+01	4.0E+02		mg/kg	Yes/B
Ethylbenzene	1/19	5.00E-03 - 5.00E-03	1.50E-02 - 1.50E-02	3.16E-03	1.1E+02		mg/kg	Yes/Bio
Alpha activity	23/23		1,20E+00 - 2.50E+00	1.85E+00			pCi/g	Yes/Qual
Beta activity	23/23		3.00E+00 - 7.00E+00	4.832+00	·		pCi/g	Yes/Qual
		LOCATION-SWMU	99A MEDIA-MCNairy Ground	iwater				
	Frequency							
	of	Nondetected	Detected	Arithmetic				COPC/
Analyte	Detection	Range	Range	Mean	HI	ELCR	Units	Basis*
		.2	-					
1,1,1-Trichloroethane	1/4	5.00E-03 - 5.00E-03	1.208-03 - 1.208-03	2.188-03	5.4E-02		mg/L	No
1,1-Dichloroethene	1/4	1.00E-02 - 1.00E-02	2.29E-02 - 2.29E-02	9.48E-03	1.8E-03	9.3E-07	mq/L	Yes/P
Carbon Tetrachloride	1/4	5.008-03 - 5.008-03	2.80E-03 - 2.80E-03	2.58B-03	1.2E-04	1.5E-05	mg/L	Yes/P
Trichloroethene	3/4	5.008-03 - 5.008-03	3.00B-04 - 5.19B-01	1.31E-01	1.2E-03	1.4E-04	mg/L	Yes/P
cis-1,2-Dichloroethene	2/4	2.00E+00 - 2.00E+00	2.80B-02 - 1.15E-01	1.04E+00	2.0E-03		mg/L	Yes/P
Alpha activity	2/2		2.60E+00 - 2.90E+00	2.75E+00			pCi/L	Yes/Qual
Beta activity	2/2		2.30E+01 - 3.50E+01	2.90E+01			pCi/L	Yes/Qual
Technetium-99	2/2		1.00E+01 - 1.90E+01	1.458+01		2.85+01	pCi/L	No
		LOCATION-SWM	U 99A MEDIA-RGA Groundwa	ater				
	Frequency		*		•			
	of	Nondetected	Detected	Arithmetic				COPC/
Analyte	Detection	Range	Range	Mean	HI	ELCR	Unite	Basis*
Aluminum	16/35	2.00E-01 - 1.00E+00	2.00E-01 - 6.59E+02	2.17E+00	1.5E+00		mg/L	Yes/P
Arsenic	4/27	5.008-03 - 5.008-03	5.00E-03 - 1.00E-02	3.56E-03	4.5E-04	3.5E-06	mg/L	Yes/P
Barium	39/39		1.30E-01 - 3.30E+00	5.418-01	1.0E-01		mg/L	Yes/P
Beryllium	8/35	5.00E-03 - 2.50E-02	8.00E-03 - 1.00E-01	3.87E-03	2.6B-03	1.0E-06	mg/L	Yes/P
Calcium	39/39	·- •	2.10E+01 - 1.20E+02	4.14E+01			mg/L	No
Chloride	9/9		5.68E+01 - 1.20E+02	6.83E+01			mg/L	Yes/E
Chromium	11/39	5.00E-02 - 6.00E-02	6.00E-02 - 1.78E+00	4.39E-02	4.2E-03		mg/L	Yes/P
Cobalt	20/37	1.00E-02 - 1.00E-01	1.00E-02 - 5.70E-01	5.64E-02	9.1E-02		mg/L	Yes/P
Copper	9/35	1.00E-02 - 1.00E-01	7.00E-02 - 6.40E-01	3.18E-02	6.0E-02		mg/L	Yes/PE
Fluoride	8/8		1.70E-01 - 2.00E-01	1.84E-01	9.1E-02		mg/L	No
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*P= > FRG, B= > Background, E= > Essential Nutrient, Bio=Bioaccumulates, Qual=Qualitative analyte

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Table	1.10.	Summary	of	data	evaluation

------ LOCATION-SHMU 99A MEDIA-RGA Groundwater

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Analyte	Frequency of Detection	Nondetected Range	Detected Range	Arithmetic Mean	ні	ELCR	Units	COPC/ Basis*
Iron	31/39	2.00E-01 - 3.55E-01	2.10E-01 - 1.20E+03	9.19E+00	4.5E-01		mg/L	Yes/PE
lead	6/29	5.00B-02 - 2.50B-01	5.00E-02 - 4.10E-01	4.03E-02	1.58-07		mg/L	Yes/P
Lithium	6/23	5.00B-02 - 5.00B-02	5.00B-02 - 1.70B-01	4.45E-02	3.0E-02		mg/L	Yes/P
lagnesium	39/39		8.38E+00 - 4.97E+01	1.64E+01			mg/L	Yes/E
langanese	37/39	1.00E-01 - 1.00E-01	4.30E-02 - 4.60E+00	1.13E+00	6.7E-02		mg/L	Yes/P
lercury	5/25	2.00B-04 - 2.00E-04	2.00E-04 - 2.00E-02	6.33B-05	4.4B-04		mq/L	Yes/P
lickel	16/39	5.00E-02 - 1.00E-01	5.00B-02 - 9.10B-01	9.25E-02	3.0E-02		mg/L	Yes/P
litrate as Nitrogen	7/9	1.00E+00 - 1.00E+00	1.00E+00 - 2.10E+00	1.12E+00	2.4E+00		mg/L	No
otassium	24/39	2.00E+00 - 1.05E+01	2.08E+00 - 2.17E+01	4.17E+00			mg/L	No
ilica	9/9		1.50B+01 - 2.50E+01	1.87E+01			mg/L	Yes/Qua
lodium	39/39		1.50E+01 - 7.24E+01	5.34E+01			mg/L	Yes/Qua
Strontium	30/30		1.20E-01 - 4.70E-01	2.49E-01	9.0E-01		mg/L	No No
Bulfate	2/2		1.75B+01 - 1.92B+01	1.84E+01			mg/L	Yes/Qua
Tetraoxo-sulfate(1-)	7/7		1.10E+01 ~ 2.20E+01	1.67E+01			mg/L	Yes/Qua
Vanadium	10/28	1.00E-01 - 1.00E-01	8.50E-02 - 2.15E+00	1.498-01	9.3E-03		ng/L	Yes/P
linc	10/35	3.008-02 - 2.50E-01	1.10B-02 - 2.55E+00	8.96E-02	4.5E-01		mg/L	Yes/P
,1-Dichloroethene	7/33	1.00E-03 - 5.00E-02	8.00E-03 - 6.50E-02	1.70E-02 [,]	1.8E-03	9.3E-07	mg/L	Yes/P
richloroethene	41/43	1.00E-03 - 5.00E-03	2.00E-04 - 2.37E+00	3.47E-01	1.2E-03	1.4E-04	mg/L	Yes/P
is (2-Ethylhexyl) phthalate	5/10	2.00E-02 - 2.00E-02	6.00E-03 - 1.60E-02	1.31E-02	2.6B-02	3.1E-04	mg/L	Yes/P
is-1,2-Dichloroethene	10/33	1.00E-03 - 2.00E+00	3.00B-04 - 3.48E-02	3.70E-03	2.0E-03		mg/L	Yes/P
rans-1,2-Dichloroethene	3/33	1.00B-03 - 2.00E+00	3.00E-04 - 6.00E-04	5.49E-04	4.0E-03		mq/L	No
lpha activity	33/39	-2.20E+00 - 2.60E+00	-2.50B+00 - 5.38B+01	4.56E+00			pC1/L	Yes/Qua
Beta activity	39/39		3.00E+00 - 1.37E+02	3.16E+01			pCi/L	Yes/Qua
ladon-222	4/4		2.86E+02 - 6.75E+02	4.75E+02		1.4E+00	pCi/L	Yes/P
Technetium-99	34/40	4.10E+00 - 1.70E+01	3.00E+00 - 1.39E+02	3.48E+01		2.8E+01	pCi/L	Yes/P

Analyte	Frequency of Detection	Nondetected Range	Detected Range	Arithmetic Mean	HI	ELCR	Units	COPC/ Basis*
Aluminum	22/22		1.80E+03 - 1.41E+04	7.55E+03	7.3E+02		mg/kg	Yes/PB
Antimony	5/22	2.00E+01 - 2.00E+01	1.70E+00 - 2.90E+00	2.16E+00	6.4B-02		mg/kg	Yes/PB
Arsenic	11/22	5.00E+00 - 5.00E+00	2.40E+00 - 8.55E+00	4.22E+00	6.9E-01	9.2E-03	mg/kg	Yes/PB
Barium	22/22		2.03E+01 - 2.47E+03	1.53E+02	3.7E+01		mg/kg	Yes/PB
Beryllium	11/22	5.00E-01 - 5.00E-01	2.20E-01 - 8.90E-01	4.32E-01	1.6E-01	1.0E-04	mg/kg	Yes/PB
Cadmium	5/22	2.00E+00 - 2.00E+00	7.508-01 - 8.30E-01	8.19E-01	3.8E-01	3.3E+03	mg/kg	Yes/PB
Calcium	20/20		1.14E+03 - 2.87E+05	1.66E+05			mg/kg	No

*P= > PRG, B= > Background, E= > Essential Nutrient, Bio=Bioaccumulates, Qual=Qualitative analyte

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		LOCATION-SWM	J 99A MEDIA-Subsurface S	oil			<i></i>	
			(continued)					
	Frequency				•			
	of	Nondetected	Detected	Arithmetic				COPC/
Analyte	Detection	Range	Range	Mean	HI	ELCR	Units	Basis*
Chromium	22/22		7.00E+00 - 4,57E+01	1.44E+01	4.8E-01	4.9E+02	mg/kg	Yes/P
Cobalt	20/22	1.00E+00 - 1.00E+00	1.68E+00 - 1.19E+01	4.97E+00	2.1E+02		mg/kg	No
Copper	21/22	2.00B+00 - 2.00B+00	3.80B+00 - 1.64E+01	7.318+00	7.4E+01		mg/kg	No
Cyanide	2/16	1.00B+00 - 1.00B+00	4.40E-01 - 5.40E-01	4.99E-01	2.3E+01		ng/kg	No
Iron	22/22		1.45E+03 - 2.33E+04	1.18E+04	3.1E+02		mg/kg	No
Lead	6/22	2.00B+01 - 2.00B+01	7.008+00 - 4.73E+01	1.36E+01	1.0E-04		mg/kg	Yes/PB
Lithium	17/17		2.82B+00 - 1.29E+01	7.38E+00	7.0E+01		mg/kg	No
Magnesium	22/22		3.97E+02 - 2.73E+04	6.49E+03			mg/kg	No
Manganese	22/22		3.93E+01 - 1.46E+03	3.02E+02	1.4E+01		mg/kg	Yes/PB
Mercury	5/22	2.00B-01 - 2.00E-01	8.00E-02 - 1.20E-01	9.95B-02	1.4E-01		mg/kg	No
Nickel	17/22	5.00E+00 ~ 5.00E+00	2.50E+00 - 2.58E+01	9.72E+00	3.4B+01		wg/kg	No
Potassium	22/22		2.28E+02 - 1.12B+03	5.27E+02			mg/kg	No
Selenium	5/20	1.005+00 - 1.002+00	2.90E-01 - 3.20E-01	3.11E-01	1.22+01		wg/kg	No
Silver	5/22	4.00E+00 - 4.00E+00	6.40B-01 ~ 7.10B-01	6.99B-01	6.1E+00		ng/kg	No
Sodium	14/22	2.00E+02 - 2.51E+02	6.63E+01 - 3.93E+02	2.14E+02			mg/kg	Yes/B
Strontium	17/17	• • • • • •	8.882+00 - 5.142+02	2.25E+02	8.0E+02		ng/kg	No
Thallium	5/22	1.50E+01 - 1.50E+01	5.30E-01 - 5.90E-01	5.75E-01			mg/kg	Yes/B
Vanadium	22/22		4.48E+00 - 3.55E+01	1.79E+01	5.6E-01		mg/kg	No
Zinc	21/22	4.76E+01 - 4.76E+01	1.16E+01 - 1.63E+02	6.96E+01	4.0E+02		mg/kg	Yes/B
1,1,1-Trichloroethane	5/8	1.00E-02 - 1.00E-02	6.00E-03 - 6.00E-03	5.63E-03	1.2E+02		mg/kg	No
1,1,2,2-Tetrachloroethane	5/8	1.00E-02 - 1.00E-02	6.00 <u>8-03</u> - 6.00E-03	5.63B-03		1.8E-02	mg/kg	No
1,1,2-Trichloroethane	5/8	1.00E-02 - 1.00E-02	6.00E-03 - 6.00E-03	5.63B-03	3.1B+00	4.6E-02	ng/kg	No
1,1-Dichloroethane	5/8	1.00E-02 - 1.00E-02	6.00E-03 - 6.00E-03	5.63B-03	6.7E+01		ng/kg	No
1,1-Dichloroethene	5/10	1.98E-01 - 5.27E-01	6.00E-03 - 6.00E-03	8.91E-02	3.5E+00	3.9E-03	mg/kg	Yes/P
1,2,4-Trichlorobenzene	5/22	5.00E-01 - 5.00E-01	3.60E-01 - 4.10E-01	2.83E-01	2.5E+01		mg/kg	Yes/Bio
1,2-Dichlorobenzene	5/22	5.008-01 - 5.008-01	3.60E-01 - 4.10E-01	2.83E-01	7.6E+01		mg/kg	Yes/Bio
1,2-Dichloroethane	5/8	1.00E-02 - 1.00E-02	6.00E-03 - 6.00E-03	5.63B-03	4.3E+00	2.28-02	mg∕kġ	No
1,2-Dichloroethene	5/5		6.00B-03 - 6.00B-03	6.00B-03	1.0E+01		mg/kg	No
1,2-Dichloropropane	5/8	1.00E-02 - 1.00E-02	6.00B-03 - 6.00E-03	5.63E-03	1.6E+00	8.7 <u>8</u> -02	mg/kg	No
1,3-Dichlorobenzene	5/22	5.00B-01 - 5.00B-01	3.60E-01 - 4.10E-01	2.83E-01	3.3E+01		mg/kg	Yes/Bio
1,4-Dichlorobenzene	5/22	5.00Ê-01 - 5.00B-01	3.60E-01 - 4.10E-01	2.83E-01	1.1E+03	2.9E-01	mg/kg	Yes/P
2,4,5-Trichlorophenol	5/22	5.00E-01 - 5.00E-01	1.80E+00 - 2.10E+00	6.43E-01	1.6E+02		mg/kg	Yes/Bio
2,4,6-Trichlorophenol	5/22	5.00E-01 - 5.00E-01	3.60E-01 - 4.10E-01	2.83E-01		8.1E-01	mg/kg	Yes/Bio
2,4-Dichlorophenol	5/22	5.00B-01 - 5.00B-01	3.60E-01 - 4.10E-01	2.83E-01	6.8E+00		mg/kg	No
2,4-Dimethylphenol	5/22	5.00B-01 - 5.00B-01	3.60B-01 - 4.10E-01	2.83E-01	3.1E+01		ug/kg	No
2,4-Dinitrophenol	5/6	4.80B-01 - 4.80E-01	1.80E+00 - 2.10E+00	1.69E+00	5.0B+00		mg/kg	No
2,4-Dinitrotoluene	5/22	5.00E-01 - 5.00E-01	3.60E-01 - 4.10E-01	2.83B-01	4.7E+00	2.1E-02	mg/kg	Yes/P
2,6-Dinitrotoluene	5/22	5.00B-01 - 5.00B-01	3.60E-01 - 4.10E-01	2.83B-01	2.3E+00	2.1B-02	mg/kg	Yes/P
2-Butanone	5/8	1.00E-02 - 2.50E-01	6.00E-03 - 1.20E-02	1.158-02	3.9B+02		mg/kg	No
2-Chloronaphthalene	5/22	5.00E-01 - 5.00E-01	3.60E-01 - 4.10E-01	2.838-01	1.1E+02		mg/kg	Yes/Bio

*P= > PRG, B= > Background, E= > Essential Mutrient, Bio=Bioaccumulates, Qual=Qualitative analyte

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(continued) Frequency of Nondetected Detected Arithmetic COPC/ Detection Mean Analyte Range Range HI ELCR Units Basis* 5/22 2-Chlorophenol 5.00E-01 - 5.00E-01 3.60E-01 - 4.10E-01 2.83E-01 7.0E+00 mg/kg No 2-Hexanone 5/8 1.00E-02 - 1.00E-02 1.10E-02 - 1.20E-02 9.258-03 Yes/Qual mg/kg 2-Methyl-4,6-dinitrophenol 5/22 5.00E-01 - 5.00E-01 1.80E+00 - 2.10E+006.43B-01 mg/kg Yes/Qual 2-Methylnaphthalene 5/22 5.00E-01 - 5.00E-01 3.60E-01 - 4.10E-01 2.83E-01 Yes/Qual mg/kg 2-Methylphenol 5/22 5.00E-01 - 5.00E-01 3.60E-01 - 4.10E-01 2.83B-01 7.88+01 mg/kg No 2-Nitroaniline 5/22 5.00E-01 - 5.00E-01 1.80E+00 - 2.10E+00 6.43E-01 7.0E-02 mg/kg Yes/P 2-Nitrophenol 5/22 5.00E-01 - 5.00E-01 3.60E-01 - 4.10E-01 2.83E-01 Yes/Qual mg/kg 3.3'-Dichlorobenzidine 5/22 5.00E-01 - 5.00E-01 7.20E-01 - 8.20E-01 3.738-01 2.18-02 Yes/P mg/kg 1.80E+00 - 2.10E+00 5/22 5.00E-01 - 5.00E-01 3-Nitroaniline 6.43E-01 mg/kg Yes/Qual 2/2 2.00E-02 - 3.50E-02 4,4'-DDD 2.75E-02 5.18-02 mg/kg Yes/Bio 4,4'-DDE 2/2 2.00E-02 - 3.50E-02 2.75E-02 3.6E-02 Yes/Bio mg/kg 2/2 2.00E-02 - 3.50E-02 2.758-02 1.0E+00 3.6E-02 4.4'-DDT mg/kg Yes/Bio 4-Bromophenyl phenyl ether 5/22 5.00E-01 - 5.00E-01 3.60E-01 - 4.10E-01 2.83B-01 Yes/Oual mg/kg 5.00E-01 - 5.00E-01 4-Chloro-3-methylphenol 5/22 3.60E-01 - 4.10E-01 2.83E-01 mg/kg Yes/Qual 5/22 5.00B-01 - 5.00E-01 3.60E-01 - 4.10E-01 2.83E-01 6.3E+00 4-Chloroaniline mg/kg No 3.60B-01 - 4.10E-01 4-Chlorophenyl phenyl ether 5/22 5.00E-01 - 5.00E-01 2.83E-01 Yes/Qual mg/kg 1.00E-02 - 2.50E-01 1.10E-02 - 1.20E-02 1.198-02 4-Methyl-2-pentanone 5/8 3.3E+01 mq/kq No 5/22 5.00B-01 - 5.00B-01 3.60E-01 - 4.10E-01 2.83B-01 4-Methylphenol 9.6E+00 mg/kg No 6.43E-01 4-Nitroaniline 5/22 5.00B-01 - 5.00B-01 1.80E+00 - 2.10E+00 mg/kg Yes/Qual 4-Nitrophenol 5/22 5.00B-01 - 5.00B-01 1.80E+00 - 2.10E+00 6.43E-01 1.6E+02 mg/kg No Acenaphthene 7/22 5.00E-01 - 5.00E-01 3.00E-01 - 4.10E-01 3.89E-01 6.4B+01 Yes/Bio mg/kg Acenaphthylene 6/22 5.00E-01 - 5.00E-01 3.60E-01 - 6.10E-01 4.32E-01 mg/kg Yes/Qual Acetone 5/8 1.00E-02 - 2.50E-01 1.20E-02 - 5.30E-02 2.47E-02 9.2E+01 mg/kg No Aldrin 2/2 9.80E-03 - 1.70E-02 1.34E-02 4.8E-02 5.5B-04 mg/kg Yes/P Anthracene 7/22 5.00B-01 - 5.00B-01 3.60B-01 - 7.50B-01 4.42E-01 6.5E+02 Yes/Bio mg/kg 8/22 5.00B-01 - 5.00B-01 2.20E-01 - 1.70E+00 4.40E-01 Benz (a) anthracene 8.5E-03 mg/kg Yes/P 1.00B-02 - 1.00B-02 6.00E-03 - 6.00E-03 5/8 5.63B-03 1.8E+00 5.18-02 Benzene wq/kq No 7/22 5.00B-01 - 5.00B-01 3.60B-01 - 2.10E+00 Benzo (a) pyrene 4.38E-01 8.5E-04 Yes/P mg/kg 1.70B-01 - 5.70E+00 11/22 5.00B-01 - 5.00B-01 5.03E-01 Benzo (b) fluoranthene 8.5E-03 mg/kg Yes/P 7/22 5.00B-01 - 5.00B-01 Benzo (ghi) perylene 3.60E-01 - 1.18E+00 4.40E-01 mg/kg Yes/Qual Benzo(k)fluoranthene 8/22 5.00B-01 - 5.00B-01 3.60E-01 - 7.90E-01 4.52E-01 8.5E-02 mg/kg Yes/P

*Pa > PRG, Ba > Background, Ea > Essential Nutrient, Bio-Bioaccumulates, Qual=Qualitative analyte

1.80E+00 - 2.10E+00

3.60B-01 - 4.10B-01

6.00E-03 - 6.00E-03

6.00E-03 - 6.00E-03

1.10E-02 - 1.20E-02

3.60E-01 - 4.10E-01

6.00E-03 - 6.00E-03

6.00B-03 - 6.00E-03

6.00E-03 - 6.00E-03

1.98E+00

3.94E-01

5.63B-03

5.63E-03

1.05B-02

3.68E-01

5.632-03

5.63E-03

5.63E-03

9.8E+03

5.8E+02

1.9E+01

1.6E+01

6.2E-01

3.7E+02

4.6E+01

2.7E-01

5.6E+00

mg/kg

mg/kg

mg/kg

mg/kg

mg/kg

mg/kg

mg/kg

mg/kg

mg/kg

1.2E-01

5.4E-01

1.6E-02

NO

No

No

No

No

No

No

No

Yes/Bio

Benzoic Acid

Bromoform

Bromomethane

Chlorobenzene

Carbon Disulfide

Benzyl Alcohol

Bromodichloromathane

Butyl benzyl phthalate

Carbon Tetrachloride

1.00B-02 - 1.00B-02

1.00E-02 - 1.00E-02

1.00B-02 - 2.00B-02

4.80E-01 - 4.80E-01

1.00E-02 - 1.00E-02

1.00B-02 - 1.00B-02

1.00E-02 - 1.00E-02

5/5

5/5

5/B

5/8

5/8

5/6

5/8

5/8

5/8

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 LOCATION-SWMU 99	A MEDIA-Subsurface	Soil	 	
(C	ontinued)			

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Analyte	Frequency of Detection	Nondetected Range	Detected Range	Arithmetic Mean	HI	ELCR	Units	COPC/ Basis*
Chloroethane	5/8	1.00E-02 - 2.00E-02	1.10E-02 - 1.20E-02	1.058-02	2.8E+02		mg/kg	No
Chloroform	5/8	1.00E-02 - 1.00E-02	6.00B-03 - 6.00B-03	5.63E-03	2.4E+00	2.1B-02	mg/kg	No
Chloromethane	5/8	1.00B-02 - 2.00B-02	1.10E-02 - 1.20E-02	1.058-02		1.38-01	mg/kg	No
Chrysene	7/22	5.008-01 - 5.00E-01	3.60E-01 - 2.10E+00	4.38E-01		8.5B-01	mg/kg	Yes/P
Di-n-butylphthalate	5/22	5.00B-01 - 5.00E-01	3.50E-01 - 4.10E-01	2.838-01	2.6E+02		₩ġ/kg	Yes/Bio
Di-n-octylphthalate	5/22	5.00E-01 - 5.00E-01	3.60E-01 - 4.10E-01	2.83E-01	4.9E+01		mq/kq	Yes/Bio
Dibenz (a, h) anthracene	6/22	5.008-01 - 5.008-01	3.60E-01 - 4.80E-01	2.938-01		8.58-04	mg/kg	Yes/P
Dibenzofuran	6/22	5.00B-01 - 5.00E-01	1.23E-01 - 4.10E-01	2.77E-01	6.3E+00	•	mg/kg	Yes/Bio
Dibromochloromethane	5/8	1.00E-02 - 1.00E-02	6.00E-03 - 6.00E-03	5.63B-03	1.5E+01	5.9E-02	mg/kg	No
Dieldrin	2/2		2.00B-02 - 3.50B-02	2.75E-02	8.0E-02	5.8B-04	ng/kg	Yes/P
Diethylphthalate	5/22	5.00B-01 - 5.00B-01	3.60E-01 - 4.10E-01	2.83E-01	2.0E+03		mg/kg	No
Dimethylphthalate	5/22	5.00E-01 - 5.00E-01	3.60E-01 - 4.10E-01	2.83E-01	2.4E+04		mg/kg	No
Endosulfan I	2/2		9.80E-03 - 1.70E-02	1.34E-02			mg/kg	Yes/Qual
Endosulfan II	2/2		2.00E-02 - 3.50E-02	2.75E-02			ng/kg	Yes/Oual
Endosulfan Sulfate	2/2		2.00E-02 - 3.50E-02	2.75B-02			ng/kg	Yes/Qual
Endrin	2/2		2.00E-02 - 3.50E-02	2.75E-02	2.4E-02		mg/kg	Yes/P
Endrin Ketone	2/2		2.00E-02 - 3.50E-02	2.75B-02			mg/kg	Yes/Qual
Ethylbenzene	5/8	1.00E-02 - 1.00E-02	6.00E-03 - 6.00E-03	5.63E-03	1.1E+02		mg/kg	Yes/Bio
Fluoranthene	9/22	5.00E-01 - 5.00E-01	1.40B-01 - 2.66E+00	4.23B-01	4.3E+01		mg/kg	Yes/Bio
Fluorene	6/22	5.00B-01 - 5.00B-01	2.19E-01 - 4.10E-01	3.80B-01	6.3B+01		mg/kg	Yes/Bio
Heptachlor	2/2		9.80E-03 - 1.70E-02	1.34E-02	1.1B+00	2.8E-03	mg/kg	Yes/P
Heptachlor Epoxide	2/2		9.80B-03 - 1.70E-02	1.34B-02	2.7B-02	1.4E-03	mg/kg	Yes/P
Hexachlorobenzene	5/22	5.00E-01 - 5.00E-01	3.60B-01 - 4.10E-01	2.832-01	1.3E+00	5.4E-03	mg/kg	Yes/P
Hexachlorobutadiene	5/22	5.00B-01 - 5.00B-01	3.60B-01 - 4.10B-01	2.83E-01	3.0E-01	1.0E-01	ng/kg	Yes/P
Hexachlorocyclopentadiene	5/22	5.00B-01 - 5.00B-01	3.60E-01 - 4.10E-01	2.83E-01	1.02+00		mg∕kg	Yes/Bio
Hexachloroethane	5/22	5.00E-01 - 5.00E-01	3.60E-01 - 4.10E-01	2.83E-01	1.5B+00	5.8B-01	tag∕kg	Yes/Bio
Indeno (1,2,3-cd) pyrene	7/22	5.00E-01 - 5.00B-01	3.60B-01 - 1.05E+00	4.468-01		8.58-03	mg/kg	Yes/P
Isophorone	5/22	5.00E-01 - 5,00E-01	3.605-01 - 4.10E-01	2.83E-01	3.0E+02	9.9B+00	mg/kg	No
Methoxychlor	2/2		9.80E-02 - 1.70E-01	1.34E-01	8.0E+00		ng/kg	Yes/Bio
Methylene Chloride	5/8	1.00E-02 - 1.00E-02	2.00B-03 - 8.00B-03	5.20B-03	7.0E+01	5.0E-01	mg/kg	No
N-Nitroso-di-n-propylamine	5/22	5.00E-01 - 5.00E-01	3.60E-01 - 4.10E-01	2.83E-01		7.3B-04	mq/kq	Yes/P
N-Nitrosodiphenylamine	5/22	5.00B-01 - 5.00B-01	3.60B-01 - 4.10E-01	2.83E-01		1.0E+00	mg/kg	Yes/Bio
Naphthalene	5/22	5.00B-01 - 5.00B-01	3.60B-01 - 4.10E-01	2.83E-01	1.3E+01		mg/kg	Yes/Bio
Nitrobenzene	5/22	5.00E-01 - 5.00E-01	3.60B-01 - 4.10B-01	2.838-01	6.1E-01		mg/kg	No
PCB-1016	3/23	1.02E-01 - 1.28E-01	9.80B-02 - 1.87B+00	2.54B-02	2.3E-01	9.9E-03	ng/kg	Yes/P
PCB-1221	2/23	1.02E-01 - 5.45E-01	9.80E-02 - 1.70E-01	7.82E-02		1.1E-02	nng/kg	Yes/P
PCB-1232	2/23	1.02E-01 - 5.45E-01	9.808-02 - 1.70E-01	7.828-02		1.1E-02	mg/kg	Yes/P
PCB-1242	2/23	1.02E-01 - 5.45E-01	9.80E-02 - 1.70E-01	7.828-02		9.7B-03	mg/kg	Yes/P
PCB-1248	2/23	1.02E-01 - 5.45E-01	9.808-02 - 1.708-01	7.828-02		1.1E-02	mg/kg	Yes/P
PCB-1254	3/23	1.02K-01 - 5.45E-01	9.60E-02 - 3.50E-01	5.92E-02	6.6E-02	9.9E-03	mg/kg	Yes/P
	-,							

*F= > FRG, B= > Background, E= > Essential Nutrient, Bio=Bioaccumulates, Qual=Qualitative analyte

		Location-sym	U 99A MEDIA-Subsurface & (continued)	Soil				
Analyte	Frequency of Detection	Nondetected Range	Detected Range	Arithmetic Mean	HI	ELCR	Units	COPC/ Basis*
PCB-1260	7/23	1.02B-01 - 5.45B-01	6.00B-02 - 6.31B-01	1.04E-01		9.8E-03	mg/kg	Yes/P
Pentachlorophenol	5/22	5.00E-01 - 5.00E-01	1.80E+00 - 2.10E+00	6.43B-01	7.9E+01	1.3B-01	wg/kg	Yes/P
Phenanthrene	7/22	5.00B-01 - 5.00E-01	3.60E-01 - 1.63E+00	4.41E-01			wg/kg	Yes/Qual
Phenol	5/22	5.00E-01 - 5.00E-01	3.60E-01 - 4.10E-01	2.83E-01	1.4B+03		mg/kg	No
Pyrene	8/22	5.00B-01 - 5.00B-01	1.30E-01 - 2.70E+00	4.25E-01	3.2E+01		mg/kg	Yes/Bio
Styrene	5/8	1.00E-02 - 1.00E-02	6.00B-03 - 6.00B-03	5.63B-03	1.9E+02		mg/kg	No
Tetrachloroethene	5/8	1.00E-02 - 1.00E-02	6.00E-03 - 6.00E-03	5.638-03	1.2E+01	1.3B-01	mg/kg	No
Toluene	5/8	1.00E-02 - 1.00E-02	6.00E-03 - 6.00E-03	5.63B-03	9.8E+01		mg/kg	No
Toxaphene	2/2		2.00E-01 - 3.50E-01	2.75E-01		8.5E-03	mg/kg	Yes/P
Trichloroethene	6/10	1.98E-01 - 5.27E-01	4.808-03 - 6.00E-03	1.36B-01	1.2E+00	9.1E-02	tng/kg	No
Vinyl Acetate	5/5		1.10E-02 - 1.20E-02	1.18E-02	9.5E+01		mg/kg	No
Vinyl Chloride	5/10	1.98E-01 - 1.00E+01	1.10E-02 - 1.20E-02	1.20E-02		1.5B-03	mg/kg	Yes/P
Xylene	5/5		4.90E-03 - 6.90E-03	5.60E-03	1.7E+03		mg/kg	Yes/Bio
alpha-BHC	2/2		9.80B-03 - 1.70B-02	1.34E-02		2.4E-03	mg/kg	Yes/P
alpha-Chlordane	2/2		9.80B-02 - 1.70B-01	1.34E-01			mg/kg	Yes/Qual
beta-BEC	2/2		9.80E-03 - 1.70E-02	1.34E-02		8.28-03	mg/kg	Yes/P
bis(2-Chloroethoxy)methane	5/22	5.00E-01 - 5.00E-01	3.60E-01 - 4.10E-01	2.83E-01			mg/kg	Yes/Qual
bis (2-Chloroethyl) ether	5/22	5.00E-01 - 5.00E-01	3.60E-01 - 4.10E-01	2.83E-01		5.9B-03	mg/kg	Yes/P
bis (2-Chloroisopropyl) ether	5/22	5.00B-01 - 5.00B-01	3.60B-01 - 4.10B-01	2.83E-01		1.1E-01	mg∕kg	Yes/P
bis(2-Ethylhexyl)phthalate	5/22	5.00E-01 - 5.00E-01	7.90E-02 - 3.60E-01	2.37B-01	1.4E+01	2.8E-01	mg/kg	Yes/P
cis-1,3-Dichloropropene	5/8	1.00B-02 - 1.00B-02	6.00B-03 - 6.00B-03	5.63B-03			mg/kg	Yes/Qual
	- / -							

9.80E-03 - 1.70E-02

9.80E-03 - 1.70E-02

9.80E-02 - 1.70E-01

6.00E-03 - 6.00E-03

9.708+00 - 1.428+02

6.708+00 - 2.73E+03

1.10B+00 - 1.90B+00

-2.00E-03 - 1.28E+01

-5.00E-03 - 6.00E-03

-1.30E+00 - 2.65E+03

5.80B-01 - 6.70E-01

5.30E+01 - 5.30E+01

1.80E-01 - 1.64E+01

7.20E-03 - 4.10E-02

2.308-01 - 5.178+01

1.34E-02

1.34B-02

1.34E-01

5.63E-03

2.33B+01

6.15B+01

3.77B-01

3.208+00

1.00E-03

1.19B+02

6.30E-01

1.58E+01

4.39E+00

4.25E+00

1.37E+01

7.7E-01

mg/kg

mg/kg

¤g/kg

mg/kg

pCi/g

pCi/g

pCi/g

pCi/g

pCi/g

pCi/g

pCi/g

pCi/g

pCi/g

pCi/g

pCi/g

1.2B-02

1.6E-02

6.8E-02

2.0E+00

4.4E+02

1.6E+01

7.2E+00

1.4E+01

1.2E-01

4.7B-01

Yes/Qual

Yes/Qual

Yes/Qual

Yes/Qual

Yes/Qual

Yes/PB

Yes/P

Yes/PB

Yes/P

Yes/PB

Yes/PB

No

No

No

Yes/P

*P= > PRG, B= > Background, E= > Essential Nutrient, Bio=Bioaccumulates, Qual=Qualitative analyte

A-152

delta-BHC

gamma-BHC (Lindane)

trans-1,3-Dichloropropene

gamma-Chlordane

Alpha activity

Beta activity

Neptunium-237

Plutonium-239

Technetium-99

Thorium-230

Thorium-234

Uranium-234

Uranium-235

Uranium-238

Cesium-137

2/2

2/2

2/2

5/8

20/21

21/21

3/21

4/4

3/3

6/23

3/3

1/21

4/4

3/24

4/4

1.00E-02 - 1.00E-02

3.10E+00 - 3.10E+00

3.80E-01 - 3.50E+00

0.00E+00 - 3.73E+00

5.30B+00 - 2.20B+01

1.30E+00 - 9.90E+00

----- LOCATION=5WMU 99A MEDIA=Surface Soil ------

Analyte	Frequency of Detection	Nondetected Range	Detected Range	Arithmetic Mean	HI	RLCR	Units	COPC/ Basis*
maryce	2010-1202		30				0	0-010
Aluminum	13/13		1.80E+03 - 1.29E+04	6.19E+03	7.3E+02		ng/kg	No
Arsenic	6/13	5.008+00 - 5.008+00	5.55E+00 - 8.55E+00	4.47E+00	6.9B-01	9.2E-03	mg/kg	No
Barium	13/13		2.08E+01 - 2.47E+03	2.11E+02	3.7E+01		mg/kg	Yes/PB
Beryllium	5/13	5.00B-01 - 5.00E-01	5.20E-01 - 8.90E-01	5.38E-01	1.6E-01	1.0E-04	mg/kg	Yes/PB
Calcium	11/11		6.10E+03 - 2.87E+05	2.44B+05			mg/kg	No
Chromium	13/13		7.00B+00 - 4.57B+01	1.478+01	4.8E-01	4.9E+02	mg/kg	Yes/P
Cobalt	11/13	1.00B+00 - 1.00B+00	1.68E+00 - 9.67E+00	3.70E+00	2.1E+02		mg/kg	No
Copper	12/13	2.00B+00 - 2.00B+00	4.37E+00 - 1.22E+01	6.66E+00	7,4E+01		mg/kg	NO
Iron	13/13		1.458+03 - 2.33B+04	1.09E+04	3.1E+02		mg/kg	No
Lithium	13/13		2.82E+00 - 1.29E+01	7.582+00	7.08+01		mg/kg	No
Magnesium	13/13		1.35E+03 - 2.73E+04	1.09E+04			ng/kg	No
Manganese	13/13		3.93E+01 - 3.87E+02	1.91E+02	1.45+01		mg/kg	No
Nickel	. 8/13	5.008+00 - 5.00 <u>8</u> +00	5.47E+00 - 2.16E+01	8.52E+00	3.48+01		mg/kg	No
Potassium	13/13		2.91E+02 - 1.12E+03	5.47E+02			mg/kg	No
Sodium	6/13	2.00E+02 - 2.51E+02	2.17E+02 - 3.66E+02	1.95B+02			mg/kg	Yes/B
Strontium	13/13		1.46B+01 - 5.14B+02	2.71E+02	8.0E+02		mg/kg	No
Vanadium	13/13		4.488+00 - 3.558+01	1.54B+01	5.6E-01		mg/kg	No
Zinc	12/13	4.76E+01 - 4.76E+01	4.71E+01 - 1.63E+02	8.24E+01	4.0E+02		mg/kg	Yes/B
Acenaphthene	2/13	5.00B-01 - 5.00B-01	3.00E-01 - 3.30E-01	3.228-01	6.4E+01		mg/kg	Yes/Bio
Acenaphthylene	1/13	5.008-01 - 5.002-01	6.108-01 - 6.108-01	2.78B-01	<i>c</i> ==		mg/kg	Yes/Qual
Anthracene	2/13	5.00E-01 - 5.00E-01	4.918-01 - 7.508-01	4.03E-01	6.52+02		mg/kg	Yes/Bio
Benz (a) anthracene	3/13	5.00B-01 - 5.00B-01	2.20B-01 ~ 1.70B+00	3.38B-01		8.5B-03	mg/kg	Yes/P
Benzo (a) pyrene	2/13	5.00B-01 - 5.00B-01	1.70B+00 - 2.10E+00	5.04B-01		8.5E-04	mg/kg	Yes/P
Benzo (b) fluoranthene	6/13	5.00E-01 - 5.00E-01	1.70B-01 - 5.70B+00	5.31B-01		8.5E-03	mg/kg	Yes/P
Benzo (ghi) perylene	2/13	5.008-01 - 5.008-01	5.50B-01 - 1.18E+00	3.14E-01			mg/kg	Yes/Qual
Benzo (k) fluoranthene	3/13	5.00E-01 - 5.00E-01	4.66B-01 - 7.90B-01	4.53E-01		8.5E-02	mg/kg	Yes/P
Chrysene	2/13	5.00E-01 - 5.00E-01	1.36E+00 - 2.10E+00	4.782-01		8.5E-01	mg/kg	Yes/P
Dibenz (a, h) anthracene	1/13	5.00B-01 - 5.00B-01	4.80E-01 - 4.80E-01	2.68E-01	6	8.55-04	mg/kg	Yes/P
Dibenzofuran	1/13	5.008-01 - 5.008-01	1.23B-01 - 1.23E 01	2.40B-01	6.3B+00		mg/kg	Yes/Bio
Fluoranthene	4/13	5.00B-01 - 5.00B-01	1.40E-01 - 2.66E+00	3.38E-01	4.3B+01		mg/kg	Yes/Bio
Fluorene	1/13	5.00E-01 - 5.00E-01	2.19E-01 - 2.19B-01	2.488-01	6.3E+01		mg/kg	Yes/Bio
Indeno (1, 2, 3-cd) pyrene	2/13	5.00B-01 ~ 5.00B-01	7.80B-01 - 1.05E+00	3.528-01	a a a	8.5E-03	ng/kg	Yes/P
PCB-1016	1/16	1.02E-01 - 1.28E-01	1.87E+00 - 1.87E+00	1.728-01	2.3E-01	9.9E-03	mg/kg	Yes/P
PCB-1254	1/16	1.02E-01 - 5.45E-01	9.60E-02 - 9.60E-02	1.34B-01	6.6E-02	9.9E-03	mg/kg	Yes/P
PCB-1260	5/16	1.02B-01 - 5.45B-01	6.00E-02 - 6.31E-01	1.01E-01		9.8E-03	mg/kg	Yes/P
Phenanthrene	2/13	5.00B-01 - 5.00B-01	8.50B-01 - 1.63E+00	2.45E-01			mg/kg	Yes/Qual
Pyrene	3/13	5.00E-01 - 5.00E-01	1.30E-01 - 2.70E+00	2.74E-01	3.28+01		mg/kg	Yes/Bio
Alpha activity	15/16	3.10E+00 - 3.10E+00	9.708+00 - 1.428+02	2.53E+01			pCi/g	Yes/Qual
Beta activity	16/16		6.70 8 +00 - 2.73 8 +03	8.582+01			pCi/g	Yes/Qual
Cesium-137	3/16	3.80E-01 - 3.50E+00	1.10B+00 - 1.90B+00	4.50B-01		1.6E-02	pCi/g	Yes/PB
Neptunium-237	1/1		1.285+01 - 1.285+01	1.28E+01		6.8E-02	pCi/g	Yes/PB

*P= > PRG, B= > Background, E= > Essential Nutrient, Bio=Bioaccumulates, Qual=Qualitative analyte

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Table	1.10.	Summary	of	data	eva	luati	on
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LOCATION=SWMU 99A MEDIA=Surface Soil ------ LOCATION=SWMU 99A MEDIA=Surface Soil -----

Analyte	Frequency of Detection	Nondetected Range	Detected Range	Arithmetic Mean	HI	ELCR	Dnits	COPC/ Basis*
Technetium-99 Thorium-234	3/16 1/16	0.008+00 - 3.738+00 5.308+00 - 2.208+01	1.66E+01 - 2.65E+03 5.30E+01 - 5.30E+01	1.71E+02 1.67E+01		4.4E+02 7.2E+00	pCi/g pCi/g	Yes/PB Yes/P
Uranium-234 Uranium-238	1/1 1/1	3.304700 2.202701	1.64E+01 - 1.64E+01 5.17E+01 - 5.17E+01	1.64E+01 5.17E+01		1.4E+01 4.7E-01	pCi/g pCi/g pCi/g	Yes/PB Yes/PB

------ LOCATION-SHMU 99B MEDIA-RGA Groundwater -----

Analyte	Frequency of Detection	Nondetected Range	Detected Range	Arithmetic Mean	HI	ELCR	Units	COPC/ Basis*
Barium	7/7		2.00B-01 - 2.70E+00	4.78E-01	1.0E-01		mg/L	Yes/P
Calcium	7/7		2.84E+01 - 3.27E+01	3.04E+01			mg/L	No
Chloride	7/7		8.29E+01 - 1.08E+02	9.59E+01			mg/L	No
Chromium	1/7	5.00E-02 - 6.00E-02	2.60B-01 - 2.60B-01	5.62B-02	4.22-03		mg/L	Yes/P
Copper	1/7	2.50B-02 - 1.00B-01	4.00E-02 - 4.00E-02	3.25E-02	6.0E-02		mg/L	No
Fluoride	7/7		1.60E-01 - 2.10E-01	1.77E-01	9.1E-02		mg/L	No
Iron	3/7	3.00E-01 - 3.60E-01	2.94E-01 - 3.34E+00	6.08E-01	4.5E-01		mg/L	Yes/PE
Magnesium	7/7		1.15E+01 - 1.31E+01	1.232+01			mg/L	No
Manganese	5/7	1.00E-01 - 1.00E-01	6.00E-02 - 2.90E-01	1.65B-01	6.72-02		mg/L	Yes/P
Nitrate as Nitrogen	7/7		1.708+00 - 2.108+00	1,842+00	2.4E+00		mg/L	No
Silica	7/7		1.50E+01 - 2.00E+01	1.728+01			mg/L	Yes/Qual
Sodium	7/7		6.32E+01 - 7.86E+01	6.99E+01			mg/L	Yes/Qual
Sulfate	2/2		1.758+01 - 2.678+01	2.21E+01			wg/L	Yes/Qual
Tetraoxo-sulfate(1-)	5/5		1.92E+01 - 2.90E+01	2.46E+01			mg/L	Yes/Qual
Zinc	2/7	3.00E-02 - 2.50E-01	3.008-02 - 6.00E-02	3.72E-02	4.5E-01		mg/L	Yes/Bio
Trichloroethene	16/16		1.30E+00 - 2.30E+00	1.94E+00	1.2E-03	1.4B-04	mg/L	Yes/P
Alpha activity .	12/16	-2.03E+00 - 4.60E+00	-4.20E+00 - 4.20E+00	7.42B-01			pCi/L	Yes/Qual
Beta activity	16/16		3.00E+00 - 4.50E+01	1.20E+01			pCi/L	Yes/Qual
Radon-222	4/4		2.57E+02 - 4.12E+02	3.66E+02		1.4E+00	pCi/L	Yes/P
Technetium-99	12/17	-3_00E+00 - 1.17E+01	-2.00E+00 - 1.90E+01	5.51E+00		2.8E+01	pCi/L	No

*Pa > PRG, Ba > Background, Bo > Essential Nutrient, Bio-Bioaccumulates, Qual-Qualitative analyte

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Table 1.10. Summary of data evaluation

apalyte	Prequency of Detection	Nondetected Range	Detected Range	Arithmetic Mean	HI	ELCR	Units	COPC/ Basis
luminum	8/8		9.31E+03 - 1.70E+04	1.258+04	7.3E+02		mg/kg	Yes/P
Arsenic	2/6	5.00B+00 - 5.00B+00	6.89E+00 - 8.05E+00	4.16E+00	6.9E-01	9.2E-03	mg/kg	Yes/P
Barium	8/8		6.50E+01 - 1.55E+02	9.63E+01	3.7E+01		mg/kg	No
Beryllium	6/8	5.00B-01 - 5.00E-01	5.70E-01 - 1.00E+00	5.66B-01	1.6E-01	1.0E-04	mg/kg	Yes/F
alcium	6/6		5.03E+02 - 7.17E+03	2.51E+03			mg/kg	No
hromium	8/8		1.18E+01 - 2.61E+01	1.79E+01	4.8E-01	4.9E+02	ing/kg	Yes/J
obalt	8/8		1.91E+00 - 6.94E+00	4.17E+00	2.1E+02	-	mg/kg	No
opper	8/8	· .	5.25E+00 - 1.30E+01	8.66E+00	7.4E+01		mg/kg	No
rón	8/8		9.66B+03 - 1.81B+04	1.48E+04	3.1E+02		mg/kg	No
itbium	8/8	-	6.50E+00 - 1.14E+01	8.62B+00	7.0E+01		mg/kg	No
agnesium	8/8		1.10E+03 - 2.53E+03	1.76E+03			mg/kg	No
inganese	8/8	-	6.32E+01 - 5.24E+02	2.43E+02	1.4E+01		mg/kg	No
ckel	5/8	5.00E+00 - 5.00E+00	7.27E+00 - 2.51E+01	1.05E+01	3.4E+01		mg/kg	No
tassium	8/8	4	3.37E+02 - 1.04E+03	6.44E+02			mg/kg	No
dium	3/8	2.008+02 - 2.008+02	2.11E+02 - 3.09E+02	1.58E+02			mg/kg	No
rontium	8/8	····	9.46E+00 - 2.22E+01	1.61B+01	8.0E+02		mg/kg	No
anadium	8/8		1.978+01 - 3.448+01	2.46B+01	5.6B-01		mg/kg	No
Inc	8/8		1.96B+01 - 5.22E+01	3.74E+01	4.08+02		mg/kg	No
cetone	1/7	1.20E+00 - 1.20E+00	5.50B-01 - 5.50B-01	5.93B-01	9.2E+01	3 A. A.	mg/kg	No
ethylene Chloride	.3/7	1.20E+00 - 1.20E+00	1.20E+00 - 1.20E+00	8.57E-01	7.0E+01	5.0B-01	mg/kg	Yes/
lpha activity	8/8	· · ·	1.33E+01 - 2.14E+01	1.73E+01			pCi/g	Yes/
Seta activity	8/8		1.48E+01 - 2.26E+01	1.86E+01			pC1/g	Yes/

*P= > PRG, B= > Background, E= > Essential Nutrient, Bio-Bioaccumulates, Qual-Qualitative analyte

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Table 1.11. Representative Concentrations* of COPCs in media

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Analyte	RGA Groundwater (mg/L or pC1/L)	McNairy Groundwater (mg/L or pCi/L)	Surface soil (mg/kg or pCi/g)	Subsurface soil (mg/kg or pCi/g
1,1-Dichloroethane	5.00B+00			
1,1-Dichloroethene	4.00E-02			4.00E-02
PCB-1254	2.50E-02			2.50E-02
PCB-1260	2.50E-02			2.50E-02
Polychlorinated biphenyl	1.70E-01			1.00E-01
Tetrachloroethene	6.41E-01			8.10E-01
Trichloroethene	5.51E-01		-	4.17E-01
Vinyl Chlorida	1.00E-04			
cis-1,2-Dichloroethene	6.00E-03			
Alpha activity	6.06E+00			1.85E+01
Beta activity	4.95E+00			2,61E+01

LOCATION-SWMU 193A -----

Analyte	RGA Groundwater (mg/L or pCl/L)	McNairy Groundwater (mg/L or pCi/L)	Surface soil (mg/kg or pCi/g)	Subsurface soil (mg/kg or pCi/g
Aluminum				6.14E+03
Ammonia	3.00E-01			
Beryllium				6.38E-01
Chromium			1.225+01	1,12E+01
Fluoride	4.20B-01			
Iron	3.02B+01	1.328+02		
Silica	1.905+01			•
Tetraoxo-sulfate(1-)	1.02E+02	4.11E+01		
Zinc	9.87E-02	-		
1,1-Dichloroethene	2.00E-04	-		
Anthracene		•	1.16E-01	1.16E-01
Benz (a) anthracene		•	1.80R-01	1.80E-01
Benzo (a) pyrene			2.50B-01	2.50E-01
Benzo (b) fluoranthene			5.10R-02	5.10E-02
Benzo (ghi) perylene			1.708-01	1.70E-01
Chrysena			· 1.70E-01	1.70E-01
Di-n-butylphthalate			7.70E-02	7.70E-02
Di-n-octylphthalate			1.20B-01	1.20E-01
Dibenz (a, h) anthracene			1.30E-01	1.30E-01
Fluoranthene			2.738-01	3.105-01
Indeno (1,2,3-cd) pyrene	•		1.60E-01	1.60E-01
Pentachlorophenol	8.47E-03			
Pyrene			2.95E-01	2.66E-01
Trichloroethene	1.69E-01	3.72B-03		
bis (2-Ethylhexyl) phthalate	1.29E-02		1.708-01	1.708-01
cis-1,2-Dichloroethene	2.91E-03	1.70E-01		
Alpha activity	4.725+00	1.50E+01	1.70E+01	1.99E+01
Beta activity	1.73E+02	4.842+01	2.37B+01	2.14E+01
Tachnetium-99	1.928+02	3.69B+01		
Dranium-238		1.32B+00		

Analyte	RGA Groundwater	McNairy Groundwater	Surface soil	Subsurface soil
	(mg/L or pC1/L)	(mg/L or pCi/L)	(mg/kg or pCi/g)	(mg/kg or pCi/g)
Beryllium Chromium Vanadium 1,1-Dichloroethene Acetone Carbon Tetrachloride Di-n-butylphthalate	1.62E-03 3.30E-02 5.50E-03 1.02E-02		1.57%+00 8.87%+01 6.50%+01	7.02E-01 3.84E+01 2.89E+01

* Smaller of maximum detect and UCL95

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Table 1.11. Representative concentrations* of COPCs in media

Analyte (mg/L or pCl/L) (mg/L or pCl/L) (mg/kg or pCl/g) (mg/kg or Autimony 3.8228-01 5.1624 Antimony 1.143-01 5.1624 Arsenic 1.222-02 6.8324 Bartium 2.4428-01 6.8324 Cadmium 3.5428-02 2.0554 Chronium 1.432-02 6.8324 Chronium 3.5428-02 2.0554 Chronium 1.6326-01 5.4724+00 2.1554 Chronium 5.3225-02 1.0324 1.0324 Kend 2.508-01 2.4524+01 1.3564 Manganese 1.0574-02 3.8124 3.8124 Manganese 2.008-04 3.8124 3.8124 Manganese 2.008-01 2.4524-01 1.3564 Manganese 2.008-01 2.6524-01 1.3624 Manganese 2.008-01 2.6524-01 1.6224 Silter 3.322-02 7 7 Tetracoro-sulfate(1-) 6.55958+00 1.7754 <t< th=""><th>net1um-99</th><th>2.736+01</th><th></th><th>•</th><th>•</th></t<>	net1um-99	2.736+01		•	•
RGA Groundwater (mg/L or pCL/L) McMairy Groundwater (mg/L or pCL/L) Surface soil (mg/Kg or pCL/g) Subsurface (mg/Kg or pCL/g) Aluminum 3.822+01 5.1624 Antimony 1.122-02 6.832 Arsenic 1.122-02 6.832 Barium 2.442-01 2.542 Cadmium 1.432-02 6.832 Cadmium 3.542-02 2.554 Chronium 1.432-02 0.1354 Chronium 5.892+01 1.392 Iron 5.892+01 1.392 Iron 5.892+01 1.392 Manganese 1.362+00 3.812 Manganese 1.352+02 1.354 Molybdennm 4.622-02 1.354 Molybdennm 6.402+03 1.778 Molybdennm 6.402+03 1.778 Molybdennm 2.007-04 1.778 Molybdennm 2.008-03 1.778 Molybdennm 2.008-03 1.778 Molybdennm 2.008-03 1.778 Molybdennm	netium-99	2.73E+01			
RGA Croundwater (mg/L or pCl/L) McMairy Groundwater (mg/L or pCl/L) Surface soil (mg/Kg or pCl/g) Subsurface (mg/Kg or science) Aluminum 1.82E-01 5.1624 Antimony 1.122E-02 6.83E Arsenic 1.122E-02 6.83E Earlum 2.44E-01 5.155E Cadmium 1.43E-02 5.47E+00 2.155E Choshit 5.32E-02 1.035E 1.035E Iron 5.89E+01 1.33E 1.35E Iron 5.89E+01 1.35E 1.35E Manganese 1.36E+00 3.81E 1.35E Manganese 1.36E+00 3.81E 1.35E Manganese 1.36E+00 3.81E 1.35E Manganese 2.00F-04 3.81E 1.35E Molybdennm 6.40E-03 1.77E 1.35E Wolybdennm 6.40E-03 1.77E 1.77E Silvar 2.03E-01 4.16E+01 5.95E Vinckal 5.02E-03 1.72E 1.77E Silvar <td< td=""><td></td><td>LOC</td><td>ATION-SHMU 193C</td><td></td><td></td></td<>		LOC	ATION-SHMU 193C		
Analyte (mg/L or pCl/L) (mg/L or pCl/L) (mg/kg or pCl/g) (mg/kg or pCl/g) Antimoum 3.828+01 5.1624 Antimouy 1.428-01 5.1624 Arsenic 1.228-02 6.818- Baryina 2.448-01 5.052 Baryina 1.128-02 6.818- Cadadum 3.648-02 2.0584 Chronium 1.132-02 6.818- Cadadum 3.648-02 2.0584 Chronium 5.1328-02 1.0324 Kosait 5.328-01 1.3524 Manganese 2.508-01 2.498+01 1.3524 Marcury 2.008-04 0.8184 0.8184 Molybdemm 4.622-02 1.6184 Mickal 5.378-02 1.8184 Silivar 3.328-02 1.21840 Trainium 6.402-03 1.2784 Zinac 2.002-04 1.2784 Zinac 2.002-03 1.2784 Zinac 2.002-03 1.2784 Zinachu					Subsurface soil
Antinory 1.148-01 Arsenic 1.22E-02 Barium 2.44E-01 Beryllium 3.54E-02 Cadmium 3.54E-01 Chromium 1.43E-01 Chromium 3.54E-02 Chromium 1.43E-01 Chromium 5.69E+01 Lead 5.69E+01 Manganese 1.36E+00 Marganese 1.36E+00 Marganese 3.32E-02 Marken 2.69E+01 Marken 2.60E+01 Marken 2.60E+02 Marken 3.32E-02 Marken 5.77E-02 Marken 5.39E+00 Thellium 5.39E+00 Thellium 1.21E-01 Trains 6.40E-01 Vanadium 6.36E-01 1.2.12E-01 5.95E+02 1.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2.2	yte				(mg/kg or pCi/g
Arsenic 1.228-02 Barylinum 2.448-01 Baryllium 1.118-02 6.838- Cadmium 3.548-02 2.0578- Chromium 1.438-01 5.472+00 2.158- Cubalt 5.328-02 1.012- Iron 5.328-02 1.012- Manganese 1.367-02 1.358- Manganese 1.367-02 3.818- Marcury 2.008-04 3.818- Marcury 2.008-04 3.818- Marcury 2.008-04 3.818- Marcury 2.008-04 3.818- Marcury 2.008-03 3.818- Marcury 3.328-02 5.178-02 Silica 6.012+00 5.178-02 Silver 3.328-01 4.168+01 5.958-03 Trainum 6.408-03 1.278-01 1.278-01 Dranium 2.508-03 1.278-01 1.378-01 1,1-2-Trichloroethane 2.508-03 1.278-01 1.278-01 1,2-Dichloroethane 5.628-01 1.278-03 2.508-03 1,2-Dichloroethane		·			5.16E+03
Barium 2.448-01 Beryllium 1.118-02 6.838-01 Cadmium 3.648-02 2.0584 Chromium 1.438-01 5.47E+00 2.1584 Chromium 5.328-02 1.0124 1.0124 Iron 5.898+01 1.0124 1.0124 Iron 2.508-01 2.498+01 1.3684 Manganess 1.367400 3.6184 3.6184 Marcury 2.008-04 3.6184 3.6184 Molyddenum 4.628-02 Mickel 3.6184 Silivar 3.328-02 7 7 Tetraxxo-sulfate(1-) 6.598-03 1.2784 7 Xinac 2.038-01 4.168+01 5.9584 1, 1, 2-Trichloroethane 2.508-03 1.2784 7 JDichloroethane 2.508-03 1.2784 1.2784 Zinde 2.508-03 2.508-03 1.2784 JDichloroethane 2.508-03 1.2784 1.2784 Striphonorethane 2.508-03 1.2784		· · ·			
Beryllium 1.112-02 6.83E Candmium 3.54E-02 2.05E Chromium 1.43E-01 5.47E+00 2.15E Cobalt 5.32E-02 1.03E Cobalt 5.32E-02 1.03E Lead 2.00E-04 1.35E Manganese 1.05E+01 2.45E+01 1.35E Manganese 1.05E+01 2.45E+01 1.35E Marganese 2.00F-04 3.81E 3.81E Marganese 6.01E+00 3.81E 3.81E Marganese 6.01E+00 3.81E 3.81E Marganese 6.01E+00 3.81E 3.81E Marganese 6.01E+00 3.81E 3.81E Marganese 6.01E+00 3.81E 3.81E Silver 3.12E-02 3 3.81E 3.81E Silver 3.22E-02 3 3.81E 3.81E Vanadium 1.23E-01 4.16E+01 5.95E 5.95E 3.81E Vanadium 6.30E-03 1.4.16E+01 5.95E 5.95E 3.95E 3.95E 3.95E 3.9					
Cadmium 3.548-02 2.05F Chromium 1.43E-01 5.47E+00 2.15F Cobalt 5.32E-02 1.03E Iron 5.89F+01 2.15F Cadmium 2.50E-01 2.49E+01 1.33E Nanganese 2.30F+04 3.81E Morcury 2.00F+04 3.81E Morcury 2.00F+04 3.81E Morcury 2.00F+04 3.81E Mickal 5.37E-02 3.81E Silica 6.01E+00 3.81E Mickal 5.37E-02 3.81E Silica 8.01E+00 3.81E Silica 6.01E+00 5.91E+00 Thallium 1.23E+01 1.27E Urantium 6.40E+03 1.27E Vanadium 6.36E+01 1.27E J.1.2-Dichloroethane 2.50E+03 1.27E J.2-Dichloroethane 2.50E+03 1.27E J.2-Dichloroethane 2.50E+03 1.20E J.2-Dichloroethane 2.50E+03 1.20E J.2-Dichloroethane 2.50E+03 1.20E Strandichloroethane 2.50E+03 1.20E Polychlorinated biphenyl 1.00E+04 1.00E+04 Tetrachloroethane 2.50E+03					
Chromium 1.43E-01 5.47E+00 2.15E+ Cobalt 5.32E-02 1.01E+ Lead 5.89E+01 2.15E+ Manganese 2.50E-01 2.45E+01 1.36E+ Manganese 2.00E-04 3.81E+ Mercury 2.00E-04 3.81E+ Mickal 5.37E-02 511ea Silica 8.01E+00 3.81E+ Silica 8.01E+00 5.37E-02 Silica 8.01E+00 5.37E-02 Silica 8.01E+01 5.37E-02 Silica 8.01E+01 5.57E+00 Thallium 1.23E-01 1.27E+ Trainim 6.40E-03 1.27E+ Vanadium 2.50E-03 1.27E+ 1,2-Dichloroethane 2.50E-03 1.27E+ 1,2-Dichloroethane 5.62E-01 2.50E-03 1,2-Dichloroethane 2.50E-03 1.27E+ Sthybensene 2.50E-03 1.27E+ Bornoodichloromethane 2.50E-03 1.27E+ Chloroform 2.50E-03 1.62E-01 Polychlorothane 1.62E-01 1.					6.83B-01
Cobalt 5.32E-02 1.0124 Iron 5.89E+01 1.3954 Iron 2.50E-01 2.49E+01 1.36E Marganese 1.36E+00 3.81E Mercury 2.00E-04 3.81E Mickel 5.37E-02 3.81E Silica 6.01E+00 5.89E+00 Silver 3.32E-02 7 Tetracoro-sulfate(1-) 6.59E+00 1.23E-01 Uranium 6.40E+00 1.27E+02 Vanadium 1.23E-01 1.7E+01 Uranium 6.36E-01 1.77E+01 Vanadium 2.03E-01 4.16E+01 5.95E+1 1,1-Dichloroethane 2.50E-03 1.21E+01 5.95E+1 1,2-Dichloroethane 2.50E-03 2.50E-03 2.50E-03 1,2-Dichloroethane 2.50E-03 2.50E-03 2.50E-03 Carbon Tetrachloride 2.50E-03 2.50E-03 2.50E-03 Chloroform 2.50E-03 2.50E-03 2.50E-03 Chloroform 2.50E-03 2.50E-03 2.50E-03 Chloroform 2.50E-03 2.50E-03 2.50E				5 477 - 00	
Tron 5.892+01 1.392+ Lead 2.502-01 2.492+01 1.362+ Manganese 1.362+00 3.813+ Molyhdemum 4.622-02 3.814+ Nickel 5.372-02 3.814+ Silver 3.322-02 7 Tetracoxo-sulfate(1-) 6.592+00 3.122-02 Thallium 1.322-02 7 Tranium 6.402-03 1.278+ Vanadium 6.402-03 1.278+ J., 2-Trichloroethane 2.502-03 1.278+ 1, 2-Trichloroethane 2.502-03 1.278+ 1, 2-Dichloroethane 2.502-03 1.278+ 1, 2-Dichloroethane 2.502-03 1.278+ 1, 2-Dichloroethane 2.502-03 1.278+ 1, 2-Dichloroethane 2.502-03 1.278+ Score-03 2.502-03 1.278+ Carbon Tetrachlorida 2.502-03 1.278+ Chloroform 2.502-03 1.278+ Polychlorinated biphenyl 1.002-04 1.022-03 Trichloroethene 2.502-03 1.728+ Polychlorinated bip		•		5.4/4+00	1.01E+01
Lead 2.50E-01 2.49E+01 1.36E+ Marganese 1.36E+00 3.81E4 Marganese 2.00E-04 3.81E4 Molyhdenma 4.62E-02 3.81E4 Mickel 5.37E-02 3.81E4 Silica 8.01E+00 5.37E-02 Silvar 3.32E-02 7 Tetracoc-sulfate(1-) 6.59E+00 1.27E+02 Thallium 1.32E-01 1.27E+1 Vanadium 6.36E-01 4.16E+01 5.95E+0 1,1-2.Trichloroethane 2.50E-03 1.27E+1 1,2-Trichloroethane 2.50E-03 1.27E+1 1,2-Dichloroethane 2.50E-03 1.27E+1 1,2-Dichloroethane 2.50E-03 1.25E-03 Barsene 2.50E-03 2.50E-03 Bromodichloroethane 2.50E-03 2.50E-03 Chloroform 2.50E-03 2.50E-03 Ethylbansene 2.50E-03 2.50E-03 Polychlorinated biphenyl 1.00E-04 1.62E-01 Trichloroethane 1.62E-01 1.33E-03<					1.39E+04
Marcury 2.008-04 Molyhdenna 4.628-02 Molyhdenna 5.378-02 Silica 8.018+00 Silver 3.128-02 Tetracoc-sulfate(1-) 6.592+00 Thallium 1.215-01 Uranium 6.408-03 Vanadium 8.368-01 1.1.215-01 1.278-4 Zina 2.038-01 4.168+01 1.1.215-01 1.278-4 Zina 2.038-01 4.168+01 1.1.215-01 2.508-03 1.278-4 J.1-Dichloroethane 2.508-03 1.278-4 J.2-Dichloroethane 2.508-03 1.278-4 J.2-Dichloroethane 2.508-03 2.508-03 J.2-Dichloroethane 2.508-03 2.508-03 Chloroform 2.508-03 2.508-03 Bensena 2.508-03 2.508-03 Chloroform 2.508-03 2.508-03 Ethylbansena 2.508-03 2.508-03 Polychlorinated biphenyl 1.002-04 1.0025-04 Trichloroet			2.50B-01	2.49B+01	1.36E+01
Molyhdemma 4.62E-02 Nickel 5.37E-02 Nickel 6.01E+00. Silver 3.32E-02 Tetraoxo-sulfate(1-) 6.59E+00 Thallium 1.23E-01 Uranium 6.40E-03 Vanadium 6.36E-01 4.16E+01 Silver 2.03E-01 4.16E+01 5.95E+0 Vanadium 2.50E-03 1.27E+ 1,1-Dichloroethane 2.50E-03 1.27E+ 1,2-Dichloroethane 2.50E-03 1.27E+ 1,2-Dichloroethane 2.50E-03 1.27E+ 1,2-Dichloroethane 2.50E-03 1.27E+ Stronodichloroethane 2.50E-03 1.27E+ Bernsene 2.50E-03 2.50E-03 Chloroform 2.50E-03 2.50E-03 Chloroform 2.50E-03 2.50E-03 Polychlorinated biphenyl 1.00E-04 1.00E-04 Trichloroethane 1.62E-01 1.23E+03 Vinyl Chloride 9.19E+03 2.64E+ Trichloroethene 1.62E+03 2.64E+<					3.81E+02
Silica 5.37E-02 Silica 6.01E+00 Silver 3.32E-02 Tetracco-sulfate(1-) 6.59E+00 Thallium 1.23E-01 Uranium 6.46E-03 Vanadium 8.36E-01 1.27E+ Zinc 2.03E-01 4.16E+01 5.95E+0 1,1-2.Trichloroethane 2.50E-03 1.27E+ 1,1-Dichloroethane 2.50E-03 1.27E+ 1,2-Dichloroethane 2.50E-03 1.27E+ 1,2-Dichloroethane 2.50E-03 1.27E+ 1,2-Dichloroethane 2.50E-03 1.27E+ Sansene 2.50E-03 1.27E+ Bensene 2.50E-03 2.50E-03 Bromodichloromethane 2.50E-03 2.50E-03 Carbon Tetrachloride 2.50E-03 2.50E-03 Ethylbanzene 2.50E-03 2.50E-03 Polychlorinated biphenyl 1.00E-04 1.62E-01 Trichloroethene 1.62E-01 1.23E-03 Vinyl Chloride 9.19E-03 2.84E-03 Xylene 5.00E-03 2.84E-03 Cia-1, 2.Dichloroethene 5.					
Silica 8.01E+00. Silver 3.32E-02 Tetraoxo-sulfate(1-) 6.59E+00. Thallium 1.23E-01 Uranium 6.40E-03 Vanadium 2.03E-01 Zinc 2.03E-01 1,1.7.2-Trichloroethane 2.50E-03 1,2-Dichloroethane 2.50E-03 1,2-Dichloroethane 5.62E-01 Bensene 2.50E-03 2.50E-03 2.50E-03 Carbon Tetrachloride 2.50E-03 Chloroothane 2.50E-03 Chloroothane 2.50E-03 Chloroothane 2.50E-03 Chloroothane 2.50E-03 Chloroothane 2.50E-03 Chloroothane 2.50E-03 Chloroothane 2.50E-03 Chloroothane 2.50E-03 Chloroothane 2.50E-03 Chloroothane 2.50E-03 Chloroothane 2.50E-03 Chloroothane 2.50E-03 Chloroothane 2.50E-03 Trichloroethane 1.62E-01 Silvelae 3.15E-03 Vinyl Chloride <td< td=""><td></td><td></td><td>-,</td><td>-</td><td></td></td<>			-,	-	
Silver 3.328-02 Tetracxco-sulfate(1-) 6.598+00 Thallium 1.23E-01 Uranium 6.408-03 Vanadium 6.36E-01 1.278+ Zinc 2.03E-01 4.16E+01 5.95E+ 1,1,2-Trichloroethane 2.50E-03 1,278+ 1,1-Dichloroethane 2.50E-03 1,278+ 1,2-Dichloroethane 2.50E-03 1,278+ 1,2-Dichloroethane 2.50E-03 1,278+ 1,2-Dichloroethane 2.50E-03 1,278+ 1,2-Dichloroethane 2.50E-03 1,278+ 1,2-Dichloroethane 2.50E-03 2.50E-03 Barsena 2.50E-03 2.50E-03 Carbon Tetrachloride 2.50E-03 2.50E-03 Chloroform 2.50E-03 2.50E-03 Ethylbansena 2.50E-03 2.50E-03 Polychloriated biphenyl 1.00E-04 1.00E-04 Tetrachloroethane 1.62E-01 1.23E-03 Yinyl Chloride 9.19E-03 2.84E-03 Cis-1,2-Dichloroethane 5.00E-03 2.84E-03 cis-1,2-Dichloroethane 5.00E-03					
Tetracxo-sulfate(1-) 6.595+00 Thallium 1.235-01 Uranium 6.405-03 Vanadium 2.038-01 4.168+01 Zinc 2.038-01 4.168+01 5.9584 1,1-Dichloroethane 2.508-03 1.2784 1,2-Dichloroethane 2.508-03 1.218 1,2-Dichloroethane 2.508-03 1.218 1,2-Dichloroethane 2.508-03 1.218 1,2-Dichloroethane 2.508-03 1.218 1,2-Dichloroethane 2.508-03 2.508-03 2,2-Dichloroethane 2.508-03 2.508-03 Carbon Tetrachloride 2.508-03 2.508-03 Chloroform 2.508-03 2.508-03 Polychlorinated biphenyl 1.008-04 1.628-03 Tetrachloroethane 1.628-01 1.238-03 Vinyl Chloride 9.198-03 2.848-03 Vinyl Chloroethane 5.008-03 2.848-03 cis-1, 2-Dichloroethane 5.008-03 2.848-03 cis-1, 2-Dichloroethane 5.008-03 2.848-03 cis-1, 2-Dichloroethane 5.008-03 2.848-03					•
Thallium 1.23E-01 Uranium 6.40E-03 Vanadium 8.36E-01 1.27E4 Zinc 2.03E-01 4.16E+01 5.95E4 1,1,2-Trichloroethane 2.50E-03 1.127E4 1,2-Dichloroethane 2.50E-03 1.27E4 1,2-Dichloroethane 2.50E-03 1.27E4 1,2-Dichloroethane 2.50E-03 1.27E4 1,2-Dichloroethane 5.62E-01 1.27E4 Bensena 2.50E-03 1.27E4 Bromodichloromethane 2.50E-03 1.27E4 Carbon Tetrachlorida 2.50E-03 2.50E-03 Carbon Tetrachlorida 2.50E-03 2.50E-03 Chloroform 2.50E-03 2.50E-03 Polychlorinated biphenyl 1.00E-04 1.00E-04 Tetrachloroethene 1.62E-01 1.23E-03 Vinyl Chloride 9.19E-03 2.84E-03 Vinyl Chloroethene 5.00E-03 2.84E-03 cis-1, 2-Dichloroethene 5.00E-03 2.84E-01 cis-1, 2-Dichloroethene 5.00E-03 2.84E-03 Carbon Tide 9.19E-03 2.84E-03 <					· ·
Uranium 6.405-03 Vancium 8.36E-01 1.27E+ Zinc 2.03E-01 4.16E+01 5.95E+ 1,1,2-Trichloroethane 2.50E-03 1.27E+ 1,1-Dichloroethane 2.50E-03 1.27E+ 1,2-Trichloroethane 2.50E-03 1.27E+ 1,2-Dichloroethane 2.50E-03 1.27E+ 1,2-Dichloroethane 2.50E-03 1.27E+ 1,2-Dichloroethane 2.50E-03 1.27E+ Benzena 2.50E-03 1.27E+ Benzena 2.50E-03 2.50E-03 Chloroform 2.50E-03 2.50E-03 Chloroform 2.50E-03 2.50E-03 Polychlorinated biphenyl 1.00E-04 1.00E-04 Tetrachloroethene 1.62E-01 1.23E-03 Vinyl Chloride 9.19E-03 2.84E- Xylena 5.45E-03 2.84E- cis-1, 2-Dichloroethene 5.00E-03 2.84E- trans-1, 2-Dichloroethene 5.00E-03 2.84E- Alpha activity 1.57E+02 5.02E+ <td></td> <td></td> <td></td> <td></td> <td></td>					
Zinc 2.03E-01 4.16E+01 5.95E+ 1,1,2-Trichloroethane 2.50E-03 1,1-Dichloroethane 2.50E-03 1,2-Dichloroethane 2.50E-03 1,2-Dichloroethane 2.50E-03 1,2-Dichloroethane 5.62E-01 2.50E-03 Benzena 2.50E-03 2.50E-03 Bromodichloromethane 2.50E-03 2.50E-03 Carbon Tetrachloride 2.50E-03 2.50E-03 Chloroform 2.50E-03 2.50E-03 Polychlorinated biphenyl 1.00E-04 2.50E-03 Tetrachloroethene 2.50E-03 7richloroethene Vinyl Chloride 9.19E-03 2.84E-03 Xylene 5.45E-03 2.84E-03 cis-1,2-Dichloroethene 5.00E-03 2.84E-03 Xylene 5.45E-03 2.84E-03 cis-1,2-Dichloroethene 5.00E-03 2.84E-03 Xylene 5.00E-03 2.84E-03 Cis-1,2-Dichloroethene 5.00E-03 2.84E-03 Xylene 5.00E-03 2.84E-03 Cis-1,2-Dichloroethene 5.00E-03 1.72E4 Beta activity 1.57E+02 5.02E+01			6.405-03		
1,1,2-Trichloroethane 2.50E-03 1,1-Dichloroethane 2.50E-03 1,2-Dichloroethane 2.50E-03 1,2-Dichloroethane 5.62E-01 Benzene 5.62E-03 Bromodichloromethane 2.50E-03 Carbon Tetrachloride 2.50E-03 Carbon Tetrachloride 2.50E-03 Carbon Tetrachloride 2.50E-03 Carbon Tetrachloride 2.50E-03 Chloroform 2.50E-03 Polychlorinated biphenyl 1.00E-04 Tetrachloroethane 2.50E-03 Trichloroethane 1.62E-01 Vinyl Chloride 9.19E-03 Xylene 5.45E-03 Cis-1, 2-Dichloroethane 5.00E-03 trans-1, 2-Dichloroethane 5.00E-03 trans-1, 2-Dichloroethane 5.00E-03 trans-1, 2-Dichloroethane 5.00E-03 trans-1, 2-Dichloroethane 5.00E-03 trans-1, 2-Dichloroethane 5.00E-03 trans-1, 2-Dichloroethane 5.00E-03 trans-1, 2-Dichloroethane 5.00E-03 trans-1, 2-Dichloroethane 5.00E-03 Beta activity 1.72E4 <td>dium</td> <td></td> <td></td> <td></td> <td>1.27E+01</td>	dium				1.27E+01
1,1-Dichloroethane 2.50E-03 1,2-Dichloroethane 2.50E-03 1,2-Dichloroethane 5.62E-01 Benzene 2.50E-03 Bromodichloromethane 2.50E-03 Carbon Tetrachloride 2.50E-03 Carbon Tetrachloride 2.50E-03 Carbon Tetrachloride 2.50E-03 Chloroform 2.50E-03 Polychlorinated biphenyl 1.00E-04 Tetrachloroethene 2.50E-03 Trichloroethene 1.62E-01 Vinyl Chloride 9.19E-03 Xylene 5.45E-03 cis-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 Beta activity 1.72E4				4.168+01	5.95B+01
1,2-Dichloroethane 2.50E-03 1,2-Dichloroethane 5.62E-01 Benzene 2.50E-03 Bromodichloromethane 2.50E-03 Carbon Tetrachloride 2.50E-03 Chloroform 2.50E-03 Chloroform 2.50E-03 Chloroform 2.50E-03 Sthylbenzene 2.50E-03 Polychlorinated biphemyl 1.00E-04 Tetrachloroethene 2.50E-03 Vinyl Chloride 9.19E-03 Xylene 5.45E-03 cis-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 Beta activity 1.57E+02 5.02E+01		•		•	
1,2-Dichloroethene 5.62E-01 Benzene 2.50E-03 Bromodichloromethane 2.50E-03 Carbon Tetrachloride 2.50E-03 Carbon Tetrachloride 2.50E-03 Chloroform 2.50E-03 Ethylbenzene 2.50E-03 Polychlorinated biphenyl 1.00E-04 Tetrachloroethene 2.50E-03 Trichloroethene 2.50E-03 Vinyl Chloride 9.19E-03 Xylene 5.45E-03 cis-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 Beta activity 1.57E+02 5.02E+01		•			
Benzena 2.50E-03 Bromodichloromethane 2.50E-03 Carbon Tetrachloride 2.50E-03 Chloroform 2.50E-03 Ethylbenzene 2.50E-03 Polychlorinated biphenyl 1.00E-04 Tetrachloroethene 2.50E-03 Trichloroethene 2.50E-03 Trichloroethene 2.50E-03 Trichloroethene 2.50E-03 Trichloroethene 2.50E-03 Trichloroethene 2.50E-03 Trichloroethene 9.19E-03 Zylene 5.45E-03 cis-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 Beta activity 1.57E+02 5.02E+01	Dichloroethane	5 628-01	2.304-03		
Bromedichloromethane2.50E-03Carbon Tetrachloride2.50E-03Chloroform2.50E-03Chloroform2.50E-03Ethylbenzene2.50E-03Polychlorinated biphenyl1.008-04Tetrachlorosthene2.50E-03Trichlorosthene1.62E-01Vinyl Chloride9.19E-03Xylene5.45E-03cis-1,2-Dichlorosthene5.00E-03trans-1,2-Dichlorosthene5.00E-03Jpha activity2.40E+01Beta activity1.57E+02Sould activity5.02E+01		J.UAH-VL	2.50B-03	•	
Chloroform 2.50E-03 Sthylbenzene 2.50E-03 Polychlorinated biphenyl 1.00E-04 Tetrachloroethene 2.50E-03 Trichloroethene 1.62E-01 Vinyl Chloride 9.19E-03 Xylene 5.45E-03 cis-1,2-Dichloroethene 5.00E-03 trans-1,2-Dichloroethene 5.00E-03 Alpha activity 2.40E+01 1.72E4 Beta activity 1.57E+02 5.028+					
Ethylbanzena 2.50E-03 Polychlorinated biphenyl 1.008-04 Tetrachloroathene 2.50E-03 Trichloroathene 1.62E-01 Trichloroathene 9.19E-03 Xylene 5.45E-03 Cis-1, 2-Dichloroathene 5.00E-03 trans-1, 2-Dichloroathene 5.00E-03 Alpha activity 2.405±01 1.72E4 Bata activity 1.57E+02 5.02E					
Scipitorinated biphenyl1.00E-04Tetrachloroethene2.50E-03Trichloroethene1.62E-01Trichloroethene9.19E-03Vinyl Chloride5.45E-03Zylene5.45E-03cis-1,2-Dichloroethene5.00E-03trans-1,2-Dichloroethene5.00E-03Alpha activity1.57E+02Beta activity1.57E+02		•			
Tetrachlorostheme 2.50E-03 Trichlorostheme 1.62E-01 Trichlorostheme 9.19E-03 Vinyl Chlorida 9.19E-03 Xyleme 5.45E-03 cis-1,2-Dichlorostheme 5.00E-03 trans-1,2-Dichlorostheme 5.00E-03 Alpha activity 2.40E+01 1.72E+ Beta activity 1.57E+02 5.02E+					
Trichloroethene 1.62E-01 1.23E-03 Vinyl Chloride 9.198-03 2.84E- Xylene 5.45E-03 2.84E- cis-1,2-Dichloroethene 5.00E-03 2.84E- trans-1,2-Dichloroethene 5.00E-03 1.72E+ Alpha activity 2.40E+01 1.72E+ Beta activity 1.57E+02 5.02E+	chlorinated biphenyl				
Vinyl Chloride 9.19E-03 2.84E- Xylene 5.45E-03 2.84E- cis-1,2-Dichloroethene 5.00E-03 2.40E+01 trans-1,2-Dichloroethene 2.40E+01 1.72E+ Beta activity 1.57E+02 5.02E+					•
Xylene 5.45E-03 2.84E- cis-1,2-Dichloroethene 5.00E-03 1.72E+ trans-1,2-Dichloroethene 5.00E+03 1.72E+ Alpha activity 1.57E+02 5.02E+01		1.645-01			
A yields 5.00E-03 cis-1, 2-Dichloroethene 5.00E-03 trans-1, 2-Dichloroethene 5.00E-03 Alpha activity 2.40E+01 1.72E+ Beta activity 1.57E+02 5.02E+	1 Chioride			,	2.848-03
trans-1,2-Dichloroethene 5.00E-03 1.72E Alpha activity 2.40E+01 1.72E Beta activity 1.57E+02 5.02E	1.2-Dichloroethene				
Alpha activity 2.40E+01 1.72E+ Beta activity 1.57E+02 5.02E+			5.00B-03		· · · ·
Beta activity 1.57E+02 5.02E+	a activity		2.405+01	· · · · · · · · · · · · · · · · · · ·	1.72E+00
Radon-222 1.578+02	activity				5.028+00
			1.578+02		
					- 1
	•				
LOCATION#SKMU 194		LC	CATION=SWMU 194		
		RGA Groundwater	McNairy Groundvater		Subsurface soil (mg/kg or pCi/g

* Smaller of maximum detect and UCL95

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	LC	CATION-SWMU 194 (continued)		
Analyte	RGA Groundwater (mg/L or pCi/L)	McNairy Groundwater (mg/L or pCi/L)	Surface soil (mg/kg or pCi/g)	Subsurface soil (mg/kg or pCi/g)
Beryllium				1.285+00
Cadmium				2.275+00
Chromium				1.058+01
Lead			•	1.58E+01
Zinc				5.09E+01
Ethylbenzene Alpha activity				3.22E-03
Beta activity				1.99E+00 5.22E+00
-				,-
	LC	CATION-SWMU 99A		
••	RGA Groundwater	McNairy Groundwater	Surface soil	Subsurface soil
Analyte	(mg/L or pCi/L)	(mg/L or pCi/L)	(mg/kg or pCi/g)	(mg/kg or pCi/g)
Aluminum	1.082+01			4.50E+03
Antimony				2.52E+00
Arsenic	5.512-03 3.438-01		2.08E+02	3,002+00
Barium Beryllium	1.04E-02		6.67E-01	1.168+02 3.058-01
Cadmium	1.044-04		0.074-01	8.308-01
Chromium	1.278-01	-	9.40E+00	8.415+00
Cobalt	9.448-02			
Copper	7.988-02			
Iron	2.328+01			
Lead	8.13B-02			1.81E+01
Lithium	7.44E-02			
Manganese	1.662+00	•		2.17E+02
Mercury	4.328-04	•.		
Nickal	1.53B-01 1.04B+01			_
Silica Sulfate	1.198+01	••		
Tetracco-sulfate(1-)	9.848+00			
Thallium				5,90E-01
Vanadium	3.678-01			
Zinc	2.258-01		1.13E+02	9.10E+01
1,1-Dichloroethene	1.79E-02	1.04E-02		6.00E-03
1,2,4-Trichlorobenzene	•			4.108-01
1,2-Dichlorobenzene		•		4.10E-01 4.10E-01
1,3-Dichlorobenzene 1,4-Dichlorobenzene				4.108-01
2,4,5-Trichlorophenol				5.358-01
2,4,6-Trichlorophenol				4.105-01
2,4-Dinitrotoluene				4.10E-01
2.6-Dinitrotoluene				4.10E-01
2-Chloronaphthalene	-			4,10E-01
2-Hexanone				5.892-03
2-Methyl-4,6-dinitrophenol				5.358-01 4.10E-01
2-Methylnaphthalene				5.35E-01
2-Nitroaniline	•		· ·	4.108-01
2-Nitrophenol 3,3'-Dichlorobenzidine			•	3.068-01
3,3'-Dichloropenzicine 3-Nitroaniline				5.358-01
4,4'-DDD				3.50B-02
4,4'-DDE				3.508-02
4,4'-DDT				3.50E-02
4-Bromophenyl phenyl ether				4.102-01
4-Chloro-3-methylphenol				4.108-01
4-Chlorophenyl phenyl ether			:•.	4.10B-01 5.35B-01
4-Nitroaniline			3.30E-01	5.35E-01 2.42E-01
Acenaphthene			3.342-UT	.
	•:			

Table 1.11. Representative concentrations* of COPCs in media

* Smaller of maximum detect and UCL95

Table 1.11. Representative concentrations* of COPCs in media

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		(continued)		
	RGA Groundwater	McNairy Groundwater	Surface soil	Subsurface soil
nalyte	(mg/L or pCi/L)	(mg/L or pCi/L)	(mg/kg or pCi/g)	(mg/kg or pCi/g
cenaphthylene			2.628-01	2.50E-01
ldrin	•			1.708-02
nthracene			5.93R-01	
enz (a) anthracene			7.96E-01	4.888-01
enzo (a) pyrene			4.898-01	5.87E-01
				6.378-01
enzo (b) fluoranthene			1.15E+00	7.70E-01
enzo (ghi) perylene			7.468-01	5.248-01
enzo (k) fluoranthene			5.72R-01	4.988-01
utyl benzyl phthalate				2.20E-01
arbon Tetrachloride		2.80E-03		
hrysene			1.31E+00	6.21E-01
i-n-butylphthalate		· .		4.108-01
i-n-octylphthalate				4.10E-01
ibenz (a, h) anthracene			2.51E-01	4.45E-01
ibenzofuran			1.23B-01	2.46B-01
ieldrin				3.50E-02
ndosulfan I				1.70E-02
ndosulfan II				3.508-02
ndosulfan Sulfate		•		
				3.50E-02
ndrin	•			3.508-02
ndrin Ketone				3.508-02
thylbenzene				4.44E-03
luoranthene			8.518-01	6.37E-01
luorene			2.19B-01	2.45E-01
eptachlor				1.70B-02
eptachlor Epoxide				1.705-02
exachlorobenzene				4.10E-01
lexachlorobutadiene				4.10E-01
exachlorocyclopentadiene				4.108-01
exachloroethane	•			4.108-01
ndenc (1,2,3-cd) pyrene			8.01E-01	5.348-01
ethaxychlor				1.708-01
-Nitroso-di-n-propylamine		•		4.108-01
-Nitrosodiphenylamine				4,10E-01
				4.10E-01
aphthalene			2.385-01	1.698-01
CB-1016			2.304-VT	1.23E-01
CB-1221				1.23E-01
CB-1232				
CB-1242				1.238-01
CB-1248				1.23E-01
CB-1254			9.608-02	1.408-01
CB-1260			1.878-01	1.758-01
entachlorophenol				5.358-01
henanthrene			9.92E-01	5.718-01
yrene			9.768-01	6.54E-01
waphene				3.508-01
richloroethene	6.762-01	4.358-01		
inyl Chloride				1.208-02
ylene				3.238-03
lpha-BHC				1.708-02
lpha-Chlordane				1.708-01
ata-BHC				1.708-02
La (2-Chloroethoxy) methane				. 4.10E-01
	•			4.108-01
is (2-Chloroethyl) ether				4.108-01
is (2-Chloroisopropyl) ether				2.412-01
is (2-Ethylhexyl) phthalate	9.56E-03			6.1LB-V4
is-1,2-Dichloroethene	7.04E-03	1,15E-01		4 449-03
is-1,3-Dichloropropene				4.448-03
elta-BHC				1.708-02
anna-BHC (Lindana)				1.702-02
amma-Chlordane				1.70E-01

* Smaller of maximum detect and UCL95

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Table 1.11. Representative concentrations* of COPCs in media

LOCATION-SWMU 99A -----

		(continued)		
Analyte	RGA Groundwater (mg/L or pCi/L)	McNairy Groundwater (mg/L or pCi/L)	Surface soil (mg/kg or pCi/g)	Subsurface soil (mg/kg or pCi/g)
trans-1,3-Dichloropropene				4.448-03
Alpha activity	7.14E+00	2.90E+00	3.64R+01	3.072+01
Beta activity	4.20E+01	3.50R+01	1,808+02	1.072+02
Cesium-137			1.06E+00	9.512-01
Neptunium-237	•.		1.288+01	1.07E+01
Radon-222	6.62E+02	•		
Technetium-99	4.50E+01		4.61E+02	3.175+02
Thorium-234			2.16E+01	1.952+01
Dranium-234			1.642+01	1.38E+01
Tranium-236 ·			5.17E+01	4.356+01

----- LOCATION-SWMU 998 -----

Analyte	RGA Groundwater (mg/L or pCi/L)	McNairy Groundwater (mg/L or pCi/L)	Surface soil (mg/kg or pCi/g)	Subsurface soil (mg/kg or pCi/g)
Aluminum				7.04E+03
 Arsenic		•		3.46E+00
Barium	4.91R-01			
Beryllium				7.892-01
Chromium	6.19B-02			1.06R+01
Iron	2.248+00			
Manganese	2.60R-01			
Silīca	9.17E+00			
Sulfate	2.56E+01			
Tetraoxo-sulfate (1-)	1.458+01			
Zinc	5.838-02			
Methylene Chloride	.•			6.00E-01
Trichloroethene	2.08E+00			-
Alpha activity	1.848+00	•		1.92E+01
Beta activity	1.69E+01	•		2.05R+01
Radon-222	4.128+02			

* Smaller of maximum detect and UCL95



Table 1.12. Reasonable maximum exposure assumptions and human intake factors for ingestion of water by a rural resident^a

Chemical Intake (mg /	$ka - day = \frac{C_w \times IR \times EF \times ED}{W}$
Chemical Intake (ing /	BW x AT

Radionuclide Intake (pCi) = $A_w \times IR \times EF \times ED$

Parameter	Units	Value used	References
Chemical concentration in water = C _w	mg/L	Chemical-specific	
Radiological activity = A _w	pCi/L	Chemical-specific	
Ingestion rate = IR	L⁄d	2 (adult) 1 (child)	[14]
Exposure frequency = EF	d/year	350	[14]
Exposure duration = ED	years	34 (adult) 6 (child)	[14]
Body weight = BW	kg	70 (adult) 14.5 (child)	[14]
Averaging time = AT	yr × day/yr	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]

Equations

* Equation from:[1]. * References: follow Table 1.38.

	. <u>1</u>	Endpoint		
Cohort	Chemical carcinogen ^a	 Chemical noncarcinogen ^a	Radionuclide carcinoger	
Adult	1.33×10^{-2}	2.74×10^{-2}	2.38×10^4	
Child	5.67 × 10 ⁻³	6.61 ×10 ⁻²	2.10×10^{3}	

*Chemical concentration in water (mg/L) times intake factor [L/(kg · day)] yields the default RME dose for the associated endpoint. ^b Radionuclide concentration in water (pCi/L) times the intake factor (L) yields the default RME dose.

Equation Absorbed Dose (mg/kg- day) = $\frac{C_w \times SA \times P_c \times CF \times ED \times EF \times ET}{BW \times AT}$			
Parameter	Units	Value used	References ^b
Concentration in water = C_w	mg/L	Chemical-specific	
Skinisurface area exposed ^e = SA	m ²	1.815 (adult) 0.72 (child)	[14]
Skin permeability constant = P _e	cm/hr	Chemical-specific	
Conversion factor = CF	(L-m)/(cm-m³)	1Ő	
Exposure duration = ED	years	34 (adult) 6 (child)	[14]
Exposure frequency = EF	baths/yr	350	[14]
Exposure time = ET	hrs/bath	0:2	[14]
Body weight = BW	kg	70 (adult) 14.5 (child)	[[]4]
Averaging time = AT	ут × day/ут	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]

Table 1.13. Reasonable maximum exposure assumptions and human intake factors for dermal contact with water while showering by a rural resident^a

Equation

^a Equation from [1]. ^b References follow Table 1.38.

* Entire surface area of body for both adult and child.

Notes:

Human intake factors for dermal contact with groundwater during showering by a rural resident

	· · · · · · · · · · · · · · · · · · ·	Endpoint	
Cohort	Chemical carcinogen*	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b
Aduit	2.42×10^{-2}	4.97×10^{-2}	Not applicable
Child	8:16 × 10 ⁻³	9.52×10^{-2}	Not applicable

* Chemical concentration in water (mg/L) times chemical "Pc" (cm/hr) times intake factor [(L • hr)/(cm • kg • day] yields default RME dose for associated endpoint.

^b Dermal absorbed dose is not applicable to radionuclides per guidance found in [1].

Equations Chemical Intake (mg/	Chemical Intake (mg/kg- day) = $\frac{C_{\text{those of }} \times IR_{\text{sir}} \times EF \times ED \times ET}{BW \times AT}$		
		R _{air} x EF x ED x IEF	
$C_{abower} (mg/m^3) = \frac{\left[\left(\frac{C_{amax}}{2}\right)t_1\right]}{t_1 + t_2}$	+ [Camax t 2]	$C_{amax} (mg/m^3) = \frac{C_{gw} x f x}{V}$	$\frac{x F_w x t_l}{a}$
Parameter	Units	Value used	References
Time-adjusted concentration in shower = C_{abower}	mg/m ³	Chemical-specific	Calculated
Indoor inhalation rate = IR _{str}	m³/hour	0.6	[14]
Exposure frequency = EF	day/year	350	[14]
Exposure duration = ED	years	34 (adult) 6 (child)	[14]
Exposure time = ET	hours/day	0.2	[14]
Body weight = BW	kg	70 (adult) 14.5 (child)	[14]
Averaging time = AT	yr × day/yr	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]
Activity in groundwater = Agw	pCi/Ĺ	Chemical-specific	
Inhalation exposure factor = IEF	(L-hr)/(m ³ -day)	0.2064 (tritium) 5.6 (radon) 0 (all other radionuclides)	[15] [15]
Maximum air concentration = C_{amax}	mg/m ³	Chemical-specific	Calculated
Time of shower = t_1	hour	0.1	[14]
Time after shower = t_2	hour	0.1	[14]
Concentration in groundwater = C_{gw}	mg/L	Chemical-specific	
Fraction volatilized = f	unitless	0.75	[14]
Water flow rate = $\mathbf{F}_{\mathbf{w}}$	L/h	890	[14]
Bathroom volume = V_a	m³	11	[14]

Table 1.14. Reasonable maximum exposure assumptions and human intake factors for inhalation of volatile organic compounds in water while showering by a rural resident^a

^a Equation from [1]. ^bReferences follow Table 1.38.

Human intake factors for inhalation of volatile organic compounds in water while showering by a rural resident

Endpoint			
Cohort	Chemical carcinogen ^a	Chemical noncarcinogen*	Radionuciide carcinogen ^b
Adult	3.63×10^{-3}	7.48 × 10 ⁻³	7.14×10^{3}
Child	3.10×10^{-3}	3.61×10^{-2}	1.26×10^{3}

* Chemical concentration in water (mg/L) times intake factor [L/(kg • day)] yields default RME dose for the associated endpoint.

^b Radionuclide concentration in water (pCi/L) times "IEF" [(L • hr/(m³ • day)] times intake factor [(m³ • day)/hr] yields default RME dose.

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Table 1.15. Reasonable maximum exposure assumptions and human intake factors for inhalation of volatile organic compounds in water during household use by a rural resident^a

Equations	2 Character X	IR x EF x ED:x ET	
Chemical Intake (n	$ng/m^3 - day) = \frac{C_{house} x}{2}$	BW x AT	
	$ntake(pCi) = A_{gw} \times IR_{a}$		
		HF x f	
Cho	$use (mg/m^3) = \frac{C_{gw} \times W}{HV \times ER}$	R x MC	
Parameter	Units	Value used	References ^b
Concentration in household air = Chouse	mg/m ³	Chemical-specific	Calculated
Indoor inhalation rate = IR _{str}	m³/hour	0.833	[14]
Exposure frequency = $\mathbf{E}\mathbf{F}$	day/year	350	[14]
Exposure duration = ED	years	34 (adult) 6 (child)	[14]
Exposure time = \mathbf{ET}	hours/day	24	[14]
Body weight = BW	kg	70 (adult) 14.5 (child)	[14]
Averaging time = AT	ут × day/yr	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]
Activity in groundwater = A_{gw}	pCi/L	Chemical-specific	
Inhalation exposure factor = IEF	(L-hr)(m ³ -day)	0.2802 (tritium) 7.6030 (radon) 0 (all other radionuclides)	[15]
Concentration in groundwater = C_{gw}	mg/L	Chemical-specific	
Water flow rate = WHF	L/day	890	[14]
Fraction volatilized = f	unitless	0.75	[14]
House volume = HV	m ³ /change	450	[14]
Exchange rate = ER	changes/day	10	[14]
Mixing coefficient = MC	unitless	0:5	[14]

*Equation from [1] and [14]. •References follow Table 1.38.

Human intake factors for inhalation of volatile organic compounds in water
during household use by a rural resident

		Endpoint	
Cohort	Chemical carcinogen ^a	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b
Adult	3.95 × 10 ⁻²	6.77 × 10 ⁻⁴	9.91×10^{3}
Child	3.36×10^{-2}	3.27×10^{-3}	1.75×10^{3}

* Chemical concentration in water (mg/L) times intake factor [m³/(kg • day)] yields default RME dose for associated endpoint.

^b Radionuclide concentration in water (pCi/L) times "IEF" [(L • hr)/(m³ • day)] times intake factor [(m³ • day)/hr] yields default RME dosė.

Table 1.16. Reasonable maximum exposure assumptions and human intake factors for incidental ingestion of soil by a rural resident^a

Equations

Chemical Intake (mg/kg-day) = $\frac{C \times CF \times EF \times FI \times ED \times IR \times AC}{BW \times AT}$

Parameter	Units	Value used	References
Chemical concentration in soil = C_s	mg/kg	Chemical-specific	
Radiological activity = A _s	pCi/g	Chemical-specific	
Conversion factor = CF	kg/mg	10-6	
Conversion factor = CF _{rad}	g/mg	10-3	
Exposure frequency = EF	days/yr	350	[14]
Fraction ingested = FI	unitless	I	[14]
Exposure duration = ED	years	34 (adult) 6 (child)	[14]
Ingestion rate of soil = IR	mg/d	100 (adult) 200 (child)	[14]
Area of contact ^c = AC	unitless	AS/AG	*****
Area of SWMU≔ AS	acres	SWMU-specific	
Area of garden = AG	acres	0.25	[26]
Body weight = BW	kg	70 (adult) 14.5 (child)	[14]
Averaging time = AT	(yr × day/yr)	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]

Radionuclide Intake (pCi) = $A_x \times CF_{rad} \times EF \times FI \times ED \times IR \times AC$

^b Equation from [1]. ^b References follow Table 1.38. ^c AC cannot be>1.

Human intake factors for incidental ingestion of soil by a rural resident

-		Endpoint	
Cohort	Chemical carcinogen'	Chemicai noncarcinogen ^a	Radionuclide carcinogen ^b
Adult	6.65 ×10 ⁻⁷	1.37 × 10 ⁻⁶	1.19×10^{3}
Child	1.13×10^{-6}	1.32×10^{-5}	4.20×10^{2}

* Chemical concentration in soil (mg/kg) times "AC" (unitless) times intake factor [kg/(kg • day)] yields default RME dose for associated endpoint.

^b Radionuclide concentration is soil (pCi/g) times "AC" (unitless) times intake factor (g) yields default RME dose.

Absorbed Dose (r	$ng/kg \cdot day) = \frac{C_{i} \times CF_{d} \times S_{d}}{C_{i} \times CF_{d} \times S_{d}}$	BW x AT	
Parameter	Units	Value used	References ^b
Concentration in soil = C_s	mg/kg	Chemical-specific	
Conversion factor = CF _d	(kg- cm²)/(mg-m²)	0.01	
Surface area ^c = SA	m²/day	0.350 (adult) 0.373 (child)	[14]
Adherence factor = AF	mg/cm ²	1	[14]
Absorption factor ^d = ABS	unitless	0:25 (volatile organic) 0.1 (semivolatile organic) 0:05 (inorganic)	[14]
Exposure frequency = EF	day/yr	350	[14]
Exposure duration = ED	years	34 (adult) 6 (child)	[14]
Body weight = BW	lkg.	70 (adult) I4.5 (child)	[14]
Averaging time = AT	ут × day/ут	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]

Table 1.17. Reasonable maximum exposure assumptions and human intake factors for dermal contact with soil by a rural resident^a

Equation

^a Equation from [[1]: ^b References follow Table: 1.38.

* Includes hands and arms for adults and arms, hands, feet, and legs for children.

^d Listed default factors used unless chemical-specific absorption factors are available. Chemical-specific absorption factors used are 0.03 for dioxins [16], [17], 0.06 for polychlorinated biphenyls [16], [17], 0.01 for cadmium [16], [17], and 0.25 for carbon disulfide [18].

~ •	·	Endpoint	
Cohort Chemical carcinogen ^a Chemical noncarcinogen ^a			Radionuclide carcinogen ^b
Adult	2.33×10^{-5}	4.79 × 10 ⁻⁵	Not applicable
Child	2.11×10 ⁻⁵	2.47 × 10 ⁻⁴	Not applicable

Human Intake factors for derm	al contact with soil by a rural resident

* Chemical concentration in soil (mg/kg) times chemical "ABS" (unitless) times intake factor [kg/(kg • day)] yields default RME dose for associated endpoint. ^b Dermal absorbed dose is not applicable to radionuclides per guidance found in [1].

Table 1.18. Reasonable maximum exposure assumptions and human intake factors for inhalation
of vapors and particulates emitted from soil by a rural resident ^a

Equations

Chemical Intake (mg/m³ - day) =
$$\frac{C_{s} \times EF \times ED \times ET \times CF\left(\frac{1}{VF} + \frac{1}{PEF}\right)}{AT}$$
Radionuclide Intake (pCi) = A, x EF x ED x ET x CF₁ x $\left(\frac{1}{VF} + \frac{1}{PEF}\right)$ x IR_{si}

Parameter	Units	Value used	References
Concentration in soil = C_s	mg/kg	Chemical-specific	
Activity in soil = A_s	pCi/g	Chemical-specific	**
Exposure frequency = EF	day/year	350	[14]
Exposure duration ≓ ED	years	34 (adult) 6 (child)	[14]
Exposure time = ET	hours/day	24	[14]
Conversion factor = CF	day/hour	1/24	
Conversion factor = CF_1	g/kg	10 ³	
Volatilization factor = VF	m ³ /kg	Chemical-specific	[19]
Particulate emission factor = PEF	m³/kg	3.21 × 10 ¹⁰	[19]
Total inhalation rate = IR _{air}	m³/hour	0.833 (radionuclides only)	[14]
Averaging time = AT	yr × day/yr	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]

^a Equation from [20]. ^b References follow Table 1.38.

Human intake factors for inhalation of vapors and particulates emitted from soil by a rural resident

.		Endpoint	
Cohort	Chemical carcinogen [*]	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b
Adult	4.66 × 10 ⁻¹	9.59 × 10 ⁻¹	2.38 × 10 ⁸
Child	8.22×10^{-2}	9.59 × 10 ⁻¹	4.20×10^{7}

* Chemical concentration in soil (mg/kg) times "1/VF + 1/PEF" [(m³/kg)¹] times intake factor [unitless)] yields default RME dose for associated endpoint. ^bRadionuclide activity in soil (pCi/g) times "1/VF + 1/PEF" [(m³/kg)⁻¹] times intake factor [(g • m³)/kg] yields default RME dose.

Table 1.19. Reasonable maximum exposure assumptions and human intake factors for external exposure to ionizing radiation from soil by a rural resident^a

Absorbed Dose (pCi- year/g) = $A_s \times ED \times EF \times (1 - S_e) \times T_e \times AC$

Parameter	Units	Value used	Referencesb
Activity in soil = A _s	pCi/g	Chemical-specific	
Exposure duration = ED	year	34 (adult) 6 (child)	[14]
Exposure frequency = EF	day/day	350/365	[14]
Gamma shielding factor = S_e	unitless	0.2	[20]
Gamma exposure time factor = T _e	hr/hr	24/24	[20]
Area of $contact^{c} = AC$	unitless	AS/AG	
Area of SWMU = AS	acres	SWMU-specific	
Area of garden = AG	acres	0.25	[26]

Equation

[#]Equation from [20]: ^b References follow Table 1.38.

• AC cannot be >1.

Human intake factors for external exposure to ionizing radiation from soil by a rural resident

6 1 1		Endpoint	
Cohort	Chemical carcinogen ^a	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b
Adult	Not applicable	Not applicable	2.61×10^{11}
Child	Not applicable	Not applicable	4.60 × 10 ⁰

Exposure route is not applicable to chemicals not emitting ionizing radiation.
 Radionuclide activity in soil (pCi/g) times "AC" (unitless) times intake factor (yr) yields default RME dose.

Table 1.20. Reasonable maximum exposure assumptions and human intake factors for ingestion of
homegrown vegetables by a rural resident ^a

Equations

Chemical Intake (mg/kg- day) = $\frac{C_v \times FI_v \times IR_v \times EF \times ED}{BW \times AT}$

Radionuclide Intake (pCi) = $A_x \times FI_x \times IR_x \times EF \times ED \times CF$

Parameter	Units	Value used	References ^b
Chemical concentration in vegetables = C_v	mg/kg	Chemical-specific	See Table 1.40
Radiological activity = A_v	pCi/g	Chemical-specific	See Table 1.40
Diet fraction = $\mathbf{FI}_{\mathbf{v}}$	unitless	0.4	[21]
Ingestion rate = $\mathbf{IR}_{\mathbf{v}}$	kg/d	0.130 (child 3–5) 0.1995 (adult 20–39)	[22]
Exposure frequency = EF	d/year	350	[14]
Exposure duration = ED	years	6 (child) 34 (adult)	[14]
Body weight = BW	kg	14.5 (child) 70 (adult)	[14]
Averaging time = AT	yr × day/yr	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]
Conversion factor = CF	g/kg	1000	

* Equation from [1]. These intake rates are for those people that eat vegetables and should not be combined with the intake rates for other media.

^b References follow Table 1.38.

		Endpoint	
Cohort	Chemical carcinogen [*]	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b
Adult	3.44 × 10 ⁻⁴	1.09 × 10 ⁻³	6.14×10^{5}
Teen	2.26×10^{-4}	1.32×10^{-3}	2.49×10^{5}
Child	2.95×10^{-4}	3.44×10^{-3}	1.09×10^{5}

	Human intake factors for	ngestion of home-grown	vegetables by a rural resident
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• Chemical concentration in vegetables (mg/kg) (see Table 42) times intake factor [kg/(kg • day)] yields default RME dose for associated endpoint.

^b Radionuclide activity in soil (pCi/g) (see Table 42) times intake factor (g) yields default RME dose.

. .

Parameter	Units	Value used	Referencesb
Chemical concentration in venison = C_d	mg/kg	Chemical-specific	See Table 1.40
Radiological activity in venison = A_d	pCi/g	Chemical-specific	See Table 1.40
Venison ingestion rate ^c = IR _d	kg/day	0.032 (adult) 0.032 (teen) 0.007 (child)	See footnote b
Conversion factor = CF	g/kg	1000	
Diet fraction = FI	unitless	1	[5]
Exposure frequency = EF	day/yr	350	See footnote b
Exposure duration = ED	years	22 (adult) 12 (teen) 6 (child)	[]] 4]
Body weight = BW	kg	70 (adult) 43 (teen) 14.5 (child)	[[14]
Averaging time = AT	yr x∶day/yr	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14];

Table 1.21. Reasonable maximum exposure assumptions and human intake factors for consumption of venison by a recreational user^a

Chemical Intake (mg/kg- day) = $\frac{C_d \times IR_d \times FI \times EF \times ED}{BW \times AT}$

Equation from [1].

Equations

^b References follow Table 1.38.

⁶ Based on two deer maximum per year in the state of Kentucky, 50% success rate (Kentucky Department of Fish and Wildlife, 1992). Deer Surveys. Project No: W-45-24.); dressed weight averaging 108:5 pounds per deer for Ballard and McCracken counties, 60% of venison recovered per deer, 2.5 persons per household in Ballard and McCracken counties, and a child consumption rate 20% of that for adults.

	Endpoint		
Cohort	Chemical carcinogen ^a	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b
Adult	1.38 × 10 ⁻⁴	4.38 × 10 ⁻⁴	2.46×10^{5}
Teen	1.22×10^{-4}	7.14 × 10 ⁻⁴	1.34 × 10 ⁵
Child	3.97 × 10 ⁻⁵	4.63×10^{-4}	1.47×10^{4}

Human intake factors for consumption of venison by a recreational user

*Chemical concentration in venison (mg/kg) (see Table 11.46) times human intake factor [kg/(kg.e.day)] yields default RME dose for associated endpoint.

^b Radionuclide concentration in venison (pCi/g) (see Table 1.46) times human intake factor (g) yields RMS dose.

Table 1.22. Reasonable maximum exposure assumptions and human intake factors for consumption of rabbit by a recreational user^a

Equations

Chemical Intake (mg/kg-day) = $\frac{C_r \times IR_r \times FI \times EF \times ED}{BW \times AT}$

Radionuclide Intake (pCi) = $A_r \times CF \times IR_r \times FI \times EF \times ED$

Parameter	Units	Value used	Referencesb
Chemical concentration in rabbit = C _r	mg/kg	Chemical-specific	See Table 1.47
Radiological activity in rabbit = A _r	pCi/g	Chemical-specific	See Table 1.47
Rabbit ingestion rate ^c = IR _r	kg/meal	0.0165 (adult) 0.0082 (teen) 0.0033 (child)	See footnote c
Conversion factor = CF	g/kg	1000	
Diet fraction = FI	unitless	1	[5]
Exposure frequency = EF	meals/yr	350	See footnote c
Exposure duration = ED	years	22 (adult) 12 (teen) 6 (child)	[14]
Body weight = BW	kg	70 (adult) 43 (teen) 14.5 (child)	[14]
Averaging time = AT	ут × day/ут	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]

 ⁴ Equation from [1].
 ^b References follow Table 1.38.
 ^c Based on 20 rabbits bagged per year at WKWMA, personal communication stating dressed weight equals 60% of average 1.2 kg rabbit. 2.5 persons per household in Ballard and McCracken counties, a child consumption rate 20% of that for adults, and a teen consumption rate 50% of that for adults.

Cohort		Endpoint	
	Chemical carcinogen"	Chemical:noncarcinogen*	Radionuciide carcinogen ^t
Adult	7.10 × 10 ⁻⁵	2.26 × 10 ⁻⁴	1.27×10^{5}
Teen	3.13 × 10 ⁻⁵	1.83 × 10 ⁻⁴	3.44×10^{4}
Chiid	1.87 × 10 ⁻⁵	2.18×10^{-4}	6.93×10^{3}

* Chemical concentration in rabbit (mg/kg) (see Table 1.47) times human intake factor [kg/(kg • day)] yields default RME dose for associated endpoint.

^b Radionuclide concentration in rabbit (pCi/g) (see Table 1.47) times human intake factor (g) yields RMS dose.

Equations Chemical Intake (mg/kg-day) = $\frac{C_q \times IR_q \times FI \times EF \times ED}{BW \times AT}$					
Cnemical Intak	e(mg/kg-day) =	BW x AT			
Radionuclide In	Radionuclide Intake (pCi) = A _q x CF x IR _q x FI x EF x ED				
Parameter	Units	Value used	References ^b		
Chemical concentration in quail = C_q	mg/kg	Chemical-specific	See Table 1.38		
Radiological activity in quail = A _q	pCi/g	Chemical-specific	See Table 1.38		
Quail ingestion rate ^c = IR_q	kg/meal	0.0047 (adult) 0.0024 (teen) 0.00094 (child)	See footnote c		
Conversion factor = CF	g/kg	1000			
Diet fraction = FI	unitless	1	[5]		
Exposure frequency = EF	meals/yr	350	See footnote c		
Exposure duration = ED	years	22 (adult) 12 (téen) 6 (child)	[14]		
Body weight = BW	kg	70 (adult) 43 (teen) 14.5 (child)	[14]		
Averaging time = AT	yr,× day/yr	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]		

Table 1.23. Reasonable maximum exposure assumptions and human intake factors for consumption of quail by a recreational user^a

^a Equation from [[1²]. ^b References follow Table 1.38.

⁶ Based on 20 quail bagged per year at WKWMA, personal communication stating dressed weight equals 75% of average 0.183 kg quail, 2.5 persons per household in Ballard and McCracken counties, a child consumption rate 20% of that for adults, and a teen consumption rate 50% of that for adults.

		Endpoint	
Cohort	Chemical carcinogen ^a	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b
Adult	2:02:× 10 ⁻⁵	6.44 × 10 ⁻⁵	3.62 ×∘10 ⁴
Teen	9.17 × 10 ⁻⁶	5.35 × 10 ⁻⁵	1.01×10^{4}
Child	5.33 × 10 ⁻⁶	6.22×10^{-5}	1.97×10^{3}

Human int	ake factors	for consum	ption of qua	ail by a recreational	user
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* Chemical concentration in quail (mg/kg) (see Table 1.38) times intake factor [kg/(kg • day)] yields default RME dose for associated endpoint. ^b Radionuclide concentration in quail (pCi/g) (see Table 1.38) times intake factor (g) yields default RME dose for associated endpoint.

Table 1.24. Reasonable maximum exposure assumptions and human intake factors for ingestion of water by an industrial worker^a

Equations

Chemical Intake (mg/kg- day) = $\frac{C_{*} \times IR_{*} \times EF \times ED}{BW \times AT}$

Radionuclide Intake (pCi) = $A_{w} \times IR_{w} \times EF \times ED$

Parameter	Units	Value used	References ^b
Concentration in groundwater = C_w	mg/L	Chemical-specific	
Activity in groundwater = A_w	pCi/L	Chemical-specific	
Ingestion rate = IR _w	L/day	1	[14]
Exposure frequency = EF	day/yr	250	[14]
Exposure duration = ED	year	25	[14]
Body weight = BW	kg	70	[14]
Averaging time = AT	yr × day/yr	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]

^a Equation from [1]. ^b References follow Table 3.38.

Human intake factors for ingestion of water by an industrial worker

		Endpoint	
Cohort	Chemical carcinogen ^a	Chemical noncarcinogen [®]	Radionuclide carcinogen ^b
Worker	3.49 × 10 ⁻³	9.78 × 10 ⁻³	6.25×10^{3}

* Chemical concentration in water (mg/L) times intake factor [L/(kg • day)] yields default RME dose for associated endpoint. ^b Radionuclide concentration in water (pCi/L) times intake factor (L) yields default RME dose.

Table 1.25.]	Reasonable maximum exposure assumptions and human intake factors for dermal
	contact with water while showering by an industrial worker ⁴

Equation	
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Absorbed Dose (Absorbed Dose (mg/kg- day) = $\frac{C_w \times P_c \times SA \times EF \times ED \times ET \times CF}{BW \times AT}$			
Parameter	Units	Value used	Referencesb	
Concentration in water = C _w	mg/L	Chemical-specific		
Skin permeability constant = P _e	cm/hr	Chemical-specific		
Skin surface area exposed ^c = SA	m²	1.815	[14]	
Exposure frequency = EF	showers/yr	250	[14]	
Exposure duration = ED	years	25	[14]	
Exposure time = ET	hrs/shower	0.2	[14]	
Conversion factor = CF	(L-m)/(cm-m ³)	10		
Body weight = BW	kg	70	[14]	
Averaging time = AT	yr × day/yr	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]	

^a Equation from [1]. ^b References follow Table 1.38. ^c Entire surface area of body.

Human intake factors for dermal contact with water while showering by an industrial worker				
Endpoint				

		Chapon	
Cohort	Chemical carcinogen [®]	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b
Worker	i.27 × 10 ⁻²	3.55 × 10 ⁻²	Not Applicable

* Chemical concentration (mg/L) times chemical "Pe" (cm/hr) times intake factor [(L • hr)/(cm • kg • day)] yields default RME dose for associated endpoint. ^b Dermal absorbed dose is not applicable to radionuclides per guidance found in [[1].

Equations Chemical Intake	$(mg/kg-day) = \frac{C_{shower} x}{2}$	IR # x EF x ED x ET BW x AT	
Radionuclide	Intake (pCi) = A _{sv} x IR ,	" x EF x ED x IEF	
$C_{\text{shower}} (\text{mg/m}^3) = \frac{ (C_{\text{amax}}/2)t_1 + t_1}{t_1 + t_2}$	$\frac{1}{2} \left[C_{amax} t_2 \right]$	$C_{amax} (mg/m^3) = \frac{C_{gw} x f x}{V_a}$	$F_w \times t_1$
Parameter	Units	Value used	References ^b
Concentration in shower = C_{shower}	mg/m ³	Chemical-specific	Calculated
Indoor inhalation rate = IR _{sir}	m³/hour	0.6	[14]
Exposure frequency = EF	day/year	250	[14]
Exposure duration = ED	years	25	[14]
Exposure time = ET	hours/day	0.2	[14]
Body weight = BW	kg	70	[14]
Averaging time = AT	yr × day∕yr	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]
Activity in groundwater = A _{gw}	pCi/L	Chemical-specific	
Inhalation exposure factor = IEF	(L-hr)/(m ³ -day)	0:206 (tritium) 5.6 (radon) 0.00 (other radionuclides)	[15]
Maximum concentration = C_{amax}	mg/m ³	Chemical-specific	
Time of shower = t_1	hours	0.1	[14]
Time after shower $= t_2$	hours	0.1	[14]
Concentration in groundwater = C _{gw}	mg/L	Chemical-specific	
Fraction volatilized = f	unitless	0.75	[14]
Water flow rate = $\mathbf{F}_{\mathbf{w}}$	L/h	890	[14]
Bathroom volume = V_a	m ³	11	.[(14]

Table 1.26. Reasonable maximum exposure assumptions and human intake factors for inhalation of volatile organic compounds in water while showering by an industrial worker^a

Equations

^a Equation after [1] and [14]. ^b References follow Table 1.38.

Human intake factors for inhalation of volatile organic compounds in water while showering by an industrial worker

		Endpoint	
Cohort	Chemical carcinogen*	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b
Worker	1.91 × 10 ⁻³	5.34 × 10 ⁻³	3.75×10^{3}

^a Chemical concentration in water (mg/L) times intake factor [L/(kg \bullet day)] yields default RME dose for the associated endpoint. ^b Radionuclide concentration in water (pCi/L) times "IEF" [(L \bullet hr)/(m³ \bullet day)] times intake factor [(m³ \bullet day)/hr] yields default RME dose.

Table 1.27. Reasonable maximum exposure assumptions and human intake factors for incidental ingestion of soil by an industrial worker^a

Equations

Chemical Intake (mg/kg-day) = $\frac{C_s \times IR_s \times FI \times EF \times ED \times AC \times CF}{BW \times AT}$

Radionuclide Intake (pCi) = $A_s x IR_s x FI x EF x ED x AC x CF_{rad}$

Parameter	Units	Value used	References ^b
Concentration in soil = C_s	mg/kg	Chemical-specific	
Activity in soil = A_s	pCi/g	Chemical-specific	
Ingestion rate = IR,	mg/day	50	[14]
Fraction ingested = FI	unitless	1	[14]
Exposure frequency = EF	day/yr	250	[14]
Exposure duration = ED	year	25	[14]
Area of contact ^c = AC	unitless	AS/AW	,
Area of SWMU = AS	acres	SWMU-specific	
Area worker ranges = AW	acres	0.5 (or size of site, whichever is less)	[28]
Conversion factor = CF	kg/mg	10.6	
Conversion factor = CF _{rad}	g/mg	10 ⁻³	
Body weight = BW	kg	70	[14]
Averaging time = AT	yr × day/yr	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]

⁶ Equation from [4]. ^b References follow Table 1.38. ^c "AC" cannot be > 1.

Human intake factors for incidental ingestion of soll by an industrial worker

		Endpoint	
Cohort	Chemical carcinogen*	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b
Worker	1.75 × 10 ⁻⁷	4.89 × 10 ⁻⁷	3.13×10^2

*Chemical concentration in soil (mg/kg) times "AC" (unitless) times intake factor [kg/(kg • day)] yields default RME dose for associated endpoint. "AC" cannot be > 1.

^b Radionuclide concentration (pCi/g) times "AC" (unitless) times intake factor (g) yields default RME dose. "AC" cannot be > 1.

Table 1.28.	Reasonable maximum exposure assumptions and human intake factors for dermal	
contact with soil by an industrial worker ^a		

Absorbed Dose (mg/kg- day) = $\frac{C_s \times CF_d \times SA \times AF \times ABS \times EF \times ED}{BW \times AT}$				
Parameter	Units	Value used	References	
Concentration in soil = C_s	mg/kg	Chemical-specific		
Conversion factor-dermal = CF _d	(kg-cm ²)/(mg- m ²)	0.01		
Surface area ^c = SA	m²/day	0.43	[1]4]	
Adherence factor = AF	mg/cm ²	1	[4]4]	
Absorption factor ^d = ABS	unitless	0.25 (volatile organic) 0.10 (semivolatile organic) 0.05 (inorganic)	[14]	
Exposure frequency = EF	day/yr	250	[14]	
Exposure duration = ED	years	25	[14]	
Body weight = $\mathbf{B}\mathbf{W}$	kg	70	[14]	
Averaging time = AT	ут × day/уг	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]	

Equation

^a Equation after [1]. ^b References follow Table 1.38. ^c Area of hands, arms, and head.

^d Listed default factors used unless chemical-specific absorption factors are available. Chemical-specific absorption factors used are 0.03 for dioxins [16] and [17], 0.06 for polychlorinated biphenyls [16] and [17], 0.01 for cadmium [16] and [17], and 0.25 for carbon disulfide [18].

Human intake factors for dermal contact with soil and sediment by an industrial worker

<u> </u>	Endpoint		
Cohort	Chemical carcinogen ⁴	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b
Worker	1.50×10 ⁻⁵	4.21 × 10 ⁻⁵	Not Applicable

* Chemical concentration is soil or sediment (mg/kg) times chemical "ABS" (unitless) times intake factor [kg/(kg • day)] yields default RME dose for associated endpoint.

^b Dermal absorbed dose is not applicable to radionuclides per guidance found in [1].

Table 1.29. Reasonable maximum exposure assumptions and human intake factors for inhalation of vapors and particulates emitted from soil by an industrial worker^a

Equations

Chemical Intake (mg/kg - day) =
$$\frac{C_s \times EF \times ED \times ET \times \left(\frac{1}{VF} + \frac{1}{PEF}\right)}{AT}$$

Radionuclide Intake (pCi) = $A_s \times CF_i \times EF \times ED \times ET \times \left(\frac{1}{V}\right)$	$\frac{1}{F} + \frac{1}{PEF}$	xIRair
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Parameter	Units	Value used	References
Concentration in soil = C_s	mg/kg	Chemical-specific	
Activity in soil or = A_s	pCi/g	Chemical-specific	
Conversion factor = CF	day/hr	1/24	
Conversion factor = CF_{i}	g/kg	103	+
Exposure frequency = EF	day/year	250	[14]
Exposure duration = ED	years	25	[14]
Exposure time = ET	hour/day	8	[14]
Volatilization factor = VF	m³/kg	Chemical-specific	[19]
Particulate emission factor = PEF	m³/kg	4.28 × 10 ⁹	[19]
Total inhalation rate = IR _{air}	m ³ /hour	2.5 (for radionuclides)	[14]
Averaging time = AT	yr × day/yr	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]

* Equation from [20].

^b References follow Table 1.38.

Human intake factors for inhalation of vapors and particulates emitted from soil by an industrial worker

		Endpoint	· · · · · · · · · · · · · · · · · · ·
Cohort	Chemical carcinogen ^a	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b
Worker	8.15 × 10 ⁻²	2.28×10^{-1}	1.25×10^{8}

Chemical concentration in soil (mg/kg): times "1/VF + 1/PEF" [(m³/kg)¹] times intake factor [unitless)] yields default RME dose for associated endpoint.

^b Radionuclide activity in soil (pCi/g) times "1/VF + 1/PEF" [(m³/kg)¹] times intake factor [(g • m³)/kg] yields default RME dose.

Table 1.30. Reasonable maximum exposure assumptions and human intake factors for external exposure to ionizing radiation from soil by an industrial worker^a

Parameter	Units	Value used	References
Activity in soil = A_s	pCi/g	Chemical-specific	*
Exposure frequency = $\mathbf{EF}_{\mathbf{x}}$	day/day	250/365	[14]
Exposure duration = ED	year	25	[14]
Gamma shielding factor = S _e	unitless	0.2	[20]
Gamma exposure time factor = T _e	hr/hr	8/24	[20]
Area of contact ^c = AC	unitless	AS/AW	
Area of SWMU = AS	acres	SWMU-specific	
Area worker ranges = AW	acres	0.5	[28]

Equation

* Equation after [20]. * References follow Table 1.38.

^c AC cannot be > 1.

Human intake factors for external exposure to ionizing radiation from soil by an industrial worker

<u> </u>		Endpoint	
Cohort	Chemical carcinogen ^a	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b
Worker	Not applicable	Not applicable	$4.57 \times 10^{\circ}$

* Exposure not applicable to chemicals not emitting ionizing radiation. *Radionuclide activity in soil (pCi/g) times "AC" (unitless) times intake factor (yr) yields default RME dose.

Table 1.31. Reasonable maximum exposure assumptions and human intake factors for incidental ingestion of soil by an excavation worker^a

Equations

Chemical Intake (mg/kg- day) = $\frac{C_s \times CF \times IR_s \times EF \times ED \times FI}{BW \times AT}$

Radionuclide Intake (pCi) = A_s , x CF_{rad} x IR_s x EF x ED x FI

Parameter	Units	Value used	References
Concentration in soil or sediment = C_s	mg/kg	Chemical-specific	·
Conversion factor = CF	kg/mg	10-6	. .
Activity in soil or sediment = A _s	pCi/g	Chemical-specific	
Conversion factor = CF _{rad}	g/mg	10-3	. =====
Ingestion rate = IR,	mg/day	480	[14]
Exposure frequency = EF	day/yr	185	[14]
Exposure duration = ED	year	25	[20]
$\mathbf{Fraction}$ ingested = \mathbf{FI}	unitless	1	[14]
Body weight = BW	kg	70	[14]
Averaging time = AT	yr × day/yr	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]

^a Equation after [1]. ^b References follow Table 1.38.

	Human intake factors for incidental ingestion of soil by an excavation worker			
.		Endpoint		
Cohort	Chemical carcinogen [®]	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b	
Excavation worker	1.24 × 10 ⁻⁶	3.48 × 10 ⁻⁶	2.22×10^{3}	

* Chemical concentration in soil (mg/kg) times intake factor [kg/(kg • day)] yields default RME dose for associated endpoint. * Radionuclide concentration in soil (pCi/g) times intake factor (g) yields default RME dose.

Equation	C, x CF a x S	A x AF x ABS x EF x ED BW x AT	
Absorbed Dose (mg/k	g- day) =		
Parameter	Units	Value used	References
Concentration in soil or sediment = C_s	mg/kg	Chemical-specific	
Conversion factor-dermal = CF _d	(kg-cm²)/(mg- m²)	0:01	
Surface area ^c = SA	m²/day	0.43	[14]
Adherence factor = AF	mg/cm ²	1:	[14]
Absorption factor ^d = ABS	unitless	0.25 (volatile organic) 0.10 (semivolatile organic) 0.05 (inorganic)	[14]
Exposure frequency = EF	day/yr	185	[14]
Exposure duration = ED	years	25	[20]
Body weight = BW	kg	70	[14]
Averaging:time = AT	ут × day/yr	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]

Table 1.32. Reasonable maximum exposure assumptions and human intake factors for dermal contact with soil by an excavation worker^a

^a Equation from [1]. ^b References follow Table 1.38.

⁶ Includes skin area of arms, hands, and head. ^a Listed default factors used unless chemical-specific absorption factors are available. Chemical-specific absorption factors used are 0.03 for dioxins [16] and [17], 0.06 for polychlorinated biphenyls [16] and [17], 0.01 for cadmium [16] and [17], and 0.25 for carbon disulfide [18].

Human intake factors for dermal contact with soil by an excavation worker
 Endpoint

.	Endpoint			
Cohort -	Chemical carcinogen ^a	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b	
Excavation worker	1.11.×10 ⁻⁵	3.11 × 10 ⁻⁵	Not applicable	

* Chemical concentration in soil (mg/kg) times chemical "ABS" (unitless) times intake factor [kg/(kg • day)] yields default RME dose for the associated endpoint.

^b Dermal absorbed dose is not applicable to radionuclides per guidance found in [1].

Table 1.33. Reasonable maximum exposure assumptions and human intake factors for inhalation of vapors and particulates emitted from soil by an excavation worker^a

Eq	uations	
- 1		

Chemical Intake (mg/ m ³ - day) =	$C_{s} x EF x ED x ET x CF x \left(\frac{1}{VF} + \frac{1}{PEF}\right)$
Chemical make(mg/m/-day)	AT

Radionuclide Intake (pCi) =
$$A_s \times CF_2 \times EF \times ED \times ET \times \left(\frac{1}{VF} + \frac{1}{PEF}\right) \times IR_{air}$$

Parameter	Units	Value used	References ^t
Concentration in soil or sediment = C_s	mg/kg	Chemical-specific	÷
Activity in soil or sediment = A_s	pCi/g	Chemical-specific	
Conversion factor = CF	day/hour	1/24	
Conversion factor = CF_2	g/kg	10 ³	
Exposure frequency = EF	day/yr	185	[14]
Exposure duration = $\mathbf{E}\mathbf{D}$	years	25	[20]
Exposure time = ET	hours/day	8	[ii4]
Volatilization factor = VF	m ³ /kg	Chemical-specific	[19]
Particulate emission factor = PEF	m³/kg	3.21×10^{10}	[19]
Inhalation rate = IR _{air}	m³/hour	2.5 (for radionuclides)	[14]
Averaging time = AT	yr × day/yr	70 × 365 (carcinogen) ED × 365 (noncarcinogen)	[14]

* Equation from [20]. * References follow Table 1.38:

Human intake factors for inhalation of volatile organic compounds and particulates emitted from soll by an excavation worker

		Endpoint	
Cohort	Chemical carcinogen ^a	Chemical noncarcinogen ^a	Radionuclide carcinogen ^b
Excavation worker	6.03×10^{-2}	1.695 × 10 ⁻¹	9.25 × 10 ⁷

* Chemical concentration in soil (mg/kg) times "1/VF + 1/PEF" [(m³/kg)¹] times intake factor [m³/(kg • day)] yields default RME dose for associated endpoint.

¹⁶ Radionuclide activity in soil (pCi/g) times "1/VF + 1/PEF" [(m³/kg)¹] times intake factor [(g • m³)/kg] yields default RME dose.

Table 1.34. Reasonable maximum exposure assumptions and human intake factors for external exposure to ionizing radiation from soil by an excavation worker^a

Equation Absorbed Dose (pCi- year/g) = $A_s \times EF \times ED \times (1-S_e) \times T_e$				
Parameter	Units	Value used	References ^b	
Activity in soil or sediment = A _s	pCi/g	Chemical-specific		
Exposure frequency = EF	day/day	185/365	[14], [20]	
Exposure duration = ED	year	25	[20]	
Gamma shielding factor = S _e	unitless	0.2	[20]	
Gamma exposure time factor = T _e	hr/hr	8/24	[20]	

Equation from [20].
 References follow Table 0.38.

Human intake factors for external exposure to ionizing radiation from soil by an excavation worker

	Endpoint		
Cohort -	Chemical carcinogen*	Chemical noncarcinogen*	Radionuclide carcinogen ^b
Excavation	Nint and Each la	Nict condicable	2.28 - 109
worker	Not applicable	Not applicable	3.38 × 10°

* Exposure not applicable to chemicals not emitting ionizing radiation.

^b Radionuclide concentration in soil (pCi/g) times intake factor (yr) yields default RME dose.

Table 1.35. Reasonable maximum exposure assumptions for concentration or activity of COPCs
in homegrown vegetables ^a

Ir x F x Bywer x [1-exp(-) = x +)]	$Ir \mathbf{\hat{x}} \mathbf{F} \mathbf{x} \mathbf{I}_{\mathbf{f}} \mathbf{x} \mathbf{T} \mathbf{x} \mathbf{[} \mathbf{i} \mathbf{l} - exp(-$	λ _E ×to)]	
$Irr_{rup} = \frac{Ir \times F \times Bv_{wet} \times [1 - exp(-\lambda_B \times t_b]}{P \times \lambda_B}$	$\frac{\mathbf{I}}{\mathbf{I}_{\mathrm{rrdep}}} = \frac{\mathbf{I}_{\mathrm{r}} \times \mathbf{F} \times \mathbf{I}_{\mathrm{f}} \times \mathbf{T} \times [[\mathbf{I} - \exp(-\lambda_{\mathrm{E}} \times \mathbf{t}_{\mathrm{v}})]]}{\mathbf{Y}_{\mathrm{v}} \times \lambda_{\mathrm{E}}}$			
Intra =	Ir x F x MLF x [1-e	xp(- λ _B x t _b)]		
111 res	Ρ×λ _Β	l,		
Parameter	Units	Value used	Referencesb	
Concentration in vegetable = C,	mg/kg or pCi/g	Chemical-specific	Calculated	
Concentration in groundwater = C_w	mg/L or pCi/L	Chemical-specific	 .	
Root uptake from irrigation = Irr _{rup}	L/kg	Chemical-specific	Calculated	
Conversion factor for radionuclides = \mathbf{CF}_{rad}	kg/g	10-3		
Concentration in soil = C_{\bullet}	mg/kg or pCi/g	Chemical-specific		
Area of contact ^c = AC	unitless	AS/AG		
Area of SWMU = AS	acres	SWMU-specific		
Area of garden = \mathbf{AG}	acres	0.25	[26]	
Wet root uptake for leafy vegetables = \mathbf{R}_{upv}	kg/kg	Chemical-specific	÷-	
Resuspension from irrigation = Irr _{res}	Ľ/kg	Chemical-specific	Calculated	
Resuspension multiplier = \mathbf{R}_{es}	unitless	0.26	[9]	
Aerial deposition from irrigation = Irr _{dep}	l⊥/kg	Chemical-specific	Calculated	
Irrigation rate = Ir	L/m ² -day	3.62	[10]	
Irrigation period = F	unitless	0.25	[10]; 3 months a year	
Soil to plant uptake, wet weight = \mathbf{Bv}_{wet}	kg/kg	Chemical-specific or 7.7 × K _{ow} - ^{0.58}	[1 1]	
Effective rate for removal = λ_B	l/day	$\lambda_i + \lambda_{HL}$	[11]	
$Decay = \lambda_1$	1/day	0.693/T _r	[1 1]	
$Half-life = T_r$	day	Chemical-specific		
Soil leaching rate = λ_{HL}	1/day	2.7×10 ⁻⁵	[1 1]	
Long-term deposition and build-up = t_b	day	10,950	[2]	
Area density for root zone = P	kg/m ²	240	[8], [12], [13]	
Plant mass leading factor = MLF	unitless	0.26	[9]	
Interception fraction = $\mathbf{I}_{\mathbf{f}}$	unitless	0.42	[7]	
Translocation factor = T	unitless	1	[2]	
Decay for removal on produce = λ_E	1/day	λ _i /# (0.693/t _w)	[14]	
Weathering half-life = t _w	day	14	[2]	
Above ground exposure time = t_v .	day	60	[2]	
Plant yield (wet) = Y.	kg/m²	2	[2]	

* Equations after [1], [2], [3] and [4], * References follow Table 4.38. * AC cannot be greater than 4.

Table 1.36. Reasonable maximum exposure assumptions for concentration or activity of COPCs in deer^a

Equations

$C_{d} = F_{d} x \left[(C_{forage} x AC x f_{s} x Q_{f}) + (C_{s} x AC x Q_{s}) + (C_{sw} x CF_{rad} x Q_{sw}) \right]$

 $C_{\text{forage}} = (C_s \times R_{upp}) + (C_s \times R_{es})$

Parameter	Units	Value used	References ^b
Chemical concentration in deer = C_d	mg/kg or pCi/g	Chemical-specific	Calculated
Forage-deer transfer factor = F_d	day/kg	Chemical-specific	
Chemical concentration in forage = C_{forage}	mg/kg or pCi/g	Chemical-specific	Calculated
Area of contact ^c = AC	unitless	AS/AD	
Area of SWMU = AS	acres	SWMU-specific	. <u></u>
Area of deer range = AD	acres	494	[27]
Fraction of deer's food from site when on site = f_s	unitless	1.0	[5]
Quantity of forage ingested daily by deer = Q_f	kg/day	1.74	[7]
Chemical concentration in soil or sediment = C_s	mg/kg or pCi/g	Chemical-specific	
Quantity of soil ingested daily by deer = Q_s	kg/day	0:034	[6]; 2% of forage
Contaminant concentration in surface water = C_{sw}	mg/L or pCi/L	Chemical-specific	
Conversion factor for radionuclides = CF _{rad}	kg/g	10-3	
Quantity of surface water ingested daily by deer = Q_{sw}	L/day	3.61	[8]
Soil to plant uptake (dry) = R _{upp}	unitless	Chemical-specific or 38 × K _{ow} - ^{0.58}	[8]
Soil resuspension multiplier = \mathbf{R}_{es}	unitless	0.25	[3]

⁴ Equations after [1], [2], [3] and [4]. ^b All references follow Table 1.38. ^c AC cannot be > 1.

Table 1.37. Reasonable maximum exposure assumptions for concentration or activity of COPCs in rabbits^a

Equations

$C_r = F_r \times [(C_{forage} \times AR \times f_s \times Q_f) + (C_s \times AR \times Q_s) + (C_{sw} \times CF_{rad} \times Q_{sw})]$

Parameter	Units	Value used	References ^b
Chemical concentration in rabbit = \mathbb{C}_r	mg/kg or pCi/g	Chemical-specific	Calculated
Forage-rabbit transfer factor = $\mathbf{F}_{\mathbf{r}}$	day/kg	Chemical-specific	
Chemical concentration in forage = C_{forage}	mg/kg or pCi/g	Chemical-specific	Calculated
Area of contact ^c = AC	unitless	AS/AD	-
Area of $SWMU = AS$	acres	SWMU-specific	
Area of rabbit range = AR	acres	7.7	[23]
Fraction of rabbit food from site when on site = f_s	unitless	1.0	
Quantity of forage ingested daily by rabbit = Q_f	kg/day	0.237	[24]
Chemical concentration in soil or sediment = C_s	mg/kg or pCi/g	Chemical-specific	
Quantity of soil ingested daily by rabbit = Q_s	kg/day	0.0149	[24]; 6.3% of forage
Contaminant concentration in surface water = c_{sw}	mg/L or pCi/L	Chemical-specific	
Conversion factor for radionuclides = CF _{rad}	kg/g	10-3	
Quantity of surface water ingested daily by rabbit = Q_{sw} .	L/day	0,116	[24]
Soil to plant uptake (dry) = R _{upp}	unitless	Chemical-specific or 38 × K _{ow} - ^{0.58}	[8]
Soil resuspension multiplier = R _{es}	unitless	0.25	[3]

 $C_{\text{forage}} = (C_s \times R_{upp}) + (C_s \times R_{es})$

Equations after [1], [2], [3] and [4].
 All References follow Table 1.38.
 AC cannot be > 1.

Table 1.38. Reasonable maximum exposure assumptions for concentration or activity of COPCs in quail^a

Equations

$C_{q_i} = F_{q_i} x \left[(C_f x A Q x f_s x Q_f) + (C_s x A Q x Q_s) + (C_{sw} x C F_{rad} x Q_{sw}) + (C_i x A Q x Q_i) \right]$

 $C_{\text{forage}} = (C_s \times R_{upp}) + (C_s \times R_{es})$

 $C_i = C_s \times BAF_i$

Parameter	Units	Value used	References ^b
Chemical concentration in quail = C_q	mg/kg or pCi/g	Chemical-specific	Calculated
Forage-quail transfer factor = F _d	day/kg	Chemical-specific	
Chemical concentration in forage = C _{forage}	mg/kg or pCi/g	Chemical-specific	Calculated
Area of contact ^c = AC	unitless	AS/AQ	
Area of SWMU = AS	acres	SWMU-specific	
Area of quail range = AQ	acres	25.5	[23]
Fraction of quails food from site when on site = f_s	unitless	1.0	
Quantity of forage ingested daily by quail = Q_f	kg/day	0.01499	[23]; 88.2% of total food
Chemical concentration in invertebrates = Ci	mg/kg∶or pCi/g	Chemical-specific	
Quantity of invertebrates ingested daily by quail = Q_1	kg/day	0,002006	[23]; 11.8% of total food
Chemical concentration in soil or sediment = C,	mg/day or pCi/L	Chemical-specific	
Quantity of soil ingested daily by quail = Q_s	kg/day	0.00158	[25]; 9.3% of total food (same as turkey)
Contaminant concentration in surface water = C _{sw}	mg/L or pCi/L	Chemical-specific	
Conversion factor for radionuclides = CF _{rad}	kg/g	10-3	
Quantity of surface water ingested daily by deer = Q_{sw}	L/day	3.61	[8]
Soil to plant uptake (dry) = \mathbf{R}_{upp}	unitless	Chemical-specific or 38 × K _{ow} - ^{0.58}	[8]
Soil resuspension multiplier = Res	unitless	0.25	[3]

^a Equations after [1], [2], [3] and [4]. ^b All References follow Table 1.38. ^c AC cannot be > 1.

REFERENCES FOR EXPOSURE EQUATIONS

- [1] United States Environmental Protection Agency (EPA). 1989. Risk Assessment Guidance for Superfund: Volume I Human Health Evaluation Manual. EPA/540/1-89/002.
- [2] National Council on Radiation Protection and Measurements (NCRP). 1984. Radiological Assessment: Predicting the Transport, Bioaccumulation, and Uptake by Man of Radionuclides Released to the Environment. NCRP Report No. 76.
- [3] Hinton, T. G. 1992. Contamination of plants by resuspension: a review, with critique of measurement methods. *Sci. Total Environ.* 121:177–193.
- [4] Whelan, G., D. L. Strenge, J. G. Droppo, Jr., B. L. Steelman, and J. W. Buck. 1987. The Remedial Action Priority System (RAPS): Mathematical Formulations. DOE/RL/87-09.
 Pacific Northwest Laboratory, Richland, WA.
- [5] Maximum Value used; equivalent to 100%
- [6] Beyer, W. N., E. E. Connor, and S. Gerould. 1994. Estimates of soil ingestion by wildlife. J. Wildl. Manage. 58:375-382.
- [7] Miller, C. W. 1980. An analysis of measured values for the fraction of a radioactive aerosol intercepted by vegetation. *Health Phys.* 38:705–712.
- [8] McKone, T. E. 1994. Uncertainty and variability in human exposure to soil contaminants through home-grown food: a Monte Carlo assessment. *Risk Anal.* 14:449–463.
- [9] Pinder, J. E., and K. W. McLeod. 1989. Mass loading of soil particles on plant surfaces. Health Phys. 57:935-942.
- [10] Personal communication with the Roane County, Tennessee, Extension Agent.
- [11] National Council on Radiation Protection Measurement (NCRP). 1989. Screening Techniques for Determining Compliance with Environmental Standards. Releases of Radionuclides to the Atmosphere. Bethesda, Maryland.
- Peterson, H. T., Jr. 1983. Terrestrial and aquatic food chain pathways. pp. 5-1 to 5-156 in J. E. Till and H. R. Meyer, eds., *Radiological Assessment: A Textbook on Environmental Dose Analysis*. United States Nuclear Regulatory Commission, Washington, D.C.
- [13] Hoffman, F. O., R. H. Gardner, and K. F. Eckerman. 1982. Variability in Dose Estimates Associated with the Food Chain Transport and Ingestion of Selected Radionuclides. NUREG/CR-2612. Oak Ridge National Laboratory, Oak Ridge, Tennessee.
- [14] The Natural Resources and Environmental Protection Cabinet, Department for Environmental Protection, Frankfurt, Kentucky. October 1995. *Risk Assessment Guidance*. 401 KAR 100:050 (Amended After Hearing).
- [15] McKone, T. E. 1987. Human Exposure to Volatile Organic Compounds in Household Tap Water: The Indoor Inhalation Pathway. *Environ. Sci. Technol.*, 21:1194–1201

- [16] United States Environmental Protection Agency (EPA). 1992. Dermal Exposure Assessment: Principles and Application. Interim Report. EPA/600/8-91/011B.
- [17] United States Environmental Protection Agency (EPA). 1992. Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual, Supplemental Guidance, Dermal Risk Assessment Interim Guidance. Office of Emergency and Remedial Response, U.S. Environmental Protection Agency, Washington, D.C. August 18, 1992.
- [18] Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Carbon Disulfide. 1992. ATSDR/TP-91/09.
- [19] United States Environmental Protection Agency (EPA). 1991. Appendix D of Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual (Part B, Development of Risk-based Preliminary Remediation Goals). OSWER Directive 9285.7-01B.
- [20] United States Environmental Protection Agency (EPA). 1991. Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual (Part B, Development of Risk-based Preliminary Remediation Goals). OSWER Directive 9285.7-01B.
- [21] United States Environmental Protection Agency (EPA). 1989. Exposure Factors Handbook. EPA/600/8-89/043.
- [22] USDA. (1987-1988) Dataset: Nationwide Food Consumption Survey 1987/88 Household Food Use. United States Department of Agriculture 1987/88 NFCS Database.
- [23] EPA. 1993. Wildlife Exposure Factors Handbook. Vol. I. Washington, D.C. EPA/600/R-93/187a.
- [24] DOE. 1995. Remedial Investigation/Feasibility Study for the Clinch River/Poplar Creek Operable Unit. DOE/OR/01-1393.
- [25] Beyer, W.N., E.E. Connor, and S. Gerald. 1994. "Estimates of soil ingestion by Wildlife." J. Wildl. Manage. 58:375-382.
- [26] Personal Communications with Ballard and McCracken Counties Agricultural Extension Agents reporting average garden size.
- [27] Marchington, R.L., and D.H. Hirth. 1984. Behavior. Chapter 6. Pages 129-168 in L.K. Halls, Ed. White-tailed deer: ecology and management. Stackpole Books, Harrisburg, P.A.
- [28] Neptune, D., E. P. Brantly, M. J. Messner, and D. I. Michael. 1990. Quantitative decision making in Superfund: A data quality objectives case study, Hazardous Materials Control 3(3):18-27, May/June.

Table 1.35. Miscellaneous	ractors used to	calculate chi	onic daily intakes	of COPCs
		Vegetable		Particulate
	Permeability	bio-uptake	Volatilization	emission
	constant	factor	factor	factor
Analyte	(cm/hr)	(kg/kg)	(m^3/kg)	(m^3/kg)
Aluminum	1.00E-03	1.00E-03		
Ammonia	1.50E-03	7.70E+00		3.21E+10
Antimony	1.00E-03	1.00E-02		3.21E+10
Arsenic	1.00E-03	1.00E-02		3.21E+10
Barium	1.00E-03	3.00E-03		3.21E+10
Beryllium	1.00E-03	2.50E-03		3.21E+10
Cadmium Coromium	1.00E-03 1.00E-03	1.38E-01 1.00 E -04		3.21E+10
Cobalt	1.00E-03	2.32E-02		3.21E+10 3.21E+10
Copper	1.00E-03	8.00E-02		JAAATIO
Fluoride	1.00E-03			
Iron	1.00E-03	4.00E-04	5.47E+01	3.21E+10
Lead Lithium	1.00E-03	7.60E-04 6.25E-03		3.21E+10
Manganese	1.00E-03 1.00E-03	6.88E-02		3.21E+10
Mercury	1.00E-03	3.00E-01		J.ZIATIU
Molybdenum	1.00E-03	8.00E-02	•	
Nickel	1.00E-03	5.00E-02		
Silica				
Silver	1.00E-03	2.16E-05		
Sulfate Tetracxo-sulfate (1-)	1.00E-03			
Thallium	1.00E-03	1.00E-03		3.21E+10
Uranium	1.00E-03	6.31E-04	•	
Vanadium	1.00E-03	1.38E-03		3.21E+10
Zinc	1.00E-03	2.64E-01		3.21E+10
1,1,2-Trichloroethane	8:40E-03	5.33E-01		
1,1-Dichloroethane 1,1-Dichloroethene	8.90E-03 8.90E-03	6.96E-01 6.96E-01	1.52E+03	3.21E+10
1,2,4-Trichlorobenzene	1.00E-01	- 3.69E-02	4.56E+04	3.21E+10
1,2-Dichlorobenzene	6.10E-02	8.21E-02	1.55E+04	3.21E+10
1,2-Dichloroethane	5.30E-03	1.04E+00		
1,2-Dichloroethene	1.07E-03	4.06E+00		
1,3-Dichlorobenzene 1,4-Dichlorobenzene	. 8.70E-02	6.29E-02 8.21E-02	! 1.37E+04	3.21E+10 3.21E+10
2,4,5-Trichlorophenol	6.20E-02 5.94E-02	4.81E-02	7.375+05	3.21E+10
2,4,6-Trichlorophenol	5.90E-02	5.50E-02	2.72E+05	3.21E+10
2,4-Dinitrotoluene	3.80E-03	5.33B-01	3.752+05	3.21E+10
2,6-Dinitrotoluene	2.50E-03	7.95E-01	3.058+05	3.21E+10
2-Chloronaphthalene	1.58E-01	3.228-02	2.978+04	3.21E+10 3.21E+10
2-Hexanone 2-Methyl-4,6-dinitrophenol	4.60E-03 9.75E-03	1.19E+00 2.09E-01	7.24E+05	3.21E+10
2-Mathylraphthalene	1.52E-01	4,218-02	4.51E+05	3.21E+10
2-Nitroaniline	5.19E-03	6.96E-01	1.25E+04	3.21E+10
2-Nitrophenol		· ·		3.21E+10
3,3'-Dichlorobenzidine	1.702-02	7.198-02	1.36E+06	3.21E+10
3-Nitroaniline	2.57E-03	1.24E+00 3.33E-03	2.46E+07	3.21E+10 3.21E+10
4., 4'-DDD 4., 4'-DDE	2.80E-01 2.40E-01	3.81E-03	2.63E+07	3.21E+10
4,4'-DDT	4.30E-01	1.58E-03	3.225+07	3,21E+10
4-Bromophenyl phenyl ether	2.40E-01	8.48E-03		3.21E+10
4-Chloro-3-methylphenol				3.21E+10
4-Chlorophenyl phenyl ether				3.21E+10
4-Nitroaniline	2.66E-03	1.20E+00 2.47E-02	2.31E+05	3.21E+10 3.21E+10
Acenaphthene	2.47E-01 9.55E-02	2.47E-02 . 5.50E-02	2.31E+05 3.14E+05	3.21E+10 3.21E+10
Acenaphthylene Acetona	5.69E-04	1.06E+01	* • * 20 TV#	
Aldrin	1.60E-03	1.40E-01	7.32E+06	3.21E+10
Anthracene	2,24E-01	2.16E-02	8.27E+05	3.21E+10
Benz (a) anthracene	8.10E-01	3,81E-03	1.00E+07	3.21E+10
Benzene	2.10E-02	4.66E-01	2 608.07	2 215.10
Benzo(a)pyrene	1.20E+00	2.23E-03	2.60E+07	3.21E+10

Table 1.39. Miscellaneous factors used to calculate chronic daily intakes of COPCs^a

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14 1		Vegetable		Particulate
	Permeability	bio-uptake	Volatilization	emission
·	constant	factor	factor	factor
Analyte	(cm/hr)	(kg/kg)	(m^3/kg)	(m^3/kg)
Benzo(b)fluoranthene	1.232+00	2.23E-03	4.90E+06	3.21E+10
Benzo(ghi)perylene	1.82E+00	1.14E-03	•.	3.21E+10
Benzo (k) fluoranthene	6.00E-01	8.76E-04	4.17E+07	3.21E+10
Bromodichloromethane	5.80E-03	4.66E-01	-	
Butyl benzyl phthalate	7.14E-02	1.11E-02	8.88E+06	3.21E+10
Carbon Tetrachloride	2.20E-02	1.83E-01		
Chloroform	8.90E-03	5.33E-01	0.007.00	
Chrysene Di - butulabbhalaba	8.102-01 1.152-01	3.81E-03 1.11E-02	2.888+06 8.55E+06	3.21E+10
Di-n-butylphthalate Di-n-octylphthalate	2.69E+01	3.55E-05	6.35E+07	3.21E+10
Dibenz (a, h) anthracene	2.70E+00	8.76E-04	1.11E+08	3.21E+10 3.21E+10
Dibenzofuran	9.338-01	3.83E-03	4.29E+05	3.21E+10
Dieldrin	1.60E-02	1.65E-02	2.28E+06	3.21E+10
Endosulfan I				3.21E+10
Endosulfan II				3.21E+10
Endosulfan Sulfate				3.21E+10
Endrin	1.60E-02	1.65E-02	2.388+06	3.21E+10
Endrin Ketone				3.21E+10
Ethylbenzene	7.40E-02	1.23E-01	5.71E+03	3.21E+10
Fluoranthene	3.60E-01	1.11E-02	3.24E+06	3.21E+10
Fluorene	2.46E-01	2.16E-02	5.41E+05	3.21E+10
Heptachlor	1.10E-02	2.47E-02	2.39E+06	3.21E+10
Heptachlor Epoxide	5.48E-02	5.68E-03	5.462+06	3.21E+10
Hexachlorobenzene	2.10E-01	6.49E-03	1.95E+05	3.21E+10
Rexachlorobutadiene	1.208-01	1.27E-02	7.628+04	3.21E+10
Hexachlorocyclopentadiene	2.86E-02	3.69E-02	1.51E+05 9.57E+04	3.21E+10
Hexachloroethane	4.20E-02 1.90E+00	4.21E-02 1.14E-03	6.05E+07	3.21E+10 3.21E+10
Indeno (1,2,3-cd) pyrene	1.97E-02	2.16E-02	4.31E+06	3.21E+10
Methoxychlor Methylene Chloride	4.50E-03	1.36E+00	2.64E+03	3.21E+10 3.21E+10
N-Nitroso-di-n-propylamine	2.80E-03	1.19E+00	1.17E+05	3.21E+10
N-Nitrosodiphenylamine	1.95E-02	1.23E-01	6.08E+05	3.21E+10
Naphthalene	6.90E-02	9.39E-02	5.91E+04	3.21E+10
PCB-1016	7.87B-01	2.91E-03	5.02E+05	3.21E+10
PCB-1221	9.22E-02	3.22E-02		3.21E+10
PCB-1232	1.37E-02	1.07E-01		3.21E+10
PCB-1242	3.65E-02	3.22E-02	3.838+05	3.21E+10
PCB-1248	3.70E-01	3.33E-03		3.21E+10
PCB-1254	3.46E-01	2.55E-03	5.62E+05	3.21E+10
PCB-1260	1.07E+00	5.878-04	4.74E+05	3.21E+10
Pentachlorophenol	6.50B-01	2.91E-03	1.25E+06	3.21E+10
Phenanthrene	2.70E-01	1.65E-02	1 007 07	3.21E+10
Polychlorinated biphenyl	3.46E-01	2.55E-03	1.00E+06 4.02E+06	3.21E+10 3.21E+10
Pyrene	3.24E-01	1.11E-02		3.21E+10 3.21E+10
Tetrachloroethene	3.70E-01	2.39E-01 1.27E-02	2.70E+03 1.24E+07	3.21E+10 3.21E+10
Toxaphene Trichloroethene	1.50E-02 1.60E-02	3.12E-01	3.45E+03	3.21E+10
	1.60E-02 7.30E-03	1.19E+00	1.10E+03	3.21E+10
Vinyl Chloride	9.47E-02	9.39E-02	7.23E+03	3.21E+10
Xylene alpha-BHC	1.88E-02	4.21E-02	5.988+05	3.21E+10
alpha-chlordane				3.21E+10
beta-BHC	2.228-02	3.69E-02	1.41E+06	3.21E+10
bis (2-Chloroethoxy) methane	1.40E-03	1.36E+00		3.21E+10
bis (2-Chloroethyl) ether	2,10E-03	1.36E+00	3.54E+04	3.21E+10
bis (2-Chloroisopropyl) ether	1.17E-02	2.46E-01	3.07E+04	3.21E+10
bis (2-Ethylhexyl) phthalate	2.34E-02	1.11E-02	2,29E+08	3.21E+10
cis-1,2-Dichloroethene	1.00E-02	6.09E-01		
cis-1,3-Dichloropropene			•	3.21E+10
delta-BHC	3.12E-03	1.83E-01		3.21E+10
gamma-BHC (Lindane)	1.40E-02	5.50E-02	4.94E+05	3.21E+10
gamma-Chlordane				3.21E+10
trans-1, 2-Dichloroethene	1.07E-03	4.06E+00		

Table 1.39. Miscellaneous factors used to calculate chronic daily intakes of COPCs





Analyte	Permeability constant (cm/hr)	Vegetable bio-uptake factor (kg/kg)	Volatilization factor (m^3/kg)	Particulate emission factor (m^3/kg)
trans-1,3-Dichloropropene				3.21E+10
Cesium-137		1.67E-02		3.21E+10
Neptunium-237		3.51E-03		3.21E+10
Radon-222		9.69E-03		
		2.08E+02		3.21E+10
Technetium-99		1.37E-04		3.21E+10
Thorium-234		6.31E-04		3.21E+10
Uranium-234 Uranium-238		2.02E-03		3.,21E+10

Table 1.39. Miscellaneous factors used to calculate chronic daily intakes of COPCs

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^a Source: Risk Assessment Information System, available online at http://tisk.lsd.ornl.gov/tox/nap_toxp.htm

------ LOCATION=AOC 204 Soil vegetable conc. RGA GW veg. conc. McNairy GW veg. conc. Soil rabbit conc. Soil quail conc. Soil deer conc. (mg/kg or pCi/g) (mg/kg or pCi/g) (mg/kg or pCi/g) (mg/kg or pCi/g) Analyte - 🦈 (mg/kg or pCi/g) (mg/kg or pCi/g) 1,1-Dichloroethane 1.89E+02 1,1-Dichloroethene 1.51E+00 PCB-1254 3.26E-01 3.24E-01 PCB-1260 Polychlorinated biphenyl 2.21E+00 Tetrachloroethene 1.38E+01 1.33E+01 Trichloroethene Vinyl Chloride 5.54E-03 cis-1,2-Dichloroethene 2.08E-01 ------ LOCATION-BWMU 193A ------Soil vegetable conc. RGA GW veg. conc. McNairy GW veg. conc. Soil rabbit conc. Soil quail conc. Soil deer conc. (mg/kg or pCi/g) (mg/kg or pCi/g) (mg/kg or pCi/g) (mg/kg or pCi/g) (mg/kg or pCi/g) (mg/kg or pCi/g) Analyte 8.65E+01 Ammonia 3.18E+00 9.218-03 4.57E-04 2.09E-03 Chromium 3.91E+02 1.71B+03 Iron 2.21E+00 Zinc 7.568-03 1,1-Dichloroethene 3.27E-02 7.24E-06 Anthracene 1.68E-06 4.75E-02 1.77E-04 3.99E-05 Benz(a) anthracene 6.56B-02 6.04E-04 1.65E-03 1.35E-04 Benzo (a) pyrene 1.23E-04 1.34E-02 2.76E-05 Benzo (b) fluoranthene 1.28E-03 4.44E-02 2.85E-04 Benzo (ghi) perylene 4.48E-02 1.67E-04 3.76E-05 Chrysene 1.33B-05 3.04E-06 2.09E-02 Di-n-butylphthalate 3.53B-01 7.86E-02 Di-n-octylphthalate 3.12E-02 Dibenz (a, h) anthracene 1.54E-03 3.39E-02 3.44E-04 4.72E-05 Fluoranthene 7.40E-02 1.08E-05 1.20E-03 Indeno (1,2,3-cd) pyrene 4.18E-02 2.69E-04 Pentachlorophenol 1.10E-01 8.00E-02 5.10B-05 1.16E-05 Pyrene 4.07E+00 8.978-02 Trichloroethene 1.72E-01 2.948-05 6.71E-06 bis (2-Ethylhexyl) phthalate 4.61E-02 1.01E-01 5.90E+00 cis-1.2-Dichloroethene 1.43E+03 2.74E+02 Technetium-99 1.72E-02 Uranium-238

Table 1.40. Representative concentrations and activities of COPCs in vegetables, deer, rabbit, and quail

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Analyte	Soil vegetable conc. (mg/kg or pCi/g)	RGA GW veg. conc. (mg/kg or pCi/g)	McNairy GW veg. conc. (mg/kg or pCi/g)	Soil rabbit conc. (mg/kg or pCi/g)	Soil quail conc. (mg/kg or pCi/g)	Soil deer conc. (mg/kg or pCi/g)
Beryllium	4.12E-01			6.71E-05	1.78E-06	6.65E-06
Chromium	2.31E+01			3.73E-02	8.19E-04	3.74E-03
Vanadium	1,70E+01		•	6.85E-03	1.52E-04	6.77E-04
1,1-Dichloroethene		6.12 B -02				
Ačetone		1,298+01				
Carbon Tetrachloride		1.078-01			-	
Di-n-butylphthalate		1.358-01				
Frichloroethene		1.208+01	3.13E-01			
bis (2-Ethylhexyl) phthalate		1.34B-01				
cis-1,2-Dichloroethene		2.85E-01	7.98E-01	•		
Technetium-99		2.04E+02				ڊ .
		roci	ATION-SWMU 193C		·	
Analyte	Soil vegetable conc. (mg/kg or pCi/g)	RGA GW veg. conc. (mg/kg or pCi/g)	McNairy GW veg. conc. (mg/kg or pCi/g)	Soil rabbit conc. (mg/kg or pCi/g)	Soil quail conc. (mg/kg or pCi/g)	Soil deer conc. (mg/kg or pCi/g)
Aluminum			4.96E+02			
Antimony			1.52E+00			
Arsenic			1.62E-01			
Barium			3.19E+00	,		
Beryllium			1.458-01			
Cadmium			6.31B-01			
Chromium	1.42E+00		1.85E+00	4.12E-03	3.00E-04	4.67E-03
Cobalt			7.328-01			
Iron	•		7.625+02			
Lead	6.50E+00		3.24E+00	9.52E±04	6.80E-05	1.10E-03
Manganese	•		2.10E+01			
Mercury			4.73E-03			
Molybdenum			7.29E-01			•
Nickel			7.918-01			
Silver			4.308-01			
Thallium			1,602+00			•
Uranium			B.30E-02			
Vanadium			1.092+01			
Zinc	2.18E+01		4.558+00	1.2BE+00		1.61E+00
1,1,2-Trichloroethane			7.998-02			+ · 0 + 0 + 0 0
1,1-Dichloroethene			9.45E-02			
1,2-Dichloroethane			1.25B-01			
		B.88E+01				
1,2-Dichloroethene		D.885+01				

Table 1.40. Representative concentrations and activities of COPCs in vegetables, deer, rabbit, and quail

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			(continued)			
Analyte	Soil vegetable conc. (mg/kg or pCi/g)	RGA GW veg. conc. (mg/kg or pCi/g)	McNairy GW veg. conc. (mg/kg or pCi/g)	Soil rabbit conc. (mg/kg or pCi/g)	Soil quail conc. (mg/kg or pCi/g)	Soil deer conc. (mg/kg or pCi/g
Bromodichloromethane			7.40E-02			
Carbon Tetrachloride			4.87E-02			
Chloroform		·	7.99E-02			
Ethylbenzene			4.33E-02			
Polychlorinated biphenyl			1.30E-03			
Tetrachloroethene		5	5.37B-02			
Trichloroethene		3.90E+00	2.97E-02			
Vinyl Chloride		·	5.09E-01			
Xylene			8.88E-02			
cis-1,2-Dichloroethene		-	1.73E=01			
trans-1, 2-Dichloroethene			7.90E-01			
Radon-222 '			1.30E-01			
		LÓC	ATION=SWMU 99A			
Analyté	Soil vegetable conc. (mg/kg or pCi/g)	RGA GW veg. conc. (mg/kg or pCi/g)	McNairy GW veg. conc. (mg/kg or pCi/g)	Soil rabbit conc. (mg/kg or pCi/g)	Soil quail conc. (mg/kg or pCi/g)	
Aluminum		1.40E+02				
Arsenic		7.32E-02				
Barium	5.47E+01	4.48E+00		1.27E-03	1.22E-03	1.30E-04
Beryllium	1.75E-01	1.358-01		1.598-05	4.23Ē-07	1.58E-06
Chromium	2.45E+00	1.64B+00		2.21E-03	4.85E-05	2.21E-04
Cobalt		1.30E+00				
Copper		1.26E+00				
Iron		3.00Ê+02		•		
Lead		1.05E+00				
Lithium		9.79E-01				
Manganese		2.56E+01				
Mercury		1.02E-02				
Nickel		2.26E+00				
Vanadium	÷	4.77E+00	•			· .
Zinc	5.92E+01	5.042+00		1.09E+00		1.20E-01
1,1-Dichloroethene		6.76E-01	3.93E-01			
Acenaphthene	9.39E-02			5.29E-06		5.45E-07
Acenaphthylene	8.25E-02			1.42E-06		1.50E-07
Anthracene	1.67E-01			1.15E-05		1.18E-06
Benz (a) anthracene	2.108-01			2.44E-04		2.43E-05
Benzo (a) pyrene	1.28E-01			3.68E-04 8.67E-04	4.46E-04	3.65E-05
Benzo(b)fluoranthene	3.02E-01					8.59E-05

Table 1.40. Representative concentrations and activities of COPCs in vegetables, deer, rabbit, and quail

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			(continued)			
Analyte	Soil vegetable conc. (mg/kg or pCi/g)	RGA GW veg. conc. (mg/kg or pC1/g)	McNairy GW veg. conc. (mg/kg or pCi/g)	Boil rabbit conc. (mg/kg or pCi/g)	Soil quail conc. (mg/kg or pCi/g)	Soil deer conc. (mg/kg or pCi/g)
Benzo (ghi) perylene	1.958-01			1.75E-03		1.73E-04
Benzo(k)fluoranthene Carbon Tetrachloride	1.49E-01	,	5.45E-02	2.128-03		2.09E-04
Chrysene	3.45E-01			4.02E-04		4-00E-05
Dibenz (a, h) anthracene	6.548-02			9.26E-04		9.158-05
Dibenzofuran	3.25E-02			3.748-05	•	3.72E-06
luoranthene	2.31E-01			4.59E-05		4.63E-06
Fluorene	6.178-02			4.268-06		4.37E-07
Indeno (1,2,3-cd) pyrene	2.098-01			1.87E-03		1.85E-04
PCB=1016	6.25B-02	•		1.148-04	4.36E-04	1.13E-05
PCB-1254	2.528-02			5.778-05	1.758-04	5-728-06
PCB-1254	4.888-02			1.378-03	3.36E-04	1.36E-04
Phenanthrene	2.74E-01			2.888-05	1.658-04	2.93E=06
Pyrene	2.65E-01			5.26E-05	1.038-04	5.31E-06
Frichloroethene	2.000.02	1.63E+01	1.05E+01	5.202.05		J.JIE-08
bis (2-Ethylhexyl) phthalate		1.27E-01	1.034401			
cis-1,2-Dichloroethene		2.44B-01	4.008+00			
Cesium-137	2.92B-01	2.335-01	4.005400	2.05E-03		
Neptunium-237	3.37E+00			2.05E-03 3.67E-04	1 007 05	2.15E-04
Radon-222	3.3/8+00	5.49E-01		3.675-04	1.02E-05	3.73E-05
Technetium-99	9.603+04	3.35E+02		2.60B-01		
	5.61E+00	3.356+02				2.97E-02
Thorium-234				5.16E=05	1.52E-06	5.11E-06
Dranium-234	4.27E+00			1.22E-04	9.04E-03	1.22E-05
Uranium-238	1.358+01			5.642-04	2.975-02	5.66E-05
		LOC	ATIONESWMU 998			
Analyte	Soil vegetable conc. (mg/kg or pCi/g)	RGA GW veg. conc. (mg/kg or pC1/g)	McNairy GW veg. conc. (mg/kg or pCi/g)	Soil rabbit conc. (mg/kg or pCi/g)	Soil quail conc. (mg/kg or pCi/g)	Soil deer conc. (mg/kg or pCi/g)
Barium		6.40E+00				
Chromium		8.01E-01				
Iron		2.908+01				
Manganese		4.00B+00				
Zinc		1.31E+00				
Trichloroethene		5.01B+01		-		
Radon-222		3.41E-01				

Table 1.40. Representative concentrations and activities of COPCs in vegetables, deer, rabbit, and quail

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Exposed populations	Exposure route, medium, and exposure point	Route quantified?	Reason for selection or dismissal
Current land us	Se		
Industrial			
	Ingestion of groundwater	No	Groundwater not in use
	Dermal contact with groundwater while showering	No	Groundwater not in use
	Inhalation of vapors while showering in groundwater	No	Groundwater not in use
	External exposure to ionizing radiation from groundwater while showering	No	Groundwater not in use
	Ingestion of soil	Yes	Soil may be ingested
	Dermal contact with soil	Yes	Soil may adhere to skin
	Inhalation of vapors and particulates emitted from soil	Yes	Vapors and particulates may be emitted from soil
	External exposure to ionizing radiation from soil	Yes	Radionuclides may be in soi
	Ingestion of waste	No	Waste not at site
	Dermal contact with waste	No	Waste not at site
	Inhalation of vapors and particulates emitted from waste	No	Waste not at site
	External exposure to ionizing radiation from waste	No	Waste not at site
	Ingestion of sediment	No	Sediment not at site
	Dermal contact with sediment	No	Sediment not at site
	Inhalation of particulates and vapors in sediment	No	Sediment not at site
	External exposure from creek sediment	No	Sediment not at site
	Ingestion of surface water	No	Surface water not at site
	Dermal contact with surface water	No	Surface water not at site
	Inhalation of vapors in surface water	No	Surface water not at site
	External exposure to ionizing radiation emitted from surface water	No	Surface water not at site

Table 1.41. Summary of reasons for selection or dismissal of exposure routes for quantitative evaluation for WAG 28 locations

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Exposed populations	Exposure route, medium, and exposure point	Route quantified?	Reason for selection or dismissal
Potential future	aland use		
Industrial			
	Ingestion of groundwater	Yes	Groundwater may be used in future
	Dermal contact with groundwater while showering	Yes	Groundwater may be used in future
	Inhalation of vapors while showering in groundwater	Yes	Groundwater may be used in future
	External exposure to ionizing radiation from groundwater while showering	No	Water is a natural radiation shield
	Ingestion of soil	Yes	Soil may be ingested
	Dermal contact with soil	Yes	Soil may adhere to skin
	Inhalation of vapors and particulates emitted from soil	Yes	Vapors and particulates may be emitted from soil
	External exposure to ionizing radiation from soil	Yes	Radionuclides may be in soi
	Ingestion of waste	No	Waste not at site
	Dermal contact with waste	No	Waste not at site
	Inhalation of vapors and particulates emitted from waste	No	Waste not at site
	External exposure to ionizing radiation from waste	No	Waste not at site
	Ingestion of sediment	No	Sediment not at site
	Dermal contact with sediment	No	Sediment not at site
	Inhalation of particulates and vapors in sediment	No	Sediment not at site
	External exposure from sediment	No	Sediment not at site
	Ingestion of surface water	No	Surface water not at site
	Dermal contact with surface water	No	Surface water not at site
	Inhalation of vapors emitted by surface water	No	Surface water not at site
	External exposure to ionizing radiation emitted from surface water	No	Surface water not at site

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Table 1.41. (Continued)

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Exposed populations	Exposure route, medium, and exposure point	Route quantified?	Reason for selection or dismissal
Potential future	land use (continued)		
Excavation			
	Ingestion of soil	Yes	Soil may be ingested
	Dermal contact with soil	Yes	Soil may adhere to skin
	Inhalation of vapors and particulates emitted from soil	Yes	Vapors and particles may be emitted from soil or waste
	External exposure to ionizing radiation in soil	Yes	Soil may contain radionculides
	Ingestion of sediment	No	Sediment not at site
	Dermal contact with sediment	No	Sediment not at site
	Inhalation of vapors and particulates emitted from sediment	No	Sediment not at site
	External exposure to ionizing radiation in sediment	No	Sediment not at site
	Ingestion of waste	No	Waste not at site
	Dermal contact with waste	No	Waste not at site
	Inhalation of vapors and particulates emitted from waste	No	Waste not at site
	External exposure to ionizing radiation in waste	No	Waste not at site

Exposed populations	Exposure route, medium, and exposure point	Route quantified?	Reason for selection or dismissal
Potential future	land use (continued)		
Recreational			
	Ingestion of soil	No	Repeated contact by recreational users with soil unlikely/exposure time limited
	Dermal contact with soil	No	Repeated contact by recreational users with soil unlikely/exposure time limited
	Inhalation of particles and vapors emitted from soil	No	Repeated contact by recreational users with soil unlikely/exposure time limited
	External exposure to ionizing radiation from soil	No	Repeated contact by recreational users with soil unlikely/exposure time limited
	Ingestion of waste	No	Waste not at site
	Dermal contact with waste	No	Waste not at site
	Inhalation of vapors and particulates emitted from waste	No	Waste not at site
	External exposure to ionizing radiation from waste	No	Waste not at site
	Ingestion of sediment	No	Sediment not at site
	Dermal contact with sediment	No	Sediment not at site
	Inhalation of particulates and vapors emitted from sediment	No	Sediment not at site
	External exposure to ionizing radiation from creek sediment	No	Sediment not at site
	Ingestion of surface water	No	Surface water not at site
	Dermal contact with surface water	No	Surface water not at site
	Inhalation of vapors emitted from surface water	No	Surface water not at site
	External exposure to ionizing radiation from creek surface water	No	Surface water not at site
	Ingestion of fish from creek surface water	No	Surface water not at site
	Ingestion of game	Yes	Deer, rabbit, and quail harvest is significant in the area

Exposed populations	Exposure route, medium, and exposure point	Route quantified?	Reason for selection or dismissal
Potential future	e land use (continued)		
Residential			
	Ingestion of groundwater	Yes	Groundwater may be used in future
	Dermal contact with groundwater while showering	Yes	Groundwater may be used in future
	Inhalation of vapors while showering in groundwater	Yes	Groundwater may be used in future
	Inhalation of vapors during household use of groundwater	Yes	Groundwater may be used in future
	External exposure to ionizing radiation from groundwater while showering	No	Water is natural radiation shield
	Ingestion of soil	Yes	Contaminated soil may be ingested on site
	Dermal contact with soil	Yes	Contaminated soil on site may adhere to skin
	Inhalation of vapors and particulates emitted from soil	Yes	Vapors and particulates may be emitted from soil on site
	External exposure to ionizing radiation from soil	Yes	Radionuclides may be in soi on site
	Ingestion of waste	No	Waste not at site
	Dermal contact with waste	No	Waste not at site
. •	Inhalation of vapors and particulates emitted from waste	No	Waste not at site
	External exposure to ionizing radiation from waste	No	Waste not at site
	Ingestion of sediment	No	Sediment not at site
	Dermal contact with sediment	No	Sediment not at site
	Inhalation of particulates and vapors in sediment	No	Sediment not at site
	External exposure from creek sediment	No	Sediment not at site
	Ingestion of surface water	No	Surface water not at site
	Dermal contact with surface water	No	Surface water not at site
	Inhalation of vapors in surface water	No	Surface water not at site
	External exposure to ionizing radiation emitted from surface water	No	Surface water not at site

Exposed populations	Exposure route, medium, and exposure point	Route quantified?	Reason for selection or dismissal
Potential future	and use (continued)		
Residential (cor	nt.)		
	Ingestion of vegetables	Yes	Site is large enough for garden
	Ingestion of beef and dairy products	No	Concentrations may change
	Ingestion of pork	No	Concentrations may change
	Ingestion of poultry and eggs	No	Concentrations may change
	Ingestion of fish raised in ponds filled with groundwater	No	Currently no such types of ponds present
	Dermal contact with groundwater while swimming in a pond	No	Currently no such types of ponds present
	Inhalation of vapors in groundwater while swimming in a pond	No	Currently no such types of ponds present
	Inhalation of vapors during irrigation with groundwater	No	Mixing volume too large
:	External exposure to ionizing radiation from groundwater while swimming in a pond filled with groundwater	No	Currently no such types of ponds present
	Ingestion of sediment while swimming in ponds filled with groundwater	No	Currently no such types of ponds present
	Dermal contact with sediment while swimming in a pond filled with groundwater	No	Currently no such types of ponds present
	Inhalation of vapors and particulates emitted from sediment in a pond filled with groundwater	No	Currently no such types of ponds present
	External exposure to ionizing radiation from sediment while swimming in a pond filled with groundwater	No	Currently no such types of ponds present

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----- LOCATION=SWMU 193A MEDIA=Surface Soil ------

Analyte	Ingestion	Dermal	Inhalation of volatiles and particulates
Chromium	5.99E-06	2.57E-05	8.70E-11
Anthracene	5.68E-08	4.88E-07	3.20E-08
Benz (a) anthracene	8.81E-08	7.57E-07	4.11E-09
Benzo (a) pyrene	1.22E-07	1.05E-06	2.20E-09
Benzo(b) fluoranthene	2.50E-08	2.15E-07	2.37E-09
Benzo (ghi) perylene	8.32E-08	7.15E-07	1.21E-12
Chrysene	8.32E-08	7.15E-07	1.35E-08
Di-n-butylphthalate	3.77E-08	3.24E-07	2.06E-09
Di-n-octylphthalate	5.87E-08	5.05E-07	4.33E-10
Dibenz (a, h) anthracene	6.36E-08	5.47E-07	2.69E-10
Fluoranthene	1.34E-07	1.15E-06	1.93E-08
Indeno (1,2,3-cd) pyrene	7.83E-08	6.73E-07	6.05E-10
Pyrene	1.44E-07	1.24E-06	1.67E-08
bis (2-Ethylhexyl) phthalate	8.32E-08	7.15E-07	1.71E-10

----- LOCATION-SWMU 193B MEDIA-Surface Soil ------

Analyte	Ingestion	Dermal contact	Inhalation of volatiles and particulates
Beryllium	7.68E-07	3.30E-06	1.12E-11
Chromium	4.34E-05	1.87E-04	6.31E-10
Vanadium	3.18E-05	1.37E-04	4.62E-10

--- LOCATION=SWMU 193C MEDIA=Surface Soil ------

Ingestion	Dermal contact	Inhalation of volatiles and particulates
2.68E-06	1.15E-05	3.89E-11
1.22E-05	5.25E-05	1.77E-10
2.04E-05	8.75E-05	2.96E-10
	2.68E-06 1.22E-05	Ingestion contact 2.68E-06 1.15E-05 1.22E-05 5.25E-05

----- LOCATION-SWMU 99A MEDIA-Surface Soil -----

Analyte	Ingestion	Dermal contact	Inhalation of volatiles and particulates
Barium	1.02E-04	4.38E-04	1.48E-09
Beryllium	3.26E-07	1.40E-06	4.74E-12
Chromium	4.60E-06	1.98E-05	6.692-11
Zinc	5.52E-05	2.38E-04	8.03E-10
Acenaphthene	1.61E-07	1.39E-06	3.26E-07
Acenaphthylene	1.28E-07	1.10E-06	1.90E-07
Anthracene	2.90E-07	2.49E-06	1.64E-07
Benz (a) anthracene	3.89E-07	3.35E-06	1.82E-08
Benzo (a) pyrene	2.39E-07	2.06E-06	4.31E-09
Benzo (b) fluoranthene	5.63E-07	4.84E-06	5.36E-08
Benzo (ghi) perylene	3.65E-07	3.14E-06	5.31E-12
Benzo (k) fluoranthene	2.80E-07	2.41E-06	3,14E-09
Chrysene	6.41E-07	5.51E-06	1.04E-07

----- LOCATION=SWMU 99A MEDIA=Surface Soil ----- (continued)

Analyte	Ingestion	Dermal contact	Inhalation of volatiles and particulates
Dibenz(a,h) anthracene	1.235-07	1.05E-06	5.19E-10
Dibenzofuran	6.02E-08	5.18E-07	6.55E-08
Fluoranthene	4.17E-07	3.58E-06	6.01E-08
Fluorene	1.07E-07	9.21E-07	9.25E-08
Indeno (1, 2, 3-cd) pyrene	3.92E-07	3.37E-06	3.03E-09
PCB-1016	1.16E-07	6.00E-07	1.08E-07
PCB-1254	4.70E-08	2.42E-07	3.90E-08
PCB-1260	9.16E-08	4.72E-07	9.01E-08
Phenanthrene	4.85E-07	4.17E-06	7.05E-12
Pyrene	4.78E-07	4.11E-06	5.54E-08
Cesium-137			
Neptunium-237	•		
Technetium-99			
Thorium-234			
Uranium-234			
Uranium-238			•

A-204

----- LOCATION=SWMU 193A MEDIA=Surface Soil -----Inhalation of volatiles Dermal and External Analyte Ingestion contact particulates exposure Chromium 2.14E-06 9.20E-06 3.11E-11 Anthracene 2.03E-08 1.74E-07 1.14E-08 3.15E-08 Benz (a) anthracene 2.70E-07 1.47E-09 Benzo (a) pyrene 4.37E-08 3.76E-07 7.86E-10 8.91E-09 Benzo (b) fluoranthene 7.66E-08 8.48E-10 Benzo (ghi) perylene 2.97E-08 2.55E-07 4.32E-13 Chrysene 2.97E-08 2.55E-07 4.81E-09 Di-n-butylphthalate 1.16E-07 7.34E-10 1.35E-08 Di-n-octylphthalate 2.10E-08 1:80E-07 1.54E-10 Dibenz (a, h) anthracene 2.27E-08 1.95E-07 9.62E-11

----- LOCATION-SWMU 193B MEDIA-Surface Soil ------

4.77E-08

2.80E-08

5.15E-08

2.97E-08

Fluoranthene

Pyrene

Indeno (1,2,3-cd) pyrene

bis(2-Ethylhexyl)phthalate

4.10E-07

2.40E-07

4.43E-07

2.55E-07

6.88E-09

2.16E-10

5.98E-09

6.10E-11

Analyte	Ingestion	Dermal contact	Inhalation of volatiles and particulates	External exposure
Beryllium	2.74E-07	1.18E-06	3.99E-12	
Chromium	1.55E-05	6.66E-05	2.25E-10	
Vanadium	1.14E-05	4.88E-05	1.65E-10	

----- LOCATION=SWMU 193C MEDIA=Surface Soil ------

Analyte	Ingestion	Dermal contact	Inhalation of volatiles and particulates	External exposure
Chromium	9.56E-07	4.11E-06	1.39E-11	
Lead	4.36E-06	·1.87E-05	6.33E-11	
Zinc	7.27E-06	3.13E-05	1.06E-10	

-- LOCATION-SWMU 99A MEDIA-Surface Soil ----

		Inhalation of volatiles		
		Dermal	and	External
Analyte	Ingestion	contact	particulates	exposure
Barium	3.63E-05	1.56E-04	5.28E-10	
Beryllium	1.16E-07	5.01E-07	1.69E-12	
Chromium	1.64E-06	7.07E-06	2.39E-11	
Zinc	1.97E-05	8.48E-05	2.87E-10	
Acenaphthene	5.77E-08	4.96E-07	1.16E-07	
Acenaphthylene	4.57E-08	3.93E-07	6.79E-08	
Anthracene	1.04E-07	8.91E-07	5,85E-08	
Benz(a) anthracene	1.39E-07	1.20E-06	6.49E-09	
Benzo (a) pyrene	8.55E-08	7.35E-07	1.54E-09	
Benzo (b) fluoranthene	2.01E-07	1.73E-06	1.91E-08	
Benzo (ghi) perylene	1.30E-07	1.12E-06	1.89E-12	
Benzo(k) fluoranthene	1.00E-07	8.60E-07	1.12E-09	
Chrysene	2.29E-07	1.97E-06	3.71E-08	

----- LOCATION=SWMU 99A MEDIA=Surface Soil ----- (continued)

			Inhalation of volatiles	
N m = 1 mm =	The second sector it areas	Dermal	and	External
Analyte	Ingestion	contact	particulates	exposure
Dibenz (a, h) anthracene	4.38E-08	3.77E-07	1.86E-10	
Dibenzofuran	2.15E-08	1.85E-07	2.34E-08	
Fluoranthene	1.49E-07	1.28E-06	2.15E-08	
Fluorene	3.83E-08	3.29E-07	3.30E-08	
Indeno (1,2,3-cd) pyrene	1.40E-07	1.20E-06	1.08E-09	
PCB-1016	4.16E-08	2.14E-07	3.86E-08	
PCB-1254	1.68E-08	8.66E-08	1.39E-08	
PCB-1260	3.27E-08	1.69E-07	3.228-08	
Phenanthrene	1.73E-07	1.49E-06	2.52E-12	
Pyrene	1.71E-07	1.47E-06	1.98E-08	
Cesium-137	3.30E+02		4.12E-03	4.83E+00
Neptunium-237	4.00E+03		4.98E-02	5.84E+01
Technetium-99	1.448+05		1.79E+00	2.10E+03
Thorium-234	6.74E+03		8.40E-02	9.85E+01
Uranium-234	5.13E+03		6.38E~02	7.49E+01
Uranium-238	1.62E+04		2.01E-01	2.36E+02

----- LOCATION=AOC 204 MEDIA=RGA Groundwater -----

Analyte	Ingestion	Dermal	Inhalation of volatiles and particulates	Inh. of volatiles while showering
1,1-Dichloroethane	4.89E-02	1.58E-03		2.67E-02
1,1-Dichloroethene	3.91E-04	1.26E-05		2.14E-04
PCB-1254	2.45E-04	3.07E-04		
PCB-1260	2.45E-04	9.46E-04		
Polychlorinated biphenyl	1.66E-03	2.09E-03		
Tetrachloroethene	6.27E-03	8.42E-03		3.43E-03
Trichloroethene	5.40E-03	3.13E-04		2.95E-03
Vinyl Chloride	9.78E-07	2.59E-0B		5.34E-07
cis-1,2-Dichloroethene	5.87E-05	2.13E-06		3.21E-05

----- LOCATION=SWMU 193A MEDIA=McNairy Groundwater ------

Analyte	Ingestion	Dermal contact	of volatiles and particulates	Inh. of volatiles while showering
Iron	1.29E+00	4.69E-03		
Tetraoxo-sulfate(1-)	4.02E-01			
Trichloroethene	3.64E-05	2.12E-06		1.99E-05
cis-1,2-Dichloroethene	1.66E-03	6.04E-05		9.08E-04
Technetium-99				
Tranium-238				

----- LOCATION-SWMU 193A MEDIA-RGA Groundwater ------

		Dermal	Inhalation of volatiles and	Inh. of volatiles while
Analyte	Ingestion	contact	particulates	showering
Ammonia	2.948-03	1.60E-05		
Fluoride	4.11E-03	1.49E-05		
Iron	2.96E-01	1.07E-03		
Silica	1.86E-01			
Tetraoxo-sulfate(1-)	9.98E-01			
Zinc	9.66E-04	3.51E-06		
1,1-Dichloroethene	1.96E-06	6.32E-08		1.07E-06
Pentachlorophenol	8.29E-05	1.96E-04		
Trichloroethene	1.65E-03	9.60E-05		9.03E-04
bis(2-Ethylhexyl)phthalate	1.26E-04	1.07E-05		
cis-1,2-Dichloroethene Technetium-99	2.84E-05	1.03E-06		1.55E-05

----- LOCATION-SWMU 193A MEDIA-Surface Soil ------

			Inhalation	Inh. of
Analyte	Ingestion	Dermal	of volatiles and particulates	volatiles while showering
Chromium	5.99E-06	2.57E-05	8.70E-11	
Anthracene	5.68E-08	4.88E-07	3.20E-08	
Benz (a) anthracene	8.81E-08	7.57E-07	4.11E-09	
Benzo (a) pyrene	1.22E-07	1.05E-06	2.20E-09	
Benzo (b) fluoranthene	2.50E-08	2.15E-07	2.37E-09	
Benzo (ghi) perylene	8.32E-08	7.15E-07	1.21E-12	

LOCATION-SWMU 193	A MEDIA=Surface	Soil	-	
(con	tinued)			•

Analyte	Ingestion	Dermal contact	Inhalation of volatiles and particulates	Inh. of volatiles while showering
Chrysene	8.32E-08	7.15E-07	1.35E-08	
Di-n-butylphthalate	3.77E-08	3.24E-07	2.06E-09	
Di-n-octylphthalate	5.87E-08	5.05E-07	4.33E-10	
Dibenz (a, h) anthracene	6.36E-08	5.47E-07	2.69E-10	
Fluoranthene	1.34E-07	1.15E-06	1.93E-08	
Indeno (1, 2, 3-cd) pyrene	7.83E-08	6.73E-07	6,05E-10	
Pyrene	1.44E-07	1.24E-06	1.67E-08	
bis (2-Ethylhexyl) phthalate	8.32E-08	7.15E-07	1.71E-10	

LOCATION=SWMU 193B MEDIA=McNairy Groundwater ----

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Analyte	Ingestion	Dermal contact	Inhalation of volatiles and particulates	Inh. of volatiles while showering
Trichloroethene	1.27E-04	7.39E-06		6.95E-05
cis-1,2-Dichloroethene	2.25E-04	8.17E-06		1.23E-04

--- LOCATION=SWMU 193B MEDIA=RGA Groundwater -----

Analyte	Ingestion	Dermal contact	Inhalation of volatiles and particulates	Inh. of volatiles while showering
1,1-Dichloroethene	1.58E-05	5.12B-07		8.65E-06
Acetone	3.23E-04	6.67E-07		1.76E-04
Carbon Tetrachloride	5.38E-05	4.30E-06	•	2.94E-05
Di-n-butylphthalate	9.94E-05	4.15E-05		
Trichloroethene	4.89E-03	2.84E-04		2.67E-03
bis (2-Ethylhexyl) phthalate	9.87E-05	8.38E-06		
cis-1,2-Dichloroethene Technetium-99	8.04E-05	2.928-06		4.39E-05

----- LOCATION-SWMU 193B MEDIA-Surface Soil

Analyte	Ingestion	Dermal	Inhalation of volatiles and particulates	Inh. of volatiles while showering
Beryllium	7.68E-07	3.30E-06	1.12E-11	
Chromium	4.34E-05	1.878-04	6.31E-10	
Vanadium	3,18E-05	1.37E-04	4.62E-10	

- LOCATION-SWMU 193C MEDIA-McNairy Groundwater ---

Analyte	Ingestion	Dermal contact	of volatiles and particulates	volatiles while showering	
Aluminum Antimony	3.74E-01 1.12E-03	1.36E-03 4.06E-06			

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------ LOCATION=SWMU 193C MEDIA=McNairy Groundwater ------(continued)

hun]uto	Ingestion	Dermal	Inhalation of volatiles and	Inh. of volatiles while
Analyte	indesciou	CONLACE	particulates	showering
Arsenic	1,19E-04	4.34E-07		•
Barium	2.39E-03	8,68E-06		
Beryllium	1.09E-04	3.94E-07		
Cadmium	3.46E-04	1.26E-06		•
Chromium	1.40E-03	5.09E-06		
Cobalt	5.20E-04	1.89E-06		-
Iron	5.76E-01	2.09E-03		
Lead	2.45E-03	8.88E-06		
Manganese	1.33E-02	4.84E-05		
Mercury	1.96E-06	7.10E-09		
Molybdenum	4.52E-04	1.64E-06	1	· · · ·
Nickel	5.26E-04	1.91E-06		
Silica	7.83E-02			•
Silver	3.25E-04	1.18E-06	•	
Tetraoxo-sulfate(1-)	6.45E-02			
Thallium	1.20E-03	4.37E-06		
Uranium	6.26E-05	2.27E-07		•
Vanadium	8.18E-03	2.97E-05		
Zinc	1.99E-03	7.22E-06		· .
1,1,2-Trichloroethane	2.45E-05	7.46E-07		1.34E-05
1,1-Dichloroethene	2.45E~05	7.90E-07		1.34E-05
1,2-Dichloroethane	2.45E-05	4.71E-07		1.34E-05
Benzene	2.45E-05	1.86E-06		1.34E-05
Bromodichloromethane	2.45E-05	5.15E-07		1.34E-05
Carbon Tetrachloride	2.45E-05	1.95E-06		1.34E-05
Chloroform	2.45E-05	7.90E-07		1.34E-05
Ethylbenzene	2.45E-05	6.57E-06		1.34E-05
Polychlorinated biphenyl	9.78E-07	1,23E-06	:	
Tetrachloroethene	2.45E-05	3.29E-05		1.34E-05
Trichloroethene	1,21E-05	7.01E-07		6.59E-06
Vinyl Chloride	8.99E-05	2.38E-06		4.91E-05
Xylene	5.33E-05	1.83E-05		2.91E-05
cis-1,2-Dichloroethene	4.89E-05	1.78E-06		2.67E-05
trans-1,2-Dichloroethene Radon-222	4.89E-05	1.90E-07		2.67E-05

-- LOCATION-SWMU 193C MEDIA-RGA Groundwater -

Analyte	Ingestion	Dermal contact	Inhalation of volatiles and particulates	Inh. of volatiles while showering
1,2-Dichloroethene Trichloroethene	5.50E-03 1.59E-03	2.14E-05 9.21E-05		3.00E-03 8.66E-04
•	···.	•		

- LOCATION-SWMU 193C MEDIA-Surface Soil -

Analyte	Ingestion	Dermal contact	Inhalation of volatiles and particulates	Inh. of volatiles while showering
Chromium	2.68E-06	1.15E-05	3.89E-11	· ·
Lead	1.22E-05	5.25E-05	1.77E-10	
Zinc	2.04E-05	8.75E-05	2.96E-10	

----- LOCATION=SWMU 99A MEDIA=McNairy Groundwater -----

Analyte	Ingestion	Dermal	Inhalation of volatiles and particulates	Inh. of volatiles while showering
1,1-Dichloroethene	1.02E-04	3.29E-06		5.56E-05
Carbon Tetrachloride	2.74E-05	2.19E-06		1.50E-05
Trichloroethene	4.26E-03	2.47E-04		2.33E-03
cis-1,2-Dichloroethene	1.13E-03	4.10E-05		6.17E-04

- LOCATION=SWMU 99A MEDIA=RGA Groundwater

Analyte	Ingestion	Dermal	Inhalation of volatiles and particulates	Inh. of volatiles while showering	
Aluminum	1.06E-01	3.85E-04			
Arsenic	5.39E-05	1.96E-07			
Barium	3.36E-03	1.22E-05		•	
Beryllium	1.01E-04	3.68E-07			
Chromium	1.24E-03	4.50E-06			
Cobalt	9.24E-04	3.35E-06		•	
Copper	7.81E-04	2.83E-06			
Iron	2.27E-01	8.23E-04			
Lead	7.95E-04	2.89E-06			
Lithium	7.28E-04	2.64E-06			
Manganese	1.63E-02	5.90E-05			
Mercury	4.22E-06	1,53E-08			
Nickel	1.50E-03	5.45E-06			
Silica	1.01E-01				
Sulfate	1.16E-01	4.21E-04			
Tetraoxo-sulfate(1-)	9.62B-02				
Vanadium	3.59E-03	1.30E-05			
Zinc	2.20E-03	8.00E-06			
1,1-Dichloroethene	1.75E-04	5.65E-06		9.56E-05	
Trichloroethene	6.61E-03	3.84E-04	•	3.61E-03	
bis(2-Ethylhexyl)phthalate	9.36E-05	7.95E-06	-		
cis-1,2-Dichloroethene Radon-222 Technetium-99	6.898-05	2.508-06		3.76E-05	

- LOCATION-SWMU 99A MEDIA-Surface Soil

Analyte	Ingestion	Dermal	Inhalation of volatiles and particulates	Inh. of volatiles while showering
Barium	1.02E-04	4.38E-04	1.48E-09	
Beryllium	3.26E-07	1.40E-06	4.74E-12	
Chromium	4.60E-06	1.98E-05	6.69E-11	
Zinc	5.52E-05	2.38E-04	8.03E-10	
Acenaphthene	1.61E-07	1.39E-06	3.26E-07	
Acenaphthylene	1.28E-07	1.10E-06	1.90E-07	
Anthracene	2.90E-07	2.49E-06	1.64E-07	
Benz (a) anthracene	3.89E-07	3.35E-06	1.82E-08	
Benzo (a) pyrene	2.39E-07	2.06E-06	4.315-09	
Benzo(b) fluoranthene	5.63E-07	4.84E-06	5.365-08	
Benzo(ghi)perylene	3.65E-07	3.14E-06	5.315-12	
Benzo(k)fluoranthene	2.80E-07	2.41E-06	3.14E-09	
Chrysene	6.41E-07	5.51E-06	1.04E-07	
Dibenz(a,h) anthracene	1.23E-07	1.05E-06	5.19E-10	

----- LOCATION=SWMU 99A MEDIA=Surface Soil ------(continued)

Analyte	Ingestion	Dermal	Inhalation of volatiles and particulates	Inh. of volatiles while	,
murice	Indeperou	contact	parciculates	showering	
Dibenzofuran	6.02E-08	5.18E-07	6.55E-08		
Fluoranthene	4.17E-07	3.58E-06	6.01E-08		
Fluorene	1.07E-07	9.21E-07	9.25E-08		
Indeno(1,2,3-cd)pyrene	3.92E-07	3.37E-06	3.03E-09		
PCB-1016	1.16E-07	6.00E-07	1:08E-07		
PCB-1254	4.70E-08	2.42E-07	3.90E-08	•	
PCB-1260	9.16E-08	4.72E-07	9.01E-08		
Phenanthrene	4.85E-07	4.17E-06	7.05E-12		
Pyrene	4.78E-07	4.11E-06	5.54E-08		
Cesium-137					
Neptunium-237					
Technetium-99					
Thorium-234					
Uranium-234					
Uranium-238				•	

----- LOCATION=SWMU 99B MEDIA=RGA Groundwater ------

Analyte	Ingestion	Dermal contact	Inhalation of volatiles and particulates	Inh. of volatiles while showering
Barium	4.80E-03	1.74E-05		
Chromium	6.06E-04	2.20E-06		
Iron	2.19E-02	7.96E-05		
Manganese	2.54E-03	9.22E-06		
Silīca	8.97E-02			
Sulfate	2.50E-01	9.08E-04		
Tetraoxo-sulfate(1-)	1.42E-01			
Zinc	5.71E-04	2.07E-06		
Trichloroethene Radon-222	2.03E-02	1.18E-03		1.11E-02

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----- LOCATION=AOC 204 MEDIA=RGA Groundwater -----

Analyte	Ingestion	Dermal contact	Inhalation of volatiles and particulates	Inh. of volatiles while showering	External exposure
1,1-Dichloroethane	1.75E-02	5.64E-04		9.54E-03	
1,1-Dichloroethene	1.40E-04	4.52E-06		7.63E-05	
PCB-1254	8.74E-05	1.10E-04			
PCB-1260	8.74E-05	3.38E-04			
Polychlorinated biphenyl	5.94B-04	7.46E-04			
Tetrachloroethene	2.24E-03	3.01E-03		1.22E-03	
Trichloroethene	1.93E-03	1.12E-04		1.05E-03	
Vinyl Chloride	3.49E-07	9.26E-09		1.91E-07	
cis-1,2-Dichloroethene	2.10E-05	7.61B-07		1.15E-05	

----- LOCATION-SWMU 193A MEDIA-MCNairy Groundwater -----

,	Analyte	Ingestion	Dermal	Inhalation of volatiles and particulates	Inh. of volatiles while showering	External exposure
	Iron	4.622-01	1.68E-03			
	Tetraoxo-sulfate(1-)	1.44E-01				
	Trichloroethene	1.308-05	7.56E-07		7.11E-06	
•	cis-1,2-Dichloroethene	5.94E-04	2.16E-05		3.24E-04	
	Technetium-99	2.30E+05				
	Uranium-238	8.25E+03				

----- LOCATION-SWMU 193A MEDIA-RGA Groundwater -----

Analyte	Ingestion	Dermal contact	Inhalation of yolatiles and particulates	Inh. of volatiles while showering	External exposure
Ammonia	1.05E-03	5.71E-06			
Fluoride	1.47E-03	5.33B-06			
Iron	1.06E-01	3.83E-04			
Silica	6.64E-02			,	
Tetraoxo-sulfate (1-)	3.56E-01	• •		•.	
Zinc	3.45E-04	1.25E-06		•	
1,1-Dichloroethene	6.99E-07	2.26E-08		3.82E-07	
Pentachlorophenol	2.96E-05	6. 98E-05			
Trichloroethene	5.91E-04	3.43E-05	•	3.23E-04	
bis (2-Ethylhexyl) phthalate	4.51E-05	3.83E-06			
cis-1,2-Dichloroethene	1.02E-05	3.69E-07		5.54E-06	
Technetium-99	1.20E+06				

----- LOCATION-SWMU 193A MEDIA-Surface Soil -----

Analyte	Ingestion	Dermal	Inhalation of volatiles and particulates	Inh. of volatiles while showering	External exposure	
Chromium	2.14E-06	9. 20E-06	3.11E-11			
Anthracene	2.03E-08	1.74E-07	1.14E-08			
Benz (a) anthracene	3.15E-08	2.70E-07	1.476-09			
Benzo (a) pyrene	4.37E-08	3.76E-07	7.86E-10			
Benzo (b) fluoranthene	8.91E-09	7.66E-08	8.48E-10			
Benzo (ghi) perylene	2.97E-08	2.55E-07	4.32E-13			

Table 1.45. Carcinogenic chronic daily intakes for future industrial worker ----- LOCATION=SWMU 193A MEDIA=Surface Soil -----(continued) . . Inhalation Inh. of of volatiles volatiles Dermal and while External Analyte Ingestion contact particulates showering exposure 2.97E-08 4.81E-09 Chrysene 2.55E-07 Di-n-butylphthalate 1.35E-08 1.16E-07 7.34E-10 Di-n-octylphthalate 2.10E-08 1.805-07 1.54E-10 Dibenz (a, h) anthracene 2.27E-08 1.956-07 9.62E-11 Fluoranthene 4.77E-08 4.10E-07 6.88E-09 Indeno (1,2,3-cd) pyrene 2.80E-08 2.40E-07 2.16E-10 Pyrene 5.15E-08 4.43E-07 5.98E-09 bis(2-Ethylhexyl)phthalate 2.97E-08 2.55E-07 6.10E-11 ----- LOCATION=SWMU 193B MEDIA=McNairy Groundwater ------Inhalation Inh. of of volatiles volatiles Dermal and while External Analyte Ingestion contact particulates showering exposure Trichloroethene 4.54E-05 2.64E-06 2.48E-05 cis-1,2-Dichloroethene 8.04E-05 2.92E-06 4.398-05 LOCATION-SWMU 193B MEDIA-RGA Groundwater --Inhalation Inh. of of volatiles volatiles Dermal and while External particulates Ingestion Analyte contact showering exposure 5.66E-06 1,1-Dichloroethene 1.83E-07 3.098-06 1.15E-04 Acetone 2.385-07 6.30E-05 Carbon Tetrachloride 1.92E-05 1.538-06 1.05E-05 Di-n-butylphthalate 3.55E-05 1.48E-05 Trichloroethene 1.75E-03 1.01E-04 9.54E-04 bis(2-Ethylhexyl)phthalate 3.52E-05 2.99E-06 cis-1,2-Dichloroethene 2.87E-05 1.04E-06 1.578-05 ·1.71E+05 Technetium-99 - LOCATION-SWMU 1938 MEDIA-Surface Soil ------Inhalation Inh. of of volatiles volatiles Dermal anđ while External Analyte. Ingestion contact particulates showering exposure 3.99E-12 Beryllium 2.74E-07 1.18E-06 Chromium 1.55E-05 6.66E-05 2.25E-10 Vanadium 1.65E-10 4.88E-05 1.14E-05 ---- LOCATION-SWMU 193C MEDIA-McNairy Groundwater -----Inh. of Inhalation of volatiles volatiles while External and Dermal exposure particulates showering Analyte Ingestion contact 4.85E-04 Aluminum 1.34E-01

A-213

1.45E-06

4.00E-04

Antimony