

APPENDIX A REFERENCES

Bowers, T.S., B.D. Beck and H.S. Karam, 1994. Assessing the relationship between environmental lead concentrations and adult blood lead levels. *Risk Analysis*. 14(2): 183-189.

CATI Inc., 2006. CATI Incorporated. Final Sampling Summary Report Follow-On Closure Activities. Lanham, Maryland.

CDC, 1991. Centers for Disease Control and Prevention. Preventing Lead Poisoning in Young Children. U.S. Department of Health and Human Services, Public Health Service, Atlanta, Georgia. Available on-line at: <http://wonder.cdc.gov/wonder/prevguid/p0000029/p0000029.asp>

EPA, 2006. Integrated Risk Information System (IRIS). Available on-line at: www.epa.gov/iris/index.html

EPA, 2005. Integrated Exposure Uptake Biokinetic Model for Lead in Children (IEUBK) Version 1.0 Build 264. Available on-line at: <http://www.epa.gov/superfund/lead/products.htm#ieubk>

EPA, 2004. Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual, Part E, Supplemental Guidance for Dermal Risk Assessment Final. Office of Superfund Remediation and Technology Innovation, OSWER 9285.7-02EP PB99-963312, July 2004.

EPA, 2003a. Adult Lead Model. Available on-line at: www.epa.gov/oswer/riskassessment/tools.htm

EPA, 2003b. Recommendations of the Technical Review Workgroup for Lead for an Interim Approach to Assessing Risks Associated with Adult Exposures to Lead in Soil. Washington, D.C. EPA/540/R-03/001.

EPA, 2002. Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites. Office of Emergency and Remedial Response, OSWER 9355.4-24, December 2002.

EPA, 1997. Exposure Factors Handbook. Available on-line at: <http://cfpub.epa.gov/ncea/cfm.recordisplay.cfm>

Health Risk Assessment No. 39-DA-07ZE-08, Camp Pedricktown Reserve Enclave, NJ, Jan 08

EPA, 1994. OSWER Directive: Revised Interim Soil Lead (Pb) Guidance for CERCLA Sites and RCRA Corrective Action Facilities, OSWER Directive #9355.4-12, August 1994. Available on-line at: <http://www.epa.gov/oerrpage/superfund/programs/lead/products/oswerdir.pdf>

EPA, 1992. Dermal Exposure Assessment: Principles and Applications, Office of Research and Development, Washington, D.C. EPA/600/8-91/011B.

EPA, 1990. National Oil and Hazardous Substances Pollution Contingency Plan. Federal Register 55:8667-8865.

EPA, 1989. Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual. Office of Emergency and Remedial Response, Washington, D.C. EPA/540/1-89/001.

EPA, 1986. Air Quality Criteria for Lead. Vol. I. Draft Final. EPA-600/8-83/028aF, June 1986.

Kemron Environmental Services, 2005. Final Site Investigation of Specific Areas of Potential Environmental Concern at the Reserve Enclave at Camp Pedricktown, Vienna, Virginia.

USACHPPM, 2006. Continued Site Investigation Addendum No. 38-EH-0606-07, U.S. Army Reserve Command, Camp Pedricktown Reserve Enclave, Building 434 and AOPEC Nos. 12 and 16, Oldmans Township, New Jersey, 25 October – 14 December 2006.

URS Corporation, 2003. Final Environmental Baseline Survey Report, Camp Pedricktown Reserve Enclave, Seattle, Washington.

Versar Inc., 1993. Final Expanded Site Inspection Report of Pedricktown Support Facility, Langhorne, Pennsylvania.

APPENDIX B RISK ASSESSMENT

B-1. INTRODUCTION.

a. The health threat from a site can be estimated through the use of risk assessment techniques. These estimates are useful in supporting whether health effects could be anticipated from the evaluated use of the site. Such calculations have also proved valuable in developing and supporting planning decisions about the need for remedial actions on sites thought or known to be affected by activities involving chemical releases.

b. This appendix presents a risk assessment performed for evaluating the health implications of future residents, construction workers and industrial site workers at the Camp Pedricktown Reserve Enclave in Oldman's Township, New Jersey. The risk assessment is limited to these receptors because they represent a maximally exposed individual and they are the focus of the future use of the area.

c. This risk assessment will follow the same methods used for conducting baseline risk assessments at EPA hazardous waste sites with the exception that the evaluation will be focused on the expected receptors using the analytical results determined in the previous investigations (Kemron, 2005; CATI, 2006; and USACHPPM, 2006). This risk assessment focused mainly on the AOPECs identified in the Site Investigation report that had elevated soil and ground-water samples. These areas include AOPEC 10, AOPEC 15, and AOPEC 16, which are located in the Warehousing Area and AOPEC 12 which is located in the Military Vehicle Parking Area. Due to their close proximity to each other and their separation from the other main divisions of the property along with the ground-water flow in a northwestern direction, it can be assumed that the contamination among these areas is self contained. This contamination should not affect the other areas of the site which are located to the south and east of the AOPEC in question.

d. Three points about a risk assessment should be emphasized:

(1) First, an estimate of carcinogenic risk or noncarcinogenic hazard is dependent upon the assumptions and numerical values used in the risk characterization, toxicity evaluation, and exposure assessment components. Risk assessment estimates should not be taken as absolute measures of an individual's probability of an adverse health effect. Rather, the estimates should be viewed as a threshold of concern for the receptor populations. Since most exposure parameters incorporate methods designed to yield a high-end estimate plus some degree of safety factor, the estimate of risk most likely represents an overestimate.

(2) Second, these estimates do not indicate that an adverse outcome actually will occur; they only indicate the likelihood or probability that such outcomes might occur under very specific exposure conditions. However, the flexibility to adjust exposure

assumptions and values allows risk managers to analyze a number of different exposure conditions and reach a more informed decision than if a risk assessment was not conducted.

(3) Third, a comprehensive risk assessment is only one of several tools that can provide useful information for risk management decisions. Risk assessment results only contribute to a final risk management solution; they are not the final solution. When all uncertainties associated with the assumptions and exposure values are identified, however, a comprehensive risk assessment can assist policy developers and risk managers in reaching a more informed risk management decision about available management options.

B-2. **METHODOLOGY AND ORGANIZATION OF DOCUMENT.** The methodology employed for this risk assessment follows EPA guidance. Four steps in the risk assessment process are outlined below. These steps are discussed in more detail in Sections B-3 through B-6.

a. Identification of Chemicals of Concern (Section B-3). This section provides site-related data along with background chemical data. Detailed summaries and statistical analyses of these data are provided in this section. All chemicals with detections in the applicable environmental media were evaluated in the risk assessment. This section discusses the reasons for eliminating chemicals from further evaluation in the risk assessment.

b. Exposure Assessment (Section B-4). For human exposure to occur, a pathway must be complete. This includes: a source, a transport media (e.g., soil), an exposure point (location), and an exposure route (e.g., ingestion). This section includes derivation and presentation of the exposures used in the human health risk assessment. Examples of scenarios which may be active on this site are future adult and child residents, future adult construction workers and industrial site workers. Chemical intake values are calculated based on exposure pathways, specific exposure values, and assumptions. Equations used to calculate intakes for all applicable exposure pathways are presented in this section.

c. Toxicity Assessment (Section B-5). This section presents the toxicity values used in the human health risk calculations. Reference to the appropriate data sources such as the Integrated Risk Information System (IRIS) (EPA, 2006) is provided to support the toxicity values.

d. Risk Characterization (Section B-6). This section presents the risk calculations for all complete human health exposure pathways. Noncarcinogenic and carcinogenic risk estimates are summarized for each receptor and exposure pathway. In all scenarios, the calculated risk values apply to a hypothetical individual on the site and represents an upper-bound (reasonable maximum) risk estimate. Thus, the calculated risk is not directly applicable to actual individuals working on the site. All of the exposure

assumptions have been chosen to protect the maximum reasonably exposed individual. This provides a conservative estimate of risk which tends to overestimate the maximum risk to any actual individual.

e. Lead Risk Characterization (Section B-7). A separate method of calculating the concentration of lead in blood is used. This has been developed due to the relation between chronic health effects from lead exposure and elevated blood lead levels.

B-3. IDENTIFICATION OF CHEMICALS OF POTENTIAL CONCERN (COPCs).

a. Introduction. The data used in this risk assessment was collected during field operations conducted in the previous 2005 Site Investigation (Kemron, 2005), the 2006 Final Sampling Summary Report (CATI, 2006), and the 2006 Continued Site Investigation Addendum (USACHPPM, 2006). The data collected for this site consisted of ground-water samples and surface and subsurface soil samples. This risk assessment focused mainly on the AOPECs identified in the Site Investigation report that had elevated soil and ground-water samples. These areas include AOPEC 10, AOPEC 15, and AOPEC 16 which are located in the Warehousing Area and AOPEC 12 which is located in the Military Vehicle Parking Area. Due to their close proximity to each other and their separation from the other main divisions of the property along with the ground-water flow in a northwestern direction, it can be assumed that the contamination among these areas is self contained. This contamination should not affect the other areas of the site which are located to the south and east of the AOPEC in question. The collected data was evaluated for suitability of use in the risk assessment as discussed in Risk Assessment Guidance for Superfund (RAGS) (EPA, 1989). The samples were analyzed for various metals, PAHs, PCBs, VOCs and SVOCs, and chlorinated pesticides and herbicides. All reported detections were considered for inclusion in a preliminary risk screening. All of the samples from the Site Investigation report that had a "B" with the value were eliminated from the data due to possible lab contamination, and no blank sample results were found for this report to determine if they can be included or not. Many of the ground-water samples taken for the Site Investigation report at AOPECs 10, 12, 15, and 16 were collected using a Geoprobe which is not very effective at collecting representative ground-water samples for metal analysis. Typically, the ground-water screening samples collected using a Geoprobe contain a higher colloid content which will artificially increase the concentrations of metals (Kemron, 2005). The ground-water samples collected using the low-flow sample collection method did not contain concentrations of metals above the laboratory reporting limits. The low-flow sample collection method is designed to collect a representative ground-water sample. Therefore, the ground-water samples collected from the monitoring wells are considered representative of the metal content for the AOPECs in this risk assessment. Table B-1 lists the analyses used in the different data collection studies.

Table B-1. Analytes and Methods for Soil Samples.

Analysis	Method (Ground Water)	Method (Soil)
Petroleum Hydrocarbons		Method 418.1
Volatile Organic Compounds	Method 624	EPA 8260B
Semivolatile Compounds	Method 625	EPA 8270C
Polychlorinated Biphenyls	Method 608	EPA 8082
Chlorinated Pesticides	Method 608	EPA 8082, EPA 8081A
Metals	EPA CLP method	EPA 6010B, SW846 7471A, and SW846 7470A

b. Risk Screening. A risk screening evaluates the exposure and health implications from all site chemicals, individually as well as collectively. The screening compares the site chemical concentrations to those levels which a residential receptor could be safely exposed to over a lifetime. A risk screening uses fewer details by focusing on a reasonable worst-case exposure to receptors that are more sensitive indicators of a health concern. As such, the 95th UCL of each chemical is calculated using a statistics application created by the State of Washington Department of Ecology called the MTCA stat 97 site module. To account for exposure a mixture of surface and subsurface soils and/or exposure directly to subsurface soils, all soil data was combined to determine the 95th UCL for each chemical. The EPA Region 3 risk-based concentrations (RBCs) for a residential receptor were used to compare the calculated 95th UCL. The elimination of a chemical from further evaluation is based on calculating the percent contribution of each detected analyte to the total risk and hazard index. Only chemicals that were not detected above the detection limit were excluded from the evaluation. Individual data analyses that were below the detection limit were replaced with ½ the detection limit for calculation. For chemicals that had varying detection limits among the different reports, the lowest reported detection limit was used for the 95th UCL calculation. The maximum value for quality assurance duplicate samples was used in the calculations. Table B-2 summarizes the soil data in the data collection reports while Table B-3 summarizes the ground-water data from the previous reports. Background risk-equivalent concentrations were listed in Tables B-2 and B-3 for those metals with available background concentrations (Versar Inc., 1993).

Table B-2. COPC Soil Data Summary (concentrations in units of mg/kg).

Parameter	Number of Detections	Detection Limit	Maximum Conc. Detected	Minimum Conc. Detected	Back-ground Conc.	Notes
Total Diesel Range Organic Compounds (DRO)	1-19	7.3	23.2	23.2		Not evaluated further, no RBCs
Total Petroleum Hydrocarbons (TPH)	1-1		25.4	25.4	19.3	Not evaluated further, no RBCs
TPH-DRO	1-5	13	13	13		Not evaluated further, no RBCs
Sulfate	4-10	54	150	59		Not evaluated further, no RBCs
Arsenic	56-56	0.41	224	1.4	3.695	
Cadmium	2-35	0.09	0.66	0.2	0.52	
Chromium	33-33	0.5	16	3.6	15.71	
Copper	11-13	2.5	19.1	2.6	6.22	
Lead	33-33	0.004	210	1.6	29.01	
Mercury (elemental)	2-13	0.017	0.13	0.05	0.035	Not evaluated further, no RBCs
Nickel	3-13	4	6.7	4.4	7.56	
Zinc	12-13	2	48.7	4.9	29.82	
Boron	2-20	4.6	13	6.8		
Molybdenum	1-18	1	1.2	1.2	1	
Acenaphthene	1-8	0.37	0.0086	0.0086		
Benzo(a)anthracene	1-8	0.037	0.016	0.016	0.53	
Benzo(a)pyrene	1-8	0.037	0.014	0.014	0.115	
Benzo(b)fluoranthene	1-8	0.037	0.019	0.019	0.19	
Benzo(g,h,i)perylene	1-8	0.039	0.014	0.014		Not evaluated further, no RBCs
Benzo(k)Fluoranthene	1-8	0.037	0.021	0.021	0.03	
Bis(2-ethylhexyl)phthalate	4-9	0.37	1.1	0.63	1	
Chrysene	1-8	0.01	0.029	0.029	0.125	
Dibenz(a,h) Anthracene	1-8	0.01	0.039	0.039	0.38	
Fluoranthene	1-8	0.01	0.02	0.02	0.225	

Table B-2. COPC Soil Data Summary (concentrations in units of mg/kg) (continued).

Parameter	Number of Detections	Detection Limit	Maximum Conc. Detected	Minimum Conc. Detected	Back-ground Conc.	Notes
Phenanthrene	1-8	0.37	0.014	0.014	0.11	Not evaluated further, no RBCs
Pyrene	1-8	0.37	0.02	0.02	0.155	
3,3-Dichlobenzidine	1-5	0.037	0.016	0.016		
Indeno(1,2,3-cd)pyrene	1-8	0.037	0.011	0.011	0.11	

Table B-3. COPC Ground-Water Data Summary (concentrations in units of µg/L).

Parameter	Number of Detections	Detection Limit	Maximum Conc. Detected	Minimum Conc. Detected	Back-ground Conc.	Notes
TPH-DRO	8-8	27	210	48		Not evaluated further, no RBCs
Sulfate	6-6	1	48	13		Not evaluated further, no RBCs
Arsenic	12-25	0.02	4.34	0.0633	14.53	
Chromium	1-23	1.1	22.6	22.6	87.53	
Lead	7-22	1.8	22.1	5.14	29.69	
Zinc	5-8	5.8	37.1	23.7	213.2	
Boron	13-13	0.1	229	115		
Bis(2-ethylhexyl)phthalate	1-37	0.6	0.7	0.7	1	
1,1,2-Trichloroethane	1-33	0.3	0.3	0.3		
Tetrachloroethene	6-33	0.3	2	0.5		
Toluene	1-38	0.2	1.1	1.1		
Endosulfanl	1-30	0.1	0.019	0.019		
Heptachlor Epoxide	1-30	0.1	0.027	0.027		

(1) Carcinogenic Risk Estimates. The risk screening for carcinogenic risk is performed by taking the site-specific concentration (95th UCL) for each chemical and dividing by the RBC concentration designated for carcinogenic evaluation then multiplying this ratio by 1E-6 to estimate the chemical-specific risk for a reasonable maximum exposure for each chemical. The total site carcinogenic risk estimate is derived by adding the estimated risks for each chemical. A total site carcinogenic risk of 1E-6 or less is considered safe. A total site carcinogenic risk exceeding 1E-6 suggests further evaluation to determine whether the site risk is related to site activities.

(2) Noncarcinogenic Risk Estimates. To determine noncarcinogenic risk estimates, the site-specific concentration (95th UCL) for each chemical is divided by its respective noncarcinogenic RBC and the ratios for multiple chemicals are then summed. The cumulative ratio represents a noncarcinogenic risk or hazard index (HI). An HI of 1 or less is generally considered safe. A ratio of greater than 1 suggests further evaluation is needed to determine whether the site risk is related to site activities. Table B-4 shows the results of the risk screening for soil, while Table B-5 shows the risk screening results for ground water.

(3) Results of Risk Screening. The total site carcinogenic risk for soil is 1.72E-04, and the total site carcinogenic risk for ground water is 4.85E-05. Both of these values are greater than 1E-06. The noncarcinogenic risk estimate for soil is 5.24E-02 and for ground water is 5.83E-02, which are less than 1. Thus, in order to determine a list of COPCs, a chemical's percentage of the total risk was calculated. If a chemical posed less than 1% of the risk, it was eliminated from further examination in the assessment. The highlighted chemicals and risk percentages can be found in Tables B-4 and B-5, while a complete list of the COPCs retained for this risk assessment are shown in Table B-6. Although lead is listed as a COPC, it will be addressed separately in this assessment. Only the chemicals that exceeded 1% of the risk were retained as COPCs.

Table B-4. Risk Screening for Chemicals of Potential Concern in Soil (concentrations in units of mg/kg) (Chemicals in bold have been retained as COPC).

Parameter	Distribution	95th UCL	Region 3 RBC (nc/ca)	Screening Risk (CA)	Screening Risk (NC)	Background Risk Equiv	CA Risk %	NC Risk %
Arsenic	lognormal	73.24	0.43ca	1.7E-04	-	8.59E-06ca	99.06%	
Cadmium	Non-para	0.10	78nc	-	1.32E-03	6.67E-03nc	-	2.52%
Chromium	lognormal	9.82	234.64nc	-	4.18E-02	6.70E-02nc	-	79.78%
Copper	lognormal	11.3	3100nc	-	3.65E-03	2.01E-03nc	-	6.95%
Nickel	Non-para	4.56	1600nc	-	2.85E-03	4.73E-03nc	-	5.44%
Zinc	lognormal	22.30	23000nc	-	9.70E-04	1.30E-03nc	-	1.85%
Boron	Non-para	4.09	16000nc	-	2.55E-04	na	-	0.49%
Molybdenum	Non-para	0.60	390nc	-	1.55E-03	2.56E-03nc	-	2.95%
Acenaphthene	Non-para	0.009	4700nc	-	1.83E-06	na	-	0.00%
Benzo(a)Anthracene	Non-para	0.016	0.22ca	7.27E-08	-	2.41E-06ca	0.04%	-
Benzo(a)Pyrene	Non-para	0.014	0.022ca	6.36E-07	-	5.23E-06ca	0.37%	-
Benzo(b)Fluoranthene	Non-para	0.019	0.22ca	8.64E-08	-	8.64E-07ca	0.05%	-
Benzo(k)Fluoranthene	Non-para	0.019	2.2ca	8.64E-09	-	1.36E-08ca	0.01%	-
Bis(2-Ethylhexyl)Phthalate	normal	0.770	45.62ca	1.69E-08	-	na	0.01%	-
Chrysene	Non-para	0.013	22ca	5.91E-10	-	5.68E-09ca	0.00%	-
Dibenzo(a,h)Anthracene	Non-para	0.016	0.022ca	7.27E-07	-	1.73E-05ca	0.42%	-
Fluoranthene	Non-para	0.01	3100nc	-	3.23E-06	7.26E-05nc	-	0.01%
Pyrene	Non-para	0.02	2300nc	-	8.70E-06	6.74E-05nc	-	0.02%
3,3-Dichlorobenzidine	Non-para	0.016	1.4ca	1.13E-08	-	na	0.01%	-
Indeno(1,2,3-cd)Pyrene	Non-para	0.011	0.22ca	5.00E-08	-	5.00E-07ca	0.03%	-
Total	-	-	-	1.72E-04	5.24E-02	-	-	-

Notes:

1. NC is used to represent noncarcinogenic and CA is used to represent carcinogenic.
2. Risk is calculated by dividing the exposure concentration or background level by the appropriate RBC.
3. Non-para is used to denote that the data did not show a normal or log-normal distribution. A nonparametric method was used to estimate the 95th UCL.
4. Chromium VI values were used to screen chromium since they are stricter, thus making this a more conservative approach.
5. The background levels were taken from the Final Expanded Site Investigation Report of the Pedricktown Support Facility by Versar Inc. in December 1993.

Table B-5. Risk Screening for Chemicals of Potential Concern in Ground Water
(concentrations in units of mg/kg) (Chemicals in bold have been retained as COPC).

Parameter	Distribution	95th UCL	Region 3 RBC (nc/ca)	Screening Risk (CA)	Screening Risk (NC)	Background Risk Equiv	CA Risk %	NC Risk %
Arsenic	Non-para	19.44	4.5E-02ca	4.35E-04	-	3.25E-04ca	85.48%	-
Chromium	Non-para	3.327	1.1E+02nc	-	2.99E-02	7.99E-01nc	-	51.25%
Zinc	lognormal	559	1.1E+04nc	-	5.11E-02	1.95E-02nc	-	5.02%
Boron	lognormal	185.20	7.3E+03nc	-	2.54E-02	-	-	43.54%
1,1,2-Trichloroethane	Non-para	0.16	1.9E-01ca	8.62E-07	-	-	1.78%	-
Tetrachloroethene	Non-para	0.53	1.0E-01	5.12E-06	-	-	10.56%	-
Toluene	Non-para	0.18	2.3E+03nc	-	7.93E-05	-	-	0.14%
Bis(2-Ethylhexyl)Phthalate	Non-para	0.33	4.8E+00nc	6.88E-08	-	2.09E-07ca	0.14%	-
Endosulfanl	Non-para	0.01	2.2E+02nc	-	2.74E-05	-	-	0.05%
Heptachlor Epoxide	Non-para	0.01	7.4E-03ca	9.51E-07	-	-	1.96%	-
Total	-	-	-	4.85E-05	5.83E-02	-	-	-

Notes:

1. NC is used to represent noncarcinogenic and CA is used to represent carcinogenic.
2. Risk is calculated by dividing the exposure concentration or background level by the appropriate RBC.
3. Non-para is used to denote that the data did not show a normal or log-normal distribution. A nonparametric method was used to estimate the 95th UCL.
4. Chromium VI values were used to screen chromium since they are stricter, thus making this a more conservative approach.
5. The background levels were taken from the Final Expanded Site Investigation Report of the Pedricktown Support Facility by Versar Inc. in December 1993.

Table B-6. Chemicals of Potential Concern.

Chemical	Carcinogen (ca) or Noncarcinogen (nc)	Media
Cadmium	nc	Soil
Copper	nc	Soil
Molybdenum	nc	Soil
Arsenic*	ca	Soil and groundwater
Chromium*	nc	Soil and groundwater
Zinc*	nc	Soil and groundwater
Nickel**	nc	Soil
Boron	nc	Ground water
Heptachlor Epoxide	ca	Ground water
Tetrachloroethene	ca	Ground water
1,1,2-Trichloroethane	ca	Ground water
Lead	nc	Soil and ground water

* - these chemicals have been excluded from groundwater based on background screening.

** - this chemical has been excluded from soil based background screening

c. Background Screening. The background screening compared the site chemical concentrations with the average background concentration for each chemical taken at the site during a previous investigation (Versar Inc., 1993). The soil samples included both surface and subsurface samples. A chemical was excluded from the COPC list if its

concentration at the site was below the site's background concentration. There were three chemicals, Arsenic, Chromium, and Zinc with concentrations below the background values for groundwater, while only Nickel was lower than background concentrations in soil. Even though, Chromium and Zinc in the soil had 95th UCL concentrations below the background concentrations, they were retained as COPCS because their maximum concentrations were above background values thus taking a more conservative approach. It should be noted that although Arsenic is a COPC in the soil, the source of the Arsenic at this site is believed to be the coal slag that was used as road bed and has apparently been in place for quite a few years. It was assumed that if the Arsenic was going to migrate to the groundwater, it would already have been seen elevated above background by now.

B-4. EXPOSURE ASSESSMENT.

a. Overview and Characterization of Exposure Setting. The objective of the exposure assessment was to estimate the type and magnitude of exposures to the COPCs that are present at the site. This component of the risk assessment can be performed either qualitatively or quantitatively. Quantitative assessment is preferred when toxicity factors necessary to characterize a COPC are available. The exposure assessment consists of three steps (EPA, 1989):

(1) Characterize Exposure Setting: This step contains general information concerning the physical characteristics of the site as it pertains to potential considerations affecting exposure. The physical setting involves climate and vegetation. All potentially exposed populations and subpopulations therein (receptors) are assessed relative to their potential for exposure. This step is a qualitative one aimed at providing a general site perspective and offering insight on the surrounding population.

(2) Identify Exposure Pathways: All exposure pathways, ways in which receptors can be exposed to site chemicals, are reviewed in this step. Exposure points of human contact and exposure routes are discussed before quantifying the exposure pathways in the next step.

(3) Quantify Exposure: In this final step, the receptor intakes are calculated for each exposure pathway and receptor. These calculations follow EPA guidance for assumptions of intake variables or exposure factors for each exposure pathway and EPA-recommended calculation methods.

b. Land Use and Potentially Exposed Populations.

(1) Land Use. The site is currently unoccupied and provided grounds and buildings to support the administration, supply, training, and maintenance activities of the U.S. Army Reserve (USAR). The site is broken up into four main areas: an

administrative area, housing and recreation area, warehousing area, and military vehicle parking area.

(2) Potentially Exposed Populations.

(a) For purposes of this risk assessment, four potentially exposed populations were considered. Since the use of this site is proposed for change in the foreseeable future and no one currently works at the site, the site exposure will be modeled for the future potentially exposed populations. As such, the risk assessment will evaluate a construction worker, an industrial worker, and a residential scenario (adult and child). These exposures will be more sensitive to detecting any health problem than a receptor that would spend less time at the site (i.e., trespasser or occasional visitor).

(b) The four exposed populations are a construction worker, an industrial site worker, an adult resident, and a child resident. Each scenario is evaluated for the default duration. Construction workers are assumed to work on the site for 250 days for 1 year, while industrial workers are assumed to work on the site 250 days a year for 25 years. The adult and child residents are evaluated separately for both carcinogens and noncarcinogens. The child scenario is based on 6 year duration, while the adult scenario is based on 30 year duration. The residents, whether a child or an adult, are assumed to live onsite, 24 hours a day, 350 days a year. Other factors defining the exposure of an individual follow the current default values determined by EPA guidance (EPA, 1989, 2002, and 2004).

c. Identification of Exposure Pathways.

(1) Exposure Estimates.

(a) Exposures are estimated only for plausible completed exposure pathways. A complete exposure pathway is comprised of three main elements: a source and mechanism for chemical release, an environmental transport medium (exposure point), and a feasible route of exposure to a human receptor. In order for there to be a need for a risk evaluation, an exposure pathway must be potentially complete.

(b) An exposure pathway is the way in which a chemical of concern potentially comes in contact with a receptor. Generally, exposure pathways include inhalation, ingestion, and dermal contact. This assessment considers all three exposure routes in regards to soil and ingestion and dermal pathways for ground-water exposure at the site. For the groundwater pathway, the two volatile organic COPCs, Tetrachloroethene and 1,1,2-Trichloroethane, were not evaluated for inhalation since their combined risk only represented about 12 percent of the total risk in the risk screening. Also, the ingestion pathway would typically account for most of the risk when evaluating exposure to drinking water. Surface water was not evaluated as an exposure pathway because no surface water exists at the site.

(2) Sources and Receiving Media.

(a) Prior to the USAR's use of the property the Army has used the site for various different activities over the past 80 years ranging from an Ordnance Depot and a backup ammunitions storage facility to a disposal facility and an Army Air Defense Command Post. The property was then transferred to Fort Dix in 1962 and finally to the USAR in 1993 (URS, 2003). There are several buildings that still exist at the site. The sources that contribute to the chemical nature of the surface and subsurface soil are discussed in more detail in the data collection reports (URS, 2003; Kemron, 2005; USACPPM, 2006, CATI, 2006) and will not be duplicated here. Arsenic has been identified in a previous report as being associated with the gravel material containing Coal Slag used to pave the roads near the warehousing area (USACHPPM, 2006).

(b) This risk assessment does not attempt to characterize the source or the transformations which may occur during transport from the source to the receptors. All measurements were made at or near the receptor points of exposure to estimate the type and dose of the chemicals acting upon the receptor. Thus, no determination of the chemical transformations is necessary.

d. Quantification of Exposure.

(1) In this section, each receptor's potential exposures to the COPCs are quantified for each of the exposure pathways. In each case, the exposures are calculated following methods recommended in EPA guidance documents such as the RAGS (EPA, 1989), the Supplemental Risk Assessment Guidance for Dermal Risk Assessment (EPA, 2004), and the Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites (EPA, 2002). These calculations generally involve two steps. First, representative chemical concentrations in the environment, or exposure point concentrations (EPCs), are determined for each pathway and receptor. From these EPC values, the amount of chemical, which an exposed person may take into his/her body, is then calculated. This value is referred to as the human intake. This section describes the exposure scenarios, exposure assumptions, and exposure calculation methods used in this risk assessment.

(2) Risk assessment as a whole and the exposure assessment step in particular are designed to be health protective. The exposure calculations require estimates and assumptions about certain human exposure parameters such as ingestion rates. Generally, values are selected which tend to overestimate exposure. EPA recommends two types of exposure estimates be used for Superfund risk assessments: a reasonable maximum exposure (RME) and central tendency exposure (CT). The RME is defined as the highest exposure that could reasonably be expected to occur for a given exposure pathway at a site, and is intended to account for both uncertainty in the chemical concentration and variability in the exposure parameters (such as exposure frequency or averaging time). The CT may also be evaluated for comparison purposes and is generally based on mean exposure parameters. Only RME scenarios have been evaluated in this risk assessment.

(3) Estimates of pathway-specific human intakes for each COPC involve assumptions about patterns of human exposure to the media being evaluated. These assumptions are integrated with the EPCs to calculate intakes. Intakes are normally expressed as the amount of chemical at the environment-human receptor exchange boundary in milligrams per kilogram of body weight per day (mg/kg-day), which represents an exposure normalized for body weight over time. The total exposure is divided by the time period of interest to obtain an average exposure. The averaging time is a function of the health endpoint. For noncarcinogenic effects, it is the exposure time (specific to the scenario being assessed) and for carcinogenic effects, it is lifetime (70 years).

e. Exposure Assumptions.

(1) An important aspect of the exposure assessment is the determination of assumptions regarding how receptors may be exposed to chemicals. The EPA guidance on exposure factors is extensive and was followed throughout this exposure assessment. Standard scenarios and default assumptions were used where appropriate.

(2) The exposure scenarios in this assessment involve four receptors: construction worker, industrial worker, adult, and child resident. The exposure assumptions for these scenarios are intended to approximate the frequency, duration, and manner in which receptors are exposed to environmental media. However, each parameter tends to have a safety factor imbedded into its determination such that they tend to overestimate exposure and therefore risk. Details of the exposure assumptions and parameters for each exposure scenario are shown in Table B-7.

f. Exposure Scenarios. The four exposure scenarios and their respective exposure assumptions in this assessment are described below.

(1) Construction Worker. These receptors spend each day of this construction project at the site (5 days/week for 50 weeks, RME). This exposure period lasts for 1 year. For the dermal exposure, we are assuming a work outfit of short sleeves shirt, long pants, and shoes, thus producing an average exposed skin area of 3300 cm². The ground-water exposure pathway for the construction worker was not evaluated in this assessment due to there being a very minor risk associated with the exposure to ground water based

Table B-7. Exposure Parameters.

Parameter	Construction Worker	Industrial Worker	Adult Resident	Child Resident
Dermal Contact Rate*	3300 cm ²	3300 cm ²	5700 cm ²	2800 cm ²
Dermal Exposure Frequency*	250 days/year	250 days/year	350 days/year	350 days/year
Inhalation Exposure Frequency**	250 days/year	250 days/year	350 days/year	350 days/year
Inhalation Rate**	20 m ³ /day	20 m ³ /day	20 m ³ /day	10 m ³ /day***
Ingestion Rate**	330 mg/day	100 mg/day	100 mg/day	200 mg/day
Exposure**	250 days	250 days	350 days	350 days
Duration**	1 year	25 years	30 years	6 years
Body Weight**	70 kg	70 kg	70 kg	15 kg
Lifetime**	25550 days	25550 days	25550 days	25550 days
Particulate Emission Factor (PEF)**	4.40E+8 m ³ /kg	1.36E+9 m ³ /kg	1.36E+9 m ³ /kg	1.36E+9 m ³ /kg
Adherence Factor*	0.3 mg/cm ²	0.2 mg/cm ²	0.07 mg/cm ²	0.2 mg/cm ²
Ground-Water Ingestion Rate**	NA	2 L/day	2 L/day	1 L/day
Ground-Water Dermal Contact Rate**	NA	800 cm ² ****	18,000 cm ²	6,600 cm ²

Notes:

* Values taken from EPA Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual, Part E, Supplemental Guidance for Dermal Risk Assessment. July 2004.

** Values taken from EPA Supplemental Guidance for Developing Soil Screening Levels for Superfund Sites, December 2002.

*** Value taken from EPA Exposure Factors Handbook, August 1997.

**** Best professional judgment, based on the mean surface area for hands for men and women.

on the short exposure duration and amount of contact with ground water. Since no drinking water wells exist on the property, the ground-water pathway for the construction worker would be incomplete.

(2) Industrial Worker. These receptors spend each day working at the site (5 days/week for 50 weeks, RME). This exposure period lasts for 25 years. For the dermal exposure, we are assuming a work outfit of a short sleeve shirt, long pants, and shoes. This produces an average exposed skin area of 3300 cm². The ground-water exposure ingestion rate used for the industrial worker is the adult default exposure rate of 2 L/day with a ground-water dermal contact rate of 800 cm² which was based on the assumption that the industrial worker is only at the site 5 days out of the week and would be washing only their hands rather than their entire body.

(3) Adult Resident. These receptors spend each day living at the site (7 days/week for 50 weeks, RME). This exposure period lasts for 30 years. For the dermal exposure, we are assuming a summer attire of short sleeves, shorts, and shoes. This produces an average exposed skin area of 5700 cm². The ground-water ingestion rate for the residential adult is 2 L/day, the default exposure rate. For the ground-water dermal contact rate it is assumed that the total body surface area for adults is exposed when bathing, thus the default adult exposure rate of 18,000 cm² was used in this assessment.

(4) Child Resident. These receptors spend each day living at the site (7 days/week for 50 weeks, RME). This exposure period lasts for 6 years of childhood. For the dermal exposure, we are assuming a summer attire of short sleeves, shorts, and shoes. This produces an average exposed skin area of 2800 cm². The ground-water ingestion rate for children used in this assessment was the default exposure rate of 1 L/day while the ground-water dermal contact rate for children is 6,600 cm², the default exposure rate based on a total body surface area while bathing.

g. Incidental Ingestion of Soil.

(1) The equation for intake is as follows:

$$\text{Intake (mg/kg-day)} = \frac{\text{CS} \times \text{IR} \times \text{CF} \times \text{FI} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}}$$

Where:

CS	=	Chemical Concentration in Soil (mg/kg soil)
IR	=	Ingestion Rate (mg soil/day)
CF	=	Conversion Factor (1 kg/10 ⁶ mg)
FI	=	Fraction Ingested from Contaminated Source (unit less) (1)
EF	=	Exposure Frequency (days/years)
ED	=	Exposure Duration (years)
BW	=	Body Weight (kg)
AT	=	Averaging Time (period over which exposure is averaged -- days) (i.e., ED X 365 days/year for noncarcinogenic effects, and 70-year lifetime for carcinogenic effects, i.e., 70 years X 365 days/year)

(2) Table B-8 shows the results of these calculations for the ingestion intake.

Table B-8. Chemical Intakes for Ingestion Route by Receptor (all values are in mg/kg-day).

Soil				
Chemical	Industrial Worker	Construction Worker	Adult Resident	Child Resident
Arsenic	7.17E-05nc 2.56E-05ca	2.36E-04nc 3.38E-06ca	1.00E-04nc 4.30E-05ca	9.36E-04nc 8.03E-05ca
Cadmium	1.01E-07nc 3.60E-08ca	3.33E-07nc 4.75E-09ca	1.41E-07nc 6.05E-08ca	1.32E-06nc 1.13E-07ca
Chromium	9.61E-06nc 3.43E-06ca	3.17E-05nc 4.53E-07ca	1.34E-05nc 5.76E-06ca	1.26E-04nc 1.08E-05ca
Copper	1.11E-05nc 3.95E-06ca	3.65E-05nc 5.21E-07ca	1.55E-05nc 6.64E-06ca	1.45E-04nc 1.24E-05ca
Zinc	2.18E-05nc 7.79E-06ca	7.20E-05nc 1.03E-06ca	3.06E-05nc 1.31E-05ca	2.85E-04nc 2.44E-05ca
Molybdenum	5.90E-07nc 2.11E-07ca	1.95E-06nc 2.78E-08ca	8.26E-07nc 3.54E-07ca	7.71E-06nc 6.61E-07ca

Note: nc is used to represent noncarcinogenic and ca is used to represent carcinogenic.

h. Incidental Ingestion of Ground Water.

(1) The equation for intake is as follows:

$$\text{Intake (mg/kg-day)} = \frac{\text{CW} \times \text{IR} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}}$$

Where:

CW = Chemical Concentration in Ground Water (mg/L)
 IR = Ingestion Rate (L/day)
 EF = Exposure Frequency (days/years)
 ED = Exposure Duration (years)
 BW = Body Weight (kg)
 AT = Averaging Time (period over which exposure is averaged -- days)
 (i.e., ED X 365 days/year for noncarcinogenic effects, and 70-year lifetime
 for carcinogenic effects, i.e., 70 years X 365 days/year)

(2) Table B-9 shows the results of these calculations for the ingestion intake.

Table B-9. Chemical Intakes for Ingestion Route by Receptor (all values are in
mg/kg-day).

Ground Water				
Chemical	Industrial Worker	Adult Resident	Child Resident	Construction Worker
Boron	3.62E-03nc 1.29E-03ca	5.07E-03nc 2.17E-03ca	1.18E-02nc 1.01E-03ca	na
1,1,2-Trichloroethane	3.17E-06nc 1.13E-06ca	4.44E-06nc 1.90E-06ca	2.22E-06nc 8.88E-07ca	na
Tetrachloroethene	1.04E-05nc 3.70E-06ca	1.45E-05nc 6.22E-06ca	3.39E-05nc 2.90E-06ca	na
Heptachlor Epoxide	1.37E-07nc 4.89E-08ca	1.92E-07nc 8.22E-08ca	4.47E-07nc 3.84E-08ca	na

Note: nc is used to represent noncarcinogenic and ca is used to represent carcinogenic.

i. Dermal Contact with Soils.

(1) The same receptors considered to have the potential to ingest soil may also contact the same soils dermally. The equation for the absorbed dose from dermal exposure is as follows, based on EPA guidance (EPA, 1997).

$$\text{Absorbed Dose (mg/kg-day)} = \frac{\text{CS} \times \text{CF} \times \text{AF} \times \text{ABS} \times \text{SA} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}}$$

Where:

CS = Chemical Concentration in Soil (mg/kg soil)
 CF = Conversion Factor (10⁻⁶ kg/mg)
 AF = Soil to Skin Adherence Factor (mg/cm²)
 ABS = Absorption Factor (unit less)

(0.03 for Arsenic, and 0.001 for metals)
 SA = Skin Surface Area Available for Contact (cm²)
 EF = Exposure Frequency (days/year)
 ED = Exposure Duration (years)
 BW = Body Weight (kg)
 AT = Averaging Time (period over which exposure is averaged -- days)
 (i.e., ED X 365 days/year for noncarcinogenic effects, and 70-year lifetime
 for carcinogenic effects, i.e., 70 years X 365 days/year)

(2) Table B-10 shows the results of the calculations for dermal absorbed dose.

Table B-10. Chemical Intake Results for the Dermal Route by Receptor (all values are in mg/kg-day).

Soil				
Chemical	Industrial Worker	Construction Worker	Adult Resident	Child Resident
Arsenic	1.42E-05nc 5.07E-06ca	2.13E-05nc 3.04E-07ca	1.20E-05nc 5.15E-06ca	7.87E-05nc 6.74E-10ca
Cadmium	6.65E-10nc 2.38E-10ca	9.98E-10nc 1.43E-11ca	5.63E-10nc 2.41E-10ca	3.69E-09nc 3.16E-10ca
Chromium	6.34E-08nc 2.26E-08ca	9.51E-08nc 1.36E-09ca	5.37E-08nc 2.30E-08ca	3.51E-07nc 3.01E-08ca
Copper	7.30E-08nc 2.61E-08ca	1.10E-07nc 1.56E-09ca	6.18E-08nc 2.65E-08ca	4.05E-07nc 3.47E-08ca
Zinc	1.44E-07nc 5.14E-08ca	2.16E-07nc 3.09E-09ca	1.22E-07nc 5.22E-08ca	7.98E-07nc 6.84E-08ca
Molybdenum	3.99E-09nc 1.39E-09ca	5.84E-09nc 8.34E-11ca	3.30E-09nc 1.41E-09ca	2.16E-08nc 1.85E-09ca

Note: nc is used to represent noncarcinogenic and ca is used to represent carcinogenic.

(3) Dermal exposure to soil involves several unique exposure factors. Toxicity values that are expressed as administered doses are adjusted to absorbed doses for comparison. Because there are few toxicity values for dermal exposure, oral values are frequently used to assess risk from dermal exposure. The dermal exposure calculation considers the amount of exposed skin, the amount of soil which adheres to the skin, and the degree to which a chemical may be adsorbed through the skin. The surface area of exposed skin depends on the size of an individual (especially adult vs. child), clothing worn, and the specific parts of the body which may directly contact the medium of concern (e.g., soil). In regards to the dermal soil calculations the default assumption of complete or 100 percent, oral absorption was made thus an adjustment to the oral toxicity

factor was not performed. EPA risk assessment guidance recommendations were followed to select exposed skin surface areas for each scenario in this assessment.

j. Dermal Contact with Ground Water.

(1) The same receptors considered to have the potential to ingest ground water may also contact the same ground water dermally. The equation for the absorbed dose from dermal exposure is as follows, based on the EPA RAGS Part E, supplemental guidance for dermal risk assessment (EPA, 2004).

$$\text{Dermal Absorbed Dose (mg/kg-day)} = \frac{DA_{\text{event}} \times EV \times ED \times EF \times SA}{BW \times AT}$$

Where:

DA_{event}	=	Absorbed dose per event (mg/cm ² -event)
EV	=	Event Frequency (events/day)
ED	=	Exposure Duration (years)
EF	=	Exposure Frequency (days/year)
SA	=	Skin Surface Area Available for Contact (cm ²)
BW	=	Body Weight (kg)
AT	=	Averaging Time (period over which exposure is averaged -- days) (i.e., ED X 365 days/year for noncarcinogenic effects, and 70-year lifetime for carcinogenic effects, i.e., 70 years X 365 days/year)

The equation to calculate DA_{event} for Organic and Inorganic Compounds is listed below.

DA_{event} (mg/cm²-event) is calculated for organic compounds as follows:

$$\text{If } t_{\text{event}} \leq t^*, \text{ then } DA_{\text{event}} = 2 FA \times K_p \times C_w \sqrt{[(6 \tau_{\text{event}} \times t_{\text{event}})/\pi]}$$

$$\text{If } t_{\text{event}} > t^*, \text{ then } DA_{\text{event}} = FA \times K_p \times C_w [(t_{\text{event}} / 1+B) + 2 \tau_{\text{event}} \{(1+3B+3B^2)/(1+B)^2\}]$$

DA_{event} (mg/cm²-event) is calculated for inorganic compounds as follows:

$$DA_{\text{event}} = K_p \times C_w \times t_{\text{event}}$$

Where:

DA_{event}	=	Absorbed dose per event (mg/cm ² -event)
FA	=	Fraction absorbed water (dimensionless)
K_p	=	Dermal permeability coefficient of compounds in water (cm/hr)

C_w	=	Chemical concentration in water (mg/cm ³)
τ_{event}	=	Lag time per event (hr/event)
t_{event}	=	Event duration (hr/event)
t^*	=	Time to reach steady-state (hr) = 2.4 τ_{event}
B	=	Dimensionless ratio of the permeability coefficient of a compound through the stratum corneum relative to its permeability coefficient across the viable epidermis

(2) Table B-11 shows the results of the calculations for dermal absorbed dose.

Table B-11. Chemical Intake Results for Dermal Route by Receptor (all values are in mg/kg-day).

Ground Water				
Chemical	Industrial Worker	Adult Resident	Child Resident	Construction Worker
Boron	1.45E-08nc 5.17E-09ca	2.65E-07nc 1.14E-07ca	7.81E-07nc 6.69E-08ca	na
1,1,2-Trichloroethane	1.74E-08nc 6.21E-09ca	1.70E-09nc 7.29E-10ca	9.37E-09nc 8.03E-10ca	na
Tetrachloroethene	3.20E-09nc 1.14E-09ca	1.01E-07nc 4.32E-08ca	2.97E-07nc 2.55E-08ca	na
Heptachlor Epoxide	1.94E-11nc 6.93E-12ca	6.11E-10nc 2.62E-10ca	1.80E-09nc 1.54E-10ca	na

Notes: *nc is used to represent noncarcinogenic and ca is used to represent carcinogenic.

* In the calculation of Heptachlor Epoxide, the values of "B" and "FA" for Heptachlor were used due to no values being available for Heptachlor Epoxide specifically.

(3) Dermal exposure to ground water involves several unique exposure factors. Toxicity values that are expressed as administered doses are adjusted to absorbed doses for comparison. Because there are few toxicity values for dermal exposure, oral values are frequently used to assess risk from dermal exposure. The dermal exposure calculation considers the amount of exposed skin, the amount of ground water which adheres to the skin, and the degree to which a chemical may be adsorbed through the skin. The surface area of exposed skin depends on the size of an individual (especially adult vs. child), clothing worn, and the specific parts of the body which may directly contact the medium of concern (e.g., ground water during showering). EPA recommendations were followed to select exposed skin surface areas for each scenario in this assessment. The event frequency value used in this assessment was 0.01; this value was calculated based on a 15-minute shower exposure and the understanding that a person will dry off after showering.

k. Inhalation of Soil Particles.

(1) The same receptors considered to have the potential to ingest soil and touch soil may also breathe the soil particles that are suspended in the air. The equation for the exposure from the inhalation of fugitive dust is as follows, based on EPA guidance (EPA, 1997).

$$\text{Intake (mg/kg-day)} = \frac{\text{CS} \times \text{IR} \times \text{EF} \times \text{ED}}{\text{PEF} \times \text{BW} \times \text{AT}}$$

Where:

- CS = Chemical Concentration in Soil (mg/kg soil)
- IR = Inhalation Rate (m³/day)
- EF = Exposure Frequency (days/year)
- ED = Exposure Duration (years)
- PEF = Particulate Emission Factor (m³/kg)
- BW = Body Weight (kg)
- AT = Averaging Time (period over which exposure is averaged -- days)
(i.e., ED X 365 days/year for noncarcinogenic effects, and 70-year lifetime for carcinogenic effects, i.e., 70 years X 365 days/year)

(2) Table B-12 shows the results of the calculations for intake from inhalation of soil particles.

Table B-12. Chemical Intake Results for the Inhalation of Soil Particles by Receptor
(all values are in mg/kg-day).

Soil				
Chemical	Industrial Worker	Construction Worker	Adult Resident	Child Resident
Arsenic	1.05E-08nc 3.76E-09ca	1.30E-10nc 4.65E-10ca	1.48E-08nc 6.32E-09ca	3.44E-08nc 1.48E-08ca
Cadmium	1.48E-11nc 5.29E-12ca	1.83E-13nc 6.54E-13ca	2.07E-11nc 8.89E-12ca	4.84E-11nc 2.07E-11ca
Chromium	1.41E-09nc 5.05E-10ca	1.75E-11nc 6.24E-11ca	1.98E-09nc 8.48E-10ca	4.61E-09nc 1.98E-09ca
Copper	1.63E-09nc 5.81E-10ca	2.01E-11nc 7.18E-11ca	2.28E-09nc 9.76E-10ca	5.31E-09nc 2.28E-09ca
Zinc	3.21E-09nc 1.15E-09ca	3.97E-11nc 1.42E-10ca	4.49E-09nc 1.93E-09ca	1.05E-08nc 4.49E-09ca
Molybdenum	8.68E-11nc	1.07E-12nc	1.21E-10nc	2.83E-10nc

	3.10E-11ca	3.83E-12ca	5.21E-11ca	1.21E-10ca
--	------------	------------	------------	------------

Note: nc is used to represent noncarcinogenic and ca is used to represent carcinogenic.

B-5. TOXICITY ASSESSMENT. The objective of the toxicity assessment is to weigh available evidence regarding the potential of the chemicals to cause adverse effects in exposed individuals, and to provide, where possible, an estimate of the relationship between the extent of exposure to a chemical and the increased likelihood and/or severity of adverse effects. The types of toxicity information considered in this assessment include the oral reference dose (RfD) and inhalation RfD used to evaluate noncarcinogenic effects, and the slope factor and unit risk to evaluate carcinogenic potential. Most toxicity information used in this evaluation was obtained from the Integrated Risk Information System (IRIS) (EPA, 2006). Table B-13 summarizes the toxicity factors used in this evaluation for both carcinogenic and noncarcinogenic effects. The lead toxicity assessment can be found in Section B-7 of this report.

Table B-13. Carcinogenic Slope Factors and Reference Doses for COPCs.

Parameter	CAS No.	Reference Dose (RfD) oral (mg/kg-day)	Carcinogenic Slope Factor (CSF) oral (mg/kg-day) ⁻¹	RfD inhalation (mg/kg-day)	SF inhalation (mg/kg-day) ⁻¹	Source
Arsenic	7440-38-2	3.00E-04	1.50E+00	na	1.51E+01	IRIS
Cadmium	7440-43-9	1.00E-03	na	5.7E-05	6.30E+00	IRIS
Chromium	7440-47-3	3.00E-03	na	3.00E-05	4.10E+01	IRIS
Copper	7440-50-8	4.00E-02	na	na	na	Heast
Zinc	7440-66-6	3.00E-01	na	na	na	IRIS
Molybdenum	7439-98-7	5.00E-03	na	na	na	IRIS
Boron	7440-42-8	2.00E-01	na	5.70E-03	na	IRIS / Heast
Tetrachloroethene	127-18-4	1.00E-02	5.4E-01	8.0E-02	2.00E-02	IRIS / ATSDR MRL
Heptachlor Epoxide	1024-57-3	1.30E-05	9.10E+00	9.10E+00	na	IRIS
1,1,2-Trichloroethane	79-00-5	4.00E-03	5.70E-02	na	5.60E-02	IRIS

Notes:

na – not applicable

IRIS – Integrated Risk Information System – online

PPRTV- Provisional Peer Reviewed Toxicological Values

ATSDR MRL – Agency for Toxic Substances and Disease Registry Minimal Risk Levels for Hazardous Substances

Chromium IV values were used for chromium since they are stricter, thus making this a more conservative approach.

a. Noncarcinogenic Effects.

(1) For chemicals that exhibit noncarcinogenic effects, authorities consider organisms to have repair and detoxification capabilities that must be exceeded by some critical concentration (threshold) before the health effect is manifested. For example, an organ can have a large number of cells performing the same or similar functions that must be significantly depleted before the effect on the organ is seen. This threshold view holds that a range of exposures from just above the zero to some finite value can be tolerated by the organism without an appreciable risk of adverse effects.

(2) Health criteria for chemicals exhibiting noncarcinogenic effects for use in risk assessment are generally developed using EPA RfDs developed by the EPA's RfD/Reference Concentration (RfC) Work Group and included in the IRIS. In general, the RfD is an estimate of an average daily exposure to an individual (including sensitive individuals) below which there will not be an appreciable risk of adverse health effects. The RfD is derived using uncertainty factors (e.g., to adjust from animals to humans and to protect sensitive subpopulations) to ensure that it is unlikely to underestimate the potential for adverse noncarcinogenic effects to occur. The purpose of the RfD is to provide a benchmark against which an intake (or an absorbed dose in the case of dermal contact) from human exposure to various environmental conditions might be compared. Intakes of doses which are significantly higher than the RfD may indicate that an inadequate margin of safety could exist for exposure to that substance and that an adverse health effect could occur.

(3) Reference Doses.

(a) The types of toxicity values used to evaluate the noncarcinogenic effects of chemicals include RfDs which represent thresholds for toxicity. They are derived such that human lifetime exposure to a given chemical via a given route at levels at or below the RfD, as appropriate, should not result in adverse health effects, even for the most sensitive members of the population. The chronic RfD for a chemical is ideally based on studies where either animal or human populations were exposed to a given chemical by a given route of exposure for the major portion of the life span (referred to as a chronic study). Various effect levels may be determined in a study; however, the preferred effect level for calculating noncarcinogenic toxicity values is the no-observed-adverse-effect level or NOAEL. Second to the NOAEL is the lowest-observed-adverse-effect level or LOAEL.

(b) The oral RfD is derived by determining dose-specific effect levels from all the available quantitative studies, and applying uncertainty factors and/or a modifying factor to the most appropriate effect level. Uncertainty factors are intended to account for: 1) the variation in sensitivity among members of the human population, 2) the uncertainty in extrapolating animal data to humans, 3) the uncertainty in extrapolating from data obtained in a study that is less than lifetime exposure, 4) the uncertainty in using LOAEL data rather than NOAEL data, and 5) the uncertainty resulting from inadequacies in the database. The modifying factor may be used to account for other uncertainties such as inadequacy of the number of animals in the critical study. Usually each of these uncertainty factors is set equal to 10, while the modifying factor varies between 1 and 10. RfDs are reported as doses in milligrams of chemical per kilogram body weight per day (mg/kg-day).

(4) Exposure Periods. As mentioned earlier, chronic RfDs are intended to be set at levels such that human lifetime exposure at or below these levels should not result in adverse health effects, even for the most sensitive members of the population. These values are ideally based on chronic exposure studies in humans or animals. Chronic exposure for humans is considered to be exposure of roughly 7 years or more, based on exposure of rodents for 1 year or more in animal toxicity studies.

b. Carcinogenic Effects.

(1) For chemicals that exhibit carcinogenic effects, most authorities recognize that one or more molecular events can evoke changes in a single cell or a small number of cells that can lead to tumor formation. This is the nonthreshold theory of carcinogenesis, which purports that any level of exposure to a carcinogen can result in some finite possibility of generating the disease. Generally, regulatory agencies assume the nonthreshold hypothesis for carcinogens in the absence of information concerning the mechanisms of action for the COPC. The EPA uses a relative potency factor (RPF) approach for assessing the carcinogenic impacts of PAHs individually. The carcinogenic slope factor (CSF) for benzo(a)pyrene is multiplied by the PAH's RPF to determine potential carcinogenic impacts for the other PAHs.

(2) EPA's Carcinogen Risk Assessment Verification Endeavor (CRAVE) has developed CSFs and unit risks (i.e., dose-response values) for estimating excess lifetime carcinogenic risks associated with various levels of lifetime exposure to potential human carcinogens. The CSFs can be used to estimate the lifetime excess carcinogenic risk associated with exposure to a potential carcinogen. Risks estimated using slope factors are considered unlikely to underestimate actual risks, but they may overestimate actual risks. Excess lifetime carcinogenic risks are generally expressed in scientific notation. An excess lifetime carcinogenic risk of 1×10^{-6} or 1E-06 (one in a million), for example,

represents the probability of an individual developing cancer over a lifetime as a result of exposure to the specific carcinogenic chemical. The EPA considers total excess lifetime carcinogenic risks within the range of 1×10^{-4} (one in ten thousand) to 1×10^{-6} (EPA, 1989) to be acceptable when developing remedial alternatives for cleanup of Superfund sites. In practice, slope factors are derived from the results of human epidemiology studies or chronic animal bioassays. The data from animals studies are fitted to the linearized, multistage model and a dose-response curve is obtained. The upper limit of the 95th percentile confidence-interval slope of the dose-response curve is subjected to various adjustments, and an interspecies scaling factor is applied to conservatively derive the slope factor for humans. This linearized multistage procedure leads to a plausible upper limit of the risk that is consistent with some proposed mechanisms of carcinogenesis. Thus, the actual risks associated with exposure to a potential carcinogen are not likely to exceed the risks estimated using these slope factors, but they may be much lower. Dose-response data derived from human epidemiological studies are fitted to dose time-response curves on an ad-hoc basis. These models provide rough but plausible estimates of the upper limits on lifetime risk. Slope factors based on human epidemiological data are also derived using very conservative assumptions and, as such, are considered unlikely to underestimate risks. In summary, while the actual risks associated with exposures to potential carcinogens are unlikely to be higher than the risks calculated using a slope factor, they could be considerably lower.

(3) Slope factors and unit risks are developed by the EPA based on epidemiological or animal bioassay data for a specific route of exposure, either oral or inhalation. For some chemicals, sufficient data are available to develop route-specific slope factors for inhalation and ingestion. For chemicals with only one route-specific slope factor but for which carcinogenic effects may also occur via another route, the available slope factor may be used by the EPA to evaluate risks associated with several potential routes of exposure (EPA, 1992).

B-6. RISK CHARACTERIZATION.

a. Introduction. To characterize risk, toxicity and exposure assessments were summarized and integrated into quantitative and qualitative expressions of risk. To characterize potential carcinogenic effects, probabilities that a hypothetical individual will develop cancer over a lifetime of exposure are estimated from projected intakes and chemical-specific dose-response information. Major assumptions, scientific judgments, and, to the extent possible, estimates of the uncertainties embodied in the assessment are also presented. For the residential risk, the adult exposure was estimated for a 30-year time period and the child exposure was estimated for a 6-year time period.

b. Noncarcinogenic Effects.

(1) The potential for noncarcinogenic effects is evaluated by comparing an exposure level over a specified time period with an RfD derived for a similar exposure period. This ratio of exposure to toxicity is called a hazard quotient (HQ) according to the following equation:

$$\text{Noncarcinogenic Hazard Quotient} = E/\text{RfD}$$

Where:

E = Exposure level or intake (mg/kg-day)

RfD = Reference dose (mg/kg-day)

(2) The noncarcinogenic HQ assumes that there is a level of exposure (i.e., an RfD) below which it is unlikely for even sensitive populations to experience adverse health effects. If the exposure level (E) does not exceed the threshold (i.e., if E/RfD does not exceed unity), there is no concern for potential noncarcinogenic effects.

(3) To assess the overall potential for noncarcinogenic effects posed by more than one exposure pathway, an HI approach has been developed by the EPA. This approach assumes that simultaneous subthreshold exposures to several exposure pathways could result in an adverse health effect. It also assumes that the magnitude of the adverse effect will be proportional to the sum of the ratios of the subthreshold exposures to respective acceptable exposures.

(4) This is expressed as:

$$\text{HI} = E1/\text{RfD}1 + E2/\text{RfD}2 + \dots + E_i/\text{RfD}_i$$

Where:

E_i = Exposure level or intake of the I pathway

RfD_i = Reference dose for the ith pathway

(5) If an HI is greater than 1, the COPCs are subdivided into categories based on the target organ affected by exposure (e.g., liver, kidney) in accordance with EPA guidance (EPA, 1989). A target organ screen was not performed for this assessment because it was mainly arsenic that was contributing the majority of the risk.

c. Carcinogenic Effects.

(1) For carcinogens, risks are estimated as the incremental probability of a hypothetical individual developing cancer over a lifetime as a result of exposure to the potential carcinogen (i.e., excess individual lifetime carcinogenic risk). The slope factor converts estimated daily intakes averaged over a lifetime of exposure directly to incremental risk of a hypothetical individual developing cancer. It can generally be assumed that the dose-response relationship will be linear in the low-dose portion of the multistage model dose-response curve. Under this assumption, the slope factor is a

constant, and risk will be directly related to intake. Thus, the following linear low-dose equation was used in this assessment:

$$\text{Risk} = \text{CDI} \times \text{SF}$$

Where:

- Risk = A unitless probability of a hypothetical individual developing cancer
CDI = Chronic Daily Intake over 70 years (mg/kg-day)
SF = Slope Factor (mg/kg-day)⁻¹

(2) Because the slope factor is often an upper 95th percentile confidence limit of the probability of a response and is based on animal data used in the multistage model, the carcinogenic risk will generally be an upper-bound estimate. This means that the "true risk" is not likely to exceed the risk estimate derived through this model and is likely to be less than predicted. For simultaneous exposure by more than one pathway, the EPA assumes that the risks are additive. That is to say:

$$\text{RiskT} = \text{Risk1} + \text{Risk2} + \dots + \text{Riski}$$

Where:

- RiskT = Total carcinogenic risk, expressed as a unitless probability
Riski = Risk estimate for the ith pathway

(3) According to guidance in the National Contingency Plan (EPA, 1990), the target overall lifetime carcinogenic risks from exposures for determining clean-up levels should range from 1×10^{-4} to 1×10^{-6} .

d. Risk Summary. Carcinogenic and noncarcinogenic risks were calculated for all applicable exposure routes and are presented below in Tables B-14 through B-20.

(1) Soil. The total carcinogenic risk from all soil exposure routes is within the EPA target range of 1×10^{-4} to 1×10^{-6} for all receptors except for the child which was slightly above. The noncarcinogenic risk for the adult resident, and industrial and construction workers do not exceed the EPA's threshold level of 1, but the HI value for the future child resident is over the threshold level (EPA, 1989). This indicates that exposure to this soil does not pose a health risk to future adult residents, and industrial and construction workers but it may pose a potential risk to the future child resident at the site. Most of the total risk is due to arsenic, which has been identified in a previous report to be associated with gravel material containing Coal Slag used to pave the roads near the warehousing area (USACHPPM, 2006). Based on the conservative assumptions used in this risk assessment, the estimated risk for the site is likely an overestimate. Table B-14 shows the results of these risk calculations for incidental ingestion of soil. The results of the risk calculations for dermal absorbed dose are shown in Table B-15, and the risks from inhalation of soil can be found in Table B-16.

(2) Ground Water. The total carcinogenic risk levels for future site residents and industrial workers were found to be within the levels considered safe by the EPA at the site. These carcinogenic risks are within the EPA's acceptable range of 1×10^{-4} to 1×10^{-6} (EPA, 1989). The total HI or noncarcinogenic risk ranged from 0.04 for the adult resident and 0.09 for the child resident to 0.03 for the industrial worker. The HI value for the adult and child resident and the industrial worker does not exceed the EPA's threshold level of 1 (EPA, 1989). This indicates that exposure to this ground water does not pose a health risk to future residents (adult and child) and industrial workers at the site. Table B-17 shows the results of these risk calculations for incidental ingestion of ground water. The results of the risk calculations for dermal absorbed dose are shown in Table B-18.

(3) Risk Results. Table B-19 summarizes the calculated noncarcinogenic risks for all receptors and exposure routes considered in this risk assessment. Table B-20 summarizes the calculated carcinogenic risks for all receptors and exposure routes in this risk assessment.

Table B-14. Carcinogenic and Noncarcinogenic Risk from the Ingestion Exposure Route for all Receptors.

Soil				
Chemical	Industrial Worker	Construction Worker	Adult Resident	Child Resident
Arsenic	2.39E-01nc 3.84E-05ca	7.88E-01nc 5.07E-06ca	3.34E-01nc 6.45E-05ca	3.12E+00nc 1.20E-04ca
Cadmium	1.01E-04nc	3.33E-04nc	1.41E-04nc	1.32E-03nc
Chromium	3.20E-05nc	1.06E-02nc	4.48E-03nc	4.18E-02nc
Copper	2.77E-04nc	9.13E-04nc	3.87E-04nc	3.61E-03nc
Zinc	7.27E-05nc	2.40E-04nc	1.02E-04nc	9.51E-04nc
Molybdenum	1.18E-04nc	3.89E-04nc	1.65E-04nc	1.54E-03nc

Note: nc is used to represent noncarcinogenic and ca is used to represent carcinogenic

Table B-15. Carcinogenic and Noncarcinogenic Risk from the Dermal Exposure Route for All Receptors.

Soil				
Chemical	Industrial Worker	Construction Worker	Adult Resident	Child Resident
Arsenic	4.73E-02nc 7.60E-06ca	7.09E-02nc 4.56E-07ca	4.00E-02nc 7.72E-06ca	2.62E-01nc 1.01E-05ca
Cadmium	6.65E-07nc	9.98E-07nc	5.63E-07nc	3.69E-06nc
Chromium	2.11E-05nc	3.17E-05nc	1.79E-05nc	1.17E-04nc
Copper	1.83E-06nc	2.74E-06nc	1.54E-06nc	1.01E-05nc
Zinc	4.80E-07nc	7.20E-07nc	4.06E-07nc	2.66E-06nc

Molybdenum	7.79E-07nc	1.17E-06nc	6.59E-07nc	4.32E-06nc
------------	------------	------------	------------	------------

Note: nc is used to represent noncarcinogenic and ca is used to represent carcinogenic

Table B-16. Carcinogenic and Noncarcinogenic Risk from the Inhalation of Soil Particles for All Receptors.

Soil				
Chemical	Industrial Worker	Construction Worker	Adult Resident	Child Resident
Arsenic	5.65E-08ca	6.98E-09ca	9.49E-08ca	1.90E-08ca
Cadmium	9.10E-07nc 3.33E-11ca	1.13E-08nc 4.12E-12ca	1.27E-06nc 5.60E-11ca	1.27E-06nc 1.12E-11ca
Chromium	1.73E-04nc 2.12E-08ca	2.14E-06nc 2.62E-09ca	2.42E-04nc 3.56E-08ca	2.42E-04nc 6.95E-09ca
Copper	na	na	na	na
Zinc	na	na	na	na
Molybdenum	na	na	na	na

Note: nc is used to represent noncarcinogenic, ca is used to represent carcinogenic, and na is defined as not applicable

Table B-17. Carcinogenic and Noncarcinogenic Risk from the Ingestion Exposure Route for all Receptors.

Ground Water				
Chemical	Industrial Worker	Adult Resident	Child Resident	Construction Worker
Boron	1.81E-02nc	2.54E-02nc	5.92E-02nc	na
1,1,2-Trichloroethane	7.93E-04nc 6.45E-08ca	1.11E-03nc 1.08E-07ca	5.55E-04nc 5.06E-08ca	na
Tetrachloroethene	1.04E-03nc 2.00E-06ca	1.45E-03nc 3.36E-06ca	3.39E-03nc 1.57E-06ca	na
Heptachlor Epoxide	1.05E-02nc 4.45E-07ca	1.48E-02nc 7.48E-07ca	3.44E-02nc 3.94E-07ca	na

Note: nc is used to represent noncarcinogenic, ca is used to represent carcinogenic, and na is defined as not applicable

Table B-18. Carcinogenic and Noncarcinogenic Risk from the Dermal Exposure route for All Receptors.

Ground Water				
Chemical	Industrial Worker	Adult Resident	Child Resident	Construction Worker
Boron	8.05E-08nc	1.47E-06nc	4.34E-06nc	na
1,1,2-Trichloroethane	4.34E-06nc 3.54E-10ca	5.25E-07nc 1.20E-10ca	2.89E-06nc 5.65E-11ca	na
Tetrachloroethene	3.20E-07nc 6.17E-10ca	1.01E-05nc 5.44E-08ca	2.97E-05nc 1.38E-08ca	na

Heptachlor Epoxide	2.95E-15nc 8.76E-11ca	6.53E-05nc 7.72E-09ca	1.92E-04nc 1.95E-09ca	na
--------------------	--------------------------	--------------------------	--------------------------	----

Note: * nc is used to represent noncarcinogenic, ca is used to represent carcinogenic, and na is defined as not applicable

Table B-19. Total Noncarcinogenic Risk Results.

Soil				
Pathway	Industrial Worker	Construction Worker	Adult Resident	Child Resident
Ingestion	2.43E-01	7.09E-02	3.39E-01	3.17E+00
Dermal	4.72E-02	2.40E-03	4.00E-02	2.62E-01
Inhalation	1.74E-04	2.18E-06	2.43E-04	2.43E-04
TOTAL SITE RISK	2.90E-01	8.71E-01	3.79E-01	3.43E+00
Ground Water				
Pathway	Industrial Worker	Construction Worker	Adult Resident	Child Resident
Ingestion	3.05E-02	na	4.27E-02	9.76E-02
Dermal	1.47E-05	na	4.22E-06	7.33E-04
TOTAL SITE RISK	3.05E-02	na	4.30E-02	9.83E-02

Note: na = not applicable

Table B-20. Total Carcinogenic Risk Results.

Soil				
Pathway	Industrial Worker	Construction Worker	Adult Resident	Child Resident
Ingestion	3.84E-05	5.07E-06	6.45E-05	1.20E-04
Dermal	7.60E-06	4.56E-07	7.72E-06	1.01E-05
Inhalation	7.77E-08	9.60E-09	1.31E-07	2.61E-08
TOTAL SITE RISK	4.61E-05	5.53E-06	7.23E-05	1.31E-04
Ground Water				
Pathway	Industrial Worker	Construction Worker	Adult Resident	Child Resident
Ingestion	2.51E-06	na	3.26E-04	1.97E-06
Dermal	1.39E-09	na	9.16E-08	1.77E-08
TOTAL SITE RISK	2.51E-06	na	4.31E-06	1.99E-06

Note: na = not applicable

B-7. LEAD RISK CHARACTERIZATION.

a. Introduction.

(1) The intake of lead has a wide variety of effects on humans. Blood lead concentrations of 10 µg/dL or more have been associated with adverse health effects in children (EPA, 1986; CDC, 1991). Exposure to lead should be limited such that the probability of a typical (or hypothetical) child, or group of similarly exposed children, having or exceeding the 10 µg/dL blood lead concentration is less than 5% (EPA, 1994).

(2) A separate method of calculating the concentration of lead in blood is used. This has been developed due to the relation between chronic health effects from lead exposure and elevated blood lead levels.

(3) Blood lead levels are dependent on both background exposure to lead and site-related exposures. Since adverse effects of lead are dependent upon the age of the exposed individual, measures of blood lead levels are believed to be more accurate counterparts for potential effects of lead than are average daily exposure levels (daily doses).

b. Potential Lead Toxicity.

(1) The potential for adverse effects from lead exposure was evaluated using the EPA IEUBK and the EPA ALM. The IEUBK is designed to predict blood lead levels in children (0-6 years old) and cannot be used to predict blood lead levels in adults (EPA, 2005). The EPA ALM was selected to estimate blood lead levels of future receptors because of the current and potential future industrial land use of the site (EPA, 2003a). The EPA ALM is based on models by Bowers et al. (1994) and was developed by the Technical Review Workgroup to provide a consistent approach for evaluating long-term nonresidential exposures to lead in soil. Both models take into account intake and uptake components of lead exposure using site-specific data to predict concentrations of lead in blood.

(2) Potential blood lead increments for commercial/industrial, construction/utility, and maintenance workers were used to assess potential subchronic risk associated with incidental exposure to lead in soil under the various scenarios. The central estimate of blood lead concentrations ($PbB_{adult,central}$) for receptors that have site exposures to lead in soil were determined using the following equation and parameters (EPA, 2003b):

$$PbB_{adult,central} = PbB_{adult,baseline} + \frac{PbS \cdot BKSF \cdot IR_s \cdot AF_s \cdot EF_s}{AT}$$

Where:

$PbB_{adult,central}$ = Central estimate of blood lead concentrations ($\mu\text{g/dL}$) in receptors that have site exposures to soil lead at concentration, PbS .

$PbB_{adult,baseline}$ = Typical blood lead concentration ($\mu\text{g/dL}$) in adults in the absence of exposures to the site that is being assessed.

PbS = Soil lead concentration (mg/kg) (appropriate average concentration for individual).

$BKSF$ = Biokinetic slope factor relating (quasi-steady state) increase in typical adult blood lead concentration to average daily lead uptake ($\mu\text{g/dL}$ blood lead increase per $\mu\text{g/day}$ lead uptake).

IR_s = Intake rate of soil, including both outdoor soil and indoor soil-derived dust (g/day).

AF_s = Absolute gastrointestinal absorption fraction for ingested lead in soil and lead in dust derived from soil (dimensionless).

EF_s = Exposure frequency for contact with assessed soils and/or dust derived in part from these soils (days of exposure during the averaging period); may be taken as days per year for continuing, long term exposure.

AT = Averaging time; the total period during which soil contact may occur; 365 days/year for continuing long-term exposures.

(3) To determine the average soil lead concentration (PbS) to enter into the ALM and IEUBK, the 95th UCL for lead was used. Refer to the Site Investigation report (Kemron, 2005), the Continued Site Investigation Addendum (USACHPPM, 2006) and the Final Sampling Summary Report (CATI, 2006) for a comprehensive list of the lead sample identification numbers, lead concentrations, and date of collection at the site. The 95th UCL lead concentration in soil for the site is 38.352 mg/kg .

(4) In order to determine if the receptor blood lead level exceeds 10 $\mu\text{g/dL}$, the geometric standard deviations (GSD) of 1.8 for homogeneous populations and 2.1 for heterogeneous populations were used. Specific soil ingestion rates for an industrial worker (100 mg/day) and a construction worker (330 mg/day) were used in the EPA ALM calculations in order to be consistent with the recommended default values. Table B-21 shows the estimated blood lead level in receptors according to the EPA ALM and

Table B-22 shows the estimated child blood levels according to the EPA IEUBK model. Refer to Appendix C for the complete lead model tables.

$$PbB_{fetal} = R_{fetal/maternal} \bullet PbB_{adult}$$

(5) The estimated blood lead level is used to estimate the blood lead level in the fetus of the child-bearing receptor (i.e., commercial/industrial worker) ($PbB_{fetal,central}$) using the following equation:

Where:

PbB_{fetal} = Fetal blood lead concentration ($\mu\text{g/dL}$) (which, like PbB_{adult} , is a variable quantity having the specified probability distribution).

$R_{fetal/maternal}$ = Constant of proportionality between fetal and maternal blood lead concentrations.

PbB_{adult} = Adult blood lead concentration ($\mu\text{g/dL}$), estimated with parameters appropriate to women of child bearing age.

Table B-21. Blood Lead Levels Using the EPA ALM.

Exposure Variable	Blood Lead Levels	
	GSDi=1.8	GSDi=2.1
PbB adult, central (Industrial worker)	2.11	2.11
PbB adult, 0.95 (Industrial worker)	5.55	7.15
PbB fetal, 0.95 (Industrial worker)	5.00	6.44
PbB adult, central (Construction worker)	2.36	2.36
PbB adult, 0.95 (Construction worker)	6.22	8.01
PbB fetal, 0.95 (Construction worker)	5.60	7.21

Table B-22. Future Child Blood Lead Levels Using the EPA IEUBK.

Year	Blood Lead Levels ($\mu\text{g}/\text{dL}$)
0.5-1	0.9
1-2	1.3
2-3	1.2
3-4	1.1
4-5	1.0
5-6	0.9
6-7	0.8

c. Lead Toxicity Summary. As Table B-21 shows, the EPA ALM 95th UCL blood lead level for both the construction worker and industrial worker heterogeneous and homogeneous populations are below the EPA threshold of 10 μg Pb/dL. The estimated fetal blood concentrations for both the construction worker and industrial worker heterogeneous and homogeneous populations do not exceed the EPA threshold. The estimated adult central blood concentrations for both the construction worker and the industrial worker heterogeneous and homogeneous populations are also below the EPA threshold. The estimated blood levels for the future child resident, calculated using the IEUBK, are all well below the EPA threshold of 10 μg Pb/dL for the ages of 0-7 years. Based on the results of these models, the site does not pose a lead exposure risk.

B-8. UNCERTAINTY ASSESSMENT. All risk assessments involve the use of assumptions, judgments, and imperfect data to varying degrees. This results in uncertainty in the final estimates of risk. There are uncertainties associated with each component of the risk assessment from data collection through risk characterization. For example, there is uncertainty in the initial selection of substances used to characterize exposures and risk on the basis of the sampling data and available toxicity information. Other sources of uncertainty are inherent in the toxicity values for each substance and the exposure assessments used to characterize risk. Finally, additional uncertainties are incorporated into the risk assessment when exposures across multiple pathways are summed. Areas of uncertainty in each risk assessment step are discussed below.

a. Uncertainty in Data Collection and Evaluation.

(1) Uncertainties in the data collection/evaluation step of the risk assessment limit determining whether enough samples were collected to adequately characterize the risk, and if sample analyses were conducted in a qualified manner to maximize the confidence in the results. Results of the sample analyses were used to develop a database, which includes a complete list of the chemicals in the soil and their representative concentrations used in the risk assessment. The sampling and analysis addressed various objectives in addition to the risk assessment. Therefore, the samples were not collected randomly but were collected from areas of the site with the greatest likelihood to contain the COPCs. This type of nonrandom sampling biases the data collected toward overestimating chemical concentrations from the site.

(2) Chemicals that were never detected were eliminated from the assessment. This practice may slightly underestimate risks due to low levels (i.e., below the sample quantitation limit) of eliminated chemicals; however, it is unlikely that the addition of these chemicals would lead to a significant health risk.

(3) When calculating the 95th UCL there is an uncertainty associated with chemicals that have numerous nondetects, because this can cause the 95th UCL to be unreliable thus having to default to using the maximum concentration as the 95th UCL value.

(4) During the risk screening estimate calculations, the metal samples were checked against the background soil levels sampled in a previous report conducted by Versar Inc. in 1993 for the entire Camp Pedricktown and not the Reserve enclave in particular. These levels may or may not be indicative of onsite naturally occurring metal concentrations.

b. Uncertainty in Exposure Assessment.

(1) A large part of the risk assessment is the estimation of risks for a broad set of exposure scenarios and pathways. If exposure does not occur, no risks are present. This assessment does not factor in the probability of the exposure occurring. For certain pathways, exposure may be extremely unlikely. This assumption yields an overestimate of risk.

(2) Once pathways are identified, EPCs must be estimated. There is always some doubt as to how well an exposure model approximates the actual conditions receptors will be exposed to at a given site. Key assumptions in estimating EPCs and exposure assumptions and their potential impact on the assessment are described in the following paragraphs.

(a) There are many factors which determine the level of exposure for each exposure pathway. These factors include ingestion rates, exposure frequencies, exposure durations, and body weight. The values for these exposure factors must be selected by the risk assessor to represent each receptor. For the scenarios in this risk assessment,

upper-bound values were selected for each exposure factor. In the calculations of exposure, these multiple upper-bound exposure factors estimates compound to yield intakes which overestimate likely exposure levels.

(b) The EPCs derived from the measured chemical concentrations are assumed to persist without change for the entire duration of each exposure scenario. It is likely that chemical concentrations in the soil will change over time. Unfortunately, it is not known whether the quality will improve or degrade. Therefore, this steady state assumption could tend to under or overestimate exposure levels.

c. Uncertainty in the Toxicity Assessment. There is considerable uncertainty inherent in the toxicity values for both carcinogens and noncarcinogens. Many of the studies are based on animals and extrapolated to humans, and in some cases, subchronic studies must be used to assess chronic effects. Most CSFs are calculated using a model which extrapolates low-dose effects from high-dose animal studies. Because toxicity constants are generally based on the upper limit of the 95th percentile confidence interval or incorporate safety factors to compensate for uncertainty, chemical-specific risks may be overestimated.

d. Uncertainty in Risk Characterization. Uncertainties in the toxicity assessment are compounded under the assumption of dose additivity for multiple substance/pathway exposure. That assumption ignores possible synergism and antagonisms among chemicals, and assumes similarity in mechanisms of action and metabolism. Overall, these assumptions could tend to under or overestimate risk. Similarly, risks summed for chemicals having different target organs may also tend to overestimate risk.

B-9. CONCLUSIONS.

a. Carcinogenic and Noncarcinogenic Risks.

(1) Soil. The total carcinogenic risk levels for future site adult residents, and industrial and construction workers were found to be within the levels considered safe by the EPA at the site. The total carcinogenic risk levels ranged from 7×10^{-5} for adult residents and 5×10^{-5} for the industrial worker to 6×10^{-6} for the construction worker. These carcinogenic risks are within the EPA's acceptable range of 1×10^{-4} to 1×10^{-6} (EPA, 1989). The future child resident risk value of 1.3×10^{-4} slightly exceeds the EPA's acceptable range of 1×10^{-4} to 1×10^{-6} . The total HI or noncarcinogenic risk ranged from 0.87 for the construction worker and 0.29 for the industrial worker to 0.38 for the adult resident and 3.43 for the child resident. The HI values for the adult resident, and industrial and construction workers do not exceed the EPA's threshold level of 1, but the HI value for the future child resident is over the EPA threshold level (EPA, 1989). This indicates that exposure to this soil does not pose a health risk to future adult residents, and industrial and construction workers but it could pose a potential risk to the future child resident at the site. Most of the total risk is due to arsenic, which has been identified in a previous report to be associated with gravel material containing Coal Slag

used to pave the roads near the warehousing area (USACHPPM, 2006). Table B-14 shows the results of these risk calculations for incidental ingestion of soil. The results of the risk calculations for the dermal absorbed dose are shown in Table B-15, and the risks from inhalation of soil can be found in Table B-16.

(2) Ground Water. The total carcinogenic risk levels for future site residents and industrial workers were found to be within the levels considered safe by the EPA at the site. These carcinogenic risks are within the EPA's acceptable range of 1×10^{-4} to 1×10^{-6} (EPA, 1989). The total HI or noncarcinogenic risk ranged from 0.04 for the adult resident and 0.09 for the child resident to 0.03 for the industrial worker. The HI value for the adult and child resident and the industrial worker does not exceed the EPA's threshold level of 1 (EPA, 1989). This indicates that exposure to this ground water does not pose a health risk to future residents (adult and child) and industrial workers at the site. Table B-17 shows the results of these risk calculations for incidental ingestion of ground water. The results of the risk calculations for the dermal absorbed dose are shown in Table B-18.

(3) Total Risk Results. Table B-19 summarizes the calculated noncarcinogenic risks for all receptors and exposure routes considered in this risk assessment and Table B-20 summarizes the calculated carcinogenic risks for all receptors and exposure routes in this risk assessment.

b. Lead Toxicity. The EPA ALM 95th percentile blood lead level for both the construction worker and industrial worker heterogeneous and homogeneous populations are below the EPA threshold of 10 $\mu\text{g Pb/dL}$. The estimated fetal blood concentrations for both the industrial worker and construction worker heterogeneous and homogeneous populations do not exceed the EPA threshold. The estimated adult central blood concentrations for both the construction worker and the industrial worker heterogeneous and homogeneous populations are also below the EPA threshold. The estimated blood levels for the future child resident, calculated using the IEUBK, are all well below the EPA threshold of 10 $\mu\text{g Pb/dL}$ for the ages of 0-7 years. Based on the results of these models, this site does not pose a lead exposure risk.

**APPENDIX C
LEAD MODEL TABLES**

Table C-1: Lead Exposure to the Industrial Worker from the Camp Pedricktown Reserve Enclave using the EPA Adult Lead Model.

Exposure Variable	Description of Exposure Variable	Values for Two Generic Sites (days)	
		One site	
$PbB_{adult,central}$			
$PbB_{adult,0.95}$	adult central PbB	GSDi = 1.8	GSDi = 2.1
$PbB_{fetal,0.95}$	95 th percentile adult PbB	2.11045376	2.11045376
$R_{fetal/maternal}$	95 th percentile PbB in fetus	5.550078325	7.151984771
BKSF	Fetal/maternal PbB ratio	4.995070492	6.436786294
	Biokinetic Slope Factor	0.9	0.9
	Geometric standard deviation PbB	0.4	0.4
	Baseline PbB	1.8	2.1
	Soil ingestion rate (including soil-derived indoor dust)	2.0	2.0
	Weighting factor; fraction of time at site 1	0.100	0.100
	Absorption fraction	--	--
	Exposure frequency soil site 1	0.12	0.12
	The averaging time (AT) is a fixed value of 365 days/yr.	219	219
		38	38
		365	365

Soil site 1 is the 95th UCL value for lead in soil at the site
 GSDi=1.8 is the default value given for a homogeneous population
 GSD=2.1 is the default value given for a heterogeneous population

Integrated Exposure Uptake Biokinetic Model (IEUBK) for Lead in Children

Model Version: 1.0 Build 264

The time step used in this model run: 2 – Daily (once a day)

----Air----

Indoor Air PB concentration: 0 percent of outdoor

Other Air Parameters:

Age	Time Outdoors(hr)	Ventilation Rate (m ³ /day)	Lung Absorption (%)	Outdoor Air Pb Concentration (ug Pb/ m ³)
0.5-1	1.0	2.0	32	0.100
1-2	2.0	3.0	32	0.100
2-3	3.0	5.0	32	0.100
3-4	4.0	5.0	32	0.100
4-5	4.0	5.0	32	0.100
5-6	4.0	7.0	32	0.100
6-7	4.0	7.0	32	0.100

-----Diet-----

Age	Diet intake (ug/day)
0.5-1	0.00
1-2	0.00
2-3	0.00
3-4	0.00
4-5	0.00
5-6	0.00
6-7	0.00

----Drinking Water----

Age	Water (L/day)
0.5-1	0.200
1-2	0.500
2-3	0.520
3-4	0.530
4-5	0.550
5-6	0.580
6-7	0.590

Drinking Water Concentration: 6.575 ug Pb/L

-----Soil and Dust-----

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0.5-1	38.352	0.00
1-2	38.352	0.00
2-3	38.352	0.00
3-4	38.352	0.00
4-5	38.352	0.00
5-6	38.352	0.00
6-7	38.352	0.00

-----Alternate Intake-----

Age	Alternate (ug Pb/day)
0.5-1	0.00
1-2	0.00
2-3	0.00
3-4	0.00
4-5	0.00
5-6	0.00
6-7	0.00

-----Maternal Contribution: Infant Model-----

Maternal Blood Concentration: 2.500 ug Pb/dL

Calculated Blood Lead and Lead Uptakes

Age	Air (ug/day)	Diet (ug/day)	Alternate (ug/day)	Water (ug/day)	Soil+Dust (ug/day)	Total (ug/day)	Blood (ug/dL)
0.5-1	0.003	0.00	0.00	0.646	0.960	1.609	0.9
1-2	0.008	0.00	0.00	1.600	1.512	3.120	1.3
2-3	0.020	0.00	0.00	1.670	1.517	3.207	1.2
3-4	0.027	0.00	0.00	1.707	1.522	3.256	1.1
4-5	0.027	0.00	0.00	1.780	1.132	2.939	1.0
5-6	0.037	0.00	0.00	1.880	1.021	2.939	0.9
6-7	0.037	0.00	0.00	1.915	0.966	2.918	0.8

Health Risk Assessment No. 39-DA-07ZE-08, Camp Pedricktown Reserve Enclave, NJ,
Jan 08