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Primary Document

Feasibility Study for the Groundwater Operable Unit at Paducah Gaseous Diffusion Plant Paducah, Kentucky

Volume 3. Appendix B Baseline Risk Assessment

Cleared for Public Release

SCIENCE APPLICATIONS INTERNATIONAL CORPORATION

contributed to the preparation of this document and should not be considered an eligible contractor for its review.

DOE/OR/07-1857&D2
Primary Document

Feasibility Study
for the Groundwater Operable Unit
at Paducah Gaseous Diffusion Plant
Paducah, Kentucky

Volume 3. Appendix B
Baseline Human Health Risk Assessment

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By
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managing the

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EXECUTIVE SUMMARY

This baseline human health risk assessment (BHHRA) utilizes information collected during a number of previous investigations to characterize the baseline risks posed to human health from contact with contaminants in groundwater at and around the Paducah Gaseous Diffusion Plant (PGDP). (Baseline doses from ingestion of groundwater are also characterized in an attachment to this BHHRA.) This BHHRA also uses information from fate and transport modeling to estimate the baseline risks posed to human health through contact with groundwater and other media impacted by contaminants migrating from the various sources at the PGDP to selected points of exposure. Generally, baseline risks are defined as those that may be present now or in the future in absence of corrective or remedial actions.

The methods and presentations used in this BHHRA are consistent with those in *Methods for Conducting Human Health Risk Assessments and Risk Evaluations at the Paducah Gaseous Diffusion Plant* (DOE/OR/07-1506&D1 as modified by regulatory comments) (DOE 1996a). The Methods Document, which integrates the human health risk assessment guidance from the United States Environmental Protection Agency (EPA) with that from the Kentucky Department of Environmental Protection (KDEP) and incorporates the various instructions contained in regulatory agency comments on earlier risk assessments performed for the PGDP, received final approval from the Commonwealth of Kentucky for use in environmental investigations and restoration activities at the PGDP in February 1998 (KDEP 1998). As noted in the Methods Document, the methods used here are consistent with those in *Risk Assessment Guidance for Superfund* (RAGS) (EPA 1989a) and additional guidance developed and distributed by EPA and KDEP subsequent to the release of RAGS (*e.g.*, EPA 1989b, 1990a-b, 1991a-c, 1992a-c, 1993a, 1995a).

To facilitate data aggregation and to focus results on specific areas, this BHHRA derives risk estimates for several area and depth data aggregates and individual sampling stations. The areas are as follows:

- Area a – Inside TCE contaminated area at C-400 Building – Inside industrialized area
- Area b – Inside the Northwest TCE Plume – Inside industrialized area (i.e., west main plant)
- Area c – Inside the Northeast TCE Plume – Inside industrialized area (i.e., east main plant)
- Area d – Outside the TCE Plumes – South of C-400 in industrialized area
- Area e – Inside the Northwest TCE Plume – Outside industrialized area
- Area f – Inside the Northeast TCE Plume – Outside industrialized area
- Area g – Outside the TCE Plumes – West of industrialized area (i.e., west of plume)
- Area h – Outside the TCE Plumes – East of industrialized area (i.e., east of plume)
- Area i – Outside the TCE Plumes – North of industrialized area (i.e., between the plumes)
- Area j – Outside the TCE Plumes - Tennessee Valley Authority area (TVA)
- Area k – Outside the TCE Plumes – South of industrialized area above terrace
- Area l – Inside plant area – Composed of Areas a, b, c, and d
- Area m – Outside plant area – Composed of Areas e, f, g, h, i, j, and k
- Area n – All groundwater – Composed of Areas l and m

The depth classifications used were based upon a combination of the depth at which the sample was collected and the characteristics of the subsurface in the area of the sampling station. These groups and their definitions are summarized as follows:

- HU1 – data from a sample collected in Hydrogeological Unit 1

- HU2 – data from a sample collected in Hydrogeological Unit 2

- HU3 – data from a sample collected in Hydrogeological Unit 3
- HU4 – data collected from a sample collected in Hydrogeological Unit 4
- HU5 – data collected from a sample collected in Hydrogeological Unit 5
- HU6 – data collected from a sample collected in Hydrogeological Unit 6
- Other – data from a sample collected from a hydrogeological unit not included above (i.e., Terrace Gravel, Porters Creek Clay, and Eocene Sands)
- UCRS – data from samples assigned to HU1, HU2, or HU3
- RGA – data from samples assigned to HU4 or HU5
- McNairy Formation – data from samples assigned to HU6

Consistent with regulatory guidance and previous agreements, the area assessment in this BHHRA evaluates scenarios that encompass current use and several hypothetical future uses of groundwater at the PGDP. These and the exposure routes considered under each are as follows:

Industrial worker

- ingestion of groundwater,
- dermal contact with groundwater while showering, and
- inhalation of vapors emitted by groundwater while showering.

Recreational user

- incidental ingestion of water while swimming in ponds filled with groundwater,
- dermal contact with water while swimming in ponds filled with groundwater,
- dermal contact with water while wading in ponds filled with groundwater,
- consumption of fish raised in ponds filled with groundwater,
- consumption of venison from deer drinking groundwater,
- consumption of meat from rabbits drinking groundwater, and
- consumption of meat from quail drinking groundwater.

Rural resident

- 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,
- consumption of vegetables,
- consumption of beef from cows drinking groundwater,
- consumption of milk from cows drinking groundwater,
- consumption of meat from chickens and turkeys drinking groundwater,
- consumption of eggs from chickens drinking groundwater, and
- consumption of pork from swine drinking groundwater.

Major conclusions and observations of the BHHRA are presented below.

LAND USES OF CONCERN

For the area assessment, not all area/depth classifications were found to have land use scenarios of concern for both systemic toxicity and ELCR. However, the RGA was found to be of concern for all uses in all areas, and the UCRS was found to be of concern for residential and industrial use in all areas where data were available and for recreational use in all but Areas c, f, h, and j.

The McNairy Formation had more areas where the land uses assessed were not of concern than the UCRS and RGA. Under the industrial worker scenario, Areas a, c, d, f, and i, were not of concern; under the recreational user, Areas a, c, d, f, h, and i were not of concern; and under the rural resident, Areas a, b, and f were not of concern. (Note that data were not available for the McNairy Formation in Areas a and b. Also, the McNairy Formation did not apply to Area k.)

Area k (i.e., groundwater taken to the south of the PGDP on the terrace) was of concern for each land use for systemic toxicity and ELCR.

CONTAMINANTS OF CONCERN

Multiple COCs were found for each of the land uses. Combining the results for systemic toxicity and ELCR and considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs were identified as "priority COCs" in UCRS groundwater across all use scenarios (excluding Area k):

- Inorganic chemicals – arsenic, antimony, beryllium, cadmium, chromium, iron, lead, manganese, nickel, and vanadium.
- Organic compounds – 1,1-dichloroethene, benzene, chloroform, ethylbenzene, naphthalene, *trans*-1,2-dichloroethene, *cis*-1,2-dichloroethene, TCE, and vinyl chloride.
- Radionuclides – ^{222}Rn.

For Area k, the "priority COCs" in groundwater across all use scenarios were:

- Inorganic chemicals – antimony, beryllium, cadmium, iron, lead, manganese, and vanadium
- Organic compounds – 1,1-dichloroethene, 1,2-dichloroethene, naphthalene, *cis*-1,2-dichloroethene, TCE, and vinyl chloride.
- Radionuclides – ^{222}Rn.

For the RGA, the following COCs were identified as "priority COCs" in RGA groundwater across all use scenarios:

- Inorganic chemicals – antimony, arsenic, beryllium, cadmium, chromium, iron, lead, manganese, molybdenum, and vanadium.
- Organic compounds – 1,1-dichloroethene, acrylonitrile, carbon tetrachloride, Aroclor-1254, tetrachloroethene, *cis*-1,2-dichloroethene, *trans*-1,2-dichloroethene, TCE, and vinyl chloride

- Radionuclides – ^{226}Ra and ^{222}Rn.

For the McNairy Formation, the following COCs were identified as "priority COCs" in McNairy Formation groundwater across all use scenarios:

- Inorganic chemicals – antimony, arsenic, beryllium, cadmium, chromium, iron, manganese, molybdenum, and vanadium.

- Organic compounds – TCE.

- Radionuclides – ^{222}Rn.

(Note that "priority COCs" are those that present either a chemical-specific HI or ELCR at one or more areas, across all land uses, that exceeds 1 or 1×10^{-4}, respectively.)

PATHWAYS OF CONCERN

All direct contact exposure routes (i.e., those involving ingestion, dermal contact, and inhalation) and the sum of the biota consumption exposure routes were of concern for at least one area/depth classification combination. However, specific biota consumption routes were determined to not be of concern for some areas. Biota consumption routes for the recreational user not of concern in any area were consumption of venison, rabbit, and quail. Biota consumption routes for the resident not of concern in any area were consumption of eggs and consumption of pork. Biota consumption routes for the recreational user and resident that were of concern for virtually all area and depth classification combinations were consumption of fish and consumption of vegetables, respectively.

OVERALL CONCLUSION

When the risk results and uncertainties are integrated, the conclusion reached during the earlier Site Investigation Phase II risk assessment is valid for this GWOU BHHRA as well. In general, the contamination problem posing the greatest risk from use of groundwater at the PGDP is the presence of TCE and its breakdown products in the aquifer. Although several inorganic chemicals and some radionuclides contribute significantly to total risk, these contaminants may be related to sampling or other biases and be of less relative importance. However, the other contaminants and contamination in source areas need to be considered when developing remedies for groundwater contamination and its sources at the PGDP, because modeling results indicate that unacceptable risks may develop if contaminants are allowed to continue to migrate from these source areas. However, because the modeling results are very uncertain, the appropriate risk management decision may be to address the TCE contamination in the short-term.

1. INTRODUCTION AND RESULTS OF PREVIOUS INVESTIGATIONS

1.1 INTRODUCTION

This baseline human health risk assessment (BHHRA) utilizes information collected during a number of previous investigations to characterize the baseline risks posed to human health from contact with contaminants in groundwater at the Paducah Gaseous Diffusion Plant (PGDP). This BHHRA also uses information from fate and transport modeling to estimate the baseline risks posed to human health through contact with groundwater and other media impacted by contaminants migrating from the various sources at the PGDP to selected points of exposure. Generally, baseline risks are defined as those that may be present now or in the future in absence of corrective or remedial actions.

The methods and presentations used in this BHHRA are consistent with those presented in *Methods for Conducting Human Health Risk Assessments and Risk Evaluations at the Paducah Gaseous Diffusion Plant* (DOE/OR/07-1506&D1 as modified by regulatory comments) (DOE 1996a). The Methods Document, which integrates the human health risk assessment guidance from the U.S. Environmental Protection Agency (EPA) with that from the Kentucky Department of Environmental Protection (KDEP) and incorporates the various instructions contained in regulatory agency comments on earlier risk assessments performed for the PGDP, received final approval from the Commonwealth of Kentucky for use in environmental investigations and restoration activities at the PGDP in February 1998 (KDEP 1998). As noted in the Methods Document, the methods used here are consistent with those in *Risk Assessment Guidance for Superfund* (RAGS) (EPA 1989a) and additional guidance developed and distributed by EPA and KDEP subsequent to the release of RAGS (e.g., EPA 1989b, 1990a-b, 1991a-c, 1992a-c, 1993a, 1995a).

Consistent with the Methods Document, this BHHRA is presented in nine sections. The first section introduces the BHHRA, reviews the results of previous risk assessments that are useful in understanding the risks posed to human health by groundwater contaminants, and presents sources of information that were used to complete the exposure assessment contained in this BHHRA. The second section describes the data set used in this BHHRA and presents the methods used to identify the chemicals of potential concern (COPCs) for groundwater. The third section documents the exposure assessment for the GWOU, 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. The fourth section presents the toxicity assessment of this BHHRA, including information on the noncarcinogenic and carcinogenic effects of the COPCs and the uncertainties in the toxicity information. The fifth section reports the results of the risk characterization for current and various future land uses and identifies contaminants, pathways, and land use scenarios of concern. The sixth section contains qualitative and quantitative analyses of the uncertainties affecting the results of the BHHRA. The seventh section summarizes the methods used in the BHHRA and presents the BHHRA's conclusions and observations. The eighth section uses the results of the BHHRA to develop site-specific risk-based remedial goal options (RGOs). The ninth section contains references.

Because of their length, all tables cited within this BHHRA are presented in Attachment 1 of this BHHRA. However, because some reviewers have noted that such a presentation makes the tables difficult to access, this BHHRA also includes exhibits. These exhibits are shorter tables that are presented within the text of the BHHRA and summarize much of the material presented in the longer tables. Similarly, in response to comments received from reviewers, all figures cited in this BHHRA are presented in the text. However, to be consistent with past assessments, some of these figures are also presented in Attachment 2 of this BHHRA. Other attachments are Attachment 3, SAS Programs; Attachment 4, Complete Toxicity Profiles; Attachment 5, Lead Modeling; Attachment 6, Filtered BHHRA Results; Attachment 7, Modeled

Concentration BHHRA Results; Attachment 8, Well-by-Well BHHRA Results; Attachment 9, Summary of the BERA for Northwest Dissolved Phase Plume; Attachment 10, Radiation Dose Assessment for Residential Groundwater Use; and Attachment 11, Risk Characterization for the Southeast Plume.

1.2 RESULTS OF PREVIOUS INVESTIGATIONS

Several previous studies were conducted investigating groundwater contamination at the PGDP and its sources. These studies fall into two general categories: (1) source control unit investigations and (2) integrator unit investigations. These investigations and their human health risk results are discussed in the following material. Additional information about the units included in the various investigations is provided in the Data Summary Report (Vol. 2, App. A) in the Groundwater Operable Unit (GWOU) Feasibility Study (FS).

1.2.1 Previous Source Control Unit Investigations

Fourteen previous investigations contain risk assessment results that address contamination migrating to groundwater or in groundwater at the various source control units at the PGDP. These reports are listed below by their date of release.

- *Results of the Public Health and Ecological Assessment, Phase II* (CH2M Hill 1991a) [This report is Vol. 6 of *Results of the Site Investigation, Phase II, at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky* (CH2M Hill 1992)

- *Baseline Risk Assessment for the Underground Storage Tanks at the C-200, C-710, and C-750 Buildings, Paducah Gaseous Diffusion Plant, Paducah, Kentucky* (DOE 1992a)

- *Remedial Investigation Addendum for Waste Area Grouping 22, Burial Grounds, Solid Waste Management Units 2 and 3, at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky* (DOE 1994a)

- *Remedial Investigation Addendum for Waste Area Grouping 23, PCB Sites, at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky* (DOE 1994b)

- *Resource Conservation and Recovery Act Facility Investigation/Remedial Investigation Report for Waste Area Groupings 1 and 7 at Paducah Gaseous Diffusion Plant, Paducah, Kentucky* (DOE 1996b)

- *Baseline Risk Assessment for Exposure to Polycyclic Aromatic Hydrocarbons at Underground Storage Tanks C-750 A&B, Paducah Gaseous Diffusion Plant, Paducah, Kentucky* (DOE 1996c)

- *Baseline Risk Assessment for Underground Storage Tanks 130, 131, 132, 133, and 134 as presented in the WAGs 1&7 RFI/RI, Paducah Gaseous Diffusion Plant, Paducah, Kentucky, UST Facility/Site Identification Number 6319073* (DOE 1996d)

- *Data Summary and Interpretation Report for Interim Remedial Design at Solid Waste Management Unit 2 of Waste Area Grouping 22 at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky* (DOE 1997a)

- *Remedial Investigation for Solid Waste Management Units 7 and 30 of Waste Area Grouping 22 at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky* (DOE 1998a)

- *Remedial Investigation Report for Waste Area Grouping 6 at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky* (DOE 1999a)

- *Remedial Investigation Report for Waste Area Grouping 27 at the Paducah Gaseous Diffusion Plant Paducah, Kentucky* (DOE 1999b)

- *Residual Risk Evaluation for Waste Area Grouping 23 and Solid Waste Management Unit 1 of Waste Area Grouping 27 at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky* (DOE 1999c)

- *Remedial Investigation Report for Waste Area Grouping 28 at the Paducah Gaseous Diffusion Plant Paducah, Kentucky* (DOE 2000a)

- *Remedial Investigation Report for Waste Area Grouping 3 at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky* (DOE 2000b)

The following subsections present the risk assessment and risk evaluation results found in these reports. Note that the methods used in some of the previous risk assessments are not consistent with those prescribed in the Methods Document (DOE 1996a). Therefore, the results presented in the following subsections should be used for comparison only and be considered preliminary to the results reported later in this BHHRA.

1.2.1.1 WAG 6 (from material in DOE 1999a)

In 1997, the U.S. Department of Energy (DOE) conducted a Remedial Investigation (RI)/Resource Conservation and Recovery Act (RCRA) Facility Investigation (RFI) for solid waste management units (SWMUs) 11, 26, 40, 47, and 203 in Waste Area Grouping (WAG) 6 at the PGDP. In addition, this RI included areas surrounding the C-400 Building that are not part of any recognized SWMU. The overall purpose of this activity was to determine the presence, nature, and extent of contamination at each of the SWMUs and in the C-400 area. The primary focus of the RI was to collect sufficient information about contamination in surface and subsurface soil and in shallow groundwater [i.e., in the Upper Continental Recharge System (UCRS)] to support an assessment of risks to human health and the environment and the selection of actions to reduce these risks. In addition, contamination in the Regional Gravel Aquifer (RGA) and McNairy Formation was characterized to determine if contamination there acted as a secondary source of contamination to groundwater. Investigative activities included sampling and analysis of surface and subsurface soils, groundwater, and investigation-derived waste.

To facilitate data aggregation and to focus results on specific areas, the BHHRA derived risk estimates for several sectors in addition to the whole of WAG 6. The sectors and their definitions are as follows:

- Sector 1 – the area under the C-400 Building.

- Sector 2 – the area to the northeast of the C-400 Building. This sector contains SWMU 40.

- Sector 3 – the area to the east of the C-400 Building. This sector does not contain a SWMU.

- Sector 4 – the area to the southeast of the C-400 Building. This sector contains SWMU 11.

- Sector 5 – the area to the southwest of the C-400 Building. This sector does not contain a SWMU.

- Sector 6 – the area to the west of the C-400 Building. This sector contains SWMU 47.

- Sector 7 – the area to the northwest of the C-400 Building. This sector contains SWMU 203.

- Sector 8 – the area to the far north and northwest of the C-400 Building. This sector contains SWMU 26.

- Sector 9 – the area to the far east and northeast of the C-400 Building. This sector does not contain a SWMU.

Consistent with regulatory guidance and previous agreements, the BHHRA evaluated scenarios that encompassed current use and several hypothetical future uses of the WAG 6 area and areas to which contaminants from WAG 6 may migrate. These scenarios are listed below.

- Current on-site industrial – direct contact with surface soil [soil found 0 to 1 ft bgs (below ground surface)].

- Future on-site industrial – direct contact with surface soil at and use of groundwater drawn from aquifers below the WAG 6 area.

- Future on-site excavation scenario – direct contact with surface and subsurface soil (soil found 1 to 15 ft bgs).

- Future on-site recreational user – consumption of game exposed to contaminated surface soil.

- Future off-site recreational user – direct contact with surface water impacted by contaminants migrating from sources and consumption of game exposed to this surface water.

- Future on-site rural resident – direct contact with surface soil at and use of groundwater drawn from aquifers below the WAG 6 area, including consumption of vegetables that were posited to be raised in this area.

- Future off-site rural resident – use in the home of groundwater drawn from the RGA at the DOE property boundary.

Note that this report also contains a baseline ecological risk assessment (BERA) for nonhuman receptors that may come into contact with contaminated media at or migrating from sources in the WAG 6 area. Results from this BERA are not discussed here.

Major conclusions and observations of the BHHRA are as follows:

- For all sectors and the C-400 area as a whole, the cumulative human health excess lifetime cancer risk (ELCR) and systemic toxicity exceeds the acceptable standards of KDEP and EPA for one or more scenarios when assessed using default exposure parameters. The results for each scenario and sector combination are summarized graphically in Exhibit 1.1 and presented in more detail in Exhibit 1.2.

- ELCR and systemic toxicity [hazard index (HI) in Exhibit 1.2] for use of groundwater drawn from the RGA and McNairy Formation were greater than upper end of the EPA risk range (i.e., 1×10^{-4} and 1 for ELCR and HI, respectively) for both the future industrial worker and potential future on-site resident. Contaminants in groundwater driving risk were trichloroethene (TCE), vinyl chloride, and ^{210}Pb. Contaminants in groundwater driving systemic toxicity were iron and TCE.

  Because there was considerable uncertainty in some of the exposure parameters, exposure pathways and toxicity values, a quantitative uncertainty analysis was performed. In this analysis, approved toxicity values and site-specific exposure parameters and exposure pathways were used to calculate

Exhibit 1.1. Land uses of concern for WAG 6

	Location (Sector Number)									
Scenario	**WAG 6**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**
Results for systemic toxicity[a]										
Current Industrial Worker	X[d]	NA	–	–	–	X	X	X[d]	–	X
Future Industrial Worker										
Exposure to Soil	X[d]	NA	–	–	–	X	X	X[d]	–	X
Exposure to Water[b]	X[d]									
Future Excavation Worker	X[d]	X	X	X[e]	X[d]	X[d]	X	X[d]	X[d]	X[d]
Future Recreational User	X[e]	NA	–	–	–	–	–	–	–	–
Future On Site Resident										
Exposure to Soil	X[d]	NA	X	X	X	X	X	X[d]	X	X
Exposure to Water[b]	X[d]									
Future Off Site Resident Exposure to Water[c]	X	–	–	–	X	X	–	X	X	–
Results for excess lifetime cancer risk										
Current Industrial Worker	X	NA	X	X	X	X	X	X	X	X
Future Industrial Worker										
Exposure to Soil	X	NA	X	X	X	X	X	X	X	X
Exposure to Water[b]	X									
Future Excavation Worker	X	X	X	X	X	X	X	X	X	X
Future Recreational User	X	NA	–	X	–	X	X	–	X	–
Future On Site Resident										
Exposure to Soil	X	NA	X	X	X	X	X	X	X	X
Exposure to Water[b]	X									
Future Off Site Resident Exposure to Water[c]	X	NA	X	X	X	X	X	X	X	–

Notes:
Duplicate of Table ES.1 of the WAG 6 BRA.
Scenarios where risk exceeded the benchmark levels 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, the future teen recreational user results are used. For the future onsite resident, the results for exposure to a child are used.
[b] In the WAG 6 BHHRA, the risk from exposure to water was assessed on an area basis; therefore, these risks are not summed with those from exposure to soil. Additionally, the BHHRA assessed risks from use of water drawn from the RGA separately from use of water drawn from the McNairy Formation. The result reported here is for use of water from the RGA.
[c] Based on results of contaminant transport modeling. X indicates that the location contains a source of unacceptable offsite contamination.
[d] Even if contribution from lead is not considered, these scenarios remain of concern.
[e] If contribution from lead is not considered, then the total HI falls below 1, and the scenario is not of concern.

Exhibit 1.2. Summary of risk results for WAG 6 without lead as a COPC

Area	Use Scenario									
	Current Worker		Future Worker		Excavation Worker		Recreational User [a]		Rural Resident [a]	
	ELCR	HI	ELCR	HI	ELCR	HI	ELCR	HI	ELCR	HI
WAG 6 (soil only)	3.3×10^{-4}	1.8	3.3×10^{-4}	1.8	2.6×10^{-3}	3.25	1.1×10^{-4}	<0.1	1.3×10^{-2}	89.6
WAG 6 (RGA only)	NA	NA	2.7×10^{-3}	37.7	NA	NA	NA	NA	6.4×10^{-2}	475
WAG 6 (McN only)	NA	NA	4.5×10^{-3}	20.6	NA	NA	NA	NA	3.5×10^{-2}	224
Sector 1	NA	NA	NA	NA	2.0×10^{-6}	1.7	NA	NA	NA	NA
Sector 2	1.7×10^{-5}	0.4	1.7×10^{-5}	0.4	1.6×10^{-4}	1.2	4.7×10^{-7}	<0.1	8.1×10^{-4}	10.6
Sector 3	8.5×10^{-5}	0.3	8.5×10^{-5}	0.3	1.2×10^{-4}	0.7	5.9×10^{-6}	<0.1	8.2×10^{-3}	13.3
Sector 4	3.7×10^{-6}	1.0	3.7×10^{-6}	1.0	3.6×10^{-4}	1.6	1.5×10^{-7}	<0.1	1.9×10^{-4}	24.8
Sector 5	4×10^{-4}	1.8	4×10^{-4}	1.8	2.3×10^{-4}	1.6	2.5×10^{-5}	<0.1	1.4×10^{-2}	85.5
Sector 6	1.1×10^{-3}	1.2	1.1×10^{-3}	1.2	5.5×10^{-4}	2.1	3.2×10^{-5}	<0.1	5.0×10^{-2}	119
Sector 7	1.2×10^{-4}	1.6	1.2×10^{-4}	1.6	1.3×10^{-4}	1.7	5.1×10^{-7}	<0.1	1.5×10^{-3}	53.6
Sector 8	2.4×10^{-4}	1.0	2.4×10^{-4}	1.0	2.3×10^{-4}	4.4	1.3×10^{-6}	<0.1	2.1×10^{-3}	18.8
Sector 9	5.2×10^{-6}	1.3	5.2×10^{-6}	1.3	1.5×10^{-4}	2.7	2.7×10^{-7}	<0.1	2.7×10^{-4}	36.8

Notes:
Taken from the WAG BHHRA.
NA = No land use scenarios of concern or media not present to assess use scenario.

[a] Values are for the child.

risk estimates for the current and future industrial worker. The results of this analysis are presented in Exhibits 1.3 through 1.6. Most important to the GWOU BHHRA is the information shown in Exhibits 1.4 and 1.6. In these exhibits, the ELCR and systemic toxicity posed to the future worker through use of groundwater are seen to vary little as the various uncertainties are considered. In fact, the ELCRs posed by use of RGA and McNairy Formation groundwater vary by factors of 1.4 and 2.6, respectively, and the systemic toxicity (as indicated by the value of HI) does not vary at all once the effect of lead is removed. [Note that the effect of lead is due to the use of a provisional reference dose (RfD). Please see Sect. 6 of this BHHRA for a discussion of the effect of the use of this provisional value.]

- Screening level modeling indicated that WAG 6 is a potential source of off-site groundwater contamination. As shown in Exhibits 1.7, 1.8, and 1.9, modeling identified WAG 6 as being the potential source of unacceptable concentrations of nine organic compounds and four inorganic chemicals. The most prominent organic compounds were TCE and its breakdown products. [Note that the modeling did not consider potential sources of TCE and ^{99}Tc located in the RGA below WAG 6. The modeling did not consider these because it was determined *a priori* that off-site sources of both of these contaminants existed in the RGA below WAG 6. (See pages 1-22 and 1-24 of the WAG 6 BHHRA.)]

1.2.1.2 WAG 27 (from material in DOE 1999b)

In 1998, the DOE conducted a RI/RFI for WAG 27. WAG 27 includes SWMUs 1, 91, 196, and the C-720 Area at the PGDP. The overall purpose of this activity was to determine the presence, nature, and extent of contamination at SWMUs 1, 91, 196, and the C-720 Area. The primary focus of the investigation was to collect sufficient information about contamination in surface soil, subsurface soil, surface water, sediment and the shallow groundwater of the UCRS to support an assessment of risks to human health and the environment and the selection of actions to reduce these risks. In addition, contamination in the RGA and McNairy Formation was characterized to determine if contamination there acted as a secondary source of contamination to groundwater. The SWMUs that were assessed for risk to human health and the environment were: SWMUs 1, 91, 196, and the C-720 Area.

To facilitate data aggregation and to focus results on specific areas, this baseline risk assessment derived risk estimates for the following SWMUs or areas. The SWMUs and areas and their definitions are as follows.

- SWMU 1 – the C-747-C Oil Land Farm
- SWMU 91 – the UF6 Cylinder Drop Test Area
- SWMU 196 - the C-746-A Septic System
- C-720 Area (includes SWMU 209 - the compressor pit sump)

Consistent with regulatory guidance and agreements contained in the Methods Document, the BHHRA evaluated scenarios that encompassed current use and several hypothetical future uses of the WAG 27 SWMUs and the areas to which contaminants from the WAG 27 SWMUs may migrate. These scenarios are listed below.

- Current on-site industrial – direct contact with sediment and surface soil (soil found 0 to 1 ft bgs).

- Future on-site industrial – direct contact with sediment, surface soil, and use of groundwater drawn from aquifers below the WAG 27 SWMUs.

- Future on-site excavation scenario – direct contact with surface soil combined with subsurface soil (soil found 0 to 15 ft bgs).

Exhibit 1.3. Summary of risk and uncertainty results for current industrial worker for WAG 6

Location	Default ELCR[a]	Site-specific ELCR[b]	Default ELCR minus common laboratory contaminants	Default ELCR calculated using EPA default dermal absorption values[c]	Default ELCR minus analytes infrequently detected	Lower-bound ELCR[d]
WAG 6	3.3×10^{-4}	2.1×10^{-5}	3.3×10^{-4}	4.1×10^{-5}	3.3×10^{-4}	2.6×10^{-6}
Sector 1	NV	NV	NV	NV	NV	NV
Sector 2	1.7×10^{-5}	1.1×10^{-6}	1.7×10^{-5}	3.8×10^{-6}	1.7×10^{-5}	2.4×10^{-7}
Sector 3	8.5×10^{-5}	5.4×10^{-6}	8.5×10^{-5}	3.0×10^{-5}	8.5×10^{-5}	1.9×10^{-6}
Sector 4	3.7×10^{-6}	2.3×10^{-7}	3.7×10^{-6}	5.9×10^{-7}	3.7×10^{-6}	3.8×10^{-8}
Sector 5	4.0×10^{-4}	2.6×10^{-5}	4.0×10^{-4}	4.5×10^{-5}	4.0×10^{-4}	2.9×10^{-6}
Sector 6	1.1×10^{-3}	7.3×10^{-5}	1.1×10^{-3}	1.5×10^{-4}	1.1×10^{-3}	9.8×10^{-6}
Sector 7	1.2×10^{-4}	7.9×10^{-6}	1.2×10^{-4}	5.7×10^{-6}	1.2×10^{-4}	3.7×10^{-7}
Sector 8	2.4×10^{-4}	1.5×10^{-5}	2.4×10^{-4}	9.8×10^{-6}	2.4×10^{-4}	6.2×10^{-7}
Sector 9	5.2×10^{-6}	3.3×10^{-7}	5.2×10^{-6}	3.7×10^{-6}	5.2×10^{-6}	2.3×10^{-7}

Notes:

Duplicate of Table ES.12 of the WAG 6 BHHRA.

NV indicates that a value is not available because the sector encompasses the area below the C-400 Building.

[a] These values were derived using the default exposure rates for the reasonable maximum exposure scenario approved by regulatory agencies.

[b] These values were derived using site-specific exposure rates for general maintenance workers at PGDP.

[c] The values were calculated using the soil dermal absorption rates suggested by EPA.

[d] These values were derived using site-specific exposure rates for general maintenance workers at PGDP and EPA default dermal absorption values and omitting contributions from common laboratory contaminants and infrequently detected analytes. The values should be used as a lower-bound estimates of risk when considering the appropriate actions to address contamination at WAG 6.

Exhibit 1.4. Summary of risk and uncertainty results for future industrial worker for WAG 6

Location	Default ELCR[a]	Site-specific ELCR[b]	Default ELCR minus common laboratory contaminants	Default ELCR calculated using EPA default dermal absorption values[c]	Default ELCR minus analytes infrequently detected	Lower-bound ELCR[d]
WAG 6 McNairy[e]	4.5×10^{-3}	4.5×10^{-3}	4.5×10^{-3}	4.5×10^{-3}	1.7×10^{-3}	1.7×10^{-3}
WAG 6 RGA[e]	2.7×10^{-3}	2.7×10^{-3}	2.7×10^{-3}	2.7×10^{-3}	2.1×10^{-3}	2.0×10^{-3}
WAG 6 soil	3.3×10^{-4}	3.3×10^{-4}	3.3×10^{-4}	4.1×10^{-5}	3.3×10^{-4}	4.1×10^{-5}
Sector 1	NV	NV	NV	NV	NV	NV
Sector 2	1.7×10^{-5}	1.7×10^{-5}	1.7×10^{-5}	3.8×10^{-6}	1.7×10^{-5}	3.8×10^{-6}
Sector 3	8.5×10^{-5}	8.5×10^{-5}	8.5×10^{-5}	3.0×10^{-5}	8.5×10^{-5}	3.0×10^{-5}
Sector 4	3.7×10^{-6}	3.7×10^{-6}	3.7×10^{-6}	5.9×10^{-7}	3.7×10^{-6}	5.9×10^{-7}
Sector 5	4.0×10^{-4}	4.0×10^{-4}	4.0×10^{-4}	4.5×10^{-5}	4.0×10^{-4}	4.5×10^{-5}
Sector 6	1.1×10^{-3}	1.1×10^{-3}	1.1×10^{-3}	1.5×10^{-4}	1.1×10^{-3}	1.5×10^{-4}
Sector 7	1.2×10^{-4}	1.2×10^{-4}	1.2×10^{-4}	5.7×10^{-6}	1.2×10^{-4}	5.7×10^{-6}
Sector 8	2.4×10^{-4}	2.4×10^{-4}	2.4×10^{-4}	9.8×10^{-6}	2.4×10^{-4}	9.8×10^{-6}
Sector 9	5.2×10^{-6}	5.2×10^{-6}	5.2×10^{-6}	3.7×10^{-6}	5.2×10^{-6}	3.7×10^{-6}

Notes:

Duplicate of Table ES.13 in the WAG 6 BHHRA.

NV indicates that a value is not available because the sector encompasses the area below the C-400 Building.

[a] These values were derived using the default exposure rates for the reasonable maximum exposure scenario approved by regulatory agencies.

[b] These values were also derived using the default exposure rates for the reasonable maximum exposure scenario because it is unknown what the site-specific exposure rates may be in the future.

[c] These values were calculated using the soil dermal absorption rates suggested by EPA.

[d] These values were derived using default exposure rates for the reasonable maximum exposure scenario and EPA default dermal absorption values and omitting contributions from laboratory contaminants and infrequently detected analytes. The values should be used as lower-bound estimates of risk when considering the appropriate actions to address contamination at WAG 6.

[e] Values are for groundwater use by the future industrial worker.

Exhibit 1.5. Summary of systemic toxicity and uncertainty results for current industrial worker for WAG 6

Location	Default HI[a]	Default HI w/o lead	Site-specific HI w/o lead[b]	Default HI minus common laboratory contaminants w/o lead	Default HI calculated EPA default dermal absorption values w/o lead[c]	Default HI minus analytes infrequently detected w/o lead	Lower-bound Hid[d]
WAG 6	1,160	1.8	<1	1.8	<1	1.8	<1
Sector 1	NV	NV	NV	NV	NV	NV	NV
Sector 2	<1	<1	<1	<1	<1	<1	<1
Sector 3	<1	<1	<1	<1	<1	<1	<1
Sector 4	<1	<1	<1	<1	<1	<1	<1
Sector 5	1.8	1.8	<1	1.8	<1	1.8	<1
Sector 6	1.2	1.2	<1	1.2	<1	1.2	<1
Sector 7	1,890	1.6	<1	1.6	<1	1.6	<1
Sector 8	1.0	1.0	<1	1.0	<1	1.0	<1
Sector 9	1.3	1.3	<1	1.3	<1	1.3	<1

Notes:

Duplicate of Table ES.14 from the WAG 6 BHHRA.

NV indicates that a value is not available because the sector encompasses the area below the C-400 Building.

<1 indicates that the hazard index is less than the *de minimis* level.

[a] These values were derived using the default exposure rates for the reasonable maximum exposure scenario approved by regulatory agencies.

[b] These values were derived using site-specific exposure rates for general maintenance workers at PGDP.

[c] The values were calculated using the soil dermal absorption rates suggested by EPA.

[d] These values were derived using site-specific exposure rates for general maintenance workers at PGDP and EPA default dermal absorption values and omitting contributions from common laboratory contaminants and infrequently detected analytes. The values should be used as lower-bound estimates of risk when considering the appropriate actions to address contamination at WAG 6.

Exhibit 1.6. Summary of systemic toxicity and uncertainty results for future industrial worker for WAG 6

Location	Default HI[a]	Default HI w/o lead	Site-specific HI w/o lead[b]	Default HI minus common laboratory contaminants w/o lead	Default HI calculated using EPA default dermal absorption values w/o lead[c]	Default HI minus analytes infrequently detected w/o lead	Lower-bound HI[d]
WAG 6 McNairy[e]	11,500	20.6	20.6	20.6	20.6	20.6	20.6
WAG 6 RGA[e]	3,320	37.7	37.7	37.7	37.7	37.7	37.7
WAG 6 soil	1,160	1.8	1.8	1.8	<1	1.8	<1
Sector 1	NV	NV	NV	NV	NV	NV	NV
Sector 2	<1	<1	<1	<1	<1	<1	<1
Sector 3	<1	<1	<1	<1	<1	<1	<1
Sector 4	<1	<1	<1	<1	<1	<1	<1
Sector 5	1.8	1.8	1.8	1.8	<1	1.8	<1
Sector 6	1.2	1.2	1.2	1.2	<1	1.2	<1
Sector 7	1,890	1.6	1.6	1.6	<1	1.6	<1
Sector 8	1.0	1	1	1.0	<1	1.0	<1
Sector 9	1.3	1.3	1.3	1.3	<1	1.3	<1

Notes:

Duplicate of Table ES.15 from the WAG 6 BHHRA.

NV indicates that a value is not available because the sector encompasses the area below the C-400 Building.

<1 indicates that the hazard index is less than the *de minimis* level.

[a] These values were derived using the default exposure rates for the reasonable maximum exposure scenario approved by regulatory agencies.

[b] These values were also derived using the default exposure rates for the reasonable maximum exposure scenario because it is unknown what the site-specific exposure rates may be in the future.

[c] These values were calculated using the soil dermal absorption rates suggested by EPA.

[d] These values were derived using default exposure rates for the reasonable maximum exposure scenario and EPA default dermal absorption values and omitting contributions from laboratory contaminants and infrequently detected analytes. The values should be used as lower-bound estimates of risk when considering the appropriate actions to address contamination at WAG 6.

[e] Values are for groundwater use by the future industrial worker.

Exhibit 1.7. Comparison between maximum modeled concentrations at the PGDP fence boundary and residential use risk-based concentrations (RBCs) for WAG 6 sources

Contaminant[a]	Source[b]	Maximum Concentration[c]	Residential Use RBC[d]		Exceed?[e]
			Cancer	Systemic Toxicity	
Organic Chemicals (mg/L)					
1,1-Dichloroethene	Southeast; Area 4	4.14E-03	1.62E-06	1.34E-02	Cancer
1,2-Dichloroethene	West; Area 6	7.64E-02	NV	1.36E-02	ST
2-Methylnaphthalene	West; Area 6	1.27E-06	NV	NV	NC
2,4-Dinitrotoluene	Far North; Area 8	1.07E-01	7.69E-06	3.00E-03	Both
Acenaphthylene	Southwest; Area 5	3.18E-04	NV	NV	NC
Carbon tetrachloride	Southeast; Area 4	4.87E-04	2.07E-05	2.03E-04	Both
N-Nitroso-di-n-propylamine	Northeast; Area 2	2.17E-02	7.39E-07	NV	Cancer
Phenanthrene	Southwest; Area 5	1.02E-04	NV	NV	NC
Tetrachloroethene	Southeast; Area 4	6.44E-04	5.91E-05	9.87E-03	Cancer
trans-1,2-Dichloroethene	West; Area 6	7.64E-02	NV	3.02E-02	ST
Trichloroethene	Southeast; Area 4	5.00E+00	2.01E-04	7.86E-03	Both
Vinyl chloride	Southeast; Area 4	1.14E-03	2.04E-06	NV	Cancer
Inorganic Chemicals (mg/L)					
Antimony	Northwest; Area 7	5.73E-03	NV	5.64E-04	ST
Copper	Far North; Area 8	1.50E-01	NV	6.02E-02	ST
Chromium	RGA	6.91E-05	NV	7.05E-03	No
Cobalt	RGA	2.74E-02	NV	9.06E-02	No
Iron	RGA	8.18E+01	NV	4.49E-01	ST
Manganese	RGA	5.71E-01	NV	6.81E-02	ST
Thallium	Southwest; Area 5	4.74E-01	NV	NV	NC
Radionuclides (pCi/L)					
Americium-241	Far North; Area 8	2.97E-21	1.18E-02	NA	No
Neptunium-237	Far North; Area 8	4.30E-06	1.29E-02	NA	No
Plutonium-239	Southeast; Area 4	1.22E-08	1.22E-02	NA	No
Technetium-99	Northwest; Area 7	5.35E-06	2.76E+00	NA	No
Thorium-230	Southeast; Area 4	2.23E-23	1.03E-01	NA	No
Uranium-234	Far North; Area 8	1.34E-06	8.70E-02	NA	No
Uranium-235	Southwest; Area 5	7.95E-07	8.21E-02	NA	No
Uranium-238	Far North; Area 8	1.04E-05	9.04E-02	NA	No

[a] All contaminants with an identified source and a modeled concentration are listed.
[b] Area in which the source contributing the maximum modeled concentration is located.

Exhibit 1.7 (continued)

[c] Maximum modeled contaminant concentration among all sources modeled.

[d] All residential use risk-based concentrations were taken from Table 2 in Appendix 1 of *Methods for Conducting Human Health Risk Assessments and Risk Evaluations at the Paducah Gaseous Diffusion Plant* (1996b). All cancer RBCs are based on a 40 year exposure; all systemic toxicity RBCs are based on chronic exposure by a child aged 1 to 7. 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 1×10^{-7} because more than 5 contaminants are present. Target HI for all systemic toxicity RBCs is 0.1 because more than 5 contaminants are present. "NV" indicates an RBC for the endpoint is not available because toxicity information is lacking. "NA" indicates that the endpoint is no applicable (radionuclides only). The RBC for chromium is for exposure to chromium VI. The RBCs for neptunium-237, uranium-235, and uranium-238 include contributions from short-lived daughters.

[e] "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.
"NC" indicates that a comparison could not be performed because neither a cancer nor a systemic toxicity RBC is available.
"No" indicates that neither RBC is exceeded by the maximum modeled concentration.

Exhibit 1.8. Summary of sources and maximum modeled concentrations for contaminants that have a source within the WAG 6 area that exceeds a residential use risk-based concentration (RBC)

Contaminant[a]	Source[b]	Maximum Concentration[c]	Residential Use RBC[d]		Exceed?[e]
			Cancer	Systemic Toxicity	
Organic Chemicals (mg/L)					
1,1-Dichloroethene	Southeast; Area 4	4.14E-03	1.62E-06	1.34E-02	Cancer
1,2-Dichloroethene	West; Area 6	7.64E-02	NV	1.36E-02	ST
2,4-Dinitrotoluene	Far North; Area 8	1.07E-01	7.69E-06	3.00E-03	Both
Carbon tetrachloride	Southeast; Area 4	4.87E-04	2.07E-05	2.03E-04	Both
N-Nitroso-di-n-propylamine	Northeast; Area 2	2.17E-02	7.39E-07	NV	Cancer
Tetrachloroethene	Southeast; Area 4	6.44E-04	5.91E-05	9.87E-03	Cancer
trans-1,2-Dichloroethene	West; Area 6	7.64E-02	NV	3.02E-02	ST
Trichloroethene	East; Area 3	2.91E-02	2.01E-04	7.86E-03	Both
	Southeast; Area 4	5.00E+00			Both
	Southwest; Area 5	2.53E-01			Both
	West; Area 6	9.58E-03			Both
	Northwest; Area 7	4.92E-03			Cancer
Vinyl chloride	Southeast; Area 4	1.14E-03	2.04E-06	NV	Cancer
	Southwest; Area 5	8.04E-04			Cancer
Inorganic Chemicals (mg/L)					
Antimony	Northwest; Area 7	5.73E-03	NV	5.64E-04	ST
Copper	Far North; Area 8	1.50E-01	NV	6.02E-02	ST
Iron	RGA	8.18E+01	NV	4.49E-01	ST
Manganese	RGA	5.71E-01	NV	6.81E-02	ST

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

[b] Maximum modeled concentration reported for sources within a area. Areas not listed do not contain a source of the contaminant.

[c] Maximum modeled contaminant concentration for source.

[d] All residential use risk-based concentrations were taken from Table 2 in Appendix 1 of *Methods for Conducting Human Health Risk Assessments and Risk Evaluations at the Paducah Gaseous Diffusion Plant* (1996b). All cancer RBCs are based on a 40-year exposure; all systemic toxicity RBCs are based on chronic exposure by a child aged 1 to 7. 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 1×10^{-7} because more than 5 contaminants are present. Target HI for all systemic toxicity RBCs is 0.1 because more than 5 contaminants are present. "NV" indicates an RBC for the endpoint is not available because toxicity information is lacking.

[e] "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.

Exhibit 1.9. Summary of time required to reach maximum modeled concentrations at the PGDP fence boundary for contaminant sources within the WAG 6 area that contribute maximum contaminant concentrations exceeding residential use risk-based concentrations (RBCs)

Contaminant[a]	Source[b]	Maximum Concentration[c]	Year[d]
Organic Chemicals (mg/L)			
1,1-Dichloroethene	Southeast; Area 4	4.14E-03	62
1,2-Dichloroethene	West; Area 6	7.64E-02	21
2,4-Dinitrotoluene	Far North; Area 8	1.07E-01	47
Carbon tetrachloride	Southeast; Area 4	4.87E-04	386
N-Nitroso-di-n-propylamine	Northeast; Area 2	2.17E-02	24
Tetrachloroethene	Southeast; Area 4	6.44E-04	285
trans-1,2-Dichloroethene	West; Area 6	7.64E-02	21
Trichloroethene	East; Area 3	2.91E-02	105
	Southeast; Area 4	5.00E+00	105
	Southwest; Area 5	2.53E-01	105
	West; Area 6	9.58E-03	105
	Northwest; Area 7	4.92E-03	89
Vinyl chloride	Southeast; Area 4	1.14E-03	54
	Southwest; Area 5	8.04E-04	54
Inorganic Chemicals (mg/L)			
Antimony	Northwest; Area 7	5.73E-03	707
Copper	Far North; Area 8	1.50E-01	9510
Iron	RGA	8.18E+01	377
Manganese	RGA	5.71E-01	633

[a] Only contaminants which have a maximum modeled contaminant concentration over all sources that exceed either RBC are listed.
[b] Maximum modeled concentration reported for sources within a area. Only areas 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.

- Future on-site recreational user – direct contact with sediment and consumption of game exposed to contaminated surface soil.

- Future off-site recreational user – direct contact with surface water impacted by contaminants migrating from sources and consumption of game exposed to this surface water.

- Future on-site rural resident – direct contact with surface soil at and use of groundwater drawn from aquifers below the WAG 27 SWMUs, including consumption of vegetables that were posited to be raised in this area.

- Future off-site rural resident – use in the home of groundwater drawn from the RGA at the DOE property boundary.

Note that this report also contains a BERA for nonhuman receptors that may come into contact with contaminated media at or migrating from sources in the WAG 27 area. Results from this BERA are not discussed here.

Major conclusions and observations of the BHHRA are as follows.

- For all SWMUs, the cumulative human health ELCR and systemic toxicity exceed the accepted standards of the KDEP and 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 excess lifetime cancer risk of 1×10^{-6} or a cumulative HI of 1) are summarized in Exhibit 1.10. More detailed information is in Exhibit 1.11.

- ELCR and systemic toxicity (HI in Exhibit 1.11) for use of groundwater drawn from the RGA and McNairy Formation were greater than upper end of the EPA risk range (i.e., 1×10^{-4}) for both the future industrial worker and potential future on-site resident. Contaminants in groundwater driving risk and systemic toxicity varied between SWMUs and groundwater source. Over all SWMUs and groundwater sources, arsenic, beryllium, 1,1-dichloroethene (DCE), ^{241}Am, ^{222}Rn, ^{99}Tc, ^{237}Np, and ^{238}U were determined to drive ELCR, and arsenic, chromium, iron, manganese, uranium, and TCE were determined to drive systemic toxicity.

- Because there was considerable uncertainty in some of the exposure parameters, exposure pathways and toxicity values, a quantitative uncertainty analysis was performed. In this analysis, approved toxicity values and site-specific exposure parameters and exposure pathways were used to calculate risk estimates for the current and future industrial worker. The results of this analysis are presented in Exhibits 1.12 through 1.15. Most important to the GWOU BHHRA is the information shown in Exhibits 1.13 and 1.15. In these exhibits, the ELCR and systemic toxicity posed to the future worker through use of groundwater are seen to vary little as the various uncertainties are considered. In fact, the ELCRs posed by use of RGA groundwater vary by factors approximately equal to 1.1, and the systemic toxicity varies by factors ranging from 1.3 to 2.5 once the effect of lead is removed. (Note that the effect of lead is due to the use of a provisional RfD. Please see Sect. 6 of this BHHRA for a discussion of the effect of the use of this provisional value.)

- Screening level modeling indicated that WAG 27 is a potential source of off-site groundwater contamination. As shown in Exhibits 1.16, 1.17, and 1.18, modeling identified WAG 27 as being the potential source of unacceptable concentrations of four organic compounds and five inorganic chemicals. The most prominent organic compounds were TCE and its breakdown products.

Exhibit 1.10. Land uses of concern for WAG 27

Scenario	Location			
	SWMU 1	SWMU 91	SWMU 196	C-720 Area
Results for excess lifetime cancer risk:				
Current Onsite Industrial Worker				
Exposure to Soil	NA	NA	X	NA
Exposure to Sediment	X	X	X	NA
Future Onsite Industrial Worker				
Exposure to Soil	NA	NA	X	NA
Exposure to Sediment	X	X	X	NA
Exposure to Groundwater[b]	X	X	–	X
Future Onsite Excavation Worker				
Exposure to Soil	X	X	X	X
Future Onsite Recreational User				
Exposure to Game	NA	NA	–	NA
Exposure to Sediment	X	X	X	NA
Future Offsite Recreational User				
Exposure to Surface Water	–	–	–	–
Future Onsite Rural Resident				
Exposure to Soil	NA	NA	X	NA
Exposure to Groundwater[b]	X	X	–	X
Future Offsite Rural Resident				
Exposure to Groundwater[c]	X	X	X	X
Results for systemic toxicity[a]:				
Current Onsite Industrial Worker				
Exposure to Soil	NA	NA	–	NA
Exposure to Sediment	X	X	X	NA
Future Onsite Industrial Worker				
Exposure to Soil	NA	NA	–	NA
Exposure to Sediment	X	X	X	NA
Exposure to Groundwater[b]	X	X	–	X
Future Onsite Excavation Worker				
Exposure to Soil	X	X	X	–
Future Onsite Recreational User				
Exposure to Game	NA	NA	–	NA
Exposure to Sediment	X	X	X	NA
Future Offsite Recreational User				
Exposure to Surface Water[c]	–	–	–	–
Future Onsite Rural Resident				
Exposure to Soil	NA	NA	X	NA
Exposure to Groundwater[b]	X	X	–	X
Future Offsite Rural Resident				
Exposure to Groundwater[c]	X	X	X	X

Notes:
Duplicate of Table ES.2 in the WAG 27 BHHRA.
Scenarios where risk exceeded the benchmark levels 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 onsite resident, the child results are used.
[b] The BHHRA assessed risks from use of water drawn from the RGA separately from use of water drawn from the McNairy Formation. The value reported here is for use of water from the RGA.
[c] Based on results of contaminant transport modeling. X indicates that the location contains one or more sources of offsite contamination that exceeded benchmark levels and – indicates that the location is not a source of offsite contamination.

Exhibit 1.11. Summary of risk results for WAG 27 without lead as a COPC

Area	Use Scenario									
	Current Worker		Future Worker		Excavation Worker		Recreational User [a]		Rural Resident [a]	
	ELCR	HI	ELCR	HI	ELCR	HI	ELCR	HI	ELCR	HI
SWMU 1 (soil only)	NA	NA	NA	NA	2.4×10^{-4}	1.92	NA	NA	NA	NA
SWMU 1 (sediment)	1.3×10^{-4}	1.7	1.3×10^{-4}	1.7	NA	NA	1.7×10^{-4}	3.36	NA	NA
SWMU 1 (RGA only)	NA	NA	1.9×10^{-3}	14.2	NA	NA	NA	NA	1.6×10^{-2}	152
SWMU 1 (McN only)	NA	NA	3.9×10^{-4}	2.99	NA	NA	NA	NA	2.8×10^{-3}	32.3
SWMU 91 (soil only)	NA	NA	NA	NA	1.5×10^{-4}	2.03	NA	NA	NA	NA
SWMU 91 (sediment)	5.8×10^{-4}	1.96	5.8×10^{-4}	1.96	NA	NA	2.3×10^{-4}	4.06	NA	NA
SWMU 91 (RGA only)	NA	NA	1.0×10^{-3}	4.24	NA	NA	NA	NA	8.2×10^{-3}	48.1
SWMU 91 (McN only)	NA	NA	6.9×10^{-4}	0.334	NA	NA	NA	NA	4.4×10^{-3}	3.88
SWMU 196 (soil only)	4.8×10^{-6}	0.521	4.8×10^{-6}	0.521	5.8×10^{-4}	3.0	1.7×10^{-7}	<0.1	3.7×10^{-4}	23.9
SWMU 196 (sediment)	8.7×10^{-5}	2.1	8.7×10^{-5}	2.1	NA	NA	1.3×10^{-4}	4.68	NA	NA
SWMU 196 (RGA only)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
SWMU 196 (McN only)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
C-720 (soil only)	NA	NA	NA	NA	7.9×10^{-5}	0.388	NA	NA	NA	NA
C-720 (sediment)	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
C-720 (RGA only)	NA	NA	6.0×10^{-4}	3.03	NA	NA	NA	NA	6.0×10^{-3}	47.5
C-720 (McN only)	NA	NA	6.6×10^{-4}	9.75	NA	NA	NA	NA	4.9×10^{-3}	105

Notes:
Taken from the WAG 27 BHHRA.
NA = No land use scenarios of concern or media not present to assess use scenario.

[a] Values reported are for the child.

Exhibit 1.12. Summary of risk and uncertainty results for the current industrial worker for WAG 27

Location	Default ELCR[a]	Site-specific ELCR[b]	Default ELCR minus common laboratory contaminants[c]	Default ELCR calculated using EPA default dermal absorption values[d]	Default ELCR minus analytes infrequently detected[e]	Lower-bound ELCR[f]
SWMU 1 (sediment)	1.3×10^{-4}	8.3×10^{-6}	1.3×10^{-4}	2.7×10^{-5}	1.3×10^{-4}	1.6×10^{-6}
SWMU 91 (sediment)	5.8×10^{-4}	3.7×10^{-5}	5.8×10^{-4}	4.7×10^{-4}	5.8×10^{-4}	3.0×10^{-5}
SWMU 196 (sediment)	8.7×10^{-5}	8.7×10^{-5}	8.7×10^{-5}	1.1×10^{-5}	8.7×10^{-5}	9.3×10^{-6}
SWMU 196 (soil)	4.8×10^{-6}	4.8×10^{-6}	4.8×10^{-6}	4.8×10^{-6}	4.8×10^{-6}	4.8×10^{-6}

Notes:
Duplicate of Exhibit 1.64a from the WAG 27 BHHRA.

[a] These values are identical to the values presented in Exhibit 1.27 in the WAG 27 BHHRA.
[b] These values are identical to the values presented in Exhibit 1.62 in the WAG 27 BHHRA.
[c] These values are identical to the values presented in Table 1.110 in the WAG 27 BHHRA.
[d] These values are identical to the values presented in Exhibit 1.61 in the WAG 27 BHHRA.
[e] These values are identical to the values presented in Table 1.108 in the WAG 27 BHHRA.
[f] These values were derived using site-specific exposure rates for general maintenance workers at PGDP and EPA default dermal absorption values and omitting contributions from common laboratory contaminants and infrequently detected analytes.

Exhibit 1.13. Summary of risk and uncertainty results for the future industrial worker for WAG 27

Location	Default ELCR[a]	Site-specific ELCR[b]	Default ELCR minus common laboratory contaminants[c]	Default ELCR calculated using EPA default dermal absorption values[d]	Default ELCR minus analytes infrequently detected[e]	Lower-bound ELCR[f]
SWMU 1 (RGA)	1.9×10^{-3}	1.9×10^{-3}	1.9×10^{-3}	1.9×10^{-3}	1.9×10^{-3}	1.7×10^{-3}
SWMU 91 (RGA)	1.0×10^{-3}	1.0×10^{-3}	1.0×10^{-3}	1.0×10^{-3}	1.0×10^{-3}	9.6×10^{-4}
C-720 (RGA)	6.0×10^{-4}	6.0×10^{-4}	6.0×10^{-4}	6.0×10^{-4}	5.9×10^{-4}	5.3×10^{-4}

Notes:
Duplicate of Exhibit 1.64b from the WAG 27 BHHRA.

[a] These values are identical to the values presented in Exhibit 1.37 in the WAG 27 BHHRA.
[b] These values are identical to the values presented in Exhibit 1.62 in the WAG 27 BHHRA.
[c] These values are identical to the values presented in Table 1.110 in the WAG 27 BHHRA.
[d] These values are identical to the values presented in Exhibit 1.61 in the WAG 27 BHHRA.
[e] These values are identical to the values presented in Table 1.108 in the WAG 27 BHHRA.
[f] These values were derived using site-specific exposure rates and default EPA dermal absorption rates and omitting contributions from common laboratory contaminants and infrequently detected analytes.

Exhibit 1.14. Summary of systemic toxicity and uncertainty results for the current industrial worker for WAG 27

Location	Default HI[a]	Default HI w/o lead[b]	Site-specific HI w/o lead[c]	Default HI calculated EPA default dermal absorption values w/o lead[d]	Default HI minus analytes infrequently detected w/o lead[e]	Lower-bound Hif
SWMU 1 (sediment)	1,160	1.71	<1	<1	1.71	<1
SWMU 91 (sediment)	1,190	1.96	<1	<1	1.96	<1
SWMU 196 (sediment)	2,000	2.1	2.1	<1	2.1	<1
SWMU 196 (soil)	3,160	<1	<1	<1	<1	<1

Notes:
Duplicate of Exhibit 1.65a from the WAG 27 BHHRA.

[a] These values are identical to the values presented in Exhibit 1.25 in the WAG 27 BHHRA.
[b] These values are identical to the values presented in Table 1.94 in the WAG 27 BHHRA.
[c] These values are identical to the values presented in Exhibit 1.63 in the WAG 27 BHHRA.
[d] These values are identical to the values presented in Exhibit 1.61 in the WAG 27 BHHRA.
[e] These values are identical to the values in Table 1.108 in the WAG 27 BHHRA.
[f] These values were derived using site-specific exposure rates for general maintenance workers at PGDP and EPA default dermal absorption rates and omitting infrequently detected analytes.

Exhibit 1.15. Summary of systemic toxicity and uncertainty results for the future industrial worker for WAG 27

Location	Default HI [a]	Default HI w/o lead [b]	Site-specific HI w/o lead [c]	Default HI calculated EPA default dermal absorption values w/o lead [d]	Default HI minus analytes infrequently detected w/o lead [e]	Lower-bound HI [f]
SWMU 1 (RGA)	5,390	14.2	14.2	14.2	14.2	11
SWMU 91 (RGA)	962	4.24	4.24	4.24	4.24	1.8
C-720 (RGA)	546	3.03	3.03	3.03	2.8	1.2

Notes:
Duplicate of Exhibit 1.65b of the WAG 27 BHHRA.

[a] These values are identical to the values presented in Exhibit 1.29 in the WAG 27 BHHRA.
[b] These values are identical to the values presented in Table 1.96 in the WAG 27 BHHRA.
[c] These values are identical to the default HI values (w/o lead) because site-specific exposure rates for the future industrial worker are unknown.
[d] These values are identical to the values presented in Exhibit 1.61 in the WAG 27 BHHRA.
[e] These values are identical to the values in Table 1.108 in the WAG 27 BHHRA.
[f] These values were derived using default exposure rates and default EPA dermal absorption rates and omitting infrequently detected analytes.

Exhibit 1.16. Comparison between maximum modeled concentrations at the PGDP fence boundary and residential use RBCs for WAG 27 sources

Contaminant[a]	Source[b]	Maximum Concentration[c]	Residential Use RBC[d]		Exceed?[e]
			Cancer	Systemic Toxicity	
Organic Chemicals (mg/L)					
Bis(2-ethylhexylphthalate)	C-720 subsurface soil	3.67E-12	3.1E-04	2.6E-02	No
Di-n-butylphthalate	SWMU 91, RGA	5.38E-06	NV	1.3E-01	No
Phenanthrene	SWMU 91, UCRS	3.85E-05	NV	NV	NC
Trans-1,2-dichloroethene	C-720 subsurface soil	7.22E+00	NV	4.0E-03	ST
Trichloroethene	C-720, RGA	3.1E+02	1.4E-04	1.2E-03	Both
Vinyl chloride	SWMU 1, UCRS	8.19E-02	1.7E-06	NV	Cancer
Xylenes	SWMU 1, UCRS	1.193E-04	NV	1.54E-01	No
Inorganic Chemicals (mg/L)					
Antimony	C-720, subsurface soil	2.55E-01	NV	5.6E-04	ST
Cadmium	C-720 subsurface soil	4.075E-06	NV	6.6E-04	No
Cobalt	C-720 subsurface soil	1.3E-02	NV	9.1E-02	No
Copper	C-720 subsurface soil	7.88E-03	NV	6.0E-02	No
Lead	SWMU 196, subsurface soil	6.963E-29	NV	1.5E-07	No
Manganese	SWMU 1, UCRS	1.73E-01	NV	6.7E-02	ST
Nickel	SWMU 196, subsurface soil	1.004E-23	NV	3.0E-02	No
Silver	C-720 subsurface soil	6.3E-02	NV	7.5E-03	ST
Thallium	C-720 subsurface soil	1.935E+00	NV	NV	NC
Vanadium	C-720 subsurface soil	2.39E-02	NV	9.3E-03	ST

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

[b] Media for each SWMU in which the source contributing the maximum modeled concentration is located.

[c] Maximum modeled contaminant concentration among all sources modeled.

[d] All residential use risk-based concentrations were taken from Table 1.40 in Appendix A of the WAG 27 BHHRA. All cancer RBCs are based on a 40 year exposure; all systemic toxicity RBCs are based on chronic exposure by a child aged 1 to 7. 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 1×10^{-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. "NA" indicates that the endpoint is no applicable (radionuclides only). The RBC for chromium is for exposure to chromium VI. The RBCs for neptunium-237, uranium-235, and uranium-238 include contributions from short-lived daughters.

[e] "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.
"NC" indicates that a comparison could not be performed because neither a cancer nor a systemic toxicity RBC is available.
"No" indicates that neither RBC is exceeded by the maximum modeled concentration.

Exhibit 1.17. Summary of sources and maximum modeled concentrations for contaminants that have a source within the WAG 27 area that exceeds a residential use risk-based concentration (RBC)

Contaminant[a]	Source[b]	Maximum Concentration[c]	Residential Use RBC[d]		Exceed?[e]
			Cancer	Systemic Toxicity	
Organic Chemicals (mg/L)					
Phenanthrene	SWMU 91, UCRS	3.85E-05	NV	NV	NC
Trans-1,2-dichloroethene	C-720 subsurface soil	7.22E+00	NV	4.0E-03	ST
Trichloroethene	SWMU 1, UCRS	2.044E+1	1.4E-04	1.2E-03	Both
	C-720 subsurface soil	12.7E-01	1.4E-4	1.2E-3	Both
	C-720 RGA	3.1E+02	1.4E-4	1.2E-3	Both
Vinyl chloride	SWMU 1, UCRS	8.19E-02	1.7E-06	NV	Cancer
	C-720 subsurface soil	3.63E-03	1.7E-06	NV	Cancer
Xylenes	SWMU 1, UCRS	1.193E-04	NV	1.54E-01	No
Inorganic Chemicals (mg/L)					
Antimony	SWMU 1, UCRS	6.43E-02	NV	5.6E-04	ST
	SWMU 1, RGA	1.67E-02	NV	5.6E-04	ST
	SWMU 91, UCRS	4.2E-02	NV	5.6E-04	ST
	SWMU 196, subsurface soil	1.826E-03	NV	5.6E-04	ST
	SWMU 196, surface soil	4.81E-04	NV	5.6E-04	No
	C-720, subsurface soil	2.55E-01	NV	5.6E-04	ST
Manganese	SWMU 1, UCRS	1.73E-01	NV	6.7E-02	ST
Silver	C-720 subsurface soil	6.3E-02	NV	7.5E-03	ST
Thallium	SWMU 196, subsurface soil	1.541E-03	NV	NV	NC
	C-720 subsurface soil	1.935E+00	NV	NV	NC
Vanadium	C-720 subsurface soil	2.39E-02	NV	9.3E-03	ST

[a] Only contaminants which have a maximum modeled contaminant concentration over all sources that exceed either RBC are listed.
[b] Maximum modeled concentration reported for sources. Areas not listed do not contain a source of the contaminant.
[c] Maximum modeled contaminant concentration for source.
[d] All residential use risk-based concentrations were taken from Table 1.40 in Appendix A of the WAG 27 BHHRA. All cancer RBCs are based on a 40-year exposure; all systemic toxicity RBCs are based on chronic exposure by a child aged 1 to 7. 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 1×10^{-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.
[e] "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.

Exhibit 1.18. Summary of time required to reach maximum modeled concentrations at the PGDP fence boundary for contaminant sources within the WAG 27 area that contribute maximum contaminant concentrations exceeding residential use risk-based concentrations (RBCs)

Contaminant[a]	Source[b]	Maximum Concentration[c]	Year[d]
Organic Chemicals (mg/L)			
Phenanthrene	SWMU 91, UCRS	3.85E-05	4,877
Trans-1,2-dichloroethene	C-720 subsurface soil	7.22E+00	25
Trichloroethene	SWMU 1, UCRS	2.044E+01	120
	C-720 subsurface soil	12.7E-01	72
	C-720, RGA	3.1E+02	9.2
Vinyl chloride	SWMU 1, UCRS	8.19E-02	57
	C-720 subsurface soil	3.63E-03	54
Xylenes	SWMU 1, UCRS	1.193E-04	159
Inorganic Chemicals (mg/L)			
Antimony	SWMU 1, UCRS	6.43E-02	794
	SWMU 1, RGA	1.67E-02	7
	SWMU 91, UCRS	4.2E-02	498
	SWMU 196, subsurface soil	1.826E-03	6,543
	C-720, subsurface soil	2.55E-01	229
Manganese	SWMU 1, UCRS	1.73E-01	2,334
Silver	C-720 subsurface soil	6.3E-02	847
Thallium	SWMU 196, subsurface soil	1.541E-03	394
	C-720 subsurface soil	1.935E+00	31
Vanadium	C-720 subsurface soil	2.39E-02	3,797

[a] Only contaminants which have a maximum modeled contaminant concentration over all sources that exceed either RBC are listed.
[b] Maximum modeled concentration reported for sources. Only areas 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.

1.2.1.3 WAG 28 (from material in DOE 2000a)

In 1999, DOE conducted an RI/RCRA Facility Investigation for WAG 28. WAG 28 includes SWMUs 99, 193, and 194 and AOC 204 at the 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 99, 193, and 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 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 RGA and McNairy Formation groundwater was characterized to determine if contamination in the sites acted as a secondary source of contamination to groundwater. The sites that were assessed for risk to human health and the environment were SWMUs 99a, 99b, 193a, 193b, 193c, and 194 and AOC 204.

To facilitate data aggregation and to focus results on specific areas, this baseline risk assessment derived risk estimates for the following SWMUs or areas. The SWMUs and areas and their definitions are as follows.

- SWMU 99a – the former C-745 Kellogg Buildings.
- SWMU 99b – the former septic tank and leach field used by the Kellogg Buildings.
- SWMU 193a – the former Millwright Shop.
- SWMU 193b – the former Pipe Fabrication Shop.
- SWMU 193c – the former location of temporary building used during the construction of PGDP and a leach field.
- SWMU 194 – the former site of the administrative portion of the McGraw construction facilities and two leach fields.
- AOC 204 – the former staging area or construction debris area associated with the original construction of the plant.

Consistent with regulatory guidance and agreements contained in the Methods Document, the BHHRA evaluated scenarios that encompassed current use and several hypothetical future uses of the WAG 28 SWMUs and the areas to which contaminants from the WAG 28 SWMUs may migrate. These scenarios are listed below.

- Current on-site industrial—direct contact with surface soil (0–1 ft below ground surface).
- Future on-site industrial—direct contact with surface soil and use of groundwater drawn from aquifers below WAG 28.
- Future on-site excavation scenario—direct contact with surface and subsurface soil (0–15 ft below ground surface).
- Future on-site 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.

Note that this report also contains a BERA for nonhuman receptors that may come into contact with contaminated media at or migrating from sources in the WAG 28 area. Results from this BERA are not discussed here.

Major conclusions and observations of the BHHRA are as follows.

- For all SWMUs, the cumulative human health ELCR and systemic toxicity exceed the accepted standards of the KDEP and 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 excess lifetime cancer risk of 1×10^{-6} or a cumulative HI of 1) are summarized in Exhibit 1.19. More detailed information is in Exhibit 1.20.

- ELCR and systemic toxicity (HI in Exhibit 1.20) for use of groundwater drawn from the RGA and McNairy Formation were greater than upper end of the EPA risk range (i.e., 1×10^{-4}) for both the future industrial worker and potential future on-site resident. Contaminants in groundwater driving risk and systemic toxicity varied between SWMUs and groundwater source. Over all SWMUs and groundwater sources, arsenic, beryllium, 1,1-DCE, ^{241}Am, ^{222}Rn, ^{99}Tc, ^{237}Np, and ^{238}U were determined to drive ELCR, and arsenic, chromium, iron, manganese, uranium, and TCE were determined to drive systemic toxicity.

- Because there was considerable uncertainty in some of the exposure parameters, exposure pathways and toxicity values, a quantitative uncertainty analysis was performed. In this analysis, approved toxicity values and site-specific exposure parameters and exposure pathways were used to calculate risk estimates for the current and future industrial worker. The results of this analysis are presented in Exhibits 1.21 through 1.24. Most important to the GWOU BHHRA is the information shown in Exhibits 1.22 and 1.24. In these exhibits, the ELCR and systemic toxicity posed to the future worker through use of groundwater are seen to vary little as the various uncertainties are considered. 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.

- The systemic toxicity varies by factors ranging from <1 to 32.1 once the effect of lead is removed. (Note that the effect of lead is due to the use of a provisional RfD. Please see Sect. 6 of this BHHRA for a discussion of the effect of the use of this provisional value.) The lower bound HI estimates still exceed an HI of 1 at several locations in RGA and McNairy groundwater.

- Screening level modeling indicated that WAG 28 is a potential source of off-site groundwater contamination. As shown in Exhibits 1.25, 1.26, and 1.27, modeling identified WAG 28 as being the potential source of unacceptable concentrations of one organic compound, four inorganic chemicals, and one radionuclide.

1.2.1.4 WAG 3 (from material in DOE 2000b)

In 1999, DOE conducted an RI/RCRA Facility Investigation for WAG 3. WAG 3 includes SWMUs 4, 5, and 6 at the PGDP in Paducah, Kentucky. The overall purpose of this investigation was to determine the presence, nature, and extent of contamination at SWMUs 4, 5, and 6. The primary focus of the RI was to collect sufficient information about surface soil, subsurface soil, and the shallow groundwater of the

Exhibit 1.19. Land uses of concern for WAG 28

Scenario	Site						
	SWMU 99a	SWMU 99b	SWMU 193a	SWMU 193b	SWMU 193c	SWMU 194	AOC 204
Excess lifetime cancer risk							
Current industrial worker							
Exposure to soil	X	NA	X	X	–	NA	NA
Future industrial worker							
Exposure to soil	X	NA	X	X	–	NA	NA
Exposure to RGA groundwater	X	X	X	X	X	NA	X
Exposure to McNairy groundwater	X	NA	X	–	X	NA	NA
Future on-site rural resident[f]							
Exposure to soil	X	NA	X	X	–	NA	NA
Exposure to RGA groundwater	X	X	X	X	X	NA	X
Exposure to McNairy groundwater	X	NA	X	X	X	NA	NA
Off-site rural resident							
Exposure to groundwater[e]	–	–	–	–	–	–	X[e]
Future recreational user[f]							
Exposure to soil	X	NA	X	–	–	NA	NA
Future excavation worker							
Exposure to soil	X	X	X	X	X	X	X
Systemic toxicity[a]							
Current industrial worker							
Exposure to soil	–	NA	–	X[b]	X[c]	NA	NA
Future industrial worker							
Exposure to soil	–	NA	–	X[b]	X[c]	NA	NA
Exposure to RGA groundwater	X[d]	X[b]	X[b]	X[b]	X[b]	NA	X[b]
Exposure to McNairy groundwater	X[b]	NA	X[b]	–	X[d]	NA	NA
Future on-site rural resident[a]							
Exposure to soil	X[b]	NA	X[b]	X[b]	X[d]	NA	NA
Exposure to RGA groundwater	X[d]	X[b]	X[b]	X[b]	X[b]	NA	X[b]
Exposure to McNairy groundwater	X[b]	NA	X[b]	X[b]	X[d]	NA	NA
Off-site rural resident							
Exposure to groundwater[e]	X[e]	–	X[e]	–	X[e]	X[e]	X[e]
Future recreational user[a]							
Exposure to soil	–	NA	–	–	X[c]	NA	NA
Future excavation worker							
Exposure to soil	X[d]	–	–	X[b]	X[d]	X[c]	–

Notes:
Taken from Table ES.2 in the WAG 28 BRA.
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 "–."
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.

Exhibit 1.20. Summary of risk results for WAG 28 without lead as a COPC

Area	Use Scenario									
	Current Worker		Future Worker		Excavation Worker		Recreational User [a]		Rural Resident [a]	
	ELCR	HI	ELCR	HI	ELCR	HI	ELCR	HI	ELCR	HI
SWMU 99a (RGA only)	NA	NA	5.6×10^{-4}	5.1	NA	NA	NA	NA	5.6×10^{-3}	97.3
SWMU 99a (McN only)	NA	NA	7.6×10^{-5}	1.6	NA	NA	NA	NA	1.7×10^{-3}	53.1
SWMU 99b (RGA only)	NA	NA	2.6×10^{-4}	7.0	NA	NA	NA	NA	2.3×10^{-3}	208
SWMU 193a (RGA only)	NA	NA	2.6×10^{-5}	1.6	NA	NA	NA	NA	2.4×10^{-3}	28.6
SWMU 193a (McN only)	NA	NA	1.1×10^{-6}	4.7	NA	NA	NA	NA	4.1×10^{-4}	59.9
SWMU 193b (RGA only)	NA	NA	4.4×10^{-5}	1.7	NA	NA	NA	NA	1.0×10^{-3}	55.5
SWMU 193b (McN only)	NA	NA	8.4×10^{-7}	<0.1	NA	NA	NA	NA	1.2×10^{-5}	2.7
SWMU 193c (RGA only)	NA	NA	1.0×10^{-5}	1.4	NA	NA	NA	NA	1.5×10^{-4}	80.7
SWMU 193c (McN only)	NA	NA	4.2×10^{-4}	9.9	NA	NA	NA	NA	4.0×10^{-3}	103
AOC 204 (RGA only)	NA	NA	1.3×10^{-3}	33.3	NA	NA	NA	NA	1.5×10^{-2}	279
SWMU 99a (soil only)	3.1×10^{-4}	0.5	3.1×10^{-4}	0.5	2.1×10^{-4}	1.5	2.7×10^{-6}	<0.1	1.4×10^{-1}	17.2
SWMU 99b (soil only)	NA	NA	NA	NA	2.1×10^{-4}	0.6	NA	NA	NA	NA
SWMU 193a (soil only)	1.5×10^{-5}	0.4	1.5×10^{-5}	0.4	1.7×10^{-4}	0.5	3.6×10^{-6}	<0.1	7.1×10^{-4}	6.3
SWMU 193b (soil only)	5.1×10^{-4}	5.3	5.1×10^{-4}	5.3	1.7×10^{-4}	1.8	4.4×10^{-8}	<0.1	3.0×10^{-3}	66.7
SWMU 193c (soil only)	1.7×10^{-10}	0.2	1.7×10^{-10}	0.2	1.7×10^{-4}	2.1	NV	<0.1	1.1E-9	3.0
SWMU 194 (soil only)	NA	NA	NA	NA	3.1×10^{-4}	0.6	NA	NA	NA	NA
AOC 204 (soil only)	NA	NA	NA	NA	1.1×10^{-6}	<0.1	NA	NA	NA	NA

Notes:
Taken from the WAG 28 BHHRA.
NA = No land use scenarios of concern or media not present to assess use scenario.

[a] Values reported are for the child.

Exhibit 1.21. Summary of risk and uncertainty results for the current industrial worker for WAG 28

Location	Default ELCR[a]	Default ELCR minus infequently detected analytes[b]	Default ELCR minus common laboratory contaminants[c]	Default ELCR omitting contaminants with provisional or withdrawn toxicity values[d]	ELCR computed using EPA Region 4 absorption factors[e]	Lower bound ELCR[f]
SWMU 99a (soil)	3.1×10^{-4}	3.0×10^{-4}	3.1×10^{-4}	7.5×10^{-5}	6.7×10^{-5}	5.8×10^{-5}
SWMU 193a (soil)	1.5×10^{-5}	1.5×10^{-5}	1.5×10^{-5}	9.2×10^{-6}	2.0×10^{-6}	1.2×10^{-6}
SWMU 193b (soil)	5.1×10^{-4}	5.1×10^{-4}	5.1×10^{-4}	2.7×10^{-9}	1.1×10^{-5}	2.7×10^{-9}
SWMU 193c (soil)	1.7×10^{-10}	1.7×10^{-10}	1.7×10^{-10}	1.7×10^{-10}	1.7×10^{-10}	1.7×10^{-10}

Notes: Duplicate of Exhibit 1.60 from the WAG 28 BHHRA.

[a] These values are identical to the values presented in Exhibit 1.19 in the WAG 28 BHHRA.
[b] These values are identical to the values presented in Table 1.82 in the WAG 28 BHHRA.
[c] These values are identical to the values presented in Table 1.84 in the WAG 28 BHHRA.
[d] These values are identical to the values presented in Table 1.86 in the WAG 28 BHHRA.
[e] These values are identical to the values presented in Exhibit 1.55 in the WAG 28 BHHRA.
[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.

Exhibit 1.22. Summary of risk and uncertainty results for the future industrial worker for WAG 28

Location	Default ELCR[a]	Default ELCRs minus infrequently detected contaminants[b]	Default ELCR minus laboratory contaminants[c]	Default ELCR omitting contaminants with provisional or withdrawn toxicity values[d]	ELCR computed using EPA Region 4 dermal toxicity values	Lower bound ELCR[e]
SWMU 99a (RGA)	5.6×10^{-4}	5.6×10^{-4}	5.6×10^{-4}	3.1×10^{-4}	NA	3.1×10^{-4}
SWMU 99a (McNairy)	7.6×10^{-5}	7.6×10^{-5}	7.6×10^{-5}	5.3×10^{-5}	NA	5.3×10^{-5}
SWMU 99b (RGA)	2.6×10^{-4}	2.6×10^{-4}	2.6×10^{-4}	1.5×10^{-4}	NA	1.5×10^{-4}
SWMU 193a (RGA)	2.6×10^{-5}	1.4×10^{-5}	2.6×10^{-5}	1.7×10^{-5}	NA	3.6×10^{-6}
SWMU 193a (McNairy)	1.1×10^{-6}	1.1×10^{-6}	1.1×10^{-6}	8.8×10^{-7}	NA	8.8×10^{-7}
SWMU 193b (RGA)	4.4×10^{-5}	4.4×10^{-5}	4.3×10^{-5}	1.7×10^{-5}	NA	1.7×10^{-5}
SWMU 193b (McNairy)	8.4×10^{-7}	8.4×10^{-7}	8.4×10^{-7}	1.5×10^{-7}	NA	1.5×10^{-7}
SWMU 193c (RGA)	1.0×10^{-5}	1.0×10^{-5}	1.0×10^{-5}	1.9×10^{-6}	NA	1.9×10^{-6}
SWMU 193c (McNairy)	4.2×10^{-4}	4.2×10^{-4}	4.2×10^{-4}	2.0×10^{-4}	NA	2.0×10^{-4}
AOC 204 (RGA)	1.3×10^{-3}	1.3×10^{-3}	1.3×10^{-3}	1.0×10^{-3}	NA	1.0×10^{-3}

Notes:
Duplicate of Exhibit 1.61 from the WAG 28 BHHRA.
NA = Not Applicable.

[a] These values are identical to the values presented in Exhibit 1.29 in the WAG 28 BHHRA.
[b] These values are identical to the values presented in Table 1.82 in the WAG 28 BHHRA.
[c] These values are identical to the values presented in Table 1.84 in the WAG 28 BHHRA.
[d] These values are identical to the values presented in Table 1.86 in the WAG 28 BHHRA.
[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.

Exhibit 1.23. Summary of systemic toxicity and uncertainty results for the current industrial worker for WAG 28

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 omitting contaminants with provisional or withdrawn toxicity values w/o lead[d]	HI computed using U.S. EPA Region 4 absorption factors w/o lead[e]	Lower bound HI[f]
SWMU 99a (soil)	<1	<1	<1	<1	<1	<1	<1
SWMU 193a (soil)	<1	<1	<1	<1	<1	<1	<1
SWMU 193b (soil)	5.25	<1	5.25	5.25	<1	<1	<1
SWMU 193c (soil)	3620	<1	<1	<1	<1	<1	<1

Notes:
Duplicate of Exhibit 1.58 from the WAG 28 BHHRA.

[a] These values are identical to the values presented in Exhibit 1.17 in the WAG 28 BHHRA.
[b] These values are identical to the values presented in Table 1.82 in the WAG 28 BHHRA.
[c] These values are identical to the values presented in Table 1.84 in the WAG 28 BHHRA.
[d] These values are identical to the values presented in Table 1.86 in the WAG 28 BHHRA.
[e] These values are identical to the values presented in Exhibit 1.55 in the WAG 28 BHHRA.
[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 1.24. Summary of systemic toxicity and uncertainty results for the future industrial worker for WAG 28

Location	Default HI[a]	Default HI w/o lead[a]	Default HIs minus infrequently detected contaminants w/o lead[b]	Default HI minus laboratory contaminants w/o lead[c]	Default HI omitting contaminants with provisional or withdrawn toxicity values w/o lead[d]	Lower bound HI[e]
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	<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	<1
SWMU 193b (McNairy)	<1	<1	<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

Notes:
Duplicate of Exhibit 1.59 in the WAG 28 BHHRA.

[a] These values are identical to the values presented in Exhibit 1.21 in the WAG 28 BHHRA.
[b] These values are identical to the values presented in Table 1.82 in the WAG 28 BHHRA.
[c] These values are identical to the values presented in Table 1.84 in the WAG 28 BHHRA.
[d] These values are identical to the values presented in Table 1.86 in the WAG 28 BHHRA.
[e] 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 1.25. Comparison between maximum modeled concentrations at the PGDP fence boundary and residential use RBCs for WAG 28 sources

Contaminant[a]	Source[b]	Maximum concentration[c]	Residential use RBC[d]		Exceed?[e]
			Cancer	Systemic toxicity	
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

[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 of the WAG 28 BHHRA. 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.
[e] "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.
[g] The RBCs for radionuclides include contributions from short-lived daughters.

Exhibit 1.26. Summary of sources and maximum modeled concentrations for contaminants that have a source within the WAG 28 area that exceeds a residential use risk-based concentration (RBC)

Contaminant[a]	Source[b]	Maximum concentration[c]	Residential Use RBC[d] Cancer	Residential Use RBC[d] Systemic toxicity	Exceed?[e]
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

[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 of the WAG 28 BHHRA. 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.
[e] "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.

Exhibit 1.27. Summary of time required to reach maximum modeled concentrations at the PGDP fence boundary for contaminant sources within the WAG 28 area that contribute maximum contaminant concentrations exceeding residential use risk-based concentrations (RBCs)

Contaminant[a]	Source[b]	Maximum concentration[c]	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	9854–10,834
Organic chemicals (mg/L)			
Trichloroethene	AOC 204 UCRS soil	1.428E+1[e]	111
Radionuclides (pCi/L)[g]			
Technetium-99	SWMU 99a surface soil	1.81E+2	1570

[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.

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 RGA and McNairy Formation groundwater was characterized to determine if contamination in the sites acted as a secondary source of contamination to groundwater. The sites that were assessed for risk to human health and the environment were SWMUs 4, 5, and 6.

To facilitate data aggregation and to focus results on specific areas, this baseline risk assessment derived risk estimates for the following SWMUs or areas. The SWMUs and areas and their definitions are as follows:

- SWMU 4 – C-747 Contaminated Burial Yard
- SWMU 5 – C-746-F Classified Burial Yard
- SWMU 6 – C-747-B Burial Ground

Consistent with regulatory guidance and agreements contained in the Methods Document, the BHHRA evaluated scenarios that encompassed current use and several hypothetical future uses of the WAG 3 SWMUs and the areas to which contaminants from the WAG 3 SWMUs may migrate. These scenarios are listed below.

- Current on-site industrial—direct contact with surface soil (0–1 ft below ground surface).

- Future on-site industrial—direct contact with surface soil and use of groundwater drawn from aquifers below WAG 3.

- Future on-site excavation scenario—direct contact with surface and subsurface soil (0–15 ft below ground surface).

- Future on-site 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 3, and ingestion of vegetables grown in this area.

- Off-site rural resident—use of groundwater drawn from aquifers at the PGDP fence boundary.

Note that this report also contains a BERA for nonhuman receptors that may come into contact with contaminated media at, or migrating from, sources in the WAG 3 area. Results from this BERA are not discussed here.

Major conclusions and observations of the BHHRA are as follows:

- For all SWMUs, the cumulative human health ELCR and systemic toxicity exceed the accepted standards of the KDEP and 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 ECLR of 1×10^{-6} or a cumulative HI of 1) are summarized in Exhibit 1.28. More detailed information is in Exhibit 1.29.

- ELCR and systemic toxicity (HI in Exhibit 1.29) for use of groundwater drawn from the RGA and McNairy Formation were greater than the upper end of the EPA risk range (i.e., 1×10^{-4}) for both the future industrial worker and potential future on-site resident. Contaminants in groundwater driving risk and systemic toxicity varied between SWMUs and groundwater source. Over all SWMUs and groundwater sources, arsenic, beryllium, 1,1-DCE, carbon tetrachloride, chloroform, trichloroethene, vinyl chloride, ^{99}Tc, and ^{226}Ra were determined to drive ELCR, and aluminum, arsenic, chromium, iron, manganese, uranium, vanadium, carbon tetrachloride, and TCE were determined to drive systemic toxicity.

Exhibit 1.28. Land uses of concern for WAG 3

	Site		
Land use scenario	**SWMU 4**	**SWMU 5**	**SWMU 6**
Systemic toxicity[a]			
Current industrial worker			
Exposure to soil	X[b]	–	–
Future industrial worker			
Exposure to soil	X[b]	–	–
Exposure to RGA groundwater	X[c]	X[c]	X[c]
Exposure to McNairy groundwater	X[c]	X[c]	X[c]
Future on-site rural resident[a]			
Exposure to soil	X[b]	X[b]	X[b]
Exposure to RGA groundwater	X[c]	X[c]	X[c]
Exposure to McNairy groundwater	X[c]	X[c]	X[c]
Off-site rural resident			
Exposure to groundwater[e]	X	X	X
Future recreational user[a]			
Exposure to soil	–	–	–
Future excavation worker			
Exposure to soil and waste	X[c]	X[b]	X[c]
Excess lifetime cancer risk			
Current industrial worker			
Exposure to soil	X	X	X
Future industrial worker			
Exposure to soil	X	X	X
Exposure to RGA groundwater	X	X	X
Exposure to McNairy groundwater	X	X	X
Future on-site rural resident[e]			
Exposure to soil	X	X	X
Exposure to RGA groundwater	X	X	X
Exposure to McNairy groundwater	X	X	X
Off-site rural resident[d]			
Exposure to groundwater	X[d]	–	–
Future recreational user[e]			
Exposure to soil	–	X	–
Future excavation worker			
Exposure to soil and waste	X	X	X

Notes:

Taken from Table ES.2 in the WAG 3 BRA.
Scenarios where risk exceeded the benchmark levels (HI of 1/ELCR of 1.0E-06) are marked with an "X."
Scenarios where risk did not exceed a benchmark level are marked with a "–."

[a] Results for a child are presented for systemic toxicity for the future recreational user and the future on-site rural resident.
[b] These scenarios are of concern even though lead was not detected.
[c] Lead is present, and the scenario is of concern whether or not the element is included in the assessment.
[d] Based on the results of contaminant transport modeling, "X" indicates that the location contains a source of unacceptable off-site contamination.
[e] Values for excess lifetime cancer risk for the future recreational user and the future on-site rural resident are for lifetime exposure.

Exhibit 1.29. Summary of risk results for WAG 3 without lead as a COPC

Area	Use Scenario									
	Current Worker		Future Worker		Excavation Worker		Recreational User [a]		Rural Resident [a]	
	ELCR	HI	ELCR	HI	ELCR	HI	ELCR	HI	ELCR	HI
SWMU 4 (RGA only)	NA	NA	4.7×10^{-4}	32.6	NA	NA	NA	NA	7.0×10^{-3}	487
SWMU 4 (McN only)	NA	NA	3.1×10^{-3}	75.9	NA	NA	NA	NA	$>1.0 \times 10^{-2}$	798
SWMU 5 (RGA only)	NA	NA	5.4×10^{-4}	26.8	NA	NA	NA	NA	3.9×10^{-3}	283
SWMU 5 (McN only)	NA	NA	1.2×10^{-3}	63.0	NA	NA	NA	NA	8.2×10^{-3}	680
SWMU 6 (RGA only)	NA	NA	2.3×10^{-4}	19.1	NA	NA	NA	NA	2.3×10^{-3}	223
SWMU 6 (McN only)	NA	NA	7.8×10^{-4}	41.7	NA	NA	NA	NA	5.7×10^{-3}	451
SWMU 4 (soil only)	5.4×10^{-4}	3.6	5.4×10^{-4}	3.6	2.7×10^{-3}	2.6	5.3×10^{-7}	<0.1	4.3×10^{-3}	98.2
SWMU 5 (soil only)	4.1×10^{-4}	1.0	4.1×10^{-4}	1.0	2.9×10^{-4}	2.2	1.0×10^{-5}	<0.1	$>1.0 \times 10^{-2}$	46.2
SWMU 6 (soil only)	2.4×10^{-4}	0.6	2.4×10^{-4}	0.6	2.3×10^{-4}	2.4	1.7×10^{-7}	<0.1	2.4×10^{-3}	9.4

Notes:
Taken from the WAG 3 BHHRA.
NA = No land use scenarios of concern or media not present to assess use scenario.

[a] Values reported are for the child.

McN = McNairy Formation

- Because there was considerable uncertainty in some of the exposure parameters, exposure pathways, and toxicity values, a quantitative uncertainty analysis was performed. In this analysis, approved toxicity values, site-specific exposure parameters, and exposure pathways were used to calculate risk estimates for the current and future industrial worker. The results of this analysis are presented in Exhibits 1.30 through 1.33. Most important to the GWOU BHHRA is the information shown in Exhibits 1.31 and 1.33. In these exhibits, the ELCR and systemic toxicity posed to the future worker through use of groundwater are seen to vary by up to two orders of magnitude (ELCR for use of water drawn from the RGA at SWMU 5) as the various uncertainties are considered. While HI for the industrial worker is less than 1 if all uncertainties are considered (see lower bound HI in Exhibit 1.33), the total ELCR for all SWMU and groundwater source combinations remains above the *de minimis* level at all sites (see lower bound ELCR in Exhibit 1.31).

Exhibit 1.30. Summary of risk and uncertainty results for current industrial worker for WAG 3

Location	Default ELCR[a]	Default ELCR minus infequently detected analytes[b]	Default ELCR minus common laboratory contaminants[c]	Default ELCR omitting contaminants with provisional or withdrawn toxicity values[d]	ELCR computed using EPA Region 4 absorption factors[e]	Lower bound ELCR[f]
SWMU 4 (soil)	5.4×10^{-4}	5.4×10^{-4}	5.4×10^{-4}	1.4×10^{-5}	2.5×10^{-5}	1.4×10^{-5}
SWMU 5 (soil)	4.1×10^{-4}	4.1×10^{-4}	4.1×10^{-4}	2.1×10^{-4}	3.3×10^{-5}	2.8×10^{-5}
SWMU 6 (soil)	2.4×10^{-4}	2.4×10^{-4}	2.4×10^{-4}	2.4×10^{-5}	8.0×10^{-6}	3.1×10^{-6}

Notes:
[a] These values are identical to the values presented in Exhibit 1.25 in the WAG 3 BHHRA.
[b] These values are identical to the values presented in Table 1.58 in the WAG 3 BHHRA.
[c] These values are identical to the values presented in Table 1.59 in the WAG 3 BHHRA.
[d] These values are identical to the values presented in Table 1.74 in the WAG 3 BHHRA.
[e] These values are identical to the values presented in Exhibit 1.71 in the WAG 3 BHHRA.
[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.

- The systemic toxicity varies by factors ranging from <1 to 3.6 once the effect of lead is removed. (Note that the effect of lead is due to the use of a provisional RfD. Please see Sect. 6 of this BHHRA for a discussion of the effect of the use of this provisional value.) As noted above, the lower bound HI estimates are less than 1.

- Screening level modeling indicated that WAG 3 is a potential source of off-site groundwater contamination. As shown in Exhibits 1.34, 1.35, and 1.36, modeling identified WAG 3 as being the potential source of unacceptable concentrations of five organic compounds, seven inorganic chemicals, and eight radionuclides.

Exhibit 1.31. Summary of risk and uncertainty results for future industrial worker for WAG 3

Location	Default ELCR[a]	Default ELCRs minus infrequently detected contaminants[b]	Default ELCR minus laboratory contaminants[c]	Default ELCR omitting contaminants with provisional or withdrawn toxicity values[d]	ELCR computed using EPA Region 4 dermal toxicity values	Lower bound ELCR[e]
SWMU 4 (RGA)	4.7×10^{-4}	4.7×10^{-4}	4.7×10^{-4}	1.7×10^{-4}	NA	1.7×10^{-4}
SWMU 4 (McNairy)	3.1×10^{-3}	3.1×10^{-3}	3.1×10^{-3}	5.6×10^{-4}	NA	5.6×10^{-4}
SWMU 5 (RGA)	5.4×10^{-4}	1.9×10^{-4}	5.4×10^{-4}	3.5×10^{-4}	NA	6.4×10^{-6}
SWMU 5 (McNairy)	1.2×10^{-3}	1.2×10^{-3}	1.2×10^{-3}	7.2×10^{-4}	NA	7.2×10^{-4}
SWMU 6 (RGA)	2.3×10^{-4}	2.3×10^{-4}	2.3×10^{-4}	3.9×10^{-5}	NA	3.9×10^{-5}
SWMU 6 (McNairy)	7.8×10^{-4}	7.8×10^{-4}	7.8×10^{-4}	1.9×10^{-4}	NA	1.9×10^{-4}

Notes:
NA = Not Applicable.

[a] These values are identical to the values presented in Exhibit 1.35 in the WAG 3 BHHRA.
[b] These values are identical to the values presented in Table 1.58 in the WAG 3 BHHRA.
[c] These values are identical to the values presented in Table 1.59 in the WAG 3 BHHRA.
[d] These values are identical to the values presented in Table 1.74 in the WAG 3 BHHRA.
[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.

Exhibit 1.32. Summary of systemic toxicity and uncertainty results for current industrial worker for WAG 3

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 omitting contaminants with provisional or withdrawn toxicity values w/o lead[d]	HI computed using U.S. EPA Region 4 absorption factors w/o lead[e]	Lower bound HI[f]
SWMU 4 (soil)	3.6	3.6	3.6	3.6	2.8	<1	<1
SWMU 5 (soil)	1.0	1.0	<1	<1	<1	<1	<1
SWMU 6 (soil)	<1	<1	<1	<1	<1	<1	<1

Notes:

[a] These values are identical to the values presented in Exhibit 1.23 in the WAG 3 BHHRA.
[b] These values are identical to the values presented in Table 1.58 in the WAG 3 BHHRA.
[c] These values are identical to the values presented in Table 1.59 in the WAG 3 BHHRA.
[d] These values are identical to the values presented in Table 1.74 in the WAG 3 BHHRA.
[e] These values are identical to the values presented in Exhibit 1.71 in the WAG 3 BHHRA.
[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 1.33. Summary of systemic toxicity and uncertainty results for future industrial worker for WAG 3

Location	Default HI[a]	Default HI w/o lead[a]	Default HIs minus infrequently detected contaminants w/o lead[b]	Default HI minus laboratory contaminants w/o lead[c]	Default HI omitting contaminants with provisional or withdrawn toxicity values w/o lead[d]	Lower bound HI[e]
SWMU 4 (RGA)	16,000	32.6	32.5	32.6	6.6	6.4
SWMU 4 (McNairy)	216,000	75.9	75.9	75.9	24.9	24.9
SWMU 4 (RGA)	19,600	26.8	26.5	26.8	6.1	5.8
SWMU 4 (McNairy)	71,000	63.0	63.0	63.0	10.4	10.4
SWMU 6 (RGA)	22,700	19.1	18.6	19.1	6.0	5.5
SWMU 6 (McNairy)	70,000	41.7	41.7	41.7	8.2	8.2

Notes:

[a] These values are identical to the values presented in Exhibit 1.27 in the WAG 3 BHHRA.
[b] These values are identical to the values presented in Table 1.58 in the WAG 3 BHHRA.
[c] These values are identical to the values presented in Table 1.59 in the WAG 3 BHHRA.
[d] These values are identical to the values presented in Table 1.86 in the WAG 3 BHHRA.
[e] 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 1.34. Comparison between maximum modeled concentrations at the PGDP fence boundary and residential use risk-based concentrations (RBCs) for WAG 3 sources

Contaminant[a]	Source[b]	Maximum concentration[c]	Residential use RBC[d] Cancer	Residential use RBC[d] Systemic toxicity	Exceed?[e]
Inorganic chemicals (mg/L)					
Arsenic	SWMU 4 UCRS WP1	1.86E-01	3.50E-06	4.50E-04	Both
Chromium	SWMU 4 UCRS WP1	1.15E-37	NV	4.20E-03	None
Cobalt	SWMU 4 UCRS WP1	3.29E+00	NV	9.10E-02	ST
Copper	SWMU 4 UCRS WP1	7.32E+00	NV	6.00E-02	ST
Iron	SWMU 4 UCRS WP1	1.16E+03	NV	4.50E-01	ST
Lead	SWMU 4 UCRS WP1	8.45E-42	NV	1.50E-07	None
Lithium	SWMU 4 UCRS WP1	1.76E-03	NV	3.00E-02	None
Manganese	SWMU 4 UCRS WP1	5.13E+01	NV	6.70E-02	ST
Nickel	SWMU 4 UCRS WP1	1.45E-01	NV	3.00E-02	ST
Strontium	SWMU 4 UCRS WP1	2.54E-05	NV	9.00E-01	None
Vandium	SWMU 4 UCRS WP1	5.53E-02	NV	9.30E-03	ST
Organic chemicals (mg/L)					
1,1-Dichloroethene	SWMU 4 UCRS WP1	2.58E-01	9.30E-07	1.80E-03	Both
1,2-Dichloroethene	SWMU 4 UCRS WP1	2.24E-03	NV	1.80E-03	ST
2-Methylnaphthalene	SWMU 5 surface soil	3.88E-05	NV	NV	NA
Acenaphthylene	SWMU 5 surface soil	4.35E-03	NV	NV	NA
Carbon tetrachloride	SWMU 4 UCRS WP1	5.94E-04	1.50E-05	1.20E-04	Both
Pentachlorophenol	SWMU 4 UCRS WP1	3.35E-18	2.10E-05	2.30E-02	None
Phenanthrene	SWMU 5 surface soil	2.62E-03	NV	NV	NA
Toluene	SWMU 5 UCRS WP2	2.78E-05	NV	2.40E-02	None
Trichloroethene	SWMU 4 UCRS WP1	2.26E+01	1.40E-04	1.20E-03	Both
Vinyl chloride	SWMU 4 UCRS WP1	3.31E-01	1.70E-06	NV	Cancer
Radionuclides (pCi/L)[f]					
Neptunium-237	SWMU 4 UCRS WP1	4.88E+02	1.30E-01	NV	Cancer
Plutonium-239	SWMU 4 UCRS WP1	1.09E+01	1.22E-01	NV	Cancer
Radium-226	SWMU 4 UCRS WP1	2.21E-01	1.30E-01	NV	Cancer
Technetium-99	SWMU 4 UCRS WP1	6.34E+04	2.80E+01	NV	Cancer
Thorium-230	SWMU 4 UCRS WP1	3.56E-28	1.03E+00	NV	None
Total uranium[g]	SWMU 4 UCRS WP1	6.46E+03	6.23E-01	NV	Cancer
Uranium-234	SWMU 4 UCRS WP1	4.51E+03	8.70E-01	NV	Cancer
Uranium-235	SWMU 4 UCRS WP1	4.75E+01	8.21E-01	NV	Cancer
Uranium-238	SWMU 4 UCRS WP1	8.33E+02	6.23E-01	NV	Cancer

[a] All contaminants with an identified source and a modeled concentration are listed.
[b] Media for each SWMU in which the source contributing the maximum modeled concentration is located. The "WP" prefix was used in the WAG 3 BHHRA to delineate multiple UCRS sources.
[c] Maximum modeled contaminant concentration among all sources modeled.
[d] All residential use RBCs are from Table A.4 in Appendix A of the WAG 3 BHHRA. 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 1.0E-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 that 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.
[e] "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 RBCs for radionuclides include contributions from short-lived daughters.
[g] The maximum detected activity of uranium in SWMU 4 was from a sample reported as "total uranium" rather than as specific isotopes; therefore, it was assessed as U-238 because naturally occurring uranium contains approximately 99.3% U-238, 0.7% U-235, and 0.005% U234.

Exhibit 1.35. Summary of sources and maximum modeled concentrations for contaminants that have a source within the WAG 3 area that exceeds a residential use risk-based concentration (RBC)

Contaminant[a]	Source[b]	Maximum concentration[c]	Residential Use RBC[d] Cancer	Residential Use RBC[d] Systemic toxicity	Exceed?[e]
Inorganic chemicals (mg/L)					
Arsenic	SWMU 4 UCRS WP1	1.86E-01	3.50E-06	4.50E-04	Both
Cobalt	SWMU 4 UCRS WP1	3.29E+00	NV	9.10E-02	ST
	SWMU 5 UCRS WP2	1.89E-03	NV	9.10E-02	None
	SWMU 6 UCRS WP2	1.66E-03	NV	9.10E-02	None
	SWMU 6 UCRS WP1	8.06E-05	NV	9.10E-02	None
	SWMU 5 UCRS WP1	2.51E-05	NV	9.10E-02	None
Copper	SWMU 4 UCRS WP1	7.32E+00	NV	6.00E-02	ST
	SWMU 4 surface soil	4.40E-04	NV	6.00E-02	None
	SWMU 6 UCRS WP1	3.13E-11	NV	6.00E-02	None
	SWMU 6 surface soil	2.56E-12	NV	6.00E-02	None
Iron	SWMU 4 UCRS WP1	1.16E+03	NV	4.50E-01	ST
	SWMU 5 UCRS WP2	4.64E+02	NV	4.50E-01	ST
	SWMU 6 UCRS WP1	6.01E+01	NV	4.50E-01	ST
	SWMU 5 UCRS WP1	4.98E+01	NV	4.50E-01	ST
	SWMU 6 UCRS WP2	3.28E+01	NV	4.50E-01	ST
	SWMU 4 surface soil	1.97E+00	NV	4.50E-01	ST
Manganese	SWMU 4 UCRS WP1	5.13E+01	NV	6.70E-02	ST
	SWMU 5 UCRS WP2	1.56E+01	NV	6.70E-02	ST
	SWMU 6 UCRS WP1	4.08E-01	NV	6.70E-02	ST
	SWMU 5 UCRS WP1	2.32E-01	NV	6.70E-02	ST
Nickel	SWMU 4 UCRS WP1	1.45E-01	NV	3.00E-02	ST
	SWMU 4 surface soil	2.53E-03	NV	3.00E-02	None
Vanadium	SWMU 4 UCRS WP1	5.53E-02	NV	9.30E-03	ST
Organic chemicals (mg/L)					
1,1-Dichloroethene	SWMU 4 UCRS WP1	2.58E-01	9.30E-07	1.80E-03	Both
1,2-Dichloroethene	SWMU 4 UCRS WP1	2.24E-03	NV	1.80E-03	ST
Carbon tetrachloride	SWMU 4 UCRS WP1	5.94E-04	1.50E-05	1.20E-04	Both
Trichloroethene	SWMU 4 UCRS WP1	2.26E+01	1.40E-04	1.20E-03	Both
Vinyl chloride	SWMU 4 UCRS WP1	3.31E-01	1.70E-06	NV	Cancer
Radionuclides (pCi/L)[f]					
Neptunium-237	SWMU 4 UCRS WP1	4.88E+02	1.30E-01	NV	Cancer
	SWMU 6 waste cell	1.68E-01	1.30E-01	NV	Cancer
	SWMU 6 UCRS WP1	5.97E-02	1.30E-01	NV	None
	SWMU 4 surface soil	5.33E-02	1.30E-01	NV	None
Plutonium-239	SWMU 4 UCRS WP1	1.09E+01	1.22E-02	NV	Cancer
	SWMU 4 surface soil	4.16E-04	1.22E-02	NV	None
Radium-226	SWMU 4 UCRS WP1	2.21E-01	1.30E-01	NV	Cancer
	SWMU 5 UCRS WP1	5.59E-03	1.30E-01	NV	None
	SWMU 5 UCRS WP2	5.33E-02	1.30E-01	NV	None
Technetium-99	SWMU 4 UCRS WP1	6.34E+04	2.80E+01	NV	Cancer
	SWMU 5 UCRS WP2	2.29E+02	2.80E+01	NV	Cancer
	SWMU 6 waste cell	9.15E+01	2.80E+01	NV	Cancer
	SWMU 5 surface soil	5.78E+01	2.80E+01	NV	Cancer
	SWMU 6 UCRS WP1	1.16E+01	2.80E+01	NV	None
	SWMU 6 surface soil	9.71E+00	2.80E+01	NV	None

Exhibit 1.35. Summary of sources and maximum modeled concentrations for contaminants that have a source within the WAG 3 area that exceeds a residential use risk-based concentration (RBC) (continued)

Contaminant[a]	Source[b]	Maximum concentration[c]	Residential Use RBC[d] Cancer	Residential Use RBC[d] Systemic toxicity	Exceed?[e]
Total uranium[g]	SWMU 4 UCRS WP1	6.46E+03	6.23E-01	NV	Cancer
Uranium-234	SWMU 4 UCRS WP1	4.51E+03	8.70E-01	NV	Cancer
	SWMU 4 surface soil	1.37E+00	8.70E-01	NV	Cancer
Uranium-235	SWMU 4 UCRS WP1	4.75E+01	8.21E-01	NV	Cancer
Uranium-238	SWMU 4 UCRS WP1	8.33E+02	6.23E-01	NV	Cancer
	SWMU 4 surface soil	2.67E+00	6.23E-01	NV	Cancer
	SWMU 5 UCRS WP2	9.95E-19	6.23E-01	NV	None
	SWMU 5 UCRS WP1	5.14E-19	6.23E-01	NV	None
	SWMU 6 waste cell	4.80E-19	6.23E-01	NV	None
	SWMU 6 UCRS WP1	3.49E-19	6.23E-01	NV	None

[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 SWMU. Sites not listed do not contain a source of the contaminant. The "WP" prefix was used in the WAG 3 BHHRA to delineate multiple UCRS sources.
[c] Maximum modeled contaminant concentration for source.
[d] All residential use RBCs are from Table A.4 in Appendix A of the WAG 3 BHHRA. 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 1.0E-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 that an RBC for the endpoint is not available because toxicity information is lacking.
[e] "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 RBCs for radionuclides include contributions from short-lived daughters.
[g] The maximum detected activity of uranium in SWMU 4 was from a sample reported as "total uranium" rather than as specific isotopes; therefore, it was assessed as U-238 because naturally occurring uranium contains approximately 99.3% U-238, 0.7% U-235, and 0.005% U234.

Exhibit 1.36. Summary of time required to reach maximum modeled concentrations at the PGDP fence boundary for contaminant sources within the WAG 3 area that contribute maximum contaminant concentrations exceeding residential use risk-based concentrations (RBCs)

Contaminant[a]	Source[b]	Maximum concentration[c]	Year[d]
Inorganic chemicals (mg/L)			
Arsenic	SWMU 4 UCRS WP1	1.86E-01	1853
Cobalt	SWMU 4 UCRS WP1	3.29E+00	787.5
Copper	SWMU 4 UCRS WP1	7.32E+00	7992
Iron	SWMU 4 UCRS WP1	1.16E+03	1738
	SWMU 5 UCRS WP2	4.64E+02	1873
	SWMU 6 UCRS WP1	6.01E+01	1966
	SWMU 5 UCRS WP1	4.98E+01	1411
	SWMU 6 UCRS WP2	3.28E+01	1787
	SWMU 4 surface soil	1.97E+00	1337
Manganese	SWMU 4 UCRS WP1	5.13E+01	2248
	SWMU 5 UCRS WP2	1.56E+01	4097
	SWMU 6 UCRS WP1	4.08E-01	3690
	SWMU 5 UCRS WP-1	2.32E-01	3870
Nickel	SWMU 4 UCRS WP1	1.45E-01	5019
Vanadium	SWMU 4 UCRS WP1	5.53E-02	9411
Organic chemicals (mg/L)			
1,1-Dichloroethene	SWMU 4 UCRS WP1	2.58E-01	62.86
1,2-Dichloroethene	SWMU 4 UCRS WP1	2.24E-03	18.8
Carbon tetrachloride	SWMU 4 UCRS WP1	5.94E-04	300.6
Trichloroethene	SWMU 4 UCRS WP1	2.26E+01	101.6
Vinyl chloride	SWMU 4 UCRS WP1	3.31E-01	56.6
Radionuclides (pCi/L)			
Neptunium-237	SWMU 4 UCRS WP1	4.88E+02	316.4
	SWMU 6 waste cell	1.68E-01	330.2
Plutonium-239	SWMU 4 UCRS WP1	1.09E+01	8665
Radium-226	SWMU 4 UCRS WP1	2.21E-01	8208
Technetium-99	SWMU 4 UCRS WP1	6.34E+04	111.4
	SWMU 5 UCRS WP2	2.29E+02	130.1
	SWMU 6 waste cell	9.15E+01	118.6
	SWMU 5 surface soil	5.78E+01	109.5
	SWMU 6 UCRS WP1	1.16E+01	118.6
	SWMU 6 surface soil	9.71E+00	105.1
Total uranium[e]	SWMU 4 UCRS WP1	6.46E+03	4330
Uranium-234	SWMU 4 UCRS WP1	4.51E+03	4329
	SWMU 4 surface soil	1.37E+00	4355
Uranium-235	SWMU 4 UCRS WP1	4.75E+01	5141
Uranium-238	SWMU 4 UCRS WP1	8.33E+02	4330
	SWMU 4 surface soil	2.67E+00	4356

[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 SWMU. The "WP" prefix was used in the WAG 3 BHHRA to delineate multiple UCRS sources.
[c] Maximum modeled contaminant concentration for source.
[d] All dates taken from MEPAS modeling results and are years from present.
[e] The maximum detected activity of uranium in SWMU 4 was from a sample reported as "total uranium" rather than as specific isotopes; therefore, it was assessed as U-238 because naturally occurring uranium contains approximately 99.3% U-238, 0.7% U-235, and 0.005% U-234.

1.2.1.5 WAG 22

SWMUs 7 and 30 (from material in DOE 1998a)

In 1996, the DOE conducted an RI/RFI at SWMUs 7 and 30 in WAG 22 at the PGDP. The purpose of this activity was to determine the presence, nature, and extent of contaminants at each of the units. The investigation focused on source characterization of the surrounding soils and the potential impacts of contaminants on adjoining surface waters and groundwater. Investigative activities included sampling and analysis of surface and subsurface soils, surface waters, groundwater, and waste.

The BHHRA utilized information collected during the remedial investigation and earlier investigations to characterize the baseline risks posed to human health from contact with contaminants in soil, sediment, groundwater, surface water, and buried waste at SWMUs 7 and 30 and from contact with media impacted by contaminants migrating from these units. To assess the risk posed by contaminants migrating from burial pits at SWMUs 7 and 30 to the RGA, fate and transport modeling was used. Fate and transport modeling was also used to assess the risks posed by contaminants migrating from SWMUs 7 and 30 to surrounding ditches. Note that although the SWMUs are bordered by ditches that collect and direct surface water runoff, the flow in these ditches was determined to be intermittent; therefore, all sediment samples collected from ditches were assessed as soil and not sediment in this BHHRA.

To facilitate data aggregation and to focus results on specific areas, this baseline risk assessment derived risk estimates for the following SWMUs or areas. The SWMUs and areas and their definitions are as follows:

- SWMU 7 – C-747-A Burial Ground.
- SWMU 30 – C-747A Burn Area.
- North Ditch – ditch along the north side of SWMUs 7 and 30.
- South Ditch – ditch along the south side of SWMUs 7 and 30.

Consistent with regulatory guidance and previous agreements, the BHHRA evaluated scenarios that encompassed current use and several hypothetical future uses of the SWMUs 7 and 30 area and areas to which contaminants from SWMUs 7 and 30 may migrate. These scenarios are listed below.

- Current on-site industrial – direct contact with surface soil (0 to 1 ft).

- Future on-site industrial – direct contact with surface soil (0 to 1 ft) and use of RGA groundwater below the SWMU.

- Future on-site excavation scenario – direct contact with waste and subsurface soil (0 to 10 ft). [Note, exposure was combined for all pits within a SWMU for this BHHRA. However, a pit-specific baseline risk assessment is presented in an appendix to the feasibility study for SWMUs 7 and 30 (DOE 1997b).]

- Future on-site recreational user – consumption of game exposed to surface soil (0 to 1 ft).

- Future off-site recreational user – direct contact with surface water and consumption of game exposed to surface water.

- Future on-site rural resident – direct contact with surface soil (0 to 1 ft), use of RGA groundwater below the SWMU, and consumption of vegetables.

- Future off-site rural resident – use of RGA groundwater at the DOE property boundary.

Note that this report contains a screening ecological risk assessment (SERA) for nonhuman receptors that may come into contact with contaminated media at or migrating from SWMUs 7 and 30. Results from this SERA are not discussed here.

Major conclusions and observations of the risk assessment are as follows.

- For SWMUs 7 and 30 and the associated ditches, ELCR and systemic toxicity posed by contaminants often exceed accepted standards of KDEP and EPA for one or more scenarios when assessed using default exposure parameters. Summaries of the BHHRA results for all land uses are in Exhibit 1.37.

- ELCR and systemic toxicity for use of groundwater drawn from the RGA and McNairy Formation were greater than the upper end of the EPA risk range (i.e., 1×10^{-4}) for both the future industrial worker and the potential future resident. Contaminants in groundwater driving risk were arsenic, beryllium, TCE, carbon tetrachloride, vinyl chloride, and ^{239}Pu. Contaminants in groundwater driving systemic toxicity were aluminum, arsenic, iron, manganese, 1,2-dichloroethene, Aroclor-1254, and TCE.

- Because there was considerable uncertainty in some of the exposure parameters, exposure pathways, and toxicity values, a quantitative uncertainty analysis was performed. In this analysis, approved toxicity values and site-specific exposure parameters and pathways were used to calculate risk estimates for the various use scenarios. The results of this analysis are in Exhibits 1.38 though 1.39. As shown there, neither the ELCR for the future industrial worker nor the ELCR for the future rural resident (onsite) were reduced to acceptable levels by assuming no groundwater use. Similarly, the HIs for these receptors were not reduced to acceptable levels by assuming no groundwater use.

- Fate and transport modeling determined that SWMUs 7 and 30 were potential sources of off-site groundwater contamination. Contaminants determined to potentially be contributed at rates leading to concentrations that are unacceptable were vinyl chloride and ^{99}Tc.

SWMU 2 (from material in DOE 1997a and DOE 1994a)

In 1989, DOE conducted an investigation of SWMU 2 (C-749 Uranium Burial Ground) of WAG 22 as part of the Phase II Site Investigation (CH2M Hill 1992). Subsequently, the results for SWMU 2 in CH2M Hill 1992 were reissued in an RI addendum (DOE 1994a) and a feasibility study report (DOE 1995a), proposed remedial action plan (DOE 1995b), and record of decision (DOE 1995c) were produced. As part of the record of decision, which was for interim action, DOE agreed to conduct additional investigations at SWMU 2 to verify the conceptual site model used to support the interim remedial actions in the record of decision. The risk material reported here is taken from the report developed following the later investigation (i.e., DOE 1997a).

Unlike the reports discussed earlier, DOE 1997a did not contain a BHHRA. In that report, detected analyte concentrations and summary statistics developed from them were compared to human health risk-based concentrations developed using procedures in the Methods Document. In addition, modeled concentrations for exposure to water drawn from the RGA at down-gradient exposure points were compared to these human health risk-based concentrations. Exhibits 1.40, 1.41, and 1.42 present some of the results of these analyses. Significant conclusions from these analyses are as follows:

- Several analytes were detected in soil and sediment at SWMU 2 that exceed human health risk-based concentrations for industrial use. These analytes include several metals (i.e., arsenic, beryllium, chromium, manganese, uranium, and vanadium), polychlorinated biphenyls (PCBs), TCE and its breakdown products, and uranium radioisotopes.

Exhibit 1.37. Summary of risk results for SWMUs 7 & 30 without lead as a COPC

Area	Use Scenario									
	Current Worker		Future Worker		Excavation Worker		Recreational User [a]		Rural Resident [a]	
	ELCR	HI	ELCR	HI	ELCR	HI	ELCR	HI	ELCR	HI
SWMU 7	4×10^{-3}	5	6×10^{-3}	62	2×10^{-3}	5	1×10^{-5}	<0.1	5×10^{-2}	1320
SWMU 30	4×10^{-3}	4	4×10^{-3}	12	1×10^{-3}	4	2×10^{-5}	<0.1	4×10^{-2}	334
North ditch	4×10^{-4}	5	4×10^{-4}	5	NA	NA	1×10^{-6}	<0.1	9×10^{-3}	229
South ditch	4×10^{-4}	5	4×10^{-4}	5	NA	NA	2×10^{-6}	<0.1	1×10^{-2}	334
30 year future	NA	NA	NA	NA	NA	NA	NA	NA	5×10^{-5}	<0.1
100 year future	NA	NA	NA	NA	NA	NA	NA	NA	2×10^{-4}	0.3

Notes:
Information taken from the WAG 22/SWMU 7 & 30 BHHRA.
NA = No land use scenarios of concern or media not present to assess use scenario.

[a] Values reported are for the child.

Exhibit 1.38. Summary of risk results and uncertainties for SWMUs 7 & 30

SWMU	Total Excess Lifetime Cancer Risk							
	Total ELCR derived using all default exposure values	Total ELCR derived using site-specific or average exposure values	Total ELCR without groundwater contribution	Total ELCR using EPA default dermal absorption exposure values	Total ELCR without contribution of lead	Total ELCR without consumption of game contribution	Total ELCR without consumption of vegetables contribution (soil + gw)	Total site-specific or average ELCR without groundwater, with EPA default dermal values, without lead, without game, and without vegetable contribution
Current Industrial Worker at Current Concentrations								
7	4×10^{-3}	2×10^{-4}	NA	2×10^{-4}	NA	NA	NA	1×10^{-5}
30	4×10^{-3}	2×10^{-4}	NA	2×10^{-4}	NA	NA	NA	1×10^{-5}
North ditch	4×10^{-4}	2×10^{-5}	NA	3×10^{-5}	NA	NA	NA	2×10^{-6}
South ditch	4×10^{-4}	2×10^{-5}	NA	5×10^{-5}	NA	NA	NA	3×10^{-6}
Future Industrial Worker at Current Concentrations								
7	6×10^{-3}	NA	4×10^{-3}	2×10^{-3}	NA	NA	NA	2×10^{-4}
30	4×10^{-3}	NA	4×10^{-3}	5×10^{-4}	NA	NA	NA	2×10^{-4}
North ditch	4×10^{-4}	NA	NA	3×10^{-5}	NA	NA	NA	3×10^{-5}
South ditch	4×10^{-4}	NA	NA	5×10^{-5}	NA	NA	NA	5×10^{-5}
Future Recreational User at Current Concentrations								
7	1×10^{-5}	NA	NA	NA	NA	0.0e+00	NA	0.0e+00
30	1×10^{-5}	NA	NA	NA	NA	0.0e+00	NA	0.0e+00
North ditch	1×10^{-6}	NA	NA	NA	NA	0.0e+00	NA	0.0e+00
South ditch	2×10^{-6}	NA	NA	NA	NA	0.0e+00	NA	0.0e+00

Table 1.38 (continued)

SWMU	Total Excess Lifetime Cancer Risk							
	Total ELCR derived using all default exposure values	Total ELCR derived using site-specific or average exposure values	Total ELCR without groundwater contribution	Total ELCR using EPA default dermal absorption exposure values	Total ELCR without contribution of lead	Total ELCR without consumption of game contribution	Total ELCR without consumption of vegetables contribution (soil + gw)	Total site-specific or average ELCR without groundwater, with EPA default dermal values, without lead, without game, and without vegetable contribution
Future Rural Resident at Current Concentrations								
7	5×10^{-2}	1×10^{-2}	3×10^{-2}	4×10^{-2}	NA	NA	2×10^{-2}	1×10^{-3}
30	4×10^{-2}	9×10^{-3}	3×10^{-2}	3×10^{-2}	NA	NA	1×10^{-2}	8×10^{-4}
North ditch	9×10^{-3}	2×10^{-3}	NA	8×10^{-3}	NA	NA	1×10^{-3}	3×10^{-4}
South ditch	1×10^{-2}	3×10^{-3}	NA	1×10^{-2}	NA	NA	1×10^{-3}	5×10^{-4}
Future Excavation Worker at Current Concentrations								
7	2×10^{-3}	1×10^{-5}	NA	9×10^{-4}	NA	NA	NA	7×10^{-6}
30	1×10^{-3}	6×10^{-5}	NA	1×10^{-4}	NA	NA	NA	7×10^{-6}
North ditch	NA	NA	NA	NA	NA	NA	NA	NA
South ditch	NA	NA	NA	NA	NA	NA	NA	NA

Notes:
Duplicate of Table ES.6 of the WAG 22/SWMU 7 & 30 BHHRA.
NA = Uncertainty not evaluated or not appropriate for this land use.

Exhibit 1.39. Summary of systemic toxicity results and uncertainties for SWMUs 7 & 30

SWMU	Systemic Toxicity							
	Total HI derived using all default exposure values with lead	Total HI derived without lead toxicity	Total HI derived using site-specific or average exposure values without lead	Total HI without groundwater contribution without lead	Total HI using EPA default dermal absorption exposure values without lead	Total HI without contribution from consumption of game	Total HI without contribution from consumption of vegetables (soil + gw)	Total site-specific or average HI without groundwater, with EPA default dermal values, without lead, without game, and without vegetable contribution
Current Industrial worker at Current Concentrations								
7	5×10^{3}	5×10^{0}	3×10^{-1}	NA	3×10^{-1}	NA	NA	2×10^{-2}
30	4×10^{3}	4×10^{0}	3×10^{-1}	NA	2×10^{-1}	NA	NA	1×10^{-2}
North ditch	3×10^{3}	5×10^{0}	3×10^{-1}	NA	2×10^{-1}	NA	NA	1×10^{-2}
South ditch	1×10^{4}	5×10^{0}	3×10^{-1}	NA	2×10^{-1}	NA	NA	1×10^{-2}
Future Industrial Worker at Current Concentrations								
7	5×10^{4}	6×10^{1}	NA	5×10^{0}	6×10^{1}	NA	NA	3×10^{-1}
30	2×10^{4}	1×10^{1}	NA	4×10^{0}	8×10^{0}	NA	NA	2×10^{-1}
North ditch	3×10^{3}	5×10^{0}	NA	NA	2×10^{-1}	NA	NA	2×10^{-1}
South ditch	1×10^{4}	5×10^{0}	NA	NA	2×10^{-1}	NA	NA	2×10^{-1}
Future Child Recreational User at Current Concentrations								
7	3×10^{0}	7×10^{-2}	NA	NA	NA	0.0e+00	NA	0.0e+00
30	2×10^{0}	4×10^{-2}	NA	NA	NA	0.0e+00	NA	0.0e+00
North ditch	2×10^{-1}	4×10^{-3}	NA	NA	NA	0.0e+00	NA	0.0e+00
South ditch	5×10^{-1}	5×10^{-3}	NA	NA	NA	0.0e+00	NA	0.0e+00

Table 1.39 (continued)

SWMU	Systemic Toxicity							
	Total HI derived using all default exposure values with lead	Total HI derived without lead toxicity	Total HI derived using site-specific or average exposure values without lead	Total HI without groundwater contribution without lead	Total HI using EPA default dermal absorption exposure values without lead	Total HI without contribution from consumption of game	Total HI without contribution from consumption of vegetables (soil + gw)	Total site-specific or average HI without groundwater, with EPA default dermal values, without lead, without game, and without vegetable contribution
Future Child Rural Resident at Current Concentrations								
7	9×10^5	1×10^3	1×10^3	4×10^2	1×10^3	NA	4×10^2	2×10^0
30	5×10^5	3×10^2	2×10^2	3×10^2	3×10^2	NA	7×10^1	2×10^0
North ditch	2×10^5	2×10^2	2×10^2	NA	2×10^2	NA	3×10^1	1×10^0
South ditch	7×10^5	3×10^2	2×10^2	NA	3×10^2	NA	3×10^1	2×10^0
Future Excavation Worker at Current Concentrations								
7	7×10^3	5×10^0	4×10^{-2}	NA	1×10^0	NA	NA	8×10^{-3}
30	5×10^3	4×10^0	2×10^{-1}	NA	9×10^{-1}	NA	NA	4×10^{-2}
North ditch	NA	NA	NA	NA	NA	NA	NA	NA
South ditch	NA	NA	NA	NA	NA	NA	NA	NA

Notes:
Duplicate of Table ES.7 in the WAG 22/SWMU 7 & 30 BHHRA.
NA = Uncertainty not evaluated or not appropriate for this land use.

Exhibit 1.40. Summary of comparison of concentration of analytes detected in soil and sediment at SWMU 2 to industrial use preliminary remediation goals

Analyte [a]	Sediment[b]	Soil				Waste Cell [g]
		Surface[c]	UCRS [d]	RGA [e]	McNairy [f]	
Arsenic	PBR	PBR	PBR	X	X	X
Barium			PB	X	X	X
Beryllium		PBR	PBR	X	X	X
Cadmium	B			X	X	X
Chromium	PBR	PR	PBR	X	X	X
Manganese	PR	PR	PBR	X	X	X
Nickel	B	B	B	X	X	X
Silver		B	B	X	X	X
Thallium	B		B	X	X	X
Uranium	PBR	X	PBR			X
Vanadium	P	P	PB	X	X	X
Polychlorinated Biphenyls	PR	PR	PR	X	X	
cis-1,2-Dichloroethene		X	PR	X	X	X
trans-1,2-Dichloroethene		X		X	X	X
Trichloroethene			PR			X
Vinyl Chloride			PR	X	X	X
Americium-241						
Neptunium-237	B	B				
Plutonium-239	PB	B				
Protactinium-234						P
Technetium-99	B	B				
Thorium-230	B	B	B		B	
Thorium-234	X	X	X	X	X	
Uranium-234	B	B	PB			
Uranium-235	PB	PB	PB			
Uranium-238	PB	PB	PB		B	P

Table 1.40 (continued)

Codes:		
	Blank	Analyses were performed, and analyte was either not detected or detected at a concentration below all preliminary remediation goals.
	X	Analyses for the analyte were not performed on samples taken from medium.
	P	Maximum detected concentration of the analyte exceeds one or both of the industrial use risk-based preliminary remediation goals.
	B	Maximum detected concentration of the analyte exceeds the background value.
	R	Maximum detected concentration exceeds the soil screening value contained in Kentucky (1995).

[a] The analytes listed are those contained in the final Sampling and Analysis Plan for SWMU 2 (DOE 1996e).

[b] The analyte list for sediment presented in the Sampling and Analysis Plan included the CERCLA Total Analyte List/Total Compound List, radiological analytes, polychlorinated biphenyls, and pesticides. For brevity, only the results for the SAP analytes are presented here. Complete results for sediment are presented in Table 5.6 of the SWMU 2 Data Summary Report. Sediment samples are those collected from 0 to 1 foot below surface at ditch and at low area.

[c] Surface soil samples are those collected from 0 to 1 foot below current ground surface.

[d] UCRS soil samples are those collected from Hydrogeologic Units 1, 2a, 2 Confining, 2b, and 3, exclusive of surface soil.

[e] RGA soil samples are those collected from Hydrogeologic Units 4 and 5.

[f] McNairy soil samples are those collected from the McNairy Formation.

[g] Waste cell samples are materials collected from within waste cells.

Exhibit 1.41. Comparison of estimated maximum concentrations of contaminants in RGA water at the PGDP fence line originating from soil and waste cells at SWMU 2 to residential preliminary remediation goals

Analyte	Maximum Concentration[a]	Time of Maximum Concentration[b]	Preliminary Remediation Goals				Criteria Exceeded[g]	Units
			ELCR[c]	HI[d]	Regulatory Value[e]	Background[f]		
Arsenic	4.93E-04	1505	3.50E-06	4.52E-04	5.00E-02	1.10E-02	P	mg/L
Barium	0.00E+00	35		1.04E-01	2.00E+00	2.90E-01	No	mg/L
Beryllium	6.45E-33	9975	1.05E-06	6.61E-03	4.00E-03	9.30E-03	No	mg/L
Cadmium	2.75E-07	9975		6.61E-04	5.00E-03	2.10E-02	No	mg/L
Chromium	8.20E-06	9975		7.05E-03	1.00E-01	1.30E-01	No	mg/L
Manganese	1.74E-02	2765		6.81E-02	1.59E-01*	1.60E-01	No	mg/L
Nickel	3.48E-06	9975		3.01E-02	6.19E-02	6.20E-02	No	mg/L
Silver	1.97E-04	1715		7.50E-03	1.00E-01*	1.10E-01	No	mg/L
Thallium	1.07E-03	35			2.00E-03	1.10E-01	No	mg/L
Uranium	4.86E-03	665		4.53E-03	2.00E-02*		No	mg/L
Vanadium	3.08E-04	8015		9.25E-03		1.40E-01	No	mg/L
Aroclor-1016	3.22E-31	9975		4.69E-05	5.00E-04		No	mg/L
Aroclor-1221	1.37E-06	4305	5.83E-07		5.00E-04		P[h]	mg/L
Aroclor-1232	9.95E-06	595	6.67E-07		5.00E-04		P[h]	mg/L
Aroclor-1242	1.26E-06	4725	6.40E-07		5.00E-04		P[h]	mg/L
Aroclor-1248	8.13E-40	9975	4.03E-07		5.00E-04		No	mg/L
Aroclor-1254	3.43E-43	9975	4.13E-07	4.30E-05	5.00E-04		No	mg/L
Aroclor-1260	0.00E+00	35	2.27E-07		5.00E-04		No	mg/L
1,1-Dichloroethene	4.78E-06	35	1.62E-06	1.34E-02	7.00E-03		P[h]	mg/L
1,2-Dichloroethene	5.35E-05	35	1.49E-02[i]		7.00E-02[i]		No	mg/L
Trichloroethene	5.64E-02	105	2.01E-04	7.86E-03	5.00E-03		PR	mg/L
Vinyl Chloride	7.74E-05	35	2.04E-06		2.00E-03		P	mg/L
Actinium-225	1.55E-06	3535	2.72E-01				No	pCi/L
Actinium-227	1.89E-04	735	6.17E-02[j]				No	pCi/L
Americium-241	6.28E-03	665	1.18E-01				No	pCi/L
Bismuth-210	6.94E-07	4025	5.30E+00				No	pCi/L
Neptunium-237	5.27E-02	35	1.29E-01[j]				No	pCi/L
Protactinium-231	1.97E-04	735	2.59E-01				No	pCi/L
Protactinium-233	5.27E-02	35	8.23E+00				No	pCi/L

Table 1.41 (continued)

Analyte	Maximum Concentration[a]	Time of Maximum Concentration[b]	Preliminary Remediation Goals				Criteria Exceeded[g]	Units
			ELCR[c]	HI[d]	Regulatory Value[e]	Background[f]		
Lead-210	6.94E-07	4025	3.82E-02[j]				No	pCi/L
Polonium-210	6.94E-07	4025	1.18E-01				No	pCi/L
Plutonium-239	2.66E-02	175	1.22E-01				No	pCi/L
Radium-223	1.89E-04	735	1.65E-01				No	pCi/L
Radium-225	1.55E-06	3535	2.46E-01				No	pCi/L
Radium-226	4.00E-02	1155	1.30E-01[j]				No	pCi/L
Radon-222	7.09E-07	4025	1.03E+00[j]				No	pCi/L
Technetium-99	3.46E-02	1365	2.76E+01				No	pCi/L
Thorium-227	1.89E-04	735	9.56E-01				No	pCi/L
Thorium-229	1.55E-06	3535	1.08E-01[j]				No	pCi/L
Thorium-230	1.04E-01	1085	1.03E+00			1.40E+00	No	pCi/L
Thorium-231	1.34E-02	665	2.16E+01				No	pCi/L
Thorium-234	1.61E-01	665	2.00E+00				No	pCi/L
Uranium-233	3.24E-05	315	8.62E-01				No	pCi/L
Uranium-234	1.51E-01	665	8.70E-01			1.20E+00	No	pCi/L
Uranium-235	1.34E-02	665	8.21E-01[j]			1.5E-01	No	pCi/L
Uranium-238	1.61E-01	665	6.23E-01[j]			1.10E+00	No	pCi/L

Note: Blank cells indicate that value is not available or not applicable.

a Maximum concentration of analyte predicted to be in RGA water at the PGDP security fence by MEPAS. All modeling was performed over a 10,000 years.

b Time at which MEPAS predicts maximum concentration will be reached.

c Direct contact residential use risk-based preliminary remediation goal calculated using 1×10^{-7} as the target excess lifetime cancer risk (ELCR) for chemicals and 1×10^{-6} as the target ELCR for radionuclides.

d Direct contact residential use risk-based preliminary remediation goal calculated using 0.1 as the target hazard index.

e The value reported is the respective analyte's maximum contaminant level (MCL). All MCLs are Primary Drinking Water Standards except where marked with *. Marked values are either proposed Primary Drinking Water Standards or Secondary Drinking Water Standards (SMCLs).

f Concentration of analyte in uncontaminated media. For all water samples, the background values reported are those for the Regional Gravel Aquifer (RGA) as reported in the SWMU 2 Data Summary Report.

g Summary of preliminary remediation goals exceeded. In this table, maximum detected concentrations are not directly comparable to preliminary remediation goals because MEPAS only predicts the additional contamination added by migration. However, the difference in magnitude between preliminary remediation goals and the maximum predicted concentrations indicates that contaminants from SWMU 2 are unlikely to contribute significantly to contamination in water at the PGDP security fence over the next 10,000 years.

h Source term concentration based on maximum undetected concentration.

i MEPAS does not offer both cis-1,2-Dichloroethene and trans-1,2-Dichloroethene; therefore, both isomers were modeled as trans-1,2-Dichloroethene; however, the preliminary remediation goals reported are the lesser of those for the respective isomers.

j Preliminary remediation goal calculated using the toxicity value (i.e., slope factor) for parent isotope and short-lived daughters.

Exhibit 1.42. Comparison of present and future concentrations of trichloroethene in RGA water drawn at the security fence and plant boundary to residential preliminary remediation goals - contributions from potential secondary sources at SWMU 2

Time (years)	Concentration[a]	Preliminary Remediation Goals				Criteria Exceeded[f]	Units
		ELCR[b]	HI[c]	Regulatory Value[d]	Background[e]		
Results for trichloroethene at the security fence							
Present[g]	1.50E+01	2.01E-04	7.86E-03	5.00E-03	None	PR	mg/L
35	6.11E-02					PR	mg/L
105	3.94E-07					No	mg/L
Results for trichloroethene at the plant boundary							
Present[g]	1.50E+01	2.01E-04	7.86E-03	5.00E-03	None	PR	mg/L
35	4.51E-02					PR	mg/L
105	3.52E-06					No	mg/L

[a] Present concentrations are measured values; future concentrations are additional materials that will be in addition to materials migrating from other sources (i.e., contributed concentrations).

[b] Direct contact residential use risk-based preliminary remediation goal calculated using 1×10^{-7} as the target excess lifetime cancer risk (ELCR) for chemical.

[c] Direct contact residential use risk-based preliminary remediation goal calculated using 0.1 as the target hazard index.

[d] The value reported is the respective analyte's maximum contaminant level (MCL). All MCLs are Primary Drinking Water Standards.

[e] Concentration of analyte in uncontaminated media. For all water samples, the background values reported are those for the Regional Gravel Aquifer (RGA) as reported in the SWMU 2 Data Summary Report.

[f] Summary of preliminary remediation goals exceeded. In this table, contributed concentrations are not directly comparable to preliminary remediation goals because MEPAS only predicts the additional contamination added by migration. However, the difference in magnitude between preliminary remediation goals and the contributed concentrations indicates if the preliminary remediation goals may be exceeded. Definitions of codes are:

- P One or both of the residential use human health risk-based preliminary remediation goals are exceeded.
- R The regulatory value is exceeded.
- No No preliminary remediation goals are exceeded.

[g] Present concentrations were taken from analyses performed for sample from EW230 taken on 11/28/95.

- Several analytes were detected groundwater drawn from the RGA and McNairy that exceed human health risk-based concentrations for residential use. Over both aquifers these analytes include several metals (i.e., arsenic, barium, beryllium, cadmium, chromium, manganese, nickel, uranium, and vanadium), TCE and its breakdown products, and several radionuclides (i.e., ^{241}Am, ^{239}Pu, and uranium radioisotopes and their daughters).

- Fate and transport modeling identified several contaminants that may migrate from sources in soil and waste at SWMU 2 to an off-site exposure point at concentrations that exceed human health risk-based concentrations for residential use (see Exhibit 1.41). However, only one contaminant (TCE) was found to have a modeled concentration that exceeds its regulatory value (i.e., maximum contaminant level or MCL).

- Fate and transport modeling determined that TCE sources in the RGA at SWMU 2 (i.e., secondary sources) may contribute to unacceptable concentrations of TCE in RGA water at an off-site exposure points (see Exhibit 1.42). However, the contribution from these sources appeared to be minor compared to concentration of TCE currently found at the off-site exposure points.

1.2.1.6 WAGs 1&7 (from material in DOE 1996b)

In 1994, the DOE conducted a RFI at nine SWMUs in WAGs 1 and 7 at the PGDP. (See Exhibit 1.43 for a list of these SWMUs.) The purpose of this activity was to determine the presence, nature, and extent of contamination at each of the units. The investigation focused on source characterization of the surrounding soils and the potential impacts of contaminants on adjoining surface waters and groundwater. Investigative activities included sampling and analysis of surface and subsurface soils, surface waters, and groundwater.

Exhibit 1.43. SWMU descriptions for WAGs 1 and 7

WAG	SWMU	Description
1	38	C-615 Sewage Treatment Plant
1	100	C-206 Fire Training Area
1	136	C-740 TCE Spill Site
7	130–134	USTs at the C-611 Water Treatment Plant
7	8	C-746-K Sanitary Landfill

To facilitate data aggregation and to focus results on specific areas, this baseline risk assessment derived risk estimates for the following SWMUs or areas. The SWMUs and areas and their definitions are as follows:

- SWMU 38a – Ditch west of SWMU 38.
- SWMU 38b – Ditch south of SWMU 38.
- SWMU 38c – Soil and groundwater at SWMU 38.
- SWMU 100a – Ditch east of SWMU 100.
- SWMU 100b – Ditch west of SWMU 100.
- SWMU 100 – Soil and groundwater at SWMU 100.
- SWMU 136 – C-740 TCE Spill Area.
- SWMUs 130 through 134 – Underground storage tanks (USTs) at the C-611 Water Treatment Plant.
- SWMU 8a – Creek along SWMU 8.
- SWMU 8b – Soil and groundwater at SWMU 8.

Consistent with regulatory guidance and previous agreements, the BHHRA evaluated scenarios that encompass current use and several hypothetical future uses of the WAGs 1 and 7 SWMUs and areas to which contaminants from the WAGs 1 and 7 SWMUs may migrate. (Note that this report was released prior to the completion of the Methods Document. Therefore, the results reported here were derived using methods that varied from those currently used for BHHRAs at the PGDP). These are as follows:

- Current on-site industrial – direct contact with surface soil (0 to 1 ft).
- Future on-site industrial – direct contact with surface soil (0 to 1 ft) and use of RGA groundwater below the SWMU.
- Future on-site excavation scenario – direct contact with subsurface soil (0 to 10 ft).
- Future on-site recreational user – consumption of game exposed to surface soil (0 to 1 ft).
- Future off-site recreational user – direct contact with surface water and consumption of game exposed to surface water.
- Future on-site rural resident – direct contact with surface soil (0 to 1 ft), use of RGA groundwater below the SWMU, and consumption of vegetables.
- Future off-site rural resident – use of RGA groundwater at the DOE property boundary.

Note that this report contains a SERA for nonhuman receptors that may come into contact with contaminated media at or migrating from the WAGs 1 and 7 SWMUs. Results from this SERA are not discussed here.

Major conclusions and observations of the investigation are as follows:

- Using default exposure parameters, cumulative ELCR and systemic toxicity exceeds the acceptable standards of KDEP and EPA for one or more scenarios at SWMU 8, 38, 100, 133, 134, and 136. These results are summarized in Exhibit 1.44 and presented in more detail in Exhibit 1.45.
- Cumulative ELCR for residential use of groundwater drawn from the RGA was greater than the upper end of the EPA risk range (i.e., 1×10^{-4}) only at SWMU 8. Driving contaminants for ELCR in RGA water were beryllium and 1,1-dichloroethene. Cumulative HI for residential use of groundwater drawn from the RGA was unacceptable at SWMUs 8, 38, 100c, and 136. Driving contaminants for HI over all SWMUs in RGA water were aluminum, cobalt, iron, manganese, nickel, TCE, and 1,1-dichloroethene.
- Screening level modeling determined that risk posed by future use of groundwater contaminated by chemicals currently in soil and sediment at all SWMUs except SWMU 8 should not exceed EPA or KDEP acceptable standards at the off-site points of exposure. Note that SWMU 8 was not modeled because this unit is in an off-site location.

1.2.1.7 WAG 23 (from material in DOE 1994b and DOE 1999c)

In 1989, DOE conducted an investigation of the SWMUs in WAG 23 as part of the Phase II Site Investigation (CH2M Hill 1992). Subsequently, these SWMUs were assigned to WAG 23 (i.e., PCB sites) and the results from the Phase II Site Investigation report were reissued in a remedial investigation addendum (DOE 1994b). Using the information in the remedial investigation addendum, DOE prepared a feasibility study report (DOE 1996f). Subsequent to the release of the feasibility study report, DOE

Exhibit 1.44. Summary of use scenarios of concern for WAG 1 & 7 BRA

Area	Use Scenario									
	Current Worker		Future Worker		Excavation Worker		Recreational User		Rural Resident	
	ELCR	HI	ELCR	HI	ELCR	HI	ELCR	HI	ELCR	HI
8a	X	X	X	X			X	X	NA	NA
8b	X		X	X			NA	NA	X	X
38a	X	X	X	X	X		X	X	NA	NA
38b	X	X	X	X			X	X	NA	NA
38c	X	X	X	X			NA	NA	X	X
100a	X	X	X	X			X	X	NA	NA
100b	X	X	X	X			X	X	NA	NA
100c	X		X	X			NA	NA	X	X
130										
131										
132										
133									X	
134									X	
136			X						X	X

Notes:
Developed from information taken from the WAGs 1 & 7 BRA.
NA = Media not available to assess use scenario.

Exhibit 1.45. Summary of risk results for WAG 1 & 7

Area	Use Scenario									
	Current Worker		Future Worker		Excavation Worker		Recreational User [a]		Rural Resident [a]	
	ELCR	HI	ELCR	HI	LCR	HI	ELCR	HI	ELCR	HI
8a	3.2×10^{-4}	6.6	3.2×10^{-4}	6.6	NA	NA	9.0×10^{-4}	53.7	NA	NA
8b	4.1×10^{-5}	0.96	1.1×10^{-3}	44.9	1.09×10^{-6}	0.29	NA	NA	1.7×10^{-2}	642
38a	5.2×10^{-4}	5.97	5.2×10^{-4}	5.97	1.7×10^{-6}	0.55	8.4×10^{-4}	14.2	NA	NA
38b	1.6×10^{-4}	2.1	1.6×10^{-4}	2.1	NA	NA	2.6×10^{-4}	4.9	NA	NA
38c	3.8×10^{-5}	1.67	4.2×10^{-5}	1.94	NA	NA	NA	NA	4.1×10^{-3}	86.7
100a	2.9×10^{-4}	5.2	2.9×10^{-4}	5.2	NA	NA	4.6×10^{-4}	12.4	NA	NA
100b	2.2×10^{-4}	2.75	2.2×10^{-4}	2.75	NA	NA	3.6×10^{-4}	0.94	NA	NA
100c	1.4×10^{-6}	NA	1.7×10^{-4}	1.44	NA	NA	NA	NA	7.8×10^{-5}	22.7
130	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
131	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
132	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
133	NA	NA	NA	NA	NA	NA	NA	NA	9×10^{-5}	NA
134	NA	NA	NA	NA	NA	NA	NA	NA	3×10^{-6}	NA
136	NA	NA	6.9×10^{-6}	0.7	NA	NA	NA	NA	1.1×10^{-4}	10.5

Notes:
Information taken from the WAGs 1 & 7 BRA.
NA = No land use scenarios of concern or media not present to assess use scenario.

[a] Values reported are for the child.

performed a removal action at the WAG 23 SWMUs to address the direct contact risks identified in the remedial investigation addendum (DOE 1998b). Finally, a residual risk report for the WAG 23 SWMUs was prepared (DOE 1999c). Material presented below is taken from the remedial investigation addendum and the residual risk report. Note that because the BHHRA in the remedial investigation addendum was prepared prior to the release of the Methods Document, procedures used to estimate the risks in that report differ from those currently used at the PGDP. However, procedures used in the residual risk report were consistent with those in the Methods Document. Additionally, please note that the following material does not consider SWMU 1 of WAG 27 even though it is discussed in the referenced reports. This SWMU is not discussed here because the relationship of this SWMU to the GWOU is discussed in Subsect. 1.2.1.2 of this BHHRA.

To facilitate data aggregation and to focus results on specific areas, the BHHRAs for WAG 23 derived risk estimates for the following SWMUs or areas. The SWMUs and areas and their definitions are as follows:

- SWMUs 32 and 33 – C-728 Clean Waste Oil Tanks and C-728 Motor Cleaning Facility
- SWMUs 56 and 80 – C-540-A PCB Waste Staging Area and C-540-A PCB Spill Site
- SWMUs 57 and 81 – C-541-A PCB Waste Storage Area and C-541-A PCB Spill Site
- SWMU 74 – C-340 PCB Spill Site
- SWMU 79 – C-611 PCB Spill Site

Significant results from the BHHRAs are as follows:

- Prior to the removal action, risks and systemic toxicity posed to workers from direct contact with contaminated soil at SWMUs 32 and 33, SWMUs 56 and 80, and SWMUs 57 and 81 exceeded acceptable standards from EPA and KDEP. Driving contaminants were PCBs, polyaromatic hydrocarbons (PAHs), and dioxins/furans. (See Exhibit 1.46.) Risks and systemic toxicity posed to workers did not exceed the acceptable standards at SWMUs 74 and 79.

- Because contaminants found at the WAG 23 SWMUs are not expected to migrate in the subsurface due to their chemical characteristics, none of these SWMUs are expected to be a source of groundwater contamination in off-site areas.

- Sampling conducted prior to the removal action could not reproduce the nature and extent of contamination results in the Phase II Site Investigation for SWMUs 32 and 33. Therefore, a removal action was not performed at these SWMUs.

- The residual risk assessment for SWMUs 56 and 80 and SWMUs 57 and 81 determined that the removal action was successful in reducing ELCRs for industrial workers for exposure to PCB-contaminated soil to within the EPA acceptable range (i.e., $< 1 \times 10^{-4}$). (See Exhibit 1.47.)

1.2.1.8 Underground Storage Tanks (from material in DOE 1992a, DOE 1996c, and DOE 1996d)

In 1992, DOE conducted a site investigation for five USTs located near the C-200 Guard and Fire Headquarters, C-710 Technical Services, and C-750 Garage buildings. In 1994, as part of the WAGs 1 and 7 RFI, DOE conducted an investigation of contamination associated with five USTs located near the C-611 Water Treatment Plant. (See Subsect. 1.2.1.6.) Finally, in 1996, DOE reexamined the analytical results for USTs C-750A and C-750B to support closure of these units. The overall purpose of these investigations was to determine the presence, nature, and extent of contamination associated with the USTs and determine

Exhibit 1.46. Summary of WAG 23 risk results

SWMUs 32 and 33			
Direct Contact to Soil	**Future Onsite Worker (25 day/year)**	**Current Worker/Intruder (250 day/year)**	**Contaminant Contributing to Risk**
Cancer Risk Estimate	3×10^{-5}	3×10^{-4}	TCDD, PCBs
Chronic HI	0.12	1.2	TCDD
Radiological Cancer Risk Estimate	2×10^{-7}	2×10^{-6}	U-238
SWMUs 56 and 80			
Direct Contact to Soil	**Unrestricted Worker (250 day/year)**	**Worker/Intruder (25 day/year)**	**Contaminant Contributing to Risk**
Cancer Risk Estimate	3×10^{-3}	3×10^{-4}	Dioxins, PCBs, Furans
Chronic HI	35.4	3.5	Dioxins, Furans
SWMUs 57 and 81			
Direct Contact to Soil	**Unrestricted Worker (250 day/year)**	**Worker/Intruder (25 day/year)**	**Contaminant Contributing to Risk**
Cancer Risk Estimate	9×10^{-4}	9×10^{-5}	Dioxins
Chronic HI	1.3	0.13	Dioxins, Furans
SWMU 74			
Direct Contact to Soil	**Unrestricted Worker (250 day/year)**	**Worker/Intruder (25 day/year)**	**Contaminant Contributing to Risk**
Cancer Risk Estimate	2×10^{-5}	2×10^{-6}	PCBs
Chronic HI	0.01	0.001	None
SWMU 79			
Direct Contact to Soil	**Unrestricted Worker (250 day/year)**	**Worker/Intruder (25 day/year)**	**Contaminant Contributing to Risk**
Cancer Risk Estimate	3×10^{-5}	3×10^{-6}	PCBs
Chronic HI	0.05	0.005	None

Notes:
Information taken from the WAG 23 RI Addendum Report

Exhibit 1.47. Summary of the Residual Risk Report findings for WAG 23

Scenario	SWMU 1	SWMU 56 and 80	SWMU 57 and 81
Baseline risk assessment results for total cancer risk (Taken from Table 2.8 of the WAG 23 FS)			
Future Industrial Worker	5×10^{-4}	3×10^{-3}	9×10^{-4}
Current Industrial Worker	5×10^{-5}	3×10^{-4}	9×10^{-5}
Residual risk assessment results for total cancer risks			
Future Industrial Worker	4×10^{-5}	3×10^{-5}	8×10^{-5}
Current Industrial Worker	4×10^{-6}	3×10^{-6}	8×10^{-6}
Percent reduction in total cancer risk			
Future Industrial Worker			
Current Industrial Worker	91%	99%	91%

Notes:
Taken from Residual Risk Report for WAG 23.

if releases from these tanks posed unacceptable risk to human health and the environment. In these analyses, data were compiled for each UST. Therefore, over all investigations, the data aggregates were:

- C-750A – East of the C-750 building
- C-750B – East of the C-750 building
- C-750C – Northwest of the C-750 building
- C-200A – North of the C-200 building
- C-710B – East of the C-710 building
- SWMU 130 – West of C-611H building
- SWMU 131 – East of C-611H building
- SWMU 132 – North of C-611H building
- SWMU 133 – South of C-611H building
- SWMU 134 – Southeast of C-611H building

Consistent with regulatory guidance and previous agreements, the BHHRAs evaluated several scenarios that encompass current use and several hypothetical future uses of the areas at the USTs and areas to which contaminants from the USTs. (Note that these reports were released prior to the completion of the Methods Document. Therefore, the results reported here were derived using methods that vary from those currently used for BHHRAs at the PGDP).

Major conclusions and observations of the UST investigations are as follows:

- ELCR and systemic toxicity under current conditions for all USTs (i.e., under industrial scenarios) are within the acceptable range established by EPA.

- ELCR and systemic toxicity for some USTs exceed the acceptable range if contact with contaminated subsurface soil is assumed (e.g., see Exhibit 1.48).

- Contamination associated with the C-750A and B USTs and the SWMUs 130 to 134 USTs is not expected to migrate to an off-site exposure point at a concentration that would result in unacceptable ELCR and systemic toxicity.

Information supporting these conclusions are in Exhibits 1.48, 1.49, and 1.50.

Exhibit 1.48. Summary risk results from the UST BRA

Scenario (Light Industrial)	Systemic Toxicity	Excess Lifetime Cancer Risk
Site-specific Estimate	0.82	1.5×10^{-4}
Reference Estimates	1.1	7.9×10^{-5}

Notes:
Information taken from Tables 5-1 to 5-4 of the UST BRA.

Exhibit 1.49. Summary risk results from the WAGs 1 & 7 UST BRA

Scenario	Systemic Toxicity	Excess Lifetime Cancer Risk
	UST 133	
Future adult rural resident	none	9×10^{-5}
Future child rural resident	none	NA
	UST 134	
Future adult rural resident	none	3×10^{-6}
Future child rural resident	none	NA

Note:
Information taken from Tables ES.1 and ES.2 of the WAGs 1 & 7 UST BRA
NA = ELCR not applicable to child cohort. Values for adult ELCR include exposures as a child.

Exhibit 1.50. Summary risk results from the C-750 A&B UST BRA

Scenario	Systemic Toxicity	Excess Lifetime Cancer Risk
	C-750 A&B UST	
Future Excavation Worker	0.00554	4.13×10^{-6}

Notes:
Information taken from Tables 9 and 10 of the C-750 A&B UST BRA.

1.2.1.9 Summary of Source Control Unit Investigations

The source control unit investigations summarized in the previous subsections indicate that direct exposure to contaminated media may lead to unacceptable risks at all units except the USTs under one or more of the scenarios assessed. However, these investigations also indicate that not all of the units are sources of off-site groundwater contamination. The following list summarizes the units that are sources of off-site contamination and the contaminants associated with that source.

- WAG 6 – Source of antimony, copper, iron, manganese, carbon tetrachloride, tetrachloroethene, TCE, TCE breakdown products, and ^{99}Tc.

- WAG 27 – Source of antimony, manganese, silver, thallium, and vanadium, phenanthrene, xylenes, TCE and TCE breakdown products.

- WAG 28 – Source of chromium, lithium, manganese, strontium, TCE, and ^{99}Tc.

- WAG 3 – Source of arsenic, cobalt, copper, iron, manganese, nickel, vanadium, 1,1-dichloroethene, carbon tetrachloride, TCE, TCE breakdown products, ^{237}Np, ^{239}Pu, ^{226}Ra, ^{99}Tc, and uranium isotopes.

- WAG 22/SWMUs 7 and 30 – Source of the TCE breakdown product vinyl chloride and ^{99}Tc.

- WAG 22/SWMU 2 – Source of arsenic, PCBs, TCE, and TCE breakdown products.

- WAGs 1 and 7 – Not a source. (See exception for SWMU 8. Fate and transport modeling for SWMU 8 has not been completed; however, this unit is a known source of metals contamination to the creeks surrounding it.)

- WAG 23 – Not a source.

- USTs – Not a source.

Therefore, fate and transport modeling indicates that several metals, TCE and its breakdown products, and several radionuclides may be migrating through groundwater to off-site areas from source control units at the PGDP. Specifically, the contaminants include arsenic, antimony, cobalt, copper, iron, manganese, nickel, silver, thallium, vanadium, 1,1-dichloroethene, carbon tetrachloride, tetrachloroethene, PCBs, phenanthrene, xylenes, TCE, *trans*-1,2-dichloroethene, *cis*-1,2-dichloroethene, vinyl chloride, ^{237}Np, ^{239}Pu, ^{226}Ra, ^{99}Tc, and uranium isotopes.

1.2.2 Results of Previous Groundwater Integrator Unit Investigations

Four previous reports contain baseline risk assessment results that are useful in understanding the risks posed by exposure to contaminants that have migrated from source control units to the groundwater integrator unit at the PGDP. These reports are listed below by their date of release.

- *Results of the Site Investigation, Phase I at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky* (CH2M Hill 1991b);

- *Results of the Public Health and Ecological Assessment, Phase II* (CH2M Hill 1991a) [This report is Volume 6 of *Results of the Site Investigation, Phase II at the Paducah Gaseous Diffusion Plant, Paducah, Kentucky* (CH2M Hill 1992)];

- *Human Health Baseline Risk Assessment for the Northwest Plume, Paducah Gaseous Diffusion Plant, Paducah, Kentucky* (DOE 1993a); and,

- *Baseline Risk Assessment and Technical Investigation Report for the Northwest Dissolved Phase Plume, Paducah Gaseous Diffusion Plant* (DOE 1994c).

The following subsections present the risk assessment and risk evaluation results found in these reports. Note that the methods used is each of these reports are not consistent with those prescribed in the Methods Document (DOE 1996a) because they were completed prior to 1996. Therefore, the results presented in the following subsections should be used for comparison only and should be considered preliminary to the results reported later in the BHHRA.

1.2.2.1 Results of the Phase I Site Investigation

In response to the identification of PGDP-related contaminants in water drawn from off-site residential wells, DOE launched a two-phased site investigation of the nature and extent of contamination at and around the PGDP. The Phase I investigation evaluated the nature and extent of off-site contamination originating at the PGDP and determined risk presented by this contamination to off-site receptors. Although this assessment considered risk from exposure to contaminants found in all media, the following discussion focuses on the risk from exposure to contaminants found in groundwater because these results are most relevant to the BHHRA for the GWOU.

In the Phase I BHHRA, the sampling data were divided into three groups to enable the assessment to focus on particular areas and to address some of the uncertainty in well construction. These groups were data from residential wells, data from PGDP monitoring wells, and data from Tennessee Valley Authority (TVA) wells.

Consistent with the agreements at the time, the risk assessment only evaluated ELCR and systemic toxicity posed to actual and hypothetical residents from household use of groundwater under average and maximum exposure assumptions. Additionally, only the ingestion and inhalation routes of exposure were considered.

The results of the risk assessment of groundwater usage are discussed in Subsect. 6.5 "Risk Characterization" of the Phase I report and tabulated in Appendix 6C and 6D of that report. The results found there are presented in Tables 1.1, 1.2, and 1.3 of this volume and are summarized in Exhibits 1.51 and 1.52.

Exhibit 1.51. Excess lifetime cancer risk and hazard indices under residential use from chemicals in groundwater as reported in the Phase I Site Investigation

Well Category and Exposure Assumptions[a]	Excess Lifetime Cancer Risk			Hazard Index		
	Ingestion	Inhalation[b]	Total	Ingestion	Inhalation[b]	Total
Average Exposure Assumptions						
Residential	2×10^{-5}	2×10^{-5}	4×10^{-5}	0.6	0.3	0.9
Monitoring	1×10^{-5}	6×10^{-6}	2×10^{-5}	1.1	<0.1	1.1
TVA	5×10^{-5}	7×10^{-7}	6×10^{-5}	0.5	<0.1	0.5
Maximum Exposure Assumptions						
Residential	3×10^{-4}	4×10^{-4}	7×10^{-4}	2.0	0.7	2.7
Monitoring	1×10^{-4}	9×10^{-5}	2×10^{-4}	3.8	0.1	3.9
TVA	7×10^{-4}	2×10^{-5}	7×10^{-4}	1.7	<0.1	1.7

[a] See Chapter 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 Subsect. 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 Subsect. 6.4.5.1 in CH2M Hill 1991a.

Exhibit 1.52. Excess total cancer incidence under residential use from radionuclides in groundwater as reported in the Phase I Site Investigation

Well Category[a]	Average Exposure Assumptions[b]	Upperbound Exposure Assumptions
Residential	4×10^{-6}	5×10^{-5}
Monitoring	3×10^{-6}	5×10^{-5}
TVA	1×10^{-5}	3×10^{-4}

[a] See Chapter 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 Subsect. 6.5.2.2 in CH2M Hill 1991a descriptions of exposure assumptions and dose calculations.

As shown in Exhibit 1.51, total ELCRs from residential use of off-site groundwater exceed the *de minimis* level defined in the Methods Document (i.e., 1×10^{-6}) for all well categories under average and maximum exposure assumptions but are within the EPA acceptable risk range for site-related exposures (i.e., 1×10^{-6} to 1×10^{-4}; EPA 1999c) under average exposure assumptions. Also as shown in Exhibit 1.51, systemic toxicity (as indicated by HI) for all well categories exceed the *de minimis* level defined in the

Methods Document and the EPA acceptable value (i.e., 1) under maximum exposure assumptions but only for the monitoring well category under average exposure assumptions.

As shown in Tables 1.1 and 1.2, the contaminants in groundwater contributing most to ELCRs and systemic toxicity are consistent between well categories. For ELCR, the primary contaminants over all well categories are TCE, arsenic, and bis(2-ethylhexyl)phthalate. For systemic toxicity, the primary contaminants over all well categories are various metals, carbon tetrachloride, and bis(2-ethylhexyl)phthalate.

As shown in Exhibit 1.52, 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. However, total cancer incidence values are within the EPA risk range for all well categories and exposure assumptions except the TVA group under upper-bound exposure assumptions. As shown in Table 1.3, the primary contaminants in groundwater over all well categories are ^{234}U, ^{238}U, and ^{99}Tc.

1.2.2.2 Results of the Phase II Site Investigation

The Phase II investigation (CH2M Hill 1992) further evaluated the nature and extent of off-site contamination originating from PGDP and characterized source control units by identifying contaminant migration routes that may contribute to off-site contamination. The Phase II investigation used this information to develop a risk assessment described as a public health and ecological assessment (PHEA) (CH2M Hill 1991b).

In the PHEA, risks to human health from exposure to all media under several scenarios were assessed. However, because the source control unit investigations summarized in Subsects. 1.1.1 supercede the source unit information in the PHEA, this subsection will focus on the groundwater risks presented in the PHEA.

Data aggregates used in the PHEA were similar to those in the Phase I Site Investigation except sampling results from on-site monitoring wells (MW) and from individual wells were considered. (The results from the individual wells are described as being representative of contamination found in the RGA at off-site locations.) The data aggregates, including a description of the location of the individual wells, are as follows:

- On-site Monitoring Wells
- Residential Wells
- Off-site Monitoring Wells
- TVA Wells
- MW 134 – located near the center of the Northwest TCE Plume
- MW 144 – located near the center of the Northeast TCE Plume
- MW 179 – located between the Northwest and Northeast TCE Plumes in an isolated area of TCE contamination in groundwater.
- MW 200 – located to the east of the Northwest TCE Plume

As with the Phase I Site Investigation, the PHEA only assessed the residential use of groundwater. Exposure routes were ingestion of groundwater and inhalation of vapors emitted by groundwater during

household use. Results of this assessment are summarized in Exhibit 1.53 and presented in detail in Tables 1.4 and 1.5. As shown in Exhibit 1.53, the ELCR for each data aggregate exceeded the *de minimis* level established in the Methods Document and exceeded the EPA acceptable range for each of the large data aggregates and for MW 144 and 200. Contaminants driving ELCR were similar across the data aggregates and were TCE, arsenic, and beryllium. Systemic toxicity exceeded the EPA and *de minimis* level for each of the large data aggregates and for MW 200. Contaminants driving systemic toxicity were more variable across the data aggregates and included antimony, arsenic, beryllium, chromium, manganese, silver, thallium, vanadium, 1,2-DCE, and carbon tetrachloride. Excess cancer risk from exposure to radionuclides in groundwater did not exceed the EPA acceptable range for any data aggregate; however, the ELCR did exceed the *de minimis* level for all data aggregates but MW 144. Radionuclides driving ELCR across aggregates were ^{99}Tc, ^{234}U, ^{238}U, ^{237}Np, and ^{239}Pu.

Important conclusions from the PHEA were as follows.

- Much of the ELCR for groundwater drawn from wells outside the TCE plumes was from "naturally occurring" concentrations of arsenic and beryllium in unfiltered water samples. For example, the ELCR under residential use of groundwater from arsenic and beryllium in samples from reference wells (results not shown in Exhibit 1.53) was 5×10^{-4}.

- Much of the systemic toxicity was associated with metals found in the unfiltered groundwater samples. Because the distribution of metals in groundwater results did not indicate the presence of a metals plume, the concentrations of the metals and the resulting systemic toxicity may be due to sampling techniques that resulted in elevated particulate levels in the samples.

- Although on-site groundwater is not used as a source of potable water, the concentrations are expected to remain elevated above acceptable risk levels for potable use on-site based on the high source concentrations.

1.2.2.3 Results of the Northwest Plume Baseline Human Health Risk Assessment

In 1994, DOE determined that information was sufficient to implement an interim record of decision (ROD) to address the high concentration area of the Northwest Plume (DOE 1993b). To support this determination, a BHHRA addressing contamination found in the RGA in the on-site and off-site areas encompassed by the Northwest Plume was completed.

To facilitate the completion of the Northwest Plume Data, data aggregates composed of validated sampling results from monitoring wells were compiled on the basis of the concentration of TCE present in groundwater samples drawn from the well. The data aggregates differed from those used in the PHEA and are listed below. Note that, unlike the PHEA, results from individual wells were not assessed as part of the Northwest Plume BHHRA and that a separate analysis was performed for naturally occurring metals.

- High TCE/^{99}Tc Plume – wells completed in the area of highest TCE concentrations.

- TCE/^{99}Tc Plume – wells completed in the plume but outside the high TCE concentration area.

- Outside the plume – wells completed to the west of the plume.

- Reference – wells completed to the southeast of the PGDP.

- Naturally Occurring Metals – a summary completed for each of the above four data aggregates for inorganic chemicals that are naturally occurring.

Exhibit 1.53. Summary of risk estimates for current and future residential use of groundwater from the PHEA

Well Category	Chemical		Radiological
	Excess Cancer Risk	Hazard Index	Excess Cancer Risk
Onsite Monitoring Wells			
Ingestion	2×10^{-2}	22	3×10^{-5}
Inhalation	4×10^{-2}	6.5	
Total	6×10^{-2}	28	3×10^{-5}
Major Contributors	Vinyl chloride, trichloroethene	Antimony, 1,2-dichloroethene	Tc-99, U-238, U-234, Np-237
Residential Wells			
Ingestion	4×10^{-4}	2.4	2×10^{-5}
Inhalation	3×10^{-4}	0.9	
Total	7×10^{-4}	3.3	2×10^{-5}
Major Contributors	Trichloroethene, arsenic, beryllium	Carbon tetrachloride, antimony, thallium	Tc-99, U-234, U-238, Np-237
Offsite Monitoring Wells			
Ingestion	4×10^{-4}	2.5	2×10^{-5}
Inhalation	4×10^{-5}	0.08	
Total	5×10^{-4}	2.6	2×10^{-5}
Major Contributors	Arsenic, beryllium, trichloroethene	Silver, beryllium, chromium	U-234, U-238, Pu-239
TVA Wells			
Ingestion	3×10^{-3}	8.8	6×10^{-5}
Inhalation	3×10^{-7}	0.04	
Total	3×10^{-3}	8.8	6×10^{-5}
Major Contributors	Arsenic, beryllium, trichloroethene	Antimony, arsenic, manganese	U-238, U-234
MW 134			
Ingestion	6×10^{-5}	0.3	7×10^{-6}
Inhalation	1×10^{-6}	0.02	
Total	6×10^{-5}	0.3	7×10^{-6}
Major Contributors	Arsenic, beryllium, trichloroethene	Manganese, barium, arsenic	Tc-99, U-234, U-238
MW 144			
Ingestion	2×10^{-4}	0.4	3×10^{-7}
Inhalation	5×10^{-5}	0.07	
Total	3×10^{-4}	0.5	3×10^{-7}
Major Contributors	Arsenic, trichloroethene, beryllium	Arsenic, manganese, barium	Tc-99, U-234
MW 179			
Ingestion	7×10^{-5}	0.53	3×10^{-5}
Inhalation	1×10^{-6}	0.02	
Total	7×10^{-5}	0.6	3×10^{-5}
Major Contributors	Arsenic, beryllium, trichloroethene	Manganese, chromium, arsenic	Tc-99, U-238
MW 200			
Ingestion	5×10^{-4}	3.1	6×10^{-6}
Inhalation	1×10^{-6}	0.02	
Total	5×10^{-4}	3.2	6×10^{-6}
Major Contributors	Beryllium, arsenic, trichloroethene	Chromium, vanadium, beryllium	Tc-99, U-238

Notes:
Information taken from Table 3-13 of the PHEA.

As with the PHEA and the Phase I Site Investigation risk assessments, this BHHRA only considered rural residential use of groundwater. However, the exposure routes considered were more extensive and included the modeled concentrations of contaminants found in farm products. These routes are listed below.

- Ingestion of groundwater.
- Dermal contact with groundwater while bathing.
- Inhalation of vapors emitted by groundwater during household use.
- Consumption of vegetables irrigated with groundwater.
- Consumption of meat products (i.e., beef) from animals ingesting groundwater and consuming forage irrigated with groundwater.
- Consumption of milk from cows ingesting groundwater and consuming forage irrigated with groundwater.

The results of the risk assessment are summarized in Exhibits 1.54 though 1.57 and are presented in more detail in Tables 1.6 through 1.9. As shown in Exhibit 1.54, total ELCRs from residential use of groundwater taken from the Northwest Plume exceed the *de minimis* level defined in the Methods Document (i.e., 1×10^{-6}) for all well groups. However, only the High TCE/^{99}Tc Plume group has an ELCR that exceeds the upper limit of the EPA acceptable risk range. Also, as shown in Exhibit 1.55, only the High TCE/^{99}Tc Plume group has a total hazard index that exceeds the *de minimis* level defined in the Methods Document and the upper limit of the EPA acceptable risk range (i.e., 1). For both ELCR and hazard index, the exposure routes contributing most were ingestion of groundwater and consumption of vegetables irrigated with groundwater.

Exhibit 1.56 displays the contaminants in groundwater contributing most to ELCR. For the High TCE/^{99}Tc Plume category, the contaminants contributing most to ELCR were bis(2-chloroethyl)ether and TCE. However, for the TCE/^{99}Tc and Outside the Plume groups, the ELCR was driven by dieldrin (detected in only 2 of 20 samples) and uranium radioisotopes, respectively. Exhibit 1.57 shows the contaminants in groundwater contributing most to the systemic toxicity. For the High TCE/^{99}Tc Plume category, the contaminants contributing most to the HI varied from those contributing to the ELCR and were carbon tetrachloride, chloroform, and bromodichloromethane. However, for the other areas, the driving contaminants for HI were similar with the addition of 2-butanone as a driving contaminant for the TCE/^{99}Tc Plume group.

Exhibits 1.56 and 1.57 also show that naturally occurring metals pose levels of ELCR and HI that exceed the *de minimis* and EPA acceptable levels. As shown in these exhibits, the ELCR and HI for the High TCE/^{99}Tc Plume group for naturally occurring metals was 3×10^{-4} and 3.7, respectively, with arsenic driving ELCR and copper, arsenic, and cyanide driving systemic toxicity. Results for other groups were similar as shown in Tables 1.7 and 1.9.

Important conclusions from the Northwest Plume BHHRA include:

- Contaminants that are infrequently detected drive the risk for some groups. Examples are bis(2-chloroethyl)ether which is a risk driver for ELCR in the High TCE/^{99}Tc Plume group and was detected in one of 44 samples, and dieldrin which is risk driver for ELCR in the TCE/^{99}Tc Plume group and was detected in only 2 of 20 samples.

Exhibit 1.54. Excess lifetime cancer risk under residential use from chemicals in groundwater as reported in the Northwest Plume BRA

Well Category[a]	Excess Lifetime Cancer Risk					
	Ingestion	Inhalation	Dermal	Vegetables	Beef & Milk[b]	Total
High TCE/^{99}Tc Plume	3×10^{-4}	2×10^{-4}	1×10^{-5}	2×10^{-3}	1×10^{-5}	3×10^{-3}
TCE/^{99}Tc Plume	3×10^{-5}	9×10^{-6}	1×10^{-6}	5×10^{-5}	4×10^{-5}	1×10^{-4}
Outside the Plume	1×10^{-5}	NV[c]	2×10^{-7}	2×10^{-6}	3×10^{-7}	1×10^{-5}
Reference[d]	3×10^{-5}	NV	3×10^{-6}	1×10^{-5}	6×10^{-6}	5×10^{-5}
Naturally Occurring Metals[e]	2×10^{-4}	NV	6×10^{-7}	7×10^{-5}	6×10^{-6}	3×10^{-4}

[a] Wells were grouped according to the concentration of trichloroethene found in groundwater samples. See Table 2.1 *in* DOE 1993a for a list of wells by group.
[b] Risks presented are the sum of risks from consumption of milk and meat from cows drinking contaminated groundwater.
[c] NV indicates no value was reported for the exposure route in the assessment.
[d] Contaminant concentrations in other well categories were compared to concentrations in reference wells. As a result of this comparison, some contaminants were removed from the analysis; therefore, risks for the categories High trichloroethene (TCE)/^{99}Tc Plume, TCE/^{99}Tc Plume, and Outside the Plume may be greater than reported.
[e] Naturally occurring metals were assessed separately for each well category. The results presented are for the High TCE/^{99}Tc category. Results for other categories were similar.

Exhibit 1.55. Hazard indices under residential use from chemicals in groundwater as reported in the Northwest Plume BRA

Well Category[a]	Hazard Index					
	Ingestion	Inhalation	Dermal	Vegetables	Beef & Milk[b]	Total
High TCE/^{99}Tc Plume	0.4	<0.1	<0.1	1.5	<0.1	1.9
TCE/^{99}Tc Plume	0.1	<0.1	<0.1	0.4	0.1	0.6
Outside the Plume	0.3	NV[c]	<0.1	<0.1	<0.1	0.4
Reference[d]	0.3	<0.1	<0.1	0.1	<0.1	0.4
Naturally Occurring Metals[e]	1.2	NV	<0.1	1.8	0.7	4.5

[a] Wells were grouped according to the concentration of trichloroethene found in groundwater samples. See Table 2.1 *in* DOE 1993a for a list of wells by group.
[b] Risks presented are the sum of risks from consumption of milk and meat from cows drinking contaminated groundwater.
[c] NV indicates no value was reported for the exposure route in the assessment.
[d] Contaminant concentrations in other well categories were compared to concentrations in reference wells. As a result of this comparison, some contaminants were removed from the analysis; therefore, risks for the categories High TCE/^{99}Tc Plume, TCE/^{99}Tc Plume, and Outside the Plume may be greater than reported.
[e] Naturally occurring metals were assessed separately for each well category. The results presented are for the High TCE/^{99}Tc category. Results for other categories were similar.

Exhibit 1.56. Contaminants[a] contributing to excess lifetime cancer risk under residential use by well category as reported in the Northwest Plume BRA

Well Category[b]	Excess Lifetime Cancer Risk	
	Contaminants	Total Risk
High TCE/^{99}Tc Plume	bis(2-chloroethyl)ether (52%); trichloroethene (41%) bromodichloromethane (3%); carbon tetrachloride (2%) technetium-99 (1%)	3×10^{-3}
TCE/^{99}Tc Plume	dieldrin (60%); trichloroethene (18%); 1,2-dichloroethane (15%); bis(2-ethylhexyl)phthalate (4%) technetium-99 (2%)	1×10^{-4}
Outside the Plume	uranium-238 (54%); uranium-234 (21%) bis(2-ethylhexyl)phthalate (21%)	1×10^{-5}
Reference[c]	bis(2-ethylhexyl)phthalate (98%)	5×10^{-5}
Naturally Occurring Metals[d]	arsenic (100%)	3×10^{-4}

[a] Contaminants contributing more than 1% of total risk are shown.
[b] Wells were grouped according to the concentration of trichloroethene found in groundwater samples. See Table 2.1 *in* DOE 1993a for a list of wells by group.
[c] Contaminant concentrations in other well categories were compared to concentrations in reference wells. As a result of this comparison, some contaminants were removed from the analysis; therefore, total risks for the categories High TCE/^{99}Tc Plume, TCE/^{99}Tc Plume, and Outside the Plume may be greater than reported.
[d] Naturally occurring metals were assessed separately for each well category. Contaminants listed here were for naturally occurring metals found in the High TCE/^{99}Tc Plume well category.

Exhibit 1.57. Contaminants[a] contributing to hazard index under residential use by well category as reported in the Northwest Plume BRA

Well Category[b]	Hazard Index	
	Contaminants	Total Hazard
High TCE/^{99}Tc Plume	carbon tetrachloride (68%); chloroform (18%); bromodichloromethane (9%), uranium (4%)	1.9
TCE/^{99}Tc Plume	2-butanone (48%); dieldrin (34%); uranium (10%) bis(2-ethylhexyl)phthalate (6%); xylene (2%)	0.6
Outside the Plume	uranium (94%); bis(2-ethylhexyl)phthalate (6%)	0.4
Reference[c]	bis(2-ethylhexyl)phthalate (95%); uranium (5%)	0.4
Naturally Occurring Metals[d]	copper (40%); arsenic (33%); cyanide (16%); silver (6%); barium (4%); cadmium (2%)	3.7

[a] Only those contaminants contributing more than 1% of total risk are shown.
[b] Wells were grouped according to the concentration of trichloroethene found in groundwater samples. See Table 2.1 *in* DOE 1993a for a list of wells by group.
[c] Contaminant concentrations in other well categories were compared to concentrations in reference wells. As a result of this comparison, some contaminants were removed from the analysis; therefore, total risks for the categories High TCE/^{99}Tc Plume, TCE/^{99}Tc Plume, and Outside the Plume may be greater than reported.
[d] Naturally occurring metals were assessed separately for each well category. Contaminants listed here were for naturally occurring metals found in the High TCE/^{99}Tc Plume well category.

- Inorganic chemicals that may have been measured at naturally occurring levels pose considerable ELCR and systemic toxicity. Arsenic contributes significantly to both ELCR and systemic toxicity.

- The human health risk associated with the Northwest Plume is essentially a carcinogenic risk due to elevated concentrations of TCE in groundwater.

1.2.2.4 Results of the Northwest Dissolved Phase Plume Baseline Risk assessment

The most recent BHHRA completed for the GWOU was performed to support a planned interim ROD that was to address the dissolved phase of the Northwest Plume. Unlike the earlier integrator unit assessments, which estimated risk using current contaminant concentrations, this assessment estimated risk using current contaminant conditions and using concentrations derived from a numeric transport model that assumed that the on-site sources of the Northwest Plume were contained. However, similar to the Northwest Plume BHHRA discussed in Subsect. 1.2.2.3, this BHHRA used data aggregates based upon the TCE concentration currently present in the samples drawn from the RGA. These data aggregates are summarized in the following list.

- Plume Centroid – analogous to the High TCE/^{99}Tc Plume group used in the previous BHHRA except only results from wells to the north of the security fence (the assumed location of the containment system) were considered.

- Dissolved Plume – analogous to the TCE/^{99}Tc Plume group used in the previous BHHRA except only results from wells to the north of the security fence were considered.

- Outside and West of the plume – analogous to the Outside the Plume group used in the previous BHHRA.

- Near the Shawnee Steam Plant – not considered in the previous BHHRA.

- Near the Ohio River – not considered in the previous BHHRA.

[Note that the report containing the Northwest Dissolved Phase Plume BHHRA (DOE 1994c) also contains an ecological risk assessment completed "to provide a basis for decisions concerning the need for remediation based on risks to nonhuman receptors." A summary of this ecological risk assessment can be found in Attachment 9 of this baseline risk assessment for the GWOU.]

In the Northwest Dissolved Phase Plume BHHRA, two use scenarios were assessed. These were industrial and rural residential use. Under industrial use, the only exposure route considered was ingestion of groundwater. However, under rural residential use, the list of exposure routes was extensive and included several recreational use exposure routes. The routes considered under residential use are listed below.

- Ingestion of groundwater.

- Inhalation of vapors emitted from groundwater during household use.

- Dermal contact with groundwater while bathing.

- Incidental ingestion of groundwater contaminated via irrigation with groundwater.

- Dermal contact with water while swimming or wading in ponds filled with groundwater,

- Consumption of fish raised in ponds filled with groundwater.

- Consumption of vegetables and produce raised in areas irrigated with groundwater.

- Consumption of beef from cattle contaminated by consuming vegetation irrigated with groundwater, consuming soil contaminated with groundwater while on pasture, and ingestion groundwater.

- Consumption of dairy products (i.e., milk) from cows contaminated by consuming vegetation (pasture and concentrate) irrigated with groundwater, consuming soil while on pasture, and ingesting groundwater.

- Consumption of game products (i.e., venison) contaminated by consumption of vegetation irrigated with groundwater and ingestion of groundwater.

The results of the BHHRA for the residential scenario under current conditions are summarized in Exhibits 1.58 through 1.61 and presented in detail in Tables 1.10 and 1.11. As shown in Exhibit 1.58, the total ELCRs for rural residential use of groundwater for all well groups exceed the *de minimis* level defined in the Methods Document (i.e., 1×10^{-6}) and are greater than the upper end of the EPA acceptable risk range for all well groups except Outside and West of the Plume and Near the Ohio River. However, as shown in Exhibit 1.59 only the Plume Centroid, Dissolved Plume, and Near Shawnee Steam Plant well groups have a total hazard index that exceeds the *de minimis* level defined in the Methods Document and the EPA acceptable value (i.e., 1). For both ELCR and hazard index, the exposure routes contributing most were ingestion of groundwater and consumption of either vegetables or animal products (i.e., biota) raised using contaminated groundwater.

As shown in Exhibit 1.60 and 1.61, the contaminants in groundwater contributing most to ELCR and HI varied between the well groups. For the Plume Centroid group, the contaminants contributing most (i.e., more than 50% of total) to ELCR and HI were vinyl chloride and carbon tetrachloride, respectively. For the Dissolved Phase Plume group, the contaminants contributing most were dieldrin for ELCR and manganese and dieldrin for HI. For the Outside and West of the Plume group, the contaminants contributing most were ^{238}U and nitrate as nitrogen. For the Near Shawnee Steam Plant group, the contaminants contributing the most to ELCR and HI were arsenic and manganese, respectively. Finally, for the Near the Ohio River group, the driving contaminant for both ELCR and HI as 1,1,1-trichloroethane (TCA).

Conclusions from the Northwest Dissolved Phase Plume BHHRA concerning risks posed by current contaminant concentrations were similar to those for the Northwest Plume BHHRA and will not be repeated here. Conclusions concerning risks posed under modeled future concentrations were unique to this assessment and are as follows:

- ELCR from organic and radionuclide COPCs and systemic toxicity from organic COPCs will decrease over time once the sources of the Northwest Plume are isolated from the dissolved phase of the plume. However, risk and systemic toxicity from exposure to inorganic COPCs may not decrease.

- The concentrations of inorganic chemicals found and the significant risks to human health (i.e., ELCR greater than 1×10^{-4} and HI greater than 1) posed by these inorganic chemicals does not appear to be related releases from the PGDP. These inorganic chemicals may be present at or near natural concentrations in all well groups.

1.2.2.5 Summary of Groundwater Integrator Unit Investigations

The groundwater integrator unit investigations summarized in the previous subsections indicate that the dominant contaminants in groundwater at the PGDP are TCE, the TCE breakdown products, and,

Exhibit 1.58. Excess lifetime cancer risk under residential use from chemicals in groundwater as reported in the Northwest Dissolved Phase Plume BRA

Well Category[a]	Excess Lifetime Cancer Risk					
	Ingestion	Inhalation	Dermal[b]	Vegetables	Biota[c]	Total[d]
Plume Centroid	1×10^{-3}	8×10^{-5}	1×10^{-6}	3×10^{-3}	4×10^{-4}	5×10^{-3}
Dissolved Plume	2×10^{-4}	2×10^{-4}	6×10^{-6}	2×10^{-4}	2×10^{-3}	3×10^{-3}
Outside and West of Plume	9×10^{-6}	NV[e]	1×10^{-6}	2×10^{-5}	7×10^{-6}	4×10^{-5}
Near Shawnee Steam Plant	6×10^{-4}	1×10^{-5}	2×10^{-6}	5×10^{-4}	2×10^{-4}	1×10^{-3}
Near Ohio River	5×10^{-6}	1×10^{-7}	3×10^{-7}	2×10^{-6}	5×10^{-6}	1×10^{-5}

[a] Wells were grouped according to the concentration of trichloroethene found in groundwater samples and according to prominent offsite features. See Table 5.11 *in* DOE 1994a for a list of wells by group.
[b] Risks presented are the sum of risks from dermal contact while bathing and dermal contact while swimming.
[c] Risks presented are the sum of risks from consumption of milk and meat from cows drinking contaminated groundwater and eating pasture irrigated contaminated groundwater, ingestion of venison from deer drinking contaminated groundwater and eating pasture irrigated with contaminated groundwater, and consumption of fish raised in ponds filled with contaminated groundwater.
[d] Total risks also include risks from ingestion of soil contaminated through irrigation with contaminated groundwater. The soil ingestion risks are not presented separately.
[e] NV indicates no value was reported for the exposure route in the assessment.

Exhibit 1.59. Hazard indices (child) under residential use from chemicals in groundwater as reported in the Northwest Dissolved Phase Plume BRA

Well Category[a]	Hazard Index					
	Ingestion	Inhalation	Dermal[b]	Vegetables	Biota[c]	Total[d]
Plume Centroid	3.0	NV[e]	0.2	0.8	2.0	6.0
Dissolved Plume	6.0	<0.1	0.7	0.4	9.0	20.0
Outside and West of Plume	0.2	NV	<0.1	<0.1	<0.1	0.3
Near Shawnee Steam Plant	20.0	<0.1	2.0	0.7	8.0	30.0
Near Ohio River	<0.1	<0.1	<0.1	<0.1	<0.1	0.1

[a] Wells were grouped according to the concentration of trichloroethene found in groundwater samples and according to prominent offsite features. See Table 5.11 *in* DOE 1994a for a list of wells by group.
[b] Hazard indices presented are the sum of risks from dermal contact while bathing and dermal contact while swimming.
[c] Hazard indices presented are the sum of risks from consumption of milk and meat from cows drinking contaminated groundwater and eating pasture irrigated contaminated groundwater, ingestion of venison from deer drinking contaminated groundwater and eating pasture irrigated with contaminated groundwater, and consumption of fish raised in ponds filled with contaminated groundwater.
[d] Total hazard indices are rounded to one significant digit. This value also includes risks from ingestion of soil contaminated through irrigation with contaminated groundwater. The soil ingestion risks are not presented separately.
[e] NV indicates no value was reported for the exposure route in the assessment.

Exhibit 1.60. Contaminants[a] contributing to excess lifetime cancer risk under residential use by well category as reported in the Northwest Dissolved Phase Plume Report

Well Category[b]	Excess Lifetime Cancer Risk	
	Contaminants	Total Risk
Plume Centroid	vinyl chloride (81%); bis(2-chloroethyl)ether (9%); trichloroethene (5%); technetium-99 (2%)	5×10^{-3}
Dissolved Phase	dieldrin (72%); trichloroethene (17%); vinyl chloride (5%); 1,1,2-trichloroethane (1%); 1,2-dichloroethane (1%); carbon tetrachloride (1%)	3×10^{-3}
Outside and West of Plume	uranium-238 (66%); bis(2-ethylhexyl)phthalate (24%); uranium-234 (3%)	4×10^{-5}
Near Shawnee Steam Plant	arsenic (50%); vinyl chloride (48%); technetium-99 (2%)	1×10^{-3}
Near Ohio River	1,1,2-trichloroethane (100%)	1×10^{-5}

[a] Contaminants contributing more than 1% of total risk are shown.
[b] Wells were grouped according to the concentration of trichloroethene found in groundwater samples and according to prominent offsite features. See Table 5.11 *in* DOE 1994a for a list of wells by group.

Exhibit 1.61. Contaminants[a] contributing to hazard index (child) under residential use by well category as reported in the Northwest Plume Dissolved Phase Report

	Hazard Index	
Well Category[b]	**Contaminants**	**Total Hazard[c]**
Plume Centroid	carbon tetrachloride (61%); manganese (31%); copper (6%)	6.0
Dissolved Phase	manganese (47%); dieldrin (42%); carbon tetrachloride (6%); 1,1,2-trichloroethane (2%)	20.0
Outside and West of Plume	nitrate as nitrogen (71%); bis(2-ethylhexyl)phthalate (29%)	0.3
Near Shawnee Steam Plant	manganese (82%); arsenic (14%); nickel (2%); barium (1%)	30.0
Near Ohio River	1,1,2-trichloroethane (100%)	0.1

[a] Contaminants contributing more than 1% of total risk are shown.
[b] Wells were grouped according to the concentration of trichloroethene found in groundwater samples and according to prominent offsite features. See Table 5.11 *in* DOE 1994a for a list of wells by group.
[c] Values are rounded to one significant digit.

possibly, carbon tetrachloride. However, there are several other organic compounds that are infrequently detected but pose considerable risk. Additionally, these investigations indicate that although various inorganic chemicals pose considerable risk, these chemicals may actually not be related to releases from the PGDP but are at naturally occurring concentrations.

1.2.3 Other Studies

In addition to the aforementioned source control unit and integrator unit investigations, there are three reports that consider and discuss the environmental conditions around PGDP that were used in the preparation of this BHHRA. Because these studies were primarily used to complete the exposure assessment step of the BHHRA and do not contain either risk assessment or risk evaluation results, the information in these reports is not summarized in detail here. These reports are as follows.

- *Report of the Paducah Gaseous Diffusion Plant, Groundwater Investigation Phase III* (Claussen *et al.* 1992a)

- *Northeast Plume Preliminary Characterization Summary Report* (DOE 1995a)

- *Environmental Investigations at the Paducah Gaseous Diffusion Plant and Surrounding Area, McCracken County, Kentucky* (COE 1994).

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2. DATA EVALUATION

2.1 INTRODUCTION

This subsection describes the sources of data, the procedures used to screen and segregate the data to develop a list of COPCs, and the methods used to derive representative concentrations for the COPCs in environmental media and biota under both current and future conditions. Additionally, this section describes the site characterization data used in the exposure assessment performed in Sect. 3.

2.2 SOURCES OF DATA

All data used to estimate current contaminant concentrations at the various points of exposure were from the completed field investigations described in Sect. 1 of this BHHRA or from ongoing monitoring programs at PGDP. These data were taken in electronic form from the Oak Ridge Environmental Information System (OREIS) as maintained by the PGDP and are provided in electronic form in a compact disk supplied with this report. These data and geophysical and geochemical information were also used to perform environmental fate and transport modeling to provide estimates of future contaminant concentrations at selected points of exposure. Finally, the current and future contaminant concentrations were used in biological fate and transport models to estimate contaminant concentrations in animals and vegetables. Additional information concerning the environmental fate and transport modeling is in the GWOU feasibility study. Additional information concerning the biological fate and transport models is in Sect. 3 of this BHHRA.

After accessing the data on OREIS, data were placed into aggregates based on four parameters. Parameters used were:

- geographical location of sampling station,
- depth at which sample was taken,
- type of sampling station (including type of sample), and
- sample preparation.

2.2.1 Consideration of Geographical Location of Sampling Station

Based upon the geographical location of the sampling station, the data were assigned to one or more of fourteen areas. These areas and their definitions are summarized in the following list.

- Area a – Inside TCE contaminated area at C-400 Building – Inside industrialized area
- Area b – Inside the Northwest TCE Plume – Inside industrialized area (i.e., west main plant)
- Area c – Inside the Northeast TCE Plume – Inside industrialized area (i.e., east main plant)
- Area d – Outside the TCE Plumes – South of C-400 in industrialized area
- Area e – Inside the Northwest TCE Plume – Outside industrialized area
- Area f – Inside the Northeast TCE Plume – Outside industrialized area
- Area g – Outside the TCE Plumes – West of industrialized area (i.e., west of plume)
- Area h – Outside the TCE Plumes – East of industrialized area (i.e., east of plume)
- Area i – Outside the TCE Plumes – North of industrialized area (i.e., between the plumes)
- Area j – Outside the TCE Plumes - Tennessee Valley Authority area (TVA)
- Area k – Outside the TCE Plumes – South of industrialized area above terrace
- Area l – Inside plant area – Composed of Areas a, b, c, and d
- Area m – Outside plant area – Composed of Areas e, f, g, h, i, j, and k
- Area n – All groundwater – Composed of Areas l and m

Eleven of these areas (i.e., Areas a through k; see Fig. 2.1) were used in this BHHRA to ensure that the summary statistics (i.e., average contaminant concentrations) derived were comparable to those developed during the BHHRA previously completed as part of the investigations of the Northwest Plume and to let this BHHRA estimate lists of COCs for specific areas at and around the PGDP. The remaining three areas (i.e., Areas l through n) were used to investigate the average risk posed through use of water drawn from the larger areas to let this BHHRA develop plant-wide lists of COCs. Table 2.1 provides a list of the sampling stations assigned to each of these areas, Fig. 2.1 contains a map showing Areas a through k, and Plates 1 and 2 depicts the stations within each area.

2.2.2 Consideration of Depth of Sampling

Data were also assigned to one of seven groups based upon a combination of the depth at which the sample was collected and the characteristics of the subsurface in the area of the sampling station. These groups and their definitions are summarized in the following list. For a discussion of the various hydrogeological units, including diagrams, please see the Data Summary Report contained in Appendix A of the GWOU FS report.

- HU1 – data from a sample collected in Hydrogeological Unit 1
- HU2 – data from a sample collected in Hydrogeological Unit 2
- HU3 – data from a sample collected in Hydrogeological Unit 3
- HU4 – data collected from a sample collected in Hydrogeological Unit 4
- HU5 – data collected from a sample collected in Hydrogeological Unit 5
- HU6 – data collected from a sample collected in Hydrogeological Unit 6
- Other – data from a sample collected from a hydrogeological unit not included above (i.e., Terrace Gravel, Porters Creek Clay, and Eocene Sands)
- UCRS – data from samples assigned to HU1, HU2, or HU3
- RGA – data from samples assigned to HU4 or HU5
- McNairy Formation – data from samples assigned to HU6

Data were assigned to these groups to remove bias related to the geophysical and geochemical environment, to allow for the estimation of risk from use of water drawn from the two major PGDP aquifers, and to derive risk results that were comparable to those from previous integrator unit BHHRAs. Table 2.2 provides a list of samples, along with their sampling stations, assigned to each depth group. Note that some sampling stations have samples assigned to more than one depth group because these sampling stations were sampled at multiple depths.

2.2.3 Consideration of Type of Sampling Station

Data were segregated into two groups on the basis of the method used to reach groundwater for sample collection. The groups used were data from samples collected from monitoring wells and data from samples collected using driven rod technology. Data were segregated using these criteria to let the BHHRA derive risk estimates from samples that are similar to those that may be collected from

residential wells (i.e., from monitoring wells) and to allow the BHHRA to address the concentration bias associated with the higher particulate levels seen in samples collected using driven rods. Table 2.3 contains a list of samples assigned to each of these groups.

2.2.4 Consideration of Sample Preparation

Data were segregated based upon the filtering performed prior to laboratory analysis. The groups used were data from unfiltered samples and data from filtered samples. Data were segregated using these criteria to allow the BHHRA to derive risk estimates that meet the requirements set forth in the Methods Document and to let the BHHRA examine bias associated with particulate concentrations in samples. Note that consistent with the requirements in the Methods Document, all risk values presented in Sect. 5 of this report were compiled using results from unfiltered (i.e., total) samples. The results for filtered samples are only considered in the uncertainty analysis presented in Sect. 6 of this BHHRA.

2.3 GENERAL DATA EVALUATION CONSIDERATIONS

The data described previously were evaluated to ensure that the data were appropriate for use in baseline risk assessments and to reduce the data set to a list of COPCs. A general description of this evaluation is provided in this subsection. A graphical presentation of this evaluation is in Fig. 2.2.

Data evaluation was performed in eight steps:

(1) **Evaluation of sampling**. Data were examined to ensure that the samples from which the data were derived were collected using sampling methods that were adequate to determine the nature and extent of contamination.

(2) **Evaluation of analytical methods**. Methods used to analyze samples were evaluated to determine if they were those approved by EPA.

(3) **Evaluation of sample quantitation limits (SQLs).** The SQLs for each analyte and sample were examined to determine if they were below the concentration at which the contaminant may pose a threat to human health or the environment. If the SQL for an analyte was greater than the concentration that may pose a threat to human health and that analyte was not detected in any sample, then the data for that chemical were deemed of insufficient quality, and only a qualitative assessment for that chemical is presented in this assessment. In developing the qualitative assessment for such chemicals, the SQL for the chemical was used in the qualitative assessment if historical or process knowledge indicated that the chemical could potentially be present. If historical or process knowledge indicated that the chemical is 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. Tagged data were evaluated following rules in Exhibits 5-4 and 5-5 of RAGS.

(5) **Elimination of chemicals not detected**. For each sample, any chemical not detected in at least one sample using an appropriate SQL was eliminated from the data set.

(6) **Examination of toxicity of detected analytes**. Each analyte's maximum detected concentration in the data set was compared to the analyte's residential use human health risk-based screening value [i.e., residential use risk-based concentration (RBC)]. Screening values used in this comparison were

derived following methods described in the Methods Document. To ensure that the residential use RBCs used in this step were conservative, routes of exposure used to develop the criteria for chemicals were ingestion of potentially contaminated groundwater, dermal contact with potentially contaminated groundwater, and inhalation of vapors emitted by potentially contaminated groundwater during household use. Direct contact exposure routes used to develop RBCs for radionuclides were ingestion of potentially contaminated groundwater and inhalation of vapors emitted by potentially contaminated groundwater. The target cancer risks and target hazard indexes used in calculating the criteria for chemicals were set by regulatory agreement in the Methods Document at 1×10^{-7} and 0.1, respectively. The target cancer risks used in calculating the criteria for radionuclides were set by regulatory agreement in the Methods Document at 1×10^{-6}. In this screen, the lower of the residential use RBCs 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 for fish greater than 100).

(7) **Comparison of analyte maximum concentrations and activities detected in site samples to analyte concentrations and activities detected in background samples**. Consistent with procedures in the Methods Document, maximum detected concentrations were compared to background concentrations for groundwater derived as part of the GWOU FS. These values are presented in Table 2.4 and in the report entitled *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* that is presented in Appendix D. of the GWOU FS.

(8) **Examination of analyte maximum concentrations for essential human nutrients detected in site samples to Recommended Dietary Allowances (RDAs) for children**. Analytes not removed from the data set to this point were examined, and the maximum detected concentration of those 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. Analytes that were not candidates to be removed based on this screen, even though they are essential nutrients, were chromium, manganese, and zinc. Analytes that were removed regardless of the results of this screen were calcium, chloride, iodine, magnesium, phosphorus, potassium, and sodium (EPA 1995a).

2.4 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. Subsect. 2.4.1 summarizes the evaluation performed to determine representative concentrations of COPCs under current conditions. Subsect. 2.4.2 summarizes the evaluation performed to determine modeled representative concentrations of COPCs under future conditions.

2.4.1 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 set. The following material summarizes the actions performed by various programs during the evaluation. The complete programs are presented in Attachment 3 of this BHHRA.

First SAS® program (data consolidation). The first program read the groundwater data set into SAS®. This program read the data into fields to produce a data set with a uniform format to facilitate further data handling. Specific functions performed by this program were:

- Remove sampling stations from the groundwater data set that were not satisfactory for risk assessment purposes. This task included removing samples collected at hand-valve stations, at stations that were part of treatment facilities' influent and effluent systems, at residential wells where water was treated with activated carbon, as part of Lasagna™ project testing, at cooling towers, from tanks, and from test pits. Additionally, samples misidentified as water samples (e.g., air samples) were removed from the data set. After this step was completed, all data could be assigned to one of three groups. These were data collected from wells (WL), from boreholes (BH) or probes (PR), and from faucets/taps (FW).

- Retain analytical types appropriate to the station type. As noted above, the station types remaining included: well (WL), borehole (BH), Probe (PR), and faucet/tap (FW). Because of biases identified during previous work, it was determined that not all station types yield acceptable data for all analytical types (i.e., anatype). For borehole and probe stations, only the "volatile organic carbon" anatype was retained. Other anatypes were not retained for borehole stations because previous work has shown that concentrations for other anatypes tended to be biased high in borehole samples due to the high concentrations of particulates. For residential faucet/tap stations, only the "radionuclide" and "metal" anatypes were retained. Other anatypes were not retained for residential faucet/tap stations because previous work has shown that concentrations for other anatypes tended to be biased low in faucet/tap samples due to aeration at the tap. For unfiltered well stations, all anatypes were retained; however, for filtered well stations, only the "metal" and "radionuclide" anatypes were retained. Other anatypes were not retained for filtered well stations because previous work has shown that concentrations for these anatypes tended to be biased low due to their loss from water during filtration.

- Assign each unique sampling station to a single HU. If a sampling station acquired samples at different depths, as is the case with borehole/probe samples, then the unique samples were assigned to a single HU. (Please note, while the UCRS was evaluated as a drinking water source in this BHHRA per agreement with the regulatory agencies, the UCRS is not a potable aquifer.)

- Assign each sampling station to one of the eleven areas. (See Table 2.1.) As discussed earlier, data were assigned to areas to better organize the investigation of the nature and extent of contamination in groundwater at the PGDP.

- Retain all data collected after December 31, 1992. This step kept all data collected during 1993 to the present. Data collected prior to 1993 was deleted to remove a sampling bias known to exist in these data. (Samples were collected with bailers prior to 1993 and were collected with bladder pumps after that date.)

- Check spelling of all analytes and their association with CAS registry numbers. This screen allows the SAS® program to accurately merge contaminant and toxicity information later in the assessment.

- Convert units of measure to those units that will be used in the forthcoming chronic daily intake (CDI) calculations. All chemical concentrations were converted to units of mg/L, and all radionuclide activities were converted to units of pCi/L. This conversion places all chemical information upon common bases 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 match those that are included in the toxicity value data base; therefore, this conversion allows SAS® to merge the contaminant and toxicity information correctly during risk characterization.

- Distinguish between and code 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. [In this assessment, site-related analytes are trichloroethene and its breakdown products, technetium-99, uranium (metal and all radioisotopes), PCBs, and fluoride.] The second filter dropped those observations that had nondetected concentrations exceeding an analyte's maximum detected concentration. Note, the rules followed here and the filters applied are those approved in the Methods Document.

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 "cleaned-up" data set prepared by the first SAS® program. Summary statistics were calculated for each station and for each area. Because of the large number of unique sampling stations, only the area summary statistics are presented. Included in the summary (see Tables 2.5 and 2.6 in Attachment 1 of this volume) are: analyte name, frequency of detection, the range of detected values, the range of nondetected values (i.e., the range of the sample quantitation limits used in samples in which the analyte was not detected), the form of the distribution of the data, the arithmetic means of the detected concentrations, and the units of measure for the analyte. In addition, this program created a permanent SAS® data set.

Fourth SAS® program (comparison to residential use human health RBCs). The fourth program compared the maximum detected concentration of each analyte in each data aggregate to that analyte's residential-use human health RBC. This comparison was done for data from each station and area. Because of the large number of unique sampling stations, only the area assessment comparisons are presented. Residential use human health RBCs were used to comply with previous agreements with the regulatory agencies specified in the Methods Document and to recognize that many sampling stations are located outside the PGDP. As discussed earlier, the exposure routes included in the calculations of the RBCs for chemicals were ingestion of water, inhalation of emissions from water during showering and house-hold use, and dermal contact with a water while showering. The exposure routes included in the calculations of the RBCs for radionuclides were ingestion of water and inhalation of emissions from water while showering and during house-hold use. Table 2.7 presents the results of this screen.

As discussed in the Methods Document, the target HI and ELCR used in the calculation of risk-based concentrations for chemicals were 0.1 and 1×10^{-7}, respectively, and the target ELCR used in the calculation of risk-based concentrations for radionuclides was 1×10^{-6}. Also, per regulatory agreement, when performing the comparisons, the lesser of an analyte's hazard and cancer risk-based screening criteria was used.

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 bioaccumulation factor (BAF) for fish. This 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, then that analyte was not eligible for removal from the data set based on the toxicity screen. Please note, the results of the BAF screen are not reported individually in Table 2.7.

Fifth SAS® program (background and RDA screen). This program compared the maximum detected concentration of each analyte within each data aggregate against their respective background concentrations and compared the maximum detected concentration of essential nutrients in groundwater to one-fifth of

that nutrient's RDA for children. This comparison was done for data from each station and area. Because of the large number of unique sampling stations, only the area assessment comparisons are presented here. The background values used in this comparison were taken from the report in Appendix D of the GWOU FS report and are presented in Table 2.4. The results of the comparison are shown in Table 2.8. The RDAs used in this comparison are shown in Table 2.9, and the results are shown in Table 2.10.

As discussed in the Methods Document, before comparing an analyte's maximum detected concentration against one-fifth of the analyte's RDA, the analyte's concentration was converted to a daily intake for a child. 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), three analytes for which RDAs for children are available were not included in this screen. These analytes were chromium, manganese, and zinc. In addition, also per regulatory guidance (EPA 1995a), seven essential nutrients were removed from the data set even if their maximum detected value exceeded one-fifth of their RDA. These were calcium, chloride, iodine, magnesium, potassium, sodium, and phosphorus.

Sixth SAS® program (toxicity values). This program merged toxicity information with the lists of remaining analytes (i.e., lists of chemicals of potential concern).

Seventh SAS® program (output production). This program compiled the results of the previous programs and produced the tables listed earlier. These tables are as follows.

Table 2.5 Data summary for all analytes

Table 2.6 Data summary for detected analytes

Table 2.7 Comparison of maximum detected concentrations and activities to human health risk-based screening criteria

Table 2.8 Comparison of maximum detected concentrations and activities to background concentrations

Table 2.9 Recommended dietary allowances of essential human nutrients

Table 2.10 Comparison of maximum detected concentrations of essential nutrients to recommended dietary allowances for children

In addition, this program produced two additional tables that present the lists of COPCs and a summary of the data evaluation process. These tables are:

Table 2.11 Chemicals of potential concern and their frequency of detection

Table 2.12 Summary of data evaluation

Table 2.12 is a complete summary of the data evaluation process and includes a listing of all detected analytes by location and medium for the area data aggregates. In addition to the analyte's name, this table also contains the analyte's frequency of detection, range of nondetected values, range of detected values, arithmetic mean of detected values, human health systemic toxicity and ELCR-based concentrations, units of measure. The last column of this table indicates whether or not the analyte is a 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, and Qual. Definitions of these codes are as follows:

P: analyte is a COPC because the analyte's maximum detected concentration is greater than a human health risk-based concentration.

B: analyte is a COPC because the analyte's 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 analyte's RDA for children.

Qual: analyte is retained as a COPC because screening criteria used in the data evaluation were not available or because the fish bioaccumulation factor for the chemical is greater than 100.

In some cases, an analyte's basis of selection may include more than one of the aforementioned codes. In this case, the analyte was selected as a COPC because it "failed" multiple screens.

2.4.2 Evaluation of Modeled Concentrations for Groundwater

Data used to estimate risk from exposure to contaminants found in RGA water in the future were taken from modeling described in the Data Summary Report Report contained in Appendix A of the GWOU FS report. As described there, data were available for four points of exposure. These were at the PGDP fence-line, at the PGDP property boundary, at Little Bayou Creek, and at the Ohio River. Also, as discussed in the Data Summary Report, contaminants modeled were those determined to be migrating from the various SWMUs in earlier source investigations and from the secondary TCE sources (i.e., TCE present at high concentration in the RGA) believed to be present at WAG 6.

2.5 EVALUATION OF DATA FROM OTHER SOURCES

This subsection describes results of the Phase I groundwater user survey, agriculture extension agent interviews, Kentucky Department of Fish and Wildlife Resources (KDFWR) information, deer range information, exposure unit information for workers, and SWMU size information. This information was used to develop the exposure assessment in Sect. 3.

2.5.1 Groundwater User Survey Phase I (CH2M Hill 1991a)

In response to the discovery of groundwater contamination in residential wells near PGDP, a survey of users of groundwater and surface water in the vicinity of PGDP was conducted in February and March of 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 users' 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 1,988 surveys was delivered; 44% (872) of these were returned. Of the respondents, 58% 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% used well water for irrigation; 29% used well water for watering livestock; and 80% 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% 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 ft to 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 Sect. 1 of Appendix 5 in the Methods Document for a reproduction of the questionnaire.) However, 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.

2.5.2 Agriculture 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 Sect. 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.

2.5.3 Kentucky Department of Fish and Wildlife Resources 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 Sect. 3 of Appendix 5 of the Methods Document.

2.5.4 Area Size Information

In previous risk assessments, the size of each area assessed to accurately represent exposure to contaminated soil in each of the areas. However, because soil is not a media of concern for this assessment, the size of each area was not determined.

2.5.5 Exposure Unit Information for Worker

In previous risk assessments, the size of each area assessed to accurately represent exposure to contaminated soil in each of the areas. However, because soil is not a media of concern for this assessment, the size of each area was not determined.

2.5.6 Exposure Unit Information for Residents

In previous risk assessments, the size of each area assessed to accurately represent exposure to contaminated soil in each of the areas. However, because soil is not a media of concern for this assessment, the size of each area was not determined.

2.5.7 Deer Range Information

In previous risk assessments, the size of each area assessed to accurately represent exposure to contaminated soil in each of the areas. However, because soil is not a media of concern for this assessment, the size of each area was not determined.

2.6 SUMMARY OF COPCS

A general summary of COPCs in groundwater, by area and depth of sampling, for the unfiltered dataset, is presented in Exhibit 2.1. A detailed summary listing the COPCs individually for the area assessment is in Table 2.11. In Table 2.11, 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 1999a) or *Health Effects Assessment Summary Tables* (HEAST) (EPA 1999b) and is not available from the alternate approved sources listed in the Methods Document]. Finally, Table 2.12 presents information summarizing information about each detected analyte for the area assessment, including the reason for the retention of an analyte as a COPC.

Exhibit 2.1. General summary of COPCs by area and analyte type for the unfiltered data set[1]

	McNairy Formation			RGA			UCRS		
Area	**Inorganic**	**Organic**	**Radionuclide**	**Inorganic**	**Organic**	**Radionuclide**	**Inorganic**	**Organic**	**Radionuclide**
a	NR	NR	NR	12/24	7/7	6/12	18/24	6/6	5/7
b	6/16	1/1	3/8	23/32	15/20	14/17	27/32	15/22	13/16
c	NR	NR	NR	12/21	4/4	4/4	9/15	3/3	3/3
d	4/16	1/1	2/8	18/28	9/9	11/14	32/36	12/14	9/12
e	19/26	1/1	4/5	20/29	4/5	6/9	15/21	1/1	3/4
f	4/13	0/0	2/4	15/22	6/8	5/7	9/16	1/1	4/4
g	4/18	0/0	5/8	11/22	1/1	7/8	9/17	0/0	7/8
h	3/15	0/0	5/8	11/20	2/2	4/4	11/16	0/0	3/4
i	4/15	0/0	2/4	33/39	27/36	8/10	24/30	7/10	7/7
j	5/17	0/0	2/4	9/20	0/1	2/5	NR	NR	NR
k[2]	NA	NA	NA	NA	NA	NA	29/36	11/12	10/15
l	8/18	1/1	3/9	26/34	20/24	15/17	35/39	22/30	14/18
m	21/28	1/1	8/8	37/41	32/39	11/12	26/30	7/10	9/10
n	24/29	1/1	8/9	38/43	38/46	15/19	37/40	25/35	15/19

Notes:

NR indicates there are no results for the area.

NA indicates the depth classification is not applicable to the area.

[1] Values shown are number of COPCs over number of detected analytes.

[2] Area k includes water drawn from Eocene Sands, Terrace Gravels, and Porters Creek Clay.

Generally, the lists of COPCs identified in this assessment are similar to the lists of COCs identified in previous assessments (see Sect. 1). For areas affected by PGDP releases, the COPC lists are dominated by TCE and its breakdown products; the inorganic chemicals antimony, arsenic, beryllium, chromium, and manganese; and plant-related radionuclides. However, lists for areas not suspected of being impacted by plant releases (e.g., Areas g, h, and j) are dominated by inorganic chemicals.

3. EXPOSURE ASSESSMENT

3.1 INTRODUCTION

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, etc.) during a specified period. Exposure assessment is a process that uses information about the exposure setting and human activities to develop conceptual site models under current and potential future conditions. This subsection introduces the general methods used in exposure assessment, applies these methods to the GWOU 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 needs to pay special attention to 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 by RAGS (EPA 1989a), 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 the GWOU is to determine chronic intake or dose. The chronic exposure estimate is used because it allows for the estimation of the health consequences that result from long-term or unrestricted exposure to contaminants present in the GWOU. Subchronic exposures receive less attention because these exposures require the use of assumptions concerning restrictions on rates of contact with contaminated media. Such assumptions are best left to managers who can use risk management to make remedial decisions that can reduce risks from chronic exposures to acceptable levels.

3.2 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 elsewhere in the GWOU FS report, and much of that information does not bear repeating here. However, a brief physical descriptions of the GWOU taken from information presented in the GWOU FS report is included here to support later discussions of the conceptual model and its uncertainties.

3.2.1 Physical Description of the GWOU

The area encompassed by the GWOU includes all of the PGDP and continues past the northern DOE property boundary to the Ohio River. An aerial picture of this area is in Fig. 3.1. As discussed in the GWOU FS report, the GWOU specifically includes all groundwater and sources of contamination to groundwater due to DOE processes found between a subsurface feature termed “the terrace” and the Ohio River. Generally, this includes all areas inside the DOE property boundary and all areas overlying the contaminant plumes to the Ohio River. This includes the stratigraphic units underlying PGDP that have been grouped into three hydrogeologic formations: (1) the UCRS composed of fill, alluvium, loess, and the upper continental deposits; (2) the RGA, part of the lower continental deposits; and (3) the McNairy flow system consisting of combined Porters Creek Clay, the Clayton and McNairy formations, and the Eocene sands. In this BHHRA, each of these hydrogeologic units are assessed; however, samples collected from Eocene Sands, Terrace Gravels, and Porters Creek Clay to the south of the plant are assessed separately from the rest of the McNairy flow system.

Current understanding of shallow groundwater hydrology in the vicinity of PGDP is dominated by the recognized importance of the gravel facies of the continental deposits. This unit is designated as the RGA. Results of studies conducted in the mid-1960s indicate that the gravel is saturated over most of its extent in the PGDP region and that the aquifer has a prolific production capability. In general, the potentiometric surface for the RGA slopes toward the Ohio River, which suggests that groundwater flow within the aquifer is in a north-northeasterly direction.

The sand lenses interbedded in the clay facies of the UCRS are not connected to the RGA and are observed to have extremely low yields to wells. The reported discontinuous nature of these sands and ambiguities with respect to observed hydraulic heads indicates that these deposits (UCRS) may not constitute a continuous aquifer.

The PGDP site hydrogeology consists of topographically controlled recharge and discharge areas to the south and north, respectively, that bound the local flow system. One area of recharge occurs within the Eocene sands and has resulted in a groundwater divide to the southwest of PGDP. From PGDP, groundwater flows northward toward the Ohio River, which is local base level for the system. The components of the hydraulic gradient within this system are the Eocene sands (also known as the Wilcox Formation), the Pliocene terrace gravels, and the RGA (Pleistocene and Holocene components). Flow originates south of PGDP within the Eocene sands. Subsequent flow is into the Pliocene gravels that separate the Eocene sands from the RGA.

Groundwater within the Pliocene terrace gravels either discharges to local streams or recharges the RGA. Recharge to the RGA is rainfall infiltration via the overlying upper continental deposits and underflow from the terrace gravels. Recharge has been estimated to be from 11.9 to 17.7 cm/yr (4.7 to 7 in/yr), which is approximately 10 to 15% of average annual precipitation. The RGA acts as the major conduit of flow to transport water laterally to areas of discharge (ultimate discharge being to the Ohio River).

Although groundwater contamination has been found throughout the industrialized area at the PGDP, three contaminant plumes in the RGA have been defined. These are as follows:

(1) Northwest Plume – A mixed organic solvent (i.e., TCE and its breakdown products) and ^{99}Tc plume that extends from near the C-400 Building within the industrialized portion of the PGDP to the

northwest corner of the plant. From the northwest corner of the plant, the plume turns to the northeast. The leading edge of this plume approaches the Ohio River. The C-400 Building is believed to be the primary source of this plume.

(2) Northeast Plume – An organic solvent plume (i.e., TCE and its breakdown products) that extends from the central portion of the PGDP to the northeast corner of the plant. From the northeast corner of the plant, the plume continues to the northeast. The source of this plume is currently undefined.

(3) Southwest Plume – An organic solvent plume (i.e., TCE and its breakdown products) that extends from near SWMU 1 on the western side of the PGDP to the west. This plume is of limited extent and appears to hook to the north after leaving the west plant area. The source of this plume is undefined.

For the area assessment portion of this BHHRA, sampling stations were grouped based on the location of the station relative to the identified contaminant plumes and on the depth of sampling. These areas are discussed in Sect. 2 and presented in Fig. 2.1 and Plates 1 and 2. As shown in Fig. 2.1, the areas assessed includes four areas inside the industrialized portion of the PGDP (i.e., Areas a, b, c, and d) and seven areas outside the industrialized portion of the PGDP (i.e., Areas e, f, g, h, i, j, and k). In addition, as discussed earlier, three larger groupings, Areas l, m, and n, were also assessed.

Note that the "area assessment" was performed using sampling information from wells completed in the UCRS, RGA, and McNairy Formation in all areas except Area k. The assessment for Area k utilized information collected from wells completed in Eocene Sands, Terrace Gravels, and Porters Creek Clay because Area k overlies the terrace located in the southern portion of the PGDP. Exhibit 3.1 lists the number of wells assigned to each area by depth classification in the area assessment.

3.2.2 Physical Description of Area a

Area a consists of the WAG 6 area previously assessed as part of BHHRA in the WAG 6 RI report. Land use in Area a is industrial, and expected future use is also industrial. (Figs. 3.2 and 3.3 graphically presents the current and expected future land uses at and around the PGDP.) As discussed in the WAG 6 RI report, this area is dominated by roads, buildings, and utilities. This area is believed to be the source of the Northwest Plume.

3.2.3 Physical Description of Area b

Area b encompasses the area overlying the Northwest Plume between the sources at the C-400 Building and the security fence surrounding the main plant area. Current land use in this area is industrial, and expected future use is also industrial. This area is dominated by roads, buildings, and utilities. Although the primary source of groundwater contamination in this area is believed to be the C-400 Building, other sources of contamination are known to contribute to the plume in this area (e.g., SWMUs 7 and 30).

3.2.4 Physical Description of Area c

Area c encompasses the area overlying the Northeast Plume from the Area a to the security fence surrounding the main plant area. Current land use in this area is industrial, and expected future use is also industrial. The area is dominated by roads, buildings, and utilities. Although the primary source of the Northeast Plume is unknown, this area contains other sources of contamination that probably contribute to the Northeast Plume.

Exhibit 3.1. Well groupings for the unfiltered well data for the GWOU BRA, PGDP, Paducah, Kentucky

Area Name	Area code	Number of wells						
		UCRS			RGA		McN	
		HU1	HU2	HU3	HU4	HU5	HU6	Other
Inside TCE Contaminated Area at C-400 Building - Inside Industrialized Area	a	0	14	0	1	6	0	0
Inside the Northwest TCE Plume – Inside Industrialized Area (i.e., West Main Plant)	b	0	21	0	0	43	1	0
Inside the Northeast TCE Plume – Inside Industrialized Area (i.e., East Main Plant)	c	0	2	1	1	8	0	0
Outside the TCE Plumes - South of C-400 in Industrialized Area	d	0	21	1	3	18	2	1
Inside the Northwest TCE Plume – Outside Industrialized Area	e	0	1	1	0	27	3	0
Inside the Northeast TCE Plume – Outside Industrialized Area	f	0	1	0	2	17	1	0
Outside the TCE Plumes - West of Industrialized Area (i.e., West of Plume)	g	0	2	0	0	25	1	0
Outside the TCE Plumes - East of Industrialized Area (i.e., East of Plume)	h	0	1	0	0	38	1	5
Outside the TCE Plumes - North of Industrialized Area (i.e., between the Plumes)	i	1	8	5	1	47	1	0
Tennessee Valley Authority Area (TVA) - (i.e., TVA wells not in known plumes)	j	0	0	0	0	4	2	0
South of Terrace - Southern wells not in the RGA or McN	k	0	0	0	0	9	0	14[1]
All groundwater inside PGDP boundary (i.e., areas: a, b, c, and d)	l	0	58	2	5	75	3	1
All groundwater data outside PGDP boundary (i.e., e, f, g, h, i, j, and k)	m	1	13	6	3	158	9	19
All groundwater	n	1	71	8	8	233	12	20
Total by Group	–	80			241		12	20

Notes: MCN is McNairy Formation

3.2.5 Physical Description of Area d

Area d consists of the southern part of the industrialized portion of the PGDP. Generally, this area is defined as lying to the south of the C-400 Building. Current land use in this area is industrial, and expected future use is also industrial. The area is dominated by roads, buildings, and utilities. Although primary sources of the main contaminant plumes are not known to lie within this area, groundwater contamination in this area has been identified as part of earlier investigations.

3.2.6 Physical Description of Area e

Area e consists of the area overlying the Northwest Plume that is outside the secure portion of the PGDP. This area was previously assessed as part of the "Northwest Dissolved Phase Plume" BHHRA. (See Sect. 1.) Current land uses in this area are industrial near the PGDP with transition to recreational and rural residential use as distance from the plant increases. However, industrial use also occurs in the far northern part of this area at the TVA Shawnee Steam Plant. Away from the PGDP and the Shawnee Steam Plant, the area is dominated by fields, farms, and woodlots. Although groundwater contamination exists in this area, no known sources of groundwater contamination exist in Area e.

3.2.7 Physical Description of Area f

Area f consists of the area overlying the Northeast Plume that is outside the secure portion of the PGDP. Unlike Area e, an assessment for this area has not been completed previously. Current land uses in this area are industrial near the PGDP with transition to recreational and rural residential as distance from the plant increases. Future land use is expected to be recreational and rural residential. The area is dominated by fields, farms, and woodlots. Groundwater contamination in present in Area f, mostly unrelated to area sources. A small TCE DNAPL source contributes to UCRS contamination but does not appear to affect the RGA.

3.2.8 Physical Description of Area g

Area g consists of all areas lying to the west of the Northwest Plume that is also outside the industrialized portion of the PGDP. This area was assessed previously as part of the "Northwest Dissolved Phase Plume" BHHRA. (See Sect. 1.) Current land uses in this area are industrial near the PGDP with transition to recreational and rural residential as distance from the plant increases. Future land use is expected to be recreational and rural residential. The area is dominated by fields, farms, and woodlots. Neither a source to groundwater contamination nor groundwater contamination related to the PGDP are believed to exist in Area g.

3.2.9 Physical Description of Area h

Area h consists of all areas lying to the east of the Northeast Plume that are also outside the industrialized potion of the PGDP. This area was not assessed previously. Current land uses are industrial near the PGDP with transition to recreational and rural residential as distance from the plant increases. Future land use is expected to be recreational and rural residential. The area is dominated by fields, farms, and woodlots. Neither a source to groundwater contamination nor groundwater contamination related to the PGDP is believed to exist in Area h.

3.2.10 Physical Description of Area i

Area i consists of the area lying between the Northeast and Northwest Plumes that is also outside the industrialized portion of the PGDP. This area was not assessed previously. Current land uses are

industrial near the main plant and near a sanitary landfill and recreational and rural residential as distance from the plant increases. Future land use is expected to be industrial at the landfill and recreational and residential elsewhere. The area is dominated by fields, farms, and woodlots. Both a source of groundwater contamination (i.e., the landfill) and groundwater contamination are known to exist in this area.

3.2.11 Physical Description of Area j

Area j consists of the area beyond the northern edges of the Northwest and Northeast Plumes. In addition, this area includes the site of the TVA Shawnee Steam Plant. This area was assessed as part of the "Northwest Dissolved Phase Plume" BHHRA (See Sect. 1.) Current land uses in this area are industrial, recreational, and rural residential with the industrial use associated with the TVA plant. The future uses are expected to remain industrial, recreational, and rural residential. Away from the TVA plant, the area is dominated by fields, farms, and woodlots. Known groundwater contamination exists in this area. However, it is unclear if this contamination is related to sources at the PGDP (i.e., to the Northwest TCE Plume).

3.2.12 Physical Description of Area k

Area k consists of the area to the south and outside of the main industrialized portion of the PGDP. This area was not assessed previously. Neither the RGA nor the UCRS are overlain by this area. Water samples in this area were drawn from wells completed in the Eocene Sands, Terrace Gravels, or Porters Creek Clay. This area does include a portion of the process area of a World War II munitions plant (i.e., the Kentucky Ordnance Works). The current land uses in the area are industrial near the PGDP and recreational and rural residential as distance from the PGDP increases. The expected future land uses are industrial, recreational, and rural residential. The site is dominated by fields, farms, and woodlots; however, the remains of the munitions plant can still be seen throughout the area. Except near the C-401K Landfill, groundwater contamination related to the PGDP is not expected to exist in Area k. The "K-Landfill" is a known source of groundwater contamination that discharges to surrounding streams.

3.2.13 Physical Description of Area l (All groundwater inside PGDP boundary)

Area l is a combination of Areas a, b, c, and d. Therefore, this area includes all locations within the PGDP security fence. Please see the previous discussions of Areas a, b, c, and d for additional information on this area.

3.2.14 Physical Description of Area m

Area m is a combination of Areas e, f, g, h, i, j, and k. Therefore, this area includes all locations outside the PGDP security fence. Please see the previous discussions of Areas e, f, g, h, i, j, and k for additional information on this area.

3.2.15 Physical Description of Area n

Area n combines all the areas into single data aggregates for the UCRS, RGA, McNairy Formation, and other groundwater (i.e., Eocene Sands, Terrace Gravels, and Porters Creek Clay). Please see the previous discussions for additional information on this area.

3.2.16 Demography and Land Use

As shown in the physical descriptions presented above, current land use over all areas encompassed by the GWOU include recreational, industrial, and rural residential uses. However, under current use, groundwater management arrangements prohibit the use of the groundwater in the GWOU area. While

foreseeable future land use of the main plant area is expected to be industrial as well, alternative uses farther into the future are possible for the plant area as shown by the current use of the areas surrounding the main plant area. Therefore, for this BHHRA, the most sensitive land use is expected to be rural residential, and the rural residential scenario will be considered for each area in the GWOU.

The primary location of recreational use around the PGDP is in the Western Kentucky Wildlife Management Area (WKWMA). The 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 the KDFWR, manager of the 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 people, with about 50,000 people living within 10 miles, based on 1990 census data. The population of McCracken County, in which PGDP lies, is estimated at 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. The PGDP is a major employer with approximately 1,800 workers. Another major employer near the PGDP is the TVA Shawnee Steam Plant, which employs approximately 500 individuals.

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 the 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 the GWOU FS report. Therefore, the following discussions focus on points of potential human contact, types of receptors, and exposure routes.

3.3.1 Points of Human Contact – Land Use Considerations

As discussed earlier, the current land uses in the GWOU areas can be expected to continue into the foreseeable future. Therefore, all land uses discussed previously are included in the BHHRA to provide risk managers with a range of risk estimates that can be used in decision-making. Additionally, because it is not possible to identify specific locations where a individual may gain access to groundwater under future conditions, the BHHRA estimates risks under each of the uses in each of the areas. (Note that an uncertainty analysis in Sect. 6 takes this a step further by assessing risk from samples taken from individual stations.) Finally, because the depth of future wells cannot be determined, separate risk estimates are developed for each of the depth of sampling classifications (e.g., UCRS, RGA, McNairy Formation).

To simplify this assessment, it was assumed that residents are the individuals most likely to partake in recreational activities at and near the PGDP. That is, in addition to exposure from rural residential activities, a resident may also be exposed during frequent recreational activities. This assumption means that it is possible that the exposure to a rural resident may be greater than that reported later 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.

3.3.2 Potential Receptor Populations

As noted above, the receptor populations are industrial workers, rural residents, and recreational users under current conditions and under potential future conditions. Within these broad categories, rural residents contain age cohorts that need to be considered (Methods Document). For rural residents, the cohorts considered are children (aged 1 to 7) and older individuals (termed adults in this assessment). The rural resident population may also contain sensitive subpopulations such as pregnant women, young children (aged 0 to 1), the elderly, and the infirm. In this assessment, exposure to these subpopulations is not quantified because much of the information that is needed is not available; however, these subpopulations are considered qualitatively in the uncertainty discussion included in this assessment. Recreational users also have age cohorts that need to be considered (Methods Document). For the recreational user, the cohorts are children (aged 1 to 7), teens (aged 8 to 20), and adults (older than 20).

3.3.3 Delineation of Exposure Points/Exposure Routes

As 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. Note that not all exposure routes presented in the following list are quantitatively evaluated in the BHHRA; after extensive review of all possible exposure routes, only the probable exposure routes are quantified in the BHHRA.

- Ingestion of water while using 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 (i.e., vapors) emitted while using groundwater. As noted previously, 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 earlier, 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 contaminants in groundwater while showering. As noted previously, 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 organic compounds (i.e., vapors) during irrigation with contaminated 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 contaminated soil (soil and waste). Industrial processes at the PGDP have contaminated the soil. Recreational users may ingest soil while recreating, 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 contaminated soil (soil and waste). Industrial processes at the PGDP have contaminated the soil. Recreational users may get soil on their skin while recreating, 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 contaminated soil (soil and waste). Industrial processes at the PGDP 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 while recreating, 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 (i.e., vapors) emitted from contaminated soil (soil and waste). Industrial processes at the PGDP have contaminated the soil. Some of these contaminants may be volatile and released to the air as vapors. Recreational users may inhale these vapors while recreating, 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 from contaminated soil (soil and waste). Industrial processes at the PGDP have contaminated the soil. Radionuclides present in contaminated soil will, in turn, undergo decay and emit ionizing radiation. Recreational users may be exposed to this ionizing radiation while recreating, and residents may be exposed to it while gardening. Industrial workers may be exposed to the 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 agriculture land use after the Agriculture 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 Sect. 2 of Appendix 5 of the Methods Document.) Although the agents disagreed 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. Incidental ingestion of water could occur during 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. In addition, recreational use of these ponds by residents may reasonably be expected to occur. During recreational use (e.g., swimming or wading), dermal contact with water could occur. 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. In addition, recreational use of these ponds by residents may reasonably be expected to occur. During recreational activities, incidental ingestion of sediment contaminated by constituents in groundwater is possible. Rural residents are potential receptors for this exposure route.

- External exposure to ionizing radiation emitted by contaminants in groundwater while swimming or wading in privately owned fish ponds filled with groundwater. The rationale for considering ponds is presented previously. During use of these ponds by residents, exposure to ionizing radiation emitted by radionuclides in water could occur. Rural residents are potential receptors for this exposure route.

- External exposure to ionizing radiation emitted by contaminants in sediment while swimming or wading in privately owned fish ponds filled with groundwater. The rationale for considering ponds is presented previously. During use of these ponds by residents, exposure to ionizing radiation emitted by radionuclides in groundwater and sediment could occur. Rural residents are potential receptors for this exposure route.

- Consumption of fish raised in privately owned fish ponds filled with groundwater. The fish raised in ponds would 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. Recreational users (i.e., visitors) and rural residents are potential receptors for this exposure route.

- Incidental ingestion of surface water in 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. Contaminants may migrate from the PGDP through groundwater to these areas. Recreational users and industrial workers are potential receptors for this exposure route.

- Dermal contact with surface water while swimming or wading in 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. Contaminants may migrate from the PGDP through groundwater to these areas. Recreational users and industrial workers are potential receptors for this exposure route.

- Incidental ingestion of sediment while swimming or wading in 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. Contaminants may migrate from the PGDP to these areas through groundwater. Recreational users and industrial workers are potential receptors for this exposure route.

- External exposure to ionizing radiation emitted by contaminants in surface water while swimming or wading in 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. Contaminants may migrate from the PGDP to these areas though groundwater. Recreational users and industrial workers are potential receptors for this exposure route.

- External exposure to ionizing radiation emitted by contaminants in sediment while swimming or wading in 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. Contaminants may migrate from the PGDP to these areas through groundwater. Recreational users and industrial workers are potential receptors for this exposure route.

- Consumption of fish taken from creeks and ponds containing contaminated surface water. Fish living in Bayou Creek or settling ponds may accumulate contaminants in surface water in their edible tissues. Contaminants may migrate from the PGDP to these areas through groundwater. 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.

- Consumption of vegetables and produce raised in contaminated soil (soil and waste). As noted in Sect. 2 of Appendix 5 of the Methods Document, crop farming and gardening are common activities near the PGDP, and this land use pattern may be expanded to the PGDP area in the future after the industrial infrastructure is removed. Because industrial use of the PGDP has contaminated soil, plants raised in this soil may, in turn, accumulate these contaminants. Finally, humans may consume this contaminated produce. Potential receptors for this route of exposure are rural residents.

- Consumption 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, Agriculture Extension Agents for Ballard and McCracken counties indicated that small-scale cow-calf operations are common in western Kentucky. (See Sect. 2 of Appendix 5 of the Methods Document.) They further noted that slaughtering feeder cattle for home consumption is common. Beef may be contaminated by incidental ingestion of soil while on pasture, by consumption of contaminated vegetation (pasture and concentrate), and by ingestion of contaminated groundwater. Residents may eat this beef. Therefore, potential receptors for this route of exposure are rural residents.

- Consumption 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, Agriculture Extension Agents for Ballard and McCracken counties noted that dairy farming still occurs in their counties. (See Sect. 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 the PGDP is contaminated, and the vegetation may become contaminated. Therefore, dairy cattle raised at the PGDP after the industrial infrastructure is removed may become contaminated through incidental ingestion of soil while on pasture, consumption of contaminated vegetation, and ingestion of contaminated groundwater. Residents could in turn consume products made from milk from these cows. Therefore, potential receptors for this route of exposure are rural residents.

- Consumption of poultry (chickens and turkeys) given groundwater to drink. During interviews, Agriculture Extension Agents for Ballard and McCracken counties noted that commercial broiler production did occur in their counties but not near PGDP. (See Sect. 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 bought (not locally raised) feed, that normal resident time in poultry houses was 2 months, and that commercial distribution of the product occurs. However, the agents did note that the birds are most likely watered with groundwater. Therefore, 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.

- Consumption of pork from swine fed contaminated feed and water with groundwater. During interviews, Agriculture Extension Agents for Ballard and McCracken counties noted that both large commercial and small hog farms exist in their counties. (See Section 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, that farm-raised pork was consumed by farmers. Therefore, any swine raised may be contaminated through consumption of contaminated feed and groundwater, and rural residents may eat this pork. Therefore, rural residents are potential receptors for this pathway.

- Consumption of game contaminated by consumption of vegetation grown in contaminated soil (soil and waste) and ingestion of groundwater. As indicated in the Methods Document and discussed earlier, the taking 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

consumption of contaminated vegetation, soil, or groundwater. Potential receptors for this route of exposure are recreational users.

As demonstrated above, a total of 28 routes of exposure, including those that consider biota, are possible for the PGDP area. However, not all of these routes are quantified in this assessment. The routes that are quantified and the number of the table in which the equation used to quantify each route is presented, is in the Exhibit 3.2. Note that the list in Exhibit 3.2 does not include when and where exposure may occur.

Exhibit 3.2. Exposure routes quantified in the GWOU baseline human health risk assessment

Exposure Route	Table[a]
Industrial Worker	
Ingestion of groundwater while using groundwater as a drinking water source	Table 3.2
Dermal contact with groundwater while showering	Table 3.3
Inhalation of vapors emitted by groundwater while showering	Table 3.4
Recreational User	
Incidental ingestion of water while swimming in a pond filled with groundwater	Table 3.5
Dermal contact with water while wading in a pond filled with groundwater	Table 3.6
Dermal contact with water while swimming in a pond filled with groundwater	Table 3.7
Consumption of fish raised in ponds filled with groundwater	Table 3.8
Consumption of venison using ponds filled with groundwater as a drinking water source	Table 3.9
Consumption of rabbit using ponds filled with groundwater as a drinking water source	Table 3.10
Consumption of quail using ponds filled with groundwater as a drinking water source	Table 3.11
Rural Residential Use	
Ingestion of water while using groundwater as a drinking water source	Table 3.12
Dermal contact with groundwater while showering	Table 3.13
Inhalation of vapors in groundwater while showering	Table 3.14
Inhalation of vapors in groundwater during household use	Table 3.15
Consumption of vegetables irrigated with groundwater	Table 3.16
Consumption of beef watered with groundwater	Table 3.17
Consumption of milk from cattle watered with groundwater	Table 3.18
Consumption of chicken watered with groundwater	Table 3.19
Consumption of eggs from chickens watered with groundwater	Table 3.20
Consumption of pork from swine watered with groundwater	Table 3.21
Consumption of turkey watered with groundwater	Table 3.22

[a] Table in App. A where equation and exposure parameters are displayed.

As noted above, 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 discussions. Note that this information is summarized in Table 3.1.

No routes of exposure for exposure to soil or sediment were quantitatively assessed in this BHHRA because groundwater was the only medium of concern for this assessment. Sampling results for contaminated soils and sediments were not included in this BHHRA because such work would have required the assessment of individual source units, and this work has already been performed as summarized in Sect. 1 or will be performed as part of the forthcoming Surface Water, Soils, or Burial Grounds Operable Unit BHHRAs (SWOU, SOU, and BGOU, respectively). Future estimated soil and sediment contaminant concentrations from groundwater use (e.g., accretion of soil contamination via irrigation or sediment contamination via discharge to surface water) were not included because such results were deemed to add little beyond that achieved by assessing risk from direct contact with water or indirect contact through biota consumption. However, it should be recognized that cumulative risks from exposure to

contaminated soil at sources of contamination and groundwater below these sources would lead to greater risks for all current and hypothetical future receptors. This uncertainty is discussed further in Sect. 6.

All previously listed direct contact exposure routes for groundwater were assessed in this BHHRA except inhalation of vapors during irrigation by a rural resident and external exposure to ionizing radiation during exposure to water. The first route was not quantified for two reasons. First, a qualitative evaluation in *Baseline Risk Assessment and Technical Investigation Report for the Northwest Dissolved Phase Plume, Paducah Gaseous Diffusion Plant* (DOE 1994c) indicated that the volume of air in which mixing could occur out of doors 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 could be more conservatively estimated using the indoor pathways (i.e., inhalation of vapors while using groundwater in a shower and during household use). The second route was not quantified because water would provide shielding against ionizing radiation preventing a significant dose at concentration seen in groundwater considered in the GWOU BHHRA.

All previously listed direct contact to surface water exposure routes were assessed for the recreational user. In, addition, all previously listed exposure routes for biota were assessed for the water contribution component. This was done by assuming that groundwater was used to fill recreational ponds and that biota used water from these ponds as a water supply. Note that these pond scenarios were also used as a surrogate for exposure at potential discharge points along creeks or to ponds (i.e., mixing with surface water at discharge points was not considered). No exposure routes were quantitatively assessed involving residential exposure to surface water or industrial worker exposure to surface water. These routes were not assessed because it was considered that the recreational user rate of contact would be higher and provide the information needed to make appropriate risk management decisions.

3.3.4 Development of Conceptual Site Models

Using the information presented in the previous subsections, a conceptual site model was developed for the GWOU. This conceptual site model (Fig. 3.4) illustrates all sources, pathways of migration, and routes of exposure for each potential receptor. This conceptual site model is common to all areas.

3.3.5 Calculation of Representative Exposure Concentrations of COPCs

The representative exposure concentrations of COPCs in each medium under current conditions for each area were determined before the intake models presented in Subsect. 3.3.3 were used to calculate the chronic daily intakes used in the risk calculations. The concentrations for COPCs in groundwater are presented in Table 3.23. The program used to calculate these values is SAS® Program 3 in Attachment 3 of this volume.

In all cases, the representative exposure concentration for a COPC within a medium was the lesser of the maximum detected concentration of the COPC in the medium and the upper 95% confidence limit on the arithmetic mean [95% upper confidence limit (UCL)] concentration of the COPC in the medium (EPA 1992a, Methods Document). In deriving the 95% UCL concentrations for COPCs expected to be present at the GWOU and its areas (e.g., TCE and its degradation products, uranium isotopes), 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 to be present at the GWOU, 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 log-normal 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, Methods Document).

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

where:

95% UCL is the upper 95% confidence limit on the mean,
X is the arithmetic mean,
t is the Student's-t value for the appropriate number of degrees of freedom,
s is the standard deviation of the sample data,
n is the number of observations.

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

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

where:

95% UCL is the upper 95% confidence limit on the mean,
e is the base of the natural log,
X is the arithmetic mean of the log transformed values,
s^2 is the variance of the log transformed sample data,
H is the H-statistic,
n is the number of observations.

After the 95% UCL concentration of the COPC was determined, this value was compared to the maximum detected concentration of the COPC. As noted above, the representative concentration of each COPC in each medium was the lessor of the maximum detected concentration and the appropriate 95% UCL concentration. (The lessor of the maximum detected concentration and the 95% UCL were used to remain consistent with guidance in the Methods Document.)

To determine the representative concentrations of COPCs in biota, the models in Tables 3.24 to 3.34 were used. These tables present the models and the values of the input parameters. Chemical-specific parameters called out in these tables, such as biotransfer factors, are in Table 3.35. Finally, Table 3.36 presents the representative concentrations of COPCs in biota derived using these models. Note that in some cases data were not available to complete the biota modeling as indicated by the lack of values for some biota in Table 3.36.

3.4 QUANTIFICATION OF EXPOSURE

Using the human exposure models presented in Sect. 3.3.3, the conceptual site model presented in Sect. 3.3.4, and the representative exposure concentrations and uptake models discussed in Sect. 3.3.5, chronic daily intakes (CDIs) for each of the COPCs were determined. The program used to calculate the chronic daily intakes is Program 8 as described in Attachment 3; these CDIs are presented in Tables 3.37 to 3.46b. In this presentation, the CDIs used to estimate systemic toxicity (i.e., noncarcinogenic effects)

are presented first, and the CDIs used to estimate ELCR follow. Within each of these broad classifications, CDIs are presented by area, exposure route, depth classification, and exposure route. The direct contact exposure routes are presented separately from the biota consumption exposure routes for convenience.

3.5 SUMMARY OF EXPOSURE ASSESSMENT

The medium available for contact in the GWOU area is groundwater. Under current conditions, groundwater is not used within any of the GWOU areas. However, industrial land use characterizes Areas a through d and parts of Areas e through k, and rural residential and recreational land uses characterize most of Areas e through k. Under future conditions, potential human receptors for groundwater for all areas are industrial workers, recreational users (children, teens, and adults), and rural residents (children and adults).

Under future conditions, several potential routes of exposure exist. Routes quantified for the industrial worker are ingestion of groundwater, dermal contact with groundwater while showering, and inhalation of volatile compounds emitted by groundwater while showering. Routes quantified for the recreational user are incidental ingestion of water while swimming in a pond filled with groundwater, dermal contact with water while wading in a pond filled with groundwater, dermal contact with water while swimming in a pond filled with groundwater, consumption of venison ingesting water from a pond filled with groundwater as a sole drinking water source, consumption of rabbit ingesting water from a pond filled with groundwater as a sole drinking water source, consumption of quail ingesting water from a pond filled with groundwater as a sole drinking water source, and consumption of fish raised in a pond filled with groundwater. Routes quantified in the rural resident are ingestion of groundwater as a drinking water source, dermal contact with groundwater while showering, inhalation of vapors emitted by groundwater while showering, inhalation of vapors emitted by groundwater during household use, consumption of vegetables irrigated with groundwater, and consumption of beef, milk, chicken, turkey, pork, and eggs raised with groundwater as a sole drinking water source.

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4. TOXICITY ASSESSMENT

4.1 INTRODUCTION

This section summarizes the potential toxicological effects of the COPCs on exposed populations. Many of the toxicological effect summaries and most of the toxicity values in this section (except lead and a few others) were obtained from information drawn from http://risk.lsd.ornl.gov/tox/rap_toxp.htm. This website (DOE 1998a) is the Risk Assessment Information System (RAIS) prepared by the Toxicology and Risk Analysis Section of Oak Ridge National Laboratory (ORNL) 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 the 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. Complete toxicity profiles for TCE, 1,2-dichloroethene, and vinyl chloride and website addresses where complete toxicity profiles for other COCs can be found are provided in Attachment 4.

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. (The reader should note that 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; assignment of a chemical to Subgroup B2 indicates that the judgment that the chemical is a probable human carcinogen is based on animal data because 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 (RAGS). 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 4.1.

Toxicity values used in risk calculations also include the chronic RfD that 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" (RAGS). 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 4.2.

For the dermal routes of exposure (e.g., dermal exposure to contaminated water during swimming, wading, or bathing), 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 chronic daily intake for these pathways. Because the chronic daily intake 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). It 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 4.3 and 4.4, respectively.

4.2 INORGANIC COMPOUNDS

4.2.1 Aluminum (CAS 000742-90-05) (RAIS)

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

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). Aluminum content of brain, muscle, and bone increases in Alzheimer's patients. Neurofibrillary tangles (NFTs) 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. Rats and guinea pigs exposed to aluminum chlorohydrate exhibited an increase in alveolar macrophages, increased relative lung weight, and multifocal granulomatous pneumonia.

No decrease in reproductive capacity, hormonal abnormalities, or testicular histopathology was observed in male rats exposed to aluminum in drinking water for 90 days.

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.

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

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-of-evidence classification is currently not assigned. Therefore, toxicity values from IRIS or HEAST or values withdrawn from IRIS or HEAST are not available for use in the BHHRA. However, a chronic oral RfD for aluminum, 1.00 mg/(kg × day), was found in the RAIS. A chronic inhalation RfD was not found. However, because aluminum appears to have a whole body effect, a value of 1.00 mg/(kg × day) was used as the extrapolated inhalation RfD in the uncertainty discussion in Sect. 6. Similarly, a chronic absorbed RfD was not found; however, a gastrointestinal absorption factor of 10% was estimated in the RAIS. Therefore, an absorbed dose RfD of 1.00×10^{-1} mg/(kg × day) was used for dermal exposure.

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

Antimony (Sb) is a naturally occurring metal that is used in various manufacturing processes. It exists in valence states of 3 and 5. Antimony is a common urban air pollutant. Exposure to antimony may be via inhalation, oral, and dermal routes.

Antimony is sparingly absorbed following ingestion or inhalation. 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. Antimony is not metabolized but may bind to macromolecules and react covalently with sulfhydryl and phosphate groups. Excretion of antimony is primarily via the urine and feces and is also dependent upon valence state.

Acute oral exposure of humans and animals to high doses of antimony or antimony-containing compounds (antimonials) may cause gastrointestinal disorders (vomiting, diarrhea), respiratory difficulties, and death at extremely high doses. Subchronic and chronic oral exposure may affect hematologic parameters. Long-term exposure to high doses of antimony or antimonials has been shown to adversely affect longevity in animals. Limited data suggest that prenatal and postnatal exposure of rats to antimony interferes with vasomotor responses.

Acute inhalation exposure of humans may cause gastrointestinal disorders (probably due to ingestion of airborne antimony). Exposure of animals to high concentrations of antimony and antimonials (especially stibine gas) may result in pulmonary edema and death. Long-term occupational exposure of humans has resulted in electrocardiac disorders, respiratory disorders, and possibly increased mortality. Antimony levels for these occupational exposure evaluations ranged from 2.2 to 11.98 mg Sb/m^3. 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. Reproductive dysfunction has been demonstrated in rats exposed to antimony trioxide.

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. Eye irritation due to exposure to stibine gas and several antimony oxides has been reported for humans.

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

Although some data indicate that long-term exposure of rats to antimony trioxide and trisulfide increased the incidence of lung tumors, the EPA has not evaluated antimony or antimonials for carcinogenicity and a Weight-of-Evidence classification is currently unavailable.

The EPA has calculated subchronic and chronic oral RfDs of 4.00×10^{-4} mg/(kg × day) based on decreased longevity and alteration of blood chemistry in rats chronically exposed to potassium antimony tartrate in drinking water. A chronic absorbed RfD of 8.00×10^{-6} was calculated from the oral dose assuming a gastrointestinal absorption factor of 2%. A chronic inhalation RfD was not found. However, because antimony appears to have whole body effects, the chronic oral RfD [4.00×10^{-4} mg/(kg × day)] will be used as a surrogate for the inhalation RfD in the uncertainty discussion in Sect. 6. Although some data indicate that long-term exposure of rats to antimony trioxide and trisulfide increased the incidence of

lung tumors, the EPA has not evaluated antimony or antimonials for carcinogenicity, and a weight-of-evidence classification is currently unavailable.

4.2.3 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). It should be noted that 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 G.I. tract (>90%) 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% in 61 hr following oral dosing. Pentavalent arsenic is reduced to the trivalent form and then methylated in the liver to less toxic methylarsinic acids.

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.

Oral doses as low as 20-60 g/kg/day have been reported to cause toxic effects in some individuals. 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. 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. Primary target organs are the skin (hyperpigmentation and hyperkeratosis), nervous system (peripheral neuropathy) and vascular system. Anemia, leukopenia, hepatomegaly, and portal hypertension have also been reported. In addition, possible reproductive effects include a high male to female birth ratio.

In animals, acute oral exposures can cause gastrointestinal and neurological effects. Oral LD_{50} values range from about 10 to 300 mg/kg. Low subchronic doses can result in immunosuppression, and hepato-renal effects. Chronic exposures have also resulted in mild hyperkeratosis and bile duct enlargement with hyperplasia, focal necrosis, and fibrosis. Reduction in litter size, high male/female birth ratios, and fetotoxicity without significant fetal abnormalities occur following oral exposures; however, parenteral dosing has resulted in exencephaly, encephaloceles, skeletal defects, and urogenital system abnormalities.

Acute inhalation exposures to inorganic arsenic can damage mucous membranes, cause rhinitis, pharyngitis and laryngitis, and result in nasal septum perforation. Chronic inhalation exposures, such as that occurring in the workplace, can lead to rhino-pharyno-laryngitis, tracheobronchitis; dermatitis, hyperpigmentation, and hyperkeratosis; leukopenia; peripheral nerve dysfunction as indicated by abnormal nerve conduction velocities; and peripheral vascular disorders as indicated by Raynaud's syndrome and increased vasospastic reactivity in fingers exposed to low temperatures. Higher rates of cardiovascular disease have also been reported in some arsenic-exposed workers. Possible reproductive effects include a high frequency of spontaneous abortions and reduced birth weights. Arsine gas (AsH_3), 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.

Animal studies have shown that inorganic arsenic, by intratracheal instillation, can cause pulmonary inflammation and hyperplasia, lung lesions, and immunosuppression. Long-term inhalation exposures have resulted in altered conditioned reflexes and central nervous system (CNS) damage. Reductions in fetal weight and in the number of live fetuses, and increases in fetal abnormalities because of retarded osteogenesis have been observed following inhalation exposures.

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. Occupational exposure studies have shown a clear correlation between exposure to arsenic and lung cancer mortality. EPA has placed inorganic arsenic in weight-of-evidence group A, human carcinogen. A drinking water unit risk of 5×10^{-5} $(\mu g/L)^{-1}$ has been proposed; derived from drinking water unit risks for females and males that are equivalent to slope factors of 1.0×10^{-3} $(\mu g/kg/day)^{-1}$ (females) and 2.0×10^{-3} $(\mu g/kg/day)^{-1}$ (males). For inhalation exposures, a unit risk of 4.3×10^{-3} $(\mu g/m^3)^{-1}$ and a slope factor of 50 $(mg/kg/day)^{-1}$ have been derived.

The RfD for chronic and subchronic oral exposures [3.00×10^{-4} mg/(kg × day)] is based on a no-observed-adverse-effects level (NOAEL) of 0.0008 mg/(kg × day) and lowest-observed-adverse-effects level (LOAEL) of 0.014 mg/(kg × day) for hyperpigmentation, keratosis, and possible vascular complications in a human population consuming arsenic-contaminated drinking water. No subchronic and chronic RfCs have been derived for arsenic. However, because arsenic appears to have whole body effects, the oral RfD [3.00×10^{-4} mg/(kg × day)] is used as a surrogate for the inhalation RfD in the uncertainty discussion in Sect. 6. In addition, an absorbed dose RfD of 1.23×10^{-4} mg/(kg × day) was calculated by assuming a gastrointestinal absorption factor of 41%.

The EPA has placed inorganic arsenic in weight-of-evidence classification Group A, human carcinogen. Cancer slope factors for arsenic are available. The values used in the BHHRA are 1.50, 50.0, and 3.66 $[mg/(kg \times day)]^{-1}$ 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%.

4.2.4 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. The human data were used by the EPA to calculate a chronic and subchronic oral RfD of 0.07 mg/kg/day. 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 the 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. Reproduction and developmental effects were also observed. Increased fetal mortality was seen after untreated females were mated with males exposed to 5.2 mg/m^3 of barium carbonate. Similar results were obtained with female rats treated with 13.4 mg barium carbonate/m^3. The NOAEL for developmental effects was 1.15 mg/m^3 (equivalent to 0.8 mg barium/m^3). An inhalation reference concentration (RfC) of 0.005 mg/m^3 for subchronic and 0.0005 mg/m^3 for chronic exposure was calculated by the EPA based on the NOAEL for developmental effects. These effects have not been substantiated in humans or other animal systems.

Barium has not been evaluated by the EPA for evidence of human carcinogenic potential. No slope factors were used in this BHHRA for barium.

Subchronic or 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, whereas human studies identified a NOAEL of 0.21 mg/(kg × day). The human data were used by the EPA to calculate a chronic and subchronic oral RfD of 7.00×10^{-2} mg/(kg × day). EPA also has released an inhalation RfD of 1.43×10^{-4} mg/(kg × day). A gastrointestinal absorption factor of 7% was used to calculate an absorbed dose RfD of 4.90×10^{-3} mg/(kg × day).

4.2.5 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. However, the highest risk for exposure occurs among workers employed in beryllium manufacturing, fabricating, or reclamation industries. Workers encounter dusts and fumes of many different beryllium compounds; the current occupational standard for worker exposure to beryllium is 2 g/m^3 during an 8-hour workshift.

Inhaled beryllium is absorbed slowly and localizes mainly in the lungs, bone, liver and kidneys. Ingested beryllium undergoes limited absorption and localizes in liver, kidneys, lungs, stomach, spleen and the large and small intestines. Significant absorption of beryllium or its compounds through intact skin is unlikely because of its chemical properties. Beryllium per se is not biotransformed, but soluble salts may be converted to less soluble compounds in the lung. Most orally administered beryllium passes through the gastrointestinal tract unabsorbed and is excreted in the feces, whereas inhaled water-soluble beryllium salts are excreted mainly by the kidneys.

Limited data indicate that the oral toxicity of beryllium is low. No adverse effects were noted in mice given 5 ppm beryllium in the drinking water in a lifetime bioassay. The dose (converted to 0.54 mg/kg bw/day) was the NOAEL used in the calculation of the chronic oral RfD for beryllium of 0.005 mg/kg/day.

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. ATSDR estimated that, based on existing data, the disease could develop at levels ranging from approximately 2-1000 g Be/m^3. 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.

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 a few months to >=20 years after exposure. Chronic beryllium disease is a systemic disease that primarily affects the lungs and is characterized by the development of non-caseating granulomas. The disease most likely results from a hypersensitivity response to beryllium as evidenced by positive patch tests and positive lymphocyte transformation tests in exposed individuals. Granulomas may also appear in the skin, liver, spleen, lymph nodes, myocardium, skeletal muscles, kidney, bone, and salivary glands.

Epidemiologic studies have suggested that beryllium and its compounds could be human carcinogens. In a study that covered 15 regions of the U.S., 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 to conclude that these results are not proof of cause and effect relationships 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. The EPA, 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.

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. For inhalation exposure, the unit risk value is 2.4×10^{-3} $(g/m^3)^{-1}$, and the slope factor is 8.4 $[mg/(kg \times day)]^{-1}$. For oral exposure, the unit risk value is 1.2×10^{-4} $(g/L)^{-1}$ and the slope factor is 4.3 $[mg/(kg \times day)]^{-1}$.

An oral RfD of 2.00×10^{-3} mg/(kg × day) was used in this BHHRA. A gastrointestinal absorption factor of 1% was used to calculate an absorbed dose RfD of 5.0×10^{-5} mg/(kg × day). No inhalation RfD is used in this BHHRA. An oral, inhalation and absorbed dose slope factor of 4.3, 8.4, and 430 $[mg/(kg \times day)]^{-1}$ were used in this BHHRA, respectively. A gastrointestinal absorption factor of 1% was used to calculate an absorbed dose slope factor.

4.2.6 Bicarbonate (CAS 000071-52-3)

Information on the toxicity of bicarbonate (also known as hydrogen carbonate) 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 bicarbonate. Therefore, neither carcinogenicity nor systemic toxicity resulting from bicarbonate exposure is included in the BHHRA.

4.2.7 Boron (CAS 007440-42-8)

Information on the key studies utilized by EPA to set reference doses for boron and borate follows. Groups of 4 male and 4 female dogs were fed borax and boric acid in the diet for 2 years. The NOAEL was established at 350 ppm of boron equivalents (8.8 mg/kg/day), highest dose tested. In an additional study, dogs were fed 1170 ppm (29 mg/kg/day) for 38 weeks. At this dose, severe testicular atrophy and spermatogenic arrest occurred. Groups of 35 male and 35 female rats were fed borax and boric acid in the diet for 2 years at boron-equivalent doses of 117, 350, and 1170 ppm (5.9, 17.5 or 58.5 mg B/kg/day). No

treatment-related effects were seen at 5.9 or 17.5 mg/kg/day, so the highest NOAEL was selected as 17.5 mg/kg/day. The LOAEL is 58.5 mg/kg/day, based on the following: significantly decreased testes weights and testes-to-body weight ratios; atrophied seminiferous epithelium; and decreased tubular size in the testes. Brain and brain-to-body weight ratios were also significantly decreased. Schroeder and Mitchener (1975) reported a lifetime study in which mice were administered boron in drinking water at 5 mg/L (equivalent to 8.1 mg B/kg/day). No effects were observed with regard to body weight, longevity or survival. The NOAEL in this study was 8.1 mg/kg/day.

The EPA has calculated a chronic and subchronic oral RfD of 9.00×10^{-2} mg/(kg × day), but the subchronic value was withdrawn by EPA in 1998. A gastrointestinal absorption factor of 90% was used to calculate an absorbed dose RfD of 8.10×10^{-2} mg/(kg × day). An inhalation RfC of 2.00×10^{-2} (mg/m^3) from HEAST was used to calculate an inhalation RfD of 5.71×10^{-3} mg/(kg × day). An oral, inhalation and absorbed dose RfD of 9.0×10^{-2}, 5.7×10^{-3}, and 8.1×10^{-2} mg/(kg × day) were used in this BHHRA, respectively.

References:

Schroeder, H.A. and M. Mitchener. 1975. Life-term effects of mercury, methyl mercury and nine other trace metals in mice. J. Nutr. 105: 452-458.

Weir, R.J., Jr. and R.S. Fisher. 1972. Toxicological studies on borax and boric acid. Toxicol. Appl. Pharmacol. 23: 351-364.

4.2.8 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.

Cadmium is absorbed more efficiently by the lungs (30 to 60%) than by the gastrointestinal tract, the latter being a saturable process. Cadmium is transported in the blood and widely distributed in the body but accumulates primarily in the liver and kidneys. 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. Cadmium is excreted primarily in the urine.

Acute oral exposure to 20-30 g have caused fatalities in humans. Exposure to lower amounts may cause gastrointestinal irritation, vomiting, abdominal pain, and diarrhea. An asymptomatic period of one-half to one hour may precede the onset of clinical signs. Oral LD_{50} values in animals range from 63 to 1125 mg/kg, depending on the cadmium compound. 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.

Inhalation exposure to cadmium and cadmium compounds may result in effects including headache, chest pains, muscular weakness, pulmonary edema, and death. The 1-minute and 10-minute lethal concentration of cadmium for humans has been estimated to be about 2,500 and 250 mg/m^3, respectively. An 8-hour TWA (time-weighted-average) exposure level of 5 mg/m^3 has been estimated for lethal effects of inhalation exposure to cadmium, and exposure to 1 mg/m^3 is considered to be immediately dangerous to human health. Renal toxicity (tubular proteinosis) may also result from inhalation exposure to cadmium.

Chronic oral RfDs of 5×10^{-4} and 1×10^{-3} mg/kg/day have been established for cadmium exposure via drinking water and food, respectively. Both values reflect incorporation of an uncertainty factor of 10. The RfDs are based on an extensive data base regarding toxicokinetics and toxicity in both human and animals, the critical effect being renal tubular proteinuria. Confidence in the RfD and data base is high.

Inhalation RfC values are currently not available.

The target organ for cadmium toxicity via oral exposure is the kidney. For inhalation exposure, both the lungs and kidneys are target organs for cadmium-induced toxicity.

There is limited evidence from epidemiologic studies for cadmium-related respiratory tract cancer. An inhalation unit risk of 1.8×10^{-3} $(g/m^3)^{-1}$ and an inhalation slope factor of 6.1 $(mg/kg/day)^{-1}$ are based on respiratory tract cancer associated with occupational exposure. 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 5.00×10^{-4} mg/(kg × day) was used in this BHHRA for cadmium-water. A gastrointestinal absorption factor of 1% was used to calculate an absorbed dose RfD of 5.0×10^{-6} mg/(kg × day) for cadmium water. An inhalation RfD of 5.71×10^{-5} mg/(kg × day) is used in this BHHRA for cadmium-water. The only slope factor available for cadmium-water was for inhalation, 6.1. Cadmium-water is used for exposure to water.

The second variation is termed cadmium-diet. Cadmium-diet is used for exposure to soil and food. An oral RfD of 1.00×10^{-3} mg/(kg × day) was used in this BHHRA for cadmium-diet. A gastrointestinal absorption factor of 1% was used to calculate an absorbed dose RfD of 1.0×10^{-5} mg/(kg × day) for cadmium-diet. An inhalation RfD of 5.71×10^{-5} mg/(kg × day) is used in this BHHRA for cadmium-diet. The only slope factor available for cadmium-diet was for inhalation, 6.1E $[mg/(kg \times day)]^{-1}$.

4.2.9 Cerium (CAS 007440-45-1)

Information on the toxicity of cerium 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 cerium. Therefore, neither carcinogenicity nor systemic toxicity resulting from cerium exposure is included in the BHHRA.

4.2.10 Chromium III (CAS 16065-83-1) and Chromium VI (CAS 18540-29-9) (RAIS)

Elemental chromium (Cr) does not occur in nature, but is present in ores, primarily chromite ($FeOCr_2O_3$). 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. Non-occupational exposure to the metal occurs via the ingestion of chromium-containing food and water, whereas occupational exposure occurs via inhalation. Workers in the chromate industry have been exposed to estimated chromium levels of 10-50 g/m^3 for Cr(III) and 5-1000 g/m^3 for Cr(VI); however, improvements in the newer chrome-plating plants have reduced the Cr(VI) concentrations 10- to 40-fold.

Chromium(III) is poorly absorbed, regardless of the route of exposure, whereas chromium(VI) is more readily absorbed. Humans and animals localize chromium in the lung, liver, kidney, spleen, adrenals, plasma, bone marrow, and red blood cells (RBCs). 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). The main routes for the excretion of chromium are via the kidneys/urine and the bile/feces.

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 ~1.9 g/kg/day of chromic oxide [Cr(III)] (diet), 2.4 mg/kg/day of Cr(III) as chromic chloride (drinking water), or 2.4 mg/kg/day of Cr(VI) as potassium dichromate (drinking water).

The respiratory and dermal toxicity of chromium are well-documented. Workers exposed to chromium have developed nasal irritation (at <0.01 mg/m^3, acute exposure), nasal ulcers, perforation of the nasal septum (at ~2 g/m^3, subchronic or chronic exposure) and hypersensitivity reactions and "chrome holes" of the skin. Among the general population, contact dermatitis has been associated with the use of bleaches and detergents.

Compounds of both Cr(VI) and Cr(III) have induced developmental effects in experimental animals that include neural tube defects, malformations, and fetal deaths.

The subchronic and chronic oral RfD value is 1 mg/(kg × day) for Cr(III). The subchronic and chronic oral RfD for Cr (VI) are 0.02 and 0.003 mg/(kg × day), respectively. The subchronic and chronic oral RfD values for Cr(VI) and Cr(III) are derived from NOAELs of 1.47 g/kg Cr(III)/day and 25 ppm of potassium dichromate (Cr[VI]) in drinking water, respectively. 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. There is also evidence for an increased risk of developing nasal, pharyngeal, and gastrointestinal carcinomas. Quantitative epidemiological data were obtained by Mancuso and Hueper, who observed an increase in deaths (18.2%; $p<0.01$) from respiratory cancer among chromate workers compared with 1.2% deaths among controls. In a follow-up study, conducted when more than 50% of the cohort had died, the observed incidence for lung cancer deaths had increased to approximately 60% . The workers were exposed to 1-8 mg/m^3/year total chromium. Mancuso 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.

Based on sufficient evidence for humans and animals, Cr(VI) has been placed in the EPA weight-of-evidence classification A, human carcinogen. For inhalation exposure, the unit risk value is 1.2×10^{-2} $(g/m^3)^{-1}$ and the slope factor is 41 $[mg/(kg \times day)]^{-1}$.

For estimation of risk from exposure to chromium, the toxicity values associated with chromium VI were used. Chromium III values were not used because most analytical results were not specific for this ionic species. The uncertainty in using chromium III versus chromium VI in the risk assessment is discussed in Sect. 6.

An inhalation cancer slope factors for chromium of 41 was used in this BHHRA. The oral and dermal RfDs used in the BHHRA are 3.00×10^{-3} and 6.00×10^{-5} mg/(kg × day), respectively. The dermal route RfD is based on the oral RfD and a gastrointestinal absorption factor of 2%.

4.2.11 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 1990).

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 1990).

An oral RfD of 6.00×10^{-2} mg/(kg × day)was used in this BHHRA. A gastrointestinal absorption factor of 80% was used to calculate an absorbed dose RfD of 4.8×10^{-2} mg/(kg × day). No inhalation RfD is used in this BHHRA.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1990. *Draft Toxicological Profile for Cobalt*. U.S. Department of Health and Human Services. Public Health Service.

Angerer, J., and R. Heinrich. 1988. Cobalt. **In**: *Handbook on Toxicity of Inorganic Compounds*. H.G. Seiler, H. Sigel, and A. Sigel. New York: Marcel Dekker, Inc. pp. 251-264.

4.2.12 Copper (CAS 007440-50-8) (RAIS)

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 the sulfate, carbonate, cyanide, oxide, and sulfide are used as fungicides, as components of ceramics and pyrotechnics, for electroplating, and for numerous other industrial applications. 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.

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. Gastrointestinal disturbances and liver toxicity have also resulted from long-term exposure to drinking water containing 2.2-7.8 mg Cu/L. 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. In animal studies, oral exposure to copper caused hepatic and renal accumulation of copper, liver and kidney necrosis at doses of >=100 mg/kg/day; and hematological effects at doses of 40 mg/kg/day.

Acute inhalation exposure to copper dust or fumes at concentrations of 0.075-0.12 mg Cu/m^3 may cause metal fume fever with symptoms such as cough, chills and muscle ache. Among the reported effects in workers exposed to copper dust are gastrointestinal disturbances, headache, vertigo, drowsiness, and hepatomegaly.

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.

Oral or intravenous administration of copper sulfate increased fetal mortality and developmental abnormalities in experimental animals. Evidence also indicates that copper compounds are spermicidal.

Oral and absorbed dose RfDs used in this BHHRA are 4.00×10^{-2} mg/(kg × day) and 1.20×10^{-2} mg/(kg × day), respectively. EPA established an action level of 1300 µg/L for drinking water (56 FR 26460). Data were insufficient to derive a RfC for copper.

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

4.2.13 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/m^3 had serious bone damage throughout their bodies. Exposure to high levels of fluoride may also cause disturbances in calcium metabolism that 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 to 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 6.00×10^{-2} mg/(kg × day). The dermal route RfD based on the oral RfD and a gastrointestinal absorption factor of 97% is 5.8×10^{-2} mg/(kg × day) (RAIS).

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1991. *Draft Toxicological Profile for Fluoride*, U.S. Department of Health and Human Services, Public Health Service.

4.2.14 Gallium (CAS 007440-55-3)

Information on the toxicity of gallium 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 gallium. Therefore, neither carcinogenicity nor systemic toxicity resulting from gallium exposure is included in the BHHRA.

4.2.15 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% 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 3.00×10^{-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%, is 4.50×10^{-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.

References

Goodman, L.S. and A. Gilman. 1985. *The Pharmacologic Bases of Therapeutics*. 7th ed. New York, New York: MacMillan Publishing Co.

4.2.16 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.

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 µg/L in surface water and 0.005 µg/L in sea water. River sediments contain about 20,000 µg/g and coastal sediments about 100,000 µg/g. Soil content varies with the location, ranging up to 30 µg/g in rural areas, 3000 µg/g in urban areas, and 20,000 µg/g near point sources. Human exposure occurs primarily through diet, air, drinking water, and ingestion of dirt and paint chips.

The efficiency of lead absorption depends on the route of exposure, age, and nutritional status. Adult humans absorb about 10-15% of ingested lead, whereas children may absorb up to 50%, depending on whether lead is in the diet, dirt, or paint chips. More than 90% 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.

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% of the total body lead burden in adults and about 73% 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 to 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. 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 and ATSDR, 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 central nervous system (CNS) effects and peripheral nerve damage occur at blood lead levels of 40-60 µg/dL, and nonovert symptoms, such as peripheral nerve dysfunction, occur at levels of 30-50 µg/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 and cognitive deficits in lead workers with blood levels of 41-80 µg/dL.

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 µg/dL in adults and at 80-100 µg/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 µg/dL and one to two points at levels of 15-30 µg/dL among 75 black children of low socioeconomic status.

Very detailed longitudinal studies have been conducted on children (starting at the time of birth) living in Port Pirie, Australia, Cincinnati, Ohio, and Boston, Massachusetts. 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-µg/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 as blood lead increased from 10-30 µg/dL. No significant neurobehavioral deficits were noted for 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 µg/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 µg/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 µg/dL within the range of 1-25 µg/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 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), the NHANES II study, and Welsh Heart Programme 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 µg/dL. 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, 40-59-year-old white males with blood levels ranging from 7-34 µg/dL, and males and females greater than 20 years old. In addition, left ventricular hypertrophy was also positively associated with blood lead. The Welsh study did not show an association among men and women with blood lead of 12.4 and 9.6 µg/dL, respectively. Other smaller studies showed both positive and negative results. The EPA concluded that increased blood pressure is positively correlated with blood lead levels in middle-aged men, possibly at concentrations as low as 7 µg/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 µg/dL in adults and 70 µg/dL in children. The anemia is due primarily to reduced heme synthesis, which is observed in adults having blood levels of 50 µg/dL and in children having blood levels of 40 µg/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 µg/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 µg/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 Biokinetic 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 µg/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 (µg/g) for lead in soil.

Inorganic lead and lead compounds have been evaluated for carcinogenicity by the EPA. 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, KDEP has provided provisional RfDs for oral, dermal, and inhalation toxicity; they are 1.0×10^{-7}, 1.5×10^{-8}, and 2.86×10^{-4} mg/(kg × day), respectively. A gastrointestinal absorption factor of 15% can be derived from the oral and dermal RfDs. In addition, three classes of benchmarks are available and are used in the BHHRA. These are the benchmarks applied by the Integrated Exposure Uptake Biokinetic Model (10 µg/dL); the EPA screening values of 400 mg/kg and 15 µg/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 µg/l for soil and water, respectively (KDEP 1995). The results of the model and a comparison of environmental concentrations to the screening values are discussed in Sect. 5.

4.2.17 Lithium (CAS 007439-93-2) (RAIS)

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

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. 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. Lithium forms covalent bonds in organometallic compounds such as lithium stearate. Organo-lithium compounds are used as multipurpose greases, particularly in the automotive industry.

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). 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. Lithium crosses the human placenta 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 LD_{50} values for several compounds and animal species range from 422-1165 mg/kg. Case histories 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. 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. 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. Movement disorders (myoclonus, choreoathetosis), proximal muscle weakness, fasciculations, gait disturbances, incontinence, corticospinal tract signs, and a Parkinsonian syndrome (cogwheel rigidity, tremor) have been reported. 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. Permanent brain damage has occurred in several patients on long-term lithium therapy.

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. Degenerative changes may occur in the glomeruli or in the distal convoluted tubules or collecting ducts. In rare cases, acute renal failure may occur.

Cohort studies indicate that the risk of major congenital malformations among women receiving lithium during early pregnancy is slightly higher (4-12%) than that among control groups (2-4%). 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. 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/m^3 caused severe eye and nasal irritation as well as skin irritation; concentrations of 0.025 mg/m^3 or less caused no adverse effects. In animal studies, concentrations above 10 mg/m^3 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.

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% lithium carbonate and 20% 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. The LC_{50} (after 14 days) was estimated to be 1700 mg/m^3 for males and 2000 mg/m^3 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% lithium carbonate, the LC_{50} value (after 14 days) was 940 mg/m^3. Four-hour exposure to an aerosol containing primarily lithium hydroxide with 23% lithium carbonate resulted in an LC_{50} of 960 mg/m^3.

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.

The oral RfD used in this BHHRA is 2.0×10^{-2} mg/(kg × day). The dermal route RfD based on the oral RfD and a gastrointestinal absorption factor of 80% is 1.6×10^{-2} mg/(kg × day). Inhalation toxicity values were not available.

4.2.18 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 central nervous system 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/m^3 in air for inhalation exposure have been estimated as LOAELs for central nervous system 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 RfD for water and diet were calculated. A chronic and subchronic RfD for drinking water of 0.005 mg/kg/day has been calculated by EPA from a human NOAEL of 0.005 mg/kg/day; the NOAEL was determined from an epidemiological study of human populations exposed for a lifetime to manganese concentrations in drinking water ranging from 3.6-2300 μg/L. A chronic and subchronic RfD of 0.14 mg/kg/day for dietary exposure has been calculated by EPA from a human NOAEL of 0.14 mg/kg/day, which was determined from a series of epidemiological studies. 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 indicates that the chronic RfD values are pending change.

A RfC of 0.05 $\mu g/m^3$ (EPA 1995a) for chronic inhalation exposure was calculated from a human LOAEL of 0.05 mg/m^3 for impairment of neurobehavioral function from an epidemiological study by Roels et al. The study population was occupationally exposed to airborne manganese dust with a median concentration of 0.948 mg/m^3 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/m^3 × 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: D, not classifiable as to human carcinogenicity based on no evidence in humans and inadequate evidence in animals.

As noted previously, no cancer slope factors for manganese are available. Therefore, carcinogenicity from exposure to manganese is not included in the BHHRA. The oral RfDs used in the BHHRA are 4.6×10^{-2} and 1.40×10^{-1} mg/(kg × day) for the exposure through environmental media and diet, respectively. The dermal route RfD based on the oral RfD for exposure to environmental media and diet and a gastrointestinal absorption factor of 4% is 1.84×10^{-3} and 5.6×10^{-3} mg/(kg × day), respectively. The manganese RfD for inhalation exposure used in the BHHRA is 1.43×10^{-5} mg/(kg × day) for environmental media and diet.

4.2.19 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.

Absorption, distribution, metabolism, and excretion of mercury is dependent upon its form and oxidation state. 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. The urine and feces are primary excretory routes. The elimination half-life is 35 to 90 days for elemental mercury and mercury vapor and about 40 days for inorganic salts.

Ingestion of mercury metal is usually without effect. 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. Mercuric (divalent) salts are usually more toxic than are mercurous (monovalent) salts. Mercury is also known to induce hypersensitivity reactions such as contact dermatitis and acrodynia (pink disease).

Inhalation of mercury vapor may cause irritation of the respiratory tract, renal disorders, central nervous system effects characterized by neurobehavioral changes, peripheral nervous system toxicity, renal toxicity (immunologic glomerular disease), and death.

Toxicity resulting from subchronic and chronic exposure to mercury and mercury compounds usually involves the kidneys and/or nervous system, the specific target and effect being dependent on the form of mercury. Organic mercury, especially methyl mercury, rapidly enters the central nervous system resulting in behavioral and neuromotor disorders. The developing central nervous system 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. Blood mercury levels of <10 μg/dL and 300 μg/dL corresponded to mild effects and death, respectively. 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.

A subchronic and chronic oral RfD of 0.0001 mg/kg/day for methyl mercury is based on a benchmark dose of 1.1 μg/kg/day relative to neurologic developmental abnormalities in human infants. A subchronic and chronic oral RfD of 0.0003 mg/(kg × day) for mercuric chloride is based on immunologic glomerulonephritis. A LOAEL of 0.63 mg Hg/kg/day for mercuric chloride was identified. NOAELs were not available for oral exposure to inorganic mercury or methyl mercury. A subchronic and chronic inhalation RfC of 0.0003 mg Hg/m^3 for inorganic mercury is based on neurological disorders (increased frequency of intention tremors) following long-term occupational exposure to mercury vapor. The LOAELs for subchronic and chronic inhalation exposures to inorganic mercury are 0.32 and 0.03 mg Hg/m^3, respectively. NOAELs were unavailable. An 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. Weight-of-evidence classifications of C (possible human carcinogen) have been assigned to mercuric chloride and methyl mercury by EPA based upon limited evidence of carcinogenicity in rodents. No slope factors have been calculated.

The oral RfD used in this BHHRA is 3.0×10^{-4} mg/(kg × day). The dermal route RfD based on the oral RfD and a gastrointestinal absorption factor of 7% is 2.1×10^{-5} mg/(kg × day). The RfD for inhalation exposure used in the BHHRA is 8.57×10^{-5} mg/(kg × day).

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

Molybdenum (Mo) occurs naturally in various ores; the principal source being molybdenite (MoS_2). 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 μg/day for adults and older children.

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. Limited data suggest that 25 to 50% 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.

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% of the adults examined in one town and 31% 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. 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.

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. 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. Fatty degeneration of the liver occurred in rabbits dosed with 50 mg/kg/day for 6 mo and in rats dosed with 5 mg/kg/day as ammonium molybdate for 1 year. Male sterility, was reported in rats fed diets containing 80 or 140 ppm Mo. 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.

The chronic oral and dermal RfD for molybdenum and molybdenum compounds is 5.0×10^{-3} and 1.9×10^{-3} mg/(kg × day), respectively, based on biochemical indices in humans. The subchronic RfD is also 5×10^{-3} mg/(kg × day) (EPA 1992). The gastrointestinal absorption factor is 38%.

Information on the inhalation toxicity of molybdenum in humans following acute and subchronic exposures is not available. Studies of workers chronically exposed to Mo 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. Elevated levels of Mo in blood plasma and urine and high levels of ceruloplasmin and uric acid in blood serum were reported for workers exposed to Mo (8-hr TWA 9.5 mg Mo/m^3). 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. Direct pulmonary effects of chronic exposure to Mo have been reported in only one study in which 3 of 19 workers exposed to Mo and MoO_3 (1 to 19 mg/m^3) for 3-7 years were symptomatic and had X-ray findings indicative of pneumoconiosis. Adverse reproductive or developmental effects have not been observed in molybdenum workers.

In animal studies, inhalation exposures to molybdenum compounds have resulted in respiratory tract irritation, pulmonary hemorrhages, perivascular edema, and liver and kidney damage. Other effects reported in animals include diarrhea, muscle incoordination, loss of hair, loss of weight, changes in ECG, increased arterial blood pressure, increased serum lactate dehydrogenase, increased cardiac adrenaline and noradrenaline levels, and inflammation of the uterine horns with necrotic foci and endometrial atrophy. 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 Mo may have an inhibitory effect on esophageal and mammary carcinogenesis. 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. Molybdenum is placed in EPA Group D, not classifiable as to carcinogenicity in humans and calculation of slope factors is not possible.

A chronic and subchronic oral RfD of 5.00×10^{-3} mg/(kg × day) was used in the BHHRA. The dermal route RfD based on the oral RfD and a gastrointestinal absorption factor of 38% is 1.9×10^{-3} mg/(kg × day) (RAIS).

4.2.21 Nickel (CAS 007440-02-0 for soluble nickel salts) (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. 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. 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. Oral LD_{50} values for rats range from 67 mg nickel/kg (nickel sulfate hexahydrate) to >9000 mg nickel/kg (nickel powder). Toxic effects of oral exposure to nickel usually involve the kidneys with some evidence from animal studies showing a possible developmental/reproductive toxicity effect.

Inhalation exposure to some nickel compounds will cause toxic effects in the respiratory tract and immune system. Inhalation LC_{50} values for animals range from 0.97 mg nickel/m^3 for rats (6-hour exposure) to 15 mg nickel/m^3 for guinea pigs (time not specified). Acute inhalation exposure of humans to nickel may produce headache, nausea, respiratory disorders, and death. Asthmatic conditions have also been documented for inhalation exposure to nickel. Soluble nickel compounds tend to be more toxic than insoluble compounds. In addition, nickel carbonyl is known to be extremely toxic to humans upon acute inhalation exposure.

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, but Chashschin et al. reported possible reproductive and developmental effects in humans of occupational exposure to nickel (0.13-0.2 mg nickel/m^3). Although not validated by quantitative epidemiologic data or statistical analyses, the authors reported an apparently abnormal increase in spontaneous and threatening abortions (16-17% in nickel-exposed workers vs 8-9% in nonexposed workers), and an increased incidence of non-specified structural malformations (17% vs 6%) 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.

A chronic and subchronic oral RfD of 0.02 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. 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 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. 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. Inhalation studies using rats have also shown nickel subsulfate or nickel carbonyl to be carcinogenic. Based on these data, the EPA has classified nickel subsulfate and nickel refinery dust in weight-of-evidence group A, human carcinogen. Carcinogenicity slope factors of 1.7 and 8.4×10^{-1} $(mg/kg/day)^{-1}$ and unit risks of 4.8×10^{-4} $(\mu g/m3)^{-1}$ and 2.4×10^{-4} $(\mu g/m3)^{-1}$ have been calculated for nickel subsulfide and nickel refinery dust, respectively. 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. 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.

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 2.00×10^{-2} mg/(kg × day). The dermal route RfD used in the BHHRA, based on the oral RfD and a gastrointestinal absorption factor of 27%, is 5.4×10^{-3} mg/(kg × day). An inhalation RfD for soluble nickel salts was not found; however, based on potential whole body effects discussed previously, the oral RfD of 2.00×10^{-2} mg/(kg × day) is used as the surrogate inhalation RfD in the uncertainty discussion in Sect. 6.

4.2.22 Nitrate, Nitrate/Nitrite (CAS 14797-55-8) also Nitrate as Nitrogen (CAS 007727-37-9) (RAIS)

Nitrates are produced by natural biological and physical oxidations and therefore are ubiquitous in the environment. Most of the excess nitrates in the environment originate from inorganic chemicals manufactured for agriculture. Organic molecules containing nitrate groups are manufactured primarily for explosives or for their pharmacological effects. Exposure to inorganic nitrates is primarily through food and drinking water, whereas exposure to organic nitrates can occur orally, dermally, or by respiration. The primary toxic effects of the inorganic nitrate ion (NO_3-) result from its reduction to nitrite (NO_2-) by microorganisms in the upper gastrointestinal tract. Nitrite ions can also be produced with organic nitrate exposure; however, the primary effect of organic nitrate intake is thought to be dependent on the production of an active nitric oxide (NO-) radical. Organic nitrates are metabolized in the liver resulting in an increase in blood nitrites. Nitrates and nitrites are excreted primarily in the urine as nitrates.

The primary toxic effect of inorganic nitrates is the oxidation of the iron in hemoglobin by excess nitrites forming methemoglobin. Infants less than 6 months old comprise the most sensitive population. Epidemiological studies have shown that baby formula made with drinking water containing nitrate nitrogen levels over 10 mg/L can result in methemoglobinemia, especially in infants less than 2 months of age. No cases of methemoglobinemia were reported with drinking water nitrate nitrogen levels of 10 mg/L or less. A secondary target for inorganic nitrate toxicity is the cardiovascular system. Nitrate intake can also result in a vasodilatory effect, which can complicate the anoxia resulting from methemoglobinemia. Decreased motor activity was reported in mice given up to 2000 mg nitrite/L in drinking water, and persistent changes in electroencephalogram (EEG) recordings were observed in rats exposed to 100 to 2000 mg nitrite/L in drinking water. However, exposure of rats to 3000 mg nitrite/L in drinking water for

2 years did not result in any gross or microscopic changes in brain tissue. The data indicate that these central nervous system effects are not related to methemoglobin levels.

The importance of the primary and secondary targets are reversed with organic nitrates, several of which have long been used for their vasodilatory effects in the treatment of angina pectoris in humans. Large doses of organic nitrates, however, can also produce methemoglobinemia. Epidemiological studies have shown that chronic or subchronic exposure to organic nitrates results in the development of tolerance to the cardiovascular effects of these compounds. This apparent biocompensation has caused serious cardiac problems in munitions workers exposed to organic nitrates when they are suddenly removed from the source of exposure.

An epidemiological study correlated the number of congenital malformations of the central nervous system and musculoskeletal system of babies with the amount of inorganic nitrate in the mother's drinking water. Other studies, however, do not support these associations, and the presence of unidentified teratogenic factors in the environment could not be ruled out. Inorganic nitrate and nitrite have been tested for teratogenicity in rats, guinea pigs, mice, hamsters, and rabbits. No teratogenic responses were reported; however, fetotoxicity attributed to maternal methemoglobinemia was observed at high doses (4000 mg nitrate/L in drinking water).

A RfD of 1.60 mg/(kg × day) (nitrate nitrogen) for chronic oral exposure was calculated from a NOAEL of 10 mg/L and a LOAEL of 11-20 mg/L in drinking water, based on clinical signs of methemoglobinemia in 0-3-month-old infants. It is important to note, however, that the effect was documented in the most sensitive human population so no uncertainty or modifying factors were used.

The possible carcinogenicity of nitrate depends on the conversion of nitrate to nitrite and the reaction of nitrite with secondary amines, amides, and carbamates to form N-nitroso compounds that are carcinogenic. Experiments with rats have shown that when given both components, nitrite and heptamethyleneimine, in drinking water, an increase in the incidence of tumors occurs. Human epidemiological studies, however, have yielded conflicting evidence. Positive correlations between the concentration of nitrate in drinking water and the incidence of stomach cancer were reported in Columbia and Denmark. However, studies in the United Kingdom and other countries have failed to show any correlation between nitrate levels and cancer incidence. Nitrate has not been classified as to its carcinogenicity by the EPA, although it is under review.

The oral RfD for nitrate used in this BHHRA is 1.6 mg/(kg × day). The dermal route RfDfor nitrate based on the oral RfD and a gastrointestinal absorption factor of 50% is 8.0×10^{-1} mg/(kg × day). The RfD for inhalation exposure of nitrate has not been determined.

The oral RfD for nitrite used in this BHHRA is 1.0×10^{-1} mg/(kg × day). The dermal route RfDfor nitrite based on the oral RfD and a gastrointestinal absorption factor of 50% is 5.0×10^{-2} mg/(kg × day). The RfD for inhalation exposure of nitrite has not been determined.

4.2.23 Nitrogen (Kjeldahl-total (CAS007727-37-9) also Ammonia as Nitrogen (CAS 0007664-41-7))

Information on the toxicity of nitrogen 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 nitrogen. Therefore, neither carcinogenicity nor systemic toxicity resulting from nitrogen exposure is included in the BHHRA.

The inhalation RfC for ammonia given by EPA is 1.00×10^{-1} mg/m3. The inhalation RfD for ammonia calculated and used in the BHHRA is 2.86×10^{-2} mg/(kg × day).

4.2.24 Orthophosphate (CAS 0014265-44-2)

Information on the toxicity of orthophosphate (also known as monohydrogen phosphate ion, HPO4–, inorganic phosphate, Pi, and $HO_4P^-_2$) 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 phosphate. Therefore, neither carcinogenicity nor systemic toxicity because of phosphate exposure is included in the BHHRA.

4.2.25 Selenium (CAS 007782-49-2) (RAIS)

Selenium is an essential trace element important in many biochemical and physiological processes including the biosynthesis of coenzyme Q (a component of mitochondrial electron transport systems), regulation of ion fluxes across membranes, maintenance of the integrity of keratins, stimulation of antibody synthesis, and activation of glutathione peroxidase (an enzyme involved in preventing oxidative damage to cells). Recommended human dietary allowances (average daily intake) for selenium are as follows: infants up to 1 year, 10-15 g; children 1-10 years, 20-30 g; adult males 11-51+ years, 40-70 g; adult females 11-51+ years, 45-55 g; pregnant or lactating women, 65-75 g. There appears to be a relatively narrow range between levels of selenium intake resulting in deficiency and those causing toxicity.

Selenium occurs in several valence states: -2 (hydrogen selenide, sodium selenide, dimethyl selenium, trimethyl selenium, and selenoamino acids such as selenomethionine; 0 (elemental selenium); +4 (selenium dioxide, selenious acid, and sodium selenite); and +6 (selenic acid and sodium selenate). Toxicity of selenium varies with valence state and water solubility of the compound in which it occurs. The latter can affect gastrointestinal absorption rates.

Gastrointestinal absorption in animals and humans for various selenium compounds ranges from about 44% to 95% of the ingested dose. Respiratory tract absorption rates of 97% and 94% for aerosols of selenious acid have been reported for dogs and rats, respectively. Selenium is found in all tissues of the body; highest concentrations occur in the kidney, liver, spleen, and pancreas. Excretion is primarily via the urine (0-15 g/L); however, excretory products can also be found in the feces, sweat, and in expired air.

In humans, acute oral exposures can result in excessive salivation, garlic odor to the breath, shallow breathing, diarrhea, pulmonary edema, and death. Other reported signs and symptoms of acute selenosis include tachycardia, nausea, vomiting, abdominal pain, abnormal liver function, muscle aches and pains, irritability, chills, and tremors. Acute toxic effects observed in animals include pulmonary congestion, hemorrhages and edema, convulsions, altered blood chemistry (increased hemoglobin and hematocrit); liver congestion; and congestion and hemorrhage of the kidneys.

General signs and symptoms of chronic selenosis in humans include loss of hair and nails, acropachia (clubbing of the fingers), skin lesions (redness, swelling, blistering, and ulcerations), tooth decay (mottling, erosion and pitting), and nervous system abnormalities attributed to polyneuritis (peripheral anesthesia, acroparaethesia, pain in the extremities, hyperreflexia of the tendon, numbness, convulsions, paralysis, motor disturbances, and hemiplegia). In domesticated animals, subchronic and chronic oral exposures can result in loss of hair, malformed hooves, rough hair coat, and nervous system abnormalities (impaired vision and paralysis). Damage to the liver and kidneys and impaired immune responses have been reported to occur in rodents following subchronic and/or chronic oral exposures.

Selenium is teratogenic in birds and possibly also in domesticated animals (pigs, sheep, and cattle), but evidence of teratogenicity in humans and laboratory animals is lacking. However, adverse reproductive and developmental effects (decreased rates of conception, increased rates of fetal resorption, and reduced fetal body weights) have been reported for domesticated and laboratory animals.

The RfD for chronic oral exposures is 5×10^{-3} mg/(kg × day) for both selenium and selenious acid. The subchronic RfDs for these compounds are the same as the chronic RfDs.

In humans, inhalation of selenium or selenium compounds primarily affects the respiratory system. Dusts of elemental selenium and selenium dioxide can cause irritation of the skin and mucous membranes of the nose and throat, coughing, nosebleed, loss of sense of smell, dyspnea, bronchial spasms, bronchitis, and chemical pneumonia. Other signs and symptoms following acute inhalation exposures include lacrimation, irritation and redness of the eyes, gastrointestinal distress (nausea and vomiting), depressed blood pressure, elevated pulse rate, headaches, dizziness, and malaise. In animals, acute inhalation exposures also result in severe respiratory effects including edema, hemorrhage, and interstitial pneumonitis as well as in splenic damage (congestion, fissuring red pulp, and increased polymorphonuclear leukocytes) and liver congestion and mild central atrophy. Information on toxicity of selenium in humans and animals following chronic inhalation exposures is not available, and subchronic and chronic inhalation reference concentrations have not been derived.

Epidemiologic studies in humans havation between chronic oral exposures to selenium and an increased incidence of death due to neoplasms. Some studies have indicated that selenium may have anti-neoplastic properties. In studies on laboratory animals, selenites or selenates have not been found to be carcinogenic; however, selenium sulfide produced a significant increase in the incidence of hepatocellular carcinomas in male and female rats and in female mice and a significant increase in alveolar/bronchiolar carcinomas and adenomas in female mice following chronic oral exposures. EPA has placed selenium and selenious acid in Group D, not classifiable as to carcinogenicity in humans, while selenium sulfide is placed in Group B2, probable human carcinogen. Quantitative data are, however, insufficient to derive a slope factor for selenium sulfide. Pertinent data regarding the potential carcinogenicity of selenium by the inhalation route in humans or animals were not located in the available literature.

The oral RfD used in this BHHRA is 5.0×10^{-3} mg/(kg × day). The dermal route RfD based on the oral RfD and a gastrointestinal absorption factor of 44% is 2.2×10^{-3} mg/(kg × day). The RfD for inhalation exposure has not been determined.

4.2.26 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.

4.2.27 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. 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.

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. Mild allergic responses have been attributed to dermal contact with silver.

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. Hypoactivity was seen in rats subchronically exposed to silver nitrate in drinking water.

A RfD of 5×10^{-3} mg/(kg × day) for subchronic and chronic exposure was calculated from a LOAEL of 0.014 mg/(kg × day) for argyria observed in patients receiving i.v. injections of silver arsphenamine. Data are presently insufficient to derive a RfC for silver.

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.

The oral RfD used in this BHHRA is 5.0×10^{-3} mg/(kg × day). The dermal route RfD based on the oral RfD and a gastrointestinal absorption factor of 18% is 9.0×10^{-4} mg/(kg × day). The RfD for inhalation exposure has not been determined.

4.2.28 Strontium (CAS 007440-24-6)

Strontium is commonly found in igneous rocks or independently in or near sedimentary rocks such as gypsum and is also sometimes found in seawater. Only .02–.03% of the earth's crust is composed of strontium, which is used to modify the properties of low aluminum silicon-casting alloys, deoxidize copper and bronze, and improve the machinability of gray-iron castings. In addition, it is sometimes added to tin and lead alloys and toothpaste (Grayson and Eckroth 1984).

Strontium is absorbed from the gastrointestinal tract and then is deposited in the teeth and bones. No evidence suggests that strontium is hazardous in industrial conditions. With massive doses through intravenous injection, strontium can cause electrocardiographic changes and respiratory paralysis (Grayson and Eckroth 1984).

No cancer slope factors for stable strontium were found; therefore, carcinogenicity due to exposure to stable strontium is not included in the BHHRA. The oral RfD used in the BHHRA is 6.00×10^{-1} mg/(kg × day). The dermal route RfD used in the BHHRA, based on the oral RfD and a gastrointestinal absorption factor of 20%, is 1.20×10^{-1} mg/(kg × day) (RAIS). An inhalation RfD for strontium was not found.

4.2.29 Sulfate and Sulfide (CAS 012143-45-2 and 018496-25-8)

The sulfate ion, SO_4, is one of the major anions occurring in natural waters. The majority of sulfates are water soluble with the exception of lead, barium, and strontium sulfates. Therefore, dissolved sulfate is considered to be a permanent solute of water.

The major health effect observed with sulfate ingestion is laxative action, and the cation associated with the sulfate appears to have some effect on the salt’s potency as a laxative. Sulfate slowly penetrates mammalian cellular membranes and is rapidly eliminated through the kidneys. Pursuant to the Safe Drinking Water Act, the EPA has proposed Maximum Contaminant Limit Goals of either 400 or 500 mg/L to

protect infants and has identified a LOAEL of 630 mg/L based on diarrhea in infants receiving formula made with high-sulfate water. The Drinking Water Standards of the U.S. Public Health Service recommend that sulfate in water 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 to 400 mg/L, and a guidance value of 400 mg/L based on aesthetic quality has been suggested. The current EPA National Secondary Maximum Contaminant Level for sulfate, based on organoleptic effects, is 250 mg/L.

Sulfide occurs as a salt (e.g., sodium, potassium, or calcium sulfide). Sulfur compounds occur naturally in the environment. The toxicity of a sulfide is a function of the metal to which the sulfur atom is bound. No toxicity information specific to sulfide was found in the available literature.

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

4.2.30 Tetraoxo-sulfate (1-)

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 tetraxo-sulfate (1-). Therefore, neither carcinogenicity nor systemic toxicity resulting from tetraoxo-sulfate (1-) exposure is included in the BHHRA.

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

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. Human and animal chronic exposures result in alterations of the brain, spinal cord, and peripheral nerves. In both humans and animals, alopecia is the most common indicator of long-term thallium poisoning.

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. 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.

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 0.23 to 0.28 mg/(kg × day). The subchronic RfDs are 8.00×10^{-4}

(thallium sulfate, chloride, and carbonate) or 9.00×10^{-4} mg/(kg × day) (thallium nitrate and acetate), and the chronic RfDs are 8.00×10^{-5} (thallium sulfate, chloride, and carbonate) or 9.00×10^{-5} mg/(kg × day) (thallium nitrate and acetate).

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.

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

4.2.32 Thorium (007440-29-1)

Information on the toxicity of thorium as a metal (not radionuclide, please see radionuclides) 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 thorium. Therefore, neither carcinogenicity nor systemic toxicity resulting from thorium exposure is included in the BHHRA.

4.2.33 Tin (CAS 007440-31-5)

Tin is a silver-white, very malleable and ductile metal that is insoluble in water. It is used to make solder, aircraft parts, tin alloys, perfumes, and soaps. Tin is also used to make containers for food and beverages.

The probable routes of human exposure to tin are through inhalation of dust and eye and skin contact. Tin is not particularly toxic, but dust particles can irritate the eyes and respiratory system. Skin and eye irritation have been observed in both humans and animals after acute and intermediate exposure to inorganic tin compounds. Gastrointestinal effects have been observed in humans after the ingestion of tin from food or beverage containers. Chronic inhalation of dust or fume of tin oxide can cause accumulation of tin in the lungs (Stannosis), but no functional changes or systemic disease have been observed in humans or animals. Neither genotoxic effects nor carcinogenic potential has been clearly demonstrated in humans or animals after inhalation, ingestion, or dermal contact. The EPA's carcinogenic classification of inorganic tin is Group D (not classifiable as to human carcinogenicity).

The oral and dermal Reference Dose (RfD) for chronic exposures is 6.0×10^{-1} and 6.0×10^{-2} mg/(kg × day), respectively for tin. A gastrointestinal absorption factor of 10% was used.

4.2.34 Titanium (CAS 007440-32-6)

Information on the toxicity of titanium 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 titanium. Therefore, neither carcinogenicity nor systemic toxicity resulting from titanium exposure is included in the BHHRA.

4.2.35 Uranium (metal and soluble salts) (CAS 007440-61-1) (see radionuclide section, also)

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 radiation effects 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 1985). 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 3.0×10^{-3} and 2.55×10^{-3} mg/(kg × day), respectively for uranium. A gastrointestinal absorption factor of 85% was used.

References

Sittig, M. 1985. *Handbook of Toxic and Hazardous Chemicals and Carcinogens*, Noyes Publications, Park Ridge, NJ.

4.2.36 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 ($NaVO_3$), sodium orthovanadate (Na_3VO_4), vanadyl sulfate ($VOSO_4$), and ammonium vanadate (NH_4VO_3). Vanadium is used primarily as an alloying agent in steels and non-ferrous metals. 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% of dietary amount), but slightly more readily absorbed through the lungs (20-25%). Absorbed vanadium is widely distributed in the body, but short-term localization occurs primarily in bone, kidneys, and liver. 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). Vanadium is excreted primarily in the feces following oral exposures and primarily in the urine following inhalation exposures.

The toxicity of vanadium depends on its physico-chemical 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. 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. Minimal effects seen after subchronic oral exposures to animals include diarrhea, altered renal function, and decreases in erythrocyte counts, hemogloblin, and hematocrit. 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.

RfD for chronic oral exposures are: 7×10^{-3} mg/(kg × day) for vanadium; 9×10^{-3} mg/(kg × day) for vanadium pentoxide; 2×10^{-2} mg/(kg × day) for vanadyl sulfate; and 1×10^{-3} mg/(kg × day) for sodium metavanadate. The subchronic RfDs for these compounds are the same as the chronic RfDs, except for sodium metavanadate, which is 1×10^{-2} mg/(kg × day).

Inhalation exposures to vanadium and vanadium compounds result primarily in adverse effects to the respiratory system. In laboratory studies, minimal effects (throat irritation and coughing) occurred after an 8-hr exposure to 0.1 mg V/m^3. 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. Quantitative data are; however, 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.

The oral RfD used in this BHHRA is 7.0×10^{-3} mg/(kg × day). The dermal route RfD based on the oral RfD and a gastrointestinal absorption factor of 1% is 7.0×10^{-5} mg/(kg × day). The RfD for inhalation exposure has not been determined.

4.2.37 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, UV 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. In addition, zinc phosphide is used as a rodenticide.

Zinc is an essential element with recommended daily allowances ranging from 5 mg for infants to 15 mg for adult males.

Gastrointestinal absorption of zinc is variable (20-80%) and depends on the chemical compound as well as on zinc levels in the body and dietary concentrations of other nutrients. In individuals with normal zinc levels in the body, gastrointestinal absorption is 20-30%. 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 CO_2 exchange. Homeostatic mechanisms involving metallothionein in the mucosal cells of the gastrointestinal tract regulate zinc absorption and excretion.

In humans, acutely toxic oral doses of zinc cause nausea, vomiting, diarrhea, and abdominal cramps and in some cases gastric bleeding. Ingestion of zinc chloride can cause burning in the mouth and throat, vomiting, pharyngitis, esophagitis, hypocalcemia, and elevated amylase activity indicative of pancreatitis. 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. 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. There is also limited evidence that the human immune system may be impaired by subchronic exposures. In animals, gastrointestinal and hepatic lesions; pancreatic lesions; anemia; and diffuse nephrosis 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. Anemia and pancreatitis were the major adverse effects observed in chronic animal studies. Teratogenic effects have not been seen in animals exposed to zinc; however, high oral doses can affect reproduction and fetal growth.

The reference dose for chronic oral exposure to zinc is under review by EPA; the currently accepted RfD for both subchronic and chronic exposures is 0.2 mg/(kg × day) based on clinical data demonstrating zinc-induced copper deficiency and anemia in patients taking zinc sulfate for the treatment of sickle cell anemia. The chronic oral RfD for zinc phosphide is 3×10^{-4} mg/(kg × day), and the subchronic RfD is 3×10^{-3} mg/(kg × day).

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. 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. Pulmonary inflammation and changes in lung function have also been observed in inhalation studies on animals.

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/m^3. Gastrointestinal distress, as well as enzyme changes indicative of liver dysfunction, have also been reported in workers occupationally exposed to zinc; 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 reference concentration has not been derived for zinc or zinc compounds.

No case studies or epidemiologic evidence has been presented to suggest that zinc is carcinogenic in humans by the oral or inhalation route. 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; 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 F0 to the F4 generation in the tumor-resistant strain (from 0.8 to 25.7%, vs. 0.0004% in the controls), and higher tumor frequencies in two tumor-susceptible strains (43.4% and 32.4% vs. 15% in the controls).

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

The oral RfD used in this BHHRA is 3.0×10^{-1} mg/(kg × day). The dermal route RfD based on the oral RfD and a gastrointestinal absorption factor of 20% is 6.0×10^{-2} mg/(kg × day). The RfD for inhalation exposure has not been determined.

4.2.38 Zirconium (CAS 007440-32-6)

Information on the toxicity of zirconium was not found in the available literature. When information bec5mes available, it will be included in this report.

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

4.3 ORGANIC COMPOUNDS

4.3.1 1,1,2-Trichloroethane (CAS 000079-00-5) (RAIS)

1,1,2-Trichloroethane (CAS Reg. No. 79-00-5), 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.

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% of the total release is to air, 10-30% 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.

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.

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.

The oral LD_{50} for mice (378-491 mg/kg) indicates that in animals the acute oral toxicity of 1,1,2-trichloroethane is moderate. 1,1,2-Trichloroethane is a central nervous system depressant, inducing sedation in mice at oral doses of 378 mg/kg and drowsiness, incoordination, and narcosis in dogs at 289 mg/kg. 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. Decreased survival was reported in female $B6C3F_1$ mice exposed to 195 or 390 mg/kg/day for 78 weeks.

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. As noted previously, 1,1,2-trichloroethane is a central nervous system depressant inducing narcosis; death results from respiratory arrest. In mice, a concentration of 3750 ppm for 30 minutes produced central nervous system depression and significantly increased liver enzyme activity within 18 minutes and death in half the animals within 10 hours. 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. Repeated topical applications of 0.1 mL 1,1,2-trichloroethane produced erythema, edema, fissuring, and scaling of rabbit and guinea pig skin.

An oral reference dose of 0.04 mg/kg/day for subchronic exposure and 0.004 mg/kg/day for chronic exposure to 1,1,2-trichloroethane was calculated based on a NOAEL of 3.9 mg/kg/day and a lowest observed adverse effects level (LOAEL) of 44 mg/kg/day from a 90-day drinking water study with mice. Clinical chemistry alterations indicative of liver damage were identified as critical effects. An inhalation reference concentration for 1,1,2-trichloroethane is under review by EPA.

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 $B6C3F_1$ mice (195 or 390 mg/kg/day), 5 days/week for 78 weeks. 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% and 76% in the low- and high-dose groups, respectively, compared with 10% in vehicle controls, and 33% and 89% 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.

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 5.7E-2 $(mg/kg/day)^{-1}$ and the unit risk for drinking water is 1.6E-6 $(\mu g/L)^{-1}$. The inhalation slope factor and unit risk are 5.7E-2 $(mg/kg/day)^{-1}$ and 1.6E-5 $(\mu g/m3)^{-1}$, respectively.

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 $[mg/(kg \times day)]^{-1}$, 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 above, 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%.

4.3.2 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.

The available evidence indicates that 1,1-dichloroethane can be readily absorbed following inhalation and oral exposures. 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. In animal studies, orally administered 1,1-dichloroethane was excreted primarily in expired air as the unmetabolized chemical.

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, 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. A subchronic oral RfD of 1 mg/kg/day and a chronic oral RfD of 0.1 mg/kg/day (based on an inhalation

study in rats and route-to-route extrapolation) are listed in HEAST; however, an oral RfD is currently not found in IRIS. A U.S. Environmental Protection Agency reassessment of the oral RfD is pending.

At high vapor concentrations (26,000 ppm), 1,1-dichloroethane induces anesthesia and can cause cardiac arrhythmia in humans, but no fatalities have occurred. 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. 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. 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.

1,1-Dichloroethane is placed in Group C, possible human carcinogen, 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. Slope factors and unit risks have not been calculated.

4.3.3 1,1-Dichloroethene (CAS 000075-35-4) (RAIS)

1,1-Dichloroethene (CAS No. 75-35-4), also known as 1,1-dichloroethylene 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.

1,1-Dichloroethene does not occur naturally but is found in the environment because of 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-dichloroethene. Smaller amounts are released to surface waters and soils. Loss of 1,1-dichloroethene from water and soils is primarily because of volatilization. In the atmosphere, reaction with photochemically generated hydroxyl radicals is expected to be the predominant removal mechanism. Human exposure to 1,1-dichloroethene is potentially highest in workplace settings and in the vicinity of hazardous waste sites where the compound may contaminate environmental media.

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

In experimental animals, the liver and kidneys are target organs for the toxic effects of 1,1-dichloroethene. Subchronic oral exposure for 90 days to 1,1-dichloroethene in drinking water produced slight hepatotoxic effects at 200 ppm, and chronic oral exposure to drinking water for 2 years produced hepatocellular changes in males at >=100 ppm and in females at >=50 ppm. 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. Exposure by inhalation to 55 ppm 1,1-dichloroethene, 6 hours/day, 5 days/week for up to 1 year produced fatty liver changes in rats and focal degeneration and necrosis in mice.

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-dichloroethene in the drinking water. 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 >=15 ppm and in rats and rabbits at >=80 ppm and >=160 ppm, respectively.

An oral RfD of 9E-3 mg/kg/day was derived for chronic exposure and subchronic exposure to 1,1-dichloroethene, based on liver lesions seen in rats in a 2-year drinking water study. The oral RfD is currently under review and may be subject to change. An inhalation RfC for 1,1-dichloroethene is under review.

An epidemiology study using a small cohort found no association between the occurrence of cancer or cancer mortality and exposure to 1,1-dichloroethene. Oral carcinogenicity bioassays (drinking water or gavage exposures) with experimental animals gave generally negative results. In one inhalation study, 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.

Based on EPA guidelines, 1,1-dichloroethene was assigned to weight-of-evidence group C, possible human carcinogen. For oral exposure, the slope factor is 6E-1 $[mg/(kg \times day)]^{-1}$ and the unit risk is 1.7E-5 $(\mu g/L)^{-1}$. The inhalation slope factor and unit risk are 1.2E+0 $[mg/(kg \times day)]^{-1}$ and 5.0E-5 $(\mu g/m^3)^{-1}$, respectively.

The oral, inhalation, and dermal cancer slope factors used in the BHHRA for 1,1-dichloroethene are 6.00E-1, 1.20E+0, and 6.00E-1 $[mg/(kg \times day)]^{-1}$, respectively. The oral and dermal RfDs used in the BHHRA are 9.00E-3 and 9.00E-3 mg/(kg × day). An inhalation RfD was not found, and based on the localized effects discussed above, 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 100%.

4.3.4 1,2-Dichlorobenzene (CAS 000095-50-1)

The oral RfD used in this BHHRA is 9.0E-2 mg/(kg × day). The dermal route RfD based on the oral RfD and a gastrointestinal absorption factor of 80% is 7.2E-2 mg/(kg × day). The RfD for inhalation exposure used in this BHHRA is 5.71E-2 mg/(kg × day).

4.3.5 1,2-Dichloroethane (CAS 000107-06-2) (RAIS)

1,2-Dichloroethane is a clear, colorless, oily liquid with a sweet, pleasant odor. 1,2-Dichloroethane is used primarily as a chemical intermediate and a solvent in closed systems in the manufacture of vinyl chloride, as well as in the synthesis of tetrachloroethene, trichloroethene, 1,1,1-trichloroethane, vinylidene chloride, aziridines, and ethylenediamines. 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 ore flotation, in paints, coatings, adhesives, varnishes, finish removers, soaps, and scouring agents.

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. It is distributed throughout the body but may be concentrated in adipose tissue. The compound can also accumulate in breast milk and may cross the placenta. Metabolism of 1,2-dichloroethane most likely involves conjugation with glutathione. Urinary metabolites are likely to include thiodiglycolic acid, chloroacetic acid, and N-acetyl-S-carboxymethyl-L-cysteine. Excretion occurs primarily through elimination of soluble urinary metabolites.

Bronchitis, hemorrhagic gastritis and colitis, hepatocellular damage, renal tubular necrosis, central nervous system depression, and histopathological changes in the brain have been reported in cases of acute oral poisoning of humans. Animal data indicate that short-term exposures may produce immune system deficiencies, and subchronic or chronic oral exposures may affect the liver or kidney. Subchronic or chronic oral reference doses for 1,2-dichloroethane have not been adopted by the EPA; however, a provisional RfD of 0.03 mg/kg/day has been calculated by the Superfund Health Risk Technical Support Center from a NOAEL of 26 mg/kg/day for rats tested in a subchronic gavage study. 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 EEG, liver and kidney damage, pulmonary edema, and hemorrhages throughout the body. Short-term exposures to animals have resulted in central nervous system 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 immuno-deficiency. Chronic occupational exposure to 1,2-dichloroethane may result in central nervous systems 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. Subchronic or chronic inhalation exposures to animals resulted in pathological lesions in the kidney, liver, heart, lungs, and testes. A subchronic or chronic inhalation reference concentration for 1,2-dichloroethane has not been adopted and verified by EPA; however, a provisional RfC of 0.005 mg/m^3 has been calculated by the Superfund Health Risk Technical Support Center from a LOAEL (gastrointestinal disturbances and liver and gallbladder disease) of 10 mg/m^3 for occupationally exposed workers. 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. Mice treated by topical application of 1,2-dichloroethane exhibited an increased incidence of lung papillomas. The oral slope factor for 1,2-dichloroethane is 9.1E-2 $(\mu g/kg/day)^{-1}$, and the drinking water unit risk is 2.6E-6 $(\mu g/L)^{-1}$. The inhalation slope factor is 9.1E-2 $(\mu g/kg/day)^{-1}$, and the inhalation unit risk is 2.6E-5 $(\mu g/m^3)^{-1}$.

The oral, dermal, and inhalation cancer slope factors used in the BHHRA for 1,2-dichloroethane are 9.10E-2 $[mg/(kg \times day)]^{-1}$. 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% was used to derive the dermal slope factor.

4.3.6 1,2-Dichloroethene (total (CAS 00540-59-0), *cis*- (CAS 000156-59-2), and *trans*- (CAS 000156-60-5))

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. 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.

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. 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. Secondary target organs include the central nervous system and lung.

Based on an unpublished study describing decreased hemoglobin and hematocrits in rats treated by gavage for 90 days, EPA 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. The RfDs were derived from a LOAEL of 175 mg/kg/day that was based on increased serum alkaline phosphatase activity in mice that received trans-1,2-dichloroethene in their drinking water. 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 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 1,2-dichloroethene were found; therefore, carcinogenicity from exposure could not be quantified in the BHHRA. The oral and dermal RfDs for a mixture of *trans*- and *cis*-1,2-dichloroethene used in the BHHRA are 9.00E-3 and 7.20E-3, respectively. The oral and dermal RfDs for *cis*-1,2-dichloroethene used in the BHHRA are 1.00E-2 and 1.00E-2, respectively. The oral and dermal RfDs for *trans*-1,2-dichloroethene used in the BHHRA are 2.00E-2 and 2.00E-2, respectively. The RfDs were derived from a LOAEL of 175 mg/kg/day that was based on increased serum alkaline phosphatase activity in mice that received *trans*-1,2-dichloroethene in their drinking water. Inhalation RfDs were derived from RfC values and are 9.00E-03, 1.00E-02, and 2.00E-02 mg/(kg × day) for the mixture, cis-, and trans-isomers, respectively. The dermal RfD for the mixture of *trans*- and *cis*-1,2-dichloroethene was derived from the oral toxicity value using a gastrointestinal absorption factor of 80%; the complimentary value for *cis*-or *trans*- 1,2-dichloroethene is 100% .

4.3.7 1,3,5-Trimethylbenzene (CAS 000108-67-8)

1,3,5-Trimethylbenzene is also known as mesitelene or mesitylene.

No cancer slope factors for 1,3,5-trimethylbenzene were found; therefore, carcinogenicity from exposure could not be quantified in the BHHRA. The oral RfD used in this BHHRA is 5.0E-2 mg/(kg × day). The dermal route RfD based on the oral RfD and a gastrointestinal absorption factor of 80% is 4.0E-2 mg/(kg × day). The RfD for inhalation exposure used in this BHHRA is 1.7E-3 mg/(kg × day).

4.3.8 1,3-Dichloropropene, *trans*- (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.

4.3.9 1,4-Dichlorobenzene (CAS 000106-46-7) (RAIS)

1,4-Dichlorobenzene (CAS 106-46-7), 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 1993). 1,4-Dichlorobenzene is used to make mothballs, deodorant blocks used in restrooms, and in animal holding facilities to control odors (ATSDR 1993). It also has applications in fumigants, insecticides, lacquers, paints, and seed disinfection products (Leber and Benya 1994). Of the 1300 sites on the United States Environmental Protection Agency's 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 1993).

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 1993). 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 1987). Oral administration of 1200 and 1500 mg/kg/day resulted in degeneration and necrosis of rat hepatocytes. Increased incidences of hepatocellular

degeneration and individual cell necrosis were observed in male and female mice gavaged with 600-1800 mg/kg/day.

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 1987). 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%; low dose, 6%; high dose, 14%), a marginal increase in mononuclear cell leukemia in male rats (control, 10%; low dose, 14%; high dose, 22%), hepatocellular carcinomas in male mice (controls, 28%; low dose, 22.5%; high dose, 64%) and in female mice (controls, 10%; low dose, 10.4%; high dose, 38%), and hepatocellular adenomas in male mice (controls, 10%; low dose, 26.2%; high dose, 32%) and in female mice (controls, 20%; low dose, 12.5%; high dose, 42%). In this 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 (1987) that 1,4-dichlorobenzene was acting as a tumor promotor for liver tumors in male and female mice.

Reference concentrations (RfC) of 2.5 mg/m3 (0.42 ppm) for subchronic inhalation exposure (EPA 1995b) and 0.8 mg/m3 (0.13 ppm) for chronic inhalation exposure for 1,4-dichlorobenzene were derived (EPA 1995a) 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 No Observed Adverse Effects Level (NOAEL) was 301 mg/m3 (50 ppm). The LOAEL was 902 mg/m3 (150 ppm) (EPA 1995a). 1,4-Dichlorobenzene has been classified as C, possible carcinogen to humans (EPA 1995b). For oral exposure, the slope factor was 0.024 (mg/kg/day)-1, and the unit risk was 6.8E-7 (µg/L)-1 (EPA 1995 b).

The oral and dermal cancer slope factors used in the BHHRA for 1,4-dichlorobenzene are 2.4E-2 and 2.67E-2 $[mg/(kg \times day)]^{-1}$, respectively; where the dermal cancer slope factor was derived using a gastrointestinal absorption factor of 90%. The inhalation RfD used in the BHHRA is 2.29E-1. Oral and dermal RfDs were not found in the available toxicity information databases from EPA.

4.3.10 2,4-Dimethylphenol (CAS 000105-67-9)

Methylphenols (cresols) occur as several closely related compounds, including 2-methylphenol, (ortho-cresol), 3-methyl phenol, (meta-cresol), 4-methylphenol, (para-cresol) and 2,4-dimethylphenol. These compounds occur naturally and are found in many foods, human and animal urine, wood and tobacco smoke, crude oil, and coal tar. Man-made methylphenols are used as disinfectants and deodorizers, to dissolve substances, and in the manufacture of other chemicals.

Methylphenols are highly irritating and corrosive to tissue. Systemically, methylphenols can affect the central nervous system, liver, lungs, kidneys, gastrointestinal tract, eyes, and heart. Effects in the blood-forming system, respiratory irritation, and sclerosis of the lungs have been reported in rats exposed for intermediate durations. No studies are available on the potential chronic, reproductive, or teratogenic effects of methylphenols. One mutagenicity study on onion root tips reports negative results for 2-methylphenol, but 4-methylphenol produced cytological abnormalities and chromosome fragmentation. In an initiation-promotion study, methylphenols have promoted the tumorigenic action of a carcinogen when applied to mouse skin.

No cancer slope factors for any route of exposure were found for 2,4-dimethylphenol. The oral, dermal, and inhalation RfDs used in the BHHRA are 2.0E-2, 1.0E-2, and 2.0E-2 mg/(kg × day), respectively. A gastrointestinal absorption factor of 50% was used to determine the dermal RfD for 2,4-dimethylphenol.

4.3.11 4-Bromofluorobenzene (CAS 000460-00-4)

Information on the toxicity of 4-bromofluorobenzene 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 4-bromofluorobenzene. Therefore, neither carcinogenicity nor systemic toxicity resulting from 4-bromofluorobenzene exposure is included in the BHHRA.

4.3.12 4-Methyl-2-pentanone (methyl butyl ketone) (CAS 000108-10-1)

4-Methyl-2-pentanone, commonly known as hexanone or methyl isobutyl ketone, is a clear liquid with a sweet, sharp odor. It is used as a solvent in synthetic resinous paints, lacquers, and varnishes and in the manufacture of adhesives, rubber cements, 2,4-D and DDT. It is also used as an extractor in dewaxing mineral oils, refining tall oil, and cleaning metals.

4-Methyl-2-pentanone is absorbed through the respiratory and gastrointestinal tract. Inhalation of high concentrations of 4-methyl-2-pentanone can lead to irritation of the eyes and mucous membranes of the nose and throat. Still higher concentrations can lead to narcosis with the additional symptoms of weakness, headache, nausea, light headedness, vomiting, dizziness, and incoordination. Acute exposure to low concentrations in animals results in minor irritation of the eye. Higher concentrations can produce immediate signs of eye and nose irritation, salivation, lacrimation, and death. Eye irritation is the primary complaint of workers or volunteer subjects exposed chronically to 4-methyl-2-pentanone vapors. Some respiratory tract irritation is also observed. Higher exposures in the workplace have been associated with weakness, loss of appetite, headache, nausea, vomiting and diarrhea, in addition to more severe eye and throat irritation. Chronic exposures in animals have resulted in liver, kidney, and central nervous system effects. No information was located in the available literature pertaining to potential reproductive or developmental effects of 4-methyl-2-pentanone or to its mutagenic or carcinogenic potential.

No cancer slope factors for any route of exposure were found for 4-methyl-2-pentanone. The oral, inhalation, and dermal RfDs used in the BHHRA are 8.0E-2, 2.29E-2, and 6.4E-2, respectively. A gastrointestinal absorption factor of 80% was used to determine the dermal RfD for 4-methyl-2-pentanone.

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

Acetone (CAS No. 67-64-1) is a clear, colorless, highly flammable liquid with a vapor pressure of 182 mm Hg at 20°C. It is completely miscible in water and soluble in organics such as benzene and

ethanol. Its log K_{ow} 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.

Acetone may be released into the environment as stack emissions and/or fugitive emissions and in waste water effluents from facilities involved in its production and use as a chemical intermediate and solvent. Acetone is also a natural metabolic byproduct 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. Acetone has been detected in groundwater and drinking water.

Acetone can be absorbed through the lungs, digestive tract, and the skin. It is rapidly transported throughout the body and is not preferentially stored in any body tissue. 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. Ingestion of 10–20 mL (7.9–15.8 g) generally is not toxic, and consumption of 20 g/day for several days resulted in only slight drowsiness. 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 oral and dermal RfDs used in the BHHRA are 1.0E-1 and 8.3E-2, respectively. A gastrointestinal absorption factor of 83% was used to determine the dermal RfD for acetone.

Information on the inhalation toxicity of acetone to humans is derived from occupational and laboratory studies. Typical symptoms of inhalation exposure are central nervous system depression and irritation of the mucous membranes of the eyes, nose, and throat. Central nervous system 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/m^3). Irritant effects have been reported at concentrations of 500 ppm (1210 mg/m^3) and higher. Transient effects were reported in workers exposed to 600–2150 ppm (1452–5203 mg/m^3). Extremely high concentrations (> 29 g/m^3) can cause dizziness, confusion, unsteadiness, and unconsciousness. Prolonged occupational exposures to acetone vapors have not been associated with chronic systemic disorders.

Studies have shown that acetone vapor concentrations in excess of 8000 ppm (19.36 mg/m^3) are generally required to produce signs of central nervous system depression in animals, but concentrations as low as 500 ppm (1210 mg/m^3) may cause subtle behavioral changes. Little information is available on subchronic or chronic inhalation toxicity in animals.

An inhalation reference concentration (RfC) has not been derived for acetone.

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. 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. In the latter study, the incidence of malformations was not increased by exposure to acetone.

No evidence is available that suggests acetone is carcinogenic in humans or animals. 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 DNA interaction indicate that acetone is not genotoxic except under severe conditions. Acetone is classified by EPA in weight-of-evidence Group D, not classifiable as to human carcinogenicity.

No cancer slope factors for any route of exposure were found for acetone. The oral, inhalation, and dermal RfDs used in the BHHRA are 1.0E-1, 1.0E-1, and 8.3E-2, respectively. A gastrointestinal absorption factor of 83% was used to determine the dermal RfD for acetone.

4.3.14 Acrylonitrile (CAS 000107-13-1)

Acrylonitrile is also known as propenenitrile; vinyl cyanide; 2-Propenenitrile; Cyanoethylene; Fumigrain; propenonitrile; miller's fumigrain; TL 314; Propenitrile.

The oral, inhalation, and dermal cancer slope factors for acrylonitrile used in this BHHRA are 5.4E-1, 2.4E-1, and 6.75E-1 $[mg/(kg \times day)]^{-1}$, respectively. A gastrointestinal absorption factor of 80% was used to determine the dermal cancer slope factor for acrylonitrile.

The oral, inhalation, and dermal RfDs for acrylonitrile used in this BHHRA are 1.0E-3, 5.71E-4, and 8.0E-4 mg/(kg × day), respectively. A gastrointestinal absorption factor of 80% was used to determine the dermal RfD for acrylonitrile.

4.3.15 Aroclor® 1254 (CAS 011097-69-1) (RAIS)

Aroclor® 1254 is a PCB mixture containing approximately 21% $C_{12}H_6Cl_4$, 48% $C_{12}H_5Cl_5$, 23% $C_{12}H_4Cl_6$, and 6% $C_{12}H_3Cl_7$ with an average chlorine content of 54%. 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® is strongly sorbed to soil and remains immobile when leached with water; however, the mixture is highly mobile in the presence of organic solvents. PCBs are resistant to chemical degradation by oxidation or hydrolysis. However, biodegradation, especially of lower chlorinated PCBs, can occur. 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.

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.

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 post-exposure. 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.

Hepatotoxicity is a prominent effect of Aroclor-1254 that has been well characterized. 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.

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 were observed in treated monkeys. A 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. The subchronic oral RfD is 5E-05 mg/kg/day.

Data are suggestive but not conclusive concerning the carcinogenicity of PCBs in humans. The 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 the EPA to classify PCBs as group B2, probable human carcinogen.

Aroclor-1254 has two designations for toxicity values. The first is Aroclor-1254-water. Aroclor-1254-water is used for water pathways. The oral, dermal, and inhalation cancer slope factors used in the BHHRA for Aroclor-1254-water are 4.00E-1, 4.44E-1, and 4.00E-1 $[mg/(kg \times day)]^{-1}$, respectively. The oral, dermal, and inhalation RfDs used in the BHHRA for Aroclor 1254-water are 2.00E-5, 1.80E-5, and 2.00E-5 respectively. The dermal cancer slope factor and RfD were derived using a gastrointestinal absorption factor of 90%.

The second designation is Aroclor-1254-soil. Aroclor-1254-soil is used for soil and biota pathways. The oral, dermal, and inhalation cancer slope factors used in the BHHRA for Aroclor-1254-soil are 2.00E+0, 2.22E+0, and 2.00E+0 $[mg/(kg \times day)]^{-1}$, respectively. The oral, dermal, and inhalation RfDs used in the BHHRA for Aroclor-1254-soil are 2.00E-5, 1.80E-5, and 2.00E-5, respectively. The dermal cancer slope factor and RfD were derived using a gastrointestinal absorption factor of 90%.

4.3.16 Benzene (CAS 000071-43-2)

Benzene is absorbed via ingestion, inhalation, and skin application. Experimental data indicate that animals can absorb up to 95% of oral doses and that humans can absorb up to 80% of inhaled benzene (after 5 minutes of exposure). 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 to 36% enters the body through the skin.

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. 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.

Numerous studies indicate that the metabolism of benzene is required for its toxicity. The liver is the main site for the metabolism of benzene; the bone marrow, a minor site. Phenol, hydroquinone, catechol, and benzene oxide are the major metabolites. 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.

Benzene is eliminated either unchanged in expired air or as metabolites in the urine. 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. These data indicate that benzene is of low acute toxicity.

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. A four week exposure of mice to ~8 mg of benzene/kg/day in the drinking water induced the synthesis and catabolism of monoamine neurotransmitters and produced dose-related decreases in red-blood cell parameters and lymphocyte numbers. Rats and mice that were treated with benzene by gavage for 103 weeks developed a dose-related lymphocytopenia (LOAEL, 25 mg/kg/day) and mice had hyperplasia of the bone marrow and lymphoid depletion of the splenic follicles and thymus (100 mg/kg/day).

Inhalation of benzene vapor concentrations of 20,000 ppm for 5 to 10 minutes can be fatal to humans; death results from central nervous system depression. The estimated LC_{50} value for the rat is 13,700 ppm.

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. In animals, 1 week of exposure to 300 ppm induced behavioral effects, and one to four weeks of exposure to benzene concentrations ranging from 21-50 ppm suppressed the bone marrow (NOAEL, 10 ppm), the cellular immune response (NOAEL, 10 ppm), and the humoral immune response (LOAEL, 50 ppm).

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. A group of workers exposed to benzene concentrations of 30 and 150 ppm for 4 months to 1 year had increased incidences of pancytopenia. A group of patients who had been exposed to benzene concentrations of 150 to 650 ppm for 4 months to 15 years exhibited severe blood dyscrasias and eight of the 32 patients died with thrombocytopenic hemorrhage and infection. 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.

Benzene may also have long-term effects on the central nervous system. Workers exposed to benzene for 0.5 to 4 years exhibited EEG changes and atypical sleep activity consistent with neurotoxicity. Others exposed to benzene concentrations of 210 ppm for 6-8 years had peripheral nerve damage.

In humans, benzene crosses the placenta and is present in the cord blood in amounts equal to those in maternal blood; however, studies of the effects of benzene on human reproduction and development have been confounded by the presence of other chemicals in the environment. Benzene does produce developmental effects (fetal toxicity, but not malformations) in the offspring of treated animals, mostly at maternally toxic doses.

Oral and dermal reference doses/concentrations 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. An inhalation RfD of 1.71E-3 was used in this BHHRA.

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. Workers at risk were exposed in one study to 8-hour TWA concentrations ranging from 10 to 100 ppm and in another to 8-hour TWA concentrations ranging from <2 to >25 ppm. 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. In one study C57Bl/BNL mice had increased incidences of leukemia, lymphoma, and solid tumors after exposure to 300 ppm for only 16 weeks.

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. The oral and inhalation slope factors for benzene are 2.9E-2 $[mg/(kg \times day)]^{-1}$ and the oral and inhalation unit risk values are 8.3E-7 and 8.3E-6 $[\mu g/m^3]^{-1}$, respectively. A gastrointestinal absorption factor of 97% was used to calculate a dermal slope factor of 2.99E-2 $[mg/(kg \times day)]^{-1}$.

4.3.17 Bis(2-ethylhexyl)phthalate (CAS 000127-81-17) (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 ground water, surface water, drinking water, air, soil, plants, fish and animals. It is rapidly absorbed from the gastrointestinal tract primarily as mono(2-ethylhexyl)phthalate. The diester can be absorbed through the skin and from the lungs. 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.

Animal studies have indicated that the primary target organs are the liver and kidneys; however, higher doses are reported to result in testicular effects and decreased hemoglobin and packed cell volume. 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. An epidemiological study reported no toxic effects from occupational exposure to air concentrations of bis(2-ethylhexyl)phthalate up to 0.16 mg/m^3.

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. Developmental toxicity studies with rats and mice have shown that bis(2-ethylhexyl)phthalate is fetotoxic and teratogenic when given orally during gestation. Oral exposure has also been shown to result in decreased sperm count in rats. A RfD of 0.02 mg/kg/day for both subchronic and chronic oral exposure was calculated from a lowest-observed-adverse-effect level (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. A Reference Concentration (RfC) for inhalation exposure is not available.

Bis(2-ethylhexyl)phthalate is known to induce the proliferation of peroxisomes, which has been associated with carcinogenesis. 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. An increased incidence of neoplastic nodules and hepatocellular carcinomas was also reported in rats.

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 of 0.014 $(mg/kg/day)^{-1}$ for oral exposure was based on the combined incidence of hepatocellular carcinomas and adenomas in male mice. A drinking water unit risk of 4.0E-7 $(g/L)^{-1}$ was calculated based on the slope factor. A quantitative estimation of carcinogenic risk from inhalation exposure is not available.

The oral and dermal cancer slope factors used in the BHHRA for bis(2-ethylhexyl)phthalate are 1.40E-2 and 7.37E-2 $[mg/(kg \times day)]^{-1}$, 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 $[mg/(kg \times day)]^{-1}$, is used as a surrogate inhalation slope factor in the uncertainty discussion in Subsect. 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. 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% was used.

4.3.18 Bromomethane (CAS 000074-83-9)

No cancer slope factors for any route of exposure were found for bromomethane. The oral, inhalation, and dermal RfDs used in the BHHRA are 1.4E-3, 1.43E-3, and 1.12E-3 mg/(kg × day), respectively. A gastrointestinal absorption factor of 80% was used to determine the dermal RfD for bromomethane.

4.3.19 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% was used.

4.3.20 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 central nervous system 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% 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. Lung damage has also been reported in animals and humans but is not route specific and is believed to be secondary to kidney damage. 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.

Maternal toxicity and fetotoxic effects have been reported in rats following oral or inhalation exposure to carbon tetrachloride during gestation. 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.

A subchronic (RfDs) 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. Significantly higher doses caused minimal liver damage. A dose of 7.1 mg/kg/day was considered a LOAEL. A chronic RfD 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. Confidence in the oral RfD values is rated medium by EPA.

A chronic or subchronic RfC for inhalation exposure is currently under development by the EPA.

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 to 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. A drinking water unit risk of 3.7 × 10-6 (g/L)-1 and an inhalation unit risk of 1.5 x 10-5 $(g/m3)^{-1}$ have also been calculated by EPA.

The oral and dermal cancer slope factors used in the BHHRA for carbon tetrachloride are 1.30E-1 and 2.00E-1 $[mg/(kg \times day)]^{-1}$, respectively. An inhalation cancer slope factor of 5.30E-2 $[mg/(kg \times day)]^{-1}$ is used. The oral and dermal RfDs used in the BHHRA are 7.00E-4 and 4.55E-4 mg/(kg × day), respectively. An inhalation RfD of 5.71E-4 mg/(kg × day) is used. When calculating both the dermal route cancer slope factor and dermal route RfD from their respective oral values, a gastrointestinal absorption factor of 65% was used.

4.3.21 Chlorobenzene CAS (000108-90-7)

Chlorobenzene is also known as Benzene chloride; Chlorobenzol; MCB; Monochlorobenzol; chlorobenzene; Chlorobenzene Mono; monochlorobenzene**.**

No cancer slope factors were used in the BHHRA for chlorobenzene. The oral and dermal RfDs used in the BHHRA are 2.00E-2 and 6.2E-3 mg/(kg × day), respectively. An inhalation RfD of 5.71E-3 mg/(kg × day) was used. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 31% was used.

4.3.22 Chloroethane (CAS 000075-00-3)

Chloroethane, also known as ethyl chloride, is a colorless gas with a pungent odor. It is used as a refrigerant, solvent, alkylating agent, anesthetic, and in the production of tetraethyl lead, ethyl cellulose, dyes, medicinal drugs, and perfumes.

Chloroethane is readily absorbed through the lungs and skin. Acute exposure to chloroethane vapor has resulted in dizziness, lack of coordination, and analgesia. Nausea, abdominal cramps, vomiting, increased respiratory rate, respiratory paralysis, and cardiac depression have been observed in humans exposed to concentrations at or above 20,000 mg/kg. Mild eye irritation has been observed in volunteers exposed to 40,000 mg/kg. Histopathological changes in the lungs, liver, and kidney and depression of the central nervous system have been reported in animals. Women workers exposed to chloroethane and ethylenediamine, ammonia, polyethylene polyamines, and vinyl chloride had genital disorders including

cervicitis, vaginitis, and inflammation of the uterus. Chloroethane was mutagenic with or without metabolic activation in the Ames assay. In a study conducted by the National Toxicology Program, rats exposed to 15,000 mg/kg for 2 years exhibited increased incidences of basal cell carcinomas, sebaceous gland adenomas, and trichoepitheliomas. The incidence of uterine carcinomas in female mice was increased.

No cancer slope factors were available for use in the BHHRA. The oral, inhalation, and dermal RfDs used in the BHHRA are 4.00E-1, 2.86E+0, and 3.20E-1 mg/(kg × day), respectively. When calculating the dermal route RfD from the respective oral value, a gastrointestinal absorption factor of 80% was used.

4.3.23 Chloroform (CAS 67-66-3) (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. 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.

Target organs for chloroform toxicity are the liver, kidneys, and central nervous system. Liver effects (hepatomegaly, fatty liver, and hepatitis) were observed in individuals occupationally exposed to chloroform. Several subchronic and chronic studies by the oral or inhalation routes of exposure documented hepatotoxic effects in rats, mice, and dogs. Renal effects were reported in rats and mice following oral and inhalation exposures, but evidence for chloroform-induced renal toxicity in humans is sparse. Chloroform is a central nervous system depressant, inducing narcosis and anesthesia at high concentrations. Lower concentrations may cause irritability, lassitude, depression, gastrointestinal symptoms, and frequent and burning urination.

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. Chloroform may cause sperm abnormalities in mice and gonadal atrophy in rats.

A RfD of 0.01 mg/kg/day for subchronic and chronic oral exposure was calculated from a lowest-observed-adverse-effect level (LOAEL) of 15 mg/kg/day based on fatty cyst formation in the liver of dogs exposed to chloroform for 7.5 years. Development of an inhalation RfC is presently under review.

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. 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.

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 for chloroform is 6.1E-3 $(mg/kg/day)^{-1}$ for oral exposure and 8.1E-2 $(\mu g/m3)^{-1}$ for inhalation exposure. An inhalation unit risk of 2.3E-5 $(g/m3)^{-1}$ is based on hepatocellular carcinomas in mice in an oral gavage study.

The oral and dermal cancer slope factors used in the BHHRA for chloroform are 6.10E-3 and 3.05E-2 $[mg/(kg \times day)]^{-1}$, respectively. An inhalation cancer slope factor of 8.10E-2 $[mg/(kg \times day)]^{-1}$ 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 equivalent to the oral RfD of 1.0E-2 mg/(kg × day) is 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% was used.

4.3.24 Chloromethane (CAS 000074-87-3)

Chloromethane, also known as methyl chloride, is a colorless, flammable gas that has a faintly sweet odor. Chloromethane is a naturally occurring chemical that is present in air all over the world in concentrations from less than 0.001 mg/l to 0.003 mg/l. It is also manufactured in industry and used to produce silicones, agricultural chemicals and butyl rubber. Chloromethane was used as the cooling agent in refrigerators (more than 30 years ago). It is also found in cigarette smoke and smoke from burning wood, grass, coal, or certain plastics.

The principle route of exposure is through inhalation, but chloromethane can be ingested through drinking water or absorbed through the skin. The central nervous system is the major target of chloromethane toxicity. In acute exposures to high concentrations in humans, chloromethane reportedly causes headache; drowsiness; giddiness; ataxia; and ultimately convulsions, coma, and death. Single inhalation exposures in animal studies have shown respiratory, cardiovascular, and hepatic effects. Chloromethane leaking either from refrigerators or industrial cooling and refrigeration systems has caused several human deaths. Repeated exposures to lower concentrations usually cause fatigue, loss of appetite, muscular weakness, and drowsiness.

In addition to chloromethane's effects on the nervous system, effects on the liver, kidney, and cardiovascular system have been described in case reports of humans exposed for brief periods or for prolonged periods in occupational settings. In humans, chronic exposures often exert delayed effects that may last for months after exposure. Testicular atrophy, infertility, sterility (male rats), kidney tumors (male mice), and possible developmental effects (heart defects in mice) have been observed in animal studies but have not been reported in humans. Only inadequate evidence exists of carcinogenicity in humans and animals.

The oral and dermal cancer slope factors used in the BHHRA for chloromethane are 1.30E-2 and 1.63E-2 $[mg/(kg \times day)]^{-1}$, respectively. An inhalation cancer slope factor of 6.30E-3 $[mg/(kg \times day)]^{-1}$ is used. No oral, dermal, or inhalation RfDs were available. When calculating the dermal route cancer slope factor from the oral value, a gastrointestinal absorption factor of 80% was used.

4.3.25 Chrysene (CAS 000218-01-9) (also see toxicity profile for polycyclic aromatic hydrocarbons)

Chrysene, a polycyclic aromatic hydrocarbon, 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. 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% of the administered chrysene may be absorbed by oral, dermal, or inhalation routes. Following its absorption, chrysene is preferentially distributed to highly lipophilic regions of the body, most notably adipose and mammary tissue. 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. 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. 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. Hepatobiliary secretion with elimination in the feces is the predominant route of excretion.

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 reference concentration (RfC) for chrysene have not been derived. 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 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 $[mg/(kg \times day)]^{-1}$, 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%. No RfDs for chrysene were found; therefore, noncancer effects due to exposure to chrysene could not be estimated in the BHHRA.

4.3.26 Di-n-butyl phthalate (CAS 000084-74-2)

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 equivalent to the oral RfD of 1.00E-1 mg/(kg × day) was used. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 100% was used.

4.3.27 Dimethylbenzene (CAS 001330-20-7) (see toxicity profile for m,p-xylene)

Dimethylbenzene is also known as xylene and exists as three isomers (ortho, meta, and para).

No cancer slope factors were used in the BHHRA for dimethylbenzene. The oral and dermal RfDs used in the BHHRA are 2.00E+0 and 1.84E+0 mg/(kg × day), respectively. An inhalation RfD equivalent to the oral RfD of 2.00E+0 mg/(kg × day) was used. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 92% was used.

4.3.28 Ethane (CAS 000074-84-0)

Ethane is also known as dimethyl; methylmethane; ethyl hydride.

Information on the toxicity of environmentally-occurring ethane 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 environmentally-occurring ethane. Therefore, neither carcinogenicity nor systemic toxicity resulting from ethane exposure is included in the BHHRA.

4.3.29 Ethanol (CAS 000064-17-5)

Ethanol is also known as alcohol; anhydrol; methylcarbinol; ethyl hydrate; ethyl hydroxide; algrain; cologne spirit; fermentation alcohol; grain alcohol; jaysol; jaysol s; molasses alcohol; potato alcohol; spirit; spirits of wine; tecsol; Synasol.

Information on the toxicity of environmentally-occurring ethanol 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 environmentally-occurring ethanol. Therefore, neither carcinogenicity nor systemic toxicity resulting from ethanol exposure is included in the BHHRA.

4.3.30 Ethylbenzene (CAS 000100-41-4) (RAIS)

Ethylbenzene is a colorless, flammable liquid with a pungent odor. The water solubility of ethylbenzene is 0.014 g/100 mL and its vapor pressure is 10 mm Hg at 25°C. 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. Occupational exposure to ethylbenzene may occur during production and conversion to polystyrene and during production and use of mixed xylenes. The general public can be exposed to ethylbenzene in ambient air as a result of releases from vehicle exhaust and cigarette smoke.

Ethylbenzene can be absorbed through the lungs, digestive tract, and skin. It also crosses the placenta. The liver is the major organ of ethylbenzene metabolism. In humans the major metabolites of ethylbenzene are mandelic acid (64 to 70%) and phenylglyoxylic acid (25%); however, these compounds are only minor metabolites in laboratory animals. Excretion occurs primarily in the urine.

Ingestion of sublethal amounts of ethylbenzene is likely to cause central nervous system (CNS) depression, oro-pharyngeal and gastric discomfort, and vomiting; 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.

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). Concentrations of 1000 ppm (434 mg/m^3) can be highly irritating to the eyes of humans; the threshold for eye irritation has been reported to be 200 ppm (879 mg/m^3). No evidence is available to suggest that occupational exposures to ethylbenzene result in chronic toxic effects; 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/m^3) 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/m^3. The inhalation RfC for chronic exposures is 1 mg/m^3, based on developmental effects.

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; 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. Ethylbenzene is placed by EPA in Group D, not classifiable as to human carcinogenicity, based on a lack of data in humans and animals.

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% was used.

4.3.31 Ethylene (CAS 000074-85-1)

Ethylene is also known as ethene; acetene; olefiant gas; and bicarburretted hydrogen.

Information on the toxicity of environmentally-occurring ethylene 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 environmentally-occurring ethylene. Therefore, neither carcinogenicity nor systemic toxicity resulting from ethylene exposure is included in the BHHRA.

4.3.32 Fluorene (CAS 000086-73-7) (see toxicity profile for Polycyclic Aromatic Hydrocarbons)

Fluorene is also known as 9H-Fluorene; o-Biphenylenemethane; diphenylenemethane; 2,2'-methylenebiphenyl; o-biphenylmethane; 2,3-benzindene; 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 4.0E-2 and 2.0E-2 mg/(kg × day), respectively. An inhalation RfD equivalent to the oral RfD of 4.0E-2 mg/(kg × day) was used. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 50% was used.

4.3.33 Isophorone (CAS 000078-59-1)

The oral and dermal cancer slope factors used in the BHHRA for isophorone are 9.50E-4 and 1.90E-3 $[mg/(kg \times day)]^{-1}$, respectively. An inhalation cancer slope factor was not found. The oral and dermal RfDs used in the BHHRA are 2.00E-1 and 1.00E-1 mg/(kg × day), respectively. An inhalation RfD equivalent to the oral RfD of 2.00E-1 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 50% was used.

4.3.34 Methylene Chloride (CAS 000075-09-2) (RAIS)

Methylene chloride (CH_2Cl_2, CAS No. 75-09-2), 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.

Methylene chloride is readily absorbed from the lungs, the gastrointestinal tract, and to some extent through the skin. Metabolism of methylene chloride produces CO2 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. Major effects following acute inhalation exposure include fatigue, irritability, analgesia, narcosis, and death. CNS effects have also been demonstrated in animals following acute exposure to methylene chloride.

Impaired liver function has been associated with occupational exposure to methylene chloride. 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. Hepatocellular foci, fatty changes, and necrosis were reported following chronic inhalation exposure of rats and mice. Chronic oral exposure to methylene chloride via drinking water resulted in histopathological alterations of the liver in rats and mice. In addition, inhalation exposure of rats caused nonspecific degenerative and regenerative changes in the kidneys.

A subchronic and chronic oral RfD of 6E-2 mg/kg/day for methylene chloride has been calculated by EPA. This value is based on a NOAEL of 5.85 mg/kg/day derived from a chronic drinking water study with rats. This same study was adapted for the derivation of the subchronic and chronic RfC of 3E+0 mg/m^3 (NOAEL, 694.8 mg/m^3).

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. 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. Rats of both sexes exposed to concentrations of methylene chloride ranging from 500 to 4000 ppm showed increases of benign mammary tumors. An inhalation study with rats and hamsters revealed sarcomas of the salivary gland in male rats, but not in female rats or hamsters. Liver tumors observed in rats and mice that ingested methylene chloride in drinking water for 2 years provided suggestive evidence of carcinogenicity. Based on inadequate evidence of carcinogenicity in humans and on sufficient evidence in animals, EPA has placed methylene chloride in weight-of-evidence group B2, probable human carcinogen. A slope factor and unit risk of 7.5E-3 $[mg/(kg \times day)]^{-1}$ and 2.1E-7 $(\mu g/L)^{-1}$, respectively, was derived for oral exposure to methylene chloride. The inhalation unit risk is 4.7E-7 $(\mu g/m3)^{-1}$.

The oral and dermal cancer slope factors used in the BHHRA are 7.50E-3 and 7.89E-3 $[mg/(kg \times day)]^{-1}$, respectively. An inhalation cancer slope factor of 1.65E-3 was used. The oral and dermal RfDs used in the BHHRA are 6.00E-2 and 5.70E-2 $mg/(kg \times day)$, respectively. An inhalation reference dose of 8.57E-01 $mg/(kg \times day)$ 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% was used.

4.3.35 Naphthalene (CAS 000091-20-3) (RAIS)

Naphthalene (CAS Reg. No. 91-20-3), a white solid with a characteristic odor of mothballs, is a polycyclic aromatic hydrocarbon composed of two fused benzene rings. The principal end use of naphthalene is as a 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.

Naphthalene can be absorbed by the 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. Hemolytic anemia has been observed in an infant dermally exposed to naphthalene and in infants whose mothers were exposed to naphthalene during pregnancy. Infants and individuals having a congenital deficiency of erythrocyte glucose-6-phosphate dehydrogenase are especially susceptible to naphthalene-induced hemolytic anemia.

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 and in rabbits and rats exposed orally to naphthalene. A number of deaths have been reported following intentional ingestion of naphthalene-containing mothballs. 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.

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. 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. Subchronic oral exposure to 133 mg/kg/day for 90 days produced decreased spleen weights in female mice. Reduced numbers of pups/litter were observed when naphthalene was administered orally to pregnant mice. Negative results in a two-year feeding study with rats receiving 10-20 mg naphthalene/kg/day and equivocal results in a mouse lung tumor bioassay suggest that naphthalene is not a potential carcinogen.

A subchronic and chronic oral RfD of 4E-2 mg/kg/day for naphthalene has been calculated by EPA. These values are based on a NOEL of 50 mg/kg/day derived from a subchronic oral toxicity study with rats. The RfD is currently under review by EPA and may be subject to change. 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 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 and dermal RfDs used in the BHHRA are 2.00E-2 and 1.60E-2 mg/(kg × day), respectively. An inhalation RfD of 8.57E-4 mg/(kg × day) was used. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 80% was used.

4.3.36 Phenanthrene (CAS 000085-01-8) (RAIS) (see toxicity profile for Polycyclic Aromatic Hydrocarbons)

Phenanthrene is a PAH that can be derived from coal tar. Currently, there is no commercial production or use of this compound. 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 in foods.

Phenanthrene is absorbed following oral and dermal exposure. Data from structurally related PAHs suggest that phenanthrene would be absorbed from the lungs. 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.

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. 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. The chemical is not currently listed in IRIS or HEAST.

No inhalation bioassays were available to assess the carcinogenicity of phenanthrene. A single oral dose of phenanthrene did not induce mammary tumors in rats and a single subcutaneous injection did not result in treatment-related increases in tumor incidence in mice. Neonate mice administered intraperitoneal or subcutaneous injections of phenanthrene also did not develop tumors. No skin tumors were reported in two skin painting assays with mice. Phenanthrene was also tested in several mouse skin initiation-promotion assays. It was active as an initiator in one study, inactive as an initiator in four others, and inactive as a promoter in one study.

Based on no human data and inadequate data from animal bioassays, EPA 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.

4.3.37 Polychlorinated biphenyl (CAS 27323-18-8) (see toxicity profile for Aroclor-1254)

4.3.38 Polycyclic Aromatic Hydrocarbons

Polycyclic aromatic hydrocarbons are a group of chemicals that are formed during the incomplete burning of wood and fuel, including coal, oil, gas, and other organic substances (ATSDR 1989). In any medium, PAHs most often exist as complex mixtures of compounds. Exposure to PAHs may occur via inhalation, ingestion, and dermal contact. Based on toxicity, these compounds have been divided into two main groups: carcinogenic PAHs and noncarcinogenic PAHs.

Carcinogenic Polycyclic Aromatic Hydrocarbons. Based on available data, benzo[a]pyrene is one of the most potent of the carcinogenic PAHs. Other PAHs considered to be carcinogenic are benz[a]anthracene, benzo[b]fluoranthene, benzo[k]fluoranthene, chrysene, dibenz[a,h]anthracene, and indeno[1,2,3-cd]pyrene.

The arrangement of aromatic rings in the benzo[a]pyrene molecule and other PAHs gives it a “bay-region” that is often correlated with carcinogenic properties. In general, bay-region PAHs and some of their metabolites are known to react with cellular macromolecules, including DNA, which may account for the toxicity and carcinogenicity of these compounds (Francis 1992). The primary toxicological concern with exposure to this group of PAHs is carcinogenicity. No case reports or epidemiological studies concerning the significance of human exposure to individual PAHs are available. Coal tar and other materials known to be carcinogenic to humans, however, contain PAHs (Francis 1985). Lung and skin cancers in humans have been associated with chronic exposure by inhalation and dermal contact, respectively, to mixtures of compounds including carcinogenic PAHs (ATSDR 1989). Several individual PAHs administered to several animal species by various routes have been found to be carcinogenic at both local and systemic sites. Long-term experimental studies resulted in tumors in the liver, mammary gland, respiratory and gastrointestinal tracts, and skin (ATSDR 1989). Carcinogenic PAHs are also reported to be mutagenic in a variety of test systems.

Reproductive effects in mice fed benzo[a]pyrene and adverse effects in their offspring, including birth defects and decreased body weight, have been reported, although reproductive toxicity associated

with PAH exposure has not been demonstrated in humans (ATSDR 1989). Toxic effects have also been observed in rapidly dividing cells of the intestinal epithelium, testes, and ovaries (oocytes). Animal studies also indicate that exposure to bay-region PAHs can damage the hematopoietic system, leading to progressive anemia as well as agranulocytosis. The lymphoid system can also be affected, resulting in lymphopenia.

As indicated previously, available data indicate that not all of the carcinogenic PAHs are as potent as benzo[a]pyrene (ICF-Clement 1988, EPA 1992). In recent guidance published by the EPA (1993), it is recommended that a series of relative potency values (orders of magnitude) be used for the risk assessment of oral exposure to PAHs, with carcinogenic potency being compared to that of benzo[a]pyrene.

Noncarcinogenic Polycyclic Aromatic Hydrocarbons. Polycyclic aromatic hydrocarbons not considered to be carcinogenic include acenaphthene, acenaphthylene, anthracene, benzo[g,h,i]perylene, fluoranthene, fluorene, methylnaphthalene, naphthalene, phenanthrene, and pyrene.

Polycyclic aromatic hydrocarbons are toxic to the skin. For example, naphthalene is a primary skin irritant and causes erythema and dermatitis on repeated contact (Sittig 1985), and acenaphthene is irritating to the skin and mucous membranes of humans and animals (Faust 1994). Other noncarcinogenic effects of PAHs have been observed in animals; however, of these, only effects of the blood and blood-forming system and of the skin have also been reported in humans (ATSDR 1989). Animal studies indicate that PAHs may adversely affect the gastrointestinal tract, liver, kidneys, lungs, hematopoietic system, and may suppress the immune system after both short- and long-term exposure. Oral exposure of animals to acenaphthene caused reproductive effects, including decreased ovary weights, decreased ovarian and uterine activity, and fewer and smaller corpora lutea (Faust 1991, 1994). Mutagenic and carcinogenic effects of the noncarcinogenic PAHs have not been reported.

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4.3.39 Tetrachloroethene (CAS 000127-18-4) (RAIS)

Tetrachloroethene (CAS No. 127-18-4) is a halogenated aliphatic hydrocarbon with a vapor pressure of 17.8 mm Hg at 25C. The chemical is used primarily as a solvent in industry and, less frequently, in commercial dry-cleaning operations. Occupational exposure to tetrachloroethene 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.

The respiratory tract is the primary route of entry for tetrachloroethene. The chemical is rapidly absorbed by this route and reaches an equilibrium in the blood within 3 hours after the initiation of exposure. Tetrachloroethene is also significantly absorbed by the gastrointestinal (g.i.) tract, but not through the skin. The chemical accumulates in tissues with high lipid content, where the half-life is estimated to be 55 hours, and has been identified in perirenal fat, brain, liver, placentofetal tissue, and amniotic fluid. The proposed first step for the biotransformation of tetrachloroethene is the formation of an epoxide thought to be responsible for the carcinogenic potential of the chemical. Tetrachloroethene is excreted mainly unchanged through the lungs, regardless of route of administration. The urine and feces comprise secondary routes of excretion. The major urinary metabolite of tetrachloroethene, trichloroacetic acid, is formed via the cytochrome P-450 system.

The main targets of tetrachloroethene toxicity are the liver and kidney by both oral and inhalation exposure, and the central nervous system 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. 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.

Some epidemiology studies have found an association between inhalation exposure to tetrachloroethene and an increased risk for spontaneous abortion, idiopathic infertility, and sperm abnormalities among dry-cleaning workers, but others have not found similar effects. The adverse effects in humans are supported in part by the results of animal studies in which tetrachloroethene induced fetotoxicity (but did not cause malformations) in the offspring of treated dams.

Reference doses for subchronic and chronic oral exposure to tetrachloroethene are 1E-1 mg/kg/day and 1E-2 mg/kg/day, respectively. These values are based on hepatotoxicity observed in mice given 100 mg tetrachloroethene/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. The tenuous finding of an excess of liver tumors in humans is strengthened by the results of carcinogenicity bioassays in which tetrachloroethene, administered either orally or by inhalation, induced hepatocellular tumors in mice. The chemical also induced mononuclear cell leukemia and renal tubular cell tumors in rats. Tetrachloroethene was negative for tumor initiation in a dermal study and for tumor induction in a pulmonary tumor assay.

Although EPA's Science Advisory Board recommended a weight-of-evidence classification of C-B2 continuum © = possible human carcinogen; B2 = probable human carcinogen), the agency has not adopted a current position on the weight-of-evidence classification. In an earlier evaluation, tetrachloroethene 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. The unit risk and slope factor values for tetrachloroethene have been withdrawn from IRIS and HEAST. The upper bound risk estimates from the 1985 Health Assessment Document as amended by inhalation values from the 1987 addendum have not yet been verified by the IRIS-CRAVE Workgroup. For oral exposure, the slope factor is 5.2E-2 $(mg/kg/day)^{-1}$; the unit risk is 1.5E-6 $(\mu g/L)^{-1}$. For inhalation exposure, the slope factor is 2.0E-3 $(mg/kg/day)^{-1}$; the unit risk ranges from 2.9E-7 to 9.5E-7 $(\mu g/m3)^{-1}$ with a geometric mean of 5.8E-7 $(\mu g/m3)^{-1}$. When the Agency makes a decision about weight-of-evidence, the IRIS-CRAVE verification will be completed and the information put on IRIS.

The oral and dermal cancer slope factors used in the BHHRA for tetrachloroethene are 5.20E-2 and 5.2E-2 $[mg/(kg \times day)]^{-1}$, respectively. An inhalation cancer slope factor of 2.00E-3 $[mg/(kg \times day)]^{-1}$ is used. The oral and dermal RfDs used in the BHHRA are 1.00E-2 and 1.00E-2 $mg/(kg \times day)$, respectively. An inhalation RfD of 1.71E-1 $mg/(kg \times day)$ was used. When calculating the dermal route cancer slope factor from the oral value, a gastrointestinal absorption factor of 100% was used.

4.3.40 Trichloroethene (CAS 000079-01-6) (RAIS)

TCE is an industrial solvent used primarily in metal degreasing and cleaning operations. TCE can be absorbed through the lungs, mucous membranes, gastrointestinal tract, and the skin. TCE 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.

Human and animal data indicate that exposure to TCE can result in toxic effects on a number of organs and systems, including the liver, kidney, blood, skin, immune system, reproductive system, nervous system, and cardiovascular system. In humans, acute inhalation exposure to TCE causes central nervous system symptoms such as headache, dizziness, nausea, and unconsciousness. 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. The use of TCE as an anesthetic has been associated with cardiac arrhythmias.

Cases of severe liver and kidney damage, including necrosis, have been reported in humans following acute exposure to TCE, but these effects generally are not associated with long-term occupational exposures. In animals, TCE has produced liver enlargement with hepatic biochemical and/or histological changes and kidney enlargement, renal tubular alterations and/or toxic nephropathy. Also observed in animals were hematological effects and immunosuppression. Inhalation studies with rats indicate that TCE is a developmental toxicant causing skeletal ossification anomalies and other effects consistent with delayed maturation. TCE may cause dermatitis and dermographism.

RfDs and RfCs for subchronic and chronic oral and inhalation exposure to TCE are presently under review by EPA.

Epidemiologic studies have been inadequate to determine if a correlation exists between exposure to TCE and increased cancer risk. Chronic oral exposure to TCE increased the incidences of hepatocellular carcinomas in mice and renal adenocarcinomas and leukemia in rats. Chronic inhalation exposure induced lung and liver tumors in mice and testicular Leydig cell tumors in rats. Although EPA's Science Advisory Board recommended a weight-of-evidence classification of B2, the agency has not adopted a current

position on the weight-of-evidence classification. In an earlier evaluation, TCE 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. 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 $(\mu g/m3)^{-1}$, respectively.

The oral, dermal, and inhalation cancer slope factors used in the BHHRA for trichloroethene are 1.10E-2, 7.33E-2, and 6.00E-3 $[mg/(kg \times day)]^{-1}$, respectively. The oral and dermal RfDs used in the BHHRA are 6.00E-3 and 9.00E-4 mg/(kg × day), respectively. An inhalation RfD was not found for trichloroethene; however, based on the effects discussed previously, an inhalation RfD extrapolated from the oral RfD [6.00E-3 mg/(kg × day)] was used and described in the uncertainty discussion. When calculating both the dermal route cancer slope factor and dermal route RfD from their respective oral values, a gastrointestinal absorption factor of 15% was used.

4.3.41 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 polyvinyl chloride (PVC); limited quantities are used as a refrigerant and as an intermediate in the production of chlorinated compounds.

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.

In humans and animals, vinyl chloride is a CNS depressant, inducing narcosis and anesthesia at high concentrations. 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. The evidence for potential developmental effects in humans (increased fetal loss and birth defects) is equivocal. Occupational exposure to vinyl chloride has been associated with reduced sexual function in both sexes and gynecological effects in women.

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. 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 and degenerative changes of the liver and kidneys at ≥500 ppm. Exposure to higher concentrations caused proliferative changes in the lungs of mice, extensive liver and kidney damage in rats and guinea pigs, cerebral and cerebellar nephrosis in rats, and degeneration of the spleen in guinea pigs. Subchronic exposure of rats to 100 ppm vinyl chloride produced significantly decreased testes weights and testicular regeneration. Evidence of developmental toxicity was seen in rats exposed to vinyl chloride during the first trimester of gestation.

Neither an oral RfD nor an inhalation RfC have been derived for vinyl chloride.

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. 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. Oral administration of vinyl chloride induced liver, lung, and kidney tumors in rodents. Angiosarcomas observed in offspring of rats exposed by inhalation during gestation indicates that vinyl chloride has the potential to initiate cancer in utero.

EPA has classified vinyl chloride as a Group A chemical, human carcinogen. A slope factor of 1.9E+0 $(mg/kg/day)^{-1}$ and a drinking water unit risk of 5.4E-5 $(\mu g/L)^{-1}$ was calculated for oral exposure to vinyl chloride. For inhalation exposure, the slope factor and inhalation unit risk are 3.0E-1 $(mg/kg/day)^{-1}$ and 8.4E-5 $(\mu g/m3)^{-1}$, respectively. The oral slope factor and inhalation unit risk are currently under review and may be subject to change.

An oral slope factor of 1.9E+0 $[mg/(kg \times day)]^{-1}$ was calculated for vinyl chloride. For inhalation exposure, the slope factor is 3.0E-1 $[mg/(kg \times day)]^{-1}$. A gastrointestinal absorption factor of 100% was used to derive an absorbed dose slope factor of 1.90E+0. No RfDs were available.

4.3.42 Xylene (mixture (CAS 001330-20-7), ortho- (CAS 000095-47-6) , meta- (CAS 000108-38-3), para- (CAS 000106-42-3)) (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. Nonlethal levels of xylene vapor may cause eye, nose, and throat irritation, and contact with liquid may result in dermatitis. 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.

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. A chronic oral RfD 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. 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.

Inhalation of 3000 mg/m^3 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. Rib anomalies and cleft palate occurred in mouse fetuses following maternal oral exposure of 2.06 g/kg/day of mixed xylenes on gestation days 6-15. An inhalation RfC is under review by EPA.

Oral and topical carcinogenic studies with xylene in laboratory animals gave negative results. EPA 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.

No cancer slope factors were used in the BHHRA for xylenes. The oral and dermal RfDs used in the BHHRA for mixtures of xylene isomers are 2.00E+0 and 1.84E+0 $mg/(kg \times day)$, respectively. An inhalation RfD equivalent to the oral RfD of 2.00E+0 $mg/(kg \times day)$ was used. When calculating the dermal route RfD from the oral value, a gastrointestinal absorption factor of 92% was used.

4.4 RADIONUCLIDES

4.4.1 Introduction

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 deoxyribonucleic acid (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, all radionuclides are classified by the EPA 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 of concern when there is a potential for inhalation or ingestion of the radionuclide. Alpha particles 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 attributed, in part, 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 and nonstochastic effects. Stochastic effects are those in which the probability of an effect is related to dose, and nonstochastic effects are those in which, above a threshold, the severity of an effect is related to dose. The risk of development of cancer from exposure to radiation is a stochastic effect. Therefore, in this assessment, the risk of developing cancer from exposure to radiation is actually a probability that is related to dose.

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 to 500 rads (0.1 to 5.0 gray, in SI units). Acute radiation sickness is seen only after doses of >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.

4.4.2 Americium-241 (CAS 014596-10-2) (see previous discussion on radionuclides)

Americium was first discovered in 1944 at the Metallurgical Laboratory, the forerunner of Argonne National Laboratory. The isotope is named after America because europium, a similar rare-earth element, was named after the continent of its discovery. Americium-241 is used in high-precision devices and smoke detectors. It decays via alpha-particle emission to neptunium-237.

Few data exist on the distribution of americium in humans, although measurable amounts have been distributed world-wide as part of nuclear weapons testing [International Commission on Radiological Protection (ICRP) 1989]. The limited data gathered from experimental animals suggest that "americium behaves like plutonium with regard to initial partition between liver and skeleton" (ICRP 1989). For dosimetry purposes, all isotopes of americium are assumed "uniformly distributed over bone surface at all times following their deposition to the skeleton" (ICRP 1989).

Oral, inhalation, and external exposure cancer slope factors used in the BHHRA for are 3.28E-10 risk/pCi, 3.85E-08 risk/pCi, and 4.59E-09 (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 americium is not quantified in the BHHRA.

References

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4.4.3 Cesium-137 (CAS 010045-97-3) (see previous discussion on radionuclides)

Cesium occurs in nature as ^{133}Cs in the aluminosilicates, pollucite (a hydrated silicate of aluminum and cesium) and lepidolite; in the borate, rhodizite; and in other sources (Budavari 1989, Klaassen 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 (Bodavari 1989, Klaassen 1986). In addition, ^{137}Cs, along with ^{90}Sr, 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 1989, Casarett 1968). Cesium-137 decays to and reaches radioactive equilibrium with its daughter product, Barium-137m (Budavari 1989, Casarett 1968). Barium-137m is a very short-lived gamma emitter that can contribute to external gamma exposure (Budavari 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.

References

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4.4.4 Cobalt-60 (CAS 010198-40-0)

Cobalt-60 decays with 1.33 and 1.17 MeV gamma rays during a half life of 5.27 years. Cobalt-60 is made by irradiation of cobalt-59 in a nuclear reactor. This nuclide is useful for a thickness gauge of metal, a level gauge, a density gauge, a gamma radiography, and for sterilization purposes.

Cobalt-60m (metastable) has a half-life of 10.467E+6 years and the majority (99.76%) decays with electron capture (IT) at 0.059 MeV, and the remainder with beta emission at 2.883 MeV.

Oral, inhalation, and external exposure cancer slope factors used in the BHHRA for cobalt 60 are 1.89E-11 risk/pCi, 6.88E-11 risk/pCi, and 9.76E-06 [(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 for this element have been presented in the section on inorganic chemicals. However, systemic toxicity due to exposure to the radioactive isotope of cobalt (Co-60) is not quantified in the BHHRA.

4.4.5 Neptunium-237 (CAS 013994-20-2) (see previous discussion on radionuclides)

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 and its short-lived daughter products 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.

References

Moskalev, Y. I., L. A. Buldakov, A. K. Zhuravleva et al. 1979. *Toxicological and Radiobiology of Neptunium-237*, ORNL-tr-4936, Moscow Atomizdat Publishers.

4.4.6 Plutonium-239 (CAS 015117-48-3)

Plutonium is a predominantly man-made radioactive metal that is produced from nuclear reactions with uranium. Plutonium-238 has been used as a nuclear power source for satellites and in thermoelectric generation systems in spacecraft, cardiac pacemakers, and other power sources (Harley 1980, NEA/OECD 1981). Plutonium-239 is mostly associated with nuclear weapons production and testing. It is generated in irradiated uranium fuel when neutrons are captured by uranium-238 nuclei. Commerce and the military principally use plutonium-238 and plutonium-239 because of their ease of production and long radioactive half-lives (86 and 24,000 years, respectively). Both plutonium-238 and plutonium-239 are artificial, alpha-emitting isotopes of plutonium; plutonium-238 decays to radioactive uranium-234 via alphas of 5.5 MeV, and plutonium-239 decays to radioactive uranium-235 via alphas of 5.1 MeV.

Atmospheric testing of nuclear weapons has been the main source of plutonium dispersion in the environment, while accidents and routine releases from weapons production facilities are the primary sources of localized contamination. Plutonium released to the atmosphere reaches the earth's surface through wet and dry deposition to the soil and surface water. Once in these media, plutonium can sorb to soil and sediment particles or bioaccumulate in terrestrial and aquatic food chains.

Because of the low solubility of plutonium isotopes, inhalation of contaminated dust particles is considered to be the most harmful means of human exposure. Plutonium that has been inhaled may be absorbed through the lungs and deposited in other body tissues. Subsequent translocation of some of the plutonium from the lungs to tissues and organs distant from the site of entry results in radiation damage to these tissues as well as to the lung. Liver and bone are the primary sites of plutonium deposition (ICRP 1986). The assumed biological retention half-lives of plutonium isotopes accumulated in the liver and bone of the human body are 20 and 50 years, respectively (ICRP 1986). Therefore, after a single exposure, plutonium isotopes reside in the body for a long time, resulting in prolonged exposure of body organs to alpha radiation (EPA 1977). The permissible health levels for plutonium are the lowest of all the radioactive elements. This is occasioned by the concentration of plutonium directly on bone surfaces rather than the more uniform bone distribution shown by other heavy elements. This increases the possibility of damage from equivalent activities of plutonium and has led to adoption of extremely low permissible levels.

Inhaled plutonium-238 is solubilized and subsequently translocated from the lung to the bone and liver (Gillett et al. 1988). Inhaled plutonium-239 dioxide is insoluble and retained primarily in the lungs and associated lymph nodes. In laboratory tests with plutonium and animals, the pattern of nonmalignant toxicity among the species tested was similar (i.e., radiation pneumonitis and pulmonary fibrosis occurred in the higher radiation dose groups in all species tested); however, species differences in the induction of cancer were apparent. With the exception of Syrian hamsters, cancer developed in animals in the lower exposure groups or in animals that survived initial radiation damage to the lungs (ATSDR 1990).

Oral, inhalation, and external exposure cancer slope factors used in the BHHRA for plutonium-239 are 3.16E-10 risk/pCi, 2.78E-08 risk/pCi, and 1.26E-11 [(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 is not quantified in the BHHRA.

References

ATSDR (Agency for Toxic Substances and Disease Registry). 1990. *Toxicological Profile for Plutonium*, U.S. Department of Health and Human Services, Public Health Service, Atlanta, GA.

EPA (U.S. Environmental Protection Agency). 1977. *Proposed Guidance on Dose Limits for Persons Exposed to Transuranium Elements in the General Environment*, EPA520/4-77-016, Washington, D.C.

Gillet, N., B. Muggenberg, J. Mewhinney, et al. 1988. Primary Liver Tumors in Beagle Dogs Exposed by Inhalation to Aerosols of Plutonium-238 Dioxide, *American Journal of Pathology* 133:265–276.

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ICRP (International Commission on Radiological Protection). 1986. *The Metabolism of Plutonium and Related Elements*, ICRP Publication 48, Pergamon Press, Oxford.

NEA/OECD (Nuclear Energy Agency/Organization of Economic Cooperation and Development. 1981. *The Environmental and Biological Behavior of Plutonium and Some Other Transuranium Elements*, Nuclear Energy Agency, Paris.

4.4.7 Radium-226 (CAS 013982-63-3)

Radium is an alkaline earth metal and was extensively used in the past as an ingredient of luminescent paints for instrument dials, watches, and similar self-illuminating instrumentation. It also occurs naturally as a daughter product of the naturally occurring radioactive material (NORM) uranium decay chain.

Pure metallic radium is brilliant white when freshly prepared, but blackens on exposure to air, probably due to formation of the nitride. It exhibits luminescence, as do its salts, and it decomposes in water and is somewhat more volatile than barium. Radium imparts a carmine red color to a flame. Radium is extremely scarce but found in uranium ores such as pitchblende at slightly more than 1g in 10 tons of ore. It may be made on a very small scale by the electrolysis of molten radium chloride, RaCl2. This was first done using a mercury cathode, which gave radium amalgam. The metal was obtained by distillation away from the amalgam.

All isotopes of radium are radioactive. Radium emits alpha, beta, and gamma rays and when mixed with beryllium produces neutrons. Inhalation, injection, or body exposure to radium can cause cancer and other body disorders. Radium is over a million times more radioactive than the same mass of uranium.

Oral, inhalation, and external exposure cancer slope factors used in the BHHRA for radium-226 and its short-lived daughter products are 2.96E-10 risk/pCi, 2.75E-9 risk/pCi, and 6.74E-06 [(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 is not quantified in the BHHRA.

4.4.8 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 which 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% 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 × g)/(pCi × 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.

4.4.9 Technetium-99 (CAS 014133-76-7) (see previous discussion on radionuclides)

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 are stable, the existence of three technetium isotopes is well established. Two common forms of technetium, ^{97}Tc and ^{98}Tc, have half-lives of 2.6×10^6 and 1.5×10^6 years, respectively. The third isotope, ^{99}Tc, has a half-life of 2.12×10^5 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 1978, 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 applications 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 ^{99}Tc, the secondary X-rays may become important with larger amounts of the element.

Oral, inhalation, and external exposure cancer slope factors used in the BHHRA for ^{99}Tc are 1.40E-12 risk/pCi, 2.89E-12 risk/pCi, and 6.19E-13 [(risk × g)/(pCi × yr)], respectively. A dermal cancer slope factor was not calculated because this route of exposure is not evaluated in the BHHRA. Oral, dermal, and inhalation RfDs are not available for this element; therefore, systemic toxicity due to exposure to technetium-99 is not quantified in the BHHRA.

References

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4.4.10 Thorium-228 (CAS 014274-82-9) and Thorium-230 (CAS 014269-63-7) (see previous discussion on radionuclides)

Thorium is a naturally occurring, radioactive metal. Small amounts of thorium are present in all rocks, soil, above-ground 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 1990).

Thorium is a metallic element of the actinide series. It exists in several isotopic forms. The isotope ^{232}Th 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, ^{208}Pb. Isotopes ^{234}Th and ^{230}Th are produced during the decay of naturally occurring ^{238}U, the isotope ^{228}Th during the decay of ^{232}Th, and the isotopes ^{231}Th and ^{227}Th during the decay of ^{235}U. Of these naturally produced isotopes of thorium, only ^{232}Th, ^{230}Th, and ^{228}Th have long enough half-lives to be environmentally significant. More than 99.99% of natural thorium is ^{232}Th; the rest is ^{230}Th and ^{228}Th (ATSDR 1990).

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 1990).

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 the fish or seafood used commercially. More thorium may be found near uncontrolled hazardous waste sites that contain thorium which 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 1990).

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. Since 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 in the environment could result in exposure to more hazardous radioactive decay products 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 1990).

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 × g)/(pCi × 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 × g)/(pCi × 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 × 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 thorium is not quantified in the BHHRA.

References

ATSDR (Agency for Toxic Substances and Disease Registry). 1990. *Toxicological Profile for Thorium*, TP-90-25, United States Department of Health and Human Services, Public Health Service, Atlanta, GA.

4.4.11 Uranium (CAS 7440-62-2 for metal, CAS 013966-29-5 for U-234, CAS 15117-96-1 for U-235, and CAS 07440-61-1 for U-238) (see previous discussion on radionuclides)

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 1989), 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 ^{238}U (which averages 99.27% of total uranium mass), ^{235}U (0.72%), and ^{234}U (0.0056%), each of which undergoes radioactive decay. Natural uranium, therefore, contains the radionuclide daughter products from the decay of ^{238}U and ^{235}U (Bowen 1979, ATSDR 1989).

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 in addition to 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 × g)/(pCi × yr)], respectively. Oral, inhalation, and external exposure cancer slope factors used in the BHHRA for uranium-235 and its short-lived daughter products are 4.70E-11 risk/pCi, 1.30E-08 risk/pCi, and 2.65E-07 [(risk × g)/(pCi × 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 and its short-lived daughter products are 6.20E-11 risk/pCi, 1.24E-08 risk/pCi, and 6.57E-08 [(risk × g)/(pCi × 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.

References

Agency for Toxic Substances and Disease Registry (ATSDR). 1989. *Draft Toxicological Profile for Uranium and Compounds*. Prepared by Syracuse Research Corporation. Prepared for ATSDR.

Bowen, H.J.M. 1979. *Environmental Chemistry of the Elements*. Academic Press: London.

4.5 CHEMICALS FOR WHICH NO EPA TOXICITY VALUES ARE AVAILABLE

Among the inorganic COPCs included in the GWOU BHHRA, oral RfD values do not exist for the following chemicals: ammonia as nitrogen, bicarbonate, bromide, cerium, gallium, Kjeldahl (total) nitrogen, orthophosphate, silica, sulfate, sulfide, tetraoxo-sulfate (1-), thallium, thorium, titanium, and zirconium.

Oral RfDs exist for all of the organic COPCs included in the GWOU BHHRA except 1,2-dichloroethane, *trans*-1,3-dichloropropene, 1,4-dichlorobenzene, 4-bromofluorobenzene, benzene, chloromethane, chrysene, ethane, ethanol, ethylene, phenanthrene, PCB, and vinyl chloride. It should be noted that the reference dose for lead is not approved by the EPA. EPA currently recommends a lead uptake/biokinetic model to provide an alternative measure for lead. Results of this model are discussed in Sect. 5 and presented in Attachment 5.

The majority of the inorganic COPCs, with the exception of ammonia as nitrogen, barium, beryllium, boron, cadmium, chromium, cobalt, lead, manganese, and mercury, lack inhalation RfD values. In addition, *trans*-1,3-dichloropropene, 4-bromofluorobenzene, chloromethane, chrysene, ethane, ethanol, ethylene, phenanthrene, PCB, and vinyl chloride of the organic COPCs, do not have inhalation RfD values. 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 GWOU BHHRA except ammonia as nitrogen, bicarbonate, bromide, cerium, gallium, Kjeldahl (total) nitrogen, orthophosphate, silica, sulfate, sulfide, tetraoxo-sulfate (1-), thallium, thorium, titanium, and zirconium. Absorbed dose RfDs exist for all of the organic COPCs included in the GWOU BHHRA except *trans*-1,3-dichloropropene, 4-bromofluorobenzene, chloromethane, chrysene, ethane, ethanol, ethylene, phenanthrene, PCB, and vinyl chloride.

Oral slope factors for inorganic compounds are only available for arsenic and beryllium. Oral slope factors do not currently exist for 43 of the 45 inorganic COPCs included in this assessment.

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, acrylonitrile, Aroclor 1254, benzene, carbon tetrachloride, chloroform, chloromethane, chrysene, methylene chloride, PCB, tetrachloroethene, trichloroethene, and vinyl chloride.

Twenty-four COPCs have absorbed dose slope factors: 2 are inorganics (arsenic and beryllium) and 22 are organic compounds (these are identical to those analytes having oral slope factors). All fifteen radionuclide COPCs have oral, inhalation, and external exposure slope factors.

4.6 UNCERTAINTIES RELATED TO TOXICITY INFORMATION

When available, standard EPA RfDs and slope factors were used to estimate potential noncarcinogenic and carcinogenic health effects from exposure to chemical contaminants detected in the GWOU. Considerable uncertainty is associated with the basic EPA 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 re-evaluated.

4.7 SUMMARY OF TOXICITY ASSESSMENT

A breakdown of the COPCs and their available toxicity information by area sector and the GWOU area as a whole is provided in the following subsections. This summary is also presented in part in Table 2.11. In that table, chemicals and compounds marked with an asterisk lack toxicity information.

4.7.1 Area a

RGA groundwater at GWOU Area a contains 22 COPCs. Seven are organic compounds of which all have toxicity information; 11 are inorganic chemicals of which 2 have no toxicity information; and 4 are radionuclides of which all have toxicity information.

UCRS groundwater at GWOU Area a contains 25 COPCs. Six are organic compounds all of which have toxicity information; 16 are inorganic chemicals of which 2 have no toxicity information; and 3 are radionuclides of which all have toxicity information.

4.7.2 Area b

McNairy groundwater at GWOU Area b contains 7 COPCs. One is an organic compound which has toxicity information; 5 are inorganic chemicals of which 2 have no toxicity information; and 1 is a radionuclide which has toxicity information.

RGA groundwater at GWOU Area b contains 48 COPCs. Fifteen are organic compounds of which 2 have no toxicity information; 21 are inorganic chemicals of which 3 have no toxicity information; and 12 are radionuclides of which all have toxicity information.

4.7.3 Area c

RGA groundwater at GWOU Area c contains 16 COPCs. Four are organic compounds of which all have toxicity information; 10 are inorganic chemicals of which 3 have no toxicity information; and 2 are radionuclides of which all have toxicity information.

UCRS groundwater at GWOU Area c contains 12 COPCs. Three are organic compounds of which all have toxicity information; 8 are inorganic chemicals of which 2 have no toxicity information; and 1 is a radionuclide which has toxicity information.

4.7.4 Area d

McNairy Formation groundwater at GWOU Area d contains 5 COPCs. One is an organic compound which has toxicity information. 4 are inorganic chemicals of which 3 have no toxicity information.

RGA groundwater at GWOU Area d contains 33 COPCs. Nine are organic compounds of which all have toxicity information; 15 are inorganic chemicals of which 2 have no toxicity information; and 9 are radionuclides of which all have toxicity information.

UCRS groundwater at GWOU Area d contains 47 COPCs. Twelve are organic compounds of which 1 has no toxicity information; 28 are inorganic chemicals of which 6 have no toxicity information; and 7 are radionuclides of which all have toxicity information.

4.7.5 Area e

McNairy Formation groundwater at GWOU Area e contains 20 COPCs. One is an organic compound which has toxicity information; 17 are inorganic chemicals of which 3 have no toxicity information; and 2 are radionuclides which have toxicity information.

RGA groundwater at GWOU Area e contains 27 COPCs. Four are organic compounds of which all have toxicity information; 19 are inorganic chemicals of which 4 have no toxicity information; and 4 are radionuclides of which all have toxicity information.

UCRS groundwater at GWOU Area e contains 15 COPCs. One is an organic compound which has toxicity information; 13 are inorganic chemicals of which 3 have no toxicity information; and 1 is a radionuclide which has toxicity information.

4.7.6 Area f

McNairy groundwater at GWOU Area f contains 3 COPCs. All are inorganic chemicals of which 1 has no toxicity information.

RGA groundwater at GWOU Area f contains 22 COPCs. Six are organic compounds of which all have toxicity information; 13 are inorganic chemicals of which 3 have no toxicity information; and 3 are radionuclides of which all have toxicity information.

UCRS groundwater at GWOU Area f contains 11 COPCs. One is an organic compound which has toxicity information; 8 are inorganic chemicals of which 2 have no toxicity information; and 2 are radionuclides of which all have toxicity information.

4.7.7 Area g

McNairy groundwater at GWOU Area g contains 7 COPCs. Four are inorganic chemicals of which 2 have no toxicity information; and 3 are radionuclides of which all have toxicity information.

RGA groundwater at GWOU Area g contains 17 COPCs. One is an organic compound which has toxicity information; 11 are inorganic chemicals of which 2 have no toxicity information; and 5 are radionuclides of which all have toxicity information.

UCRS groundwater at GWOU Area g contains 13 COPCs. Eight are inorganic chemicals of which 2 have no toxicity information; and 5 are radionuclides of which all have toxicity information.

4.7.8 Area h

McNairy groundwater at GWOU Area h contains 6 COPCs. Three are inorganic chemicals of which 2 have no toxicity information; and 3 are radionuclides of which all have toxicity information.

RGA groundwater at GWOU Area h contains 14 COPCs. Two are organic compounds of which all have toxicity information; 10 are inorganic chemicals of which 1 has no toxicity information; and 2 are radionuclides of which all have toxicity information.

UCRS groundwater at GWOU Area h contains 11 COPCs. Ten are inorganic chemicals of which 2 have no toxicity information; and 1 is a radionuclide which has toxicity information.

4.7.9 Area i

McNairy groundwater at GWOU Area i contains 4 COPCs. All 4 are inorganic chemicals of which 2 have no toxicity information.

RGA groundwater at GWOU Area i contains 63 COPCs. Twenty-seven are organic compounds of which 3 have no toxicity information; 30 are inorganic chemicals of which 9 have no toxicity information; and 6 are radionuclides of which all have toxicity information.

UCRS groundwater at GWOU Area i contains 34 COPCs. Seven are organic compounds of which 1 has no toxicity information; 22 are inorganic chemicals of which 4 have no toxicity information; and 5 are radionuclides of which all have toxicity information.

4.7.10 Area j

McNairy groundwater at GWOU Area j contains 5 COPCs. All 4 are inorganic chemicals of which 1 have no toxicity information.

RGA groundwater at GWOU Area j contains 9 COPCs. All 9 are inorganic chemicals of which 3 have no toxicity information.

4.7.11 Area k

Area k does not lie above either the UCRS or RGA. All samples used for this BHHRA were drawn from the Eocene Sands, Terrace Gravel, or Porters Creek Clay. These data contain 45 COPCs. 11 are organic compounds of which 1 has no toxicity information; 26 are inorganic chemicals of which 5 have no toxicity information; and, 8 are radionuclides of which all have toxicity information.

4.7.12 Area l

Data for Area l were developed by combining the data sets for Area a, b, c, and d (i.e., all areas inside the security fence at the PGDP. Therefore, the summary of the toxicity information is a combination of the results discussed above.

McNairy groundwater at GWOU Area l contains 9 COPCs. One is an organic compound which has toxicity information; 7 are inorganic chemicals of which 3 have no toxicity information; and 1 is a radionuclide which has toxicity information.

RGA groundwater at GWOU Area m contains 56 COPCs. Twenty are organic compounds of which 2 have no toxicity information; 23 are inorganic chemicals of which 4 have no toxicity information; and 13 are radionuclides of which all have toxicity information.

UCRS groundwater at GWOU Area l contains 65 COPCs. Twenty-two are organic compounds of which 3 have no toxicity information; 31 are inorganic chemicals of which 6 have no toxicity information; and 12 are radionuclides of which all have toxicity information.

4.7.13 Area m

Data for Area m were developed by combining the data sets for Area e, f, g, h, i, and j (i.e., all areas outside the security fence at the PGDP except Area k). Therefore, the summary of the toxicity information is a combination of the results discussed above.

McNairy groundwater at GWOU Area m contains 26 COPCs. One is an organic compound which has toxicity information; 19 are inorganic chemicals of which 3 have no toxicity information; and 6 are radionuclides which have toxicity information.

RGA groundwater at GWOU Area m contains 75 COPCs. Thirty-two are organic compounds of which 3 have no toxicity information; 34 are inorganic chemicals of which 10 have no toxicity information; and 9 are radionuclides of which all have toxicity information.

UCRS groundwater at GWOU Area m contains 37 COPCs. Seven are organic compounds of which 1 has no toxicity information; 23 are inorganic chemicals of which 4 have no toxicity information; and 7 are radionuclides of which all have toxicity information.

4.7.14 Area n

Data for Area n were developed by combining all the groundwater data sets within there appropriate depth class. Therefore, the summary of the toxicity information is a combination of the results discussed above.

McNairy groundwater at GWOU Area n contains 29 COPCs. One is an organic compound which has toxicity information; 22 are inorganic chemicals of which 4 have no toxicity information; and 6 are radionuclides which have toxicity information.

RGA groundwater at GWOU Area m contains 86 COPCs. Thirty-eight are organic compounds of which 5 have no toxicity information; 35 are inorganic chemicals of which 10 have no toxicity information; and 13 are radionuclides of which all have toxicity information.

UCRS groundwater at GWOU Area m contains 71 COPCs. Twenty-five are organic compounds of which 4 have no toxicity information; 33 are inorganic chemicals of which 6 have no toxicity information; and 13 are radionuclides of which all have toxicity information.

5. RISK CHARACTERIZATION

5.1 INTRODUCTION

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 (1) the lifetime probability of excess cancer incidence for carcinogens and (2) the estimate of daily intake exceeding intake that may lead to toxic effects for noncarcinogens.

5.2 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 chronic daily intake 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 is the hazard quotient, dimensionless,
CDI is the chronic daily intake of a particular chemical, mg/(kg × day),
RfD is the chronic reference dose for a particular chemical and pathway, mg/(kg × day).

Care was taken when performing this calculation to ensure that the proper RfD was used for each chronic daily intake. For chronic daily intakes that reflect ingestion, the RfD used was that for administered dose. For chronic daily intakes that reflect absorption, as in dermal contact, the RfD used was that for absorbed dose. Finally, for chronic daily intakes 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 7 years; therefore, the chronic RfD was used. For all exposures to children, regardless of duration, the chronic RfD was used (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 exposure route (or pathway), guidance (RAGS, Methods Document) recommends adding the HQs of all chemicals reaching the receptor through the common pathway to calculate a hazard index (HI). This can be represented by the following equation:

$$\text{Pathway HI} = HQ_1 + HQ_2 + HQ_3 + \ldots + HQ_n ,$$

where:

Pathway HI is the sum of the individual chemical HQs, dimensionless,
HQ_1 to HQ_n are the individual chemical hazard quotients 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 or cumulative HI. The total HI is not an estimate of the systemic toxicity posed by all contaminants that may reach the receptor but can be used to

estimate if a toxic effect may result if all contaminants reaching the receptor have additive effects over all pathways. This can be represented as in the following equation:

$$\text{Total HI} = HI_1 + HI_2 + HI_3 + \ldots + HI_n ,$$

where:

Total HI is the sum of all pathways relevant to a single receptor, dimensionless,
HI_1 to HI_n are the individual pathway HIs.

Note that 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 the assumptions used in deriving the RfD. Similarly, if the pathway HI is less than 1, then 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, then 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, then 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, then 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 over pathways, the risk was further evaluated if the sum was greater than 1. In this evaluation, chemicals with similar effects were segregated to determine if the HQs of these chemicals also summed to a value greater than 1. This evaluation was performed because the belief is that (RAGS) if the sum of the HQs of chemicals with common effects is greater than 1, then there is greater confidence in stating that exposure to several chemicals within a pathway or over 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. 6.

5.3 DETERMINATION OF EXCESS LIFETIME CANCER RISK

Estimates of the potential for cancer induction are measured by calculating estimates of ELCR. Generally, ELCR can be 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. Remember that 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 could vary markedly if the exposure assumptions used to develop the conceptual site model are varied.

5.3.1 Chemical Excess Cancer Risk

The numeric estimate of the ELCR resulting from exposure to a single chemical carcinogen is derived by multiplying the chronic daily intake (CDI) through 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:

$$\text{Chemical-specific ELCR} = \text{CDI} \times \text{SF} ,$$

where:

Chemical specific ELCR is an estimate of the excess lifetime probability of developing cancer which results because of exposure to the specific chemical, dimensionless,

CDI is the chronic daily intake of the chemical [mg/(kg × day)],
SF is the slope factor for the specific chemical $[(mg/(kg \times day)]^{-1}$.

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 can be represented by the following equation:

$$\text{Pathway ELCR} = ELCR_1 + ELCR_2 + ELCR_3 + \ldots + ELCR_n ,$$

where:

Pathway ELCR is the sum of the chemical-specific ELCRs, dimensionless,
$ELCR_1$ to $ELCR_n$ are 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 can be used to estimate the total ELCR that may result if all contaminants reaching the receptor have additive effects over all pathways. This can be represented as in the following equation:

$$\text{Total ELCR} = ELCR_{P1} + ELCR_{P2} + ELCR_{P3} + \ldots + ELCR_{Pn} ,$$

where:

Total ELCR is the sum of all pathways relevant to a single receptor, dimensionless,
$ELCR_{P1}$ to $ELCR_{P2}$ is the individual pathway ELCRs.

Unlike the HQ, the pathway HI, and the total HI, the chemical-specific ELCR, the pathway ELCR, and total ELCR define a dose-response relationship. That is, the ELCRs do represent a statistical probability of the increased risk of developing cancer that exists 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 1×10^{-4}. If the total ELCR exceeds 1×10^{-4}, then chemicals contributing to the ELCR should be segregated by common effect. This analysis is performed to decrease the uncertainty in the risk presentation and raise 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. 6.

5.3.2 Radionuclide Excess Cancer Risk

Calculation of cancer risk from exposure to radionuclides is conceptually similar to calculation of risks for chemical carcinogens. In performing this calculation, ELCR from exposure to a particular radionuclide within a specific pathway is calculated by multiplying the intake of the radionuclide by the route-specific cancer slope factor. This can be represented by the following equation:

$$ELCR = CDI \times SF ,$$

where:

Radionuclide specific ELCR is an estimate of the excess lifetime probability of developing cancer which results from exposure to the specific radionuclide, dimensionless,

CDI is the ingestion and inhalation chronic daily intake of the radionuclide, pCi,
SF is 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 which 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 over multiple pathways, respectively, are calculated as illustrated for chemical carcinogens in Subsect. 5.2. These equations will not be presented here. The uncertainties related to this method of determining ELCR from exposure to radionuclides is discussed in detail in Sect. 6.

In this risk assessment, ELCRs from exposure to chemicals and radionuclides were summed within pathways and over 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. 6.

5.4 RISK CHARACTERIZATION FOR CURRENT CONCENTRATIONS BY AREA

This subsection presents the risk for each land use for each area. In previous BHHRAs for the PGDP, the current and future land use discussions were separated because the assessments focused on specific study areas (i.e., individual solid waste management units or areas of concern). However, this BHHRA presents the risk characterization for each land use without the designation of current or future because the areas assessed are very large and because multiple land uses within several of the areas are possible. Exhibits and discussion in this subsection provide the total HI or ELCR for each area for the unfiltered data and list the major exposure routes and constituents contributing to the total HI or ELCR. The risk results presented in this section focus primarily on the direct contact pathways because it was determined that the biota pathways added little to the assessment. Additionally, the hazard results focus on the child resident because previous risk assessments for the PGDP have indicated that this receptor is more sensitive to environmental contamination than the adult resident. This subsection does not select either land use scenarios of concern, pathways of concern, or COCs. The selection of land use scenarios of concern, pathways of concern, and COCs is in Subsects. 5.7.1, 5.7.2, and 5.7.3, respectively.

The information summarized in the exhibits and discussion in this subsection is presented in full in Tables 5.1 to 5.9b. Exhibit 5.1 summarizes the contents of each of these tables. 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 for the area or sampling station. The program used to calculate the risk values is Program 10 described in Attachment 3.

Exhibit 5.1. Table of contents for GWOU BHHRA risk tables

Table Number	Land use	Cohort	Risk Category	Routes
Table 5.1	Industrial	Adult	Systemic Toxicity (HI)	Direct Contact
Table 5.2	Industrial	Adult	Cancer Risk (ELCR)	Direct Contact
Table 5.3a	Recreational	Child	Systemic Toxicity (HI)	Direct Contact
Table 5.3b	Recreational	Child	Systemic Toxicity (HI)	Biota
Table 5.4a	Recreational	Teen	Systemic Toxicity (HI)	Direct Contact
Table 5.4b	Recreational	Teen	Systemic Toxicity (HI)	Biota
Table 5.5a	Recreational	Adult	Systemic Toxicity (HI)	Direct Contact
Table 5.5b	Recreational	Adult	Systemic Toxicity (HI)	Biota
Table 5.6a	Recreational	All	Cancer Risk (ELCR)	Direct Contact
Table 5.6b	Recreational	All	Cancer Risk (ELCR)	Biota
Table 5.7a	Residential	Child	Systemic Toxicity (HI)	Direct Contact
Table 5.7b	Residential	Child	Systemic Toxicity (HI)	Biota
Table 5.8a	Residential	Adult	Systemic Toxicity (HI)	Direct Contact
Table 5.8b	Residential	Adult	Systemic Toxicity (HI)	Biota
Table 5.9a	Residential	All	Cancer Risk (ELCR)	Direct Contact
Table 5.9b	Residential	All	Cancer Risk (ELCR)	Biota

Note: Excess lifetime cancer risk (ELCR) calculations consider a 40-year lifetime exposure.

5.4.1 Industrial Worker

5.4.1.1 Systemic toxicity

Exhibit 5.2 summarizes the HIs for direct contact exposure routes for the industrial worker over all areas. As shown in this exhibit, the total scenario hazard index (i.e., Location Total without lead in Exhibit 5.2) is greater than 1 for Areas b, e, j, l, m, and n for the McNairy Groundwater Formation; for Areas a, b, c, d, e, f, i, j, l, m, and n for the RGA; and Areas a, b, d, e, i, l, m, and n for the UCRS. This value is also greater than 1 for Area k. This exhibit also shows that the driving exposure route for systemic toxicity for the industrial worker across all areas and depth classifications is ingestion of groundwater. However, both dermal contact and inhalation contribute a marked portion of the total HI for some areas.

Exhibit 5.3 summarizes the contaminants contributing more than 10% of the total systemic toxicity for the industrial worker for direct contact pathways for those areas where the total systemic toxicity for the area exceeds 1 without lead considered as a COPC. As shown in this exhibit, TCE and its breakdown products are the driving contaminants for all areas inside the security fence at the PGDP (Areas a, b, c, and d). However, outside the security fence (Areas e through k), TCE is a driving contaminant only in areas delimited by the Northeast and Northwest Plumes (Areas e and f) and then only for samples from the RGA. For other areas outside the security fence, the driving contaminants are various inorganic chemicals, with vanadium, chromium, antimony, iron, manganese, and cadmium appearing most often. Additionally, the polychlorinated biphenyl, Aroclor-1254, appears as a driving contaminant for Area i in the RGA.

Exhibit 5.2. Direct contact exposure route summary for the industrial worker - systemic toxicity[1]

Location	Direct Ingestion of Groundwater	Dermal Contact while showering	Inhalation of vapors while showering	Location Total without lead	Location Total with lead[2]
Area a UCRS % of Total	136 52%	52.6 20%	74.0 28%	**264**	**7,180**
Area a RGA % of Total	753 52%	291 20%	411 28%	**1,460**	**1,460**
Area a McN % of Total	NA NA	NA NA	NA NA	**NA**	**NA**
Area b UCRS % of Total	48.9 53%	18.2 20%	26.0 28%	**93.1**	**273**
Area b RGA % of Total	6.0 55%	2.1 19%	2.7 25%	**10.9**	**4,150**
Area b McN % of Total	3.6 72%	0.9 17%	0.5 11%	**5.0**	**5.0**
Area c UCRS % of Total	0.2 83%	<0.1 12%	<0.1 6%	**0.2**	**0.2**
Area c RGA % of Total	1.8 69%	0.47 16%	0.4 15%	**2.6**	**2.6**
Area c McN % of Total	NA NA	NA NA	NA NA	**NA**	**NA**
Area d UCRS % of Total	17.8 67%	3.8 14%	4.5 17%	**26.6**	**3,490**
Area d RGA % of Total	2.2 61%	0.6 17%	0.8 22%	**3.6**	**6,750**
Area d McN % of Total	<0.1 73%	<0.1 12%	<0.1 15%	**<0.1**	**<0.1**
Area e UCRS % of Total	1.3 83%	0.3 17%	<0.1 <1%	**1.6**	**1.6**
Area e RGA % of Total	3.1 58%	1.1 19%	1.3 23%	**5.4**	**5.4**
Area e McN % of Total	2.7 84%	0.5 16%	<0.1 <1%	**3.2**	**3.2**
Area f UCRS % of Total	0.3 90%	<0.1 9%	<0.1 <1%	**0.3**	**0.3**
Area f RGA % of Total	2.4 62%	0.8 20%	0.7 18%	**3.9**	**3.9**
Area f McN % of Total	<0.1 96%	<0.1 5%	<0.1 <1%	**<0.1**	**<0.1**
Area g UCRS % of Total	0.6 84%	0.1 17%	<0.1 <1%	**0.7**	**0.7**
Area g RGA % of Total	0.8 86%	0.1 15%	<0.1 <1%	**0.9**	**6,700**
Area g McN % of Total	0.1 98%	<0.1 2%	<0.1 <1%	**0.1**	**0.1**
Area h UCRS % of Total	0.3 92%	<0.1 8%	<0.1 <1%	**0.3**	**0.3**
Area h RGA % of Total	1.0 88%	0.1 11%	<0.1 <1%	**1.1**	**1.1**
Area h McN % of Total	<0.1 100%	<0.1 <1%	<0.1 <1%	**<0.1**	**<0.1**

Exhibit 5.2 (continued)

Location	Direct Ingestion of Groundwater	Dermal Contact while showering	Inhalation of vapors while showering	Location Total without lead	Location Total with lead[2]
Area i UCRS % of Total	2.1 88%	0.3 13%	<0.1 <1%	**2.4**	**5,750**
Area i RGA % of Total	3.9 79%	0.9 19%	0.1 2%	**4.9**	**4.9**
Area i McN % of Total	0.4 79%	0.1 21%	<0.1 <1%	**0.5**	**0.5**
Area j UCRS % of Total	NA NA	NA NA	NA NA	**NA**	**NA**
Area j RGA % of Total	1.2 92%	0.1 8%	<0.1 <1%	**1.3**	**1.3**
Area j McN % of Total	4.1 98%	<0.1 2%	<0.1 <1%	**4.2**	**4.2**
Area k Terrace [3] % of Total	10.2 89%	0.8 7%	0.3 3%	**11.4**	**15,400**
Area l UCRS % of Total	60.9 53%	21.4 19%	32.7 28%	**115**	**4,780**
Area l RGA % of Total	29.4 54%	9.7 18%	15.5 28%	**54.6**	**5,010**
Area l McN % of Total	3.0 74%	0.7 17%	0.4 10%	**4.1**	**4.1**
Area m UCRS % of Total	2.8 86%	0.5 14%	<0.1 <1%	**3.3**	**5,160**
Area m RGA % of Total	4.0 68%	1.2 20%	0.7 11%	**5.9**	**5,110**
Area m McN % of Total	2.3 88%	0.3 12%	<0.1 <1%	**2.7**	**2.7**
Area n UCRS % of Total	48.1 54%	16.4 18%	24.9 28%	**89.4**	**4,920**
Area n RGA % of Total	18.4 56%	5.8 18%	8.4 26%	**32.6**	**4,980**
Area n McN % of Total	3.7 81%	0.7 16%	0.1 3%	**4.5**	**4.5**

Notes: NA indicates that there were no data for the pathway or area.

[1] Current convention is to use one significant digit for presentation of hazard indices. Three significant digits are used here when the hazard index 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's value is greater than 0.1, allows the reader to sum the route values and check the location total.

[2] The very large values are the result of the retention of lead as a COPC at a value only slightly greater than the background concentration and the use of a provisional reference dose provided in comments by KDEP.

[3] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porter's Creek Clay only.

Exhibit 5.3. Driving contaminants' summary for direct contact exposure routes for the industrial worker scenario - systemic toxicity

Location	Driving Contaminants Over All Exposure Routes	Location Total[1]
Area a UCRS	TCE (97%)	264
Area a RGA	TCE (100%)	1,460
Area a McN	NA	NA
Area b UCRS	TCE (97%)	93.1
Area b RGA	TCE (73%); *cis*-1,2-DCE (10%)	10.9
Area b McN	TCE (62%); Antimony (38%)	5
Area c UCRS	HI<1	0.2
Area c RGA	TCE (51%); Chromium (31%)	2.6
Area c McN	NA	NA
Area d UCRS	TCE (60%); Manganese (24%); Iron (10%)	26.6
Area d RGA	TCE (76%);	3.6
Area d McN	HI<1	<0.1
Area e UCRS	Vanadium (55%); Chromium (11%)	1.6
Area e RGA	TCE (81%)	5.4
Area e McN	Vanadium (27%); Cadmium (17%); Iron (16%); Chromium (15%); Arsenic (14%)	3.2
Area f UCRS	HI<1	0.3
Area f RGA	TCE (61%); Cadmium (20%)	3.9
Area f McN	HI<1	<0.1
Area g UCRS	HI<1	0.7
Area g RGA	HI<1	0.9
Area g McN	HI<1	0.1
Area h UCRS	HI<1	0.3
Area h RGA	Chromium (48%); Iron (22%); Vanadium (12%)	1.1
Area h McN	HI<1	<0.1
Area i UCRS	Vanadium (24%); Antimony (17%); Manganese (13%)	2.4
Area i RGA	Antimony (49%); Chromium (21%); Aroclor-1254 (10%)	4.9
Area i McN	HI<1	0.5
Area j UCRS	NA	NA
Area j RGA	Manganese (42%); Molybdenum (19%); Vanadium (14%); Iron (12%); Arsenic (11%)	1.3
Area j McN	Arsenic (68%); Manganese (17%); Molybdenum (15%)	4.2
Area k Terrace[2]	Iron (49%); Manganese (25%); Antimony (14%)	11.4
Area l UCRS	TCE (94%)	115
Area l RGA	TCE (81%)	54.6
Area l McN	Antimony (66%); TCE (34%)	4.1
Area m UCRS	Antimony (50%); Vanadium (13%)	3.3
Area m RGA	Antimony (39%); TCE (31%); Chromium (12%)	5.9
Area m McN	Iron (38%); Cadmium (19%); Chromium (12%); Vanadium (11%)	2.7
Area n UCRS	TCE (90%)	89.4
Area n RGA	TCE (65%); Carbon tetrachloride (12%)	32.6
Area n McN	Antimony (52%); TCE (12%); Cadmium (11%)	4.5

Notes NA indicates that there were no data for that route or area.
HI<1 indicates that total scenario hazard index is less than 1; therefore, COCs are not listed.
COCs contributing more than 10% of total HI are listed. Percentages rounded to nearest whole number.
[1] Totals are without lead as a COPC. The total HIs with lead are in Exhibit 5.2.
[2] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porter's Creek Clay only.

5.4.1.2 Excess lifetime cancer risk

Exhibit 5.4 summarizes the excess cancer risks for direct contact exposure routes for the industrial worker over all areas for the unfiltered data set. As shown in this exhibit, the total ELCR is greater than 1×10^{-6} for all areas except Areas a, c, d, f, and i for the McNairy Formation groundwater and Area j for UCRS groundwater; however, total ELCR is less than 1×10^{-6} for Areas a and c for the McNairy Formation groundwater and Area j for the UCRS because data were not available to assess these areas.

Unlike systemic toxicity where the primary driving exposure route across all areas was ingestion of groundwater, this exposure route was replaced by inhalation of vapors emitted by groundwater while showering in several areas. Generally, the dermal contact exposure route posed only a small portion of the total ELCR compared to the other two exposure routes.

Exhibit 5.5 summarizes the contaminants contributing more than 10% of the total ELCR for the industrial worker over all areas. As shown in this exhibit, the driving contaminants for areas encompassed by the Northeast and Northwest Plumes inside the security fence (Areas a and b) are TCE and its breakdown products. However, in Areas c and d both chloroform and ^{222}Rn are more important than TCE and its breakdown products, and in Areas e and f, the inorganic chemicals, arsenic and beryllium, and ^{222}Rn are of greater importance than TCE. Outside the TCE plumes, except for Area k, ^{222}Rn and the inorganic chemicals, arsenic and beryllium, drive ELCR. In Area k., ^{222}Rn and the TCE breakdown products, 1,1-dichloroethene and vinyl chloride, drive ELCR.

5.4.2 Recreational user

5.4.2.1 Systemic Toxicity

Exhibit 5.6 summarizes the HIs for direct contact exposure routes for the child recreational user over all areas. As shown in this exhibit, the total scenario HI (i.e., Location Total without lead in Exhibit 5.6) is greater than 1 for Areas b, e, i, j, l, m, and n for the McNairy Formation; all areas of the RGA; and Areas a, b, d, e, g, i, l, m, and n for the UCRS. Total HI for Area k also exceeds 1. Generally, the driving exposure route across all areas is dermal contact while wading.

Exhibit 5.7 summarizes the contaminants contributing more than 10% of the total systemic toxicity for the child recreational user for direct contact pathways for those areas where the total systemic toxicity for the area exceeds 1. As shown in this exhibit, results are similar to those for the industrial worker with TCE dominating at areas inside the plant and plumes and the inorganic chemicals antimony, manganese, vanadium, chromium, cadmium, and arsenic dominating in areas outside the plume. However, Aroclor-1254 is a driving contaminant for the RGA in Area i.

Exhibit 5.8 summarizes the HIs for biota consumption for the child recreational user over all areas and the driving contaminants for these areas. As shown in this exhibit, consumption of fish is the only biota exposure route that is significant for the child recreator. Additionally, the results for driving contaminants are seen to be similar to those for the direct exposure routes except that cadmium gains in importance in several areas and that tin appears as a driving contaminant for Area d (RGA), bis(2-ethylhexyl)phthalate appears as a driving contaminant for Area f (RGA), and mercury appears as a driving contaminant in Area g (McNairy Formation). Note that total HI for Area g (McNairy Formation) equals 1 so the importance of mercury as a diving contaminant is uncertain.

Exhibit 5.4. Direct contact exposure route summary for the industrial worker – excess lifetime cancer risk[1]

Location	Direct Ingestion of Groundwater	Dermal Contact while showering	Inhalation of vapors while showering	Location Total
Area a UCRS % of Total	3.2×10^{-3} 58%	1.2×10^{-3} 22%	1.1×10^{-3} 20%	**5.0×10^{-3}**
Area a RGA % of Total	1.8×10^{-2} 59%	6.9×10^{-3} 23%	5.3×10^{-3} 18%	**3.0×10^{-2}**
Area a McN % of Total	NA NA	NA NA	NA NA	**NA**
Area b UCRS % of Total	1.8×10^{-3} 62%	4.4×10^{-4} 15%	6.9×10^{-4} 23%	**3.0×10^{-3}**
Area b RGA % of Total	7.9×10^{-3} 88%	3.5×10^{-4} 4%	7.6×10^{-4} 8%	**9.0×10^{-3}**
Area b McN % of Total	2.4×10^{-5} 60%	8.9×10^{-6} 22%	6.9×10^{-6} 17%	**4.0×10^{-5}**
Area c UCRS % of Total	$<1 \times 10^{-6}$ 33%	$<1 \times 10^{-6}$ 3%	1.9×10^{-6} 64%	**3.0×10^{-6}**
Area c RGA % of Total	3.7×10^{-5} 8%	6.9×10^{-6} 1%	4.5×10^{-4} 91%	**5.0×10^{-4}**
Area c McN % of Total	NA NA	NA NA	NA NA	**NA**
Area d UCRS % of Total	2.9×10^{-4} 48%	8.5×10^{-5} 14%	2.3×10^{-4} 38%	**6.0×10^{-4}**
Area d RGA % of Total	6.4×10^{-5} 21%	1.4×10^{-5} 5%	2.3×10^{-4} 75%	**3.1×10^{-4}**
Area d McN % of Total	$<1 \times 10^{-6}$ 59%	$<1 \times 10^{-6}$ 23%	$<1 \times 10^{-6}$ 18%	**1.1×10^{-7}**
Area e UCRS % of Total	1.7×10^{-5} 26%	$<1 \times 10^{-6}$ <1%	4.9×10^{-5} 74%	**6.6×10^{-5}**
Area e RGA % of Total	1.6×10^{-4} 46%	5.3×10^{-5} 15%	1.3×10^{-4} 38%	**3.5×10^{-4}**
Area e McN % of Total	2.3×10^{-4} 62%	5.6×10^{-4} 15%	8.2×10^{-5} 22%	**3.7×10^{-4}**
Area f UCRS % of Total	$<1 \times 10^{-6}$ <1%	$<1 \times 10^{-6}$ <1%	1.4×10^{-4} 100%	**1.4×10^{-4}**
Area f RGA % of Total	5.6×10^{-5} 23%	1.2×10^{-5} 5%	1.8×10^{-4} 72%	**2.5×10^{-4}**
Area f McN % of Total	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	**$<1 \times 10^{-6}$**
Area g UCRS % of Total	2.4×10^{-6} 1%	$<1 \times 10^{-6}$ <1%	1.8×10^{-4} 99%	**1.8×10^{-4}**
Area g RGA % of Total	1.5×10^{-5} 7%	$<1 \times 10^{-6}$ <1%	1.9×10^{-4} 93%	**2.0×10^{-4}**
Area g McN % of Total	1.6×10^{-5} 99%	$<1 \times 10^{-6}$ <1%	$<1 \times 10^{-6}$ <1%	**1.6×10^{-5}**
Area h UCRS % of Total	$<1 \times 10^{-6}$ <1%	$<1 \times 10^{-6}$ <1%	7.9×10^{-5} 100%	**7.9×10^{-5}**
Area h RGA % of Total	1.5×10^{-5} 13%	$<1 \times 10^{-6}$ <1%	9.9×10^{-5} 87%	**1.1×10^{-4}**
Area h McN % of Total	1.8×10^{-6} 2%	$<1 \times 10^{-6}$ <1%	7.7×10^{-5} 98%	**7.9×10^{-5}**

Exhibit 5.4 (continued)

Location	Direct Ingestion of Groundwater	Dermal Contact while showering	Inhalation of vapors while showering	Location Total
Area i UCRS % of Total	3.8×10^{-5} 22%	$<1 \times 10^{-6}$ <1%	1.4×10^{-4} 78%	**1.8×10^{-4}**
Area i RGA % of Total	1.6×10^{-4} 43%	4.6×10^{-5} 12%	1.7×10^{-4} 45%	**3.8×10^{-4}**
Area i McN % of Total	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	**$<1 \times 10^{-6}$**
Area j UCRS % of Total	NA NA	NA NA	NA NA	**NA**
Area j RGA % of Total	2.2×10^{-5} 99%	$<1 \times 10^{-6}$ <1%	$<1 \times 10^{-6}$ <1%	**2.3×10^{-5}**
Area j McN % of Total	4.5×10^{-4} 99%	4.0×10^{-6} <1%	$<1 \times 10^{-6}$ <1%	**4.5×10^{-4}**
Area k Terrace [2] % of Total	2.7×10^{-4} 42%	3.8×10^{-5} 6%	3.3×10^{-4} 52%	**6.4×10^{-4}**
Area l UCRS % of Total	3.4×10^{-2} 87%	1.4×10^{-3} 4%	4.0×10^{-3} 10%	**4.0×10^{-2}**
Area l RGA % of Total	3.1×10^{-2} 88%	1.1×10^{-3} 3%	3.1×10^{-3} 9%	**3.5×10^{-2}**
Area l McN % of Total	1.8×10^{-5} 60%	6.6×10^{-6} 22%	5.1×10^{-6} 17%	**3.0×10^{-5}**
Area m UCRS % of Total	2.6×10^{-5} 21%	$<1 \times 10^{-6}$ <1%	1.0×10^{-4} 79%	**1.3×10^{-4}**
Area m RGA % of Total	2.2×10^{-4} 47%	5.2×10^{-5} 11%	1.9×10^{-4} 41%	**4.6×10^{-4}**
Area m McN % of Total	1.3×10^{-4} 55%	3.7×10^{-5} 15%	7.0×10^{-5} 29%	**2.4×10^{-4}**
Area n UCRS % of Total	3.4×10^{-2} 87%	1.3×10^{-3} 3%	3.9×10^{-3} 10%	**3.9×10^{-2}**
Area n RGA % of Total	1.2×10^{-2} 87%	5.4×10^{-4} 4%	1.4×10^{-3} 10%	**1.4×10^{-2}**
Area n McN % of Total	1.4×10^{-4} 58%	4.0×10^{-5} 17%	5.7×10^{-5} 25%	**2.3×10^{-4}**

Notes: NA indicates that there were no data for the pathway or area.
Values for ELCR greater than 1×10^{-2} fall outside the calculation bounds in EPA 1989a and are approximate values.

[1] Current convention is to use one significant digit for presentation of ELCRs. Two significant digits are used here when to enable the reader to match the numbers reported in the exhibit with those in its associated risk characterization table. Additionally, use of two significant digits allows the reader to sum the route values and check the location total.

[2] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porter's Creek Clay only.

Exhibit 5.5. Driving contaminants' summary for direct contact exposure routes for the industrial worker scenario – excess lifetime cancer risk

Location	Driving Contaminants Over All Exposure Routes	Location Total
Area a UCRS	TCE (97%)	5.0×10^{-3}
Area a RGA	TCE (99%)	3.0×10^{-2}
Area a McN	NA	NA
Area b UCRS	TCE (63%); Vinyl chloride (23%); ^{222}Rn (10%)	3.0×10^{-3}
Area b RGA	Vinyl chloride (93%)	9.0×10^{-3}
Area b McN	TCE (97%)	4.0×10^{-5}
Area c UCRS	Chloroform (71%)	3.0×10^{-6}
Area c RGA	^{222}Rn (86%)	5.0×10^{-4}
Area c McN	NA	NA
Area d UCRS	TCE (54%); ^{222}Rn (22%); 1,1-DCE (11%)	6.0×10^{-4}
Area d RGA	^{222}Rn (72%); TCE (18%)	3.1×10^{-4}
Area d McN	NA	$<1 \times 10^{-6}$
Area e UCRS	^{222}Rn (74%); Arsenic (26%)	6.6×10^{-5}
Area e RGA	Beryllium (35%); ^{222}Rn (34%); TCE (26%)	3.5×10^{-4}
Area e McN	Beryllium (57%); ^{222}Rn (22%); Arsenic (21%)	3.7×10^{-4}
Area f UCRS	^{222}Rn (100%)	1.4×10^{-4}
Area f RGA	^{222}Rn (63%); TCE (20%); 1,1-DCE (10%)	2.5×10^{-4}
Area f McN	NA	$<1 \times 10^{-6}$
Area g UCRS	^{222}Rn (99%)	1.8×10^{-4}
Area g RGA	^{222}Rn (93%)	2.0×10^{-4}
Area g McN	Arsenic (84%); ^{226}Ra (11%)	1.6×10^{-5}
Area h UCRS	^{222}Rn (100%)	7.9×10^{-5}
Area h RGA	^{222}Rn (87%); Arsenic (13%)	1.1×10^{-4}
Area h McN	^{222}Rn (98%)	7.9×10^{-5}
Area i UCRS	^{222}Rn (78%); Arsenic (20%)	1.8×10^{-4}
Area i RGA	^{222}Rn (44%); Beryllium (42%)	3.8×10^{-4}
Area i McN	NA	$<1 \times 10^{-6}$
Area j UCRS	NA	NA
Area j RGA	Arsenic (100%)	2.3×10^{-5}
Area j McN	Arsenic (100%)	4.5×10^{-4}
Area k Terrace[1]	^{222}Rn (41%); 1,1-DCE (19%); Vinyl chloride (17%); Beryllium (14%)	6.4×10^{-4}
Area l UCRS	Vinyl chloride (91%)	4.0×10^{-2}
Area l RGA	Vinyl chloride (95%)	3.5×10^{-2}
Area l McN	TCE (97%)	3.0×10^{-5}
Area m UCRS	^{222}Rn (78%); Arsenic (17%)	1.3×10^{-4}
Area m RGA	Beryllium (32%); ^{222}Rn (29%); 1,1-DCE (19%)	4.6×10^{-4}
Area m McN	Beryllium (58%); ^{222}Rn (29%); Arsenic (12%)	2.4×10^{-4}
Area n UCRS	Vinyl chloride (93%)	3.9×10^{-2}
Area n RGA	Vinyl chloride (91%)	1.4×10^{-2}
Area n McN	Beryllium (60%); ^{222}Rn (24%); Arsenic (11%)	2.3×10^{-4}

Notes NA indicates that there were no data for that route or area.
ELCR $<1 \times 10^{-6}$ indicates that total ELCR is less than 1×10^{-6}; therefore, COCs are not listed.
COCs contributing more than 10% of total ELCR are listed. Percentages rounded to nearest whole number.
Values for ELCR greater than 1×10^{-2} fall outside the calculation bounds in EPA 1989a and are approximate values.

[1] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porter's Creek Clay only.

Exhibit 5.6. Direct contact exposure route summary for the child recreator - systemic toxicity[1]

Location	Direct Ingestion of Groundwater	Dermal Contact while swimming	Dermal Contact while Wading	Location Total without lead	Location Total with lead[2]
Area a UCRS % of Total	15.4 2%	236 37%	379 60%	**631**	**3,310**
Area a RGA % of Total	85.1 2%	1,300 37%	2,100 60%	**3,490**	**3,490**
Area a McN % of Total	NA NA	NA NA	NA NA	**NA**	**NA**
Area b UCRS % of Total	5.5 3%	81.3 37%	131 60%	**218**	**288**
Area b RGA % of Total	0.6 2%	9.6 37%	15.4 60%	**25.8**	**1,630**
Area b McN % of Total	0.4 4%	3.8 37%	6.2 59%	**10.4**	**10.4**
Area c UCRS % of Total	<0.1 6%	0.1 36%	0.2 58%	**0.3**	**0.3**
Area c RGA % of Total	0.2 4%	1.8 37%	2.9 59%	**4.9**	**4.9**
Area c McN % of Total	NA NA	NA NA	NA NA	**NA**	**NA**
Area d UCRS % of Total	2.0 4%	18.1 37%	29.3 59%	**49.5**	**1,390**
Area d RGA % of Total	0.2 3%	2.8 37%	4.5 59%	**7.6**	**2,620**
Area d McN % of Total	<0.1 6%	<0.1 36%	<0.1 58%	**<0.1**	**<0.1**
Area e UCRS % of Total	0.2 4%	1.2 37%	2.0 59%	**3.4**	**3.4**
Area e RGA % of Total	0.4 3%	4.7 37%	7.6 60%	**12.7**	**12.7**
Area e McN % of Total	0.3 5%	2.2 36%	3.6 59%	**6.1**	**6.1**
Area f UCRS % of Total	<0.1 8%	0.1 35%	0.2 57%	**0.4**	**0.4**
Area f RGA % of Total	0.3 3%	3.5 37%	5.7 60%	**9.4**	**9.4**
Area f McN % of Total	<0.1 17%	<0.1 32%	<0.1 51%	**<0.1**	**<0.1**
Area g UCRS % of Total	<0.1 5%	0.5 37%	0.8 59%	**1.4**	**1.4**
Area g RGA % of Total	<0.1 4%	0.6 36%	1.0 57%	**1.7**	**2,590**
Area g McN % of Total	<0.1 31%	<0.1 26%	<0.1 42%	**<0.1**	**<0.1**
Area h UCRS % of Total	<0.1 11%	0.1 34%	0.2 55%	**0.3**	**0.3**
Area h RGA % of Total	0.1 7%	0.6 36%	0.9 57%	**1.6**	**1.6**
Area h McN % of Total	<0.1 72%	<0.1 11%	<0.1 17%	**<0.1**	**<0.1**

Exhibit 5.6 (continued)

Location	Direct Ingestion of Groundwater	Dermal Contact while swimming	Dermal Contact while Wading	Location Total without lead	Location Total with lead[2]
Area i UCRS % of Total	0.2 5%	1.4 35%	2.3 57%	**4.0**	**2,230**
Area i RGA % of Total	0.4 4%	4.1 37%	6.6 59%	**11.1**	**11.1**
Area i McN % of Total	<0.1 4%	0.5 37%	0.8 60%	**1.3**	**1.3**
Area j UCRS % of Total	NA NA	NA NA	NA NA	**NA**	**NA**
Area j RGA % of Total	0.1 10%	0.5 34%	0.7 55%	**1.3**	**1.3**
Area j McN % of Total	0.5 31%	0.4 27%	0.6 43%	**1.5**	**1.5**
Area k Terrace [3] % of Total	1.1 10%	3.7 34%	6.0 55%	**10.8**	**5,960**
Area l UCRS % of Total	6.9 3%	96.4 37%	155 60%	**259**	**2,060**
Area l RGA % of Total	3.2 3%	43.7 37%	70.4 60%	**118**	**2,040**
Area l McN % of Total	0.3 4%	3.1 37%	5.0 59%	**8.5**	**8.5**
Area m UCRS % of Total	0.3 5%	2.1 36%	3.4 58%	**5.9**	**2,000**
Area m RGA % of Total	0.4 3%	5.5 37%	8.9 59%	**15.1**	**1,990**
Area m McN % of Total	0.3 7%	1.4 36%	2.2 58%	**3.9**	**3.9**
Area n UCRS % of Total	5.4 3%	73.6 37%	119 60%	**198**	**2,070**
Area n RGA % of Total	2.0 3%	26.0 37%	42.0 60%	**70.1**	**1,980**
Area n McN % of Total	0.4 5%	3.2 37%	5.2 59%	**8.8**	**8.8**

Notes: NA indicates that there were no data for the pathway or area.

[1] Current convention is to use one significant digit for presentation of hazard indices. Three significant digits are used here when the hazard index 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's value is greater than 0.1, allows the reader to sum the route values and check the location total.

[2] The very large values are the result of the retention of lead as a COPC at a value only slightly greater than the background concentration and the use of a provisional reference dose provided in comments by KDEP.

[3] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porter's Creek Clay only.

Exhibit 5.7. Driving contaminants' summary for direct contact exposure routes for the child recreator scenario - systemic toxicity

Location	Driving Contaminants Over Direct Contact Exposure Routes	Location Total[1]
Area a UCRS	TCE (100%)	631
Area a RGA	TCE (100%)	3,490
Area a McN	NA	NA
Area b UCRS	TCE (99%)	218
Area b RGA	TCE (72%); Tetrachloroethene (19%)	25.8
Area b McN	Antimony (56%); TCE (44%)	10.4
Area c UCRS	HI<1	0.3
Area c RGA	TCE (64%); Chromium (31%)	4.9
Area c McN	NA	NA
Area d UCRS	TCE (79%); Manganese (14%)	49.5
Area d RGA	TCE (86%);	7.6
Area d McN	HI<1	<0.1
Area e UCRS	Vanadium (84%); Chromium (10%)	3.4
Area e RGA	TCE (83%); Cadmium (11%)	12.7
Area e McN	Vanadium (45%); Cadmium (29%); Chromium (15%)	6.1
Area f UCRS	HI<1	0.4
Area f RGA	TCE (61%); Cadmium (26%)	9.4
Area f McN	HI<1	<0.1
Area g UCRS	Chromium (46%); Vanadium (43%); Manganese (10%)	1.4
Area g RGA	Cadmium (60%); Chromium (34%)	1.7
Area g McN	HI<1	<0.1
Area h UCRS	HI<1	0.3
Area h RGA	Chromium (64%); Vanadium (26%)	1.6
Area h McN	HI<1	<0.1
Area i UCRS	Vanadium (46%); Cadmium (14%)	4.0
Area i RGA	Antimony (41%); Aroclor-1254 (31%); Chromium (17%)	11.1
Area i McN	Vanadium (86%); Manganese (14%)	1.3
Area j UCRS	NA	NA
Area j RGA	Manganese (44%); Vanadium (44%)	1.3
Area j McN	Manganese (50%); Arsenic (40%)	1.5
Area k Terrace[2]	Manganese (27%); Antimony (26%); Iron (19%); Cadmium (13%)	10.8
Area l UCRS	TCE (98%)	259
Area l RGA	TCE (90%)	118
Area l McN	Antimony (60%); TCE (40%)	8.5
Area m UCRS	Antimony (53%); Vanadium (23%); Cadmium (15%)	5.9
Area m RGA	Antimony (29%); TCE (29%); Aroclor-1254 (23%)	15.1
Area m McN	Cadmium (41%); Vanadium (24%); Chromium (16%); Iron (10%)	3.9
Area n UCRS	TCE (97%)	198
Area n RGA	TCE (73%)	70.1
Area n McN	Antimony (51%); Cadmium (19%); TCE (14%)	8.8

Notes NA indicates that there were no data for that route or area.
HI<1 indicates that total scenario hazard index is less than 1; therefore, COCs are not listed.
COCs contributing more than 10% of total HI are listed. Percentages rounded to nearest whole number.

[1] Totals are without lead as a COPC. The total HIs with lead are in Exhibit 5.6.
[2] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porter's Creek Clay only.

Exhibit 5.8. Biota exposure route summary for the child recreator – systemic toxicity[1]

Parameter	Consumption of Fish	Consumption of Venison	Consumption of Rabbit	Consumption of Quail
	Area a UCRS (Direct Route Total HI = 631)[2]			
Total HI	158	<0.1	<0.1	<0.1
Driving COC	TCE (98%)			
	Area a RGA (Direct Route Total HI = 3,490)[2]			
Total HI	866	<0.1	<0.1	<0.1
Driving COC	TCE (100%)			
	Area a McNairy Formation (Direct Route Total HI = NA)[2]			
Total HI	NA	NA	NA	NA
Driving COC	NA			
	Area b UCRS (Direct Route Total HI = 218)[2]			
Total HI	56.4	<0.1	<0.1	<0.1
Driving COC	TCE (94%)			
	Area b RGA (Direct Route Total HI = 25.8)[2]			
Total HI	9.1	<0.1	<0.1	<0.1
Driving COC	TCE (50%); Iron (11%)			
	Area b McNairy Formation (Direct Route Total HI = 10.4)[2]			
Total HI	8.8	<0.1	<0.1	<0.1
Driving COC	Antimony (98%)			
	Area c UCRS (Direct Route Total HI = 0.3)[2]			
Total HI	0.5	<0.1	<0.1	<0.1
Driving COC	None[3]			
	Area c RGA (Direct Route Total HI = 4.9)[2]			
Total HI	6.35	<0.1	<0.1	<0.1
Driving COC	Chromium (63%); Iron (20%); TCE (12%)			
	Area c McNairy Formation (Direct Route Total HI = NA)[2]			
Total HI	NA	NA	NA	NA
Driving COC	NA			
	Area d UCRS (Direct Route Total HI = 49.5)[2]			
Total HI	48.6	<0.1	<0.1	<0.1
Driving COC	Manganese (45%); Iron (29%); TCE (19%)			
	Area d RGA (Direct Route Total HI = 7.6)[2]			
Total HI	5.0	<0.1	<0.1	<0.1
Driving COC	TCE (32%); Tin (23%); Manganese (19%); Chromium (13%)			
	Area d McNairy Formation (Direct Route Total HI = <0.1)[2]			
Total HI	0.1	<0.1	<0.1	<0.1
Driving COC	None[3]			
	Area e UCRS (Direct Route Total HI = 3.4)[2]			
Total HI	2.3	<0.1	<0.1	<0.1
Driving COC	Chromium (38%); Iron (37%); Nickel (10%)			
	Area e RGA (Direct Route Total HI = 12.7)[2]			
Total HI	4.4	<0.1	<0.1	<0.1
Driving COC	TCE (59%); Cadmium (21%); Iron (12%)			
	Area e McNairy Formation (Direct Route Total HI = 6.1)[2]			
Total HI	7.4	<0.1	<0.1	<0.1
Driving COC	Iron (38%); Chromium (31%); Cadmium (26%)			
	Area f UCRS (Direct Route Total HI = 0.4)[2]			
Total HI	0.9	<0.1	<0.1	<0.1
Driving COC	None[3]			
	Area f RGA (Direct Route Total HI = 9.4)[2]			
Total HI	6.3	<0.1	<0.1	<0.1
Driving COC	Cadmium (26%); Chromium (25%); TCE (22%); Bis(2-ethylhexyl)phthalate (20%)			

Exhibit 5.8 (continued)

Parameter	Consumption of Fish	Consumption of Venison	Consumption of Rabbit	Consumption of Quail
	Area f McNairy Formation (Direct Route Total HI = <0.1)[2]			
Total HI	0.1	<0.1	<0.1	<0.1
Driving COC	None[3]			
	Area g UCRS (Direct Route Total HI = 1.4)[2]			
Total HI	2.3	<0.1	<0.1	<0.1
Driving COC	Chromium (76%); Manganese (21%)			
	Area g RGA (Direct Route Total HI = 1.7)[2]			
Total HI	3.1	<0.1	<0.1	<0.1
Driving COC	Chromium (49%); Cadmium (22%); Iron (18%)			
	Area g McNairy Formation (Direct Route Total HI = <0.1)[2]			
Total HI	1.0	<0.1	<0.1	<0.1
Driving COC	Mercury (100%)			
	Area h UCRS (Direct Route Total HI = 0.3)[2]			
Total HI	0.8	<0.1	<0.1	<0.1
Driving COC	None[3]			
	Area h RGA (Direct Route Total HI = 1.6)[2]			
Total HI	4.1	<0.1	<0.1	<0.1
Driving COC	Chromium (64%); Iron (34%)			
	Area h McNairy Formation (Direct Route Total HI = <0.1)[2]			
Total HI	<0.1	<0.1	<0.1	<0.1
Driving COC	None[3]			
	Area i UCRS (Direct Route Total HI = 4.0)[2]			
Total HI	5.1	<0.1	<0.1	<0.1
Driving COC	Iron (21%); Manganese (21%); Antimony (20%); Chromium (11%)			
	Area i RGA (Direct Route Total HI = 11.1)[2]			
Total HI	142	<0.1	<0.1	<0.1
Driving COC	Aroclor-1254 (91%)			
	Area i McNairy Formation (Direct Route Total HI = 1.3)[2]			
Total HI	0.7	<0.1	<0.1	<0.1
Driving COC	None[3]			
	Area j UCRS (Direct Route Total HI = NA)[2]			
Total HI	NA	NA	NA	NA
Driving COC	NA			
	Area j RGA (Direct Route Total HI = 1.3)[2]			
Total HI	2.9	<0.1	<0.1	<0.1
Driving COC	Manganese (66%); Iron (30%)			
	Area j McNairy Formation (Direct Route Total HI = 1.5)[2]			
Total HI	2.6	<0.1	<0.1	<0.1
Driving COC	Manganese (100%)			
	Area k Terrace[4] (Direct Route Total HI = 10.8)[2]			
Total HI	46.3	<0.1	<0.1	<0.1
Driving COC	Iron (67%); Manganese (21%)			
	Area l UCRS (Direct Route Total HI = 259)[2]			
Total HI	70.1	<0.1	<0.1	<0.1
Driving COC	TCE (89%)			
	Area l RGA (Direct Route Total HI = 118)[2]			
Total HI	36.9	<0.1	<0.1	<0.1
Driving COC	TCE (70%); Carbon tetrachloride (14%)			
	Area l McNairy Formation (Direct Route Total HI = 8.5)[2]			
Total HI	7.6	<0.1	<0.1	<0.1
Driving COC	Antimony (88%); TCE (11%)			

Exhibit 5.8 (continued)

Parameter	Consumption of Fish	Consumption of Venison	Consumption of Rabbit	Consumption of Quail
	Area m UCRS (Direct Route Total HI = 5.9)[2]			
Total HI	6.9	<0.1	<0.1	<0.1
Driving COC		Antimony (59%); Iron (11%)		
	Area m RGA (Direct Route Total HI = 15.1)[2]			
Total HI	141	<0.1	<0.1	<0.1
Driving COC		Aroclor-1254 (91%)		
	Area m McNairy Formation (Direct Route Total HI = 3.9)[2]			
Total HI	9.4	<0.1	<0.1	<0.1
Driving COC		Iron (61%); Chromium (17%); Cadmium (11%)		
	Area n UCRS (Direct Route Total HI = 198)[2]			
Total HI	56.2	<0.1	<0.1	<0.1
Driving COC		TCE (84%)		
	Area n RGA (Direct Route Total HI = 70.1)[2]			
Total HI	165	<0.1	<0.1	<0.1
Driving COC		Aroclor-1254 (83%)		
	Area n McNairy Formation (Direct Route Total HI = 8.8)[2]			
Total HI	10.4	<0.1	<0.1	<0.1
Driving COC		Antimony (56%); Iron (16%); Cadmium (10%)		

Notes: NA indicates that there were no data for the exposure route or area.

[1] All Total HI values do not include contribution from lead as a COPC.
[2] Direct Route Total HI is from Exhibit 5.6.
[3] No COCs because Total HI < 1.0
[4] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porters Creek Clay only.

5.4.2.2 Excess lifetime cancer risk

Exhibit 5.9 summarizes the total ELCRs for direct contact exposure routes for the recreational user over all areas for the unfiltered data set. As shown in this exhibit, the total ELCR is greater than 1×10^{-6} for all areas except Areas a, c, d, f, h, and i for the McNairy Formation and Areas c, f, g, and h for the UCRS. The total ELCR for the RGA in all areas and for groundwater in Area k has a total ELCR greater than 1×10^{-6}.

For ELCR, the dermal contact exposure routes are seen to be much more important than the ingestion exposure route for all areas except Area e (UCRS), Area g (UCRS, RGA, and McNairy Formation), Area h (RGA and McNairy Formation), and Area j (RGA and McNairy Formation). However, total ELCR in each of the areas where ingestion is of greater importance than dermal exposure has a total ELCR that is near 1×10^{-6}.

Exhibit 5.10 summarizes the contaminants contributing more than 10% of the total ELCR for the recreational user over all areas. As shown in this exhibit, the driving contaminants over most areas is similar to those for the industrial worker with TCE and its breakdown products being of greatest importance in areas associated with the TCE plumes and arsenic and beryllium being of greatest importance elsewhere. Of note is the absence or ^{222}Rn as a driving contaminant. This is the result of not considering an inhalation exposure route for the recreational user.

Exhibit 5.11 summarizes the total ELCRs for the biota consumption exposure routes for the recreational user. As shown in this exhibit, total ELCR is greater than 1×10^{-6} for Areas b, e, g, h, l, m, and n for the McNairy Formation; Areas h and j for the RGA; and Areas a, b, c, d, g, i, l, m, and n for the UCRS. The total ELCR for Area k groundwater also exceeds 1×10^{-6}. Similar to total HI for the biota consumption routes, only consumption of fish is of any importance for total ELCR. Although driving contaminants are similar to those for direct contact, ^{226}Ra, ^{99}Tc, bis(2-ethylhexyl)phthalate, ^{137}Cs, and Aroclor-1254 gain significantly in importance when the biota consumption exposure routes are considered.

5.4.3 Rural Resident

5.4.3.1 Systemic Toxicity

Exhibit 5.12 summarizes the total HIs for direct contact exposure routes for the child rural resident over all areas. As shown in this exhibit, the total HI (i.e., Location Total without lead in Exhibit 5.10) is greater than 1 for all areas except Areas a, c, d, f, g, and h for the McNairy Formation and Area j for the UCRS. Note that this result for Areas a and c for the McNairy Formation and Area j for the UCRS is the result of not being able to assess these area and depth combinations due to the lack of data.. For areas with very large total HIs, inhalation of vapors dominates the total HI; however, for areas with HIs much closer to 1, ingestion of groundwater tends to dominate the total HI.

Exhibit 5.13 summarizes the contaminants contributing more than 10% of the total systemic toxicity for the child rural resident over all areas for those areas where the total systemic toxicity for the area exceeds 1. Generally, results here are similar to those for the industrial worker with TCE being a dominant contaminant in several areas. Additionally, the same inorganic chemicals are also driving contaminants in other areas. Of note, is the appearance of acrylonitrile as a driving contaminant in Area i (RGA).

Exhibit 5.14 summarizes the total HIs for the consumption of biota exposure routes. Only the consumption of vegetables route is of any importance in most areas. However, both the consumption of beef and the consumption of milk have HIs greater than 0.1 for some areas, and the total HI for consumption of beef for Area k exceeds 1. Areas where the total HI across all areas is less than 1 are Areas a, c, d, f, g, and h for the McNairy Formation and Areas c and j for the UCRS. All areas for the

Exhibit 5.9. Direct contact exposure route summary for the recreator – excess lifetime cancer risk[1]

Location	Direct Ingestion while Swimming	Dermal Contact while Swimming	Dermal Contact while Wading	Location Total
Area a UCRS % of Total	2.1×10^{-4} 2%	5.6×10^{-3} 45%	6.5×10^{-3} 53%	**1.2×10^{-2}**
Area a RGA % of Total	1.2×10^{-3} 2%	3.1×10^{-2} 45%	3.6×10^{-2} 53%	**6.6×10^{-2}**
Area a McN % of Total	NA NA	NA NA	NA NA	**NA**
Area b UCRS % of Total	1.2×10^{-4} 3%	2.0×10^{-3} 45%	2.3×10^{-3} 53%	**4.4×10^{-3}**
Area b RGA % of Total	5.2×10^{-4} 13%	1.6×10^{-3} 40%	1.8×10^{-3} 47%	**3.9×10^{-3}**
Area b McN % of Total	1.6×10^{-6} 2%	4.0×10^{-5} 45%	4.7×10^{-5} 53%	**8.9×10^{-5}**
Area c UCRS % of Total	$<1 \times 10^{-6}$ 5%	$<1 \times 10^{-6}$ 44%	$<1 \times 10^{-6}$ 51%	**9.3×10^{-7}**
Area c RGA % of Total	2.4×10^{-6} 4%	3.1×10^{-5} 44%	3.6×10^{-5} 52%	**6.9×10^{-5}**
Area c McN % of Total	NA NA	NA NA	NA NA	**NA**
Area d UCRS % of Total	1.9×10^{-5} 2%	3.8×10^{-4} 45%	4.5×10^{-4} 53%	**8.5×10^{-4}**
Area d RGA % of Total	4.0×10^{-6} 3%	6.4×10^{-5} 45%	7.5×10^{-5} 52%	**1.4×10^{-4}**
Area d McN % of Total	$<1 \times 10^{-6}$ 2%	$<1 \times 10^{-6}$ 45%	$<1 \times 10^{-6}$ 53%	**2.5×10^{-7}**
Area e UCRS % of Total	1.1×10^{-6} 40%	$<1 \times 10^{-6}$ 27%	$<1 \times 10^{-6}$ 32%	**2.8×10^{-6}**
Area e RGA % of Total	1.0×10^{-5} 2%	2.4×10^{-4} 45%	2.8×10^{-4} 53%	**5.3×10^{-4}**
Area e McN % of Total	1.5×10^{-5} 3%	2.5×10^{-4} 45%	3.0×10^{-4} 53%	**5.7×10^{-4}**
Area f UCRS % of Total	$<1 \times 10^{-6}$ 7%	$<1 \times 10^{-6}$ 43%	$<1 \times 10^{-6}$ 50%	**2.1×10^{-7}**
Area f RGA % of Total	3.7×10^{-6} 3%	5.6×10^{-5} 45%	6.5×10^{-5} 52%	**1.2×10^{-4}**
Area f McN % of Total	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	**$<1 \times 10^{-6}$**
Area g UCRS % of Total	$<1 \times 10^{-6}$ 100%	$<1 \times 10^{-6}$ <1%	$<1 \times 10^{-6}$ <1%	**9.1×10^{-8}**
Area g RGA % of Total	$<1 \times 10^{-6}$ 42%	$<1 \times 10^{-6}$ 27%	$<1 \times 10^{-6}$ 31%	**2.2×10^{-6}**
Area g McN % of Total	$<1 \times 10^{-6}$ 46%	$<1 \times 10^{-6}$ 25%	$<1 \times 10^{-6}$ 29%	**2.1×10^{-6}**
Area h UCRS % of Total	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	**$<1 \times 10^{-6}$**
Area h RGA % of Total	$<1 \times 10^{-6}$ 40%	$<1 \times 10^{-6}$ 27%	$<1 \times 10^{-6}$ 32%	**2.4×10^{-6}**
Area h McN % of Total	$<1 \times 10^{-6}$ 100%	$<1 \times 10^{-6}$ <1%	$<1 \times 10^{-6}$ <1%	**6.9×10^{-8}**

Exhibit 5.9 (continued)

Location	Direct Ingestion while Swimming	Dermal Contact while Swimming	Dermal Contact while Wading	Location Total
Area i UCRS % of Total	2.5×10^{-6} 38%	1.9×10^{-6} 29%	2.2×10^{-6} 34%	**6.5×10^{-6}**
Area i RGA % of Total	1.1×10^{-5} 2%	2.1×10^{-4} 45%	2.4×10^{-4} 53%	**4.6×10^{-4}**
Area i McN % of Total	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	**$<1 \times 10^{-6}$**
Area j UCRS % of Total	NA NA	NA NA	NA NA	**NA**
Area j RGA % of Total	1.5×10^{-6} 43%	$<1 \times 10^{-6}$ 26%	1.0×10^{-6} 31%	**3.4×10^{-6}**
Area j McN % of Total	3.0×10^{-5} 43%	1.8×10^{-5} 26%	2.1×10^{-5} 31%	**6.8×10^{-5}**
Area k Terrace [2] % of Total	1.7×10^{-5} 5%	1.7×10^{-4} 44%	2.0×10^{-4} 52%	**3.8×10^{-4}**
Area l UCRS % of Total	2.3×10^{-3} 14%	6.3×10^{-3} 39%	7.4×10^{-3} 46%	**1.6×10^{-2}**
Area l RGA % of Total	2.1×10^{-3} 16%	5.1×10^{-3} 39%	6.0×10^{-3} 46%	**1.3×10^{-2}**
Area l McN % of Total	1.2×10^{-6} 2%	3.0×10^{-5} 45%	3.5×10^{-5} 53%	**6.6×10^{-5}**
Area m UCRS % of Total	1.7×10^{-6} 32%	1.6×10^{-6} 31%	1.9×10^{-6} 37%	**5.2×10^{-6}**
Area m RGA % of Total	1.4×10^{-5} 3%	2.3×10^{-4} 45%	2.7×10^{-4} 52%	**5.2×10^{-4}**
Area m McN % of Total	8.8×10^{-6} 2%	1.7×10^{-4} 45%	2.0×10^{-4} 53%	**3.7×10^{-4}**
Area n UCRS % of Total	2.3×10^{-3} 15%	5.7×10^{-3} 39%	6.7×10^{-3} 46%	**1.5×10^{-2}**
Area n RGA % of Total	8.1×10^{-4} 13%	2.4×10^{-3} 40%	2.8×10^{-3} 47%	**6.1×10^{-3}**
Area n McN % of Total	8.9×10^{-6} 2%	1.8×10^{-4} 45%	2.1×10^{-4} 53%	**4.0×10^{-4}**

Notes: NA indicates that there were no data for the pathway or area.
Values for ELCR greater than 1×10^{-2} fall outside the calculation bounds in EPA 1989a and are approximate values.

[1] Current convention is to use one significant digit for presentation of ELCRs. Two significant digits are used here when to enable the reader to match the numbers reported in the exhibit with those in its associated risk characterization table. Additionally, use of two significant digits allows the reader to sum the route values and check the location total.

[2] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porter's Creek Clay only.

Exhibit 5.10. Driving contaminants' summary for direct contact exposure routes for the recreator scenario – excess lifetime cancer risk

Location	Driving Contaminants Over Direct Contact Exposure Routes	Location Total
Area a UCRS	TCE (100%)	1.2×10^{-2}
Area a RGA	TCE (99%)	6.6×10^{-2}
Area a McN	NA	NA
Area b UCRS	TCE (95%)	4.4×10^{-3}
Area b RGA	Vinyl chloride (62%); Tetrachloroethene (20%)	3.9×10^{-3}
Area b McN	TCE (100%)	8.9×10^{-5}
Area c UCRS	ELCR$<1 \times 10^{-6}$	9.3×10^{-7}
Area c RGA	TCE (89%); 1.1-DCE (11%)	6.9×10^{-5}
Area c McN	NA	NA
Area d UCRS	TCE (87%); Beryllium (11%)	8.5×10^{-4}
Area d RGA	TCE (89%)	1.4×10^{-4}
Area d McN	ELCR$<1 \times 10^{-6}$	2.5×10^{-7}
Area e UCRS	Arsenic (93%)	2.8×10^{-6}
Area e RGA	Beryllium (61%); TCE (39%)	5.3×10^{-4}
Area e McN	Beryllium (98%)	5.7×10^{-4}
Area f UCRS	ELCR$<1 \times 10^{-6}$	2.1×10^{-7}
Area f RGA	TCE (90%)	1.2×10^{-4}
Area f McN	ELCR$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Area g UCRS	ELCR$<1 \times 10^{-6}$	9.1×10^{-8}
Area g RGA	Arsenic (91%)	2.2×10^{-6}
Area g McN	Arsenic (96%)	2.1×10^{-6}
Area h UCRS	NA	$<1 \times 10^{-6}$
Area h RGA	Arsenic (92%)	2.4×10^{-6}
Area h McN	ELCR$<1 \times 10^{-6}$	6.9×10^{-8}
Area i UCRS	Arsenic (81%)	6.5×10^{-6}
Area i RGA	Beryllium (94%)	4.6×10^{-4}
Area i McN	ELCR$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Area j UCRS	NA	NA
Area j RGA	Arsenic (100%)	3.4×10^{-6}
Area j McN	Arsenic (100%)	6.8×10^{-5}
Area k Terrace[1]	Beryllium (84%)	3.8×10^{-4}
Area l UCRS	Vinyl chloride (68%); TCE (31%)	1.6×10^{-2}
Area l RGA	Vinyl chloride (75%); TCE (16%)	1.3×10^{-2}
Area l McN	TCE (100%)	6.6×10^{-5}
Area m UCRS	Arsenic (61%)	5.2×10^{-6}
Area m RGA	Beryllium (75%); TCE (17%)	5.2×10^{-4}
Area m McN	Beryllium (99%)	3.7×10^{-4}
Area n UCRS	Vinyl chloride (73%); TCE (25%)	1.5×10^{-2}
Area n RGA	Vinyl chloride (62%); TCE (16%); Tetrachloroethene (13%)	6.1×10^{-3}
Area n McN	Beryllium (93%)	4.0×10^{-4}

Notes NA indicates that there were no data for that route or area.
ELCR$<1 \times 10^{-6}$ indicates that total ELCR is less than 1×10^{-6}; therefore, COCs are not listed.
COCs contributing more than 10% of total ELCR are listed. Percentages rounded to nearest whole number.
Values for ELCR greater than 1×10^{-2} fall outside the calculation bounds in EPA 1989a and are approximate values.

[1] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porter's Creek Clay only.

Exhibit 5.11. Biota exposure route summary for the recreator – excess lifetime cancer risk[1]

Parameter	Consumption of Fish	Consumption of Venison	Consumption of Rabbit	Consumption of Quail
	Area a UCRS (Direct Route Total ELCR = 1.2×10^{-2})[2]			
Total ELCR	7.0×10^{-3}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	TCE (99%)			
	Area a RGA (Direct Route Total ELCR = 6.6×10^{-2})[2]			
Total ELCR	3.8×10^{-2}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	TCE (100%)			
	Area a McNairy Formation (Direct Route Total ELCR = NA)[2]			
Total ELCR	NA	NA	NA	NA
Driving COC	NA			
	Area b UCRS (Direct Route Total ELCR = 4.4×10^{-3})[2]			
Total ELCR	2.7×10^{-3}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	TCE (89%)			
	Area b RGA (Direct Route Total ELCR = 3.9×10^{-3})[2]			
Total ELCR	6.2×10^{-3}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	Vinyl chloride (45%); ^{226}Ra (37%)			
	Area b McNairy Formation (Direct Route Total ELCR = 8.9×10^{-5})[2]			
Total ELCR	5.1×10^{-5}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	TCE (98%)			
	Area c UCRS (Direct Route Total ELCR = 9.3×10^{-7})[2]			
Total ELCR	1.1×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	^{99}Tc (40%); Chloroform (26%); TCE (22%); Benzene (12%)			
	Area c RGA (Direct Route Total ELCR = 6.9×10^{-5})[2]			
Total ELCR	5.1×10^{-5}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	TCE (67%); 1,1-Dichloroethene (29%)			
	Area c McNairy Formation (Direct Route Total ELCR = NA)[2]			
Total ELCR	NA	NA	NA	NA
Driving COC	NA			
	Area d UCRS (Direct Route Total ELCR = 8.5×10^{-4})[2]			
Total ELCR	6.7×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	TCE (62%); Beryllium (21%)			
	Area d RGA (Direct Route Total ELCR = 1.4×10^{-4})[2]			
Total ELCR	4.6×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	^{137}Cs (74%); TCE (15%)			
	Area d McNairy Formation (Direct Route Total ELCR = 2.5×10^{-7})[2]			
Total ELCR	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	None[3]			
	Area e UCRS (Direct Route Total ELCR = 2.8×10^{-6})[2]			
Total ELCR	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	None[3]			
	Area e RGA (Direct Route Total ELCR = 5.3×10^{-4})[2]			
Total ELCR	6.2×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	Beryllium (80%); TCE (19%)			
	Area e McNairy Formation (Direct Route Total ELCR = 5.7×10^{-4})[2]			
Total ELCR	8.5×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	Beryllium (100%)			
	Area f UCRS (Direct Route Total ELCR = 2.1×10^{-7})[2]			
Total ELCR	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	None[3]			

Exhibit 5.11 (continued)

Parameter	Consumption of Fish	Consumption of Venison	Consumption of Rabbit	Consumption of Quail
	Area f RGA (Direct Route Total ELCR = 1.2×10^{-4})[2]			
Total ELCR	3.1×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	Bis(2-ethylhexyl)phthalate (76%); TCE (20%)			
	Area f McNairy Formation (Direct Route Total ELCR = $<1 \times 10^{-6}$)[2]			
Total ELCR	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	None[3]			
	Area g UCRS (Direct Route Total ELCR = 9.1×10^{-8})[2]			
Total ELCR	3.6×10^{-5}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	^{226}Ra (98%)			
	Area g RGA (Direct Route Total ELCR = 2.2×10^{-6})[2]			
Total ELCR	1.4×10^{-5}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	^{226}Ra (89%)			
	Area g McNairy Formation (Direct Route Total ELCR = 2.1×10^{-6})[2]			
Total ELCR	3.3×10^{-5}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	^{226}Ra (91%)			
	Area h UCRS (Direct Route Total ELCR = $<1 \times 10^{-6}$)[2]			
Total ELCR	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	None[3]			
	Area h RGA (Direct Route Total ELCR = 2.4×10^{-6})[2]			
Total ELCR	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	None[3]			
	Area h McNairy Formation (Direct Route Total ELCR = 6.9×10^{-8})[2]			
Total ELCR	3.1×10^{-5}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	^{226}Ra (97%)			
	Area i UCRS (Direct Route Total ELCR = 6.5×10^{-6})[2]			
Total ELCR	4.2×10^{-5}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	^{226}Ra (55%); ^{137}Cs (35%)			
	Area i RGA (Direct Route Total ELCR = 4.6×10^{-4})[2]			
Total ELCR	5.1×10^{-3}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	Aroclor-1254 (69%); PCBs (16%); Beryllium (13%)			
	Area i McNairy Formation (Direct Route Total ELCR = $<1 \times 10^{-6}$)[2]			
Total ELCR	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	None[3]			
	Area j UCRS (Direct Route Total ELCR = NA)[2]			
Total ELCR	NA	NA	NA	NA
Driving COC	NA			
	Area j RGA (Direct Route Total ELCR = 3.4×10^{-6})[2]			
Total ELCR	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	None[3]			
	Area j McNairy Formation (Direct Route Total ELCR = 6.8×10^{-5})[2]			
Total ELCR	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	None[3]			
	Area k Terrace[4] (Direct Route Total ELCR = 3.8×10^{-4})[2]			
Total ELCR	6.3×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	Beryllium (78%)			
	Area l UCRS (Direct Route Total ELCR = 1.6×10^{-2})[2]			
Total ELCR	1.6×10^{-2}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	Vinyl chloride (79%); TCE (18%)			

Exhibit 5.11 (continued)

Parameter	Consumption of Fish	Consumption of Venison	Consumption of Rabbit	Consumption of Quail
	Area l RGA (Direct Route Total ELCR = 1.3×10^{-2})[2]			
Total ELCR	1.6×10^{-2}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	Vinyl chloride (73%); ^{226}Ra (10%)			
	Area l McNairy Formation (Direct Route Total ELCR = 6.6×10^{-5})[2]			
Total ELCR	3.8×10^{-5}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	TCE (98%)			
	Area m UCRS (Direct Route Total ELCR = 5.2×10^{-6})[2]			
Total ELCR	4.6×10^{-5}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	^{226}Ra (53%); ^{137}Cs (32%)			
	Area m RGA (Direct Route Total ELCR = 5.2×10^{-4})[2]			
Total ELCR	5.1×10^{-3}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	Aroclor-1254 (68%); PCBs (16%); Beryllium (12%)			
	Area m McNairy Formation (Direct Route Total ELCR = 3.7×10^{-4})[2]			
Total ELCR	5.9×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	Beryllium (96%)			
	Area n UCRS (Direct Route Total ELCR = 1.5×10^{-2})[2]			
Total ELCR	1.5×10^{-2}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	Vinyl chloride (82%); TCE (14%)			
	Area n RGA (Direct Route Total ELCR = 6.1×10^{-3})[2]			
Total ELCR	1.2×10^{-2}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	Vinyl chloride (36%); Aroclor 1254 (31%)			
	Area n McNairy Formation (Direct Route Total ELCR = 4.0×10^{-4})[2]			
Total ELCR	6.1×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC	Beryllium (94%)			

Notes: NA indicates that there were no data for the exposure route or area.

[1] Total ELCR values greater than 1×10^{-2} fall outside the calculation bounds in EPA 1989a and are estimated values.
[2] Direct Route Total ELCR from Exhibit 5.8.
[3] No COCs because Total ELCR $< 1 \times 10^{-6}$
[4] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porters Creek Clay only.

Exhibit 5.12. Direct contact exposure route summary for the child resident - systemic toxicity[1]

Location	Direct Ingestion of Groundwater	Dermal Contact while Showering	Inhalation of Vapors while Showering	Inhalation of Vapors during Household Use	Location Total without lead	Location Total with lead[2]
Area a UCRS % of Total	924 13%	140 2%	500 7%	5,430 78%	**7,000**	**53,100**
Area a RGA % of Total	5,090 13%	781 2%	2,780 7%	30,200 78%	**38,800**	**38,800**
Area a McN % of Total	NA NA	NA NA	NA NA	NA NA	**NA**	**NA**
Area b UCRS % of Total	331 13%	48.6 2%	176 7%	1,910 78%	**2,460**	**3,660**
Area b RGA % of Total	40.8 16%	5.7 <1%	18.2 7%	198 76%	**262**	**27,900**
Area b McN % of Total	24.4 35%	2.3 3%	3.6 5%	39.2 56%	**69.5**	**69.5**
Area c UCRS % of Total	1.2 54%	<0.1 3%	<0.1 4%	0.9 39%	**2.3**	**2.3**
Area c RGA % of Total	12.1 28%	1.1 2%	2.6 6%	28.1 64%	**43.9**	**43.9**
Area c McN % of Total	NA NA	NA NA	NA NA	NA NA	**NA**	**NA**
Area d UCRS % of Total	122 25%	10.9 2%	30.6 6%	332 67%	**496**	**23,600**
Area d RGA % of Total	14.7 19%	1.7 2%	5.3 7%	57.1 72%	**78.8**	**45,000**
Area d McN % of Total	<0.1 29%	<0.1 2%	<0.1 6%	0.1 64%	**0.2**	**0.2**
Area e UCRS % of Total	9.0 92%	0.7 7%	<0.1 <1%	<0.1 <1%	**9.9**	**9.9**
Area e RGA % of Total	21.2 17%	2.8 2%	8.4 7%	91.5 74%	**124**	**124**
Area e McN % of Total	18.3 93%	1.3 7%	<0.1 <1%	0.1 <1%	**19.8**	**19.8**
Area f UCRS % of Total	2.0 92%	<0.1 4%	<0.1 <1%	<0.1 4%	**2.2**	**2.2**
Area f RGA % of Total	16.4 22%	2.1 3%	4.7 6%	50.9 69%	**74.0**	**74.0**
Area f McN % of Total	0.2 98%	<0.1 2%	<0.1 <1%	<0.1 <1%	**0.2**	**0.2**
Area g UCRS % of Total	4.0 93%	0.3 7%	<0.1 <1%	<0.1 <1%	**4.3**	**4.3**
Area g RGA % of Total	5.2 91%	0.4 7%	<0.1 <1%	<0.1 <1%	**5.7**	**44,600**
Area g McN % of Total	0.8 99%	<0.1 <1%	<0.1 <1%	<0.1 <1%	**0.8**	**0.8**
Area h UCRS % of Total	2.1 97%	<0.1 3%	<0.1 <1%	<0.1 <1%	**2.2**	**2.2**
Area h RGA % of Total	6.7 93%	0.3 5%	<0.1 <1%	0.2 2%	**7.2**	**7.2**
Area h McN % of Total	0.4 100%	<0.1 <1%	<0.1 <1%	<0.1 <1%	**0.4**	**0.4**
Area i UCRS % of Total	14.2 91%	0.8 5%	<0.1 <1%	1.0 6%	**15.6**	**38,300**
Area i RGA % of Total	26.1 70%	2.4 7%	0.7 2%	8.0 21%	**37.3**	**37.3**
Area i McN % of Total	2.8 91%	0.3 9%	<0.1 <1%	<0.1 <1%	**3.1**	**3.1**

Exhibit 5.12 (continued)

Location	Direct Ingestion of Groundwater	Dermal Contact while Showering	Inhalation of Vapors while Showering	Inhalation of Vapors during Household Use	Location Total without lead	Location Total with lead[2]
Area j UCRS % of Total	NA NA	NA NA	NA NA	NA NA	**NA**	**NA**
Area j RGA % of Total	8.1 97%	0.3 3%	<0.1 <1%	<0.1 <1%	**8.4**	**8.4**
Area j McN % of Total	27.5 99%	0.2 <1%	<0.1 <1%	<0.1 <1%	**27.7**	**27.7**
Area k Terrace [3] % of Total	69.7 76%	2.2 2%	1.6 2%	17.6 19%	**91.2**	**102,000**
Area l UCRS % of Total	418 14%	57.7 2%	221 7%	2,400 78%	**3,090**	**34,200**
Area l RGA % of Total	198 13%	26.1 2%	105 7%	1,140 78%	**1,470**	**34,500**
Area l McN % of Total	20.4 38%	1.9 3%	2.7 5%	29.0 54%	**54.0**	**54.0**
Area m UCRS % of Total	19.3 86%	1.2 5%	0.1 <1%	1.6 7%	**22.4**	**34,400**
Area m RGA % of Total	31.2 36%	3.3 4%	4.5 5%	48.3 55%	**87.3**	**34,100**
Area m McN % of Total	15.8 94%	0.8 5%	<0.1 <1%	<0.1 <1%	**16.7**	**16.7**
Area n UCRS % of Total	328 14%	44.0 2%	168 7%	1,830 77%	**2,370**	**34,600**
Area n RGA % of Total	124 15%	15.6 2%	56.7 7%	616 76%	**812**	**33,800**
Area n McN % of Total	24.8 64%	1.9 5%	1.0 3%	10.9 28%	**38.6**	**38.6**

Notes: NA indicates that there were no data for the pathway or area.

[1] Current convention is to use one significant digit for presentation of hazard indices. Three significant digits are used here when the hazard index 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's value is greater than 0.1, allows the reader to sum the route values and check the location total.

[2] The very large values are the result of the retention of lead as a COPC at a value only slightly greater than the background concentration and the use of a provisional reference dose provided in comments by KDEP.

[3] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porter's Creek Clay only.

Exhibit 5.13. Driving contaminants' summary for direct contact exposure routes for the child resident scenario - systemic toxicity

Location	Driving Contaminants Over Direct Contact Exposure Routes	Location Total[1]
Area a UCRS	TCE (100%)	7,000
Area a RGA	TCE (100%)	38,800
Area a McN	NA	NA
Area b UCRS	TCE (97%)	2,460
Area b RGA	TCE (80%); *cis*-1,2-DCE	262
Area b McN	TCE (73%); Antimony (27%)	69.5
Area c UCRS	Chloroform (27%); Iron (19%); Vanadium (16%); Benzene (11%); TCE (10%)	2.3
Area c RGA	TCE (80%); Chromium (11%)	43.9
Area c McN	NA	NA
Area d UCRS	TCE (86%)	496
Area d RGA	TCE (91%);	78.8
Area d McN	HI<1	0.2
Area e UCRS	Vanadium (51%); Chromium (11%); Iron (10%)	9.9
Area e RGA	TCE (95%)	124
Area e McN	Vanadium (25%); Iron (17%); Arsenic (16%); Cadmium (16%); Chromium (15%)	19.8
Area f UCRS	Iron (41%); Vanadium (22%); Aluminum (21%)	2.2
Area f RGA	TCE (86%)	74.0
Area f McN	HI<1	0.2
Area g UCRS	Chromium (50%); Vanadium (26%); Manganese (20%)	4.3
Area g RGA	Chromium (33%); Cadmium (32%); Iron (12%); Arsenic (10%)	5.7
Area g McN	HI<1	0.8
Area h UCRS	Nickel (51%); Vanadium (20%); Iron (15%)	2.2
Area h RGA	Chromium (46%); Iron (23%); Vanadium (10%)	7.2
Area h McN	HI<1	0.4
Area i UCRS	Vanadium (20%); Antimony (16%); Manganese (12%)	15.6
Area i RGA	Antimony (40%); Acrylonitrile (22%); Chromium (17%)	37.3
Area i McN	Vanadium (65%); Manganese (35%)	3.1
Area j UCRS	NA	NA
Area j RGA	Manganese (42%); Molybdenum (20%); Iron (12%); Vanadium (12%); Arsenic (11%)	8.4
Area j McN	Arsenic (68%); Manganese (16%); Molybdenum (15%)	27.7
Area k Terrace[2]	Iron (41%); Manganese (20%); Antimony (11%)	91.2
Area l UCRS	TCE (91%)	3,090
Area l RGA	TCE (80%)	1,470
Area l McN	TCE (69%); Antimony (31%)	54.0
Area m UCRS	Antimony (46%); Vanadium (11%)	22.4
Area m RGA	TCE (60%); Antimony (17%); Acrylonitrile (10%)	87.3
Area m McN	Iron (40%); Cadmium (17%); Chromium (12%); Vanadium (10%)	16.7
Area n UCRS	TCE (90%)	2,370
Area n RGA	TCE (70%); Carbon tetrachloride (17%)	812
Area n McN	Antimony (38%); TCE (36%)	38.6

Notes NA indicates that there were no data for that route or area.
HI<1 indicates that total scenario hazard index is less than 1; therefore, COCs are not listed.
COCs contributing more than 10% of total HI are listed. Percentages rounded to nearest whole number.

[1] Totals are without lead as a COPC. The total HIs with lead are in Exhibit 5.10.
[2] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porter's Creek Clay only.

Exhibit 5.14. Biota exposure route summary for the child resident – systemic toxicity[1]

Parameter	Consumption of Vegetable	Consumption of Beef	Consumption of Milk	Consumption of Chicken	Consumption of Turkey	Consumption of Pork	Consumption of Eggs
	Area a UCRS (Direct Route Total HI = 7,000)[2]						
Total HI	1,160	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC				TCE (99%)			
	Area a RGA (Direct Route Total HI = 38,800)[2]						
Total HI	6,380	0.1	0.3	<0.1	<0.1	<0.1	<0.1
Driving COC				TCE (100%)			
	Area a McNairy Formation (Direct Route Total HI = NA)[2]						
Total HI	NA	NA	NA	NA	NA	NA	NA
Driving COC				NA			
	Area b UCRS (Direct Route Total HI = 2,460)[2]						
Total HI	413	<0.1	0.6	<0.1	<0.1	<0.1	<0.0
Driving COC				TCE (95%)			
	Area b RGA (Direct Route Total HI = 262)[2]						
Total HI	49.2	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC			TCE (68%); *cis*-1,2-Dichloroethene (16%)				
	Area b McNairy Formation (Direct Route Total HI = 69.5)[2]						
Total HI	20.6	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC				Antimony (60%); TCE (40%)			
	Area c UCRS (Direct Route Total HI = 2.3)[2]						
Total HI	0.8	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC				Iron (36%); Vanadium (24%); Chloroform (15%)			
	Area c RGA (Direct Route Total HI = 43.9)[2]						
Total HI	10.9	0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC				TCE (52%); Chromium (28%); Iron (10%)			
	Area c McNairy Formation (Direct Route Total HI = NA)[2]						
Total HI	NA	NA	NA	NA	NA	NA	NA
Driving COC				NA			
	Area d UCRS (Direct Route Total HI = 496)[2]						
Total HI	98.0	0.7	0.1	0.2	0.7	<0.1	<0.1
Driving COC				TCE (69%); Iron (13%); Manganese (10%)			
	Area d RGA (Direct Route Total HI = 78.8)[2]						
Total HI	15.0	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC				TCE (79%);			
	Area d McNairy Formation (Direct Route Total HI = 0.2)[2]						
Total HI	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC				None[3]			

Exhibit 5.14 (continued)

Parameter	Consumption of Vegetable	Consumption of Beef	Consumption of Milk	Consumption of Chicken	Consumption of Turkey	Consumption of Pork	Consumption of Eggs
	Area e UCRS (Direct Route Total HI = 9.9)[2]						
Total HI	5.8	<0.1	0.2	<0.1	<0.1	<0.1	<0.1
Driving COC	Vanadium (48%); Iron (12%); Chromium (11%); Nickel (10%)						
	Area e RGA (Direct Route Total HI = 124)[2]						
Total HI	23.0	<0.1	<0.1	<0.1	0.1	<0.1	<0.1
Driving COC	TCE (83%)						
	Area e McNairy Formation (Direct Route Total HI = 19.8)[2]						
Total HI	11.4	0.2	0.1	<0.1	0.2	<0.1	<0.1
Driving COC	Vanadium (24%); Iron (21%); Arsenic (19%); Chromium (16%); Cadmium (12%)						
	Area f UCRS (Direct Route Total HI = 2.2)[2]						
Total HI	1.3	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC	Iron (48%); Aluminum (22%); Vanadium (20%)						
	Area f RGA (Direct Route Total HI = 74.0)[2]						
Total HI	15.9	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC	TCE (65%); Cadmium (12%)						
	Area f McNairy Formation (Direct Route Total HI = 0.2)[2]						
Total HI	0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC	None[3]						
	Area g UCRS (Direct Route Total HI = 4.3)[2]						
Total HI	2.3	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC	Chromium (59%); Vanadium (28%)						
	Area g RGA (Direct Route Total HI = 5.7)[2]						
Total HI	3.2	<0.1	0.2	<0.1	<0.1	<0.1	<0.1
Driving COC	Cadmium (25%); Iron (16%); Nickel (16%); Arsenic (12%)						
	Area g McNairy Formation (Direct Route Total HI = 0.8)[2]						
Total HI	0.7	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC	None[3]						
	Area h UCRS (Direct Route Total HI = 2.2)[2]						
Total HI	1.5	<0.1	0.5	<0.1	<0.1	<.01	<0.1
Driving COC	Nickel (67%); Vanadium (13%); Iron (13%)						
	Area h RGA (Direct Route Total HI = 7.2)[2]						
Total HI	4.3	0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC	Chromium (47%); Iron (28%)						
	Area h McNairy Formation (Direct Route Total HI = 0.4)[2]						
Total HI	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC	None[3]						

Exhibit 5.14 (continued)

Parameter	Consumption of Vegetable	Consumption of Beef	Consumption of Milk	Consumption of Chicken	Consumption of Turkey	Consumption of Pork	Consumption of Eggs
	Area i UCRS (Direct Route Total HI = 15.6)[2]						
Total HI	8.3	0.1	0.1	<0.1	0.1	<0.1	<0.1
Driving COC	Vanadium (23%); Antimony (19%); Arsenic (12%); Iron (11%);						
	Area i RGA (Direct Route Total HI = 37.3)[2]						
Total HI	25.1	0.2	0.5	<0.1	<0.1	<0.1	<0.1
Driving COC	Antimony (37%); Acrylonitrile (28%); Chromium (15%);						
	Area i McNairy Formation (Direct Route Total HI = 3.1)[2]						
Total HI	1.5	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC	Vanadium (81%); Manganese (19%)						
	Area j UCRS (Direct Route Total HI = NA)[2]						
Total HI	NA	NA	NA	NA	NA	NA	NA
Driving COC	NA						
	Area j RGA (Direct Route Total HI = 8.4)[2]						
Total HI	4.3	<0.1	<0.1	<0.1	0.1	<0.1	<0.1
Driving COC	Molybdenum (33%); Manganese (19%); Iron (17%); Arsenic (14%); Vanadium (13%)						
	Area j McNairy Formation (Direct Route Total HI = 27.7)[2]						
Total HI	17.7	<0.1	0.2	<0.1	0.2	<0.1	<0.1
Driving COC	Arsenic (72%); Molybdenum (21%)						
	Area k Terrace[4] (Direct Route Total HI = 91.2)[2]						
Total HI	46.6	1.5	0.2	0.3	1.4	0.1	0.1
Driving COC	Iron (55%); Antimony (12%); 1,2-Dichloroethene (10%)						
	Area l UCRS (Direct Route Total HI = 3,090)[2]						
Total HI	546	<0.1	0.3	<0.1	<0.1	<0.1	<.01
Driving COC	TCE (84%); *cis*-1,2-Dichloroethene (11%)						
	Area l RGA (Direct Route Total HI = 1,470)[2]						
Total HI	276	<0.1	<0.1	<0.1	0.1	<0.1	<0.1
Driving COC	TCE (70%); *trans*-1,2-Dichloroethene (12%)						
	Area l McNairy Formation (Direct Route Total HI = 54.0)[2]						
Total HI	16.9	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Driving COC	Antimony (63%); TCE (36%)						
	Area m UCRS (Direct Route Total HI = 22.4)[2]						
Total HI	12.5	<0.1	0.1	<0.1	0.1	<0.1	<0.1
Driving COC	Antimony (52%); Vanadium (11%)						
	Area am RGA (Direct Route Total HI = 87.3)[2]						
Total HI	32.6	0.2	0.5	<0.1	<0.1	<0.1	<0.1
Driving COC	Antimony (27%); TCE (24%); Acrylonitrile (22%)						

Exhibit 5.14 (continued)

Parameter	Consumption of Vegetable	Consumption of Beef	Consumption of Milk	Consumption of Chicken	Consumption of Turkey	Consumption of Pork	Consumption of Eggs
	Area m McNairy Formation (Direct Route Total HI = 16.7)[2]						
Total HI	9.8	0.3	0.1	<0.1	0.3	<0.1	<0.1
Driving COC	Iron (48%); Chromium (12%); Cadmium (11%)						
	Area n UCRS (Direct Route Total HI = 2,370)[2]						
Total HI	423	<0.1	0.2	<0.1	0.1	<0.1	<0.1
Driving COC	TCE (82%); *cis*-1,2-Dichloroethene (11%)						
	Area n RGA (Direct Route Total HI = 812)[2]						
Total HI	171	0.2	0.5	<0.1	0.1	<0.1	<0.1
Driving COC	TCE (54%); *trans*-1,2-Dichloroethene (14%)						
	Area n McNairy Formation (Direct Route Total HI = 38.6)[2]						
Total HI	17.2	0.1	0.1	<0.1	0.1	<0.1	<0.1
Driving COC	Antimony (53%); TCE (13%)						

Notes: NA indicates that there were no data for the exposure route or area.

[1] All Total HI values do not include contribution from lead as a COPC.
[2] Direct Route Total HI is from Exhibit 5.12.
[3] No COCs because Total HI < 1.0
[4] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porters Creek Clay only.

RGA and Area k have a total HI that exceeds 1. Driving contaminants are similar to those for direct contact although cadmium gains in importance for some areas. Similar to the results for the direct contact exposure routes, acrylonitrile appears as a driving contaminant in Area i (RGA).

5.4.3.2 Excess lifetime cancer risk

Exhibit 5.15 summarizes the ELCRs for direct contact exposure routes for the rural resident over all areas for the unfiltered data set. As shown in this exhibit, the total ELCR is greater than 1×10^{-6} for all areas except Areas a, c, f, and i for the McNairy Formation and Area j for the UCRS. Note that total ELCR is less than 1×10^{-6} for Areas a and c (McNairy Formation) and Area j (UCRS) because information was not sufficient to assess these area/depth classification combinations. As with total HI, the driving exposure route is inhalation of vapors either during household use or while showering when total ELCR is greatly in excess of 1×10^{-6} and ingestion when total ELCR is closer to this benchmark value. Also similar to results for total HI, contribution from dermal contact is minor compared to that for other exposure routes.

Exhibit 5.16 summarizes the contaminants contributing more than 10% of the total ELCR for the rural resident over all areas. As shown in this exhibit, the driving contaminants for areas associated with the TCE plumes are TCE and its breakdown products. However, outside the plumes, including the UCRS and McNairy for Areas e and f, the driving contaminants are ^{222}Rn and the inorganic chemicals arsenic and beryllium. Of note is the identification of acrylonitrile as a driving contaminant in Area i (RGA).

Exhibit 5.17 summarizes the ELCRs and driving contaminants for the biota consumption exposure routes for the rural resident. As shown there, all areas except Areas a, c, d, f, and i for the McNairy Formation and Areas h and j for the UCRS have total ELCRs that exceed 1×10^{-6}. Routes with ELCRs greater than 1×10^{-6} are consumption of vegetables, consumption of beef, consumption of milk, consumption of chicken, and consumption of turkey. Contaminants gaining in importance when biota consumption is considered are the radionuclides ^{99}Tc and ^{226}Ra.

5.5 RISK CHARACTERIZATION FOR FUTURE CONCENTRATIONS

Risk posed by continued migration of contaminants from sources at the PGDP were characterized using the contaminant concentrations derived from fate and transport modeling. This modeling is discussed in the Data Summary Report located in Appendix B of the GWOU FS. The results of the risk characterization are depicted in Figs. 5.1 through 5.20 (see Attachment 2). The information used to create these figures is presented in Attachment 7 of this volume.

Consistent with the fate and transport modeling, risk was characterized for four potential points of exposure. For sources to the Northwest and Northeast Plumes, these exposure points were at the security fence to the north of the industrialized area of the PGDP, at the northern property line of the PGDP, at Little Bayou Creek, and at the Ohio River. For sources to the Southwest Plume, the only exposure point modeled was at the security fence to the west of the industrialized area of the PGDP. Other exposure points were not modeled for the Southwest Plume because these points match those used for sources to the Northwest and Northeast Plume. For all exposure points, residential ELCR and systemic toxicity due to household use of contaminated groundwater drawn from the RGA were estimated.

Figures 5.1 through 5.4 depict the systemic toxicity and ELCR estimates for the fence line point of exposure for sources to the Northwest and Northeast Plumes. As shown in Fig. 5.1, total HI from the TCE dense non-aqueous phase liquid (DNAPL) (i.e., associated with releases at the C-400 Building) is equal to approximately 1,000 up to about year 1000 from present. After year 1000 from present, total HI posed by

Exhibit 5.15. Direct contact exposure route summary for the resident – excess lifetime cancer risk[1]

Location	Direct ingestion of groundwater	Dermal Contact while Showering	Inhalation of Vapors while Showering	Inhalation of Vapors during Household Use	Location Total
Area a UCRS % of Total	1.7×10^{-2} 29%	3.2×10^{-3} 5%	3.7×10^{-3} 6%	3.6×10^{-2} 60%	**5.9×10^{-2}**
Area a RGA % of Total	9.2×10^{-2} 29%	1.7×10^{-2} 5%	1.9×10^{-2} 6%	1.8×10^{-1} 60%	**2.9×10^{-1}**
Area a McN % of Total	NA NA	NA NA	NA NA	NA NA	**NA**
Area b UCRS % of Total	9.8×10^{-3} 35%	1.1×10^{-3} 4%	2.0×10^{-3} 7%	1.5×10^{-2} 53%	**2.7×10^{-2}**
Area b RGA % of Total	4.2×10^{-2} 59%	8.9×10^{-4} 1%	2.6×10^{-3} 4%	2.6×10^{-2} 36%	**7.0×10^{-2}**
Area b McN % of Total	1.3×10^{-4} 30%	2.3×10^{-5} 5%	2.4×10^{-5} 6%	2.6×10^{-4} 60%	**4.4×10^{-4}**
Area c UCRS % of Total	4.7×10^{-6} 5%	$<1 \times 10^{-6}$ <1%	6.8×10^{-6} 8%	7.4×10^{-5} 86%	**8.6×10^{-5}**
Area c RGA % of Total	2.0×10^{-4} 8%	1.7×10^{-5} <1%	1.1×10^{-3} 44%	1.1×10^{-3} 47%	**2.4×10^{-3}**
Area c McN % of Total	NA NA	NA NA	NA NA	NA NA	**NA**
Area d UCRS % of Total	1.5×10^{-3} 26%	2.2×10^{-4} 4%	6.2×10^{-4} 10%	3.6×10^{-3} 60%	**6.0×10^{-3}**
Area d RGA % of Total	3.4×10^{-4} 25%	3.6×10^{-5} 3%	5.3×10^{-4} 40%	4.3×10^{-4} 32%	**1.3×10^{-3}**
Area d McN % of Total	$<1 \times 10^{-6}$ 29%	$<1 \times 10^{-6}$ 5%	$<1 \times 10^{-6}$ 6%	$<1 \times 10^{-6}$ 60%	**1.2×10^{-6}**
Area e UCRS % of Total	9.3×10^{-5} 43%	$<1 \times 10^{-6}$ <1%	1.1×10^{-4} 51%	1.2×10^{-5} 6%	**2.1×10^{-4}**
Area e RGA % of Total	8.7×10^{-4} 44%	1.4×10^{-4} 7%	3.2×10^{-4} 16%	6.4×10^{-4} 33%	**2.0×10^{-3}**
Area e McN % of Total	1.2×10^{-3} 78%	1.4×10^{-4} 9%	1.8×10^{-4} 11%	2.0×10^{-5} 1%	**1.6×10^{-3}**
Area f UCRS % of Total	1.6×10^{-6} <1%	$<1 \times 10^{-6}$ <1%	3.1×10^{-4} 90%	3.4×10^{-5} 10%	**3.5×10^{-4}**
Area f RGA % of Total	3.1×10^{-4} 19%	3.2×10^{-5} 2%	4.3×10^{-4} 26%	8.7×10^{-4} 53%	**1.6×10^{-3}**
Area f McN % of Total	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	**$<1 \times 10^{-6}$**
Area g UCRS % of Total	1.0×10^{-5} 2%	$<1 \times 10^{-6}$ <1%	4.0×10^{-4} 88%	4.3×10^{-5} 9%	**4.6×10^{-4}**
Area g RGA % of Total	7.7×10^{-5} 14%	$<1 \times 10^{-6}$ <1%	4.2×10^{-4} 77%	4.5×10^{-5} 8%	**5.4×10^{-4}**
Area g McN % of Total	8.1×10^{-5} 100%	$<1 \times 10^{-6}$ <1%	$<1 \times 10^{-6}$ <1%	$<1 \times 10^{-6}$ <1%	**8.2×10^{-5}**
Area h UCRS % of Total	$<1 \times 10^{-6}$ <1%	$<1 \times 10^{-6}$ <1%	1.8×10^{-4} 90%	1.9×10^{-5} 10%	**2.0×10^{-4}**
Area h RGA % of Total	8.0×10^{-5} 25%	$<1 \times 10^{-6}$ <1%	2.2×10^{-4} 68%	2.4×10^{-5} 7%	**3.3×10^{-4}**

Exhibit 5.15 (continued)

Location	Direct ingestion of groundwater	Dermal Contact while Showering	Inhalation of Vapors while Showering	Inhalation of Vapors during Household Use	Location Total
Area h McN % of Total	7.6×10^{-6} 4%	$<1 \times 10^{-6}$ <1%	1.7×10^{-4} 87%	1.8×10^{-5} 9%	**2.0×10^{-4}**
Area i UCRS % of Total	2.1×10^{-4} 36%	1.1×10^{-6} <1%	3.1×10^{-4} 54%	5.7×10^{-5} 10%	**5.8×10^{-4}**
Area i RGA % of Total	8.9×10^{-4} 54%	1.2×10^{-4} 7%	4.0×10^{-4} 24%	2.6×10^{-4} 15%	**1.7×10^{-3}**
Area i McN % of Total	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	$<1 \times 10^{-6}$ NA	**$<1 \times 10^{-6}$**
Area j UCRS % of Total	NA NA	NA NA	NA NA	NA NA	**NA**
Area j RGA % of Total	1.2×10^{-4} 100%	$<1 \times 10^{-6}$ <1%	$<1 \times 10^{-6}$ <1%	$<1 \times 10^{-6}$ <1%	**1.2×10^{-4}**
Area j McN % of Total	2.4×10^{-3} 100%	1.0×10^{-5} <1%	$<1 \times 10^{-6}$ <1%	$<1 \times 10^{-6}$ <1%	**2.4×10^{-3}**
Area k Terrace [2] % of Total	1.4×10^{-3} 28%	9.6×10^{-5} 2%	8.4×10^{-4} 16%	2.7×10^{-3} 54%	**5.1×10^{-3}**
Area l UCRS % of Total	1.7×10^{-1} 54%	3.6×10^{-3} 1%	1.4×10^{-2} 4%	1.3×10^{-1} 41%	**2.9×10^{-1}**
Area l RGA % of Total	1.6×10^{-1} 57%	2.9×10^{-3} <1%	1.1×10^{-2} 4%	1.1×10^{-1} 38%	**2.6×10^{-1}**
Area l McN % of Total	9.6×10^{-5} 30%	1.7×10^{-5} 5%	1.8×10^{-5} 6%	1.9×10^{-4} 60%	**3.3×10^{-4}**
Area m UCRS % of Total	1.4×10^{-4} 30%	$<1 \times 10^{-6}$ <1%	2.3×10^{-4} 49%	9.6×10^{-5} 21%	**4.7×10^{-4}**
Area m RGA % of Total	1.2×10^{-3} 29%	1.3×10^{-4} 3%	5.0×10^{-4} 12%	2.3×10^{-3} 56%	**4.1×10^{-3}**
Area m McN % of Total	7.2×10^{-4} 73%	9.5×10^{-5} 10%	1.6×10^{-4} 16%	1.8×10^{-5} 2%	**9.9×10^{-4}**
Area n UCRS % of Total	1.7×10^{-1} 55%	3.3×10^{-3} <1%	1.3×10^{-2} 4%	1.3×10^{-1} 40%	**2.9×10^{-1}**
Area n RGA % of Total	6.5×10^{-2} 55%	1.4×10^{-3} 1%	4.7×10^{-3} 4%	4.7×10^{-2} 40%	**1.1×10^{-1}**
Area n McN % of Total	7.3×10^{-4} 70%	1.0×10^{-4} 10%	1.3×10^{-4} 12%	8.6×10^{-5} 8%	**1.1×10^{-3}**

Notes: NA indicates that there were no data for the pathway or area.
Values for ELCR greater than 1×10^{-2} fall outside the calculation bounds in EPA 1989a and are approximate values.

[1] Current convention is to use one significant digit for presentation of ELCRs. Two significant digits are used here when to enable the reader to match the numbers reported in the exhibit with those in its associated risk characterization table. Additionally, use of two significant digits allows the reader to sum the route values and check the location total.

[2] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porter's Creek Clay only.

Exhibit 5.16. Driving contaminants' summary for direct contact exposure routes for the resident scenario – excess lifetime cancer risk

Location	Driving Contaminants Over Direct Contact Exposure Routes	Location Total
Area a UCRS	TCE (99%)	**5.9×10^{-2}**
Area a RGA	TCE (99%)	**2.9×10^{-1}**
Area a McN	NA	**NA**
Area b UCRS	TCE (74%); Vinyl chloride (20%)	**2.7×10^{-2}**
Area b RGA	Vinyl chloride (94%)	**7.0×10^{-2}**
Area b McN	TCE (99%)	**4.4×10^{-4}**
Area c UCRS	Chloroform (92%)	**8.6×10^{-5}**
Area c RGA	^{222}Rn (44%); 1,1-DCE (41%); TCE (13%)	**2.4×10^{-3}**
Area c McN	NA	**NA**
Area d UCRS	TCE (61%); 1,1-DCE (27%)	**6.0×10^{-3}**
Area d RGA	TCE (46%); ^{222}Rn (41%)	**1.3×10^{-3}**
Area d McN	TCE (100%)	**1.2×10^{-6}**
Area e UCRS	^{222}Rn (56%); Arsenic (43%)	**2.1×10^{-4}**
Area e RGA	TCE (52%); Beryllium (29%); ^{222}Rn (15%)	**2.0×10^{-3}**
Area e McN	Beryllium (61%); Arsenic (26%); ^{222}Rn (13%)	**1.6×10^{-3}**
Area f UCRS	^{222}Rn (99%)	**3.5×10^{-4}**
Area f RGA	1,1-DCE (37%); TCE (34%); ^{222}Rn (24%)	**1.6×10^{-3}**
Area f McN	ELCR$<1 \times 10^{-6}$	**$<1 \times 10^{-6}$**
Area g UCRS	^{222}Rn (98%)	**4.6×10^{-4}**
Area g RGA	^{222}Rn (86%); Arsenic (13%)	**5.4×10^{-4}**
Area g McN	Arsenic (87%)	**8.2×10^{-5}**
Area h UCRS	^{222}Rn (100%)	**2.0×10^{-4}**
Area h RGA	^{222}Rn (75%); Arsenic (24%)	**3.3×10^{-4}**
Area h McN	^{222}Rn (96%)	**2.0×10^{-4}**
Area i UCRS	^{222}Rn (60%); Arsenic (33%)	**5.8×10^{-4}**
Area i RGA	Beryllium (46%); ^{222}Rn (25%); Acrylonitrile (18%)	**1.7×10^{-3}**
Area i McN	ELCR$<1 \times 10^{-6}$	**$<1 \times 10^{-6}$**
Area j UCRS	NA	**NA**
Area j RGA	Arsenic (100%)	**1.2×10^{-4}**
Area j McN	Arsenic (100%)	**2.4×10^{-3}**
Area k Terrace[1]	1,1-DCE (56%); Vinyl chloride (18%); ^{222}Rn (13%); Beryllium (11%)	**5.1×10^{-3}**
Area l UCRS	Vinyl chloride (87%)	**2.9×10^{-1}**
Area l RGA	Vinyl chloride (93%)	**2.6×10^{-1}**
Area l McN	TCE (99%)	**3.3×10^{-4}**
Area m UCRS	^{222}Rn (53%); Arsenic (25%); Chloroform (15%)	**4.7×10^{-4}**
Area m RGA	1,1-DCE (53%); Beryllium (17%); TCE (10%)	**4.1×10^{-3}**
Area m McN	Beryllium (65%); ^{222}Rn (18%); Arsenic (16%)	**9.9×10^{-4}**
Area n UCRS	Vinyl chloride (88%)	**2.9×10^{-1}**
Area n RGA	Vinyl chloride (87%)	**1.1×10^{-1}**
Area n McN	Beryllium (62%); Arsenic (13%); ^{222}Rn (13%); TCE (12%)	**1.1×10^{-3}**

Notes NA indicates that there were no data for that route or area.

ELCR$<1 \times 10^{-6}$ indicates that total ELCR is less than 1×10^{-6}; therefore, COCs are not listed.

COCs contributing more than 10% of total ELCR are listed. Percentages rounded to nearest whole number. Values for ELCR greater than 1×10^{-2} fall outside the calculation bounds in EPA 1989a and are approximate values.

[1] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porter's Creek Clay only.

Exhibit 5.17. Biota exposure route summary for the resident – excess lifetime cancer risk[1]

Parameter	Consumption of Vegetable	Consumption of Beef	Consumption of Milk	Consumption of Chicken	Consumption of Turkey	Consumption of Pork	Consumption of Eggs
	Area a UCRS (Direct Route Total ELCR = 5.9×10^{-2})[2]						
Total ELCR	1.9×10^{-2}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				TCE (95%)			
	Area a RGA (Direct Route Total ELCR = 2.9×10^{-1})[2]						
Total ELCR	1.0×10^{-1}	2.7×10^{-6}	3.5×10^{-6}	$<1 \times 10^{-6}$	3.3×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				TCE (91%)			
	Area a McNairy Formation (Direct Route Total ELCR = NA)[2]						
Total ELCR	NA	NA	NA	NA	NA	NA	NA
Driving COC				NA			
	Area b UCRS (Direct Route Total ELCR = 2.7×10^{-2})[2]						
Total ELCR	1.7×10^{-2}	2.2×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	2.0×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				Vinyl chloride (46%); TCE (37%); ^{99}Tc (15%)			
	Area b RGA (Direct Route Total ELCR = 7.0×10^{-2})[2]						
Total ELCR	9.9×10^{-2}	1.3×10^{-5}	1.4×10^{-5}	$<1 \times 10^{-6}$	2.1×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				Vinyl chloride (94%)			
	Area b McNairy Formation (Direct Route Total ELCR = 4.4×10^{-4})[2]						
Total ELCR	1.5×10^{-3}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{99}Tc (91%)			
	Area c UCRS (Direct Route Total ELCR = 8.6×10^{-5})[2]						
Total ELCR	6.5×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{99}Tc (100%)			
	Area c RGA (Direct Route Total ELCR = 2.4×10^{-3})[2]						
Total ELCR	2.8×10^{-3}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{99}Tc (90%)			
	Area c McNairy Formation (Direct Route Total ELCR = NA)[2]						
Total ELCR	NA	NA	NA	NA	NA	NA	NA
Driving COC				NA			
	Area d UCRS (Direct Route Total ELCR = 6.0×10^{-3})[2]						
Total ELCR	2.9×10^{-3}	1.1×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	1.8×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{99}Tc (46%); TCE (38%)			
	Area d RGA (Direct Route Total ELCR = 1.3×10^{-3})[2]						
Total ELCR	5.2×10^{-4}	2.2×10^{-6}	1.6×10^{-6}	1.3×10^{-6}	5.4×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{99}Tc (43%); TCE (35%); Arsenic (12%)			

Exhibit 5.17 (continued)

Parameter	Consumption of Vegetable	Consumption of Beef	Consumption of Milk	Consumption of Chicken	Consumption of Turkey	Consumption of Pork	Consumption of Eggs
	Area d McNairy Formation (Direct Route Total ELCR = 1.2×10^{-6})[2]						
Total ELCR	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				None[3]			
	Area e UCRS (Direct Route Total ELCR = 2.1×10^{-4})[2]						
Total ELCR	5.4×10^{-5}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				Arsenic (99%)			
	Area e RGA (Direct Route Total ELCR = 2.0×10^{-3})[2]						
Total ELCR	5.7×10^{-3}	1.2×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{99}Tc (89%)			
	Area e McNairy Formation (Direct Route Total ELCR = 1.6×10^{-3})[2]						
Total ELCR	8.0×10^{-4}	3.2×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				Beryllium (59%); Arsenic (30%); ^{99}Tc (11%)			
	Area f UCRS (Direct Route Total ELCR =3.5×10^{-4})[2]						
Total ELCR	4.0×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{99}Tc (100%)			
	Area f RGA (Direct Route Total ELCR = 1.6×10^{-3})[2]						
Total ELCR	4.5×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				TCE (37%); ^{99}Tc (29%); 1,1-Dichloroethene (23%)			
	Area f McNairy Formation (Direct Route Total ELCR = $<1 \times 10^{-6}$)[2]						
Total ELCR	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				None[3]			
	Area g UCRS (Direct Route Total ELCR = 4.6×10^{-4})[2]						
Total ELCR	2.8×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{99}Tc (98%)			
	Area g RGA (Direct Route Total ELCR = 5.4×10^{-4})[2]						
Total ELCR	3.0×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{99}Tc (85%); Arsenic (14%)			
	Area g McNairy Formation (Direct Route Total ELCR = 8.2×10^{-5})[2]						
Total ELCR	4.7×10^{-5}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				Arsenic (87%)			
	Area h UCRS (Direct Route Total ELCR = 2.0×10^{-4})[2]						
Total ELCR	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				None[3]			

Exhibit 5.17 (continued)

Parameter	Consumption of Vegetable	Consumption of Beef	Consumption of Milk	Consumption of Chicken	Consumption of Turkey	Consumption of Pork	Consumption of Eggs
	Area h RGA (Direct Route Total ELCR = 3.3×10^{-4})[2]						
Total ELCR	2.5×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{99}Tc (82%); Arsenic (18%)			
	Area h McNairy Formation (Direct Route Total ELCR = 2.0×10^{-4})[2]						
Total ELCR	4.2×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{226}Ra (88%); ^{230}Th (12%)			
	Area i UCRS (Direct Route Total ELCR = 5.8×10^{-4})[2]						
Total ELCR	4.5×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{99}Tc (72%); Arsenic (24%)			
	Area i RGA (Direct Route Total ELCR = 1.7×10^{-3})[2]						
Total ELCR	2.1×10^{-3}	2.6×10^{-6}	2.3×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				Acrylonitrile (45%); ^{99}Tc (30%); Beryllium (18%)			
	Area i McNairy Formation (Direct Route Total ELCR < 1×10^{-6})[2]						
Total ELCR	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				None[3]			
	Area j UCRS (Direct Route Total ELCR = NA)[2]						
Total ELCR	NA	NA	NA	NA	NA	NA	NA
Driving COC				NA			
	Area j RGA (Direct Route Total ELCR = 1.2×10^{-4})[2]						
Total ELCR	7.1×10^{-5}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				Arsenic (100%)			
	Area j McNairy Formation (Direct Route Total ELCR = 2.4×10^{-3})[2]						
Total ELCR	1.4×10^{-3}	9.3×10^{-6}	2.0×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				Arsenic (100%)			
	Area k Terrace[4] (Direct Route Total ELCR = 5.1×10^{-3})[2]						
Total ELCR	2.3×10^{-3}	1.8×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				Vinyl chloride (58%); 1,1-Dichloroethene (22%); Beryllium (12%)			
	Area l UCRS (Direct Route Total ELCR = 2.9×10^{-1})[2]						
Total ELCR	3.6×10^{-1}	2.6×10^{-6}	1.4×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				Vinyl chloride (97%)			
	Area l RGA (Direct Route Total ELCR = 2.6×10^{-1})[2]						
Total ELCR	3.3×10^{-1}	1.1×10^{-5}	1.1×10^{-5}	1.6×10^{-6}	6.5×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				Vinyl chloride (98%)			

Exhibit 5.17 (continued)

Parameter	Consumption of Vegetable	Consumption of Beef	Consumption of Milk	Consumption of Chicken	Consumption of Turkey	Consumption of Pork	Consumption of Eggs
	Area l McNairy Formation (Direct Route Total ELCR = 3.3×10^{-4})[2]						
Total ELCR	1.1×10^{-3}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{99}Tc (92%)			
	Area m UCRS (Direct Route Total ELCR = 4.7×10^{-4})[2]						
Total ELCR	3.5×10^{-4}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{99}Tc (72%); Arsenic (20%)			
	Area m RGA (Direct Route Total ELCR = 4.1×10^{-3})[2]						
Total ELCR	3.3×10^{-3}	2.5×10^{-6}	2.3×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{99}Tc (42%); Acylonitrile (28%); 1,1-Dichloroethene (11%)			
	Area m McNairy Formation (Direct Route Total ELCR = 9.9×10^{-4})[2]						
Total ELCR	4.9×10^{-4}	1.8×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				Beryllium (64%); Arsenic (19%); ^{99}Tc (17%)			
	Area n UCRS (Direct Route Total ELCR = 2.9×10^{-1})[2]						
Total ELCR	3.6×10^{-1}	2.4×10^{-6}	1.4×10^{-6}	$<1 \times 10^{-6}$	1.0×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				Vinyl chloride (97%)			
	Area n RGA (Direct Route Total ELCR = 1.1×10^{-1})[2]						
Total ELCR	1.5×10^{-1}	9.0×10^{-6}	9.8×10^{-6}	1.6×10^{-6}	6.5×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				Vinyl chloride (96%)			
	Area n McNairy Formation (Direct Route Total ELCR = 1.1×10^{-3})[2]						
Total ELCR	8.6×10^{-4}	1.7×10^{-6}	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$
Driving COC				^{99}Tc (49%); Beryllium (37%)			

Notes: NA indicates that there were no data for the exposure route or area.

[1] Total ELCR values greater than 1×10^{-2} fall outside the calculation bounds in EPA 1989a and are estimated values.
[2] Direct Route Total ELCR is from Exhibit 5.15.
[3] No COCs because Total ELCR < 1×10^{-6}.
[4] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porters Creek Clay only.

contributions from the TCE DNAPL falls, but remains above, 1 until about year 7000 from present. Figure 5.1 also shows that total HI from source areas is less than that from the TCE DNAPL at all times, except for a period from year 80 to 140 (peak total HI from source areas = 5,960) and for times after year 2600 (peak total HI = 1,720). Finally, Fig. 5.2 shows that TCE from source areas is the dominant contaminant during the first period when total HI from source areas exceeds that from TCE DNAPL and that chromium is the dominant contaminant in the second period. Figure 5.3 shows that total ELCR from the use of groundwater contaminated by contributions from the TCE DNAPL is near 1×10^{-2} up to about year 1000 from present. After year 1000 from present, total ELCR posed by contributions from TCE DNAPL falls, but remains above, 1×10^{-4} until about year 7000 from present. Total ELCR from the TCE DNAPL source finally falls below 1×10^{-6} about year 7200 from present. Figure 5.3 also shows that total ELCR from source areas is less than that from TCE DNAPL at all times except for a period from year 80 to 140 (peak total ELCR = 8×10^{-2}) and for times after year 4700 (peak total ELCR = 2×10^{-4}) when total ELCR from sources is similar to that from TCE DNAPL. Figure 5.4 shows that TCE and Tc-99 from source areas are the dominant contaminants during the first period when total ELCR from source areas exceeds that from TCE DNAPL and that the uranium isotopes are the dominant contaminants during the second period.

Figures 5.17 through 5.20 depict the systemic toxicity and ELCR estimates for the fence line point of exposure for sources to the Southwest Plume. As shown in Fig. 5.17, total HI from source areas peaks at 1,865 at 110 years from present. Smaller peaks in HI occur at years 20 and 4100 from present. The contaminants driving these peak HIs are TCE; 1,2-DCE; and copper, respectively (Fig. 5.18). Total ELCR from source areas also has three peaks (Fig. 5.19). The years of peak and the total ELCRs at those years are 110 and 2×10^{-2}, 530 and 2×10^{-3}, and 4900 and 2×10^{-2}, respectively. Contaminants driving the total ELCR at these peaks are TCE; 1,1-DCE; and vinyl chloride at the first peak, ^{237}Np at the second peak, and uranium isotopes at the third peak. Technetium-99 also contributes to the first peak, but its relative contribution to total ELCR is markedly less than that from other contaminants.

Figures 5.5 through 5.8 depict the systemic toxicity and ELCR estimates for the property boundary point of exposure for sources to the Northwest and Northeast Plumes. As shown there, results for this point of exposure are similar to those for the fence line point of exposure for sources to these plumes, except that the peak values are lower. For example, as shown in Figs. 5.5 and 5.7, the peak HI and ELCR from TCE DNAPL are approximately 600 and 5×10^{-3}, respectively, for the property boundary point of exposure versus approximately 1,000 and 1×10^{-2}, respectively, for the fence line point of exposure. These results are expected because the modeled distance between the fence line and property boundary points of exposure is only 2,000 ft. (Note that a separate model was not run for this point of exposure for sources to the Southwest Plume because groundwater flow from these sources matches those for the sources to the Northwest and Northeast.)

Figures 5.9 through 5.12 depict the systemic toxicity and ELCR estimates for the Little Bayou Creek point of exposure for sources to the Northwest Plume. (Sources to the Northeast Plume do not contribute to this point of exposure.) These results are similar to those described for the fence line and property boundary points of exposure except risk from contributions from the TCE DNAPL are markedly reduced (peak value HI and ELCR at Little Bayou Creek = 3 and 3×10^{-5} , respectively). However, the first peak from other source areas is still very high and exceeds the HI and ELCR values for the TCE DNAPL from 110 to 210 years from present. This peak, which is primarily due to TCE releases from the source areas, reaches a HI of approximately 2,000 and an ELCR of approximately 1×10^{-2}.

Figures 5.13 through 5.16 depict the systemic toxicity and ELCR estimates for the Ohio River point of exposure for sources to the Northwest and Northeast Plumes. These results are different from those for the other three exposure points because contribution from TCE DNAPL is not significant (i.e., less than

HI = 0.1 and ELCR = 1×10^{-8}) and does not appear on the figures. However, contributions from source areas show patterns that are similar to those described earlier with peak HIs and ELCRs approximately equal to those discussed earlier and with the same contaminant, TCE, being primarily responsible for the size of the earlier peak.

5.6 RISK CHARACTERIZATION FOR LEAD

Unlike the other analytes included in this risk assessment, the risks from exposure to lead were estimated using a biokinetic model and through a comparison of detected concentrations to KDEP and EPA screening values in addition to characterization using an RfD. This procedure was followed to address the uncertainty in the provisional reference dose provided by KDEP, to meet the requirements of the Region 4 EPA in their guidance, and to be consistent with agreements in the Methods Document.

The model used to estimate the importance of lead was EPA's Integrated Exposure Uptake Biokinetic Model for Lead. The complete results of the modeling are in Attachment 5 of this volume. The results of this model indicate that the following area/depth classification combinations have lead concentrations that could lead to unacceptable blood lead concentrations in children.

McNairy Formation: None
RGA: Areas b, d, g, l, m, and n
UCRS: Areas a, d, i, k, l, m, and n

The KDEP and EPA screening values used in comparisons are those used in previous assessments at the PGDP. The KDEP value is 4 µg/L for water. The EPA value is 15 µg/L for water. Exhibit 5.18 presents the comparison between the exposure, minimum detected, and maximum detected concentrations of lead and the screening value, by area, for the unfiltered data. Note that the value used to determine if the screening value is exceeded is the exposure concentration and that the unfiltered background concentrations for the RGA and McNairy Formation are also provided. This exhibit shows that the lead concentrations in groundwater drawn from the RGA and McNairy Formation exceed the screening values from both regulatory agencies.

As shown in Exhibit 5.18, the screening values are exceeded in the following areas:

McNairy Formation: None
RGA: Areas b, d, g, l, m, and n
UCRS: Areas a, d, i, l, m, and n

In addition, the exposure concentration for lead in Area k also exceeds the screening values.

However, Exhibit 5.18 also shows that, in general, lead was infrequently detected even in those areas where the screening values were exceeded and that, for the RGA, the exposure concentration is less than the background concentration in all cases. These results indicate that the apparent unacceptable concentrations are most likely an artifact of the data sets and their summarization.

5.7 IDENTIFICATION OF LAND USE SCENARIOS, PATHWAYS, AND CONTAMINANTS OF CONCERN FOR AREAS

This subsection identifies the land use scenarios of concern, pathways of concern (POCs), and contaminants of concern (COCs) for each area for the assessment of the unfiltered data set. This subsection

Exhibit 5.18. Comparison of representative concentrations[1] of lead (µg/L) against regulatory values for the unfiltered data by area

Location	Frequency of Detection	Exposure Concentration	Minimum Detected Concentration	Maximum Detected Concentration	KDEP Screening Value	Exceed?	EPA Screening Value	Exceed?
UCRS								
Area a	2/4	69	3.69	69.0	4	Yes	15	Yes
Area b	1/150	1.8	1.80	1.80	4	No	15	No
Area c	0/0	NA	ND	ND	4	No	15	No
Area d	7/39	35	3.30	1,380	4	Yes	15	Yes
Area e	0/4	NA	ND	ND	4	No	15	No
Area f	0/1	NA	ND	ND	4	No	15	No
Area g	0/7	NA	ND	ND	4	No	15	No
Area h	0/1	NA	ND	ND	4	No	15	No
Area i	5/37	57	57.0	235	4	Yes	15	Yes
Area j	0/0	NA	ND	ND	4	No	15	No
Area k (Terrace[2])	14/72	153	1.60	1,780	4	Yes	15	Yes
Area l	10/93	47	1.80	1,380	4	Yes	15	Yes
Area m	5/50	51	57.0	235	4	Yes	15	Yes
Area n	15/243	48	1.80	1,380	4	Yes	15	Yes
RGA								
Background[3]		129			4	Yes	15	Yes
Area a	0/9	NA	ND	ND	4	No	15	No
Area b	8/370	41	5.00	432	4	Yes	15	Yes
Area c	0/23	NA	ND	ND	4	No	15	No
Area d	13/88	67	2.40	250	4	Yes	15	Yes
Area e	1/69	NA	60.0	60.0	4	No	15	No
Area f	0/46	NA	ND	ND	4	No	15	No
Area g	4/25	67	51.0	129	4	Yes	15	Yes
Area h	0/3	NA	ND	ND	4	No	15	No
Area i	8/445	NA	4.0	126	4	No	15	No
Area j	0/0	NA	ND	ND	4	No	15	No
Area l	21/490	50	2.40	432	4	Yes	15	Yes
Area m	13/588	51	4.00	129	4	Yes	15	Yes
Area n	34/1078	49	2.40	432	4	Yes	15	Yes
McNairy Formation								
Background[c]		50			4	Yes	15	Yes
Area a	0/0	NA	ND	ND	4	No	15	No
Area b	0/5	NA	ND	ND	4	No	15	No
Area c	0/0	NA	ND	ND	4	No	15	No
Area d	0/11	NA	ND	ND	4	No	15	No
Area e	0/6	NA	ND	ND	4	No	15	No
Area f	0/2	NA	ND	ND	4	No	15	No
Area g	0/7	NA	ND	ND	4	No	15	No
Area h	0/8	NA	ND	ND	4	No	15	No
Area i	0/1	NA	ND	ND	4	No	15	No
Area j	0/0	NA	ND	ND	4	No	15	No
Area l	0/16	NA	ND	ND	4	No	15	No
Area m	0/24	NA	ND	ND	4	No	15	No
Area n	0/40	NA	ND	ND	4	No	15	No

Notes: NA indicates that lead is not a COPC for the area. Therefore, a representative concentration is not available.
ND indicates that lead was not detected in any sample. Check the frequency of detection column to determine if analyses for lead were performed on any samples.

[1] The representative concentration or the representative exposure concentration is the lesser of the maximum detected concentration and the upper 95% confidence level on the mean concentration.
[2] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porters Creek Clay.
[3] Background values are for total or unfiltered samples and are from App. D of the GWOU FS Report.

evaluates all land use scenarios and identifies those land use scenarios, contaminants, and pathways that should be considered when choosing appropriate remedial actions. Sect. 8 presents remedial goal options 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 area are compared to benchmarks of 1 and 1×10^{-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 1×10^{-6} are deemed land use scenarios of concern for ELCR. To determine COCs, the chemical-specific HI and ELCR contributed by each COPC over all pathways within a land use scenario of concern are compared to benchmarks of 0.1 and 1×10^{-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 scenario of concern. To determine POCs, the exposure route HI and ELCR over all COPCs within the land use scenarios of concern are compared to benchmarks of 0.1 and 1×10^{-6} for exposure route HI and ELCR, respectively. Exposure routes with HIs and ELCRs that exceed these benchmarks are deemed POCs for that land use scenario of concern. Note that media of concern are not selected in this assessment because only one media was included in the BHHRA.

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 1×10^{-6}, respectively, then that land use scenario is a land use scenario of concern for the area. Exhibit 5.19 presents the land uses of concern for each location. Note that the results presented do not include contributions from lead as a COPC.

As shown in Exhibit 5.19, not all area/depth classifications have land use scenarios of concern for both systemic toxicity and ELCR. However, all land uses assessed in the RGA for systemic toxicity and ELCR are of concern across all areas. The McNairy Formation had more areas than any other where the land uses assessed were not of concern, and the UCRS was of concern for every area for the rural resident for systemic toxicity and ELCR. Finally note that Area k (i.e., groundwater taken to the south of the PGDP) was of concern for each land use for systemic toxicity and ELCR.

5.7.2 Contaminants of Concern

Only those contaminants whose chemical-specific ELCRs summed over all exposure routes within a land use scenario of concern are greater than or equal to 1×10^{-6} or whose HQs summed over all exposure routes are greater than or equal to 0.1 are COCs. The COCs across all land use scenarios for systemic toxicity for the UCRS, RGA, and McNairy Formation for the direct routes of exposure are summarized in Exhibits 5.20, 5.21, and 5.22, respectively. In these exhibits, those contaminants which are a COC within a scenario of concern and have a chemical-specific HI greater than 1 are marked with a solid cell. Those contaminants which are a COC within a scenario of concern and have a chemical-specific HI between 0.1 and 1 are marked with an "X." Those contaminants which are not a COC within a scenario are not marked (i.e., cell left blank). Similar information for COCs for ELCR is shown in Exhibits 5.23, 5.24, and 5.25 for the UCRS, RGA, and McNairy Formation, respectively. In these exhibits, all COCs across all land use scenarios for ELCR are summarized. Those contaminants which are a COC within a scenario of concern and have a chemical-specific ELCR greater than 1×10^{-4} are marked with a solid cell. Those contaminants which are a COC within a scenario of concern and have a chemical-specific ELCR between 1×10^{-6} and 1×10^{-4} are marked with an "X." Those contaminants which are not a COC within a scenario are not marked (i.e., cell left blank).

As shown in Exhibit 5.20, there is a total of 36 COCs for systemic toxicity over all the areas for the UCRS (including Area k). Of these, 21 are inorganic chemicals and 15 are organic compounds. Exhibit 5.23 shows that there is a total of 21 COCs for ELCR over all the areas for the UCRS (including Area k). Of

Exhibit 5.19. Selection of land uses of concern

Scenario (Depth Group)	Area													
	a	b	c	d	e	f	g	h	i	j	k	l	m	n
Results for systemic toxicity[1]														
Industrial Worker (UCRS)	X	X	–	X	X	–	–	–	X	NV	X[2]	X	X	X
Industrial Worker (RGA)	X	X	X	X	X	X	X	X	X	X	NA	X	X	X
Industrial Worker (McN)	NV	X	NV	–	X	–	–	–	–	X	NA	X	X	X
Child Recreational User (UCRS)	X	X	–	X	X	–	X	–	X	X	X[2]	X	X	X
Child Recreational User (RGA)	X	X	X	X	X	X	X	X	X	X	NA	X	X	X
Child Recreational User (McN)	NV	X	NV	–	X	–	–	–	X	X	NA	X	X	X
Child Rural Resident (UCRS)	X	X	X	X	X	X	X	X	X	X	X[2]	X	X	X
Child Rural Resident (RGA)	X	X	X	X	X	X	X	X	X	X	NA	X	X	X
Child Rural Resident (McN)	NV	X	NV	–	X	–	–	–	X	X	NA	X	X	X
Results for ELCR[3]														
Industrial Worker (UCRS)			X		X			X		NV	[2]			
Industrial Worker (RGA)										X	NA			
Industrial Worker (McN)	NV	X	NV	–		–	X	X	–		NA	X		
Recreational User (UCRS)			–		X	–	–	–	X	NV	[2]		X	
Recreational User (RGA)			X				X	X		X	NA			
Recreational User (McN)	NV		NV	–		–	X	–	–	X	NA	X		
Rural Resident (UCRS)			X							NV	[2]			
Rural Resident (RGA)											NA			
Rural Resident (McN)	NV		NV	X		–	X		–		NA			

Notes: Scenarios where risk did not exceed a benchmark level are marked with a –.
NA indicates that the scenario/land use combination is not appropriate for the HUs involved.
NV indicates data were not available to assess water drawn from this area/depth combination.

[1] Scenarios where total HI exceeds 1.0 without consideration of lead as a COPC are marked with an X.
[2] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porters Creek Clay.
[3] Scenarios where total ELCR exceeds 1×10^{-4} are marked with a solid square. Scenarios where total ELCR exceeds 1×10^{-6} but is less than 1×10^{-4} are marked with an X.

Exhibit 5.20. Contaminants of concern for systemic toxicity for UCRS[1] across all areas

Areas and Scenarios / Chemicals of Potential Concern[2]	a			b			c			d			e			f			g			h			i			j			k			l			m			n		
	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker
Aluminum	X			X			X			X			X			X						X			X						X			X			X			X		
Antimony			X									X														X	X									X						
Arsenic	X				X	X				X		X	X		X												X				X				X	X	X		X			X
Barium				X						X			X			X									X									X			X			X		
Beryllium																															X	X										
Cadmium				X	X	X						X														X	X						X		X	X		X	X		X	X
Chromium	X	X		X	X	X				X	X	X		X	X					X	X				X	X	X				X	X	X	X	X	X	X	X	X	X	X	X
Copper																									X												X					
Fluoride				X						X			X												X		X				X			X			X		X	X		
Iron	X		X	X		X	X				X				X	X		X				X					X									X	X		X	X		X
Lead																																										
Manganese				X			X									X			X	X	X					X	X							X	X	X	X				X	X
Mercury																																								X		
Molybdenum				X																														X						X		
Nickel	X					X				X			X											X	X						X			X			X			X		
Nitrate as Nitrogen																			X																							
Nitrate/Nitrite										X																																
Silver																									X												X			X		
Strontium										X																								X						X		
Uranium	X			X						X		X													X									X					X	X		
Vanadium		X	X		X	X	X	X				X	X		X	X	X			X	X	X	X				X					X	X	X	X	X			X	X	X	X
1,1-Dichloroethene				X						X																									X	X					X	
1,2-Dichloroethane										X																								X						X		
1,2-Dichloroethene				X						X																							X	X						X		

Exhibit 5.20 (continued)

Chemicals of Potential Concern[2] \ Areas and Scenarios	a			b			c			d			e			f			g			h			i			j			k			l			m			n		
	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker
2,4-Dimethylphenol																																		X						X		
Acetone																															X											
Benzene							X																		X												X					
Bromodichloromethane																																					X			X		
Chloroethane																																		X								
Chloroform	X						X																		X									X			X					
Dimethylbenzene																																		X						X		
Ethylbenzene										X																									X					X	X	
Naphthalene										X																																
trans-1,2-Dichloroethene				X																																X						X
cis-1,2-Dichloroethene			X		X					X												X											X									
Trichloroethene							X						X			X						X			X							X	X									

Notes: X indicates that the chemical of potential concern is a contaminant 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.

[1] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porters Creek Clay.
[2] Only chemicals of potential concern which have a chemical-specific HI greater than 0.1 for one or more land use scenarios of concern are listed.

Exhibit 5.21. Contaminants of concern for systemic toxicity for RGA[1] across all areas

Areas and Scenarios / Chemicals of Potential Concern[2]	a			b			c			d			e			f			g			h			i			j			k			l			m			n		
	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker
Aluminum				X			X												X			X						X						X								
Antimony																																										
Arsenic	X		X	X		X				X		X	X			X			X			X			X			X		X				X		X	X			X		
Barium	X			X			X			X			X			X																		X								
Beryllium				X	X								X	X											X	X								X	X		X	X		X	X	
Boron																									X												X			X		
Cadmium					X	X									X			X			X				X	X	X								X	X		X	X		X	X
Chromium		X	X	X	X	X			X	X	X	X					X	X		X	X			X											X	X			X		X	X
Fluoride	X			X						X			X												X									X			X			X		
Iron	X					X			X	X			X			X			X					X	X					X						X	X			X		X
Lead																																										
Lithium																									X												X			X		
Manganese	X			X			X				X	X				X									X				X	X				X			X			X		
Molybdenum							X						X																	X				X			X			X		
Nickel																			X						X												X			X		
Nitrate as Nitrogen																						X																				
Silver													X												X												X			X		
Uranium				X																														X								
Vanadium		X	X	X	X					X	X	X	X	X	X	X	X	X				X	X	X	X	X	X	X	X	X				X	X		X	X	X	X	X	X
1,1,2-Trichloroethane				X																														X						X		
1,1-Dichloroethene							X									X																				X						X
1,2-Dichloroethane				X																														X						X		
1,2-Dichloroethene																X																					X					
2-Butanone													X																											X		
4-Methyl-2-pentanone																									X												X			X		
Acetone																																								X		
Acrylonitrile																											X												X			X
Benzene																									X												X			X		

Exhibit 5.21 (continued)

Areas and Scenarios / Chemicals of Potential Concern[2]	a			b			c			d			e			f			g			h			i			j			k			l			m			n		
	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker
Bis(2-ethylhexyl)phthalate																X																										
Bromomethane																									X												X			X		
Carbon Tetrachloride					X	X										X																					X					
Chlorobenzene				X																														X						X		
Chloroform				X			X																											X						X		
Dimethylbenzene																																		X								
Ethylbenzene																																		X	X					X		
Aroclor-1254																											X												X			X
Tetrachloroethene			X			X																														X						X
cis-1,2-Dichloroethene			X		X		X									X						X																			X	
trans-1,2-Dichloroethene			X										X																							X	X					X
Trichloroethene																						X			X																		

Notes: X indicates that the chemical of potential concern is a contaminant 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.

[1] Area k includes wells completed in Terrace Gravels, Eocene Sands, and Porters Creek Clay. Please see Exhibit 5.16 for the COCs for this area.
[2] Only chemicals of potential concern which have a chemical-specific HI greater than 0.11 for one or more land use scenarios of concern are listed.

Exhibit 5.22. Contaminants of concern for systemic toxicity for McNairy Formation[1] across all areas

Areas and Scenarios / Chemicals of Potential Concern[2]	a			b			c			d			e			f			g			h			i			j			k			l			m			n		
	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker
Aluminum																																								X		
Antimony																												X														
Arsenic														X	X														X										X			X
Barium													X																								X			X		
Beryllium													X	X																							X	X		X	X	
Cadmium															X																								X			X
Chromium														X	X																							X	X	X	X	X
Fluoride													X																								X			X		
Iron														X	X																							X			X	X
Manganese													X	X	X											X			X	X							X	X	X	X		
Molybdenum																													X	X							X			X		
Nickel													X																								X			X		
Uranium													X																													
Vanadium															X																							X	X	X	X	X
Trichloroethene													X																								X					

Notes: X indicates that the chemical of potential concern is a contaminant 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.

[1] Area k includes wells completed in Terrace Gravels, Eocene Sands, and Porters Creek Clay. Please see Exhibit 5.16 for the COCs for this area.
[2] Only chemicals of potential concern which have a chemical-specific HI greater than 0.11 for one or more land use scenarios of concern are listed.

Exhibit 5.23. Contaminants of concern for ELCR for UCRS[1] across all areas

Areas and Scenarios / Chemicals of Potential Concern[2]	a			b			c			d			e			f			g			h			i			j			k			l			m			n		
	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker
Arsenic	X	X	X		X	X					X	X	X	X	X										X	X	X				X	X	X		X	X		X	X		X	X
Beryllium	X	X	X	X	X	X					X	X																							X	X						X
1,1-Dichloroethene		X	X		X	X					X	X																				X										
1,2-Dichloroethane										X																								X						X		
Benzene				X		X	X			X															X									X		X	X			X		X
Bromodichloromethane																									X												X		X	X		X
Chloroform	X		X				X		X																X						X			X			X			X		
Dibromochloromethane																									X												X			X		
Methylene Chloride										X															X						X			X			X			X		
Trichloroethene							X																		X						X	X	X				X					
Vinyl Chloride																																X										
Americium-241				X																														X								
Neptunium-237	X		X							X		X																			X			X						X		
Plutonium-239				X																											X			X						X		
Radium-226				X															X		X				X		X				X		X	X			X		X	X		
Radon-222															X									X															X			
Technetium-99	X			X		X	X			X						X									X									X		X				X		X
Thorium-228										X																					X		X	X						X		
Uranium-234				X		X				X		X																			X			X		X				X		X
Uranium-235										X																								X						X		
Uranium-238				X		X				X		X																			X			X		X				X		X

Notes: X indicates that the chemical of potential concern is a contaminant of concern, and chemical-specific ELCR is between 1×10^{-6} and 1×10^{-4} for the scenario.
Solid cell indicates that the chemical of potential concern is a chemical of concern, and chemical-specific ELCR is greater than 1×10^{-4} for the scenario.
Blank cell indicates that the chemical of potential concern is not a chemical of concern for the scenario.

[1] Area k includes samples from wells completed in the Terrace Gravels, Eocene Sands, and Porters Creek Clay.
[2] Only chemicals of potential concern which have a chemical-specific HI greater than 1×10^{-6} for one or more land use scenarios of concern are listed.

Exhibit 5.24. Contaminants of concern for ELCR for RGA[1] across all areas

Areas and Scenarios / Chemicals of Potential Concern[2]	a			b			c			d			e			f			g			h			i			j			k			l			m			n		
	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker
Arsenic	■	X	X	X	X	X				■	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X	■	X	X				X	X	X	X	X	X	X	X	X
Beryllium				■	■	■							■	■	■										■	■	■							■	■	■	■	■	■	■	■	■
1,1,2-Trichloroethane				X																														X						X		
1,1-Dichloroethene	■	X	X	■		X	■	X	X							■	X	X																■	X	■	X	X	X	■	X	■
1,2-Dichloroethane				X																														X						X		
Acrylonitrile																									■	X	X										■	X	X	■	X	X
Benzene																									X												X			X		
Bis(2-ethylhexyl)phthalate																X	X	X							X	X											X	X		X	X	
Carbazole																									X	X											X	X	X	X	X	
Carbon tetrachloride	■	X	X	■	X	X										X																		■	X	X	X			■	X	X
Chloroform	X			X		X	X																		X									X		X	X			X		X
Chloromethane																									X												X			X		
Chrysene																										X												X			X	
Methylene chloride										X		X																						X						X		X
Aroclor-1254																									X	X	X										X	X	X	X	X	X
Polychlorinated biphenyl																									X	X											X	X		X	X	
Tetrachloroethene	■	■	X	■	■	■				X	X	X													X	X								■	■	■	X	X		■	■	■
Trichloroethene	■	■	■	■	■	■	■	X	X	■	■	X	■	■	X	■	■	X							X									■	■	■	■	X	X	■	■	■
Vinyl Chloride				■	■	■																			X	X	X							■	■	■	X	X	X	■	■	■
Americium-241				X		X				X															X									X			X			X		
Cesium-137	X		X							X		X																						X		X				X		X
Neptunium-237	X		X																X															X		X	X			X		
Radium-226				■	X	■				X		X							X						X		X							■	X	X	X			■		X
Radon-222				■		X	■		■	■		■	■		■	■		■	■		■	■		X	■		■							■		■	■		■	■	X	■
Technetium-99	X		X	X		X	X		X				X		X									X	X									X		X	X		X	X		X
Uranium-234				X						X																								X						X		
Uranium-235																																								X		
Uranium-238				X		X				X																								X		X				X		X

Notes: X indicates that the chemical of potential concern is a contaminant of concern, and chemical-specific ELCR is between 1×10^{-6} and 1×10^{-4} for the scenario.
Solid cell indicates that the chemical of potential concern is a chemical of concern, and chemical-specific ELCR is greater than 1×10^{-4} for the scenario.
Blank cell indicates that the chemical of potential concern is not a chemical of concern for the scenario.

[1] Area k includes samples from wells completed in the Terrace Gravels, Eocene Sands, and Porters Creek Clay.
[2] Only chemicals of potential concern which have a chemical-specific HI greater than 1×10^{-6} for one or more land use scenarios of concern are listed.

Exhibit 5.25. Contaminants of concern for ELCR for McNairy[1] Formation across all areas

Areas and Scenarios / Chemicals of Potential Concern[2]	a			b			c			d			e			f			g			h			i			j			k			l			m			n		
	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker
Arsenic													■	X	X				X	X	X							■	X	■							■	X	X	■	X	X
Beryllium													■	■	X																						■	■	■	■	■	■
Trichloroethene				■	X	X				X			X																					■	X	X	X			■	X	X
Neptunium-237																			X																							
Radium-226																			X		X	X		X													X		X	X		X
Radon-222													■		X							■		X													■		X	■		X
Technetium-99				X		X																												X						X		

Notes: X indicates that the chemical of potential concern is a contaminant of concern, and chemical-specific ELCR is between 1×10^{-6} and 1×10^{-4} for the scenario.
Solid cell indicates that the chemical of potential concern is a chemical of concern, and chemical-specific ELCR is greater than 1×10^{-4} for the scenario.
Blank cell indicates that the chemical of potential concern is not a chemical of concern for the scenario.

[1] Area k includes samples from wells completed in the Terrace Gravels, Eocene Sands, and Porters Creek Clay.
[2] Only chemicals of potential concern which have a chemical-specific HI greater than 1×10^{-6} for one or more land use scenarios of concern are listed.

these, 2 are inorganic chemicals, 9 are organic compounds, and 10 are radionuclides. Over both systemic toxicity and ELCR (i.e., combining results from Exhibits 5.20 and 5.23), there is a total of 49 COCs over all the areas for the UCRS (including Area k). Of these, 21 are inorganic chemicals, 18 are organic compounds, and 10 are radionuclides.

Combining the results from Exhibits 5.20 and 5.23 and considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs can be considered "priority COCs" in UCRS groundwater across all use scenarios (excluding Area k):

- Inorganic chemicals – arsenic, antimony, beryllium, cadmium, chromium, iron, lead, manganese, nickel, and vanadium.
- Organic compounds – 1,1-dichloroethene, benzene, chloroform, ethylbenzene, naphthalene, *trans*-1,2-dichloroethene, *cis*-1,2-dichloroethene, TCE, and vinyl chloride.
- Radionuclides – ^{222}Rn.

For Area k, the "priority COCs" in groundwater across all use scenarios are as follows:

- Inorganic chemicals – antimony, beryllium, cadmium, iron, lead, manganese, and vanadium
- Organic compounds – 1,1-dichloroethene, 1,2-dichloroethene, naphthalene, *cis*-1,2-dichloroethene, TCE, and vinyl chloride.
- Radionuclides – ^{222}Rn.

Each of these COCs presents either a chemical-specific HI or ELCR at one or more areas, across all land uses, that exceeds 1 or 1×10^{-4}, respectively.

As shown in Exhibit 5.21, there is a total of 38 COCs for systemic toxicity over all areas for the RGA. Of these, 19 are inorganic chemicals and 21 are organic compounds. Exhibit 5.24 shows that there is a total of 28 COCs for ELCR over all areas. Of these, 2 are inorganic chemicals, 17 are organic compounds, and 9 are radionuclides. Over both systemic toxicity and ELCR (i.e., combining results from Exhibits 5.21 and 5.24), there is a total of 55 COCs over all areas. Of these, 19 are inorganic chemicals, 27 are organic compounds, and 9 are radionuclides.

Combining the results from Exhibits 5.21 and 5.24 and considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs can be considered "priority COCs" in RGA groundwater across all use scenarios:

- Inorganic chemicals – antimony, arsenic, beryllium, cadmium, chromium, iron, lead, manganese, molybdenum, and vanadium.
- Organic compounds – 1,1-dichloroethene, acrylonitrile, carbon tetrachloride, Aroclor-1254, tetrachloroethene, *cis*-1,2-dichloroethene, *trans*-1,2-dichloroethene, TCE, and vinyl chloride.
- Radionuclides – ^{226}Ra and ^{222}Rn.

As with the UCRS and Area k groundwater, each of these COCs presents either a chemical-specific HI or ELCR at one or more areas, across all land uses, that exceeds 1 or 1×10^{-4}, respectively.

As shown in Exhibit 5.22, there is a total of 15 COCs for systemic toxicity over all areas for the McNairy Formation. Of these, 14 are inorganic chemicals and 1 is an organic compound. Exhibit 5.25 shows that there is a total of 7 COCs for ELCR over all areas. Of these, 2 are inorganic chemicals, 1 is an organic compound, and 4 are radionuclides. Over both systemic toxicity and ELCR (i.e., combining results from Exhibits 5.22 and 5.25), there is a total of 19 COCs over all the GWOU areas. Of these, 14 are inorganic chemicals, 1 is an organic compound, and 4 are radionuclides.

Combining the results from Exhibits 5.22 and 5.25 and considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs can be considered "priority COCs" in McNairy Formation groundwater across all use scenarios:

- Inorganic chemicals – antimony, arsenic, beryllium, cadmium, chromium, iron, manganese, molybdenum, and vanadium.
- Organic compounds – TCE.
- Radionuclides – ^{222}Rn.

As before, each of these COCs presents either a chemical-specific HI or ELCR at one or more areas, across all land uses, that exceeds 1 or 1×10^{-4}, respectively.

5.7.3 Pathways of Concern

Only those exposure routes with a HI for adults or children greater than 0.1 or a pathway ELCR greater than 1×10^{-6} over all contaminants within a land use scenario of concern are POCs. The POCs for each land use scenario of concern are presented in the Exhibit 5.26, 5.27, and 5.28 for the UCRS, RGA, and McNairy Formation, respectively. As shown in these exhibits all exposure routes evaluated, including the biota consumption exposure routes, are a POC for at least one scenario in at least one area.

5.8 SUMMARY OF RISK CHARACTERIZATION

Tables 5.10 to 5.23 present summaries of the unfiltered groundwater data risk characterizations for the GWOU and its areas. Each of these tables presents 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 pathway of concern contributes to the total risk, and the percent of risk each COC contributes to the total risk.

Note that the tables that summarize the results for systemic toxicity do not include contributions from lead. The contribution from lead was not included in the calculations that generated these tables because the determination was made that to do so would make the contributions from the other COCs appear meaningless. Given the uncertainty in the provisional lead reference dose, it was believed that this was inappropriate.

Exhibit 5.26. Pathways of concern for UCRS[1]

Exposure Routes \ Areas and Scenarios	a			b			c			d			e			f			g			h			i			j			k			l			m			n		
	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker
Systemic Toxicity																																										
Direct Ingestion	■		■	■		■	■			■		■	■		■	■			■			■			■		X				■		■	■		■	■		■	■		■
Dermal Contact while Showering	■		■	■		■				■		■	X		X				X						X		X				■		X	■		■	■		X	■		■
Inhalation of Vapors while Showering	■		■	■		■				■		■																			■		X	■		■	X			■		■
Inhalation of Vapors during Home Use	■			■			X			■															■						■			■						■		
Incidental Ingestion while Swimming		■			■						■			X												X						■			■			X			■	
Dermal Contact while Swimming		■			■						■			■						X						■						■			■			■			■	
Dermal Contact while Wading		■			■						■			■						X						■						■			■			■			■	
Consumption of Biota[2]	■	■		■	■		X			■	■		■	■		■			■	■		■			■						■	■		■	■		■	■		■	■	
ELCR																																										
Direct Ingestion	■		■	■		■	X			■		■	X		X	X			X		X				■		X				■		■	■		■	■		X	■		■
Dermal Contact while Showering	■		■	■		■				■		X													X						X		X	■		■				■		■
Inhalation of Vapors while Showering	■		■	■		■	X		X	■		■	■		X	■		■	■		■	■		X	■		■				■		■	■		■	■		■	■		■
Inhalation of Vapors during Home Use	■			■			X			■			X			X			X			X			X						■			■			X			■		
Incidental Ingestion while Swimming		■			■						X			X												X						X			■			X			■	
Dermal Contact while Swimming		■			■						■															X						■			■			X			■	X
Dermal Contact while Wading		■			■						■															X						■			■			X			■	X
Consumption of Biota[2]	■	■		■	■		■			■	■		X			■			■						■	X					■	■		■	■		■	X		■	■	X

Notes: Both an X and a solid cell indicate that the exposure route is a pathway of concern (POC). If under systemic toxicity, an X indicates that the pathway-specific HI is between 0.1 and 1, and a solid cell indicates that the pathway-specific HI is greater than 1. If under ELCR, an X indicates that the pathway-specific ELCR is between 1×10^{-6} and 1×10^{-4}, and a solid cell indicates that the pathway-specific ELCR is greater than 1×10^{-4}. Under both systemic toxicity and ELCR, a blank cell indicates that the exposure route is not a POC for the scenario or is not applicable to the scenario.
Systemic toxicities for the recreational user and resident used to complete this exhibit are those for the child.
All values do not include contribution from lead as a COPC.

[1] Area k includes samples from wells completed in Terrace Gravels, Eocene Sands, and Porters Creek Clay.
[2] Sum across all biota consumption exposure routes.

Exhibit 5.27. Pathways of concern for RGA[1]

Exposure Routes \ Areas and Scenarios	a			b			c			d			e			f			g			h			i			j			k			l			m			n		
	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker
Systemic Toxicity																																										
Direct Ingestion	■		■	■		■	■		■	■		■	■		■	■		■	■			■		■	■		■	■		■	■		■	■		■	■		■	■		■
Dermal Contact while Showering	■		■	■		■	■		X	■		X	■		■	■		X	X			X		X	■		X	X		X	■		X	■		■	■		■	■		■
Inhalation of Vapors while Showering	■		■	■		■	■		X	■		X	■		■	■		X							X		X				■		X	■		■	■		X	■		■
Inhalation of Vapors during Home Use	■			■			■			■			■			■						X			■						■			■			■			■		
Incidental Ingestion while Swimming		■			X			X			X			X			X						X			X			X			■			■			X			■	
Dermal Contact while Swimming		■			■			■			■			■			■			X			X			■			X			■			■			■			■	
Dermal Contact while Wading		■			■			■			■			■			■			X			X			■			X			■			■			■			■	
Consumption of Biota[2]	■	■		■	■		■	■		■	■		■	■		■	■		■	■		■	■		■	■					■	■		■	■		■	■		■	■	
ELCR																																										
Direct Ingestion	■		■	■		■	■		X	■		X	■		■	■		X	X		X	X		X	■		■			X	■		■	■		■	■		■	■		■
Dermal Contact while Showering	■		■	■		■	X		X	X		X	■		X	X		X							■		X				X		X	■		■	■		X	■		■
Inhalation of Vapors while Showering	■		■	■		■	■		■	■		■	■		■	■		■	■		■	■		X	■		■				■		■	■		■	■		■	■		■
Inhalation of Vapors during Home Use	■			■			■			■			■			■			X			X			■						■			■			■			■		
Incidental Ingestion while Swimming		■			■			X			X			X			X									X			X			X			■			X			■	
Dermal Contact while Swimming		■			■			X			X			■			X									■						■			■			■			■	
Dermal Contact while Wading		■			■			X			X			■			X									■			X			■			■			■			■	
Consumption of Biota[2]	■	■		■	■		■	X		■	■		■	■		■	■		■			■			■	■		X			■	■		■	■		■	■		■	■	

Notes: Both an X and a solid cell indicate that the exposure route is a pathway of concern (POC). If under systemic toxicity, an X indicates that the pathway-specific HI is between 0.1 and 1, and a solid cell indicates that the pathway-specific HI is greater than 1. If under ELCR, an X indicates that the pathway-specific ELCR is between 1×10^{-6} and 1×10^{-4}, and a solid cell indicates that the pathway-specific ELCR is greater than 1×10^{-4}. Under both systemic toxicity and ELCR, a blank cell indicates that the exposure route is not a POC for the scenario or is not applicable to the scenario.
Systemic toxicities for the recreational user and resident used to complete this exhibit were those for the child.
All values do not include contribution from lead as a COPC.

[1] Area k includes samples from wells completed in Terrace Gravels, Eocene Sands, and Porters Creek Clay.
[2] Sum across all biota consumption exposure routes.

Exhibit 5.28. Pathways of concern for McNairy Formation[1]

Exposure Routes \ Areas and Scenarios	a			b			c			d			e			f			g			h			i			j			k			l			m			n		
	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker	Rural Resident	Recreational User	Industrial Worker
Systemic Toxicity																																										
Direct Ingestion				■		■							■		■										■			■		■	■		■	■		■	■		■	■		■
Dermal Contact while Showering				■		X							■		X										X			X			■		X	■		X	X		X	■		X
Inhalation of Vapors while Showering				■		X																									■		X	■		X				■		X
Inhalation of Vapors during Home Use				■									X																		■			■						■		
Incidental Ingestion while Swimming					X									X															X			■			X			X			X	
Dermal Contact while Swimming					■									■												X			X			■			■			■			■	
Dermal Contact while Wading					■									■												X			X			■			■			■			■	
Consumption of Biota[2]				■	■								■	■											■	X		■	■		■	■		■	■		■	■		■	■	
ELCR																																										
Direct Ingestion				■		X							■		■				X		X	X		X				■		■	■		■	X		X	■		■	■		■
Dermal Contact while Showering				X		X							■		■													X		X	X		X	X		X	X		X	■		X
Inhalation of Vapors while Showering				X		X							■		X							■		X							■		■	X		X	■		X	■		X
Inhalation of Vapors during Home Use				■									X									X									■			■			X			X		
Incidental Ingestion while Swimming					X									X															X			X			X			X			X	
Dermal Contact while Swimming					X									■															X			■			X			■			■	
Dermal Contact while Wading					X									■															X			■			X			■			■	
Consumption of Biota[2]				■	X								■	■					X			X						■			■	■		■	X		■	■		■	■	

Notes: Both an X and a solid cell indicate that the exposure route is a pathway of concern (POC). If under systemic toxicity, an X indicates that the pathway-specific HI is between 0.1 and 1, and a solid cell indicates that the pathway-specific HI is greater than 1. If under ELCR, an X indicates that the pathway-specific ELCR is between 1×10^{-6} and 1×10^{-4}, and a solid cell indicates that the pathway-specific ELCR is greater than 1×10^{-4}. Under both systemic toxicity and ELCR, a blank cell indicates that the exposure route is not a POC for the scenario or is not applicable to the scenario.
Systemic toxicities for the recreational user and resident used to complete this exhibit were those for the child.
All values do not include contribution from lead as a COPC.

[1] Area k includes samples from wells completed in Terrace Gravels, Eocene Sands, and Porters Creek Clay.
[2] Sum across all biota consumption exposure routes.

6. UNCERTAINTY IN THE RISK ASSESSMENT

Uncertainties are associated with each of the steps 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 these uncertainties may have significant effects upon the results of the risk characterization and subsequent risk management decisions. Types of uncertainties that are associated with the risk assessment process can be divided into four broad categories. These are uncertainties associated with data, with the exposure assessment, with the toxicity assessment, and with the risk characterization. Specific uncertainties in each of these broad categories are discussed in the following subsections. In this discussion, the magnitude of the effect of the uncertainty on risk characterization is categorized as small, moderate, or large. Uncertainties categorized as small should not cause the cumulative or total risk estimates to vary by more than one order of magnitude, uncertainties categorized as moderate may cause the total risk estimates to vary by between one and two orders of magnitude, and uncertainties categorized as large may cause the total risk estimate to vary by more than two orders of magnitude.

In evaluating these uncertainties and their estimated effect on the total risk estimates, it must be remembered that the following uncertainties are neither independent nor mutually exclusive. Therefore, the total effect of all uncertainties discussed in the following subsections on the total risk estimates is <u>not</u> the sum of the estimated effects.

6.1 UNCERTAINTIES ASSOCIATED WITH DATA AND DATA EVALUATION

Several uncertainties are associated with the data set and data evaluation. Specific uncertainties that will be discussed in the following subsections are selection of COPCs, determination of exposure point concentrations, and use of concentrations from total versus filtered samples for inorganic compounds in groundwater.

6.1.1 Selection of COPCs

Some uncertainty is involved with the selection of COPCs from the larger data set of all detected analytes. This uncertainty is derived from several sources. These sources are as follows:

- Retention of infrequently detected analytes,
- Temporal patterns in analyte detection,
- Quantitation limits used in analyte detection,
- Use of historical data,
- Retention of common laboratory contaminants,
- Lack of consideration of blank contamination,
- Use of the toxicity screen, and
- Use of the background screen.

As shown above, the first uncertainty related to the selection of COPCs to be discussed is the retention of infrequently detected chemicals in the list of COPCs. As can be seen in Tables 2.6, 2.11, and 2.12, several of the chemicals retained in the list of COPCs were detected in less than 10% of the samples taken. Of greatest concern is that some of these COPCs are retained as COCs. Table 6.1 presents the effect of removing infrequently detected analytes from the list of COPCs under residential use. Results in this table indicate that the infrequently detected COPCs had virtually no effect on the total risk or hazard estimates for most areas. Notable exceptions are Area b (McNairy) where both total HI and total ELCR

decrease by two orders of magnitude and Area i (RGA) where both total HI and total ELCR decrease by nearly an order of magnitude. The large effect for Area b (McNairy) is because all COPCs for this data aggregate, except ^{99}Tc, were detected in less than 10% of all samples. The nearly moderate effect on total HI for Area i (RGA) is because the driving COC, antimony, was only detected in 6 of 412 samples. (Antimony makes up approximately 40% of the total HI for the child resident.) Similarly, the nearly moderate effect on total ELCR for Area i (RGA) is because two of the three driving COC, beryllium and acrylonitrile, were only detected in 28 or 412 and 1 of 378 samples, respectively. (Beryllium and acrylonitrile make up approximately 46% and 18% of the total ELCR for the resident.) In any case, the estimated effect of this uncertainty on the total risk estimates is variable but tends to be small.

A related uncertainty is the retention of analytes for which analyses were infrequently performed. These analytes are not removed from the list of COPCs using any of the procedures described in Sect. 2 and may become important risk drivers if they are toxic at very low doses. The most notable example from a previous BHHRA performed for the PGDP was the retention of ^{210}Pb as a COPC for the McNairy formation in the WAG 6 report (DOE 1999a). In that BHHRA, ^{210}Pb was found to contribute over 43% of the total ELCR from use of groundwater drawn from the McNairy Formation and over 5% of the total ELCR from use of groundwater drawn from the RGA. However, only a single analysis was performed for this radionuclide making these percentages suspect for decision-making. Fortunately, the data set for the GWOU BHHRA was large enough, and the analyte list kept constant enough, that this phenomenon does not appear to be important in this BHHRA. Therefore, it is estimated that the net effect on the final risk estimates in the GWOU BHHRA is small. (See Tables 7.1 and 7.2 for additional exploration of this uncertainty.)

The second uncertainty related to selection of COPCs is that temporal patterns in the detection of analytes were not fully considered when selecting COPCs for the area assessment. (Note that these were considered when risks for future modeled concentrations from sources were estimated.) Generally, experience has shown that it is possible for contaminant concentrations to show positive or negative trends that may in turn be related to increasing or decreasing levels of risk over time. However, experience has also shown that this uncertainty is only important when the goal of a BHHRA is to estimate risks for dates far into the future (i.e., more than 100 years). Because the time frame considered in the area assessment is relatively short (i.e., 40 years), the assumed effect of this uncertainty on the risk estimates is small. However, as shown in Sect. 5.5, risks in areas associated with the TCE plumes may change significantly as contaminants continue to migrate from the TCE DNAPL and other sources.

The third uncertainty related to selection of COPCs in the BHHRA concerns the use of quantitation limits that exceed concentrations that may result in a significant health effect in humans. For example, for Area a (RGA), 85 organic compounds were never detected but are seen to have a quantitation limit in at least one sample that exceeds either their residential use HI-based RBC or their residential use ELCR-based RBC (see Table 6.2). Because the quantitation limits of these analytes exceed their RBCs, it is possible that these chemicals are present in water drawn from the Area a (RGA) at concentrations that pose risk to human health but are not retained as COPCs. However, experience at the PGDP has shown that these organic compounds tend to be unrelated to processes at GWOU. Therefore, the estimated effect of this uncertainty on the risk estimates is small.

A fourth uncertainty related to selection of COPCs is the removal of data from samples collected prior to 1993. As noted earlier, these data were removed from the data set to address a possible sampling bias caused by a change in sampling methods made around that date. The estimated effect of this uncertainty on the risk estimates is assumed to be small because the remaining data set is so much larger than that deleted and because the lists of COPCs and COCs developed in the area assessment correlate well with those produced in earlier BHHRAs.

A fifth uncertainty related to the selection of COPCs is the failure to delete common laboratory contaminants when developing in the COPC list. A cursory examination of Tables 2.6 and 2.11 shows that some common laboratory contaminants were retained as COPCs. Examples of these are benzene, phthalates, and methylene chloride. However, examination of the risk results shows that these compounds are not significant risk drivers in the area assessment. Therefore, the estimated effect of the uncertainty on the risk estimates is small. (See Tables 7.1 and 7.2 for additional consideration of this uncertainty.)

A sixth uncertainty related to the selection of COPCs is that analyte concentrations were not compared to concentrations found in blank samples to ensure that blank contamination did not impact the risk estimates. Generally, regulatory guidance allows the deletion of common laboratory contaminants and other analytes from the list of COPCs if they are also detected in blank samples at appropriate concentrations. However, for the data used in the GWOU BHHRA, sample blank data could not be matched to site data; therefore, it was not possible to perform this test. While not performing this test ensures that all analytes that may be potential laboratory contaminants are treated as site contaminants, it also results in risk estimates that may exceed the actual risk posed by use of water drawn from the various areas. However, the estimated effect of this uncertainty is small because relatively few contaminants (e.g., TCE, vinyl chloride, and ^{222}Rn) detected at significant concentrations tend to dominate most area's risks estimates, and it is unlikely that these contaminants are laboratory contaminants for all areas.

A seventh uncertainty related to the selection of the COPCs is the use of a toxicity screen to determine the final list of COPCs. In this BHHRA, the maximum detected concentrations of analytes within each area and depth classification combination were compared to residential human health RBCs, and analytes with maximum detected concentrations less than their RBC were removed from the list of COPCs. (The derivation of these criteria is explained in detail in Subsect. 1.2.) Past BHHRAs prepared for the PGDP have quantitatively examined the effect of the toxicity screen on the list of COPCs and on the resulting risk estimates by displaying marginal hazard and risk contributions. (Marginal hazard and risk contributions can be defined as the estimated increase in the final hazard and risk estimates under the residential scenario which would have been seen if the analytes removed from the list of COPCs had been left on the list.) Because the number of analyses performed in this assessment is larger than these earlier assessments, this information is not displayed here. However, as illustrated in that previous work, it is believed that the marginal contribution of the analytes removed from the COPCs list would be minimal. Therefore, the estimated effect of this uncertainty on the final risk estimates is judged to be small.

An eighth uncertainty related to the selection of the COPCs is the use of a background screen to determine the final list of COPCs for water drawn from the RGA and McNairy Formation. (Background values are not available for the UCRS or other groundwater.) In this BHHRA, the maximum detected concentrations of analytes within each area and depth classification combination were compared to background concentrations. The source of these background values is described in Subsect. 1.2. In Appendix D of the GWOU feasibility study report, these background concentrations are compared to their respective residential use RBCs. As shown there, several of the background concentrations are greater than their respective RBCs indicating that if analytes had not been removed from the list of COPCs 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 each group to the selected background concentration, it is unlikely that the final risk estimates would be significantly impacted. Therefore, the estimated effect of using the background screen to develop the list of COPCs is assumed to be small, and the resulting effect on the final risk estimates is also assumed to be small.

6.1.2 Determination of exposure point concentrations–current conditions

The uncertainty related to the calculation of exposure point concentrations under current conditions cannot be completely quantified for this BHHRA because information is lacking. For example, 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 some results that may be in error. However, because the risk estimates are driven, for the most part, by contaminants known to be present in groundwater at the PGDP, the effect of this uncertainty on the final risk estimates is believed to be small.

To examine the uncertainty in the derivation of exposure point concentrations in more detail and to refine the evaluation of the area risk estimates, risk from residential use of water drawn from each sampling station was estimated. In these "well-by-well" risk calculations, data were summarized and evaluated following the procedures discussed in Sect. 2, doses were estimated using the procedures discussed in Sect. 3, toxicity was assessed using information in Sect. 4, and risks were characterized using the methods in Sect. 5. However, note that the biota consumption exposure routes were not included in the "well-by-well" assessment because it was believed that the uncertainties inherent in the biota routes would confuse the evaluation of the risk results to too great an extent. (Also, sampling results from boreholes installed during the WAG 3 investigation were not included because these results were not available at the time the analysis was performed.)

Rather than presenting here the multitude of exhibits and tables the "well-by-well" analyses generated, a series of maps and two summary tables were generated. These maps are presented in the following figures. (Note that the risk results used to generate these maps and tables are presented in App. H.)

- Figure 6.1 – Systemic toxicity from residential use of unfiltered groundwater samples drawn from wells completed in the RGA and McNairy Formation.

- Figure 6.2 – Systemic toxicity from residential use of filtered groundwater samples drawn from wells completed in the RGA and McNairy Formation.

- Figure 6.3 – Excess lifetime cancer risk from residential use of unfiltered groundwater samples drawn from wells completed in the RGA and McNairy Formation.

- Figure 6.4 – Excess lifetime cancer risk from residential use of filtered groundwater samples drawn from wells completed in the RGA and McNairy Formation.

- Figure 6.5 – Systemic toxicity from residential use of unfiltered groundwater drawn from wells completed in the UCRS, Eocene Sands, Porters Creek Clay, and Terrace Gravels.

- Figure 6.6 – Excess lifetime cancer risk from residential use of unfiltered groundwater drawn from wells completed in the UCRS, Eocene Sands, Porters Creek Clay, and Terrace Gravels.

The tables summarizing the results of the "well-by-well" assessment are as follows:

- Table 6.3 – Summary of excess cancer risks by area from analysis of results from individual sampling points.

- Table 6.4 – Summary of hazard indices by area from analysis of results from individual sampling points.

Generally, the "well-by-well" results for ELCR are consistent with the results from the area assessments. As with the area assessments, when unfiltered samples are assessed, ELCR from TCE and its breakdown products compose the greatest portion of the total ELCR for areas inside the security fence or associated with the TCE plumes outside the fence (i.e., Areas a, b, c, d, e, and f); ELCR from arsenic and beryllium are of less importance in each of the aforementioned areas, except Area e, but these metals

compose the greatest portion of the total ELCR for Areas i, j, and k.; ELCR from ^{99}Tc and uranium tend to be of minor importance relative to the contributions of total ELCR from organic compounds and metals. However, contribution of ^{99}Tc to total ELCR is notable in Areas a and b, and contribution of uranium to total ELCR is notable in Areas b and k. Excess lifetime cancer risk results for filtered samples also are similar to those from the area assessment. Generally, ELCRs from metals are lower or not present for the filtered samples, and fewer sampling points had detectable analyte concentrations. (Note that only 7 RGA sampling points had detectable concentrations of either arsenic or beryllium or both.)

Similarly, the "well-by-well" results for HI are consistent with the results from the area assessments. As with the area assessments, when unfiltered samples are assessed, HI from TCE and its breakdown products compose the greatest portion of the total HI for areas inside the security fence or associated with the TCE plumes outside the fence; HI from metals are less important in the aforementioned areas, but metals compose the greatest portion of the total HI in Areas g, h, i, and j. Hazard indices results from analysis of filtered samples also are similar to those from the area assessments. Generally, HIs are lower for most metals, and fewer sampling points had detectable concentrations.

Another uncertainty in the determination of exposure point concentrations under current conditions is the combination of the data from the Southwest Plume with other data from Area d because the Southwest Plume was not delimited at the time the BHHRA was initiated. Generally, this results in combining data from known TCE sources with data collected downgradient from a source of inorganic chemicals (i.e., SWMU 8, C-746K Landfill). To address this uncertainty and to provide a concise risk characterization of the Southwest Plume, Attachment 11 to this BHHRA was prepared. As shown there, risks from the use of groundwater in the Southwest plume are similar to those reported for other areas. Therefore, this uncertainty had a small impact on the overall risk characterization.

To further examine the uncertainty in exposure point concentrations, the concentration of the contaminant determined to pose the greatest risk via the inhalation exposure route (i.e., vinyl chloride) was analyzed further. This analysis did not focus upon the emissions of vinyl chloride from water during use but did focus upon the possible presence of vinyl chloride in enclosed spaces due to soil vapor migration. Because previsous information concerning this condition did not exist, air sampling for vinyl chloride was condected in selected enclosed areas at the PGDP in spring 2000 (i.e., the underground cable tunnel from C-337 to C-300, the underground cable tunnel from C-331 to C-531, the underground tunnel from C-333 to the approximate location of the old millwright shop, and the C-400 basement). During this sampling, vinyl chlodride was not detected at any location and a level greater than the detection limit (0.85 ppm). Because the detection limit is below the Occupational Safety and Health Administration's eight-hour time weighted average limit (1 ppm), it is unlikely that this uncertainty had any effect upon the final risk values.

6.1.3 Determination of exposure point concentrations–future conditions

Uncertainty is involved in characterizing exposure point concentrations under future conditions in this BHHRA. However, because these uncertainties are related to the modeling performed to support the examination of migration from the various sources and because these uncertainties are covered in detail in Appendix A, the Data Summary Report, of the feasibility study report, they will not be discussed here. However, note that previous work has indicated that the effects of the modeling uncertainties on risk estimates tend to be moderate.

6.1.4 Use of concentrations from total versus filtered samples

In the main parts of the area assessment and the "well-by-well" analyses, 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 (Methods Document) but introduces an additional

uncertainty to the BHHRA due to potential sampling bias. While it is difficult to estimate the importance of this uncertainty because it is not known to what extent the quality of water (in terms of total solids) from a residential well would vary from the quality of water taken during the various sampling efforts, it is possible to quantify the reduction in risk offered by filtering. Exhibit 6.1 and Tables 6.5a through 6.5f present the risk estimates derived from the assessment of filtered data and compares these estimates to those derived from the assessment of total data. Note that the procedures used in deriving these risk estimates were those used for the total data. Risk tables for the filtered assessment are in Attachment 6.

As seen in Exhibit 6.1, results for metals from the assessment of filtered data were quite similar to those from the assessment of unfiltered water in most cases. In fact, in all but 1 of 36 cases, the change in the HI due to metals was less than 1 order of magnitude, and in all but 8 of 37 cases, the change in ELCR due to metals was less than 1 order of magnitude. While the difference for the HI exceptions was just in excess of 1 order of magnitude, the difference for some of the ELCR exceptions do exceed two orders of magnitude.

In each case where a marked difference between the filtered and unfiltered results are seen, the cause is the lack of detection in the filtered samples of a metal that drove risk in the assessment of unfiltered data. For example, a marked difference is seen in the HI results for Area b (McNairy Formation). As shown in Table 6.5c this difference is due to the lack of detection of antimony in the filtered sample. Similarly, the marked difference in the ELCR results for Area e (UCRS), Area a (RGA), Area f (RGA), Area g (RGA), Area h (RGA), Area j (RGA), and Area g (McNairy Formation) are due to the lack of detection of arsenic in the filtered sample. (See Tables 6.5d through 6.5f).

Although the differences are marked in some cases, the overall affect upon the total risk estimates is generally small in the GWOU BHHRA. This is the result of metals (i.e., inorganic chemicals) presenting little of the overall risk for the most heavily contaminated areas. For example, in Area a (UCRS) and Area a (RGA) risk due to metals (See Exhibit 6.1) makes up less than 1% of the total HI and ELCR. However, for some locations, the importance of this uncertainty is greater, and it should be considered in more detail when making risk-management decisions.

6.2 UNCERTAINTIES ASSOCIATED WITH EXPOSURE ASSESSMENT

Uncertainties associated with the exposure assessment are from four sources in the GWOU BHHRA. These are as follows:

- Biota fate and transport modeling,
- Use of the RME scenario,
- Development of the conceptual site model and selection of pathways, and
- Use of default values when estimating dermal absorbed dose.

Each of these uncertainties is discussed in the following material.

6.2.1 Uncertainties in biota fate and transport modeling

Modeling was used to estimate chemical concentrations and radionuclide activities in biota in this BHHRA. Although the models used in this assessment are industry standard models, the output from these models contain a considerable amount of uncertainty. To ensure that these models generated values which were unlikely to underestimate dose (i.e., were conservative values), default values were used in all cases. These conservative assumptions ensure that the risk values estimated tend to be conservative. However, this may result in risk estimates that overestimate the real risk.

Exhibit 6.1. Comparison of risk results (without lead) for the resident derived using unfiltered and filtered samples[1]

Area	Systemic Toxicity[2]			Excess Lifetime Cancer Risk[3]		
	Result Using Unfiltered Samples		Result Using Filtered Samples[6]	Result Using Unfiltered Samples		Result Using Filtered Samples[6]
	Total	Metals[4]		Total	Metals[4]	
UCRS Results						
Area a	7,000	8.6	2.1	5.9×10^{-2}	1.3×10^{-4}	8.6×10^{-5}
Area b	2,460	10.1	8.9	2.7×10^{-2}	5.6×10^{-4}	4.3×10^{-4}
Area c	2.3	1.2	0.6	8.6×10^{-5}	$<1\times 10^{-6}$	$<1\times 10^{-6}$
Area d	496	69.1	8.4	6.0×10^{-3}	2.7×10^{-4}	8.0×10^{-5}
Area e	9.9	9.8	3.5	2.1×10^{-4}	9.3×10^{-5}	$<1\times 10^{-6}$
Area f	2.2	2.1	0.4	3.5×10^{-4}	$<1\times 10^{-6}$	$<1\times 10^{-6}$
Area g	4.3	4.3	2.1	4.6×10^{-4}	$<1\times 10^{-6}$	$<1\times 10^{-6}$
Area h	2.2	2.2	1.7	2.0×10^{-4}	$<1\times 10^{-6}$	$<1\times 10^{-6}$
Area i	15.6	10.7	14.3	5.8×10^{-4}	1.9×10^{-4}	2.3×10^{-4}
Area j	NR	NR	NR	NR	NR	NR
Area k[5]	91.2	69.9	24.3	5.1×10^{-3}	6.5×10^{-4}	1.4×10^{-3}
Area l	3,090	14.8	7.8	2.9×10^{-1}	5.7×10^{-4}	3.6×10^{-4}
Area m	22.4	20.5	14.1	4.7×10^{-4}	1.1×10^{-4}	8.4×10^{-5}
Area n	2,370	19.3	16.0	2.9×10^{-1}	5.1×10^{-4}	3.2×10^{-4}
RGA Results						
Area a	38,800	4.7	3.2	2.9×10^{-1}	1×10^{-4}	$<1\times 10^{-6}$
Area b	262	6.1	3.8	7.0×10^{-2}	7.1×10^{-4}	5.7×10^{-4}
Area c	43.9	7.8	1.0	2.4×10^{-3}	$<1\times 10^{-6}$	$<1\times 10^{-6}$
Area d	78.8	5.2	4.9	1.3×10^{-3}	1.1×10^{-4}	6.1×10^{-4}
Area e	124	6.2	1.6	2.0×10^{-3}	6.4×10^{-4}	4.3×10^{-4}
Area f	74.0	8.5	2.4	1.6×10^{-3}	7.4×10^{-5}	$<1\times 10^{-6}$
Area g	5.7	5.6	2.3	5.4×10^{-4}	7.2×10^{-5}	$<1\times 10^{-6}$
Area h	7.2	7.0	0.9	3.3×10^{-4}	8.0×10^{-5}	$<1\times 10^{-6}$
Area i	37.3	25.6	8.8	1.7×10^{-3}	8.4×10^{-4}	4.0×10^{-4}
Area j	8.4	8.4	6.5	1.2×10^{-4}	1.2×10^{-4}	$<1\times 10^{-6}$
Area k[5]	–	–	–	–	–	–
Area l	1,470	7.3	4.2	2.6×10^{-1}	6.6×10^{-4}	5.6×10^{-4}
Area m	87.3	23.8	13.3	4.1×10^{-3}	7.6×10^{-4}	4.4×10^{-4}
Area n	812	22.4	14.0	1.1×10^{-1}	7.2×10^{-4}	4.8×10^{-4}
McNairy Formation Results						
Area a	NR	NR	NR	NR	NR	NR
Area b	69.5	19.1	1.0	4.4×10^{-4}	$<1\times 10^{-6}$	$<1\times 10^{-6}$
Area c	NR	NR	NR	NR	NR	NR
Area d	<1.0	<0.1	<0.1	1.2×10^{-6}	$<1\times 10^{-6}$	$<1\times 10^{-6}$
Area e	19.8	19.7	7.1	1.6×10^{-3}	1.4×10^{-3}	$<1\times 10^{-6}$
Area f	<1.0	0.2	0.1	$<1 \times 10^{-6}$	$<1 \times 10^{-6}$	$<1\times 10^{-6}$
Area g	<1.0	0.8	1.2	8.2×10^{-5}	7.1×10^{-5}	$<1\times 10^{-6}$
Area h	<1.0	0.4	NR	2.0×10^{-4}	$<1 \times 10^{-6}$	NR
Area i	3.1	3.1	2.9	$<1\times 10^{-6}$	$<1 \times 10^{-6}$	$<1\times 10^{-6}$
Area j	27.7	27.7	26.2	2.4×10^{-3}	2.4×10^{-3}	2.2×10^{-3}
Area k[5]	–	–	–	–	–	–
Area l	54.0	16.7	0.9	3.3×10^{-4}	$<1 \times 10^{-6}$	$<1\times 10^{-6}$
Area m	22.4	16.6	6.9	9.9×10^{-4}	8.1×10^{-4}	2.5×10^{-4}
Area n	38.6	24.6	5.1	1.1×10^{-3}	7.9×10^{-4}	2.0×10^{-4}

Note:

NR indicates that a result was not available for comparison.

"–" indicates that

[1] Results are for direct routes of exposure only.

[2] Totals do not include contribution from lead as a COPC.

[3] Values for ELCR greater than 1×10^{-2} fall outside the calculation bounds in EPA 1989a and are approximate values.

[4] Systemic toxicity (HI) and ELCR contributed by metals (i.e., inorganic chemicals) for the respective area.

[5] Area k includes water drawn from Eocene Sands, Terrace Gravels, and Porters Creek Clay. The results for this area are grouped with the UCRS for convenience.

[6] Results for filtered samples include contribution from inorganic chemicals only.

Exhibit 6.2 displays the total HIs and ELCRs estimated for the future recreational and residential use of water drawn from the RGA including and ignoring the HIs and ELCRs for biota consumption. (Note that all estimates of HI are for child exposures, and the effects of lead have been removed.) This exhibit shows that the effect of this uncertainty on the total HIs is small in all cases except that for assessment of Area m (Resident) where HI increases by an order of magnitude when the direct and biota routes are summed. Similarly, Exhibit 6.2 shows that the effect of this uncertainty on the total ELCR is small in all cases except Area i (Recreator) and Area m (Recreator) where ELCR increases by an order of magnitude when the direct and biota routes are summed.

Exhibit 6.2. Effect upon the risk results for the recreator and resident from summing the biota consumption exposure routes with those for the direct exposure routes[1]

Area	Systemic Toxicity[2]			Excess Lifetime Cancer Risk[3]		
	Direct Routes	Biota Routes	Cumulative Result	Direct Routes	Biota Routes	Cumulative Results
Recreator						
Area a	3,490	866	4,360	6.6×10^{-2}	3.8×10^{-2}	1.0×10^{-1}
Area b	25.8	9.1	34.9	3.9×10^{-3}	6.2×10^{-3}	1.0×10^{-2}
Area c	4.9	6.4	11.3	6.9×10^{-5}	5.1×10^{-5}	1.2×10^{-4}
Area d	7.6	5.0	12.6	1.4×10^{-4}	4.6×10^{-4}	6.0×10^{-4}
Area e	12.7	4.4	17.1	5.3×10^{-4}	6.2×10^{-4}	1.2×10^{-3}
Area f	9.4	6.3	15.7	1.2×10^{-4}	3.1×10^{-4}	4.3×10^{-4}
Area g	1.7	3.1	4.8	2.2×10^{-6}	1.4×10^{-5}	1.6×10^{-5}
Area h	1.6	4.1	4.7	2.4×10^{-6}	$<1 \times 10^{-6}$	2.4×10^{-6}
Area i	11.1	142	153	4.6×10^{-4}	5.1×10^{-3}	5.6×10^{-3}
Area j	1.3	2.9	4.2	3.4×10^{-6}	$<1 \times 10^{-6}$	3.4×10^{-6}
Area k[4]	10.8	46.3	57.1	3.8×10^{-4}	6.3×10^{-4}	1.0×10^{-3}
Area l	118	36.9	155	1.3×10^{-2}	1.6×10^{-2}	2.9×10^{-2}
Area m	15.1	141	156	5.2×10^{-4}	5.1×10^{-3}	5.6×10^{-3}
Area n	70.1	165	235	6.1×10^{-3}	1.2×10^{-2}	1.8×10^{-2}
Resident						
Area a	38,800	6,380	45,200	2.9×10^{-1}	1.0×10^{-1}	3.9×10^{-1}
Area b	262	49.2	311	7.0×10^{-2}	9.9×10^{-2}	1.7×10^{-1}
Area c	43.9	10.9	54.8	2.4×10^{-3}	2.8×10^{-3}	5.2×10^{-3}
Area d	78.8	15.0	93.8	1.3×10^{-3}	5.2×10^{-4}	1.8×10^{-3}
Area e	124	23.0	147	2.0×10^{-3}	5.7×10^{-3}	7.7×10^{-3}
Area f	74.0	15.9	89.9	1.6×10^{-3}	4.5×10^{-4}	2.1×10^{-3}
Area g	5.7	3.2	8.9	5.4×10^{-4}	3.0×10^{-4}	8.4×10^{-4}
Area h	7.2	4.3	11.5	3.3×10^{-4}	2.5×10^{-4}	5.8×10^{-4}
Area i	37.3	25.1	62.4	1.7×10^{-3}	2.1×10^{-3}	3.8×10^{-3}
Area j	8.4	4.3	12.7	1.2×10^{-4}	7.1×10^{-5}	1.9×10^{-4}
Area k[4]	91.2	46.6	137.8	5.1×10^{-3}	2.3×10^{-3}	7.4×10^{-3}
Area l	1,470	276	1,750	2.6×10^{-1}	3.3×10^{-1}	5.9×10^{-1}
Area m	87.3	32.6	120	4.1×10^{-3}	3.3×10^{-3}	7.4×10^{-3}
Area n	812	171	983	1.1×10^{-1}	1.5×10^{-1}	2.6×10^{-1}

[1] Results are for exposure to water drawn for the RGA except for Area k.
[2] Totals do not include contribution from lead as a COPC and are for the child.
[3] Values for ELCR greater than 1×10^{-2} fall outside the calculation bounds in EPA 1989a and are approximate values.
[4] Area k includes water drawn from Eocene Sands, Terrace Gravels, and Porters Creek Clay.

6.2.2 Uncertainties in use of reasonable maximum exposure scenarios

For each exposure pathway modeled, assumptions were made about the number of times a year an activity could occur, the routes of exposure, and the rate of intake of contaminated media. Because site-specific data were not available for most of these parameters, suggested EPA and Commonwealth of Kentucky defaults were used. Because most of these defaults are conservative to prevent the underestimation of risk estimates, the risk estimates tend to be conservative. Generally, when several upper-bound values are combined, the resulting 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, then the resulting total dose overestimates total dose.

To examine the potential effect of this uncertainty in past BHHRAs completed for the PGDP, ELCRs and HIs for the residential scenario were also 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, Review Draft*. This report is presented in the Method Document.) In those assessments, all exposure pathways evaluated were identical to those used in the RME scenarios, and all exposure equations, chemical concentrations, radionuclide activities, and toxicity values were identical to those used for the RME scenario. These assessments demonstrated that risk estimates change little when average exposure parameters versus RME exposure parameters are used. Therefore, it is assumed that the effect of this uncertainty upon the risk estimates is small, as was the case in the earlier assessments.

6.2.3 Uncertainties related to development of the site conceptual models

Generally, the level of uncertainty in the development of the site conceptual models is small. Data were collected from several previous studies and from local experts to develop these models. However, there are some uncertainties related to specific scenarios that deserve additional explanation. These uncertainties are the consideration or lack of consideration of specific pathways for some scenarios and the summation of risks across areas and across scenarios.

An uncertainty related to the consideration of specific pathways for some scenarios is the assessment of groundwater ingestion by the industrial worker and resident of water drawn from directly below the PGDP. While this is not a current use of groundwater at the PGDP, these exposure routes were included to provide risk managers with additional information about the potential risk posed by groundwater at GWOU. Specifically, this pathway was included even though PGDP does not currently use groundwater and does not plan to use groundwater in the future.

Another uncertainty to consider here is the effect of not considering exposure to contaminated soil at source units. As discussed previously, this was not done because this would have repeated the work performed in the previous source BHHRAs (see Sec. 1.). However, it must be recognized that risks at source units would be greater if exposure to contaminated soil and waste was considered.

Finally, there is some uncertainty in the biota pathways considered in the risk assessment. Pathways considered were consumption of produce from home gardens, consumption of meat from livestock (beef, pork, and poultry), consumption of milk from cows, consumption of eggs from laying hens, consumption of meat from game (deer, rabbit, and quail), and consumption of fish. Other pathways that may be important at the PGDP, but that were not quantified because information needed to quantify exposure was inadequate, were consumption of meath from turtles and amphibians. If these pathways had been included, risk estimates for the recreational user consuming meat from these biota would have been greater.

The primary uncertainty related to summarizing information is the effect of summarization upon the identification of "hot spots" or areas of very high contamination. To address this concern, an assessment of individual sampling stations (i.e., "well-by-well" assessment) was performed. This assessment allowed for a check of the area assignments and for the identification of individual hot spots. Therefore, the estimated effect of this uncertainty upon the BHHRA's results is small.

6.2.4 Uncertainties related to use of default values when estimating dermal absorbed dose

In this assessment, the default dermal absorption factors (i.e., permeability constants) for water provided by EPA guidance were used. While there is some uncertainty in these permeability constants, there is less for these parameters than the uncertainty in absorption values for soil. Additionally, there is no disagreement between the permeability constants recommended by the Commonwealth of Kentucky and those recommended by EPA. Therefore, in the GWOU BHHRA, the uncertainty related to dermal absorption is estimated to be small.

6.3 UNCERTAINTIES ASSOCIATED WITH TOXICITY ASSESSMENT

Uncertainties related to the toxicity assessment are from three sources. These are uncertainty due to 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.

6.3.1 Uncertainties due to lack of toxicity values for some chemicals

Uncertainties due to lack of toxicity values for some chemicals results 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 had a significant effect on the results of the BHHRA. Some COPCs did not have approved toxicity values, so a provisional or withdrawn value was used. Notable among these COPCs are lead and TCE. For lead, a provisional RfD was provided by KDEP in a comment package on the WAG 17 RI/BHHRA. As shown in the exhibits in Sect. 5, the systemic toxicity posed by lead dominates all land use scenarios in those sectors where lead was detected. For better interpretation of the systemic toxicity results for the rest of the COPCs in the BHHRA, results with and without contributions from lead are provided. For TCE, the standard practice at the PGDP is to use the withdrawn TCE toxicity values in assessments because TCE is the dominant organic compound contaminant found in groundwater and not including TCE in the assessment of risks would be inappropriate. In any case, the effect of using these provisional and withdrawn values on the final risk estimates was large. (See Table 7.1 and 7.2 for additional consideration of this uncertainty.)

In some earlier BHHRAs completed for the PGDP, for the PCBs (e.g., Aroclor 1254, 1260, etc.), there was uncertainty in the selection of the appropriate toxicity value for individual Aroclors because of difficulty in the identification of specific Aroclors in the laboratory, the differential media accumulation of the Aroclors over time, and weathering processes which alter Aroclors over time making the Aroclors appear to be more chlorinated than they really are. To address these concerns and to ensure that the risk estimates for Aroclors were conservative, KDEP required that all PCBs be evaluated as Aroclor 1260. The GWOU BHHRA is consistent with KDEP guidance because in this assessment the cancer toxicity values for all Aroclors were assumed to be equal to 2.0 mg/(kg-day). (See Sect. 4.) Therefore, unlike the earlier BHHRAs performed at PGDP where the effect of the uncertainty in the selection of the appropriate

toxicity values for PCBs on the final risk values may have been moderate, the effect of this uncertainty on the final risk values in the current assessment on the final risk number is small.

Including inhalation toxicity values extrapolated from toxicity values based on administered doses in the risk characterization would not have significantly affected the results of the GWOU BHHRA. While EPA guidance recommends against extrapolating between oral and inhalation toxicity values because of the differing path a chemical entering through the lungs must follow before exerting its effect versus entry through the gut, examination of this form of extrapolation as an uncertainty in assessments for the PGDP was requested by the regulatory community. Previous work at the 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.

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. The EPA has working groups that 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 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.

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 can cause a toxic or cancer 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 due to 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 assumed to be small.

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 subsections.

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 chemical-specific ELCRs over pathways and combine pathway HIs and ELCRs to calculate total HI and ELCR. The uncertainties in this method are discussed in the following text.

The method used to calculate pathway HIs and ELCRs in the BHHRA followed EPA protocols (Methods Document). This guidance calls for the simple summation 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. Specific 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, it is possible that the interaction of multiple chemicals could 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 potential for at least noncarcinogenic effects to occur can be overestimated for chemicals that act by different mechanisms and on different target organs.

Therefore, the effect of this uncertainty on the estimate of systemic toxicity depends on how many contaminants drive systemic toxicity and if 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 in Sect. 5, individual contaminants alone contribute significant levels of risk for each scenario, and the HI associated with the single contaminant alone is great enough that a systemic toxic effect may be reasonably expected. Therefore, the effect of this uncertainty on HIs is small.

Specific limitations for this approach in regard to chemical carcinogenesis have also been reported by EPA in 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. Similar to HI, 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. 4, EPA recognizes this by placing weight-of-evidence classifications on all slope factors. Those contaminants with an A weight-of-evidence 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.

Therefore, 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. Therefore, the potential effect of mixtures is reduced.

6.4.2 Combining risks from chemicals with those from radionuclides

Some uncertainty is associated with adding risks from chemical exposure to those from exposure to radionuclides. This uncertainty arises from two sources. First, as noted in Subsect. 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 Subsect. 4). This difference in mechanism of action inflates the uncertainties discussed in Subsect. 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 Subsect 6.4.1, generally one COC drives the risks. Where multiple chemicals and radionuclides drive risk, the effect of this uncertainty could be moderate.

6.5 SUMMARY OF UNCERTAINTIES

As is shown in the previous subsections, the risk estimates could vary considerably if different assumptions were used in deriving the risk estimates or if better information was available for some parameters. Exhibit 6.3 summarizes the estimated effects of each uncertainty mentioned previously.

Note that the only uncertainty with an effect estimated to be large is the use of the provisional toxicity values. Because this uncertainty was identified as being large and the effect for exposure to lead was easy to quantify, it receives greater attention in summary discussions than other uncertainties discussed in this BHHRA. This attention is not meant to imply that the authors believe that the provisional toxicity should not have been used in this assessment.

Exhibit 6.3. Summary of uncertainties

Description of Uncertainty	Estimated Effect[1]		
	Small	Moderate	Large
Uncertainties related to data and data evaluation			
Inclusion of infrequently detected analytes	X		
Inclusion of infrequently analyzed for analytes	X		
Lack of consideration of temporal patterns in detection of analytes	X		
Quantitation limits for some analytes exceeding their respective human health risk-based concentrations	X		
Inclusion of common laboratory contaminants	X		
Lack of comparison of analyte concentrations to concentrations in associated blanks	X		
Removal of analytes from the COPC list on the basis of a toxicity screen	X		
Removal of analytes from the COPC list on the basis of a background comparison	X		
Characterization of exposure point concentrations for environmental media under current conditions	X		
Characterization of exposure point concentration for environmental media under future conditions		X	
Use of results from total versus filtered samples	X		
Uncertainties related to exposure assessment			
Incorporation of biota fate and transport modeling into risk estimates		X	
Use of RME parameters versus average parameters for all exposure routes and pathways	X		
Summation of risk across areas and across scenarios	X		
Uncertainties related to toxicity assessment			
Use of provisional toxicity values for the systemic toxicity of lead			X
Use of provisional or withdrawn toxicity values for systemic toxicity and ELCR		X	
Route-to-route extrapolation in derivation of toxicity values	X		
Derivation of toxicity values			
Chemicals		X	
Radionuclides	X		
Selection of toxicity values for PCBs	X		
Calculation of absorbed dose toxicity values from administered dose toxicity values	X		
Uncertainties related to risk characterization			
Combination of chemical –specific risk values to yield route-specific risk values	X		
Combination of route-specific risk values to yield cumulative (total) risk values	X		
Combination of chemical-specific cumulative risk values with radionuclide-specific cumulative risk values to yield total risk values	X		

[1] Definitions of effects are:
Small = Uncertainty should not cause the risk estimate to vary by more than one order of magnitude.
Moderate = Uncertainty may cause the risk estimate to vary by between one and two orders of magnitude.
Large = Uncertainty may cause the risk estimate to vary by over two orders of magnitude.

7. SUMMARY AND CONCLUSIONS

This section summarizes the results of the risk assessment and draws conclusions from the results. Although 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 derived by combining the results of the risk assessment with the uncertainties affecting the GWOU BHHRA. The compilation of these observations begins the risk management process that continues in Sect. 1.2.6 of the FS Report.

7.1 DATA EVALUATION AND SELECTION OF COPCS

Chemicals of potential concern were selected from data derived from unfiltered groundwater samples collected at and around the PGDP since 1993. This groundwater data set was screened to produce a final list of COPCs grouped by geographical area (Areas a through n), depth of sampling (UCRS, RGA, McNairy, and "other"), and method of sample collection (monitoring well, driven rod, faucet, and boring). Geographical areas were defined as follows:

- Area a – Inside TCE contaminated area at C-400 Building – Inside industrialized area
- Area b – Inside the Northwest TCE Plume – Inside industrialized area (i.e., west main plant)
- Area c – Inside the Northeast TCE Plume – Inside industrialized area (i.e., east main plant)
- Area d – Outside the TCE Plumes – South of C-400 in industrialized area
- Area e – Inside the Northwest TCE Plume – Outside industrialized area
- Area f – Inside the Northeast TCE Plume – Outside industrialized area
- Area g – Outside the TCE Plumes – West of industrialized area (i.e., west of plume)
- Area h – Outside the TCE Plumes – East of industrialized area (i.e., east of plume)
- Area i – Outside the TCE Plumes – North of industrialized area (i.e., between the plumes)
- Area j – Outside the TCE Plumes - Tennessee Valley Authority area (TVA)
- Area k – Outside the TCE Plumes – South of industrialized area above terrace
- Area l – Inside plant area – Composed of Areas a, b, c, and d
- Area m – Outside plant area – Composed of Areas e, f, g, h, i, j, and k
- Area n – All groundwater – Composed of Areas l and m

Areas a through k were developed to ensure that the summary statistics (i.e., average contaminant concentrations) derived in the GWOU BHHRA were comparable to those developed during the BHHRAs previously completed as part of the investigations of the Northwest Plume and to let the GWOU BHHRA create lists of COCs for specific areas at and around the PGDP. Areas l through n were used to investigate the average risk posed through use of water drawn from the larger areas to let this BHHRA develop plant-wide lists of COCs.

In addition to the area assessment, which used the aforementioned data aggregates, two additional assessments requiring different data sets were completed. The first of these was a "well-by-well" assessment that was completed using summary statistics developed from groundwater results collected at each sampling station. This assessment was performed to examine uncertainties in the area assessment due to data summarization. The second of these was a risk assessment based upon future modeled concentrations. This assessment was performed to examine the potential contribution to risk at four integration points (i.e., PGDP security fence, PGDP property boundary, at Little Bayou Creek, and near the Ohio River) from sources of contamination identified in previous source unit investigations.

The number of COPCs identified for each area and depth classification combination varied markedly in the area assessment. Over the geographical areas, Areas a through d and Area i and k had the greatest number of COPCs, and Areas e through h and j had the fewest. Over depth classifications, the UCRS tended to have more COPCs than other depths for the areas inside the fence and in Areas i and k, but the RGA tended to have more COPCs than other depths for areas outside the fence. The McNairy Formation had the fewest COPCs in all areas.

Over all areas, the majority of the COPCs were inorganic chemicals followed in number by organic compounds and radionuclides. Specifically, for Area n (UCRS) groundwater, 37 COPCs were inorganic chemicals, 25 COPCs were organic compounds, and 15 COPCs were radionuclides; for Area n (RGA) groundwater, 38 COPCs were inorganic chemicals, 38 COPCs were organic compounds, and 15 COPCs were radionuclides; and, for Area n (McNairy Formation) groundwater, 24 COPCs were inorganic chemicals, 1 COPC was an organic compound, and 8 COPCs were radionuclides. For Area k (groundwater collected to the south of the PGDP), 29 COPCs were inorganic chemicals, 11 COPCs were organic compounds, and 10 COPCs were radionuclides.

7.2 EXPOSURE ASSESSMENT

Historical information and newly collected data were used to develop a conceptual site model for the GWOU. After consideration of all data, the scenarios selected for assessment in the area BHHRA were the industrial worker, recreational user, and rural resident. While current exposure to groundwater at all locations except Little Bayou Creek was determined to be unlikely, each of the scenarios was assumed to be equally likely under future conditions. The exposure routes assessed under each of the scenarios for the area BHHRA did not differ with geographical area or depth of sampling. These exposure routes are summarized in the following material.

Industrial worker

- ingestion of groundwater,
- dermal contact with groundwater while showering, and
- inhalation of vapors emitted by groundwater while showering.

Recreational user

- incidental ingestion of water while swimming in ponds filled with groundwater,
- dermal contact with water while swimming in ponds filled with groundwater,
- dermal contact with water while wading in ponds filled with groundwater,
- consumption of fish raised in ponds filled with groundwater,
- consumption of venison from deer drinking groundwater,
- consumption of meat from rabbits drinking groundwater, and
- consumption of meat from quail drinking groundwater.

Rural resident

- 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,
- consumption of vegetables,

- consumption of beef from cows drinking groundwater,
- consumption of milk from cows drinking groundwater,
- consumption of meat from chickens and turkeys drinking groundwater,
- consumption of eggs from chickens drinking groundwater, and
- consumption of pork from swine drinking groundwater.

For the "well-by-well" assessment and the assessment that used future modeled concentrations, only the rural resident was assessed in order to limit the results to a manageable size.

After selection of the exposure routes, chronic daily intakes (i.e., chronic doses) 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.

7.3 TOXICITY ASSESSMENT

The toxicity values used in the risk assessment were those approved by the United States Environmental Protection Agency or recommended for use by the Commonwealth of Kentucky Department of Environmental Protection. After compiling toxicity information, it was determined that the majority of the COPCs had a toxicity value available for one or more routes of exposure.

7.4 RISK CHARACTERIZATION

Risks were characterized by integrating the chronic daily intakes calculated during the exposure assessment and the toxicity values collected during the toxicity assessment. As a result of this characterization, it was determined that there are unacceptable risks associated with exposure to groundwater from virtually all area and depth classifications. Significant results of the risk characterization for the area assessment are presented below.

7.4.1 Land use scenarios of concern

For the area assessment, not all area/depth classifications were found to have land use scenarios of concern for both systemic toxicity and ELCR. However, the RGA was found to be of concern for all uses in all areas, and the UCRS was found to be of concern for residential and industrial use in all areas where data were available and for recreational use in all but Areas c, f, h, and j.

The McNairy Formation had more areas where the land uses assessed were not of concern than the UCRS and RGA. Under the industrial worker scenario, Areas a, c, d, f, and i, were not of concern; under the recreational user, Areas a, c, d, f, h, and i were not of concern; and under the rural resident, Areas a, b, and f were not of concern. (Note that data were not available for the McNairy Formation in Areas a and b. Also, the McNairy Formation did not apply to Area k.)

Area k (i.e., groundwater taken to the south of the PGDP) was of concern for each land use for systemic toxicity and ELCR.

7.4.2 Contaminants of concern

Multiple COCs were found for each of the land uses. For the UCRS, over all areas and land uses, a total of 36 COCs for systemic toxicity and 21 COCs for ELCR were identified (including Area k). Of the COCs for systemic toxicity, 21 were inorganic chemicals and 15 were organic compounds. Of the COCs for ELCR, 2 were inorganic chemicals, 9 were organic compounds, and 10 were radionuclides.

Combining the results for systemic toxicity and ELCR and considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs were identified as "priority COCs" in UCRS groundwater across all use scenarios (excluding Area k):

- Inorganic chemicals – arsenic, antimony, beryllium, cadmium, chromium, iron, lead, manganese, nickel, and vanadium.

- Organic compounds – 1,1-dichloroethene, benzene, chloroform, ethylbenzene, naphthalene, *trans*-1,2-dichloroethene, *cis*-1,2-dichloroethene, TCE, and vinyl chloride.

- Radionuclides – ^{222}Rn.

For Area k, the "priority COCs" in groundwater across all use scenarios were:

- Inorganic chemicals – antimony, beryllium, cadmium, iron, lead, manganese, and vanadium

- Organic compounds – 1,1-dichloroethene, 1,2-dichloroethene, naphthalene, *cis*-1,2-dichloroethene, TCE, and vinyl chloride.

- Radionuclides – ^{222}Rn.

For the RGA, over all areas and land uses, a total of 38 COCs for systemic toxicity and 28 COCs for ELCR were identified. Of the COCs for systemic toxicity, 19 were inorganic chemicals and 21 were organic compounds. Of the COCs for ELCR, 2 were inorganic chemicals, 17 were organic compounds, and 9 were radionuclides.

Combining the results for systemic toxicity and ELCR and considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs were identified as "priority COCs" in RGA groundwater across all use scenarios:

- Inorganic chemicals – antimony, arsenic, beryllium, cadmium, chromium, iron, lead, manganese, molybdenum, and vanadium.

- Organic compounds – 1,1-dichloroethene, acrylonitrile, carbon tetrachloride, Aroclor-1254, tetrachloroethene, *cis*-1,2-dichloroethene, *trans*-1,2-dichloroethene, TCE, and vinyl chloride

- Radionuclides – ^{226}Ra and ^{222}Rn.

For the McNairy Formation, over all areas and land uses, a total of 15 COCs for systemic toxicity and 7 COCs for ELCR were identified. Of the COCs for systemic toxicity, 14 were inorganic chemicals and 1 was an organic compound. Of the COCs for ELCR, 2 were inorganic chemicals, 1 was an organic compound, and 4 were radionuclides.

Combining the results for systemic toxicity and ELCR considering the magnitude of the chemical-specific HIs and ELCRs, the following COCs were identified as "priority COCs" in McNairy Formation groundwater across all use scenarios:

- Inorganic chemicals – antimony, arsenic, beryllium, cadmium, chromium, iron, manganese, molybdenum, and vanadium.

- Organic compounds – TCE.
- Radionuclides – ^{222}Rn.

(Note that "priority COCs" are those that present either a chemical-specific HI or ELCR at one or more areas, across all land uses, that exceeds 1 or 1×10^{-4}, respectively.)

7.4.3 Pathways of concern

All direct contact exposure routes (i.e., those involving ingestion, dermal contact, and inhalation) and the sum of the biota consumption exposure routes were of concern for at least one area/depth classification combination. However, specific biota consumption routes were determined to not be of concern for some areas. Biota consumption routes for the recreational user not of concern in any area were consumption of venison, rabbit, and quail. Biota consumption routes for the resident not of concern in any area were consumption of eggs and consumption of pork. Biota consumption routes for the recreational user and resident that were of concern for virtually all area and depth classification combinations were consumption of fish and consumption of vegetables, respectively.

7.5 OBSERVATIONS

This section presents observations based on the risk results and uncertainties discussed in the previous sections and begins the risk management process that continues in Sect. 1.2.6 of the FS Report. Note that results in this section focus upon the results of the rural residential scenario because these results receive the greatest attention in the GWOU FS Report. Also note that these observations were made after removing the contribution of lead to total systemic toxicity. As discussed previously, the use of the provisional lead RfD provided by KDEP resulted in total HIs that that exceeded 1,000 for some area/depth classification combinations. However, when this provisional value was not included in the risk characterization, total HIs were often an order of magnitude or more smaller. Because the total HIs calculated using the provisional lead RfD were dominated by the HI of lead, a quantitative uncertainty analysis in which contributions from lead are not included and should be considered when examining the hazards presented by other COCs.

Due to the uncertainty in the provisional lead RfD, the risk presented by lead may be better understood using comparisons to regulatory agency screening values and results of EPA's IEUBK lead model. These comparisons show that lead concentrations in unfiltered groundwater drawn from many area/depth classification combinations are unacceptable. Specific area/depth classifications determined to have unacceptable lead concentrations in unfiltered water by the IEUBK model were Area a (UCRS), Area b (RGA), Area d (UCRS and RGA), Area g (RGA), Area i (UCRS), Area k (all), Area l (UCRS and RGA), Area m (UCRS and RGA), and Area n (UCRS and RGA).

Area/depth classifications determined to be of concern differed only slightly when selected using the benchmarks in the Methods Document (i.e., total HI > 1 and total ELCR > 1×10^{-6}) versus using EPAs generally acceptable risk range (i.e., total HI > 1 and total ELCR > 1×10^{-4}) (EPA 1999c). Under the benchmarks used in the Methods Document, the following area/depth classifications were determined to have unacceptable risk for the residential use scenario: Area a (UCRS and RGA), Area b (all), Area c (UCRS and RGA), Area d (all), Area e (all), Area f (UCRS and RGA), Area g (all), Area h (all), Area i (all), Area j (RGA and McNairy Formation), Area k (all), Area l (all), Area m (all), and Area n (all). Under EPAs generally acceptable risk range, the following area/depth classifications were determined to have unacceptable risk for the industrial worker use scenario: Area a (UCRS and RGA), Area b (all), Area c (UCRS and RGA), Area d (UCRS and RGA), Area e (all), Area f (UCRS and RGA), Area g

(UCRS and RGA), Area h (all), Area i (all), Area j (RGA and McNairy Formation), Area k (all), Area l (all), Area m (all), Area n (all). (Note that these results are for direct contact exposure routes and for the case where contribution from lead to systemic toxicity is not considered.)

When considered as a group, the uncertainties (including that involving lead) have effects upon the final total risk estimates that are significant in some cases. These effects are demonstrated in Tables 7.1 and 7.2 where the HIs and ELCRs derived after addressing each uncertainty are presented. For example, as shown in Table 7.1, total or cumulative HI for Area a (RGA) decreases from 39,000 before addressing any uncertainties to 22.0 after removing the contribution of lead and contribution from COPCs that have provisional or withdrawn toxicity values, that are common laboratory contaminants, and that were infrequently detected. However, also as shown in Table 7.1, total or cumulative HI for Area i (RGA) changes little after addressing each of the uncertainties (i.e., 37.0 versus 33.0). Similar results for ELCR are in Table 7.2.

An exposure route found to contribute significantly to total HI and ELCR in this assessment, unlike some earlier assessments performed for the PGDP, is risk from inhalation of vapors emitted from groundwater during showering and household use. As demonstrated in Sect. 5, this exposure route was significant for all area/depth classifications where TCE and its breakdown products were detected at high concentrations and where ^{222}Rn was detected in groundwater at concentrations greater than background.

Although most COCs for groundwater identified in the area risk assessment match well with those identified in earlier source and integrator unit BHHRAs, one COC deserves special attention because it was not identified as being important in the earlier assessments and because it does not appear to be related to the common data problems (i.e., sampling bias, frequency of detection, or frequency of analysis). This COC is ^{222}Rn. As can be seen in the exhibits in Sect. 5, ^{222}Rn drives ELCR for those area/depth classifications where TCE and its breakdown products were not present at significant concentration. In fact, ^{222}Rn is an important risk driver for the resident in Area d (RGA), Area e (all), Area f (UCRS and RGA), Area g (UCRS and RGA), Area h (all), Area i (UCRS and RGA), Area k, Area m (UCRS and McNairy Formation), and Area n (McNairy Formation). To examine this phenomenon in more detail, detected concentrations of ^{222}Rn in groundwater were summarized and compared to background concentrations and human health RBCs (see Exhibit 7.1.). As shown in that exhibit, the ^{222}Rn do not appear to be higher onsite than offsite in the RGA when the comparison is between all areas inside the security fence (i.e., 430 pCi/l, Area l) and all areas outside the security fence (i.e., 447 pCi/l, Area m). Also, when RGA ^{222}Rn concentrations are summarized within area, the two highest exposure concentrations (1,450 pCi/l, Area c and 753 pCi/l, Area d) and the lowest exposure concentration (242 pCi/l, Area b) were those for areas inside the security fence. (Note that ^{222}Rn was not a COPC for Area a because the maximum detected concentration was less than the RGA background concentration.) These results indicate that the identification of ^{222}Rn as a priority COC is probably an artifact of the data analysis; however a specific cause cannot be identified. This conclusion regarding anomalous results is further supported by results in a study entitled *Paducah Groundwater Contamination, Detailed History and Summary of Future Actions* (MMES 1988). In this study, residential and monitoring wells were sampled for ^{222}Rn from 1 to 4 times over a 3-month period. The results from residential wells ranged from 143 to 481 pCi/liter (ignoring 2 outlier samples with values of 740 and 1,309 pCi/liter). The results from monitoring wells located on the PGDP ranged from 194 to 340 pCi/liter. Using these results and a comparison to results published by EPA for samples from public supply wells located near the PGDP (LaCenter 391 pCi/liter and Metropolis 550 pCi/liter), the report concludes that ^{222}Rn found "in the plant aquifer is unrelated to plant operations."

Like ^{222}Rn's contribution to total ELCR, which appears to be related to sampling bias, the contribution of arsenic and beryllium to total ELCR also appears to be related to sampling as does the contribution of several other inorganic chemicals to total HI. As shown in Table 6.5a through 6.5f, the contribution of inorganic chemicals to total HI and total ELCR decreases markedly if results of filtering

Exhibit 7.1. Concentrations of ^{222}Ra (μg/L) by area and comparison to human health risk-based concentrations and background concentrations[1]

Location	Frequency of Detection	Exposure Concentration	Minimum Detected Concentration	Maximum Detected Concentration	COPC?[2]
UCRS (Background concentration is not available; RBC = 1.4 pCi/l)[3]					
Area a	1/1	461	461	461	Yes
Area b	38/38	1,040	12.0	2,050	Yes
Area c	0/0	NA	ND	ND	No
Area d	5/5	453	135	512	Yes
Area e	21/21	165	40	253	Yes
Area f	1/1	471	471	471	Yes
Area g	7/7	630	372	695	Yes
Area h	1/1	268	268	268	Yes
Area i	5/5	469	288	519	Yes
Area j	0/0	NA	ND	ND	No
Area k (Terrace[4])	30/30	895	9.0	1,310	Yes
Area l	44/44	957	12.0	2,050	Yes
Area m	35/35	336	40.0	695	Yes
Area n	79/79	806	12.0	2,050	Yes
RGA (Background concentration = 640 pCi/l; RBC = 1.4 pCi/l)[3]					
Area a	4/4	NA	278	604	No
Area b	247/247	242	11.0	2,230	Yes
Area c	16/16	1,450	236	6,590	Yes
Area d	44/44	753	71.0	9,480	Yes
Area e	255/255	401	51.0	861	Yes
Area f	13/13	528	257	848	Yes
Area g	138/138	630	55.0	1,970	Yes
Area h	57/57	336	0.8	1,060	Yes
Area i	30/30	574	208	930	Yes
Area j	0/0	NA	ND	ND	No
Area l	311/311	430	11.0	9,480	Yes
Area m	498/499	447	0.8	1,970	Yes
Area n	809/810	431	0.8	9,480	Yes
McNairy Formation (Background concentration = 291 pCi/l; RBC = 1.4 pCi/l)[3]					
Area a	0/0	NA	ND	ND	No
Area b	31/31	NA	22	291	No
Area c	0/0	NA	ND	ND	No
Area d	13/13	NA	37.0	145	No
Area e	31/31	277	143	391	Yes
Area f	4/4	NA	173	267	No
Area g	9/9	NA	67.0	178	No
Area h	9/9	261	130	333	Yes
Area i	1/1	NA	64	64	No

Exhibit 7.1. Concentrations of ^{222}Ra (μg/L) by area and comparison to human health risk-based concentrations and background concentrations[1] (continued)

Location	Frequency of Detection	Exposure Concentration	Minimum Detected Concentration	Maximum Detected Concentration	COPC?[2]
Area j	0/0	NA	ND	ND	No
Area l	44/44	NA	22.0	291	No
Area m	54/54	239	64.0	391	Yes
Area n	98/98	187	22.0	391	Yes

Notes: NA indicates that lead is not a COPC for the area. Therefore, a representative concentration is not available.
ND indicates that lead was not detected in any sample. Check the frequency of detection column to determine if analyses for lead were performed on any samples.

[1] The minimum and maximum concentrations were taken from Table 2.5. The exposure concentration is the lesser of the maximum detected concentration and the upper 95% confidence level on the mean concentration and is taken from Table 8.1.
[2] ^{222}Ra was selected as a COPC if the maximum exposure concentration exceeded both the background concentration and the RBC.
[3] Background values are for total or unfiltered samples and are from App. D of the GWOU FS Report. The RBC is taken from those used to perform the toxicity screen performed earlier in this baseline human health risk assessment.
[4] Area k includes water drawn from wells completed in Terrace Gravels, Eocene Sands, and Porters Creek Clay.

are considered. For example, with filtering, contributions from arsenic and beryllium to total ELCR decreases to below *de minimis* levels for Area a (RGA), Area e (UCRS), Area f (RGA), Area g (RGA and McNairy Formation), Area h (RGA), and Area j (RGA). Therefore, the importance of the inorganic chemicals in the total HI and ELCR estimates may be an artifact of sampling and not be real.

With integration of the risk results and uncertainties, the conclusion reached during the earlier Site Investigation Phase II risk assessment are valid for this GWOU BHHRA as well. In general, the contamination problem posing the greatest risk is from use of groundwater at the PGDP is the presence of TCE and its breakdown products in the aquifer. Although several inorganic chemicals and some radionuclides contribute significantly to total risk, these contaminants may be related to sampling or other biases and be of less relative importance. However, the other contaminants and contamination in source areas needs to be considered when developing remedies for groundwater contamination and its sources at the PGDP because modeling results indicate that unacceptable risks may develop if contaminants are allowed to continue to migrate from these source areas. However, because the modeling results are very uncertain, the appropriate risk management decision may be to address the TCE contamination in the short-term.

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8. REMEDIAL GOAL OPTIONS

This section presents RGOs for the COCs identified in Sect. 5 and the methods used to calculate the RGOs. These RGOs should not be interpreted as clean-up goals but as risk-based values that may be used to guide the development of clean-up goals by risk managers. Clean-up goals are determined and finalized in decision documents and not in this risk assessment. RGOs were calculated for groundwater at each location. Where ingestion rates differed between adults and children, the more conservative child ingestion rates were used in the calculation of the RGOs. In addition, the MCLs for the COCs affecting each receptor are in an exhibit presented at the end of this section. Note that MCLs are also not clean-up criteria. The National Contingency Plan 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.

8.1 CALCULATION OF RGOS

EPA guidance directs that RGOs are to be calculated for all COCs identified in a baseline risk assessment. The COCs identified in this risk assessment and their RGOs are presented in Tables 8.1, 8.2, and 8.3. The program used to calculate these RGOs is Program 12 in Attachment 3 of the BHHRA.

EPA guidance (EPA 1991) 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 1×10^{-4}, 1×10^{-5}, and 1×10^{-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. While 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.

$$\frac{\text{Concentration}}{\text{Risk}} = \frac{\text{RGO}}{\text{Target Risk}}$$

where:

Concentration is the exposure concentration for the medium.
Risk is the risk posed by exposure to the contaminated medium.
RGO is the remedial goal option.
Target Risk is one of the values listed above.

8.2 PRESENTATION OF RGOS

The equation developed in the previous subsection was applied to each COC. The RGOs developed for all land use scenarios of concern, POCs, and COCs, for the BHHRA using this equation are presented in Tables 8.1, 8.2, and 8.3 along with each COCs representative (i.e., exposure) concentration. In addition, Exhibit 8.1 provides the MCLs for COCs over all areas and receptors. The MCLs were taken from the RAIS accessed on February 5, 2000.

Exhibit 8.1. Maximum contaminant levels (MCLs) for COCs identified in the GWOU BHHRA for the Paducah Gaseous Diffusion Plant, Paducah, Kentucky

Chemical	Federal and State Primary Drinking Water MCLs µg/L	Federal and State Primary Drinking Water MCLs (Radionuclides) pCi/L	Federal and State Primary Drinking Water Proposed MCLs (Radionuclides) pCi/L	Federal Secondary Drinking Water SMCLs µg/L	State Secondary Drinking Water SMCLs µg/L
Acrylonitrile	-	-	-	-	-
All other manmade radionuclides	-	4 mrem/yr[5]	4 mrem/yr[5]	-	-
Aluminum	-	-	-	50-200	50-200
Antimony	6	-	-	-	-
Aroclor 1254	-	-	-	-	-
Arsenic	50	-	-	-	-
Barium	2000	-	-	-	-
Benzene	5	-	-	-	-
Beryllium	4	-	-	-	-
Bromodichloromethane	-[3]	-	-	-	-
Cadmium	5	-	-	-	-
Carbon tetrachloride	5	-	-	-	-
Chloroform	-[3]	-	-	-	-
Chromium	100	-	-	-	-
Chrysene	-	-	-	-	-
Copper	TT[1]	-	-	1000	1000
Dibromochloromethane	-[3]	-	-	-	-
Dichloroethane, 1,2-	5	-	-	-	-
Dichloroethylene, 1,1-	7	-	-	-	-
Dichloroethylene, *cis*-1,2-	70	-	-	-	-
Dichloroethylene, *trans*-1,2-	100	-	-	-	-
Dimethylphenol, 2,4-	-	-	-	-	-
Ethylbenzene	700	-	-	30	-
Fluoride	4000	-	-	2000	2000
Iron	-	-	-	300	300
Lead	TT[1]	-	-	-	-
Manganese	-	-	-	50	50
Mercury	2	-	-	-	-
Methylene chloride	5	-	-	-	-
Naphthalene	-	-	-	-	-
Natural uranium	-	-	20	-	-
Nickel	100[2]	-	-	-	-
Nitrate (as N)	10000	-	-	-	-
Nitrate + Nitrite (as N)	10000	-	-	-	-
Polychlorinated biphenyls	0.5	-	-	-	-
Radium 226 [4]	-	-	20	-	-
Radium 226 + Radium 228 [6]	-	5	-	-	-
Radon 222	-	-	300	-	-
Silver	-	-	-	100	100
Tetrachloroethylene	5	-	-	-	-
Trichloroethane, 1,1,2-	5	-	-	-	-
Trichloroethylene	5	-	-	-	-
Vinyl chloride	2	-	-	-	-

Notes:

All values from the Risk Assessment Information System. This web site can be accessed at http://risk.lsd.ornl.gov/cgi-bin/guide/GUID_9709
Dashes indicate a value is not available under the respective column's header.

Exhibit 8.1 (continued)

[1] TT = Treatment technique. When the "action level" of 15 µg/L for lead or 1,300 µg/L for copper, measured at the 90th percentile at the consumer's tap, is exceeded, corrosion control studies and treatment requirements are applicable. However, an OSWER memoraudum (July 21, 1990) recommends a final cleanup level of 15 µg/L for lead in groundwater usable for drinking water is protective of sensitive subpopulations; this is TBC guidance, not an ARAR.

[2] EPA has deleted both the MCL and MCLG for nickel, which have been vacated by court ruling, effective February 23, 1995 (60FR 33926, June 29, 1995). However, Kentucky retains a nickel MCL of 100 µg/L in its drinking water regulations. See 401 KAR 8:250, Section 12.

[3] For total trihalomethanes (i.e., sum of concentrations of chloroform, bromodichloromethane, dibromochloromethane, and bromoform), Kentucky retains a prior MCL of 100 µg/L. (See 401 KAR 8:500, Section 4).

[4] The present MCL includes Ra-226 and excludes radon and uranium; the proposed MCL excludes all three radionuclides.

[5] These values are not MCLs but are average annual concentrations that result in the effective dose equivalent (EDE) of 4 mrem/year, the MCL for gross beta emissions. If two or more radionuclides are present, the sum of their annual dose equivalent to the total body or to any part shall not exceed 4 mrem/year.

[6] Combined ^{226}Ra and ^{228}Ra. Specific determinations of these radioisotopes are not necessary if dissolved gross alpha particle activity does not exceed 5 pCi/L.

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