6.0 PUBLIC HEALTH EVALUATION AND ENVIRONMENTAL CONCERNS

The purpose of this chapter is to fulfill the public health evaluation requirements of the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) and the Comprehensive Environmental Response, Compensation and Liability Act of 1980 (CERCLA) as amended by the Superfund Amendments and Reauthorization Act of 1986 (SARA). The standards used as criteria for evaluation of the project analytical results comply with the "Applicable or Relevant and Appropriate Requirements" (ARARs). The public health evaluation assesses the risks associated with the no-action alternative at the site and develops risk-based goals for remedial alternatives. Conduct of this evaluation is intended to ensure that the Superfund remedial actions provide adequate public health protection to reduce risks to an acceptable level. The assessment addresses both the human health and environmental consequences associated with the existing contamination. This chapter was prepared in accordance with the U. S. EPA's Superfund Public Health Evaluation Manual (14) and the U. S. EPA Superfund Exposure Assessment Manual (13).

6.1 Hazard Identification

6.1.1 Site Contamination History

The Berks Sand Pit Site originally was created by the removal of sand and gravel from the area that is now the Van Elswyck property. The local residents reportedly used the pit for refuse disposal and observed tank trucks traveling Benfield Road between September and November 1981. Therefore, industrial waste also was assumed to have been disposed of at this site.

During January 1982, groundwater contamination was detected in residential well water both by the distinguishable odor and obnoxious taste and laboratory analysis by the PADER. The following chemicals were detected:

1,1,1-Trichloroethane	> 45,000 µg/l
1,1-Dichloroethene	> 800 µg/l
1,1-Dichloroethane	> 300 µg/l
1,2-Dichloromethane	> 150 µg/l
Toluene	> 150 µg/l

The EPA conducted a clean up effort on the R-3 property during the summer of 1983. This consisted of excavating the area reported to be the sand pit and installing a water supply well

for four residences whose wells were contaminated by the previous disposal operations. The excavation did not encounter any buried drums or other objects relating to the contamination.

6.1.2 Nature and Extent of Problem

The primary risk associated with the previous disposal of liquids at the Berks Sand Pit Site is the degradation of the groundwater quality. The public health hazards associated with the disposal operations are mainly chemical in nature.

The contaminants, primarily solvents, have infiltrated into the groundwater system and thereby entered the potable water supply. Due to the highly irregularly fractured nature of the bedrock, the contaminants have dispersed to various degrees in a vertical and horizontal direction. The geologic investigation indicated that fractured bedrock was encountered at depths to 300 feet and that based on existing published information, the fractures probably extend beyond 300 feet. Continued use of the groundwater by residents not yet affected by the contaminants may have caused the chemicals to be pulled farther through the fractures than normally would have occurred.

Groundwater contamination persists to this day, as indicated by elevated levels of organic compounds such as 1,1,1-trichloroethane, tetrachloroethene, and metals such as lead, copper, and manganese. The predominant organic contaminant at the site is 1,1,1-trichloroethane and is being used as an indicator of the relative concentrations of other organics. The contaminant plume is migrating to the northeast and the rate appears to be accelerating after three private wells were taken out of service because of extreme contamination. These wells apparently were acting as barrier wells which were preventing or slowing the contaminant plume from migrating downgradient, thereby initially limiting the extent of the affected area.

6.1.3 Selection of Indicator Chemicals

A total of 40 chemicals were identified and validated at the Berks Sand Pit Site from sampling the groundwater, surface water, soils, sediments and air. The chemicals included 10 volatile organic compounds (VOCs), 10 semi-volatile organic compounds (SVOCs), and 20 metals (M). Groundwater (monitoring wells and residential wells) contained a total of 28 chemicals (eight VOCs, six SVOCs, and 14 Ms); surface water (seeps, springs, and creek samples) contained a total of 24 chemicals (five VOCs, two SVOCs, and 17 Ms); soil/sediment

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(monitoring well cuttings, test borings, and surface water sediments) contained a total of 28 chemicals (three VOCs, six SVOCs, and 19 Ms), and air contained six chemicals (6 Ms).

The large number of validated chemicals found above minimum detection level (MDL) at the Berks Sand Pit Site prevented the Public Health Evaluation process from focusing on the chemicals of greatest concern. Therefore, an indicator chemical selection process was used to identify the "highest risk" chemicals at the site. These chemicals posing the greatest potential public health risk at the site were chosen so that they represented the chemicals with the most toxic, mobile, persistent, and greatest amounts available.

The selection process consisted of the following four steps (14):

1. Identification of the chemicals present at the site above minimum detection levels.

2. Determine maximum and representative concentrations from the site monitoring data base.

3. Calculation of the indicator scores from the maximum and representative chemical concentrations and the route-specific toxicity data.

4. Selection of final indicator chemicals based on indicator scores and the chemical properties affecting exposure and risk at the site.

The minimum and representative chemical concentrations were developed from the site monitoring computer data base and included the concentration values recorded above the MDL and passing the QA/QC data validation process (see Section 4.0). The representative chemical concentration is the sum of the concentrations of a specific chemical divided by the number of positive (greater than the MDL) samples. The average positive concentration is an arithmetic mean of the samples where the chemical was detected.

The route-specific toxicity data required to calculate the indicator scores (IS) was taken from Exhibits C-3 and C-5 in Appendix C (14). The algorithm used to score each chemical measured at the site (14) was:

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$$IS_i = \sum_{j=1}^{3} (C_{ij} \cdot T_{ij})$$

where

IS_i = indicator score for chemical i (unitless)

C_{ij} = concentration of chemical i in medium j based on the maximum and representative concentrations from the site monitoring data

T_{ij} = route-specific toxicity constant for chemical i in medium j.

The IS is a ration between the maximum and representative chemical concentrations and a route-specific toxicity constant and is used to rank the chemicals at the site. The toxicity constants units are medium-specific and inverses of their respective medium-specific concentration units such that the IS is unitless.

The air sampling results (see Table 5-3) were not included in the selection process because the sampling was for a health and safety protocol for the onsite sampling program and the results were not QA/QC'd. Also, there were no volatile organics detected during the air sampling program.

Table 6-1 shows the Koc values and concentrations used in the selection process. The representative concentration was the average concentration of all values greater than zero, the average positive concentration. Table 6-2 gives the toxicity information available in Appendix C (14) for each of the chemicals. Table 6-3 and 6-4 give the results of the calculation of the concentrations (maximum and representative) times the toxicity constants and the IS scores for potential carcinogens and noncarcinogens, respectively. These last two tables also rank the IS scores for the maximum ranking and the representative ranking.

An examination of the raw data (see Appendix E), laboratory procedures and, especially, the site history of the sources of contamination allows for the elimination of many of the chemicals found at the Berks Sand Pit Site. The following paragraphs summarize the justifications used to reduce the total number of chemicals found at the site.

The chemicals methylene chloride, acetone, toluene, and the common phthalate esters are described as common contaminant compounds in the EPA Laboratory Data Validation Guidelines (94). When detected, these chemicals almost always were qualified with the letter

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Table 6-1

SCORING FOR INDICATOR CHEMICAL SELECTION: K_{oc} VALUES AND CONCENTRATIONS IN VARIOUS ENVIRONMENTAL MEDIA

Chemical	CAS #	K_{oc} Value (ml/g)	Groundwater (mg/l)							Surface Water (mg/l)						
			Max.	Min.	Samples	>0	Average	Average Positive	Ref.	Max.	Min.	Samples	>0	Average	Average Positive	Ref.
Volatile Organics																
Methylene Chloride	75092	8.8	4×10^{-1}	5×10^{-3}	63	12	1×10^{-2}	6×10^{-2}		3×10^{-2}	3×10^{-2}	25	1	1×10^{-3}	3×10^{-2}	
Acetone	67641	2.2	8×10^{-2}	2×10^{-2}	63	2	2×10^{-3}	5×10^{-2}		--	--	25	--	--	--	
1,1-Dichloroethene	75354	65	9×10^{-3}	9×10^{-3}	63	19	2×10^{-1}	5×10^{-1}		1×10^{0}	2×10^{-2}	25	8	5×10^{-2}	2×10^{-1}	
1,1-Dichloroethane	75343	30	--	--	63	--	--	--		9×10^{-3}	9×10^{-3}	25	1	4×10^{-4}	9×10^{-3}	
2-Butanone	78933	4.5	--	--	63	--	--	--		6×10^{-3}	6×10^{-3}	25	1	3×10^{-4}	7×10^{-3}	
1,1,1-Trichloroethane	71556	152	7×10^{0}	5×10^{-3}	63	32	6×10^{-1}	1×10^{0}		3×10^{0}	6×10^{-3}	25	8	2×10^{-1}	5×10^{-1}	
Benzene	71432	83	6×10^{-3}	6×10^{-3}	63	1	1×10^{-4}	6×10^{-3}		--	--	25	--	--	--	
Tetrachloroethene	127184	364	3×10^{-2}	6×10^{-3}	63	2	5×10^{-4}	2×10^{-2}		--	--	25	--	--	--	
Toluene	108883	300	7×10^{-3}	7×10^{-3}	63	1	1×10^{-4}	7×10^{-3}		--	--	25	--	--	--	
Xylene (total)	1330207	240	5×10^{-2}	8×10^{-3}	63	2	9×10^{-4}	3×10^{-2}		--	--	25	--	--	--	
Semi-Volatile Organics																
1,4-Dichlorobenzene	106467	1700	2×10^{-2}	2×10^{-2}	63	1	3×10^{-4}	2×10^{-2}		--	--	25	--	--	--	
4-Methylphenol	106445	--	--	--	63	--	--	--		--	--	25	--	--	--	
N-Nitrosodiphenylamine	86306	--	--	--	63	--	--	--		--	--	25	--	--	--	
Di-n-butyl phthalate	84742	--	1×10^{-2}	1×10^{-2}	63	1	2×10^{-4}	1×10^{-2}		--	--	25	--	--	--	
Fluoranthene	206440	38,000	--	--	63	--	--	--		--	--	25	--	--	--	
Bis(2-ethylhexyl)phthalate	117817	--	3×10^{-2}	3×10^{-2}	63	1	5×10^{-4}	3×10^{-2}		--	--	25	--	--	--	
Di-n-octyl phthalate	117840	--	3×10^{-1}	3×10^{-1}	63	1	4×10^{-3}	3×10^{-1}		--	--	25	--	--	--	
Benzo(b)fluoranthene	205992	550,000	3×10^{-4}	1×10^{-4}	63	5	2×10^{-3}	3×10^{-4}		3×10^{-4}	3×10^{-4}	25	1	3×10^{-4}	3×10^{-4}	
Benzo(k)fluoranthene	207089	550,000	3×10^{-4}	1×10^{-4}	63	5	2×10^{-3}	3×10^{-4}		3×10^{-4}	3×10^{-4}	25	1	3×10^{-4}	3×10^{-4}	
Benzo(a)pyrene	50328	5,500,000	--	--	63	--	--	--		--	--	25	--	--	--	

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Table 6-1 (Continued)

SCORING FOR INDICATOR CHEMICAL SELECTION: K_{oc} VALUES AND CONCENTRATIONS IN VARIOUS ENVIRONMENTAL MEDIA

Chemical	CAS #	K_{oc} Value (ml/g)	Groundwater (mg/l)							Surface Water (mg/l)						
			Max.	Min.	Samples	>0	Average	Average Positive	Ref.	Max.	Min.	Samples	>0	Average	Average Positive	Ref.
Metals																
Aluminum	7429905	--	2×10^{-1}	2×10^{-1}	63	2	7×10^{-3}	2×10^{-1}		1×10^{2}	5×10^{-1}	25	6	5×10^{0}	2×10^{1}	
Barium	7440393	--	2×10^{-1}	2×10^{-1}	63	1	3×10^{-3}	2×10^{-1}		3×10	3×10^{0}	25	1	1×10^{-1}	3×10^{0}	
Beryllium	7440417	--	--	--	63	--	--	--		--	--	25	--	--	--	
Cadmium	7440439	--	--	--	63	--	--	--		5×10^{-2}	5×10^{-3}	25	3	2×10^{-3}	2×10^{-2}	
Calcium	7440702	--	1×10^{2}	4×10^{0}	63	41	8×10^{0}	1×10^{2}		1×10^{2}	5×10^{0}	25	9	7×10^{0}	2×10^{1}	
Chromium	7440473	--	2×10^{-2}	1×10^{-2}	63	3	6×10^{-4}	1×10^{-2}		1×10^{-1}	1×10^{-1}	25	1	5×10^{-3}	1×10^{-1}	
Cobalt	7440484	--	--	--	63	--	--	--		--	--	25	--	--	--	
Copper	7440508	--	3×10^{-1}	3×10^{-2}	63	9	1×10^{-2}	1×10^{-1}		3×10^{1}	3×10^{-1}	25	1	1×10^{-2}	3×10^{-1}	
Iron	15438310	--	3×10^{1}	1×10^{-1}	63	21	2×10^{0}	5×10^{0}		8×10^{1}	1×10^{-1}	25	13	4×10^{0}	8×10^{0}	
Lead	7439921	--	--	--	63	--	--	--		6×10^{-2}	8×10^{-3}	25	4	4×10^{-3}	3×10^{-2}	
Cyanide	57125	--	4×10^{-2}	1×10^{-2}	63	14	5×10^{-3}	2×10^{-2}		7×10^{0}	2×10^{-2}	25	3	3×10^{-1}	3×10^{0}	
Magnesium	7439954	--	6×10^{0}	1×10^{0}	63	4	3×10^{-1}	4×10^{0}		2×10^{1}	5×10^{0}	25	2	1×10^{0}	1×10^{1}	
Manganese	7439965	--	2×10^{0}	2×10^{-2}	63	32	1×10^{-1}	2×10^{-1}		3×10^{0}	1×10^{-2}	25	13	2×10^{-1}	3×10^{-1}	
Mercury	7439976	--	4×10^{-4}	3×10^{-4}	63	2	1×10^{-5}	3×10^{-4}		2×10^{-3}	2×10^{-3}	25	1	8×10^{-5}	2×10^{-3}	
Nickel	7440020	--	3×10^{-1}	4×10^{-2}	63	4	8×10^{-3}	1×10^{-1}		2×10^{-1}	2×10^{-1}	25	1	9×10^{-3}	2×10^{-1}	
Potassium	7440097	--	2×10^{1}	2×10^{0}	63	9	1×10^{0}	9×10^{0}		8×10^{0}	8×10^{0}	25	2	6×10^{-1}	8×10^{0}	
Selenium	7782492	--	--	--	63	--	--	--		--	--	25	--	--	--	
Sodium	7440235	--	7×10^{1}	5×10^{0}	63	56	9×10^{0}	1×10^{1}		1×10^{3}	5×10^{0}	25	18	2×10^{2}	2×10^{2}	
Vanadium	7440622	--	--	--	63	--	--	--		3×10^{-1}	3×10^{-1}	25	1	1×10^{-2}	3×10^{-1}	
Zinc	7440666	--	2×10^{0}	2×10^{-2}	63	43	7×10^{-2}	1×10^{-1}		4×10^{0}	2×10^{-2}	25	6	2×10^{-1}	7×10^{-1}	

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Table 6-1 (Continued)

SCORING FOR INDICATOR CHEMICAL SELECTION: K_{oc} VALUES AND CONCENTRATIONS IN VARIOUS ENVIRONMENTAL MEDIA

Chemical	CAS #	K_{oc} Value (ml/g)	Soil (mg/kg)							Sediment (mg/kg)						
			Max.	Min.	Samples	>0	Average	Average Positive	Ref.	Max.	Min.	Samples	>0	Average	Average Positive	Ref.
Volatile Organics																
Methylene Chloride	75092	8.8	4×10^1	1×10^1	39	5	3×10^0	2×10^1		4×10^1	1×10^1	27	6	5×10^0	2×10^1	
Acetone	67641	2.2	5×10^1	6×10^0	39	3	2×10^0	2×10^1		--	--	27	--	--	--	
1,1-Dichloroethene	75354	65	--	--	39	--	--	--		--	--	27	--	--	--	
1,1-Dichloroethane	75343	30	--	--	39	--	--	--		2×10^3	2×10^3	27	1	9×10^0	2×10^3	
2-Butanone	78933	4.5	--	--	39	--	--	--		--	--	27	--	--	--	
1,1,1-Trichloroethane	71556	152	--	--	39	--	--	--		--	--	27	--	--	--	
Benzene	71432	83	--	--	39	--	--	--		--	--	27	--	--	--	
Tetrachloroethene	127184	364	--	--	39	--	--	--		--	--	27	--	--	--	
Toluene	108883	300	--	--	39	--	--	--		--	--	27	--	--	--	
Xylene (total)	1330207	240	--	--	39	--	--	--		--	--	27	--	--	--	
Semi-Volatile Organics																
1,4-Dichlorobenzene	106467	1700	--	--	39	--	--	--		--	--	27	--	--	--	
4-Methylphenol	106445	--	--	--	39	--	--	--		2×10^3	2×10^3	27	1	7×10^1	2×10^3	
N-Nitrosodiphenylamine	86306	--	--	--	39	--	--	--		4×10^3	4×10^3	27	1	1×10^2	4×10^3	
Di-n-butyl phthalate	84742	--	4×10^2	4×10^2	39	1	1×10^1	4×10^2		--	--	27	--	--	--	
Fluoranthene	206440	38,000	--	--	39	--	--	--		5×10^2	5×10^2	27	1	2×10^1	5×10^2	
Bis(2-ethylhexyl)phthalate	117817	--	6×10^2	2×10^3	39	2	2×10^2	4×10^3		--	--	27	--	--	--	
Di-n-octyl phthalate	117840	--	--	--	39	--	--	--		--	--	27	--	--	--	
Benzo(b)fluoranthene	205992	550,000	--	--	39	--	--	--		--	--	27	--	--	--	
Benzo(k)fluoranthene	207089	550,000	--	--	39	--	--	--		--	--	27	--	--	--	
Benzo(a)pyrene	50328	5,500,000	--	--	39	--	--	--		8×10^2	8×10^2	27	1	3×10^1	8×10^2	

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Table 6-1 (Continued)

SCORING FOR INDICATOR CHEMICAL SELECTION: K_{oc} VALUES AND CONCENTRATIONS IN VARIOUS ENVIRONMENTAL MEDIA

Chemical	CAS #	K_{oc} Value (ml/g)	Soil (mg/kg)							Sediment (mg/kg)						
			Max.	Min.	Samples	>0	Average	Average Positive	Ref.	Max.	Min.	Samples	>0	Average	Average Positive	Ref.
Metals																
Aluminum	7429905	--	2×10^4	4×10^3	39	30	8×10^3	1×10^4		3×10^4	2×10^3	27	27	1×10^4	1×10^4	
Barium	7440393	--	3×10^2	5×10^1	39	32	6×10^2	7×10^2		1×10^3	5×10^1	27	12	4×10^2	9×10^2	
Beryllium	7440417	--	3×10^0	1×10^0	39	7	3×10^{-1}	1×10^0		2×10^0	2×10^0	27	2	1×10^0	2×10^0	
Cadmium	7440439	--	2×10^0	2×10^0	39	1	5×10^{-2}	2×10^0		2×10^0	2×10^0	27	1	7×10^{-2}	2×10^0	
Calcium	7440702	--	--	--	39	--	--	--		2×10^4	1×10^3	27	2	7×10^2	9×10^3	
Chromium	7440473	--	8×10^1	2×10^0	39	21	8×10^0	2×10^1		4×10^1	3×10^0	27	23	1×10^1	2×10^1	
Cobalt	7440484	--	4×10^1	2×10^1	39	4	3×10^0	3×10^1		1×10^1	1×10^1	27	1	4×10^0	1×10^1	
Copper	7440508	--	1×10^2	6×10^0	39	13	8×10^0	2×10^1		7×10^1	6×10^0	27	9	8×10^0	3×10^1	
Iron	15438310	--	4×10^4	2×10^3	39	30	1×10^4	1×10^4		4×10^4	2×10^3	27	26	1×10^4	2×10^4	
Lead	7439921	--	2×10^1	1×10^0	39	35	6×10^0	7×10^0		4×10^2	4×10^0	27	19	3×10^1	4×10^1	
Cyanide	57125	--	2×10^0	6×10^{-1}	39	4	1×10^{-1}	1×10^0		2×10^1	8×10^{-1}	27	2	6×10^{-1}	8×10^0	
Magnesium	7439954	--	2×10^4	1×10^3	39	28	2×10^3	3×10^3		2×10^3	2×10^3	27	1	7×10^1	2×10^3	
Manganese	7439965	--	5×10^2	5×10^1	39	22	1×10^2	2×10^2		1×1^3	3×10^1	27	20	1×10^2	1×10^2	
Mercury	7439976	--	2×10	4×10^{-1}	39	2	7×10^{-2}	1×10^0		8×10^{-1}	2×10^{-1}	27	4	6×10^{-2}	4×10^{-1}	
Nickel	7440020	--	2×10^2	1×10^1	39	3	7×10^0	9×10^1		2×10^1	9×10^0	27	3	1×10^0	1×10^1	
Potassium	7440097	--	3×10^3	1×10^3	39	18	8×10^2	2×10^3		2×10^3	2×10^3	27	1	8×10^1	2×10^3	
Selenium	7782492	--	--	--	39	--	--	--		3×10^2	3×10^0	27	5	3×10^1	2×10^2	
Sodium	7440235	--	--	--	39	--	--	--		--	--	27	--	--	--	
Vanadium	7440622	--	1×10^2	1×10^1	39	13	1×10^1	4×10^1		7×10^1	2×10^1	27	2	3×10^0	4×10^1	
Zinc	7440666	--	7×10^1	1×10^1	39	33	3×10^1	3×10^1		3×10^2	1×10^1	27	25	4×10^1	5×10^1	

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Table 6-2

SCORING FOR INDICATOR CHEMICAL SELECTION: TOXICITY INFORMATION

Chemical	Toxic Class	Rating Category*	wT	sT	aT
Volatile Organics					
Methylene Chloride	NC	10	9.2×10^{-4}	4.6×10^{-8}	9.2×10^{-3}
Acetone					
1,1-Dichloroethene	PC	C	1.2×10^{-1}	6.1×10^{-6}	1.2
	NC	7 Oral	3.7×10^{-1}	1.9×10^{-5}	5.7
		5 Inhal			
1,1-Dichloroethane	NC	7	2.6×10^{-2}	1.3×10^{-6}	2.6×10^{-1}
2-Butanone	NC	10	7.8×10^{-3}	3.9×10^{-7}	7.8×10^{-2}
1,1,1-Trichloroethane	NC	2	7.3×10^{-4}	3.7×10^{-8}	7.3×10^{-3}
Benzene	PC	A	7.7×10^{-3}	3.9×10^{-7}	7.7×10^{-2}
	NC	5 Oral	1.2×10^{-1}	5.9×10^{-6}	1.2×10^{-2}
		10 Inhal			
Tetrachloroethene	PC	B2	8.9×10^{-3}	4.4×10^{-7}	8.9×10^{-2}
	NC	7 Oral	9.6×10^{-3}	4.8×10^{-7}	2.8×10^{-2}
		10 Inhal			
Toluene	NC	7	5.2×10^{-3}	2.6×10^{-7}	5.2×10^{-2}
Total Xylenes					
Semi-Volatile Organics					
1,4-Dichlorobenzene	NC	4 Oral	5.2×10^{-2}	2.6×10^{-6}	3.6×10^{-1}
		5 Inhal			
4-Methylphenol					
N-Nitrosodiphenylamine					
Di-n-Butyl Phthalate					

NC = Noncarcinogen
PC = Potential carcinogen
wT = Route-specific toxicity constant for water
sT = Route-specific toxicity constant for soil/sediment
aT = Route-specific toxicity constant for air

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Table 6-2 (Continued)

SCORING FOR INDICATOR CHEMICAL SELECTION: TOXICITY INFORMATION

Chemical	Toxic Class	Rating Category*	wT	sT	aT
Fluoranthene					
bis(2-Ethylhexyl)Phthalate	PC	B2	5.7×10^{-4}	2.9×10^{-8}	5.7×10^{-3}
Di-n-Octyl Phthalate					
Benzo(b)Fluoranthene					
Benzo(k)Fluoranthene					
Benzo(a)Pyrene	PC	B2	4.6	2.3×10^{-4}	4.6×10^{1}
	NC	8 Oral	2.7×10^{1}	1.3×10^{-3}	1.9×10^{1}
		6 Inhal			
Metals					
Aluminum					
Barium	NC	10	4.1	2.0×10^{-4}	4.1×10^{1}
Beryllium	PC	B1 Inhal			2.3×10^{1}
	NC	8 Inhal			1.5×10^{4}
Cadmium	PC	B1 Inhal			1.7×10^{1}
	NC	10 Oral	4.5	2.2×10^{-4}	3.6×10^{-2}
		8 Inhal			
Calcium					
Chromium	PC	A Inhal			1.1×10^{2}
	NC	8 Inhal			2.5×10^{1}
Cobalt					
Copper	NC	5	7.1×10^{-1}	3.6×10^{-5}	7.1
Iron					
Lead	NC	10	8.9×10^{-1}	4.5×10^{-5}	8.9
Cyanide					
Magnesium					
Manganese					

NC = Noncarcinogen
PC = Potential carcinogen
wT = Route-specific toxicity constant for water
sT = Route-specific toxicity constant for soil/sediment
aT = Route-specific toxicity constant for air

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Table 6-2 (Continued)

SCORING FOR INDICATOR CHEMICAL SELECTION: TOXICITY INFORMATION

Chemical	Toxic Class	Rating Category*	wT	sT	aT
Mercury	NC	7 Oral 8 Inhal	1.8×10^{1}	9.2×10^{-4}	1.9×10^{2}
Nickel	PC	A Inhal			2.9
	NC	10	4.3	2.1×10^{-4}	1.6×10^{2}
Potassium					
Selenium	NC	10	1.1×10^{2}	3.5×10^{-3}	1.1×10^{3}
Sodium					
Vanadium	NC	1	1.4×10^{-1}	7.1×10^{-6}	1.4
Zinc	NC	8	1.1×10^{-1}	5.3×10^{-6}	1.1

NC = Noncarcinogen
PC = Potential carcinogen
wT = Route-specific toxicity constant for water
sT = Route-specific toxicity constant for soil/sediment
aT = Route-specific toxicity constant for air

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Table 6-2 (Continued)

*Summarized below are the descriptions and effects for the Rating Categories (14).

EPA WEIGHT-OF-EVIDENCE
CATEGORIES FOR POTENTIAL CARCINOGENS

EPA Category	Description of Group	Description of Evidence
Group A	Human Carcinogen	Sufficient evidence from epidemiologic studies to support a causal association between exposure and cancer
Group B1	Probable Human Carcinogen	Limited evidence of carcinogenicity in humans from epidemiologic studies
Group B2	Probable Human Carcinogen	Sufficient evidence of carcinogenicity in animals, inadequate evidence of carcinogenicity in humans
Group C	Possible Human Carcinogen	Limited evidence of carcinogenicity in animals
Group D	Not Classified	Inadequate evidence of carcinogenicity in animals
Group E	No Evidence of Carcinogenicity in Humans	No evidence for carcinogenicity in at least two adequate animal tests or in both epidemiologic and animal studies

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Table 6-2 (Continued)

RATING CONSTANTS (RVe) FOR NONCARCINOGENS

Effect	Severity Rating (RVe)
Enzyme induction or other biochemical change with no pathologic changes and no change in organ weights.	1
Enzyme induction and subcellular proliferation or other changes in organelles but no other apparent effects.	2
Hyperplasia, hypertrophy or atrophy, but no change in organ weights.	3
Hyperplasia, hypertrophy or atrophy with changes in organ weights.	4
Reversible cellular changes: cloudy swelling, hydropic change, or fatty changes.	5
Necrosis, or metaplasia with no apparent decrement of organ function. Any neutropathy without apparent behavioral, sensory, or physiologic changes.	6
Necrosis, atrophy, hypertrophy, or metaplasia with a detectable decrement of organ functions. Any neuropathy with a measurable change in behavioral , sensory, or physiologic activity.	7
Necrosis, atrophy, hypertrophy, or metaplasia with definitive organ dysfunction. Any neuropathy with gross changes in behavior, sensory, or motor performance. Any decrease in reproductive capacity, any evidence of fetotoxicity.	8
Pronounced pathologic changes with severe organ dysfunction. Any neuropathy with loss of behavioral or motor control or loss of sensory ability. Reproductive dysfunction. Any teratogenic effect with maternal toxicity.	9
Death or pronounced life-shortening. Any teratogenic effect without signs of material toxicity.	10

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Table 6-3

SCORING FOR INDICATOR CHEMICAL SELECTION: CALCULATION OF CONCENTRATION X TOXICITY CONSTANTS (CT) AND INDICATOR SCORE (IS) VALUES FOR CARCINOGENIC EFFECTS

Chemical	Groundwater CT		Surface Water CT		Soil CT		Sediment CT		IS Value		Rank		
	Max.	Repres.	Max.	Repres.	Max.	Repres.	Max.	Repres.	Max.	Repres.	Orig.	Max.	Repres.
Benzo(a)pyrene	--	--	--	--	--	--	1.7×10^{-1}	1.7×10^{-1}	1.7×10^{-1}	1.7×10^{-1}	5	2	1
1,1-Dichloroethene	4.3×10^{-1}	6.6×10^{-2}	1.2×10^{-1}	2.0×10^{-2}	--	--	--	--	4.3×10^{-1}	6.6×10^{-2}	1	1	2
Tetrachloroethene	2.2×10^{-4}	1.4×10^{-4}	--	--	--	--	--	--	2.2×10^{-4}	1.4×10^{-4}	3	3	3
bis(2-ethylhexyl)phthalate	1.9×10^{-5}	1.9×10^{-5}	--	--	1.7×10^{-4}	1.2×10^{-4}	--	--	1.9×10^{-4}	1.3×10^{-4}	4	4	4
Benzene	4.9×10^{-5}	4.9×10^{-5}	--	--	--	--	--	--	4.9×10^{-5}	4.9×10^{-5}	2	5	5

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Table 6-4

SCORING FOR INDICATOR CHEMICAL SELECTION: CALCULATION OF CONCENTRATION X TOXICITY CONSTANTS (CT) AND INDICATOR SCORE (IS) VALUES FOR NONCARCINOGENIC EFFECTS

Chemical	Groundwater CT		Surface Water CT		Soil CT		Sediment CT		IS Value		Rank		
	Max.	Repres.	Max.	Repres.	Max.	Repres.	Max.	Repres.	Max.	Repres.	Orig.	Max.	Repres.
Barium	8.1×10^{-1}	8.1×10^{-1}	1.4×10^{1}	1.4×10^{1}	5.2×10^{-2}	1.5×10^{-2}	3.0×10^{-2}	1.9×10^{-2}	1.4×10^{1}	1.4×10^{1}	21	1	1
Nickel	1.1×10^{0}	5.2×10^{-1}	9.3×10^{-1}	9.3×10^{-1}	3.2×10^{-2}	1.9×10^{-2}	3.6×10^{-3}	2.9×10^{-3}	1.1×10^{0}	9.5×10^{-1}	34	4	2
Selenium	–	–	–	–	–	–	1.2×10^{0}	6.2×10^{-1}	1.2×10^{0}	6.2×10^{-1}	36	3	3
1,1-Dichloroethene	1.3×10^{0}	2.0×10^{-1}	3.7×10^{-1}	5.9×10^{-2}	–	–	–	–	1.3×10^{0}	2.0×10^{-1}	3	2	4
Copper	2.1×10^{-1}	6.8×10^{-2}	1.9×10^{-1}	1.9×10^{-1}	3.9×10^{-3}	8.6×10^{-4}	2.4×10^{-3}	9.0×10^{-4}	2.1×10^{-1}	1.9×10^{-1}	27	7	5
Benzo(a)Pyrene	–	–	–	–	–	–	1.7×10^{-1}	1.7×10^{-1}	1.7×10^{-1}	1.7×10^{-1}	19	8	6
Cadmium	–	–	2.1×10^{-1}	8.7×10^{-2}	4.7×10^{-4}	4.3×10^{-4}	4.2×10^{-4}	4.2×10^{-4}	2.1×10^{-1}	8.8×10^{-2}	23	6	7
Zinc	1.9×10^{-1}	1.0×10^{-2}	4.1×10^{-1}	7.1×10^{-2}	3.8×10^{-4}	1.6×10^{-4}	1.4×10^{-3}	2.5×10^{-4}	4.1×10^{-1}	7.2×10^{-2}	39	5	8
Vanadium	–	–	3.8×10^{-2}	3.8×10^{-2}	7.1×10^{-4}	2.8×10^{-4}	4.7×10^{-4}	3.0×10^{-4}	3.9×10^{-2}	3.8×10^{-2}	38	10	9
Mercury	6.8×10^{-3}	5.9×10^{-3}	3.5×10^{-2}	3.7×10^{-2}	2.2×10^{-3}	1.3×10^{-3}	6.9×10^{-4}	3.8×10^{-4}	3.7×10^{-2}	3.8×10^{-2}	33	11	10
Lead	–	–	5.5×10^{-2}	2.2×10^{-2}	1.0×10^{-3}	2.9×10^{-4}	1.7×10^{-2}	1.9×10^{-3}	7.2×10^{-2}	2.4×10^{-2}	29	9	11
1,1,1-Trichloroethane	5.4×10^{-3}	9.0×10^{-4}	1.9×10^{-3}	3.8×10^{-4}	–	–	–	–	5.4×10^{-3}	9.0×10^{-4}	6	12	12
1,4-Dichlorobenzene	8.3×10^{-4}	8.2×10^{-4}	–	–	–	–	–	–	8.3×10^{-4}	8.2×10^{-4}	11	13	13
Benzene	7.6×10^{-4}	7.6×10^{-4}	–	–	–	–	–	–	7.6×10^{-4}	7.6×10^{-4}	7	14	14
1,1-Dichloroethane	–	–	2.4×10^{-4}	2.4×10^{-4}	–	–	3.1×10^{-4}	3.1×10^{-4}	5.5×10^{-4}	5.5×10^{-4}	4	15	15
Tetrachloroethene	2.4×10^{-4}	1.5×10^{-4}	–	–	–		–	–	2.4×10^{-4}	1.5×10^{-4}	8	17	16
Methylene Chloride	3.7×10^{-4}	5.3×10^{-5}	2.5×10^{-3}	2.5×10^{-3}	1.9×10^{-4}	1.1×10^{-4}	1.6×10^{-4}	1.0×10^{-4}	3.7×10^{-4}	5.4×10^{-3}	1	16	17
2-Butanone	–	–	5.0×10^{-3}	5.0×10^{-3}	–	–	–	–	5.0×10^{-3}	5.0×10^{-3}	5	18	18
Toluene	3.7×10^{-3}	3.6×10^{-3}	–	–	–	–	–	–	3.7×10^{-3}	3.6×10^{-3}	9	19	19
Aluminum	–	–	–	–	–	–	–	–	–	–	20	20	20

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Table 6-4 (Continued)

SCORING FOR INDICATOR CHEMICAL SELECTION: CALCULATION OF CONCENTRATION X TOXICITY CONSTANTS (CT) AND INDICATOR SCORE (IS) VALUES FOR NONCARCINOGENIC EFFECTS

Chemical	Groundwater CT		Surface Water CT		Soil CT		Sediment CT		IS Value		Rank		
	Max.	Repres.	Max.	Repres.	Max.	Repres.	Max.	Repres.	Max.	Repres.	Orig.	Max.	Repres.
Potassium	--	--	--	--	--	--	--	--	--	--	35	30	21
Benzo(b)Fluoranthene	--	--	--	--	--	--	--	--	--	--	18	31	22
Magnesium	--	--	--	--	--	--	--	--	--	--	31	22	23
4-Methylphenol	--	--	--	--	--	--	--	--	--	--	12	32	24
Manganese	--	--	--	--	--	--	--	--	--	--	32	24	25
Chromium	--	--	--	--	--	--	--	--	--	--	25	33	26
Di-n-Butyl Phthalate	--	--	--	--	--	--	--	--	--	--	14	26	27
Sodium	--	--	--	--	--	--	--	--	--	--	37	34	28
N-Nitrosodiphenylamine	--	--	--	--	--	--	--	--	--	--	13	28	29
Fluoranthene	--	--	--	--	--	--	--	--	--	--	15	35	30
Cyanide	--	--	--	--	--	--	--	--	--	--	30	21	31
Di-n-Octyl Phthalate	--	--	--	--	--	--	--	--	--	--	16	36	32
Calcium	--	--	--	--	--	--	--	--	--	--	24	25	33
Benzo(b)Fluoranthene	--	--	--	--	--	--	--	--	--	--	17	37	34
Iron	--	--	--	--	--	--	--	--	--	--	28	29	35
Cobalt	--	--	--	--	--	--	--	--	--	--	26	27	36
Beryllium	--	--	--	--	--	--	--	--	--	--	22	23	37
Xylenes (Total)	--	--	--	--	--	--	--	--	--	--	10	38	38
Acetone	--	--	--	--	--	--	--	--	--	--	2	39	39

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"B," denoting the presence of the chemical in the laboratory blank. Bis(2-ethylhexyl) phthalate ranked only fourth overall out of five chemicals with IS scores for carcinogenic effects. Methylene chloride and toluene ranked very low for noncarcinogenic effects; the other blank contaminants did not have an IS score.

Many of the chemicals were only detected one time and included 2-butanone, benzene, xylene (total), 1,4-dichlorobenzene, 4-methylphenol, N-nitrosodiphenylamine, fluoranthene, and benzo(a)pyrene. The infrequent occurrence of these chemicals as well as the relatively low levels found in the samples indicate that these chemicals should not be classified as high risk chemicals. Benzo(a)pyrene did rank the highest chemical in the ranking of IS scores for carcinogenic effects. However, this was for a single occurrence of the chemical in sediments. In the ranking of noncarcinogenic effect IS scores, benzo(a)pyrene, benzene and 1,4-dichlorobenzene were the only chemicals with IS scores above zero. Two other chemicals, benzo(b)fluoranthene and benzo(k)fluoranthene, occurred a total of six times in the samples. However, these two chemicals did not have IS scores above zero. Collectively, the chemicals discussed above were not seen as related to the source of contamination, either chemically or in the occurrence in the samples.

The metals are the final category of chemicals to be removed by the selection process. The following metals were not considered to be toxic or pose a public health and environmental risk: aluminum, calcium, cobalt, iron, magnesium, potassium and sodium. These seven chemicals did not have IS scores above zero. Other chemicals not having an IS score above zero were beryllium, chromium and cyanide. Of the remaining metals, barium received the highest ranking of the IS scores for noncarcinogenic effects. However, this was due to a single occurrence each in a the groundwater sample and a surface water sample. Similarly, mercury and vanadium were found in only three and one water sample, respectively. Selenium was found only in sediment samples. The remaining metals, cadmium, lead, nickel and zinc, were not found in samples related to the source contamination. Nickle, which ranked second in the IS value scoring, had the highest concentration in a residential well (R-13) that did not have any corresponding detected concentrations of the suspected source contaminants.

The four chemicals that were selected as the highest risk chemicals included the volatile organics 1,1-dichloroethane, 1,1-dichloroethene, 1,1,1-trichloroethane and tetrachloroethene. These chemicals, especially 1,1-dichloroethene and 1,1,1-trichloroethane, represent the most toxic, mobile and persistent chemicals at the site given the frequency of occurrence and the

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chemical relationship to the suspected source of contamination, the industrial sovlents reportedly dumped in the Berks Sand Pit Site and frequently found in previous analyses.

6.2 Dose-Response Assessment

6.2.1 Introduction

The information required to evaluate the chemicals at the Berks Sand Pit Site includes the ARARs, toxicity data for non-carcinogens (Acceptable Intakes Chronic - AIC) and for carcinogens (Potency Factor - PF), health effects, and chemical and physical data. The sources of information included the Integrated Risk Information System (IRIS); Health Effects Assessment documents (HEA); Toxicological Profiles (TP); the Superfund Public Health Evaluation Manual (SPHEM) appendices; the Chemical, Physical, and Biological Properties of Compounds Present at Hazardous Waste Sites (CPBP); and general reference texts.

6.2.2 Toxicity and Environmental Profiles

The primary volatile organic chemicals of concern are the halogenated aliphatic compounds, specifically the alkanes and alkenes, that were reportedly dumped in the sand and gravel pit. Section 6.2.2.1 summarizes the transport and fate processes and health effects for these chemicals. Although cis- and trans-1,2-dichloroethene was deleted from the working database because of QA/QC invalidation, the chemical was included in the chemical summaries because it was an intermediate in degradation pathways. The following two paragraphs discuss generically the transport and fate processes and the health effects.

Transport and Fate. The distribution relationships for a chemical between the environmental components of air, water, soil, and biota are expressed by a series of equilibrium constants. The air-water distribution of a chemical can be expressed in three ways: the partition coefficient; relative volatility; and, most commonly, the Henry's Law constant (H). For soil-water partitioning, the soil sorption constant (K_d) relates the amount of chemical sorbed to soil to the concentration in water. Because organic matter is the key to the sorption process in many soils, the sorption characteristics of a chemical often can be predicted better using the soil organic carbon partition coefficient (K_{oc}) rather than K_d. The K_{oc} expresses the degree to which a chemical partitions between the soil and water based on the soil's organic content. Two physical constants, the n-octanol-water partition coefficient (K_{ow}) and water solubility,

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can be used to predict the K_{oc} for a chemical. The K_{ow} is inversely related to water solubility and also has been correlated with bioconcentration factors for many organic compounds. Thus, by utilizing the physical properties of a chemical (water solubility, vapor pressure, partition coefficients), it is possible to estimate a chemical's expected environmental distribution (16).

Health Effects. Without understanding a chemical's distribution in the environment, it is impossible to evaluate its public health risks. The uncertainties involved in the risk assessment process itself are numerous. To ensure the protection of public health, an EPA risk assessment is based on a series of worst case assumptions (18). For example, the reference dose (RfD) or acceptable daily intake (ADI) is an estimate of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfD assumes that thresholds exist for certain toxic effects (example: cellular necrosis), but may not exist for other toxic effects such as cancer. The uncertainty factors are obtained from multiplying factors to account for intraspecies variability, interspecies variability, and extrapolation of a subchronic effect level to its chronic equivalent. The confidence that the EPA has in a RfD (low, medium, or high) is based on the confidence and uncertainties of existing principle animal studies and other data such as epidemiological studies. To determine the risk from chronic exposure to a carcinogen, the chronic daily intake (CDI) is multiplied by the carcinogenic potency factor (the slope of the dose-response curve) (14, 17).

6.2.2.1 Chemical Summaries

The following chemical summaries discuss the implications of the chemical and physical properties on the transport and fate of the selected indicator chemicals. The sections include discussions of estimated or laboratory determined half-lives and of biotic and abiotic degradation and transformation products. The sections discuss briefly the data and procedures used to derive RfDs, the noncarcinogenic and carcinogenic effects, and the other ARARs such as MCLs and ambient water quality criteria. Tables 6-5, 6-6 and 6-7 present this data for physical and chemical properties and health related information, respectively.

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Table 6-5

BERKS SAND PIT
CHEMICAL AND PHYSICAL CONSTANTS OF INDICATOR CHEMICALS

Chemical	IS Values		Ranking		Water Solubility (mg/l)	Vapor Pressure (mm Hg)	Henry's Law Constant (atm-m^3/mole)	K_{oc}	Log K_{ow}	Half-Lives (Days)				IC*
	PC	NC	PC	NC						GW	SW	Soil	Air	
1,1-Dichloroethene					2250	600	0.034	65	1.84	-	1-6	-	2	+
1,1-Dichloroethane					5500	182	0.00431	30	1.79	-	1-5	-	45	+
1,1,1-Trichloroethane					1500	123	0.0144	152	2.5	-	0.14-7.00	-	803-1752	+
Tetrachloroethene	0.28	0.28	2	2	150	18	0.026	364	2.6	264	1-30	-	47	+

GW = Groundwater
SW = Surface Water

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Table 6-6

BERKS SAND PIT SITE
SUMMARY OF PUBLIC HEALTH AND ENVIRONMENTAL CRITERIA

	1,1-Dichloroethene	1,1-Dichloroethane	1,1,1-Trichloroethane	Tetrachloroethene
AIC - µg/kg/day				
Oral	10	120	90	10
Reference	IRIS	HEA	IRIS	HEA
Inhalation	-	138	6,000	-
Reference	-	HEA	HEA	-
PF - $(\mu g/kg/day)^{-1}$				
Oral	600	-	-	51
Reference	IRIS	-	-	HEA
Weight of Evidence	C	-	-	B2
Inhalation	1,200	-	-	1.7
Reference	IRIS	-	-	HEA
Weight of Evidence	C	-	6,000	B2
MCL - µg/l	7	-	200	-
EPA - WQC - µg/l				
Human Health				
Water and Fish	3.3×10^{-2}	9.4×10^{-1}	1.8×10^{4}	8.0×10^{-1}
Fish Only	1.85	2.43×10^{2}	1.03×10^{6}	8.85
Aquatic Organisms				
Acute	1.16×10^{4}	1.8×10^{5}	5.28×10^{4}	5.8×10^{3}
Chronic	-	2.0×10^{4}	-	8.4×10^{2}

AIC = acceptable intake chronic
MCL = maximum contaminant level
IRIS = Integrated Risk Information System

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Table 6-7

BERKS SAND PIT SITE
SUMMARY OF HEALTH EFFECTS DATA

	1,1-Dichloroethene	1,1-Dichloroethane	1,1,1-Trichloroethane	Tetrachloroethene
Oral LD_{50} - mg/kg				
Mice	200	-	11,240	8 - 11,000
Rat	200 - 1,800	725	10,300 - 12,300	>5,000
Inhalation LC_{50} - ppm				
Mice	-	17,300 (4 hours)	13,500 (10 hours)	5,200 (4 hours)
Rat	-	16,000 (8 hours)	14,000 (7 hours)	4,000 (4 hours)
CNS Depression	+	+	+	
Cardiac Sensitization	+	+		
Liver Damage	+	+	+	+
Kidney Damage	+	+	+	+

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1,1,1-Trichloroethane

- Transport and Fate

1,1,1-Trichloroethane disperses from surface waters (half-life 0.14 to 7.00 days) primarily by volatilization (14, 19). The chemical may be absorbed onto organic materials in the sediment, but it is not an important route of elimination from surface water (19). 1,1,1-Trichloroethane can readily be transported in groundwater, but the speed of transport depends on soil composition (14, 19). 1,1,1-Trichloroethane, unlike other chlorinated compounds, does not bioaccumulate in individual animals or food chains (23). Once present in soils or groundwater, 1,1,1-trichloroethane can undergo degradation by abiotic dehydrohalogenation to form acetic acid (20). Anaerobic bacteria can also biotransform 1,1,1-trichloroethane to 1,1-dichloroethane, and then finally to chloroethane (20). The half-life of 1,1,1-trichloroethane (by abiotic degradation) in water ranges from 0.5 to 1.7 years (20) while laboratory experiments have estimated the half-life by biotic degradation to be 16 days or less (21). Overall, photooxidation by reaction with hydroxyl radicals in the atmosphere is probably the principal fate process (19). The half-life range of 1,1,1-trichloroethane in air is 803 to 1,752 days (14).

- Health Effects

Noncarcinogenic Effects: The major sources of exposure to 1,1,1-trichloroethane were from contaminated water and air (23). Inhalation of 1,1,1-trichloroethane vapor through the lungs was a common route of entry into the body and when oral exposure occurred, it was absorbed rapidly and completely from the gastrointenstinal tract (87). Non-lethal acute intoxication has occurred after oral ingestion of a liquid ounce of the chemical (0.6 g/kg) (88). Excretion of 1,1,1-trichloroethane was primarily via the lungs (23). In fatalities resulting from inhalation of 1,1,1-trichloroethane, acute pulmonary congestion and edema typically was found (89, 90). The livers of exposed subjects had fatty vaculoations (90).

Several species of animals had acute LD_{50} ranging from 5.7 to 14.3 g/kg (91). The oral LD_{50} value in rats was about 11,000 mg/kg (23, 24). At 500 ppm, groups of guinea pigs showed no adverse effects compared with unexposed and air-exposed controls after exposure for seven hours/day, five days/week for six months (92). In the same

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study, groups of female guinea pigs exposed to 1,000 ppm 1,1,1-trichloroethane vapor three hours/day, five days/week for three months had fatty changes in the liver and statistically significant increased liver weights. This study defined a LOAEL of 500 ppm in guinea pigs. In another study, groups of guinea pigs were subjected to 650 ppm 1,1,1-trichloroethane vapor seven hours/day, five days/week for two to three months (93). The animals showed slight depression in weight again compared with both air-exposed and unexposed controls, thereby establishing a LOAEL of 650 ppm in guinea pigs. There was no dose-dependent effects on fertility, gestation, viability indices in mice exposed to 1,1,1-trichloroethane at dose levels from 100 to 1,000 mg/kg for 35 days.

The oral RfD of 9.0×10^{-2} mg/kg/day was determined using the LOAEL (120 mg/kg/day, converted) and the NOAEL (90 mg/kg/day, converted) identified in the above discussion (92, 93). A NOAEL of 90 mg/kg/day was divided by an uncertainty factor of 1,000 times a modifying factor of one (17). For the uncertainty factor, a factor of 10 was used for use of subchronic assay, for extrapolation from animal data, and protection of sensitive human subpopulations. Because the number of animals at each dose level was limited, the length of exposure varied with different dose levels and few toxic endpoints were examined, the confidence in the studies were low (17). The confidence in the database was medium. Therefore, the confidence in the RfD was considered medium to low (17). An AIC value of 6.0 mg/kg/day via the inhalation route has been determined by HEA (14).

A MCL of 200 µg/l was finalized in 1987. The EPA ambient water quality criteria for protection of human health was 1.8×10^{4} µg/l for water and fish consumption and 1.03×10^{6} µg/l for fish consumption only (17). The EPA ambient water quality criteria for the protection of aquatic organisms was an acute lowest effect level of 5.28×10^{4} µg/l.

Carcinogenic Effects: 1,1,1-Trichloroethane was retested for carcinogenicity because in a previous study early lethality precluded an assessment of carcinogenicity (19). Preliminary results indicated that 1,1,1-trichloroethane increased the incidence of combined hepatocellular carcinomas and adenomas in female mice when administered by gavage (22). The chemical was mutagenic in various strains of S. typhimurium, with metabolic activation although other studies with Saccharomyces cerevisiae or Schezosaccharomyces bombe were not mutagenic (23).

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Applying the EPA criteria guidelines for assessment of carcinogenic risk (69), 1,1,1-trichloroethane is classified in Group D: Not classified (23). This category is for chemicals with inadequate animal evidence of carcinogenicity.

1,2-Dichloroethene (cis and trans)

- Transport and Fate

The cis and trans isomers of 1,2-dichloroethene are expected to behave in the same manner in the environment based upon their similar physical properties (37). The primary transport process of 1,2-dichloroethenes is volatilization due to the relatively high vapor pressure (19). 1,2-Dichloroethene's half-life in surface water is 1 to 6 days (14) but, they are generally chemically stable in water (23). Aerial transport can occur and is partly responsible for its wide environmental distribution (19, 23). When released to the atmosphere, the chemicals are expected to degrade by photooxidation (23). The half-life in air for the trans isomer is 2.1 days and for the cis isomer is 1.3 days (14). 1,2-Dichloroethene is poorly adsorbed onto soils because of its low K_{oc} and log K_{ow}, but can be transported to groundwater from subsurface soils. 1,2-Dichloroethene's low log K_{ow} also suggests that bioaccumulation is relatively insignificant (14, 19, 23). Studies indicate that 1,2-dichloroethene is biologically degraded to vinyl chloride but at a very slow rate (20, 21). The 1,2-dichloroethenes have been found to co-occur with trichloroethene (23).

- Health Effects

Noncarcinogenic Effects: The 1,2-dichloroethenes are neutral, low molecular weight, lipid soluble materials that are readily absorbed by any exposure route (oral, inhalation, dermal) (63). Although kinetic data for tissue distribution are not available, if it follows the same absorption and distribution pattern as 1,1-dichloroethene, the highest concentration would be expected to be found in the liver and kidney (56). The position of the chlorine appears important in terms of metabolic activity; the cis isomer was metabolized at a faster rate than the trans isomer in an *in vitro* hepatic microsomal system (55). If excretion rate is similar to 1,1-dichloroethene, elimination would be rapid, with most of a single dose excreted in 27 to 72 hours (64).

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At high concentrations, chlorinated ethenes possess anesthetic properties with depression of the central nervous system and cardiac sensitizing effects (16, 19, 23, 25, 51). However, it appears that the trans-isomer is about twice as potent in depressing the central nervous as the cis isomer. An oral LD_{50} of 770 mg/kg of 1,2-dichloroethene mixture was reported for rats (58). A single dose of the cis isomer (400 mg/kg) caused a significant elevation of liver alkaline phosphates in rats (65). A trans isomer oral LD_{50} was 1,300 mg/kg in rats although when administered intraperitoneally, the LD_{50} was six-fold higher (67). At high exposure levels (8,000 to 16,000 ppm), the trans isomer can cause narcosis and death in rats after four hours (68). Prolonged exposure (up to 18 weeks) of rats to air containing the trans isomer (0 to 2,000 ppm) produced slight degeneration of the liver lobule (200 ppm), severe pneumonic infiltration (1,000 ppm), and significant decrease in the number of lencocytes (200 and 1,000 ppm) (67). Reproductive and developmental effects data was lacking for both isomers (23).

Although no lifetime toxicity data for 1,2-dichloroethenes exist, data from a two-year chronic drinking water study in rats with 1,1-dichloroethene (51) was used to determine the RfD. A LOAEL of 100 ppm (10 mg/kg) was identified based on an abnormal histopathology of minimal mid-zonal heptocellular fatty change and hepatocellular swelling. An oral RfD of 0.01 mg/kg/day was determined by dividing the LOAEL by an uncertainty factor of 1,000 and a modifying factor of one. For the uncertainty factor, a factor of 10 was used for use of a LOAEL, for interspecies variation, and for protection of sensitive human subpopulations. The confidence in the study was medium based on carroborative chronic and subchronic oral bioassays and use of appropriate methodology.

A MCLG of 70 µg/l was proposed in 1985 (14). The EPA ambient water quality criteria for protection of human health for water and fish consumption and for fish consumption only was 3.3×10^{-2} µg/l and 1.85 µg/l, respectively, for carcinogenicity protection. The EPA ambient water quality criteria for the protection of aquatic organisms was an acute lowest effect level of 1.16×10^{4} µg/l. These last two criteria were for the class of dichloroethenes, and not specifically for 1,2-dichlorothenes.

Carcinogenic Effects: At a medium concentration (2.3 to 2.9 mM), 1,2-dichloroethenes were not mutagenic, with or without microsomal activation, when assayed in *E. coli*

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K12 (70). In a diploid strain of Saccharomyces cerevisial, 1,2-dichloroethenes did not cause point mutation, mitotic gene conversion or mitotic recombination with or without microsomal activation (71). The isomers were not mutagenic in an in vivo (intravenous host-mediated assay) test (72).

There was no information found on the carcinogenic potential for the 1,2-dichloroethenes in a recent literature review (23). Applying the EPA criteria guidelines for assessment of carcinogenic risk (69), the 1,2-dichloroethenes are classified in Group D: Not classified (23). This category is for chemicals with inadequate animal evidence of carcinogenicity.

1,1-Dichloroethane

- Transport and Fate

Due to the high vapor pressure of 1,1-dichloroethane, volatilization from surface waters and soils is rapid and is the primary transport process (19). Similar to 1,1-dichloroethene, 1,1-dichloroethane has a low K_{oc} and log K_{ow} (19). Therefore, it is poorly absorbed onto soils and can be transported to groundwater. Most likely, bioaccumulation is relatively insignificant (14, 19). 1,1-Dichloroethane can undergo biotic degradation (by anaerobic bacteria) and form chloroethane (20). The biotic degradation rate reported in the laboratory for 1,1-dichloroethane was a bio half-life of "long" (21). At sites with 1,1,1-trichloroethane as a source compound, the concentration ratio of 1,1-dichloroethane increased with distance while the source compound decreased (21). However, chloroethane was detected when the source compound, 1,1,1-trichloroethane, was appreciably high, but further from the source, only 1,1-dichloroethane was present (21). These results were explainable by the bio half-life values for 1,1,1-trichloroethane and chloroethane of 16 and 10 days, respectively (21). Little is known about 1,1,-dichloroethane's half-life in soil, although its half-life in surface water is one to five days (14). In the air, 1,1-dichloroethane undergoes hydroxylation, and has a half-life of 45 days (14, 15).

- Health Effects

Noncarcinogenic Effects: 1,1-Dichloroethane has demonstrated liver and kidney toxicity in rodents, dogs, and monkeys (16). A number of key enzymes in the liver and

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kidney are affected by 1,1-dichloroethane (26, 27, 30). However, a review of the literature indicated that 1,1-dichloroethane may be one of the least toxic of the chlorinated ethanes (19). It can be significantly absorbed through the skin and is an experimental teratogen (74). Inhalation exposure to high doses of 1,1-dichloroethane (over 16,000 mg/m^3) caused retarded fetal development in rats (29). Central nervous system depression also occurs at high inhalation doses, and evidence suggest that the chemical is hepatotoxic in humans (19). The oral LD_{50} value in the rat is 725 mg/kg (28, 29). The inhalation LD_{50} in the mouse and rat was 17,300 ppm for two hours and 16,000 ppm for eights hours, respectively (54). However, there was no effect in the rat at 1,000 ppm, 5 x 6 hours/week for three months (54).

There was no RfD value for 1,1-dichloroethane. An HEA value for an oral route AIC was 1.2×10^{-1} mg/kg/day and for an inhalation route was 1.38×10^{-1} mg/kg/day (14). There were no values for MCLs, ambient water quality criteria for protection of human health or aquatic organisms. However, chloroethane toxicity declines with decreases in chlorination and the 1,1,1-isomer is less active than the 1,1,2-isomer (19). Therefore, 1,1-dichloroethane is probably no more toxic than 1,2-dichloroethane, which has an EPA ambient water quality criteria for the protection of aquatic organisms of 1.18×10^5 µg/l and 2.0×10^4 µg/l for acute and chronic lowest effect levels, respectively. Similarly, the 1,2-dichloroethane EPA ambient water quality criteria for the protection of human health for water and fish consumption and for fish consumption only was 9.4×10^{-1} µg/l and 2.43×10^2 µg/l, respectively, for carcinogenicity protection.

Carcinogenic Effects: A bioassay on 1,1-dichloroethane was limited by poor survival of test animals, but some marginal tumoragenic effects were seen (19). Mice appear to be more sensitive than rats to 1,1-dichloroethane and developed kidney and liver tumors, particularly in males, as well as lung and liver angiosarcomas (16). The tests in rats appeared to be negative. In addition, the closely related 1,1,1-trichloroethane is not carcinogenic (16). Studies on the S9 fractions from mouse and rat liver, particularly from males, showed mutagenic results (16).

Applying the EPA criteria guidelines for assessment of carcinogenic risk (69), 1,2-dichloroethane is classified in Group C: Possible human carcinogen (17). This category is for chemicals with limited evidence of carcinogenicity in animals. A PF of

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9.1×10^{-2} $(mg/kg/day)^{-1}$ was determined from HEA documentation by the oral route (17).

Tetrachloroethene

- Transport and Fate

Due to its relatively high vapor pressure, tetrachloroethene volatilizes rapidly (23). In the ambient atmosphere, it is expected to exist almost entirely in the vapor phase and not partition to atmosphere particulates (19). Significant evaporation from dry surfaces can be expected. Tetrachloroethene has been detected in rainwater throughout the United States, indicating that physical removal by wet deposition is an important environmental fate process (30).

Tetrachloroethene volatilizes rapidly from water, although volatilization rates will vary with temperature, depth, water movement, and associated air temperatures (30). Estimated tetrachloroethene volatilization rates for bodies of water area as follows: pond, 7 days; river, 1.4 days; and lake, 5.6 days (30). Experimental bioconcentration factor (BCF) values in fish range from 39 to 49 (32, 33, 34, 35). Other studies have shown tetrachloroethene to have low bioaccumulation potential (36, 37). Tetrachloroethene can rapidly leach into groundwater, but will not partition significantly from the water column to sediment in natural bodies of water (30, 38, 39). In natural water and soil systems, the most important transformation processes are hydrolysis and biodegradation, although these processes occur slowly (40, 41, 42, 43, 44). The biotransformed product is trichloroethene, which can be reduced further to dichloroethene, and finally to vinyl chloride (20). In surface waters tetrachloroethene is dispersed primarily by volatilization. In the air, tetrachloroethene undergoes photooxidation with atmospheric hydroxyl radicals, producing chloroacetylchlorides and phosgene (half-life approximately 96 days). Long-range global transport is likely (45, 46, 47).

- Health Effects

Noncarcinogenic Effects: Tetrachloroethene is readily absorbed after ingestion or inhalation, but poorly absorbed after dermal contact. At a concentration of 189 mg/kg, single oral doses of tetrachloroethene were absorbed completely when

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given to rats (75). In humans, the compound was initially absorbed rapidly via inhalation (72 or 144 ppm), with decreasing uptake as exposure continued (23). Once in the bloodstream, tetrachloroethene tended to distribute to body fat (23). Human ratio of fat to liver concentrations were greater than 6:1 (76) and fat to blood was about 90:1 (77). The main target organs in humans include the central nervous system (CNS), liver, and kidneys (23).

Short term exposure to mice produced 24-hour LD_{50}s/LC_{50}s of: 8.8 to 10.8 g/kg by the oral route (78); 5,200 ppm with four hours inhalation exposure (79); and 4.7 g/kg intraperitoneal (80). In rats, the 24-hour LD_{50}s/LD_{50}s were 13 g/kg oral (81) and 4,000 ppm with four hours inhalation (82). Pregnant rats exposed to 300 ppm of tetrachloroethene for seven hours/day, on days six through 15 of gestation had four to five percent reduction in body weight and twice the number of resorptions per implantation compared with controls (83). Also, the mice exhibited a significant increase in the mean relative liver weights and fetuses weighed significantly less than controls.

Delayed ossifications of skull bones and sternebrae were reported in offspring of pregnant mice exposed to 2,000 mg/m^3 of tetrachloroethane for seven hours/day on days six to 15 of gestation (49). However, these effects occurred at maternally toxic doses, and may not be significant if they were secondary to maternal toxicity. Increased fetal resorptions were observed after exposure of pregnant rats to tetrachloroethane (17, 48). Renal toxicity and hepatotoxicity have been noted following chronic inhalation exposure of rats to tetrachloroethane levels of 1,356 mg/m^3 (17, 48). During the first two weeks of a subchronic inhalation study, exposure to concentrations of 1,622 ppm (10,867 mg/m^3) of tetrachloroethane produced signs of central nervous system depression, and cholinergic stimulation was observed among rabbits, monkeys, rats, and guinea pigs (17, 19, 48).

A NOAEL of 20 mg/kg/day and a LOAEL of 100 mg/kg/day determined by exposure of mice to tetrachloroethene at doses of 0, 20, 100, 200, 500, 1,500, and 2,000 mg/kg, five days/week for six weeks were the basis for the development of an oral RfD. An oral RfD of 1 x 10^{-2} mg/kg/day was determined by multiplying the NOAEL by 5/7 (factor to convert to daily exposure) and dividing by an uncertainty factor of 1,000 and a modifying factor of one. For the uncertainty factor, a factor of 10 was chosen to account for intraspecies variability, interspecies variability, and extrapolation of a

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subchronic effect level to its chronic equivalent (23). The confidence in the oral RfD was low for the study (lack of complete histopathological examination at the NOAEL in the mouse study), medium for the database, and medium for the RfD (17).

A MCLG of 0 µg/l was proposed in 1984 (17). The EPA ambient water quality criteria for protection of human health for water and fish consumption and for fish consumption only was 8.0×10^{-1} and 8.85 µg/l, respectively (17). The EPA ambient water quality criteria for the protection of aquatic organisms had an acute lowest effect level of 5.8×10^{3} µg/l and a chronic lowest effect level of 8.4×10^{2} µg/l (17).

Carcinogenic Effects: Studies on tetrachloroethene employing the Ames Salmonella/ microsome test or modifications of this test revealed little or no evidence of mutagenic activity except at concentrations which result in greater than 90 percent bacterial toxicity (30). Tetrachloroethene was concluded to be a liver carcinogen in mice administered 386 to 1,072 mg/kg by gavage for 78 weeks (23). However, no conclusion concerning the effects on rats administered 471 to 949 mg/kg by gavage could be made because of high mortality rates (23). In inhalation bioassays, rats and mice of both sexes were exposed to tetrachloroethene (86). Male rats exhibited a significant increase in incidence of mononuclear cell leukemia and an increased incidence of renal tubular adenomas/carcinomas. Both sexes of mice had induced hepatocellular carcinomas. The relevance of mononuclear leukemia to man, a species not susceptible to this type of leukemia, has been questioned (85).

The evaluation of tetrachloroethene is under review by an inter-office EPA work group (17). The Carcinogen Assessment Group classified tetrachloroethene in Group B2: Probable human carcinogen (86). This category is for chemicals with sufficient evidence of carcinogenicity in animals but inadequate evidence of carcinogenicity in humans. However, this classification has been questioned by the Science Advisory Board, Halogenated Organics Subcommittee, which has recommended a classification of Group C: Possible human carcinogen (85). This category is for chemicals with limited evidence of carcinogenicity in animals.

The oral potency factor and inhalation potency factor for the current weight of evidence classification of B2 were 5.1×10^{-2} $(mg/kg/day)^{-1}$ and 1.7×10^{-3} $(mg/kg/day)^{-1}$, respectively, with the source identified as the HEA (14).

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1,1-Dichloroethene

- Transport and Fate

Volatilization from surface soils and aquatic systems to the atmosphere is the primary transport mechanism for 1,1-dichloroethene (23). The half-life in surface water is from one to six days (14). Aerial transport, in part, is responsible for its relatively widespread environmental distribution. Because of its low K_{oc} and log K_{ow}, 1,1-dichloroethene can be transported by groundwater (19). It can undergo abiotic or biotic degradation in soil or water to form vinyl chloride (20) and has a bio half-life of 53 days (21). 1,1-Dichloroethene is a degradation product of trichloroethene and tetrachloroethene (23). Bioaccumulation seems to be a relatively insignificant pathway (23). Photooxidation in the atmosphere is the principle environmental fate of 1,1-dichloroethene, with a half-life in air of less than one week (19, 20) and as low as two days (14).

- Health Effects

Noncarcinogenic Effects: 1,1-Dichloroethene is a hepatic toxic, causing both lipid accumulation and necrosis (51). Exposure to ketonic solvents, especially acetone, potentiates hepatic responses to 1,1-dichloroethene (23, 49, 50). Assimilation is complete after gavage with 96 to 100 percent of a single dose reportedly excreted within 72 hours (56).

Inhalation of high concentrations of 1,1-dichloroethene (≥4,000 ppm) resulted in central nervous system depression in humans (57). Reported oral LD_{50}s in adult rats range from 200 to 1,800 mg/kg (58, 59) and in the mouse and the dog were 200 mg/kg and 5,750 mg/kg (58), respectively. These studies and a review of available data indicate that the liver is the most sensitive target organ (end-point-liver damage) and that rats are the most sensitive species. In rats, the liver toxicity followed a complex dose-response pattern, with a threshold level, a rapid increase in effect and then an extended plateau (60). Long-term exposures resulted in similar health effects with the liver the principal target of 1,1-dichloroethen toxicity (23). In a three-generation rat reproductive study, concentrations of 0 to 26 mg/kg in the drinking water did not affect reproductive capacity (61). 1,1-Dichloroethene did not produce teratogenic

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effects in rats or rabbits at levels producing no maternal toxicity, but it was fetotoxic (53, 62). It is also a skin irritant (17, 23).

The drinking water exposure study (51) offers a suitable model for potential human exposure to 1,1-dichloroethene. A LOAEL of 100 ppm (10 mg/kg) was identified based on an abnormal histopathology of minimal mid-zonal hepatocellular fatty change and hepatocellular swelling. An oral RfD of 0.01 mg/kg/day was determined by dividing the LOAEL by an uncertainty factor of 1,000 and a modifying factor of one. For the uncertainty factor, a factor of 10 was used for use of a LOAEL, for interspecies variation, and for protection of sensitive human subpopulations (17). The confidence in the study, database, and RfD are considered medium based on corroborative chronic and subchronic oral bioassays and use of appropriate methodology (17).

A MCL of 7 µg/l (17) was finalized in 1987. The EPA ambient water quality criteria for protection of human health for water and fish consumption and for fish consumption was only 3.3×10^{-2} µg/l and 1.85 µg/l, respectively (17). The EPA ambient water quality criteria for the protection of aquatic organisms was an acute lowest effect level of 1.16×10^{4} µg/l (17). This value was for the class of dichloroethenes, and not specifically for 1,1-dichloroethene.

Carcinogenic Effects: There is conflicting evidence as to the carcinogenicity of 1,1-dichloroethene (23, 26, 51). In part, this could be due to variability in study design. 1,1-Dichloroethene is mutagenic and its chemical structure is similar to a known human carcinogen, vinyl chloride (17, 23). The EPA weight-of-evidence classification is C, a possible human carcinogen (17). The basis for this classification was the observed tumor growth in one mouse strain after inhalation exposure (27). Group C includes chemicals with limited evidence in animals in the absence of human data. In mice, exposure to 1,1-dichloroethene has resulted in kidney adenocarcinoma (male mice), mammary carcinomas (female mice), and pulmonary adenomas (both sexes) (17, 51). However, an epidemiologic study of 138 workers showed no carcinogenic effect associated with 1,1-dichloroethene exposure (63).

The oral potency factor and inhalation potency factor were estimated at 6×10^{-1} $(mg/kg/day)^{-1}$ and 1.2 $(mg/kg/day)^{-1}$, respectively (17). The estimate for the oral slope factor is based on a data set in which there is no significant increase in tumor

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incidence and the confidence is high that the upper limit is not greater than the oral slope factor.

6.3 Exposure Assessment

6.3.1 Introduction

The goal of the Exposure Assessment is to determine of the type and magnitude of potential human population exposure to contaminants present at, and migrating from, the Berks Sand Pit Site. There are currently four residential households that receive water from a Superfund well located upgradient of the former sand and gravel pit. This well was installed as an emergency remedial measure because of the high levels of contaminants found in local private wells. However, for purposes of the Public Health Evaluation, the Exposure Assessment will be conducted on a pre-Superfund well basis to establish no-action, baseline conditions prior to any remedial actions.

To determine if human and environmental exposure to constituents of concern might occur in the absence of remedial action, a comprehensive exposure pathway analysis in which potential exposure pathways were identified and characterized was performed. An exposure pathway is comprised of four necessary elements: 1) a source and mechanism of chemical release, 2) an environmental transport medium, 3) a human and/or environmental exposure point, and 4) a feasible human and/or environmental exposure route at the exposure point. The highest potential human health risks occurred from the exposure to volatile organic chemicals via ingestion of drinking water and inhalation of volatilized organics during showering by receptors on residential wells. The surface waters also presented a risk due to possible ingestion of contamination water. Section 6.3 defines the extent of the potential human health risks.

This section of the report evaluates the potential for completion of an exposure pathway at the Berks Sand Pit Site. The former sand and gravel pit located beneath the R-3 property in Longswamp Township, Berks County, is considered to be a potential source of contamination, although human and/or environmental exposure to contaminants associated with the site may not be solely attributed to releases from the backfilled pit.

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6.3.2 Atmospheric

6.3.2.1 Air Exposure Pathway

There are two potential release sources to be considered in evaluating the atmospheric pathway: release of contaminated particulate and volatilization from surface soil, groundwater and surface water. The release mechanisms to the air which must be considered are fugitive dust generation and volatilization; the transport mechanism is the air. The most important routes for human exposure to the contaminated air is via inhalation. Potential exposure points from the Berks Sand Pit Site are areas of human activity adjacent to the site and residential users of contaminated groundwater for showering and bathing.

6.3.2.2 Air Environmental Contamination

The sand and gravel pit, which reportedly received the contaminants, has been backfilled and developed into a residential property. As described in Section 5.2, OVA and breathing zone measurements were near background levels or only slightly elevated. There were no volatile organics detected in surface soils that would contribute to air contamination by volatilization. Air quality samples taken for the health and safety program did not show any elevated readings (see Table 5-3).

The surface water samples did contain volatile organics (Appendix E, Table E-5). Surface water samples SP-1 through SP-8 contained concentrations of 1,1-dichloroethene (DCE), 1,1-dichloroethane (DCA), and 1,1,1-trichloroethane (TCA) ranging from a low of 9.3 µg/l to a high of 2,600 µg/l. These surface water samples represent a potential source of contaminant exposure at the Berks Sand Pit Site by the air pathway. However, it is likely that the small surface area of the contaminated surface waters precludes any significant discharges of volatile contaminants to the ambient air.

The groundwater samples also contained volatile organics (Appendix E, Table E-3 and E-4). (Note: A summary of maximum, minimum, and average concentrations found for all chemicals in the residential and monitoring wells are given in Table 6-9.) These groundwater samples represent the main potential source of contaminant exposure at the Berks Sand Pit Site by the air pathway. Estimates of the expected indoor air concentrations of the volatile organic chemicals found in the groundwater were made using a mod[illegible] described in Appendix F. The objective of the model was to provide the worst case shower [illegible]

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concentrations of the volatile chemicals found in the groundwater well samples. Table 6-8 gives the results of the modelling effort. The model assumed that four people would take consecutive 15-minute showers. The model was run using the residential well results and a combination of residential and monitoring well results. The combined results and the justification for using the combined results can be found in Table 6-15 and Section 6.4.4.2 - Groundwater Dose Evaluation. The highest shower-area concentrations occurred using groundwater well data, with the concentration being 0.2183 mg/l for 1,1,1-trichloroethane. The residential well highest concentration was 0.2033 mg/l, also for 1,1,1-trichloroethane.

6.3.2.3 Air Exposed Population

The potentially exposed population from the air pathway includes the residents of the approximately 27 residential properties in the vicinity of the former sand and gravel pit, and any residents of areas more distant from the site (e.g., Mountain Village Trailer Park) that come in contact with contaminated air originating from the seeps, springs, and creeks. This would especially include small children living in the area and playing in and around the aforementioned surface waters. Also included in the potential exposed population are residential users of the contaminated groundwater for showering and bathing.

6.3.2 Soil

6.3.3.1 Soil Exposure Pathway

There are two potential release sources to be considered in the soil pathway - the contents of the sand and gravel pit, and contaminated soils. The release mechanisms to be considered are fugitive dust generation/deposition, tracking, surface runoff, and leaching; the transport medium is the surface and subsurface soils and surface water sediments. The most important routes for human exposure to the contaminated soils and sediments by the soil pathway are via ingestion, inhalation, and dermal contact. Potential exposure points from the Berks Sand Pit Site are areas of human activity adjacent to the site. Fugitive dust generation/deposition was discussed in the air exposure pathway. Site leaching is the most significant release mechanism and will be discussed in the groundwater section of the report, Section 6.3.4.

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Table 6-8

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SUMMARY OF 90-MINUTE SHOWER AIR CONCENTRATION AVERAGES USING DATA FROM GROUNDWATER WELLS

Name	Maximum ($\mu g/m^3$)	Average-Total ($\mu g/m^3$)	Average-Positives ($\mu g/m^3$)
Residential Wells			
1,1-Dichloroethene	0.018	0.0006	0.0063
1,1,1-Trichloroethane	0.2033	0.0085	0.0212
Groundwater Wells			
1,1-Dichloroethene	0.1170	0.0054	0.0178
1,1,1-Trichloroethane	0.2183	0.0186	0.0367
Tetrachloroethene	0.0007	0.0000	0.0004

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6.3.3.2 Soil Environmental Contamination

Soil and sediment samples were obtained from six monitoring wells, 17 test borings and 13 surface water sampling points (Appendix E, Tables E-1 and E-2). The sample locations are shown on Drawing 1.

The sand and gravel pit, which reportedly received the contaminants, has been backfilled and developed into a residential property. There were no volatile organics found in the surface sediments that could potentially be absorbed via dermal contact through tracking or transported by surface runoff.

The surface water sediment samples had one detected concentration of a volatile organic that could potentially adversely impact human health by dermal contact and ingestion. At SP-2, 1,1-dichloroethane had a concentration of 240 mg/kg. The sampling point SP-2 was a seep located northeast of the former sand pit in the general area of the headwaters of the West Branch of Perkiomen Creek.

6.3.3.3 Soil Exposure Population

The potentially exposed population from the soil exposure pathway includes adults and small children from the approximately 27 residential homes in the vicinity of the former sand and gravel pit and from the Mountain Village Trailer Park located approximately one mile from the site. Small children visiting or living in the site may play in the surface water seep, SP-2, and could potentially intake contaminants via the routes of ingestion and dermal contact.

Also included in the potential exposed population from the soil exposure pathway are users of groundwater contaminated by site leaching, which will be discussed in the groundwater section, and human receptors via inhalation, which was discussed in the atmospheric section.

6.3.4 Groundwater

6.3.4.1 Groundwater Exposure Pathway

There are two potential release sources to be considered in evaluating the groundwater pathway - the contaminants placed in the sand and gravel pit and contaminated soils. The release mechanism to be considered is site leaching and the transport medium is the

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groundwater in the soil overburden and bedrock aquifers. The most important routes for human exposure to the contaminated groundwater by the groundwater pathway is via ingestion, inhalation, and dermal contact. Inhalation of the volatile organics will be discussed with regard to the air pathway. Potential exposure points from the Berks Sand Pit Site are household and municipal wells in the area that withdraw contaminated groundwater linked to the hydrology of the sand and gravel pit. Additionally, groundwater discharge to surface water also may represent a pathway for contaminant migration; this transport pathway will be discussed with regard to the surface water pathway.

6.3.4.2 Groundwater Environmental Contamination

Groundwater samples were obtained from 19 residential wells and 18 monitoring wells. The results are given in Appendix E, Tables E-3 and E-4, respectively. The sampling locations are shown on Drawing 1.

A summary of maximum, minimum, and average concentrations found for the volatile organics in the residential and monitoring wells is given in Table 6-9. The maximum concentrations found for DCE and TCA in the residential wells were 540 µg/l and 6,800 µg/l, respectively, in RW-4. The maximum concentrations of DCE and TCA found in the monitoring wells were 3,500 µg/l and 7,300 µg/l, respectively in MW-4. Tetrachloroethene had a maximum concentration of 25 µg/l in MW-7

As discussed in Section 3.0 - Geology, the groundwater in the area of the former sand and gravel pit is encountered in the bedrock and in the soil overburden. The quantity and flow rate of groundwater in the bedrock are dependent on the hydraulic conductivity. The Byram Gneiss is a fractured rock media, therefore, groundwater, for practical purposes, entirely migrates along the avenues of secondary porosity, the interconnected bedrock fractures, rather than by primary porosity, the intercrystalline void space. Groundwater flow rate through the interconnected bedrock fractures can be extremely rapid compared to that in surrounding material.

Groundwater flow and consequent contaminant transport trends towards the northeast, which is consistent with the predominant direction of strike of the joints of the fractured bedrock aquifer. Groundwater elevation contours indicate steeper hydraulic gradients to the northeast, further supporting the preferred groundwater flow in this direction. However, there are also fractures that are oriented in a direction perpendicular to the predominant

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Table 6-9

BERKS SAND PIT
SUMMARY STATISTICS FOR RESIDENTIAL AND MONITORING WELL SAMPLES

Name	Maximum (µg/l)	Minimum (µg/l)	Positives	Average-Total* (µg/l)	Average-Positives* (µg/l)
Volatile Organics					
Residential Wells					
1,1-Dichloroethene	540.00	8.7	3	18.99	189.90
1,1,1-Trichloroethane	6,800.00	6.1	12	282.99	707.48
Monitoring Wells					
1,1-Dichloroethene	3,500.00	41.00	16	290.21	598.56
1,1,1-Trichloroethane	7,300.00	5.00	20	932.36	1,538.39
Tetrachloroethene	25.00	6.40	2	0.96	15.67

* The Average Total is the sum of the concentrations divided by the total number of residential well samples (32) and the Average Positives is the sum of the concentrations divided by the number of positive (>0) residential well samples.

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strike direction. Such fractures would provide a pathway for groundwater flow towards the north and northwest. This could account for the apparent migration of the contaminants in this general direction.

The combined effect of groundwater flow resulting from the predominant bedrock foliation and the perpendicular fractures to the dominant strikes results in a complex pattern of aquifer porosity, permeability, and direction; and, thus, although the regional direction of groundwater flow is considered to be to the northeast, site-specific flow is not well defined. Because of the irregularity in size, shape, and direction of fractures, attempts to model groundwater flow in the Berks Sand Pit Site area produced unreliable results. Contaminated liquids can flow through fractures and cavities largely unimpeded and undiluted by retardation or dispersion processes. The typical three-dimensional dispersion and retardation processes of an isotropic and homogeneous subsurface material for groundwater and leachate travel is disrupted by the fractures. When these conditions exist, the only reliable method of determining concentrations at seeps, springs, and wells is by monitoring. Predictions as to whether a particular point in the aquifer may be affected by plume migration can often only be made through tracer studies.

The transfer and transformation processes that remove hazardous substances from groundwater through transfer to other media or through degradation of the substance include volatilization, hydrolysis, and biodegradation. At the Berks Sand Pit Site, the halogenated aliphatic compounds, specifically the alkanes and alkenes, present the greatest threat to human health and are believed to be the principal hazardous waste disposed of at this site. Generally, the abiotic (i.e., chemical) transformation processes in groundwater, while sometimes significant, is typically slow, whereas biotic processes can be much faster.

The halogenated aliphatic compound with the highest concentration is 1,1,1-trichloroethane (TCA). The half-life of TCA undergoing abiotic transformation processes at 25°C is two years. The products of abiotic transformation of TCA are 1,1-dichloroethene (DCE) by dehydrohalogenation, and acetic acid by hydrolysis. Under methanogenic conditions, the TCA is converted to 1,1-dichloroethane; the kinetics of this biotic transformation is dependent on a sufficient nutrient level and the presence of a microbial population that can effect the transformation.

Concentration contour lines of the two halogenated aliphatic chemicals found in the groundwater, TCA and DCE, are plotted on Drawings 4 through 7. These two chemicals

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represent the greatest threat to public health (see Section 6.4, Risk Characterization). The two plumes extend due east from the former sand and gravel pit, and are very similar in appearance. The centers of both plumes are found at MW-4, representing the highest concentrations found in the groundwater samples. The contours of the plumes indicate that a source of contamination may be located in the vicinity of MW-7 (R-2 and R-3 properties) in addition to the sand and gravel pit. Also indicated by the trace contour lines is the areal extent of contamination. The trace line includes the residential wells along both sides of Benfield Road from RW-2 to RW-8, inclusive, on the eastern side of the road to RW-10, RW-11, and RW-12 on the western side of the road; and the R-9 well on the south side of Walker Road. The plumes appear to follow the general direction of groundwater flow to the east and northeast (see Drawings 4 through 7).

The trend analyses of TCA concentrations from March 1983 to March 1988 (see Section 5.0, Figures 5-1 through 5-11) support the appearance of plume migration in the direction of groundwater flow. The wells in the immediate sand and gravel pit area (RW-3, RW-2, ERT-1, ERT-2, and ERT-3) all show a general decrease in concentrations of TCA with time. Wells further from the sand and gravel pit (RW-6, RW-7, RW-9 and RW-11) all show a general increase in concentrations of TCA with time. Also, the fractured groundwater flow in the area may result in fluctuations in contaminant concentrations atypical of a more isotropic and homogeneous subsurface material.

Supplemental groundwater samples were taken for eight key volatile organics and analyzed with Method 601 (refer to Section 4.0 for sample dates and methodology). This method has a lower detection limit and serves as an indicator of low levels of contamination not observed in the regular sample analyses. Eighteen residential wells and 18 monitoring wells were sampled. The results are presented below but are not used further in the Public Health Evaluation.

6.3.4.2.1 Supplemental Groundwater Analysis

Additional groundwater samples were taken for eight key volatile organics and analyzed with Method 601 (refer to Section 4 for sample dates and methodology). This method has a lower detection limit and will serve as an indicator of low levels of contamination not observed in the regular sample analyses. Eighteen residential wells and 18 monitoring wells were sampled and the results are presented in Tables 6-10 and 6-11, respectively. Table 6-12 gives

AR300201

Table 6-10

BERKS SAND PIT
SUPPLEMENTAL RESIDENTIAL WELL SAMPLE RESULTS FOR METHOD 601
(µg/l)

Residential Well	1,1-Dichloroethene	1,1-Dichloroethane	1,2-Dichloroethane	1,1,1-Trichloroethane	Tetrachloroethene	1,1,2-Trichloroethane
RW-11		1.2		14.0		
RW-2				28.0		
RW-5				3.8		
RW-6				9.9		
RW-10				4.6		
RW-9	11.0			95.0		
RW-4	5.7	2.0		48.0		
RW-7	1.4	1.5		44.0		
RW-13						
RW-1						
RW-15						
RW-8				6.8		
RW-11				5.4		
RW-17						
RW-19						
RW-16						
RW-14						
RW-3	16.0			1,200.0		

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a summary of maximum, minimum, and average values for the chemicals found in the groundwater samples.

The residential wells had two 1,1-dichloroethene (DCE) concentrations that were above the MCL (7 µg/l); Van Elswyck (16.0 µg/l) and Thomas (11.0 µg/l). Only one well, the Van Elswyck's (1,200 µg/l), had 1,1,1-trichloroethane (TCA) concentrations above the MCL (200 µg/l). All monitoring wells that had concentrations of DCE reported were over the MCL. Eleven of the 17 monitoring wells had concentrations of TCA over the MCL.

Method 601 samples were taken at the same time as Round 3 samples were taken for the Public Health Evaluation (PHE). The results were somewhat similar; the TCA concentrations for Van Elswyck's well were 1,200 µg/l and 1,400 µg/l for Method 601 and the PHE data, respectively. However, the Van Elswyck well did not show concentrations of DCE over the MCL in the PHE, but a value of 16.0 µg/l was reported for Method 601 data. Likewise, Thomas' well did not show a problem with DCE in the PHE, but it did have 11.0 µg/l in Method 601 analysis. The monitoring well data showed similar discrepancies.

The inconsistencies between the two groundwater sampling results were probably due to the different methods of analyses, with Method 601 being more suitable for detecting trace amounts of contaminants. Therefore, Method 601 was indicating contamination of DCE in the Van Elswyck and Thomas wells that was not previously noted and which may be indicative of an increasing trend of contamination. This is especially noteworthy given the fact that the Thomas well is presently being used as a potable water source. Also, the results of the monitoring wells indicate the presence of other contaminants not identified in the PHE (1,2-dichloroethane and 1,1,2-trichloroethane).

6.3.4.5 Groundwater Exposed Population

The potentially exposed population from the groundwater pathway includes the approximately 27 residential properties with household wells in the vicinity of the former sand and gravel pit, the one well at the Mountain Village Trailer Park located approximately one mile from the site serving approximately 425 residents, and any additional wells used for domestic use that withdraw contaminated groundwater linked to the hydrology of the sand and gravel pit. However, the complex pattern of groundwater flow in the Berks Sand Pit area precludes identification of such additional wells. As previously discussed in the Introduction to the Exposure Assessment section, for purposes of developing a no-action, baseline public

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health evaluation, the potentially exposed population will include those residents now on the Superfund well. Also included in the potentially exposed population from the groundwater exposure pathway are users of groundwater discharged to surface water; these will be discussed with regard to the surface water pathway.

6.3.5 Surface Water

6.3.5.1 Surface Water Exposure Pathway

There are two potential release sources to be considered in evaluating the surface water pathway - contaminated soils and contaminated groundwater. The release mechanisms to be considered are surface runoff and groundwater seepage. The transport medium is the surface waters originating from local seeps and the headwaters of Perkiomen Creek and Swabia Creek. The most important route for human exposures to the contaminated surface waters by the surface water pathway is via dermal contact since surface waters are not known to be used as a potable water supply in this area. Potential exposure points in the Berks Sand Pit area are users (small children) of the seeps and creeks for recreational purposes.

6.3.5.2 Surface Water Environmental Contamination

The Berks Sand Pit Site area is drained by the headwaters of three creeks, the West Branch of Perkiomen Creek, Perkiomen Creek, and Swabia Creek. These creeks are all classified for cold water fishes and trout stocking. Surface water samples were obtained from 13 sampling points located within the drainage basin of the three creeks (Appendix E, Table E-5). Table 6-13 summarizes the maximum, minimum, and average concentrations found for the four organic volatiles in the surface water samples. The maximum concentrations of TCA and DCE found in the surface water samples were 2,600 µg/l and 990 µg/l, respectively, in SP-1. This seep is located along the horizontal axis of the groundwater plume previously discussed in Section 6.3.4.2 - Groundwater - Environmental Contamination. The sampling points within each drainage are discussed in the following paragraphs and shown on Drawing 1.

The discharges from seeps and springs located east and south of the site (sampling points SP-1 to SP-5) contribute to the headwaters of the West Branch of Perkiomen Creek (sampling points SP-6 to SP-8). All of these sampling points, except for SP-6, and SP-8 were included within the 200 µg/l contour line for TCA (see Drawing 4). For DCE (Drawing 5), seep SP-1 was included within the 200 µg/l contour line, and the rest of the sampling points were

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Table 6-11

BERKS SAND PIT
SUPPLEMENTAL MONITORING WELL SAMPLE RESULTS FOR METHOD 601

Monitoring Well	1,1-Dichloroethene	1,1-Dichloroethane	1,2-Dichloroethane	1,1,1-Trichloroethane	Tetrachloroethene	1,1,2-Trichloroethane
ERT-1		2.4				
ERT-2				5.8		
ERT-3	65.0	26.0		700.0		
MW-2	15.0	6.7		170.0		
MW-3				2,300.0		
MW-3	9.1	3.3		80.0		
MW-3	200.0			2,500.0		
MW-4	660.0			5,500.0		
MW-5	16.0	1.3		170.0		
MW-6	42.0	1.9	1.4	580.0	0.52	0.46
MW-7				3,900.0		
MW-8				2.6		
MW-9	170.0			2,200.0		
SW-1	88.0			1,300.0		
SW-2	260.0	40.0		2,300.0		
SW-3	13.0			160.0		
SW-4	39.0			380.0		
SW-5	32.0	1.8		570.0	0.39	
SW-7				7.0		
SW-6				0.54		

AR300205

Table 6-12

BERKS SAND PIT
SUMMARY STATISTICS FOR RESIDENTIAL AND MONITORING WELL SAMPLES
FROM METHOD 601

Name	Maximum	Minimum	Positives	Average-Total	Average-Positives
Residential Wells					
1,1-Dichloroethene	11.0	1.4	4	1.9	8.5
1,1-Dichloroethane	2.0	1.2	3	0.3	1.6
1,1,1-Trichloroethane	1,200.0	3.8	11	81.1	132.7
Monitoring Wells					
1,1-Dichloroethene	660.0	9.1	13	89.4	123.8
1,1-Dichloroethane	40.0	1.3	8	4.6	10.4
1,2-Dichloroethane	1.4	1.4	1	0.1	1.4
1,1,1-Trichloroethane	5,500.0	0.54	17	1,268.1	1,342.7
1,1,2-Trichloroethane	0.52	0.39	2	0.05	0.46
Tetrachloroethene	0.46	0.46	1	0.03	0.46

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6.4 Risk Characterization

6.4.1 Introduction

The risk characterization process involves comparison of projected intakes and acceptable intakes for chronic exposure for non-carcinogens and between calculated risk and target risk for potential carcinogens. Preliminary comparisons are made with ARARs for human health and environmental resources, where applicable.

6.4.2 Air

6.4.2.1 Air - ARAR Comparison

The ambient indoor air concentrations of the volatile organics found in the groundwater do not have ARARs for comparison, therefore, the concentrations will be evaluated in the next section. It is noted that two volatile organics had concentrations above ARARs established for ingestion of groundwater, DCE and TCA, and will be discussed in Section 6.4.4 - Groundwater.

6.4.2.2 Air - Dose Evaluation

The evaluation of human health impacts from chemical concentrations that do not have ARARs established involves converting the concentrations to a daily dose and comparing the calculated daily dose to an acceptable daily dose, acceptable daily intake, or verified reference dose. The CDI for inhalation of contaminated air per shower event was calculated using the equation (13):

$$CDI = \frac{t_e \times I \times C(X) \times F}{BW \times 2.56 \times 10^4 \, days/lifetime}$$

where

t_e = duration of an exposure event, hours/event
I = average inhalation rate of exposed person, m^3/hr
C(X) = contaminant air concentration throughout the exposure period, $\mu g/m^3$
BW = average body weight, kg
F = frequency of exposure event, number/lifetime

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The duration of an exposure event, t_e, is 0.25 hours/shower (13). The average inhalation rate of the exposed person, I, for light activity (attending to personal needs and care) is 1.3 m^3/hour for an average adult and 1.55 m^3/hour for a child (between 6 and 10)(13). The C(x) concentration is the average-positive and maximum concentrations from Table 6-8. The frequency of exposure event, F, is daily (13). The average body weight, BW, for an adult is 70 kg (14) and a child between 6 and 10 is 26.5 kg (15).

Table 6-14 gives the calculated CDI for the volatile organic chemicals found in the groundwater. Also, given in this table are the calculations for the chronic hazard index and the potential carcinogenic risk for the CDIs. A chronic hazard index value for non-carcinogens above unity is indicative of a potential adverse health impact.

A complete risk analysis, incorporating all of the exposure pathways, will be conducted in Section 6.4.6 - Risk Integration.

6.4.3 Soil

6.4.3.1 Soil - ARAR Comparison

There are no ARARs for the chemical concentrations found in sediment samples at Berks Sand Pit Site and the concentrations, therefore, will be evaluated in the next section.

6.4.3.2 Soil - Dose Evaluation

The evaluation of human health impacts from chemical concentrations that do not have ARARs established involves converting the concentrations to a daily dose and comparing the calculated daily dose to an acceptable daily dose, acceptable daily intake, or verified reference dose. The CDI for ingestion of contaminated surface water sediments was calculated using the equation:

$$CDI = \sum_{i=1}^{n} \frac{C_W \times R_i \times D_i}{BW_i \times DL}$$

AR300208

Table 6-14

BERKS SAND PIT
SUMMARY OF INHALATION CHRONIC DAILY INTAKES AND CALCULATED CHRONIC HAZARD INDEX AND POTENTIAL CARCINOGENIC RISKS FOR RESIDENTIAL AND GROUNDWATER WELL WATER

Chemical	Inhalation Chronic Daily Intake (µg/kg/day)							
	Residential				Groundwater			
	Child		Adult		Child		Adult	
	Avg.	Max.	Avg.	Max.	Avg.	Max.	Avg.	Max.
1,1-Dichloroethene	9.2×10^{-5}	2.6×10^{-4}	2.9×10^{-5}	8.4×10^{-5}	2.6×10^{-4}	1.7×10^{-3}	3.2×10^{-4}	5.4×10^{-4}
1,1,1-Trichloroethane	3.1×10^{-4}	3.0×10^{-3}	9.8×10^{-5}	9.4×10^{-4}	3.2×10^{-3}	5.4×10^{-4}	1.7×10^{-4}	1.0×10^{-3}
Tetrachloroethene	-	-	-	-	1.0×10^{-5}	5.8×10^{-6}	1.9×10^{-6}	3.2×10^{-6}

Chemical	Inhalation Hazard Index CDI:AIC							
	Residential				Groundwater			
	Child		Adult		Child		Adult	
	Avg.	Max.	Avg.	Max.	Avg.	Max.	Avg.	Max.
1,1,1-Trichloroethane	5.2×10^{-8}	5.0×10^{-7}	1.6×10^{-8}	1.6×10^{-7}	5.3×10^{-7}	9.0×10^{-8}	2.8×10^{-8}	1.7×10^{-7}

Chemical	Potential Carcinogenic Risk CDI x PF							
	Residential				Groundwater			
	Child		Adult		Child		Adult	
	Avg.	Max.	Avg.	Max.	Avg.	Max.	Avg.	Max.
1,1-Dichloroethene	5.5×10^{-8}	1.6×10^{-7}	1.7×10^{-7}	5.0×10^{-8}	1.6×10^{-7}	1.0×10^{-6}	1.9×10^{-7}	3.2×10^{-7}
Tetrachloroethene	-	-	-	-	5.1×10^{-10}	3.0×10^{-10}	9.7×10^{-11}	1.6×10^{-10}

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where

i = age group index

R_i = rate of ingestion for age group i, kg/day

D_i = total number of days in age group i in which ingestion is assumed to occur, days/lifetime

BW_i = body weight for age group i, kg

DL = days in lifetime i, days/lifetime

C_W = concentration of 1,1-dichloroethane in sediment, mg/kg

The values for each of the variables in the above equation are summarized below:

i (years)	D_i (days)	BW_i (kg)	C_W (mg/kg)	D_i (kg/day)	DL (days)
0 - 1	52	5	240	0	365
1 - 5	208	12	240	1×10^{-4}	1,460
5 - 18	676	38	240	5×10^{-5}	4,745
18 - 70	2,704	70	240	1×10^{-5}	18,980

The calculated CDI for 1,1-dichloroethane in SP-2 for children (i is 0 - 18) was 3.3×10^{-4} mg/kg/day and for adults (i is 18 - 70) was 4.9×10^{-6} mg/kg/day. Using the HEA oral route AIC value of 1.2×10^{-1} mg/kg/day, the chronic hazard index was 2.8×10^{-3} for children and 4.1×10^{-5} for adults. Using a PF of 9.1×10^{-2} $(mg/kg/day)^{-1}$ (HEA), the potential carcinogenic risk was 3.0×10^{-5} for children and 4.5×10^{-7} for adults.

The CDI for dermal contact of contaminated surface water sediments was calculated using the equation: (13)

$$CDI = \sum_{i=1}^{n} \frac{C_W \times A_i \times F \times E \times PA \times D_i}{BW_i \times DL}$$

where

i = age group index

C_W = weight fraction of substance in surface water sediments, unitless

A_i = surface area of exposed skin in age group i, cm^2

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F = flux rate of water across skin, mg/cm^2-hr

E = duration of exposure per day, hr/day

PA = percent of substance absorb, unitless

D_i = total number of days in age group i in which absorption is assumed to occur, days/lifetime

BW = body weight of age group i, kg

DL = days in lifetime i, days/lifetime

The values for each of the variables in the above equation are summarized below:

i (years)	C_W	A_i (cm^2)	F (mg/cm^2-hr)	E (hr/day)	PA	D_i (days)	BW (kg)	DL (days)
0 - 1	2.4 x 10^{-4}	700	0.5	2	0.25	52	5	365
1 - 5	2.4 x 10^{-4}	934	0.5	2	0.25	208	12	1,460
5 - 18	2.4 x 10^{-4}	1,328	0.5	2	0.25	676	38	4,745
18 - 70	2.4 x 10^{-4}	1,840	0.5	2	0.25	2,704	70	18,980

The calculated CDI for 1,1-dichloroethane in SP-2 for children (i is 0 - 18) was 2.2 x 10^{-3} mg/kg/day and for adults (i is 18 - 70) was 2.2 x 10^{-4} mg/kg/day. Using the HEA oral route AIC value (there was no AIC value for absorption) of 1.2 x 10^{-1} mg/kg/day, the chronic hazard index was 1.8 x 10^{-2} for children and 1.8 x 10^{-3} for adults. Using a PF of 9.1 x 10^{-2} (mg/kg/day)$^{-1}$ from HEA, the potential carcinogenic risk was 2.0 x 10^{-4} for children and 2.0 x 10^{-5} for adults.A complete risk analysis, incorporating all of the exposure pathways, will be conducted in Section 6.4.6, Risk Integration.

6.4.4 Groundwater

6.4.4.1 Groundwater - ARAR Comparison

The results of the groundwater sampling program showed two volatile organics that had concentrations above their ARARs, DCE and TCA. There were no ARARs for the remaining chemicals found; these will be evaluated in the following section. The groundwater samples that exceeded the ARARs for DCE and TCA are listed in Table 6-15 and Table 6-16 for the residential wells and monitoring wells, respectively.

AR300211

Table 6-15

BERKS SAND PIT
RESIDENTIAL WELL SAMPLES THAT EXCEEDED ARARs
FOR 1,1-DICHLOROETHENE (DCE) AND
1,1,1-TRICHLOROETHANE (TCA)

	DCE MCL = 7 ug/l	TCA MCL = 200 ug/l
Round 1		
RW-4	540.00	6,800.00
Round 3		
RW-2	8.70	-
RW-3	-	1,400.00
RW-4	21.00	-

AR300212

Table 6-16

BERKS SAND PIT
MONITORING WELL SAMPLES THAT
EXCEEDED ARARs FOR
1,1-DICHLOROETHENE (DCE) AND
1,1,1-TRICHLOROETHANE (TCA)

	DCE MCL = 7 ug/l	TCA MCL = 200 ug/l
Round 1		
ERT-3		2,900
Round 3		
ERT-1	250	
ERT-3	250	
MW-1	48	
MW-2	48B	
MW-3	860	2,200
MW-4	3,500	7,300
MW-5	120B	300
MW-6	340B	940
MW-7	1,300	3,700
MW-9	1,100	3,100
SW-1	850	1,900
SW-2	220	6,500
SW-3	100B	240
SW-4	240B	490B
SW-5	280	600

B = found in blank.

AR300213

6.4.4.2 Groundwater - Dose Evaluation

The evaluation of human health impacts from chemical concentrations that are above ARARs, or if no ARARs exist, involves converting the contaminant concentrations to a daily dose and then comparing these calculated daily doses to acceptable daily doses, acceptable daily intakes, or verified reference doses. The CDI was calculated for the ingestion of contaminated groundwater using the equation (14):

$$CDI = \frac{C_W \times R_I}{BW}$$

where

C_W = concentration in residential well water, µg/l
R_I = rate of ingestion, l/day
BW = body weight, kg

The C_W concentrations will represent the average-positive and maximum concentrations from Table 6-9. The R_I standard value is 2 liters for an adult and 1 liter for a child (14). The BW standard value is 70 kg for an adult and 17 kg for a child (14).

Table 6-17 gives the calculated CDIs for the volatile organic chemicals found in the residential well samples. Table 6-17 showed the calculations for the chronic health index and the potential carcinogenic risk for the CDIs. A chronic health index for non-carcinogens above unity is indicative of a potential adverse health impact.

In addition to calculating CDI values for residential well samples, CDI values were calculated for all groundwater samples. Inclusion of the monitoring well samples in the calculation of the CDI values better identified the levels of contaminants available to potential human receptors located along the path of the plume migration, east of the sand and gravel pit. The residential wells, RW-6, RW-7, RW-9 and RW-11, are showing an increasing trend (see Figures 5-1 through 5-11) in levels of TCA. Although the hydrology of the area precludes modeling to determine future plume migration, the plume concentrations may represent potential exposure point concentrations at the above residential wells in the near future.

The C_W concentrations will represent the average-positive and maximum concentrations from Table 6-18. Table 6-17 gave the calculated CDI values for the volatile organic chemicals

AR300214

Table 6-13

BERKS SAND PIT
SUMMARY STATISTICS FOR SURFACE WATER SAMPLES

Name	Maximum (µg/l)	Minimum (µg/l)	Positives	Average-Total* (µg/l)	Average-Positives* (µg/l)
Volatile Organics					
1,1-Dichloroethene	990.00	17.00	8	51.00	159.38
1,1-Dichloroethane	9.3	9.3	1	0.37	9.3
1,1,1-Trichloroethane	2,600.00	62.00	8	166.64	520.75

* The Average Total is the sum of the concentrations divided by the total number of surface water samples (27) and the Average Positives is the sum of the concentrations divided by the number of positive (>0) surface water samples.

AR300215

included within the trace contour line. Because seeps and springs are a product of the groundwater, they also indicate groundwater concentrations farther east than the monitoring wells are located.

Sampling points SP-9 through SP-12 were located in the Perkiomen Creek area northwest to west of the Berks Sand Pit Site. Sampling point SP-9 was a mine pool measuring roughly 15 feet by 15 feet. Sampling points SP-10 and SP-11 were stream samples, and SP-12 was a spring flowing through a stone culvert. Sampling points SP-9 and SP-10 had trace amounts of TCA and SP-11 had low levels of DCE.

Sampling point SP-13 was located in Swabia Creek northeast of the site, and probably not in the same drainage basin as the Berks Sand Pit Site area. The creek contained TCA and DCE levels that were below the minimum detection levels.

6.3.5.3 Surface Water Exposed Population

The potentially exposed population from the surface water pathway includes small children living in the 27 residential properties in the Berks Sand Pit area and small children living at the Mountain Village Park located approximately one mile from the site.

Small children visiting or living in the site may play in the surface waters and could potentially intake contaminants via the routes of ingestion, dermal contact, and inhalation. The inhalation exposure route was examined in the atmospheric section.

The potentially exposed population from the surface water pathway also includes users of the creeks from the same areas as defined for small children above (local residents and trailer park residents) that use the creeks for fishing and swimming purposes. There is also a potential for the creeks to be used as a source of potable water. The recreational users of the creeks could potentially intake contaminants via ingestion of fish, and through dermal contact. However, because these potential uses would be located outside of the general Berks Sand Pit area (the headwaters sampled were only 1 to 3 feet wide and inches deep), they will not be addressed in subsequent sections.

AR300216

Table 6-17

BERKS SAND PIT
SUMMARY OF ORAL DAILY INTAKES AND CALCULATED CHRONIC HAZARD INDEX AND POTENTIAL CARCINOGENIC RISKS FOR RESIDENTIAL AND MONITORING WELL WATER

Chemical	Oral Chronic Daily Intake (µg/kg/day)							
	Residential				Groundwater			
	Child		Adult		Child		Adult	
	Avg.	Max.	Avg.	Max.	Avg.	Max.	Avg.	Max.
1,1-Dichloroethene	11.2	31.8	5.4	15.4	31.4	205.9	15.3	100
1,1,1-Trichloroethane	41.6	400	20.2	194.3	72.7	429.4	35.1	208.6
Tetrachloroethene	-	-	-	-	0.9	1.5	0.5	0.7

Chemical	Oral Hazard Index CDI:AIC							
	Residential				Groundwater			
	Child		Adult		Child		Adult	
	Avg.	Max.	Avg.	Max.	Avg.	Max.	Avg.	Max.
1,1-Dichloroethene	1.1	3.2	0.5	1.6	3.1	20.6	1.5	10
1,1,1-Trichloroethane	0.5	4.4	0.2	2.2	0.8	4.8	0.4	2.3
Tetrachloroethene	-	-	-	-	0.1	0.2	0.1	0.1

Chemical	Oral Potential Carcinogenic Risk CDI x PF							
	Residential				Groundwater			
	Child		Adult		Child		Adult	
	Avg.	Max.	Avg.	Max.	Avg.	Max.	Avg.	Max.
1,1-Dichloroethene	6.7×10^{-3}	1.9×10^{-2}	3.2×10^{-3}	9.2×10^{-3}	1.9×10^{-2}	1.2×10^{-1}	9.2×10^{-2}	6×10^{-2}
Tetrachloroethene	-	-	-	-	4.6×10^{-5}	7.7×10^{-5}	2.6×10^{-5}	3.6×10^{-5}

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Table 6-18

BERKS SAND PIT
SUMMARY STATISTICS FOR GROUNDWATER SAMPLES ABOVE MINIMUM DETECTION LEVELS

Name	Maximum (µg/l)	Minimum (µg/l)	Positives	Average-Total* (µg/l)	Average-Positives* (µg/l)
Volatile Organics					
1,1-Dichloroethene	3,500.00	8.70	19	161.06	534.04
1,1,1-Trichloroethane	7,300.00	5.00	32	623.14	1,226.81
Tetrachloroethene	25.00	6.40	2	0.50	15.75

* The Average Total is the sum of the concentrations divided by the total number of groundwater samples (84) and the Average Positives is the sum of the concentrations divided by the number of positive (>0) groundwater samples.

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found in the groundwater samples. Table 6-17 showed the calculations for the chronic health index and the potential carcinogens risk for the CDIs. A chronic health index for non-carcinogens above unity is indicative of a potential adverse health impact.

The CDI for dermal contact of residential well water was calculated using the equation (13):

$$CDI = \frac{C_W \times A \times D \times F \times PA \times FL}{BW \times 2.50 \times 10^4 \; days/lifetime}$$

where

C_W = weight fraction of substance in the residential well water, unitless
A = surface area of exposed skin, cm^2
D = duration of each shower/bath event, hr/day
F = flux rate of water across skin, $\mu g/cm^2$-hour
PA = percent of substance absorb, unitless
FL = frequency of exposure events per lifetime, days/lifetime
BW = body weight, kg

The C_W concentration will represent the average-positive and maximum concentrations from Table 6-9. The surface area, A, exposed for an adult is 18,150 square centimeters of skin and for a child is 9,400 square centimeters of skin (15). The total duration, D (submersion in water), is assumed to be 0.25 hours (15 minutes) per day. The flux rate, F, is 5.0×10^2 $\mu g/cm^2$-hour (13). The BW average value for an adult is 70 kg and for a child is 17 kg (15). The percent of substance absorb is 25 percent for volatile organics (13). The frequency of exposure events per lifetime is daily.

Table 6-19 gives the calculated CDI for the volatile organic chemicals found in the residential well samples. Table 6-19 showed the calculations for the chronic health index and the potential carcinogenic risk for the CDIs. A chronic health index for non-carcinogens above unity is indicative of a potential adverse health impact.

In addition to calculating CDI values for residential samples, CDI values were calculated for all groundwater samples. The rationale for inclusion of the monitoring well samples in the calculation of the CDI values was explained above.

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Table 6-19

BERKS SAND PIT
SUMMARY OF DERMAL CONTACT CHRONIC DAILY INTAKES AND CALCULATED CHRONIC HAZARD INDEX AND POTENTIAL CARCINOGENIC RISKS FOR RESIDENTIAL AND GROUNDWATER WELL WATER

Chemical	Dermal Contact Chronic Daily Intakes (µg/kg/day)							
	Residential				Groundwater			
	Child		Adult		Child		Adult	
	Avg.	Max.	Avg.	Max.	Avg.	Max.	Avg.	Max.
1,1-Dichloroethene	3.3×10^{-3}	9.3×10^{-3}	1.6×10^{-3}	4.4×10^{-3}	9.3×10^{-3}	6.1×10^{-2}	4.3×10^{-3}	2.8×10^{-2}
1,1,1-Trichloroethane	1.2×10^{-2}	1.2×10^{-1}	5.8×10^{-3}	5.5×10^{-2}	2.1×10^{-2}	1.3×10^{-1}	1.0×10^{-2}	6.0×10^{-2}
Tetrachloroethene	-	-	-	-	2.8×10^{-4}	4.3×10^{-4}	1.3×10^{-4}	2.0×10^{-4}

Chemical	Dermal Contact Hazard Index CDI:AIC							
	Residential				Groundwater			
	Child		Adult		Child		Adult	
	Avg.	Max.	Avg.	Max.	Avg.	Max.	Avg.	Max.
1,1-Dichloroethene	3.3×10^{-4}	9.3×10^{-4}	1.6×10^{-4}	4.4×10^{-4}	9.3×10^{-4}	6.1×10^{-3}	4.3×10^{-3}	2.8×10^{-3}
1,1,1-Trichloroethane	1.3×10^{-4}	1.3×10^{-3}	6.4×10^{-5}	6.1×10^{-4}	2.3×10^{-4}	1.4×10^{-3}	1.1×10^{-4}	6.7×10^{-4}
Tetrachloroethene	-	-	-	-	2.8×10^{-5}	4.3×10^{-5}	1.3×10^{-5}	2.0×10^{-5}

Chemical	Dermal Contact Potential Carcinogenic Risk CDI x PF							
	Residential				Groundwater			
	Child		Adult		Child		Adult	
	Avg.	Max.	Avg.	Max.	Avg.	Max.	Avg.	Max.
1,1-Dichloroethene	2×10^{-6}	5.6×10^{-6}	9.6×10^{-7}	2.6×10^{-6}	5.6×10^{-6}	3.6×10^{-5}	2.6×10^{-6}	1.7×10^{-5}
1,1,1-Trichloroethane	-	-	-	-	1.4×10^{-8}	2.2×10^{-8}	6.6×10^{-9}	1.0×10^{-8}

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The CW concentrations will represent the average-positive and maximum concentrations from Table 6-18. Table 6-19 gave the calculated CDI values for the indicator found in the groundwater samples. Table 6-19 showed the calculations for the chronic health index and the potential carcinogenic risk for the CDIs. A chronic health index for non-carcinogens above unity is indicative of a potential adverse health impact.

A complete risk analyses incorporating all the exposure pathways will be conducted in Section 6.4.6 - Risk Integration.

6.4.5 Surface Water

6.4.5.1 Surface Water - ARAR Comparison

The chemical concentrations of the surface water sampling program were compared to two separate types of ARARs: drinking water standards (MCLs) for the protection of human health, and surface water quality standards and criteria for the protection of aquatic life. There currently does not exist environmental standards for wetland type areas such as the seeps and springs in the Berks Sand Pit Site area. However, application of surface water quality standards from Section 6.2.2 helps define the extent of environmental contamination. The sampling program did not extend far enough downstream of the surface water systems to estimate relative contaminant concentrations in Perkiomen Creek.

The results of the surface water sampling program showed two volatile organics (TCA and DCE) that had concentrations above their ARARs for the protection of human health. There were no ARARs for the remaining volatile organic concentrations found, and these will be evaluated in Section 6.4.5.2 - Surface Water Dose Evaluation. The surface water samples that did exceed the ARARs for the volatile organics are listed in Table 6-20. Except for SP-11, all the samples were obtained from the seeps, springs, and streams to the east of the site and contributing to the headwaters of the West Branch of Perkiomen Creek.

The results of the surface water sampling program showed that no samples had concentrations above the ARARs for the protection of aquatic life.

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Table 6-20

BERKS SAND PIT
RESULTS OF SURFACE WATER SAMPLES FOR TWO VOLATILE ORGANICS THAT EXCEEDED ARARs FOR THE PROTECTION OF HUMAN HEALTH*

	1,1-Dichloroethene	1,1,1-Trichloroethane
MCL	7	200
Round 1		
SP-3	19	
SP-4	38	
SP-7	17	
Round 2		
SP-1	990	2,600
SP-2	84	260
SP-3	41	330
SP-4	66	240
SP-5	9.3	490
SP-11	20	

*Concentrations in µg/l.

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6.4.5.2 Surface Water Dose Evaluation

The evaluation of human health impacts from chemical concentrations that are above the ARARs, of if no ARARs exist, involves converting the concentrations to a daily dose and comparing the calculated daily dose to an acceptable daily dose, acceptable daily intake, or verified reference dose. The CDI for ingestion of contaminated surface water was calculated using the equation (14):

$$CDI = \frac{C_W \times R_I \times D}{BW \times DL}$$

where

C_W = concentration in surface water, µg/l
R_I = rate of ingestion, l/day
D = total number of days in which ingestion is assumed to occur, days/lifetime interval
BW = body weight, kg
DL = days in lifetime, days/lifetime interval

The ingestion exposure rate was considered because the springs and seeps are directly linked to the contaminated groundwater and represent the easternmost extension of the plume. Also, the water may be ingested by adults or small children playing in the surface water and at least one spring may be the source of spring water for domestic use, as indicated by the presence of a stone culvert.

The C_W concentrations will represent the average-positive and maximum concentrations from Table 6-13. The R_I standard value is 0.5 liters for an adult and 0.25 liter for a child (14). The BW standard value is 70 kg for an adult and 25 kg for a child (14). The frequency of exposure, D, is two times per week for 26 weeks. The lifetime intervals are 1 - 18 for a child and 18 - 70 for an adult.

Table 6-21 gives the calculated CDI for the volatile organic chemicals found in the surface waters. Table 6-21 showed the calculations for the chronic health index and the potential carcinogens risk for the CDIs. A chronic health index for non-carcinogens above unity is indicative of a potential adverse health impact.

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Table 6-21

BERKS SAND PIT
SUMMARY OF ORAL AND DERMAL CHRONIC DAILY INTAKES AND CALCULATED CHRONIC HAZARD INDEX AND POTENTIAL CARCINOGENIC RISKS FOR SURFACE WATER SAMPLES

Chemical	Chronic Daily Intakes							
	Oral (µg/kg/day)				Dermal Contact (µg/kg/day)			
	Child		Adult		Child		Adult	
	Avg.	Max.	Avg.	Max.	Avg.	Max.	Avg.	Max.
1,1-Dichloroethene	2.3×10^{-1}	1.4	1.6×10^{-1}	1	3×10^{-8}	1.9×10^{-7}	1.4×10^{-8}	8.9×10^{-8}
1,1-Dichloroethane	1.3×10^{-2}	1.3×10^{-2}	9.5×10^{-3}	9.5×10^{-3}	1.7×10^{-9}	1.7×10^{-9}	8.4×10^{-10}	8.4×10^{-10}
1,1,1-Trichloroethane	7.4×10^{-1}	3.7	5.3×10^{-1}	2.6	9.7×10^{-8}	4.9×10^{-7}	4.7×10^{-8}	2.3×10^{-7}

Chemical	Dermal Contact Hazard Index CDI:AIC							
	Oral (µg/kg/day)				Dermal Contanct (µg/kg/day)			
	Child		Adult		Child		Adult	
	Avg.	Max.	Avg.	Max.	Avg.	Max.	Avg.	Max.
1,1-Dichloroethene	2.3×10^{-2}	0.1	1.6×10^{-2}	0.1	3×10^{-9}	1.9×10^{-8}	1.4×10^{-9}	8.9×10^{-8}
1,1-Dichloroethane	1.1×10^{-4}	1.1×10^{-4}	7.9×10^{-5}	7.9×10^{-5}	1.4×10^{-11}	1.4×10^{-11}	7.0×10^{-12}	7.0×10^{-12}
1,1,1-Trichloroethane	7.4×10^{-2}	0.4	5.3×10^{-2}	0.3	4.9×10^{-9}	4.9×10^{-9}	4.7×10^{-9}	2.3×10^{-8}

Chemical	Dermal Contact Potential Carcinogenic Risk CDI x PF							
	Oral (µg/kg/day)				Dermal Contanct (µg/kg/day)			
	Child		Adult		Child		Adult	
	Avg.	Max.	Avg.	Max.	Avg.	Max.	Avg.	Max.
1,1-Dichloroethene	1.4×10^{-4}	8.4×10^{-4}	9.6×10^{-5}	1.7×10^{-3}	5.0×10^{-11}	3.2×10^{-10}	2.3×10^{-11}	1.5×10^{-10}
1,1-Dichloroethane	1.2×10^{-6}	1.2×10^{-6}	8.6×10^{-7}	8.6×10^{-7}	1.5×10^{-13}	1.5×10^{-13}	7.6×10^{-14}	7.6×10^{-14}

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The CDI for dermal contact was calculated using the equation (13):

$$CDI = \frac{C_W \times A \times D \times F \times PA \times DE}{BW \times DL}$$

where

C_W = weight fraction of substance in surface water, unitless
A = surface area of exposed skin, cm^2
D = duration of each recreational event, hr/day
F = flux rate of water across skin, $\mu g/cm^2$ - hour
PA = percent of substance absorbed, unitless
D = total number of days in which exposure is assumed to occur, days/lifetime interval
BW = body weight, kg
DL = days in lifetime, days/lifetime interval

The C_W concentration will represent the average-positive and maximum concentrations from Table 6-13. The surface area, A, exposed for a small child is 1,328 and for an adult is 1,840 square centimeters of skin on both hands and both feet (15). The total duration, D (submersion in water), is assumed to be 2 hours per day. The flux rate, F, is 5.0×10^{-2} $\mu g/cm^2$-hr (13). The BW average value for a child is 25 kg and for an adult is 70 kg. The frequency of exposure, D, is two times per week for 26 weeks. The lifetime intervals are 1 - 18 for a child and 18 - 70 for an adult. The PA is 25 percent (15).

Table 6-21 gives the calculated CDI for the volatile organic chemicals found in the surface waters. Table 6-21 showed the calculations of the chronic health index and the potential carcinogenic risk for the CDIs. A chronic health index for non-carcinogens above unity is indicative of a potential adverse health impact.

The complete risk analyses incorporating all the exposure pathways will be described in Section 6.4.6 - Risk Integration.

6.4.6 Risk Integration

The final step in the Public Health Evaluation is the integration of the Chronic Health Index and Potential Carcinogenic Risk from each of the exposure pathways for all receptor populations. This is a compilation of the results of the previous analyses to total all exposures

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to a given chemical contacted by each receptor category. The average CDI values were used in the integration.

6.4.6.1 Surface Water and Sediment - Child Receptor

Table 6-22 sums the individual chronic health index for the oral and dermal contact exposure routes from surface water by a child receptor. The sum total of all calculated CDI:AIC ratios was 9.9×10^{-2}. A Chronic Hazard Index value above unity indicates a potential chronic adverse health impact. The values for volatile organic chemicals found would not give a chronic hazard index value of 1, indicating that these chemicals are not presenting a potential chronic adverse health impact.

Table 6-22 also sums the individual risks from potential carcinogens for the oral and dermal exposure routes. A value above 1×10^{-6} indicates risk beyond the typically acceptable levels. The two chemicals 1,1-dichloroethene and 1,1-dichloroethane did represent a significant risk, with risks of 1.4×10^{-4} and 2.8 x 10-3, respectively. 1,1-Dichloroethene was a chemical found throughout the Berks Sand Pit Site.

6.4.6.2 Surface Water and Sediment - Adult Receptor

Table 6-23 sums the individual chronic health index values for the oral and dermal contact exposure of the chemicals found in the routes from surface water by an adult receptor. The sum of the CDI:AIC ratio for all calculated CDI:AIC ratios was 7.1×10^{-2}. A chronic health index for non-carcinogens above unity indicates a potential chronic adverse health impact. The 1,1-dichloroethene and 1,1,1-trichloroethane values, when summed, do not indicate a potential chronic adverse health impact.

Table 6-23 also sums the risks from potential carcinogens for each of the organic volatiles found in the oral and dermal contact exposure route from surface water by an adult receptor. The sum of the risk (CDI x PF) for all the chemicals was 3.5×10^{-4}, indicating a value above the permissible levels. The chemical posing the most significant risk was 1,1-dichloroethene. This chemical was directly related to the Berks Sand Pit Site.

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Table 6-22

BERKS SAND PIT
TOTAL CHRONIC HAZARD INDEX AND RISK FROM POTENTIAL CARCINOGENS
FOR SURFACE WATER AND SEDIMENT EXPOSURE POINT FOR
A CHILD RECEPTOR

Chronic Hazard Index

Chemical	Oral			Dermal Contact		
	Sediment	Water	Total	Sediment	Water	Total
Volatile Organics						
1,1-Dichloroethene		2.3×10^{-2}	2.3×10^{-2}		3.0×10^{-9}	3.0×10^{-9}
1,1-Dichloroethane	3.3×10^{-4}	1.1×10^{-4}	4.4×10^{-4}	2.2×10^{-3}	1.4×10^{-11}	2.2×10^{-3}
1,1,1-Trichloroethane		7.4×10^{-2}	7.4×10^{-2}		4.9×10^{-9}	4.9×10^{-9}

Chronic Hazard Index Total = 9.9 x 10-2

Potential Carcinogenic Risk

Chemical	Oral			Dermal Contact		
	Sediment	Water	Total	Sediment	Water	Total
Volatile Organics						
1,1-Dichloroethene		1.4×10^{-4}	1.4×10^{-4}		5.0×10^{-11}	5.0×10^{-11}
1,1-Dichloroethane	2.8×10^{-3}	1.2×10^{-6}	2.8×10^{-3}	2.0×10^{-4}	1.5×10^{-13}	2.0×10^{-4}

Total Potential Carcinogenic Risk = 2.0×10^{-4}

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Table 6-23

BERKS SAND PIT
TOTAL CHRONIC HAZARD INDEX AND RISK FROM POTENTIAL CARCINOGENS
FOR SURFACE WATER AND SEDIMENT EXPOSURE FOR AN ADULT RECEPTOR

Chronic Hazard Index

Chemical	Oral			Dermal Contact		
	Sediment	Water	Total	Sediment	Water	Total
Volatile Organics						
1,1-Dichloroethene	-	1.6×10^{-2}	1.6×10^{-2}	-	1.4×10^{-9}	1.4×10^{-9}
1,1-Dichloroethane	4.1×10^{-4}	7.9×10^{-5}	4.9×10^{-4}	1.8×10^{-3}	7.0×10^{-12}	1.8×10^{-3}
1,1,1-Trichloroethane	-	5.3×10^{-2}	5.3×10^{-2}	-	4.7×10^{-9}	4.7×10^{-9}

Total Chronic Hazard Index 7.1×10^{-2}

Potential Carcinogenic Risk

Chemical	Oral			Dermal Contact		
	Sediment	Water	Total	Sediment	Water	Total
Volatile Organics						
1,1-Dichloroethene	-	9.6×10^{-5}	9.6×10^{-5}	-	2.3×10^{-11}	2.3×10^{-11}
1,1-Dichloroethane	4.5×10^{-7}	8.6×10^{-7}	1.3×10^{-6}	2×10^{-5}	7.6×10^{-14}	2×10^{-5}

Total Potential Carcinogenic Risk 3.5×10^{-4}

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6.4.6.3 Residential Wells - Child and Adult Receptors

Table 6-24 sums the individual chronic health indicies for the inhalation, oral, and dermal contact exposure routes for residential well water for a child and an adult receptor. The sum total of all calculated CDI:AIC ratios was 1.6 for a child and 0.7 for an adult. Values were above 1 and indicated a potential chronic adverse health impact. The primary chemicals contributing to the significant index value were 1,1-dichloroethene and 1,1,1-trichloroethane. The inhalation and dermal contact exposure route were insignificant components of the index. The high values for 1,1-dichloroethene and 1,1,1-trichloroethane were expected, as household wells have exceeded MCLs for these chemicals.

Table 6-24 also sums the individual risks from potential carcinogens for the inhalation, oral, and dermal contact exposure routes for residential well water for a child and an adult receptor. The sum of the risk (CDI x PF) for all the chemicals was 6.7×10^{-3} for a child and 3.2×10^{-3} for an adult, indicating values above the permissible levels. The chemical contributing entirely to the significant risk was 1,1-dichloroethene, directly related to the Berks Sand Pit Site, and found in concentrations above the MCL in residential wells.

6.4.6.4 Groundwater Wells - Child and Adult Receptors

Table 6-25 sums the individual chronic health index for the inhalation, oral, and dermal contact exposure routes from groundwater (residential and monitoring wells) for both child and adult receptors. As previously explained, the inclusion of the monitoring well data in the evaluation was justified due to the migration of the plume towards potential receptors and the fact that monitoring wells are representative of potential receptor point concentrations. However, it is noted that these concentrations do not represent actual exposure point concentrations, but rather potential concentrations.

The sum of the CDI:AIC ratio was 4 for a child and 2 for an adult. Both of these values were above 1, thereby, indicating a potential chronic adverse health impact. The primary chemicals contributing the most to the significant index were 1,1-dichloroethene and 1,1,1-trichloroethane. The inhalation and dermal contact exposure route was an insignificant component of the index. Again, as was the case in the residential wells, the high chronic hazard index was expected due to the exceedance of the MCL by these two chemicals in the residential wells and in monitoring wells.

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Table 6-24

BERKS SAND PIT
TOTAL CHRONIC HAZARD INDEX AND RISK FROM POTENTIAL CARCINOGENS FOR RESIDENTIAL WELL WATER EXPOSURE POINT FOR A CHILD AND AN ADULT RECEPTOR

Chronic Hazard Index

Chemical	Inhalation		Oral		Dermal Contact		Total	
	Child	Adult	Child	Adult	Child	Adult	Child	Adult
Volatile Organics								
1,1-Dichloroethene			1.1	0.5	3.3×10^{-4}	1.6×10^{-4}	1.1	0.5
1,1,1-Trichloroethane	5.2×10^{-8}	1.6×10^{-8}	0.5	0.2	1.3×10^{-4}	6.4×10^{-5}	0.5	0.2
Total Chronic Hazard Index							1.6	0.7

Potential Carcinogenic Risk

Chemical	Inhalation		Oral		Dermal Contact		Total	
	Child	Adult	Child	Adult	Child	Adult	Child	Adult
Volatile Organics								
1,1-Dichloroethene	5.5×10^{-8}	1.7×10^{-8}	6.7×10^{-3}	3.2×10^{-3}	2.0×10^{-6}	9.6×10^{-7}	6.7×10^{-3}	3.2×10^{-3}
Total Potential Carcinogenic Risk							6.7×10^{-3}	3.2×10^{-3}

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Table 6-25

BERKS SAND PIT
TOTAL CHRONIC HAZARD INDEX AND POTENTIAL CARCINOGENIC RISK FOR GROUNDWATER
EXPOSURE POINT FOR A CHILD AND AN ADULT RECEPTOR

Chronic Hazard Index

Chemical	Inhalation		Oral		Dermal Contact		Total	
	Child	Adult	Child	Adult	Child	Adult	Child	Adult
Volatile Organics								
1,1,1-Trichloroethane	2.6×10^{-4}	3.2×10^{-4}	3.1	1.5	9.3×10^{-4}	4.3×10^{-3}	3.1	1.5
						Total Chronic Hazard Index =	4.0	2.0

Potential Carcinogenic Risk

Chemical	Inhalation		Oral		Dermal Contact		Total	
	Child	Adult	Child	Adult	Child	Adult	Child	Adult
Volatile Organics								
1,1-Dichloroethene	1.6×10^{-7}	1.9×10^{-7}	1.9×10^{-2}	9.2×10^{-2}	5.6×10^{-6}	2.6×10^{-6}	1.9×10^{-2}	9.2×10^{-2}
Tetrachloroethene	5.1×10^{0}	9.9×10^{-11}	4.6×10^{-5}	2.6×10^{-5}	1.4×10^{-8}	6.6×10^{-9}	4.6×10^{-5}	2.6×10^{-5}
						Total Potential Carcinogenic Risk	1.9×10^{-2}	9.2×10^{-2}

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Table 6-25 sums the individual risks from potential carcinogens for the inhalation, oral, and dermal contact exposure routes from groundwater (residential and monitoring wells) by a child and an adult receptor. The sum of the risk (CDI x PF) for the volatile organics chemicals was 1.9×10^{-2} for a child and 9.2×10^{-2} for an adult, indicating values above the permissible levels. The chemicals contributing to this significant risk value were 1,1-dichloroethene and 1,1,1-trichloroethane, with the most significant being 1,1-dichloroethene. The inhalation and dermal contact exposure route was an insignificant component of the index.

6.5 Summary

The Berks Sand Pit Site's groundwater has a significant potential adverse health impact on receptor populations as calculated by the chronic health index and the risk from potential carcinogens indices. There were two complete exposure pathways identified: the groundwater exposure pathway via inhalation, ingestion ,and dermal contact by receptors on residential wells, and the surface water/sediment exposure pathway via ingestion and dermal contact.

The air pathway was not seen as a health hazard in regard to the volatilization of organics from the surface waters, from the surface soils or from the groundwater exposure pathway. In addition, the surface soils were not seen as a health hazard from dermal contact or ingestion.

The groundwater exposure pathway had significant chronic health index values and projected risks values above the target risk values for carcinogens. The compounds most responsible for the potential adverse health impacts were 1,1-dichloroethene, 1,1,1-trichloroethane and tetrachloroethene. The residential wells having levels of these two compounds of concern were RW-2, RW-3 and RW-4. The monitoring wells also showed concentrations capable of having a potential adverse health effect. The migration of the plume toward the northeast could bring the elevated concentrations found in the monitoring wells to human receptors, especially the Thomas residential well.

The surface water and sediment exposure pathway had insignificant chronic health index values for non-carcinogens, but had significant projected risks values above the target risk values for carcinogens. The compound found in this pathway, 1,1-dichloroethene had a significant potential adverse health impact. The samples SP-1 through SP-8 are primarily in line with the migrating plume and further define the extent of contamination. These surface

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water and sediments indicate the potential concentrations of contaminants to both receptors using these areas (e.g., small children) and the receptors who are using groundwater in the area.

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