RISK ASSESSMENT AT THE ROUTE 8 LANDFILL FOR AMPHENOL CORPORATION SIDNEY, NEW YORK

21 March 1989

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File No. 301-07-01-01

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INTRODUCTION

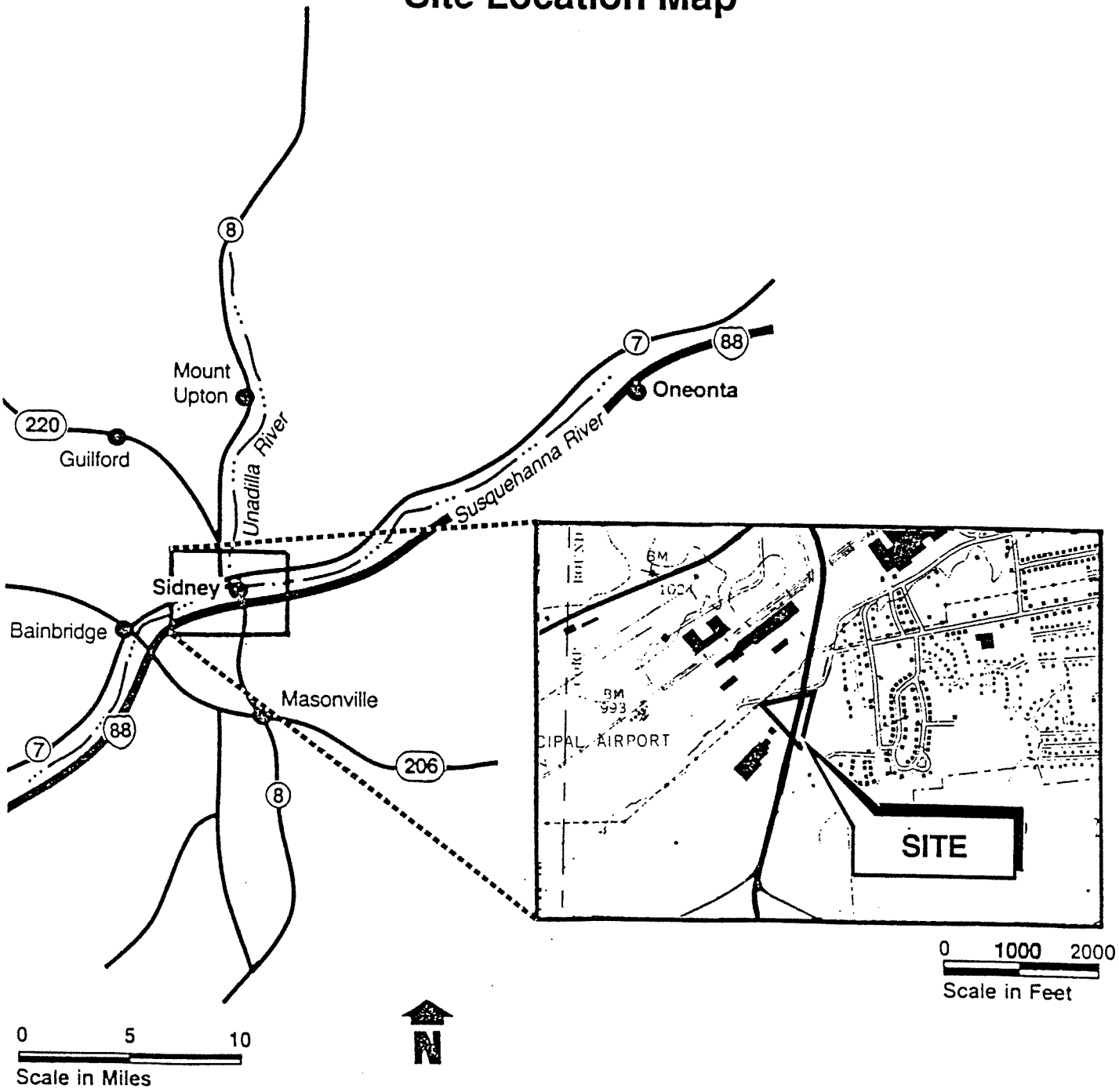
1.1 Background

Environmental Resources Management, Inc. (ERM) has been retained by Amphenol Corporation to conduct remedial investigations and feasibility studies at the former Route 8 Landfill disposal area in Sidney, New York.

This report constitutes the site Risk Assessment (RA). It evaluates the potential for the release of site-related compounds and attendant risk to human populations under conditions existing at the Route 8 Landfill. The Route 8 Landfill facility was used by the Bendix Corporation (now Amphenol) for disposal and burning of solid and liquid wastes generated at the nearby Engine Products and Electrical Components division (now Amphenol Bendix Connectors Operations) plant. The site location is shown in Figure 1-1. The site is located in Delaware County, New York, southwest of the intersection of New York State Route 8 and Gifford Road. The principal wastes disposed at the facility were reported to be plant refuse, including waste connector parts, trash, and waste oils.

In December 1983 Allied-Bendix requested that ERM collect a water sample from a spring near Gifford Road (hereinafter the Gifford Road Spring) for volatile organic compound (VOC) analysis. The results indicated the presence of 229 ppb total VOCs. Additionally, the presence of PCBs was detected in sediment

Figure 1-1
Site Location Map



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samples obtained near the spring. As a result, ERM was retained to conduct an investigation of the Route 8 Landfill area; that investigation was conducted between 1985 and 1987, in three phases, and is reported in Remedial Investigations at the Route 8 Landfill, Volume I, 10 January 1989. The Remedial Investigations of the Route 8 Landfill are summarized in the following subsection.

1.2 Summary of the Remedial Investigations

Aerial photographs of the Route 8 Landfill area from 1960 were examined, and the refuse disposal area, two potential oil disposal areas, and associated roads were identified. Figure 1-2 depicts the 1960 configuration, with an overlay of New York State Route 8, which was built over the landfill area in the early 1970s. The topography of the area, taken from a 1963 New York State Department of Transportation survey, is also shown.

Through a New York Department of Health (DOH) well survey and the data accumulated in ERM studies of other Amphenol sites, the extent of local ground water use was determined, as shown in Figure 1-3. Table 1-1 lists the wells, depths where known, aquifer in use where known, and daily usage where known. The major uses of area ground water are at the Amphenol plant 2,500 feet northeast of the landfill, at the Village of Sidney Well No. 1 which is 5,000 feet northeast of the landfill, and at the Unalam, Inc., industrial cooling water well 800 feet northwest of the landfill. Some limited domestic use is present west of the landfill.

Ground water was sampled at the Unalam Well by the DOH in 1985, and was found to contain a total of 2,933 ppb of total VOCs. Ground water at the Village of Sidney Maintenance Shop Well

Figure 1-2
Route 8 Landfill Activity
April 1960 Air Photograph

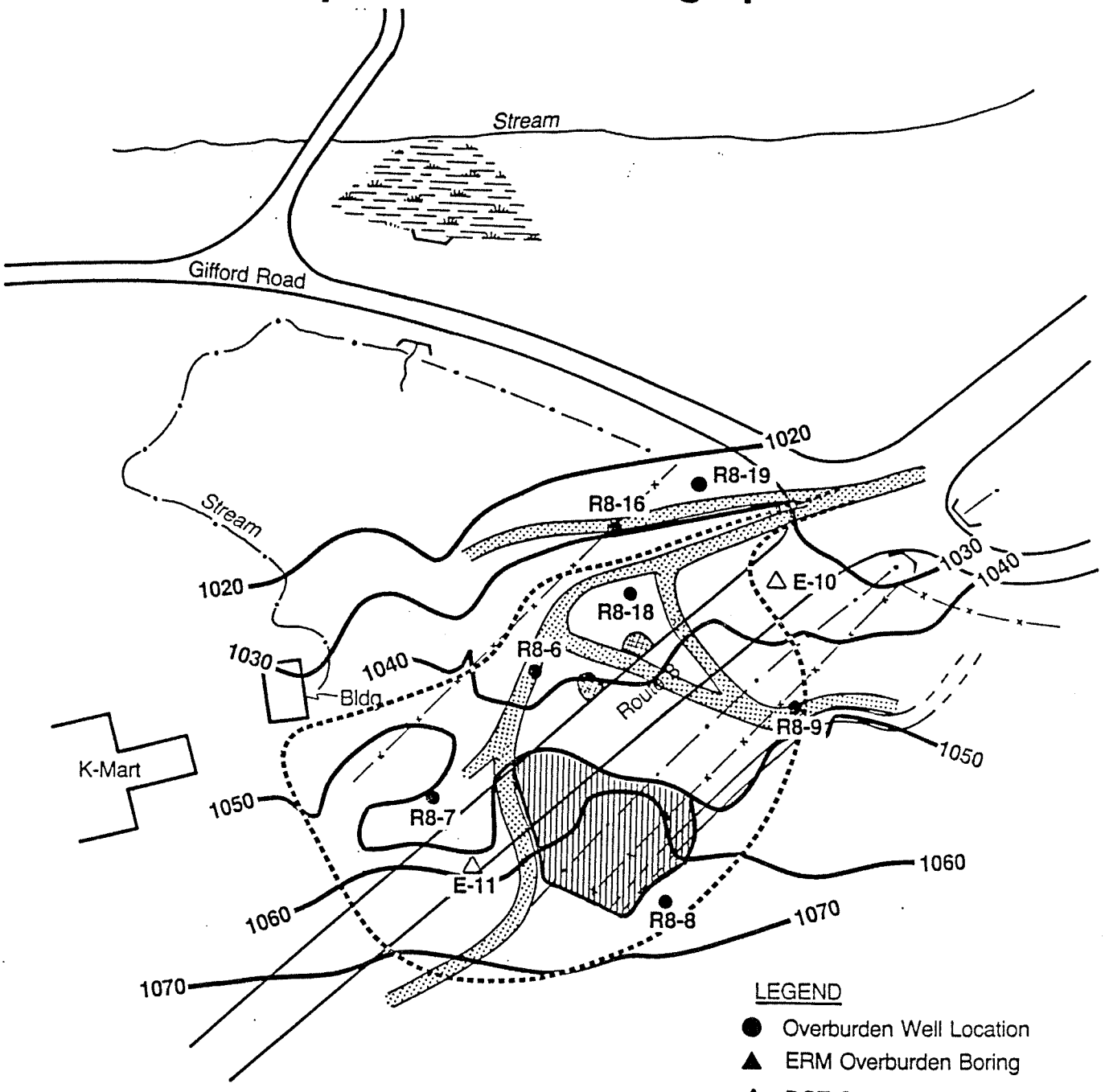

Figure 1-3
Area Ground Water Use and Surface Waters

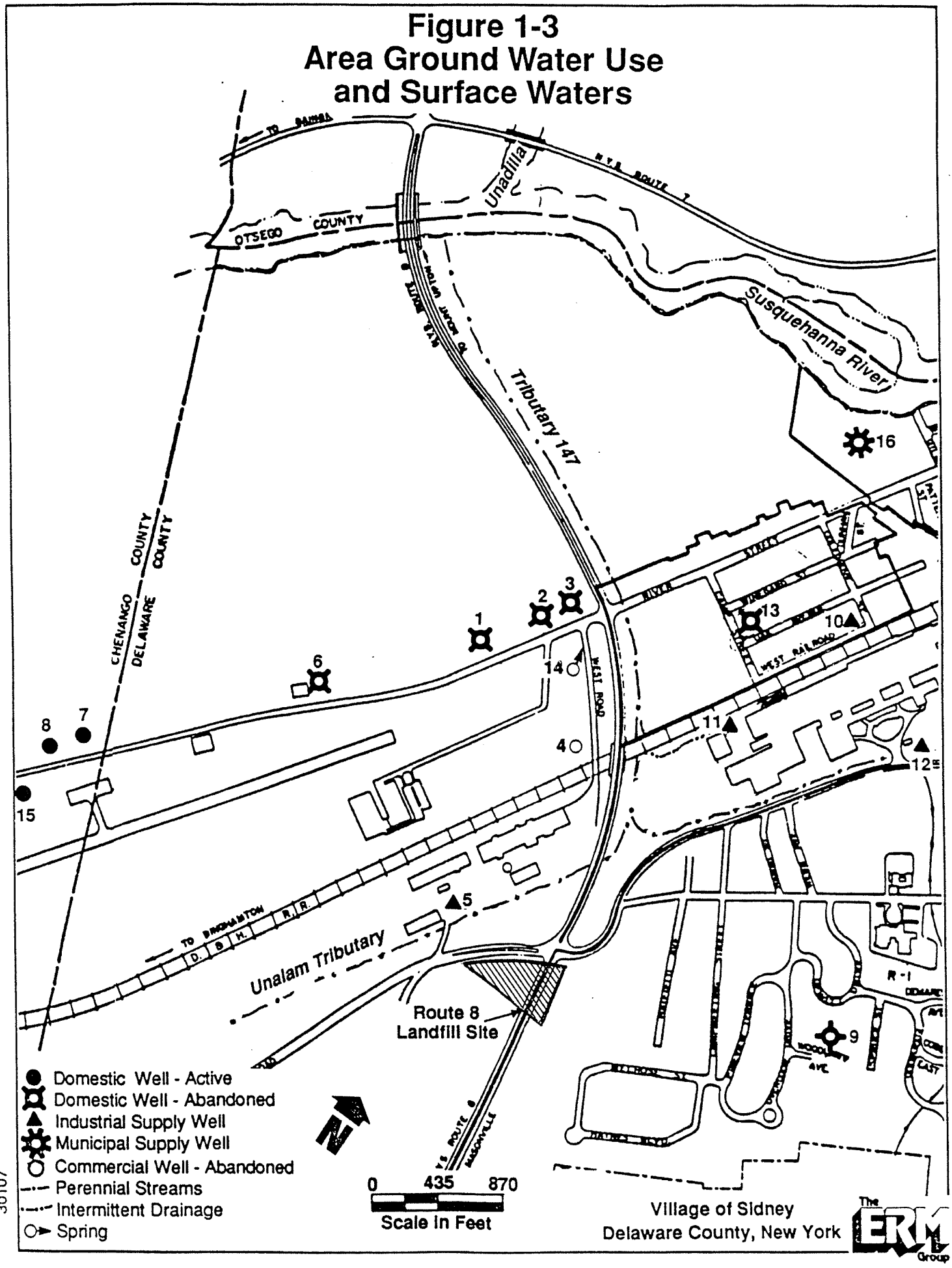

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TABLE 1-1

AREA GROUND WATER USAGE

WELL NO.	TYPE OR NAME	REPORTED DEPTH (FT)	AQUIFER	DAILY USAGE (GPD)
1	Abandoned Domestic	20	Glacial Overburden	0
2	Abandoned Domestic	15	Glacial Overburden	0
3A	Abandoned Domestic	shallow	Glacial Overburden	0
3B	Abandoned Domestic	200	Bedrock*	0
4	Village Maintenance Shop - Not Used	423	Bedrock	0
5	Unalam Products - Cooling Water	235	Bedrock	approx. 1000
6	Abandoned Airport Well	?	?	0
7	Active Domestic	?	?	350**
8	Active Domestic	?	?	350**
9	Inactive Domestic	?	?	?
10	Amphenol North Well - Process Water	155	Bedrock/Overburden	310,000
11	Amphenol West Well - Process Water	150	Bedrock/Overburden	468,000
12	Amphenol South Well - Limited Use	135	Bedrock/Overburden	<1000***
13	Limited Use Domestic	shallow	Glacial Overburden	<50
14	Domestic Spring - not used	surface	Glacial Overburden	
15	Active Domestic	178	Bedrock*	350**
16	Village of Sidney Well No. 1	approx. 200	Glacial Overburden and Bedrock	612,000

* Assumed from depth
** 350 gpd assumed for a family of 4.
*** Estimated - used only for lawn watering

northwest of the landfill was sampled by ERM and found to contain a total of 14 ppb of VOCs in a 1987 analysis. No other wells depicted in Figure 1-3 showed any VOC presence which could be attributed to the Route 8 Landfill.

1.2.1 Field Investigations

A total of 42 monitoring wells and 12 test borings were installed by ERM during the three phases of field investigations. Figure 1-4 shows the locations of the monitoring wells and test borings. A series of rising head slug tests was performed at several wells in order to evaluate the hydraulic conductivities of the overburden and bedrock flow systems. Pumping tests were also conducted to obtain additional information regarding the responses of the overburden and bedrock systems to pumping of the Unalam Well. Ground water samples were collected from the monitoring wells, the Unalam Well, and the Village of Sidney Maintenance Shop Well for VOC, PCBs, oil and grease, and Priority Pollutant metals analyses.

To assess potential PCB migration at the site, selected split spoon samples from the water-bearing zones were collected during monitoring well drilling and submitted for total PCB analysis. In addition, selected soil and waste samples obtained from test borings through the suspected Route 8 Landfill were analyzed for PCBs, oil and grease, and total VOC content. Two of the samples of the landfill material were analyzed for all constituents on the U.S. EPA Hazardous Substance List (HSL) to fully identify all possible waste materials associated with the site.

Surface water and sediment sampling was conducted at the locations indicated on Figure 1-5, for analysis of PCBs, VOCs, and oil and grease. It should be noted that the discharge to

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Figure 1 - 4
Locations of Wells, Borings and Lines Of Geologic Section

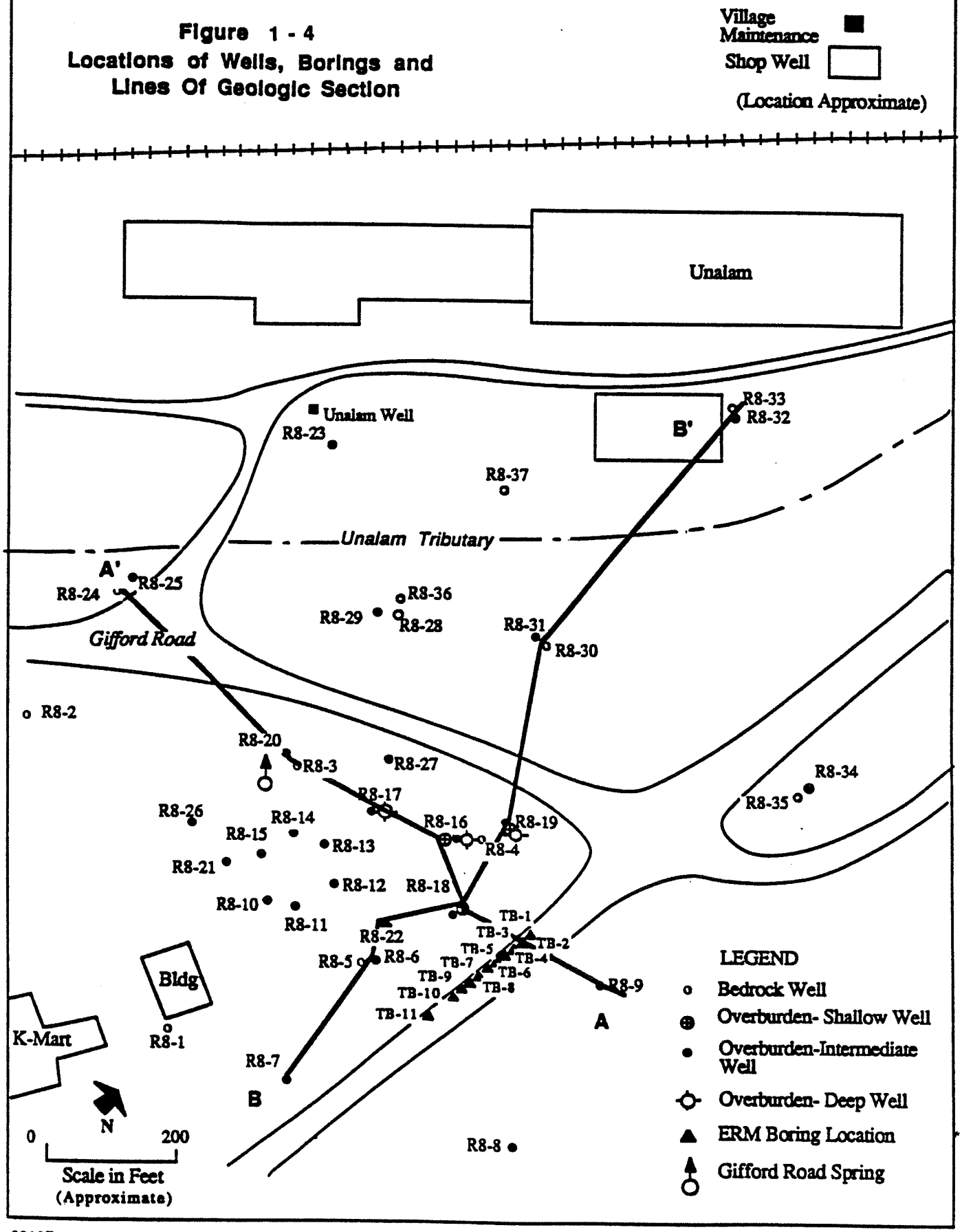

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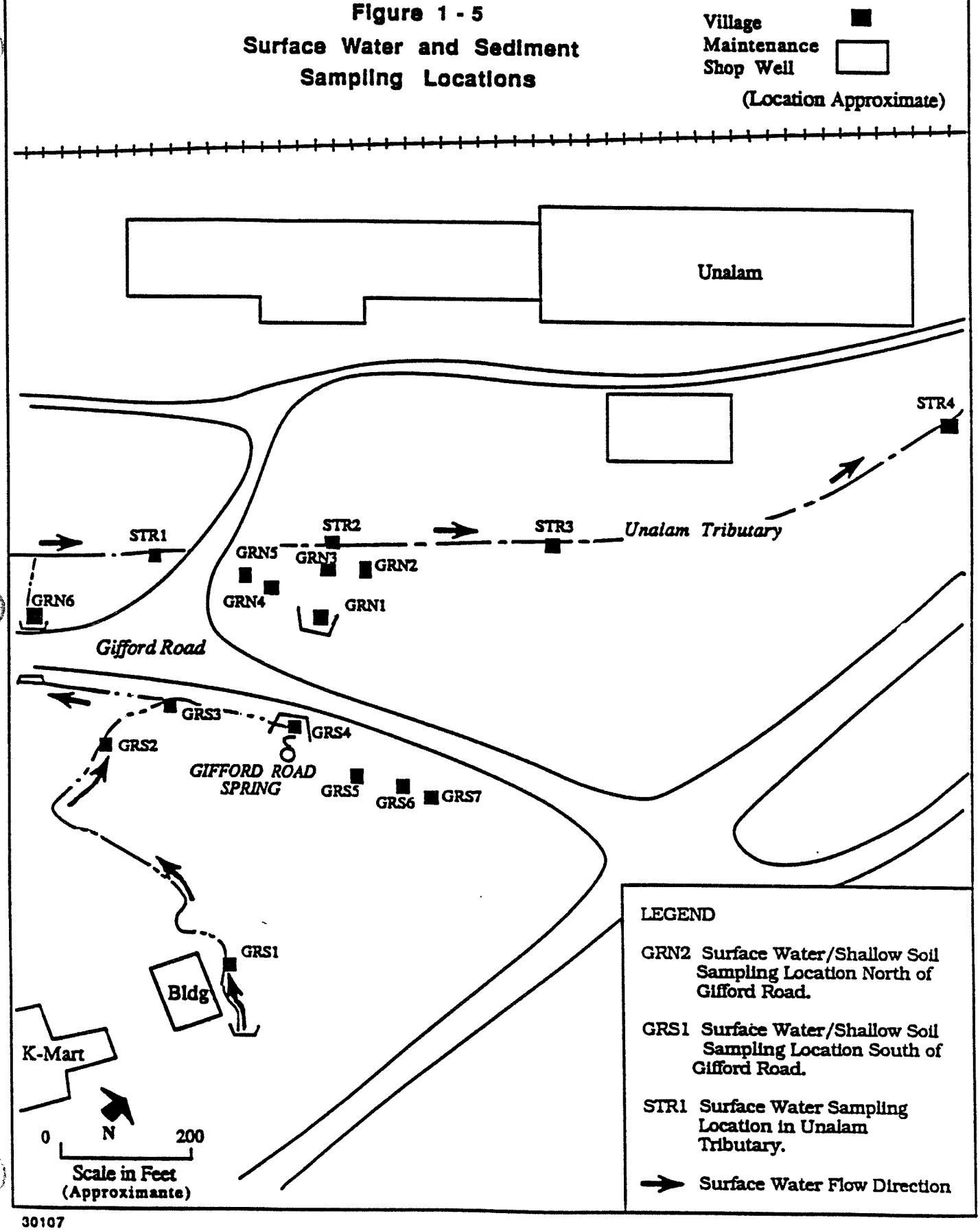
Figure 1 - 5
Surface Water and Sediment Sampling Locations



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stream location GRS1 originates at the K-Mart drain, which conveys drainage water from behind the K-Mart plaza. This water was found in the Hill Site Remedial Investigation (ERM, 1986) to contain approximately 50 to 160 ppb of total VOCs. Subsequent samples collected in November and December 1986 indicated the presence of 84 and 89 ppb total VOCs, respectively. The July 1985 sampling also indicated the presence of 0.0003 ppm of PCBs. By agreement with the New York Department of Environmental Conservation (NYSDEC), the K-Mart Drain is being included for evaluation in the Route 8 Landfill Feasibility Study (FS), due to its proximity to the site.

1.2.2 Results of the Field Investigations

1.2.2.1 Geology/Hydrogeology

Geologic cross-sections of the site have been developed from data collected during the field investigations. Figure 1-6 is an east-west geologic cross-section through the Route 8 Landfill, illustrating the geologic conditions influencing the migration of site-related compounds through the subsurface. Figure 1-7 is a south-north geologic cross-section depicting the site geology downgradient from the source areas along the axis of VOC plume migration. The locations of the lines of geologic sections are shown in the location map presented as Figure 1-4.

The site and areal geology is characterized by surficial glacial deposits averaging about 30 feet in thickness, overlying a flat-lying interbedded siltstone unit with some shale and sandstone stringers. The principal glacial units include the following:

TABLE 1-3
WASTE DISPOSAL AREA CHARACTERIZATION RESULTS (PPB)

	BORING LOCATIONS	SAMPLE DATE	PCBs (ug/kg)	OIL (Soxhlet Ext. %)	TOTAL VOLATILES (ug/kg)	BENZENE	CHLORO-BENZENE	TOLUENE	ETHYL-BENZENE	TOTAL XYLENES	TRANS-1,2-DICHLORO-ETHENE	TRICHLORO-ETHENE	1,1-DICHLORO-ETHANE	METHYLENE CHLORIDE	1,1,1-TRICHLORO-ETHANE	2-BUTANONE	ACETONE
Q	TB-1 10-12'	4/20/87	3,880	0.032 B	226			86			140						
Q	TB-3 10-12'	4/20/87	5,960	0.032 B	202			117			85						
Q	TB-4 10-12'	4/21/87	1,820	0.032 B	321			278			43						
	TB-4 12-14'	4/21/87	ND	0.041	---	---	---	---	---	---	---	---	---	---	---	---	---
Q	TB-4 18-20'	4/21/87	3,510	0.187	35			35									
	TB-4 30-32'	4/21/87	2,370	0.859	---	---	---	---	---	---	---	---	---	---	---	---	---
Q	TB-5 10-12'	4/21/87	13,300,000	6.90	361,900			260,000	7,100	82,000	7,100	5,700					
	TB-5 16-18'	4/21/87	9,120	0.209	---	---	---	---	---	---	---	---	---	---	---	---	---
	TB-5 18-20'	4/21/87	1,750	0.066	---	---	---	---	---	---	---	---	---	---	---	---	---
Q	TB-7 12-14'	4/21/87	10,900	0.033 B	954			654			160	140		33 B			
	TB-7 14-16'	4/21/87	4,630	0.055	---	---	---	---	---	---	---	---	---	---	---	---	---
Q	TB-8 10-12'	4/22/87	46,700	0.086	118.6		8.6	110 J									21 B
Q	TB-9 12-14'	4/22/87	33,400	0.126	4,630			2100 J		140	1,400	990 J				1870 B	640 B
	TB-9 16-18'	4/22/87	1,500	0.086	---	---	---	---	---	---	---	---	---	---	---	---	---
Q	TB-9 22-24'	4/22/87	26,300	0.434	8,513	379	22	5,010	2,670		22				410		
	TB-9 28-30'	4/22/87	1,290	0.496	---	---	---	---	---	---	---	---	---	---	---	---	---
Q	TB-10 12-14'	4/22/87	ND	0.054 B	7,480		27	703			2400	4200	150	27 B			
	TB-10 18-20'	4/22/87	6,260	0.259	---	---	---	---	---	---	---	---	---	---	---	---	---
Q	TB-11 10-12'	4/22/87	92,700	0.138	1,121			829 J	32 J		140	120					

All of the above results are reported on a dry weight basis.
All of the results are measured in ug/kg, unless otherwise indicated.
All blanks – none detected. ND – none detected.
Dashed lines indicate sample was not analyzed for volatiles.

Qualifier Codes:
B - This result is of questionable qualitative significance since this compound was detected in blank(s) at similar concentrations.
Q - These sampling results have undergone an ERM Quality Assurance Review.
J - This result should be considered a quantitative estimate.

TABLE 1-3
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	BORING LOCATIONS	SAMPLE DATE	PCBs (ug/kg)	OIL (Soxhlet Ext. %)	TOTAL VOLATILES (ug/kg)	BENZENE	CHLORO-BENZENE	TOLUENE	ETHYL-BENZENE	TOTAL XYLENES	TRANS-1,2-DICHLORO-ETHENE	TRICHLORO-ETHENE	1,1-DICHLORO-ETHANE	METHYLENE CHLORIDE	1,1,1-TRICHLORO-ETHANE	2-BUTANONE	ACETONE
Q	TB-1 10-12'	4/20/87	3,880	0.032 B	226			86			140						
Q	TB-3 10-12'	4/20/87	5,960	0.032 B	202			117			85						
Q	TB-4 10-12'	4/21/87	1,820	0.032 B	321			278			43						
	TB-4 12-14'	4/21/87	ND	0.041	---	---	---	---	---	---	---	---	---	---	---	---	---
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	TB-4 30-32'	4/21/87	2,370	0.859	---	---	---	---	---	---	---	---	---	---	---	---	---
Q	TB-5 10-12'	4/21/87	13,300,000	6.90	361,900			260,000	7,100	82,000	7,100	5,700					
	TB-5 16-18'	4/21/87	9,120	0.209	---	---	---	---	---	---	---	---	---	---	---	---	---
	TB-5 18-20'	4/21/87	1,750	0.066	---	---	---	---	---	---	---	---	---	---	---	---	---
Q	TB-7 12-14'	4/21/87	10,900	0.033 B	954			654			180	140		33 B			
	TB-7 14-16'	4/21/87	4,630	0.055	---	---	---	---	---	---	---	---	---	---	---	---	---
Q	TB-8 10-12'	4/22/87	46,700	0.086	118.6		8.6	110 J									21 B
Q	TB-9 12-14'	4/22/87	33,400	0.128	4,630			2100 J		140	1,400	990 J				1870 B	640 B
	TB-9 16-18'	4/22/87	1,500	0.086	---	---	---	---	---	---	---	---	---	---	---	---	---
Q	TB-9 22-24'	4/22/87	26,300	0.434	8,513	379	22	5,010	2,670		22				410		
	TB-9 28-30'	4/22/87	1,290	0.496	---	---	---	---	---	---	---	---	---	---	---	---	---
Q	TB-10 12-14'	4/22/87	ND	0.054 B	7,460		27	703			2400	4200	150	27 B			
	TB-10 18-20'	4/22/87	6,260	0.259	---	---	---	---	---	---	---	---	---	---	---	---	---
Q	TB-11 10-12'	4/22/87	92,700	0.138	1,121			829 J	32 J		140	120					

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J - This result should be considered a quantitative estimate.

TABLE 1-4
GROUND WATER ANALYSIS
PCBs / OIL & GREASE/PHENOLS

WELL	PCBs (ppb) DATE Mar-85	PCBs (ppb) DATE Sept. 1985	PCBs (ppb) DATE Apr-87	OIL & GREASE (ppm) DATE Mar-85	OIL & GREASE (ppm) DATE Sep-85	PHENOLS (ppm) DATE Oct-86
R8-1	ND	ND	NA	2	19	NA
R8-2	ND	ND	NA	ND	22	NA
R8-3	ND	ND	NA	1	5	NA
R8-4	ND	ND	NA	ND	26	NA
R8-5	ND	ND	NA	2	8	NA
R8-6	100	180	370	26	26	NA
R8-7	2.6	30	NA	ND	44	NA
R8-8	1.3	ND	NA	ND	6	NA
R8-9	44	NA	3.9	ND		NA
R8-10	2.3	ND	NA	ND	4	NA
R8-11	3.9	10	NA	ND	7	NA
R8-12	0.6	80	21.3	ND	30	NA
R8-13	190	2320	NA	270	268	NA
R8-14	0.9	ND	NA	ND	ND	NA
R8-15	0.2	ND	NA	ND	ND	NA
R8-16 S	6.5	NA	NA	4		NA
R8-16 I	0.6	ND	ND	ND	14	NA
R8-16 D	0.2	ND	ND	ND	ND	NA
R8-17 I	6.1	72	13.2	23	61	NA
R8-17 D	20	4	4.2	750	35	NA
R8-18 S	15	NA	NA	5		NA
R8-18 I	8.9	7	NA	18	10	0.019
R8-19 S	5.6	180	NA	7	617	NA
R8-19 I	2.6	ND	9	11	8	NA
R8-19 D	6.6	ND	ND	52	5	NA
R8-20	0.7	ND	ND	ND	5	NA
R8-21	0.2	ND	NA	3	7	NA
R8-23		ND	ND		14	ND
R8-24		ND	NA		13	NA
R8-25		ND	ND		14	NA
R8-26		ND	ND		ND	NA
R8-27		11	11.4		27	NA
R8-28		ND	NA		5	NA
R8-29		ND	ND		6	ND
R8-30		ND	NA		5	NA
R8-31		ND	ND		5	NA
R8-32			ND			NA
R8-34			ND			NA
R8-35			ND			
R8-36			ND			
R8-37			ND			
Unalam Well	NA	ND	ND	NA	8	ND

ND = none detected. NA = not analyzed. Blank spaces indicate that the well was not installed at that time.

TABLE 1-5
SURFACE WATER RESULTS

VOLATILE ORGANICS (ppb)

Location Designation		Laboratory Designation	Sample Date	Total Volatiles	Toluene	Benzene	Ethyl-benzene	1,1,2-Tri-chloro-ethane	Vinyl Chloride	Chloro-ethane	1,1-Dichloro-ethene	1,1-Dichloro-ethane	Trans1,2-Dichloro-ethene	Chloro-form	1,2-Dichloro-ethane	1,1,1-Trichloro-ethane	Trichloro-ethene	PCBs (ppb)	Oil/Grease (ppm)
GRN1		DW-18	7/19/84	126					2			36	57				31	NA	NA
GRN1		DW2	9/12/85	176			10			24		140			2			3.4	25
GRN1		SW2	4/21/86	393					28			110	210	8		37			NA
GRN2	Q	L3	5/12/87	52						37		15							
GRN3		SW3	4/21/86	42								18	19			5			NA
GRN4	Q	L2	5/12/87	167						8		140	14		1	4			
GRN5	Q	L1	5/12/87	280						47		230			3				3 B
GRN6		SW5	4/21/86	7									2			2	3		NA
GRS1		DW6	9/12/85	109							3	4	19		2	30	51		NA
GRS3		SW4	4/21/86	376	8		56		62			200	28			22			NA
GRS4		DW4	9/12/85	2								2						0.23	26
GRS4		SW1	4/21/86	486					32			146	243	11		54			NA
GRS5	Q	L4	5/12/87	0															3 B
STR1		DW-17	7/19/84	0														NA	NA
STR1		DW1	9/12/85	0															26
STR3		DW-16	7/19/84	0														NA	NA
STR4		DW-15	7/19/84	366				18	8			23	280			22	15	NA	NA
Gifford Spring		Gifford Spring	12/06/83	49								36				13		NA	NA
Gifford Spring		Gifford Spring	12/15/83	299								89	56			154		NA	NA
Gifford Spring		Gifford Spring	5/2/84	0														NA	NA
Gifford Spring		DW-19	7/19/84	145					17			92				36		NA	NA
Gifford Spring		Gifford Spring	3/13/85	183					20			81	34			48		4.2	39

NOTES: Blank spaces = none detected.
NA = Parameter not analyzed.

QUALIFIER CODES:

Q - These samples have undergone an ERM Quality Assurance Review.
B - This result is of questionable qualitative significance since these compounds were detected in blank(s) at similar concentrations.
J - This result should be considered a quantitative estimate.

TABLE 1-6
SEDIMENT ANALYSIS RESULTS

VOLATILE ORGANICS (PPB)

Location Designation		Laboratory Designation *	Sample Date	Total Volatiles	Toluene	Benzene	Ethyl-benzene	1,1,2-Tri-chloro-ethane	Vinyl Chloride	Chloro-ethane	1,1-Dichloro-ethene	1,1-Dichloro-ethane	Trans1,2-Dichloro-ethene	Chloro-form	1,2-Dichloro-ethane	1,1,1-Trichloro-ethane	Trichloro-ethene	PCBs (ppb)	Oil/Grease (ppm)	Moisture % by wt.
GRN1		DS-18	7/19/84															ND	0.02	
GRN1		DS-18A	7/19/84															ND	1.4	
GRN2	Q	Q L3	5/12/87	0	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	6680J	2.4 J	49.1 J
GRN4	Q	Q L2	5/12/87	32	ND	ND	ND	ND	ND	ND	ND	32	ND	ND	ND	ND	ND	ND	0.095 B	37.1
GRN5	Q	Q L1	5/12/87	0	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.181	72.4
GRS1		D5 6A 0"-6"	9/12/85															ND	0.06	
GRS1		D5 6B 10"-16"	9/12/85															ND	0.03	
GRS2		D5 5A 0"-6"	9/12/85															ND	0.02	
GRS2		D5 5B 10"-16"	9/12/85															ND	0.04	
GRS4		D5 4A 0"-6"	9/12/85															ND	0.04	
GRS4		D5 4B 10"-16"	9/12/85															ND	0.06	
GRS6	Q	L5	5/12/87	0	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	0.05 B	0.4
GRS7		D5 3A 0"-6"	9/12/85															ND	0.03	
STR1		DS-17	7/19/84															ND	0.06	
STR1		DS-17A	7/19/84															ND	0.13	
STR3		DS-16	7/19/84															ND	1.4	
STR3		DS-16A	7/19/84															ND	0.73	
STR4		DS-15	7/19/84															ND	ND	
STR4		DS-15A	7/19/84															ND	0.15	
Gifford Spring			1983															2,500	55,000	
Gifford Spring		DS-19	7/19/84															ND	0.67	
Gifford Spring		DS-19A	7/19/84															ND	0.02	

NOTES: ND = none detected.
Blank Spaces = analysis for these compounds was not conducted.
All sediment samples are reported on a dry weight basis with the exception of % moisture.

QUALIFIER CODES:

Q - These samples have undergone an ERM Quality Assurance Review.
B - This result is of questionable qualitative significance since these compounds were detected in blank(s) at similar concentrations.
J - This result should be considered a quantitative estimate.

TABLE 1-7
GROUND WATER ANALYSIS
OVERBURDEN WELLS - VOLATILE ORGANIC RESULTS (PPB)

Overburden Wells	Lab *	Date Sampled	Total Volatiles	Benzene	Toluene	Ethyl-benzene	1,1-dichloro-ethene	1,1-dichloro-ethane	trans-1,2-dichloro-ethene	Chloroform	Trichloro-ethene	Methylene Chloride	Vinyl Chloride	1,1,1-Trichloro-ethane	1,2-Dichloro-ethane	Chloroethene	Tetrachloro-ethene	Dibromochloro-methane	Carbon Tetrachloride
R8-6	F	4/18/84	117						88		8	21							
	O	3/13/85	60		11				33		16								
	L	9/12/85	16		3			4	5		3								
	L	4/2/87	18		3			3	5		5								
R8-7	F	4/18/84	ND																
	O	3/13/85	39						39										
	L	9/12/85	1						1										
R8-8	O	3/13/85	39						39										
	L	9/12/85	5						1	2	2								
R8-9	O	3/13/85	39						39										
	L	9/17/85	300					20	280										
	L	4/2/87	5						2		3								
R8-10	O	3/13/85	5,970		1,200			160	2,200		1,100		440	870					
	L	9/17/85	4,240		1,350			150	1,800				280	660					
R8-11	O	3/13/85	1,005		22			130	430		13		220	190					
	L	9/16/85	4,310		1,250	20		410	1,500				550	560	20				
R8-12	O	3/13/85	590					110	210				160	110					
	L	9/16/85	520					250	60				120	80					
	L	4/2/87	371					190	12				140	27	2				
R8-13	O	3/13/85	319		78	110		53	67				11						
	L	9/16/85	594		27			220	97				170	80					
R8-14	O	3/13/85	88					57	31										
	L	9/13/85	900					340	210				290	60					
R8-15	O	3/13/85	101					47	28					26					
	L	9/13/85	1,514		67			330	650		2		320	140	5				
R8-16S	O	3/12/85	26,440		14,000	290		410	9,700				440	1,600					
		10/16/85						400	2,700					1,200					
R8-16 I	O	3/13/85	58,770		26,000	1,600	140	930	26,000				1,300	2,900					
	L	9/18/85	80,400		33,800	3,300		1,300	36,000				2,100	3,900					
	L	4/2/87	47,600		19,700	800		900	21,000	700			1,900	2,600					
R8-16 D	O	3/12/85	49,700		16,000	1,400	350	1,300	23,000		4,700	670	880	1,400					
	L	9/18/85	64,700		11,500	2,600		1,500	34,000		1,800		1,500	1,800					
	L	4/2/87	36,100		8,900	1,000		1,000	19,000	600	100		3,500	2,000					
R8-17 I	O	3/12/85	35,290		17,000	770		620	12,000				2,500	2,400					
	L	9/17/85	40,200		14,400	1,100		900	19,000				2,200	2,600					
	L	4/2/87	33,200		17,900	1,000		1,000	7,000	300			3,900	2,100					
R8-17 D	O	3/12/85	11,730		2,100	960		520	4,700				3,100	350					
	L	9/17/85	17,200		1,300	400		900	7,900				6,300	400					
	L	4/2/87	10,700		700	300		1,300	2,800	200			5,100	300					
R8-18S (A)	O	3/12/85	763					15	180		470			98					
R8-18I (A)	O	3/12/85	67,910		33,000	1,500	210	800	26,000		450		650	5,300					
	L	9/18/85	58,800		32,000	1,000	.	1,000	18,000				1,200	5,600					

* Lab F - Friend Laboratory, Inc.; Waverly, NY
O - O'Brien & Gere Engineers, Inc.; Syracuse, NY
L - Lancaster Laboratories, Inc.; Lancaster, PA
H - NY Department of Health

(A) - These samples were tested for additional HSL parameters.

TABLE 1-7 (continued)
GROUND WATER ANALYSIS
OVERBURDEN WELLS - VOLATILE ORGANIC RESULTS (PPB)

Overburden Wells	Lab *	Date Sampled	Total Volatiles	Benzene	Toluene	Ethyl-benzene	1,1-dichloro-ethene	1,1-dichloro-ethene	Trans-1,2-dichloro-ethene	Chloroform	Trichloro-ethene	Methylene Chloride	Vinyl Chloride	1,1,1-Trichloro-ethane	1,2-Dichloro-ethane	Chloroethane	Tetrachloro-ethene	Dibromochloro-methane	Carbon Tetrachloride
R8-19S	O	3/12/85	28,570		15,000	430		370	11,000		470		200	1,100					
	L	9/18/85	61,500		35,000	6,000		800	17,000		400	400	1,200	1,700					
R8-19 I	O	3/12/85	24,200		9,400	1,100		500	12,000		180		330	690					
	L	9/18/85	25,400		11,600	500		600	11,000				800	900					
	L	4/2/87	14,286	3	2,500	2	26	500	8,600		19		1,700	900	33		3		
R8-19 D	O	3/12/85	39,100		14,000	1,500	690	880	20,000		1,900		130						
	L	9/18/85	51,900		9,300	1,200	400	1,200	27,000		11,000	900		300	600				
	L	4/2/87	22,780		2,400		40	800	17,000	520	70		1,200	680	70				
R8-20	O	3/12/85	1,270		92	18		320	47				700	93					
	L	9/18/87	1,120					490	50				530	50					
	L	4/2/87	589		2			470	13	2			88	10	4				
R8-21	O	3/12/85	2,541		420			72	1,300	39			220	490					
	L	9/18/87	2,090		400			80	960				240	410					
R8-23	L	9/12/85	5					3	2										
	L	4/2/87	230					76	37		2		110	4	1				
R8-25	L	9/12/85	342					96	170		21		44	3	3	5			
	L	4/2/87	387					210	120		26		31						
R8-26	L	9/18/85	180		1	2		18	74		12		47	25	1				
	L	4/2/87	227			3		32	70		9		86	26	1				
R8-27	L	9/18/85	5,242	6	1,250	136	31	770	1,100		33	2	1,800	110			4		
	L	4/2/87	16,900		4,100	4,900		700	2,000	100			4,100	1,000					
R8-29	L	9/18/85	11,400					1,200	5,100		600		4,200	300					
	L	4/2/87	8,422	8	13		19	1,300	3,800	84	220	2	2,700	220	29	27			
R8-31	L	9/18/85	37,200					1,200	24,000		200		11,000	800					
	L	4/2/87	23,868	9	2		46	1,000	13,000		75	12	9,000	700			3	21	
R8-32	L	4/2/87	6,020				10	180	4,300	210	1,100	50		60	40			60	10
R8-34	L	4/2/87	21		2				3		16								

* Lab
F - Friend Laboratory, Inc.; Waverly, NY
O - O'Brien & Gere Engineers, Inc.; Syracuse, NY
L - Lancaster Laboratories, Inc.; Lancaster, PA
H - NY Department of Health

(A) - These samples were tested for additional HSL parameters.

TABLE 1-7 (continued)
GROUND WATER ANALYSIS
BEDROCK WELLS - VOLATILE ORGANIC RESULTS (PPB)

Bedrock Wells	Lab *	Date Sampled	Total Volatiles	Benzene	Toluene	Ethyl-benzene	1,1-Dichloro-ethene	1,1-Dichloro-ethane	Trans-1,2-Dichloro-ethene	Chloroform	Trichloro-ethene	Methylene Chloride	Vinyl Chloride	1,1,1-Trichloro-ethane	1,2-Dichloro-ethene	Chloroethene	Tetrachloro-ethene	Dibromochloro-methane	Carbon Tetrachloride
R8-1	F	12/15/83	169								169								
	F	4/25/84	867								800			67					
	O	3/13/85	ND																
	L	9/12/85	326				5	15	160		95		39	8	4				
R8-2	F	12/15/83	63 (U)								51			12					
	F	4/26/84	132						67		54			11					
	O	3/13/85	61						39		22								
	L	9/12/85	140					12	62		17		28	17	2	2			
R8-3	O	3/12/85	5,831		950	270		650	1700				1,900	350	11				
	L	9/17/85	5,000		1,200			1100	1200				1,500						
R8-4 (A)	O	3/12/85	28,930		1,300	470	190	930	9300	710	12,000	170	130	830	2,900				
	L	9/18/85	47,000		13,000	1,100	500	1500	12000	1500	14,000	1,400	600	800	600				
R8-5	O	3/11/85	9,059		1,200	21	28	220	4900		200		590	1,900					
	L	9/17/85	11,720		4,590	260		280	4500				490	1,800					
R8-24	L	9/12/85	107					26	59		4		16	2	1				
R8-28	L	9/18/85	820		150	20		50	350				130		20		100		
R8-30	L	9/18/85	10,600		700	200		500	5900	500	600		1,900	100	200				
R8-33	L	4/2/87	840					50	650	40	90		10						
R8-35	L	4/2/87	136	1	16	4		3	32	1	77			2					
R8-36	L	4/2/87	12					2	5		3			2					
R8-37	L	4/2/87	1,740		190			120	680				750						
Unalam Well	H	4/17/85	2,930	400	10	5	9	140	1900	20	94		280	52	20				
	L	9/18/85	2,420					190	1800		60		370						
	L	4/2/87	1,720					190	1,200		20		280	30					
Village Maintenance Shop Well (150')	L	3/4/87	14					2	9		2		1						
Village Maintenance Shop Well (400')	L	3/4/87	ND																

* Lab F - Friend Laboratory, Inc.; Waverly, NY
O - O'Brien & Gere Engineers, Inc.; Syracuse, NY
L - Lancaster Laboratories, Inc.; Lancaster, PA
H - NY Department of Health

(U) - Some minor additional compounds were reported.

(A) - These samples were tested for additional HSL parameters.

TABLE 1-7 (continued)
GROUND WATER ANALYSIS
INORGANIC ANALYTICAL RESULTS (PPM)

Well	Sample Date	Antimony	Arsenic	Beryllium	Cadmium	Chromium	Copper	Lead	Mercury	Nickel	Selenium	Silver	Thallium	Zinc
R8-1	3/13/85													
R8-2	3/13/85													
R8-3	3/12/85													
R8-4	3/12/85													
R8-5	3/11/85													
R8-6	3/11/85													
R8-7	3/13/85	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
R8-8	3/13/85	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
R8-9	3/13/85	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
R8-10	3/13/85													
R8-11	3/13/85													
R8-12	3/13/85													
R8-13	3/13/85													
R8-14	3/13/85													
R8-15	3/13/85													
R8-16 D	3/12/85													
R8-16 I	3/12/85													
R8-16 S	3/12/85													
R8-17 D	3/12/85							0.05						0.03
R8-17 I	3/12/85		0.02					0.15						
R8-18 I	3/12/85		0.02					0.10						
R8-18 S	3/12/85	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
R8-19 D	3/12/85													
R8-19 I	3/12/85													
R8-19 S	3/12/85													
R8-20	3/13/85	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND
R8-21	3/13/85													
Gifford Spring	3/13/85		0.01											

All of the above inorganic results were supplied by O'Brien & Gere Engineers, Inc.
ND = none detected.
Blank spaces = not analyzed.

TABLE 1-7 (continued)
GROUND WATER ANALYSIS-HSL RESULTS
APRIL 1-3, 1987

Compound	Sample Location			
	R 8 - 4	R8-18 S	R8-18 I	* Gifford Road Spring
Volatile Organics (PPB)				
Total Volatiles	108.78	2530	73900	442
Vinyl chloride	0.7	30	2100	70
Methylene chloride	1.6	8		
Acetone	48			
1,1-Dichloroethene			100	
1,1-Dichloroethane	1.1	250	940	190
Trans-1,2-Dichloroethene	15	1900	32000	32
Chloroform	0.48	7	370	
1,2-Dichloroethane		20		
2-Butanone	5.8			
1,1,1-Trichloroethane	0.75	290	5000	9
Trichloroethene	16	25	90	
4-Methyl-2-pentanone	1.8			
Toluene	16		28000	11
Ethylbenzene	0.65		1900	
Total Xylenes	0.9		3400	130
Semi Volatile Organics (PPB)				
Naphthalene	40		200	40
Pesticides (PPB)				
Heptachlor Epoxide		0.8		
PCB-1242			38	
PCB-1254		19	39	
PCB-1260		3	5	
Metals (PPM)				
Arsenic			0.031	0.027
Barium	0.3		0.2	0.2
Calcium	19.9	72.8	53.5	25.7
Iron			5.75	23.1
Magnesium	3.24	24.1	33.4	22.1
Manganese	0.01		15.5	5.94
Potassium	5.99	3.85	5.23	3
Sodium	11.8	58.2	40.3	31.9
Zinc			0.02	

* Gifford Road Spring - Ground Water Discharge
Blank Spaces = none detected

- a glaciofluvial sand lens immediately west/northwest of the landfill area,

- a dense red glacial till underlying the glaciofluvial sand and the southern portion of the landfill area, and

- a less dense sandy green and brown glacial till underlying the northern portion of the landfill area.

Ground water flow in the overburden occurs laterally northwestward through the permeable glaciofluvial sand, and both laterally and vertically downward through the till units. The brown till, being less dense, contains more water than the red till, in which water bearing zones are limited to occasional thin gravelly seams one-foot thick or less. Ground water flow in the bedrock is also to the northwest.

The cross sections shown in Figures 1-6 and 1-7 indicate that the ground water in the glacial till and the underlying bedrock are directly hydraulically connected, with a downward gradient toward the bedrock. Mathematical analysis of the relationship indicated that up to two-thirds of the overburden flow is along the vertical component, discharging to the bedrock flow system.

The pump testing of the Unalam Well indicated the presence of apparent bedrock fractures, passing essentially east-west from the Unalam Well between monitoring wells R8-28 and R8-37, through the area of R8-30. The pump test results also indicate that the downward hydraulic gradient between the glacial overburden and the bedrock is likely increased by the pumping of the Unalam Well, particularly along fracture zones.

The hydraulic properties of the glacial overburden vary widely, with conductivities ranging from less than 0.01 ft/day in dense till sections, to almost 25 ft/day in the glaciofluvial sand (Table 1-2). In the bedrock, hydraulic conductivities ranged from less than 1 ft/day in interfracture areas, to up to 23 ft/day in fractures. Ground water flow velocities in the glacial overburden were calculated to be approximately 0.14 to 0.2 ft/day in the horizontal direction, and 0.86 ft/day in the vertical direction. In the bedrock, flow along fractures is likely to be very rapid, particularly under the influence of pumping at the Unalam Well.

1.2.2.2 Route 8 Landfill Source Areas

Figure 1-8 is a geologic cross section through the test borings augered along the west shoulder of Route 8. Test boring TB-5 penetrated a small suspected oil disposal area, estimated to be 30 to 35 feet wide and 3 feet deep. Test borings TB-9 and TB-10 penetrated the larger suspected oil disposal area, estimated to be 60 to 70 feet wide and 10 feet deep. The ground water table was encountered at a depth of about 15 to 20 feet, and was below the smaller disposal area, but within the larger one. The results of the soil and waste material analyses are shown on the Figure 1-8 cross-section and in Table 1-3. The results indicate that the primary source area at the site is the Area 1 industrial fill area penetrated by boring TB-5, and the secondary source area is area which was penetrated by borings TB-9 and TB-10. Maximum concentrations of PCBs were 33 ppm in Area 2, and 13,300 ppm in Area 1. Maximum concentrations of VOCs were 8,513 ppb in Area 2, and 361,900 ppb in Area 1. Between and surrounding the source areas, the soil was oil-stained, and contained low concentrations of PCBs and VOCs.

TABLE 1-2

RESULTS OF SLUG TESTS OF OVERBURDEN AND BEDROCK WELLS

Overburden Wells

Well Number	Hydraulic Conductivity* (ft/day)	Well TOC Elevation (ft MSL)	Open hole or Screened interval (ft)	Strata Description
R8-10	2.22	1028	15-30	Sand and gravel, silt and gravel, (Gravel unit in Red Till)
R8-11	1.78	1024	10-20	Sand and gravel, and silt and gravel (Gravel unit in Red Till)
R8-12	22.25	1028	14-24	Sand, silty sand, and gravel (Glaciofluvial Sand)
R8-13	24.85	1019	6-16	Sand and gravel, silt and gravel, (Glaciofluvial Sand)
R8-16I	0.57	1025	20-30	Sandy till layer (Brown Till)
R8-17D	0.01	1019	28-33	Dense, angular shale gravel (Basal till)
R8-19S	1.73	1025	5-10	Silty sand and gravel, firm (Brown till)
R8-19D	0.08	1026	33-38	Dense,fine sand and gravel (Basal till)
R8-20	0.04	1007	15-20	Dense,fine sand and gravel (Red Till)
R8-27	0.08	1014	15-25	Gravelly, fine sand and silt (Red Till)

Bedrock Wells

Well Number	Hydraulic Conductivity* (ft/day)	Well TOC Elevation (ft MSL)	Open section of well (ft)	Strata Description
R8-33	13.00	993	95-120	alternating layers of sandstone and siltstone, two fractures
R8-35	0.11	986	39-59	alternating layers of shale and siltstone, one fracture
R8-36	23.00	993	90-140	alternating layers of siltstone and sandstone, one fracture
R8-37	0.15	982	69-87	alternating layers of siltstone and sandstone, one fracture
R8-3	2.65	1007	20.5-49	alternating layers of red and green siltstone, several fractures
R8-5	3.20	1039	31-61	alternating layers of siltstone, shale, and sandstone, several fractures

*Hydraulic conductivity calculated from method of Bouwer and Rice (1976).

1.2.2.3 PCB Migration

The PCB-containing oils have migrated principally westward from the source areas, through the brown and red glacial tills and the permeable glaciofluvial sand. Immediately beneath the source areas, some oils and PCBs migrated vertically into the brown and red glacial till units, and are present in the small gravelly water-bearing zones. This results in the presence of PCBs in the ground water at ppb concentrations in the shallow, intermediate and deep glacial overburden monitoring wells south of Gifford Road. North of Gifford Road, and in the bedrock wells, no PCBs were detected (Table 1-4).

Lateral PCB migration is restricted, for the most part, to the glaciofluvial sand unit and the section of green-brown sandy glacial till located south of Gifford Road. Figures 1-9 and 1-10 show the configuration of the sand and sandy till units, and the distribution of PCBs in these units, respectively. Oily discharges from this unit occur during wet weather at numerous seeps both north and south of Gifford Road, and for most of the year at the Gifford Road Spring (Figure 1-5).

Surface water analyses (Table 1-5) indicate that PCBs are present only in the discharge from the Gifford Road Spring (includes GRS4), and downstream thereof in the marsh area north of Gifford Road at one of six locations sampled. In addition, very low level PCBs (0.0003 ppm) were detected in a water sample at the K-Mart Drain. However, downstream of this location, at GRS1, no PCBs were detected.

Sampling/analysis of drainageway sediments for PCBs (Table 1-6) indicated that they are present at the Gifford Road Spring discharge and in the marsh area at location GRN2, at

concentrations of less than 10 ppm. Their absence at other locations indicates that the residual PCBs tend to principally become absorbed in the discharge area sediments before the oil discharges. No PCBs were detected in the Unalam Tributary stream bed.

1.2.2.4 VOC Migration

VOC migration at the site occurs in both the glacial overburden and bedrock aquifers (Table 1-7). The principal compounds detected near the source areas are toluene, trans-1,2-dichloroethene, and 1,1,1-trichloroethane. Figures 1-11 and 1-12 are isoconcentration maps of total VOCs in the glacial overburden and bedrock, respectively. Concentrations exceeding 10,000 ppb total VOCs have migrated north of Gifford Road in both flow systems. In the bedrock, concentrations exceeding 1,000 ppb have been detected at the Unalam Well. The principal compounds detected north of Gifford Road are trans-1,2-dichloroethene and vinyl chloride. Inorganics are for the most part not detectable.

Discharges of VOCs to the site surface waters occur at the Gifford Road Spring, marsh area seeps, and at the Unalam cooling water discharge to the Unalam Tributary (Table 1-5). These range from less than 1 to a few hundred ppb at the Gifford Road Spring and marsh areas. The Unalam Tributary ranges from less than 1 ppb upstream of the Unalam Well discharge, to over 300 ppb downstream thereof (STR4). At the K-Mart Drain, VOCs of 84 to 161 ppb have been detected.

1.2.3 Conclusions

The results of the investigations have revealed that PCBs and volatile organic compounds have migrated from the Route 8

Figure 1-6
Geologic Cross Section A-A'

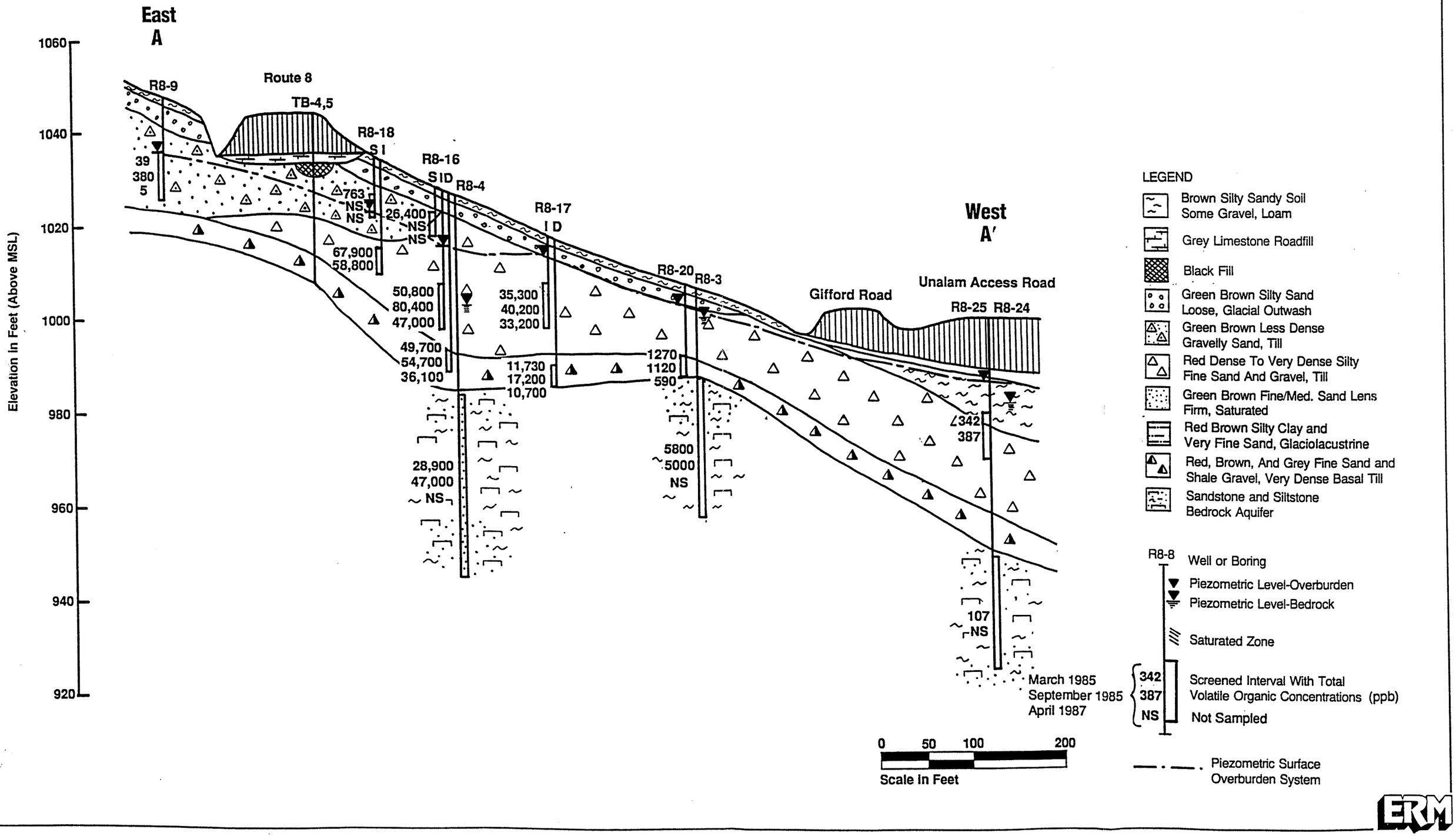
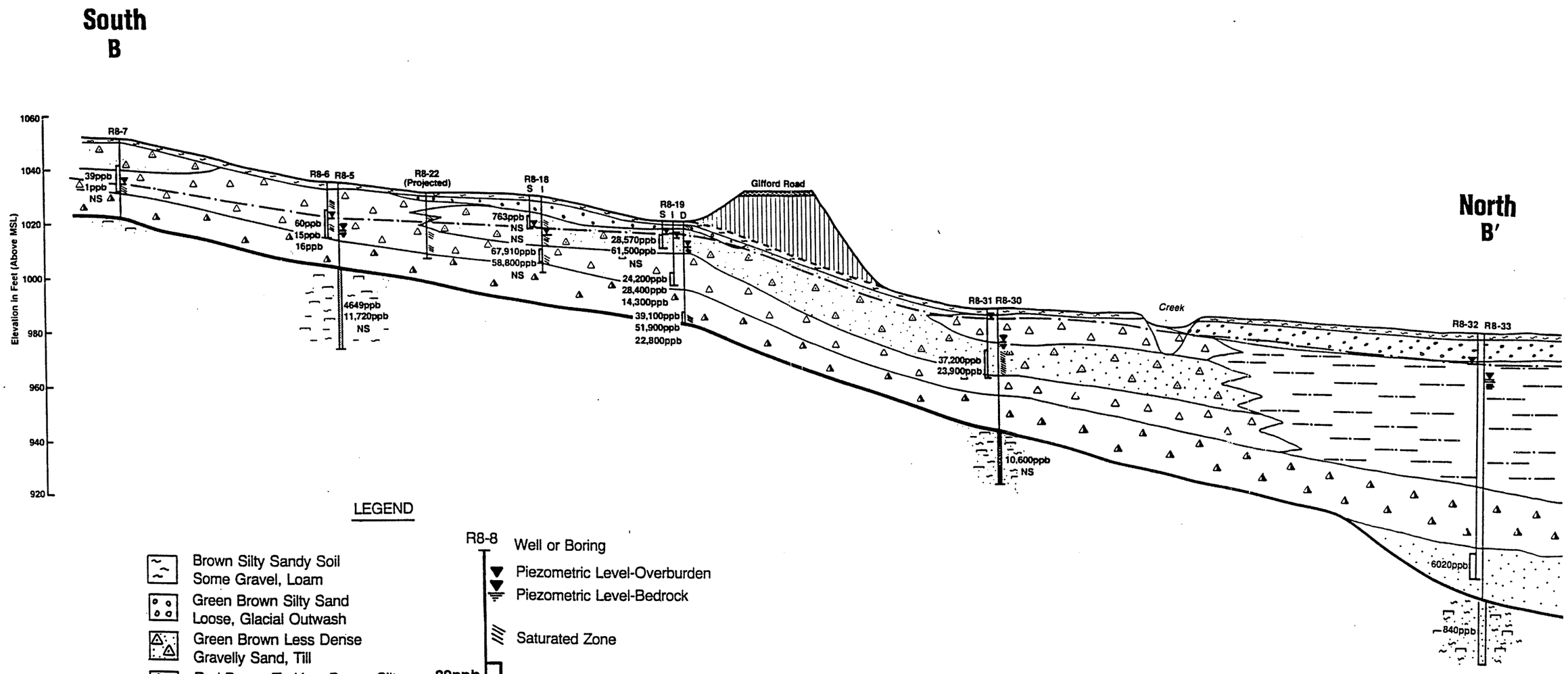


Figure 1-7
Geologic Cross Section B-B'



LEGEND

- Brown Silty Sandy Soil, Some Gravel, Loam
- Green Brown Silty Sand, Loose, Glacial Outwash
- Green Brown Less Dense Gravelly Sand, Till
- Red Dense To Very Dense Silty Fine Sand And Gravel, Till
- Green Brown Fine/Med. Sand Lens, Firm, Saturated
- Red Brown Silty Clay and Very Fine Sand, Glaciolacustrine
- Red, Brown, And Grey Fine Sand and Shale Gravel, Very Dense Basal Till
- Sandstone and Siltstone Bedrock Aquifer

R8-8 Well or Boring

Piezometric Level-Overburden

Piezometric Level-Bedrock

Saturated Zone

39ppb / 1 ppb / NS — Screened Interval With Total Volatile Organic Concentrations: Not Sampled

March 1985
September 1985
April 1987

Piezometric Surface Overburden System

0 50 100 200

Scale in Feet

Figure 1-8
Geologic Cross Section
Route 8 Test Borings

South

North

TB-11 TB-10 TB-9 TB-8 TB-7 TB-6 TB-5 TB-4 TB-3 TB-2 TB-1

1050
1040
1030
1020
1010

Elevation in Feet MSL

93 *1121*
ND *7480*
33 *4630*
47 *119*
10.9 *954*
13,300 *361,900*
1.8 *321*
ND
6.0 *202*
3.9 *226*
6.3
1.5
4.6
9.1
1.8
26 *8513*
3.5 *35*
1.3
2.4

LEGEND

- Brown, Red, and Grey Fine to Coarse Sand and Gravel, Till Used as Fill
- Grey Limestone Roadfill
- Grey to Brown Oil-stained Sand and Gravel
- Black Fill
- Brown, Less Dense Gravelly Sand, Till
- Red Dense to Very Dense Silty Fine Sand and Gravel, Till
- Red, Brown, and Grey Fine Sand and Shale Gravel, Very Dense Basal Till
- Sandstone and Siltstone Bedrock Aquifer

EXPLANATION - TEST BORING

TB-5 Boring

Depth at Which Water Table Was Encountered

6.3 PCB Concentration in Soil Sample (in ppm)

35 Total Volatile Organic Concentration in Soil Sample (in ppb)

Vertical Exaggeration 1:4

0 10 20 40

Horizontal Scale in Feet

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Figure 1 - 9
Isopach Map of Sand Unit and Brown Sandy Till

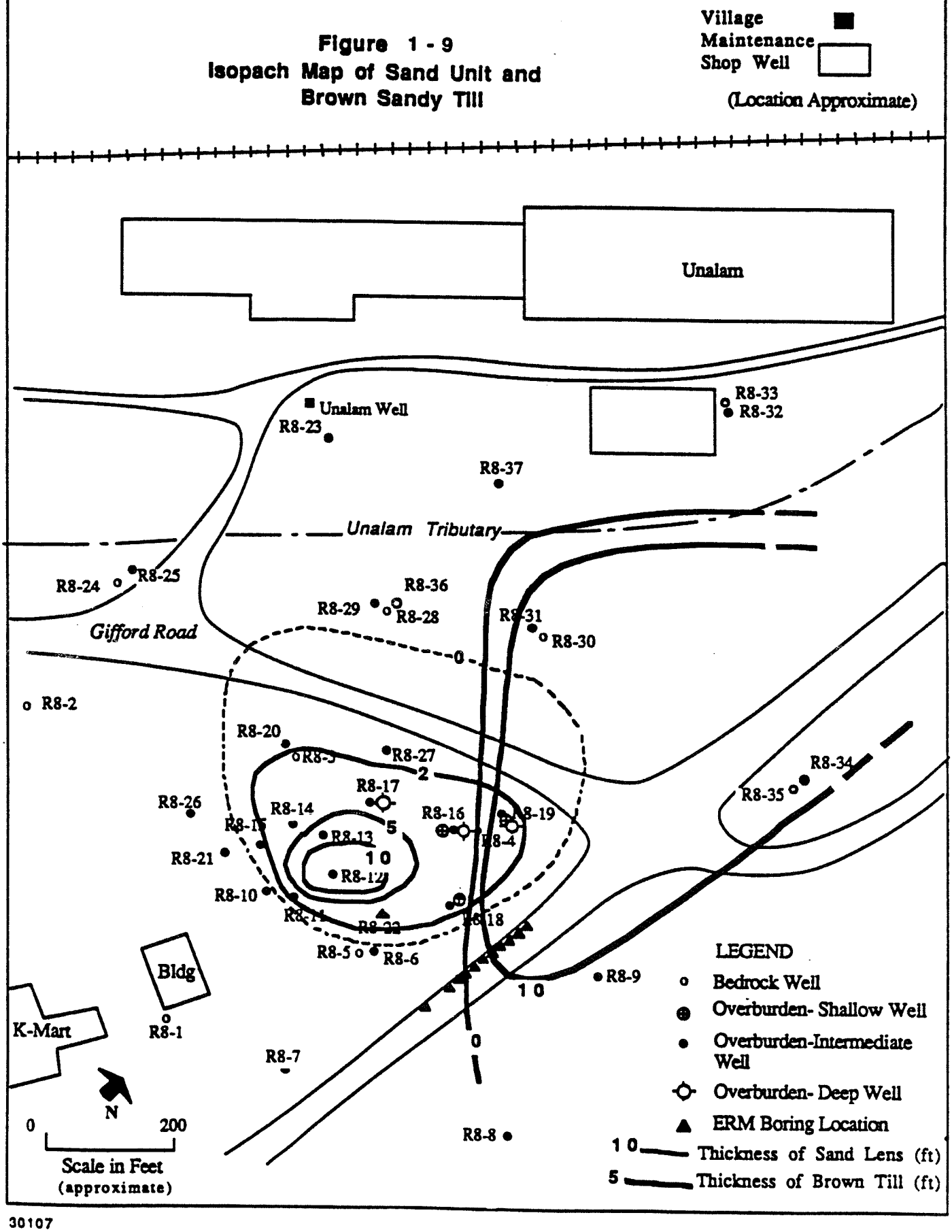

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Figure 1-10
Maximum PCB Concentrations Detected In Soil Borings
(Concentration in ppm)

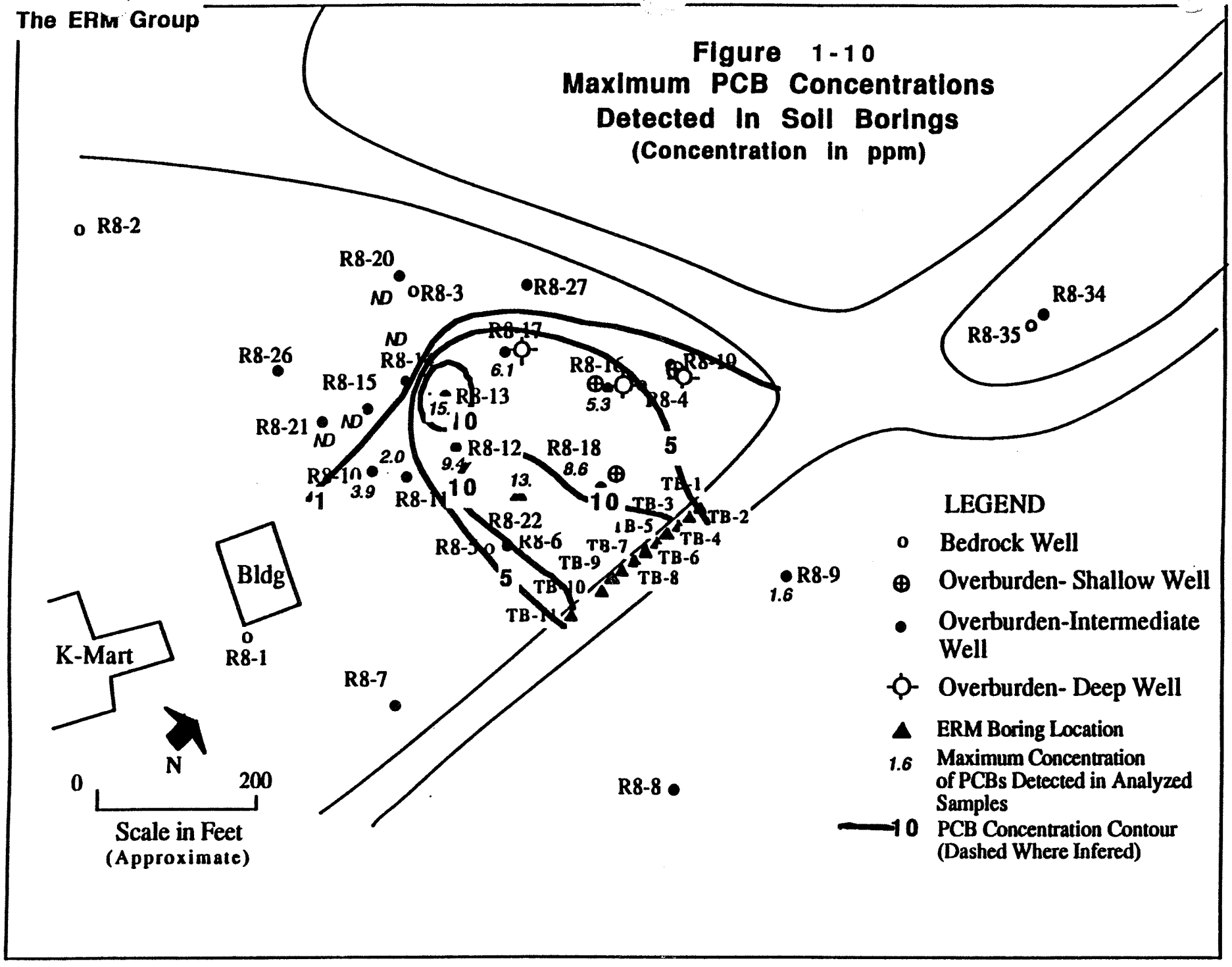

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Figure 1-12
Isoconcentration Map - Total VOCs
Bedrock System

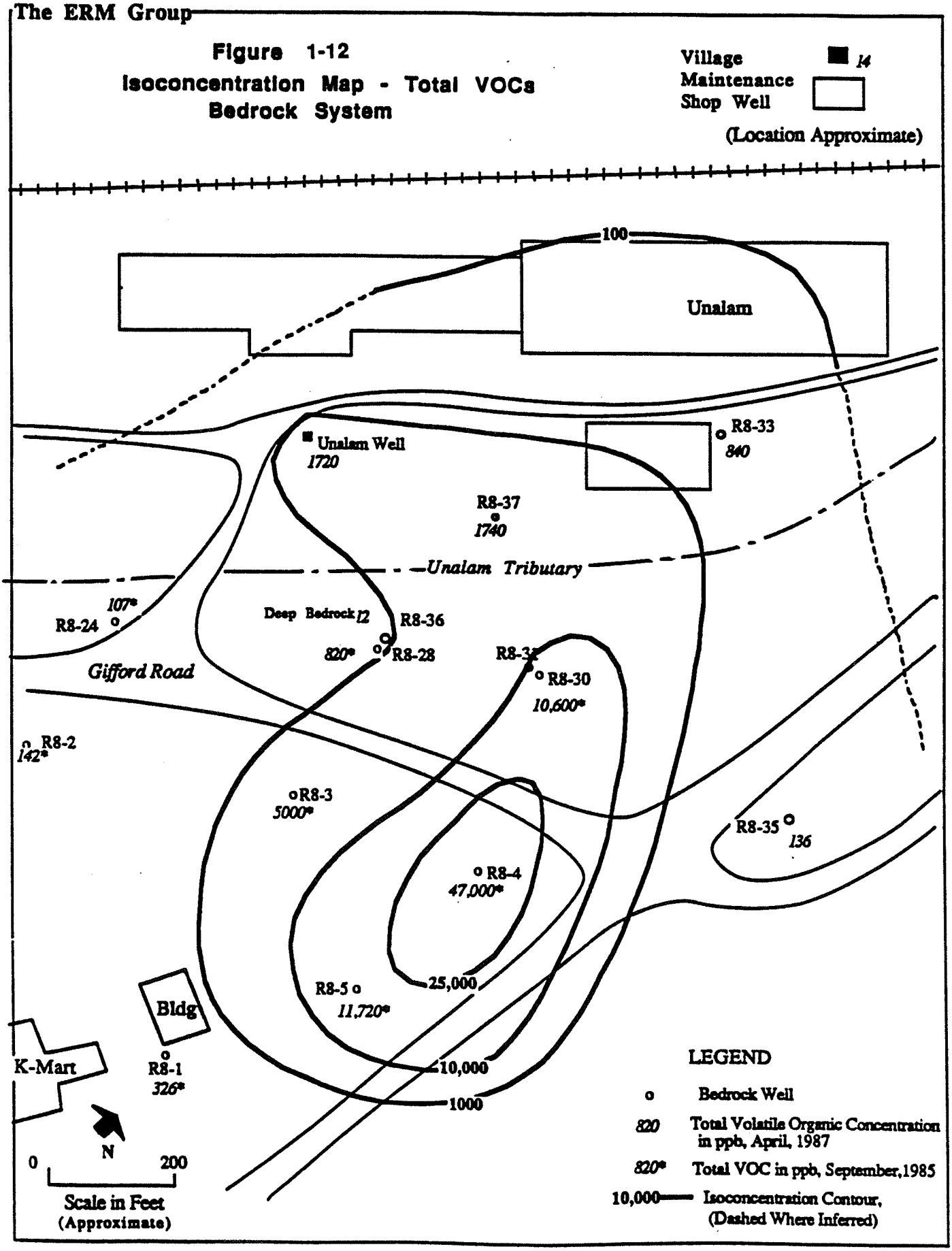

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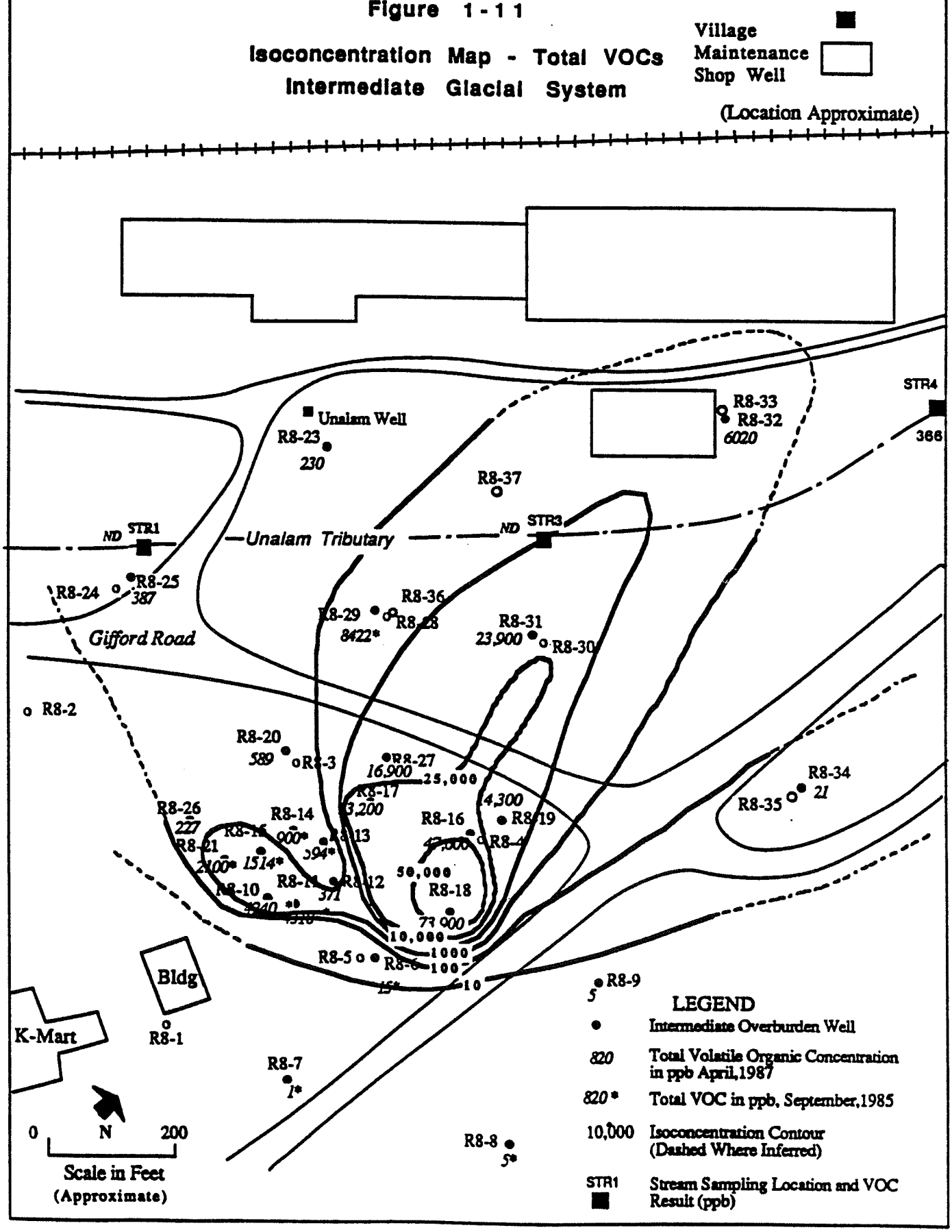
Figure 1-11

Isoconcentration Map - Total VOCs Intermediate Glacial System



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Landfill area. The principal source area is a former disposal pit or trench which now lies beneath the west shoulder of Route 8. From this area, PCBs have migrated in oil through a sand lens of limited extent, with discharge at the Gifford Road Spring approximately 300 feet west of the source area. As the PCBs have very limited solubility and adsorb onto the subsurface soils, migration in ground water and surface water has been limited, with none detected in the regional bedrock aquifer or in downgradient surface waters.

Volatile organic compounds have migrated from the source area in both a glacial overburden flow system, and the underlying regional bedrock flow system at concentrations up to several tens of thousands of ppb. The principal compounds of concern are toluene, ethylbenzene, 1,1 dichloroethane, trans 1,2 dichloroethene, trichlorethene, vinyl chloride and 1,1,1 trichlorethane. It was determined that site hydrogeologic conditions have allowed the vertical migration of the VOCs downward from the overburden, into the bedrock. In the bedrock the VOC plume is intercepted by pumping at the Unalam Corporation production well, which is used for cooling water. No wells used for potable water are affected by the Route 8 Landfill VOC plume, however.

Volatile organics are also present in seasonal site-related surface waters in the several hundred ppb concentration range. However, the Unalam Tributary, a second order tributary to the Susquehanna, appears to receive VOCs only from the Unalam cooling water discharge. The major conclusions of these investigations are:

- The principal source area for the waste constituents is a limited former pit or trench area which presently lies

beneath the west lane and shoulder of Route 8, approximately 250 feet south of the intersection of Route 8 and Delaware Avenue.

- PCB migration has occurred in solution in oil, and is concentrated in a permeable sand lens of limited extent, which discharges to the surface at the Gifford Road Spring and at smaller wet weather seeps.

- PCB migration has been limited by its low solubility and adsorption onto subsurface soils, such that little migration has occurred in site-related surface waters.

- PCBs are present in solution in shallow ground water near the source area at concentrations in the low ppb range; no PCBs were detected in the regional bedrock aquifer.

- Migration of VOCs has occurred in the concentration range of tens of thousands of ppb in the ground water beneath and downgradient of the site in both the glacial overburden and regional bedrock aquifers.

- No site-related VOCs are present in any well used for potable water.

- The downgradient limit of the overburden plume is uncertain, as it appears that a second downgradient source of VOCs may be present. However, it is apparent that the presence of the lacustrine silty clay body in the path of the plume limits the extent of its migration.

- The bedrock VOC plume is intercepted by the cyclical pumping of the Unalam Production Well.

- The Amphenol West Well pumping does not affect plume migration.

These conclusions form the basis of our understanding of the fate and transport process upon which the Risk Assessment is based. The presence of PCBs in subsurface soils, seeps, and shallow ground water and VOCs in ground water and surface water indicates that potential points of human exposure to site-related contaminants may exist. The potential for exposure to occur is addressed in the Risk Assessment.

1.3 Brief Description of the Risk Assessment Process as Applied to Hazardous Waste Site Investigations

The overall objective of a hazardous waste site Risk Assessment is to determine the magnitude and probability of actual or potential harm which the site poses to human health and the environment. Risk assessment techniques have been used in various regulatory programs employed by Federal and State agencies. The principal guidance manuals employed by the U.S. Environmental Protection Agency for performance of risk assessments at hazardous waste sites include the Superfund Public Health Evaluation Manual (U.S. EPA, 1986 a) and the Endangerment Assessment Manual (U.S. EPA, 1986 b).

Risk Assessment consists of four separate but inter-related evaluations as follows:

1. Indicator chemical selection: Identification of contaminants and selection of a set of representative chemicals which pose the major carcinogenic and non-carcinogenic risks at the site.

2. Exposure assessment: Identification of significant migration pathways and receptors, determination of current and future emission roles, analysis of environmental fate and persistence, and an exposure evaluation which includes the calculation of intakes by potentially exposed populations.

3. Toxicity assessment: Assessing the intrinsic toxicological properties of the indicator chemicals, which includes acute, subchronic, chronic, carcinogenic, and reproductive effects and development of acceptable daily intakes or cancer risk potency factors, if necessary.

4. Risk characterization: Quantitative estimations of the actual and potential hazards caused by the exposures to each indicator chemical and the possible additive effects of exposures to mixtures of chemicals.

An adequate characterization of risks at the site allows the site remediation process to be focused on actual and potential public health concerns. Exposures creating the greatest risk can be identified and mitigation measures can be selected to address these problems. In this sense, a Risk Assessment integrates the information obtained in the Remedial Investigations into a coherent set of objectives for initiation of the Feasibility Study.

1.4 Report Organization

This report is organized to present the methodology followed by the results of each stage of the Risk Assessment process in separate sections as follows:

Section 2 - Methodology

Section 3 - Indicator Chemical Selection

Section 4 - Exposure Assessment

Section 5 - Toxicity Assessment

Section 6 - Risk Characterization

SECTION

2

SECTION 2

RISK ASSESSMENT METHODOLOGY

Risk Assessment (RA) is a process which evaluates the collective demographic, geographic, physical, chemical, and biological factors at a site to determine whether or not there may be a risk to public health or the environment, and attempts to characterize that risk if it does exist. This Risk Assessment has been developed to assess conditions currently associated with the Route 8 Landfill. It is based on the results of the Remedial Investigations summarized in the previous section. It will allow the determination of areas that must be remediated to effectively minimize any site-related risks and to comply with regulatory standards.

The need to include estimates of risk in the decision-making process for disposal sites has been recognized by the U.S. EPA, and is now a required part of Comprehensive Environmental Resources, Compensation and Liability Act (CERCLA) site investigations. Although the Route 8 Landfill is not a CERCLA site, the procedures for a CERCLA risk assessment will be used since they represent the most comprehensive and nationally-recognized guidance currently available.

2.1 US EPA's Risk Assessment Process

This section provides a broad overview of the Risk Assessment (EA) process for hazardous waste sites. The discussion is not intended to be a comprehensive guide to preparing risk

assessments. The US EPA has proposed guidelines for the preparation of assessments in the Endangerment Assessment Handbook (US EPA, 1986b), Superfund Public Health Evaluation Manual (US EPA, 1986a), Exposure Assessment Manual (US EPA, 1986c), and Toxicology Handbook (US EPA, 1986d).

A Risk Assessment is normally conducted after the completion of a Remedial Investigation (RI). The RI determines the nature and extent of contamination at a site, and its results form the data base from which potential exposures can be determined and risks assessed. In addition, the RI defines whether or not the present conditions at the site are at steady state.

There are four separate evaluations which must be completed in a Risk Assessment. These steps are listed below:

1. Identification of indicator compounds, which are used to represent the majority of carcinogenic risk and noncarcinogenic hazard posed by a site;

2. Exposure assessment, which includes the calculation of doses to potentially exposed populations;

3. Toxicity assessment of the potential carcinogenicity of site indicator chemicals and of noncarcinogenic effects; and

4. Characterization of the risks of existing conditions to all affected populations due to exposure to the indicator chemicals.

2.2 Indicator Chemicals

Indicator chemicals are selected on a site-specific basis. They are intended to be those chemicals which represent the majority ($\geq$95%) of the risk posed by the site.

The selection and ranking of indicator chemicals implements the procedure outlined in the Superfund Public Health Evaluation Manual (US EPA, 1986a). As part of this selection process, toxicological information about each compound is compiled using Appendix C of the Superfund Public Health Evaluation Manual (US EPA, 1986a) and the US EPA's on-line Integrated Risk Information System (IRIS). A range and representative concentration for each compound is calculated for each appropriate medium. This information includes the following:

1. toxicology class: potential carcinogens (PC) or noncarcinogens (NC);

2. severity-of-effect ratings value for noncarcinogens;

3. weight-of-evidence ratings for carcinogens; and

4. toxicity constants for the various environmental media.

The site-related compounds are then subdivided into potential carcinogens and noncarcinogens. An indicator score (IS), the product of the chemical concentration and the toxicity constant, is calculated for each medium (subsurface soil, surface soil, surface water, and ground water) and then summed to yield a total indicator score per compound. The compounds are then ranked numerically based upon decreasing indicator scores. The top-scoring compounds (based on IS values) are then re-evaluated

based upon frequency of detection, water solubility, vapor pressure, Henry's law constant, and soil organic carbon partition coefficient (K_{OC}) to determine the final indicator compounds. This re-evaluation has a direct relationship to the IS value but selectively eliminates those compounds which are degradation products, have similar physical or chemical properties, or have comparable half-lives in the various environmental media.

2.3 Exposure Assessment

The purpose of an exposure assessment is to determine the possible intakes of each indicator compound by potentially exposed populations. Intakes are ultimately determined through a series of steps in the exposure assessment process. First, the modes of contaminant transport, leading from the sources on the site to a point of exposure, are defined. Concentrations of the indicator compounds are determined in each medium to which a population may be exposed. Potentially exposed populations are then defined, and possible exposures are determined. Finally, the possible intakes resulting from the potential exposures are calculated.

The sources of contamination at the site are defined in the RI. The exposure assessment determines the migration of contaminants from the site to potentially exposed populations through the following tasks:

- Evaluating fate and transport processes for the indicator compounds;

- Establishing exposure pathways;

- Determining possible exposures to potentially affected populations; and,

- Calculating doses and resultant intakes.

2.3.1 Evaluate Fate and Transport Processes for the Indicator Compounds

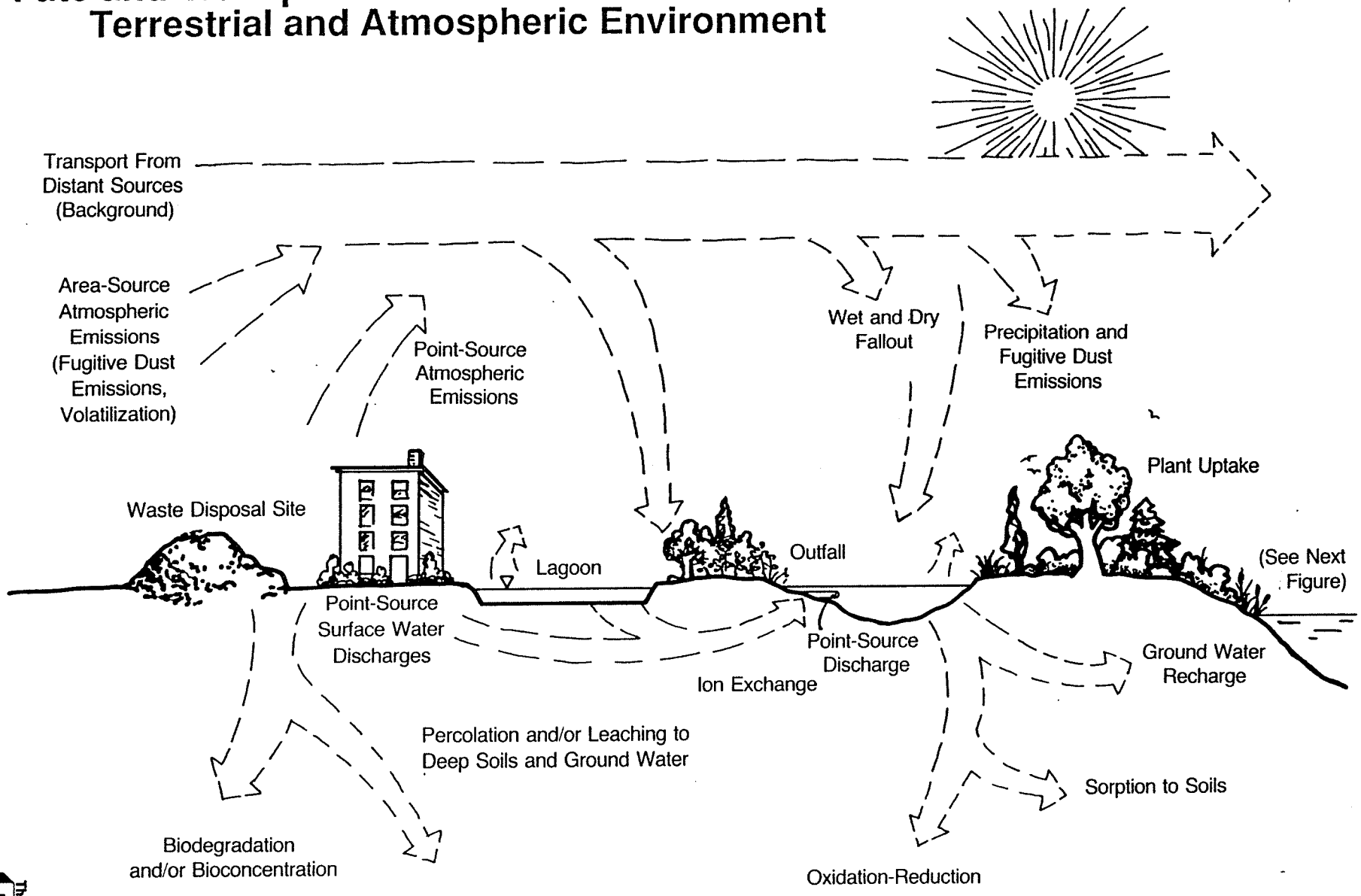
The first step in the analysis of exposure is to evaluate the fate and transport processes for the indicator compounds in a qualitative manner; this step is taken so that the potential for releases from sources of contamination can be considered. This analysis can also identify any significant intermedia transport routes that may need to be later evaluated in detail through fate and transport modeling. Examples of the fate and transport processes of chemicals in the terrestrial, atmospheric, and aquatic environments are presented in Figures 2-1 and 2-2, respectively.

Examples of the environmental fates of the Route 8 indicator chemicals include sorption by soils and sediments, volatilization into the atmosphere, photochemical degradation, and bioaccumulation. Physical and chemical constants such as solubility and octanol-water partition coefficients are tabulated so that their importance in affecting fate and mobility of the contaminants can be evaluated.

2.3.2 Establish Exposure Pathways for Each Medium

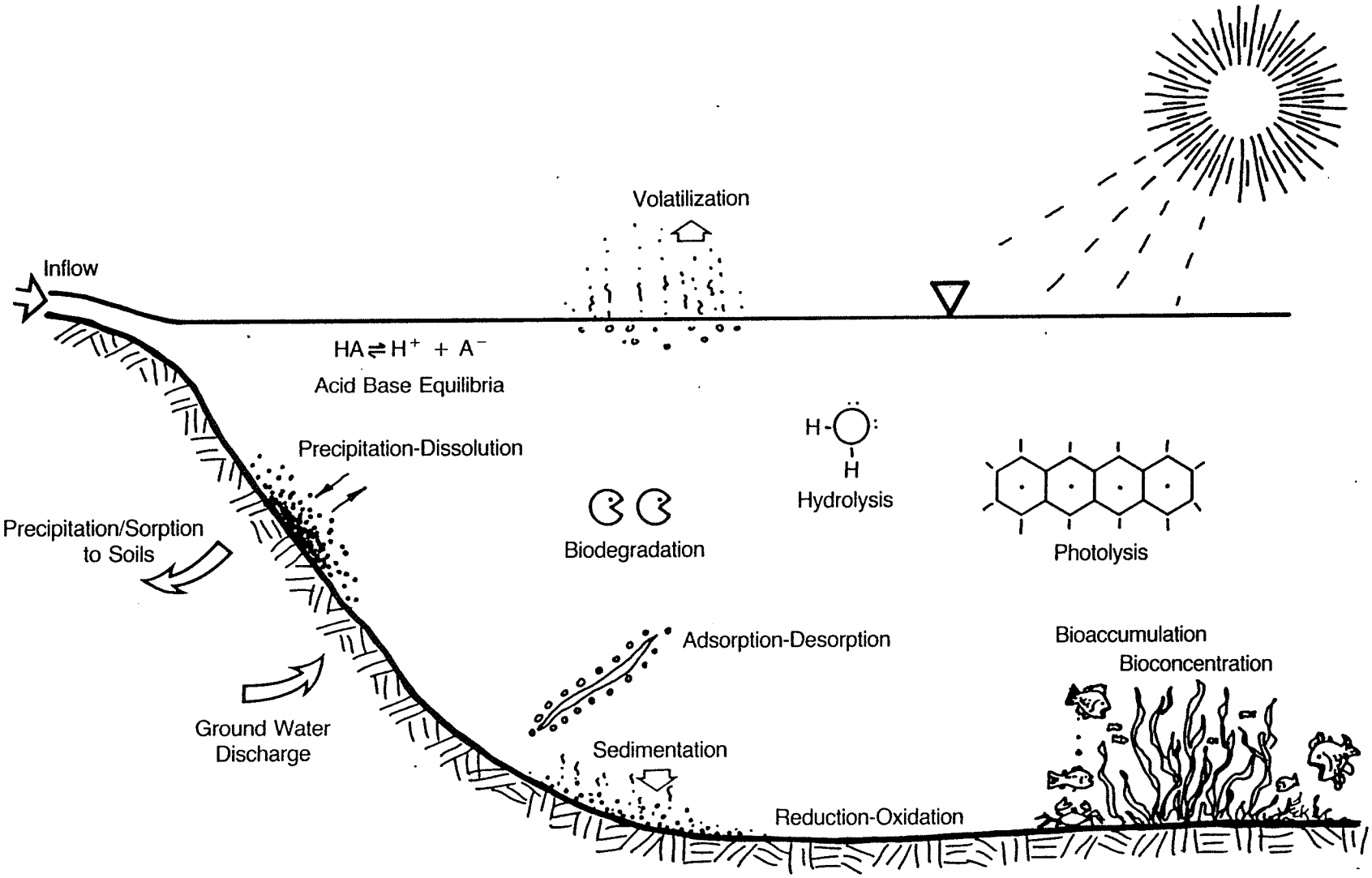
An exposure pathway qualitatively establishes the connection, through one or more environmental media, between a source of a contaminant and a human population. An exposure pathway is

Figure 2-1
Fate and Transport Processes of Chemicals in the Terrestrial and Atmospheric Environment



Source: ERM 1986

Figure 2-2
Fate and Transport Processes of Chemicals in the Aquatic Environment



Source: ERM 1986

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considered complete if it consists of the following four elements:

- source of contaminant;
- contaminant release pathway;
- transport; and
- human contact at exposure points.

Exposure pathways are determined by integrating information from the RI with knowledge about potentially exposed populations and their likely behavior.

2.3.3 Determine Exposures to Potentially Affected Populations

The next step is the quantitative determination of indicator chemical concentrations in the media of concern at the potential points of contact with human populations (i.e., exposure point concentrations). This step may be quite complicated; it requires knowledge of the contaminant source and the behavior of the contaminant in, and its effect on, the environment between the site and any potentially exposed populations. The exposed populations for each medium may also be different, as would be the case if the direction of ground water flow were opposite that of the prevailing wind.

If the transporting medium can be treated as steady-state, monitoring data may be used to represent exposure point concentrations. If no data are available or if transient or increasing concentrations are suspected, models may be used to predict concentrations.

Ground water contaminant transport through advection and dispersion is normally described in the RI. Transport in such other media as surface water and the atmosphere is not normally evaluated in the RI, and modeling assessments are often required to determine exposures. Many factors, including the fate processes reviewed previously, are considered when selecting the most appropriate model.

2.3.4 Calculate Doses to and Possible Intakes by Potentially Exposed Populations

Once exposure point concentrations in all media have been determined, the resultant doses and intakes to potentially exposed populations are calculated. Dose is defined as the amount of compound contacting body boundaries (skin, lungs, or gastrointestinal tract), and intake is the amount of chemical absorbed by the body.

Doses and intakes are normally calculated in the same step of the exposure assessment. First, for each exposure pathway under consideration, a dose per event is developed. This value quantifies the amount of contaminant contacted during each exposure event. "Event" may have different meanings depending on the nature of the scenario under consideration (e.g., each day's inhalation of contaminated air constitutes one inhalation exposure event). The quantity of contaminant absorbed per event (intake) is calculated by additionally considering any pertinent physiological parameters (such as gastrointestinal absorption rates, etc.). When extent of intake (systemic absorption) from a dose is unknown, or cannot be estimated by defensible argument, dose and intake are taken to be the same (i.e., 100% absorption from contact).

Event-based intake values are converted to final intake values by multiplying the intake per event by the frequency of exposure events over the time frame being considered. Subchronic (short-term) exposure is based on the number of exposure events that occur during the short-term time frame using maximum contaminant concentrations in the media to define dosage. Subchronic exposure values are intended to represent 10- to 90-day exposures. Chronic (long-term) exposures are based on the number of events that occur within an assumed 70-year lifetime using average contaminant concentrations in the media of concern to define dosage.

Estimates of daily intakes of contaminants are necessary to assess risk; both subchronic and chronic daily intakes (SDIs and CDIs) are calculated. Daily intake estimates are expressed in terms of mass of contaminant per unit of body mass per day (e.g., mg/kg/day). SDIs and CDIs are derived by dividing subchronic and chronic daily exposures, respectively, by an appropriate average body mass (e.g., a 70 kg adult). For assessment of carcinogenic effects, the CDI values are used. For assessment of noncarcinogenic effects, both SDIs and CDIs are used to evaluate subchronic and chronic effects.

In the Route 8 Risk Assessment, intakes resulting from seven modes of exposure were calculated: ingestion of drinking water, ingestion of soil by children (Pica), dermal exposure to contaminants in soil, surface water, and bath/shower water, inhalation of volatilized contaminants from surface water, and inhalation of volatilized contaminants during bathing. The methods by which each type of intake was calculated are given as follows.

Inhalation Exposure

Potential inhalation intakes are estimated based on the length of exposure, the inhalation rate of the exposed individual during the event, the concentration of contaminant in the air breathed, and the amount retained in the lungs. The formulae for calculating intakes during inhalation (including inhalation while bathing or showering) are given as follows:

Inhalation

$$Inh = Conc \times 1/BW \times BR \times E_t \times Abs_a$$

Where:

- Inh - Total exposure (mg/kg/day)
- Conc - Concentration of contaminant in water (mg/L)
- BW - Body weight (kg)
- BR - Breathing rate (m^3/hr)
- E_t - Length of exposure (hr/day)
- Abs_a - Percent of contaminant absorbed into the bloodstream

Inhalation While Bathing

$$Inh = ([(AW \times Conc \times E_1 \times BR)/(2 \times SV)] + [(AW \times Conc \times E_2 \times BR)/BV]) \times Abs_a \times 1/BW$$

Where:

- Inh - Total exposure (mg/kg/day)
- Conc - Concentration of contaminant in water (mg/L)
- AW - Amount of Water used during shower (L)
- BW - Body Weight (kg)

BR - Breathing rate (m^3/hr)
E_1 - Length of exposure in shower (hr)
E_2 - Length of additional exposure in enclosed bathroom (hr)
SV - Shower volume (m^3)
BV - Bathroom volume (m^3)

Subchronic (short-term) exposure resulting from inhalation is calculated using the maximum contaminant air concentration. Chronic (long-term) exposure is based on the average concentration.

Dermal Exposure

Dermal intake is determined by the concentration of compounds in a contaminated medium that is contacted, the body surface area contacted, the duration of the contact, the flux of the medium of concern across the skin surface, and the absorbed fraction. For exposure to contaminated water, dermal intake is calculated as follows:

Surface/Ground Water

$$DEX = Conc \times 1/BW \times Area \times FR \times E_t \times Abs_k$$

Where:

DEX - Total exposure (mg/kg/day)
Conc - Concentration of contaminant in soil (expressed as fraction of total weight)
BW - Body weight (kg)
FR - Mass flux rate of water across the skin surface; water based (mg/cm^2/hr)
E_t - Length of exposure (hr/day)

Abs_k - Percent of contaminant absorbed into the bloodstream
Area - Amount of skin surface area exposed (cm^2)

For exposure to chemicals in soil, dermal intake is calculated as follows:

Soils

$DEX = N_e \times Conc \times 1/BW \times Area \times DA \times Abs_s \times SM$

Where:

DEX - Total exposure (mg/kg/day)
N_e - Number of events per day (1/day)
Conc - Concentration of contaminant in soil (expressed as fraction of total weight)
BW - Body weight (kg)
Area - Amount of skin surface area exposed (cm^2)
DA - Dust adherence (mg/cm^2)
Abs_s - Skin absorption rate of compounds in soil (%)
SM - Soil matrix effect (%)

Possible subchronic intake resulting from each dermal exposure event is calculated using the maximum (short-term) contaminant concentrations in the appropriate media. Chronic intake is based on average (long-term) contaminant concentrations.

Ingestion Exposure

Potential intake resulting from ingestion of water-borne contaminants and contaminants sorbed on soil is determined by multiplying the concentration of the contaminant in the media of concern by the amount of water or soil ingested per day and the

degree of absorption (assumed to be one hundred percent), as follows:

Ingestion

$$Ing = Conc \times 1/BW \times Amt \times Abs_W$$

Where:

Ing - total exposure (mg/kg/day)
Conc - concentration of contaminant in water (mg/L) or soil (mg/kg)
BW - Body weight (kg)
Amt - Amount ingested (liters or kilograms per day)
Abs_W - Percent of contaminant absorbed into the bloodstream

2.4 Toxicity Assessment

A toxicity assessment is conducted as part of a risk assessment to quantitatively and qualitatively assess the potential for adverse human health effects from exposure to the indicator compounds. The quantitiative portion of the evaluation entails identifying the relevant indices of toxicity against which exposure point intakes can be compared during the risk characterization evaluation.

The qualitative aspect of the assessment presents summaries of the adverse human health effects, typical environmental levels or background concentrations, toxicokinetics, toxicodynamics, and ecotoxicology, associated with each indicator compound. This assessment includes the consideration of experimental studies using mammals and aquatic nonmammalian species (where available), as well as epidemiological studies. Because of its major impact

on the risk assessment, a discussion on the evaluation of carcinogenicity including the procedures used for classifying animal and human carcinogens by both US EPA and the International Agency for Research on Cancer (IARC) of the World Health Organization, and the attendant uncertainties, will be presented.

Evaluations of carcinogenicity within a risk assessment basically involve two steps: (1) the identification of potential carcinogens among the contaminants present at the site, and (2) the quantitative determination of their carcinogenic potency.

Evidence of possible carcinogenicity in humans comes primarily from long-term animal tests and epidemiological investigations. Results from these studies are supplemented with information from short-term tests, pharmacokinetic studies, comparative metabolism studies, structure-activity relationships, and other relevant information sources.

For judging the qualitative evidence of carcinogenicity, US EPA and the IARC have adopted a policy of "weight-of-evidence," meaning that the quality and adequacy of all relevant data on responses induced by a possible carcinogen using different procedures will be considered. There are three major steps in determining the weight-of-evidence for carcinogenicity:

1. characterization of the evidence from human studies and from animal studies individually,

2. combination of the two types of studies into a final indication of overall weight-of-evidence for human carcinogenicity, and

3. evaluation of all supportive information to determine whether the overall weight-of-evidence should be modified.

Further details concerning the classification systems of US EPA and IARC and the use of these data in the endangerment assessment process are presented in Appendix B.

The second phase in carcinogen assessment involves the quantification of risk. Experimental studies of carcinogenic effects that utilize the low exposure levels usually encountered in the environment generally are not feasible. Therefore, various mathematical models have to be used for extrapolation from the high doses used in animal bioassays down to the doses involved with exposure to ambient environmental concentrations. Since the resolution power of animal studies, for example, is not adequate for precise elaboration of the dose-response curve, extrapolating from a high dose to a low dose introduces a level of uncertainty which may amount to orders of magnitude. Given the recognized differences in carcinogenic response between species, and between strains of the same species, it is clear that additional uncertainties will be introduced when quantitative extrapolations, as from rodents to humans, are made. Of the various proposed models for quantitative extrapolation, US EPA recommends a linearized multistage model: "In the absence of adequate information to the contrary, the linearized multistage model will be employed" (Federal Register, Guidelines for Carcinogen Risk Assessment, 24 September 1986). The linearized multistage model assumes linearity at low doses. Alternative models do not assume a linear relationship and in general are less conservative. There is often no biologically sound basis for choosing one model over another. However, when applied to the same data, the various models can produce a wide range of risk estimates; the model recommended by US EPA usually

produces the highest estimates of risk. Moreover, this model does not provide a best estimate of risk, but rather an upper-bound probability that the risk will be less 95 percent of the time.

2.5 Risk Characterization

The risks to potentially exposed population from exposure and subsequent intake of the indicator compounds are characterized in the following tasks:

1. Calculation of noncarcinogenic hazard index, and
2. Calculation of carcinogenic risk.

2.5.1 Noncarcinogenic Hazard Index

The Hazard Index (HI) method is used for assessing the overall potential for noncarcinogenic effects posed by multiple compounds. This approach assumes that multiple subthreshold exposures could result in an adverse effect and that the magnitude of the adverse effect will be proportional to the sum of the ratios of the subthreshold exposures to acceptable exposures. This relationship can be expressed as:

$$\text{Hazard Index} = E_1/AL_1 + E_2/AL_2 + \ldots + E_i/AL_i,$$

where

E_i = Exposure level (or intake) for the i^{th} contaminant

AL_i = Acceptable level (or intake) for the i^{th} contaminant.

For a single contaminant, there may be a potential adverse health effect when the hazard index exceeds unity or one, although because the "acceptable level" itself incorporates a large margin of safety (safety factor), it is possible that no toxic effects may occur even if the "acceptable level" is exceeded. For multiple chemical exposures, hazard indices, if summed, may result in an overall hazard index that exceeds one even if no single chemical exceeds its acceptable level. However, the assumption of additivity should be made only for compounds that produce the same toxic effect by the same mechanisms of action.

US EPA has developed some preliminary information regarding Acceptable Intakes for Subchronic Exposures (AISs) and Acceptable Intakes for Chronic Exposures (AICs or Rfds) (US EPA, 1986a). Where these are available, they are used as acceptable levels for subchronic and chronic exposures, respectively. When unavailable, these intakes are calculated using approved US EPA methodology from well-designed and conducted toxicology studies on experimental animals.

2.5.2 Carcinogenic Risk

For potential carcinogens, risks are estimated as probabilities. The carcinogenic potency factor, which is the upper 95%

confidence limit of the probability of a carcinogenic response per unit intake over a lifetime of exposure, converts estimated CDIs directly to incremental risk values. This is not the only methodology to calculate risks, but it is likely to be an upper bound. In general, because only relatively low CDIs are likely to result from environmental exposures, the US EPA methodology assumes that the exposure will be in the linear portion of the dose-response curve. Based on this assumption, the slope of the dose-response curve is equivalent to the carcinogenic potency factor, and the risk is directly proportional to the CDI at low levels of exposure. The low-dose carcinogenic risk equation is

Risk = CDI x Carcinogenic Potency Factor.

2.6 Limitations Inherent in the Risk Assessment Process

The Risk Assessment process as applied at hazardous waste sites has a number of limitations. Many of these are well known, but are reluctantly accepted because there is at present no practical alternative means of making consistent quantitative estimates of risk. In fact, the additive endangerment assessment methodology was developed as a short-term stopgap until the information was available for more complete endangerment assessment methods to be developed and applied (NAS 1983). Some progress toward applying alternative methodologies is being made in on-going studies. Some other limitations are discussed below.

1. Reliance on Animal Toxicology

The use of animal models for human toxicological response, including the necessity to scale experimental dosages according to body size and metabolism, continues to draw criticism from the

other than uncontrolled epidemiological studies (e.g., inferences from "cancer clusters").

2. Identifying Critical Pollutants

With complex chemical mixtures, it is frequently necessary to screen the list of toxicants to rank them according to toxicological potency, eliminating those that contribute little to the overall additive risk. A criticism of screening is that individual substances, of little risk in themselves, may be screened out even though they may act as catalysts to activate the toxicity of other compounds in the mixture. The eventual incorporation of information on synergistic behavior into the screening process may eventually reduce this concern.

3. The Worst Case Exposure Assumption - The Maximally Exposed Individual

The inherent conservatism in the risk estimation is sometimes criticized on several grounds. The use of "reasonable worst case" exposure scenarios often results in assessing exposures that no real individual will ever actually experience. Similarly, safety factors are often incorporated in the analysis; often, the size of the safety factor is roughly proportional to the assessor's lack of certainty in the data. Together, these conservative assumptions act to remove the estimated risk from actual risk (e.g., as would be manifested by the actual incidence of cancer in a population with a known exposure to a toxicant).

All these limitations are present in this Risk Assessment for the Route 8 Landfill. In addition, site-specific concerns may contribute to uncertainty as well. These concerns include the use of RI sampling results to represent environmental

concentrations over large areas; the use of ground water modeling to estimate concentrations of contaminants in ground water at a hypothetical well; and the use of air and emissions modeling to estimate concentrations of contaminants in air at various locations in the Route 8 area.

SECTION

3

SECTION THREE

INDICATOR COMPOUNDS

3.1 Indicator Compound Selection

For the purposes of risk assessment, "indicator compounds" are selected on a site-specific basis. These are generally the compounds which have the following characteristics:

- the most toxic, persistent, and mobile;
- most prevalent; and
- provide a representative analysis of the major potential risks.

The goal of this task is to correctly identify and defend the selection of those compounds responsible for the majority ($\geq$ 95%) of endangerment posed by the site. The selection and ranking of the indicator compounds for the Route 8 Landfill generally follows the procedure outlined in the Superfund Public Health Evaluation Manual (USEPA, 1986a). All worksheets used in the selection of the indicator compounds for the Route 8 Landfill site are provided in Appendix A. Toxicological information about each site-related compound was compiled using information from Appendix C of the Superfund Public Health Evaluation Manual and the US EPA on-line Integrated Risk Information System (IRIS). This information includes toxicologic class [potential carcinogens (PC) or noncarcinogens (NC)]; the severity ratings value for noncarcinogens; the weight-of-evidence ratings for

carcinogens; and toxicity constants for the various environmental media.

The compounds identified at the site were subdivided into potential carcinogens and noncarcinogens. An indicator score (IS), equal to the compound concentration times the toxicity constant (C x T), was calculated for each environmental medium and then summed to yield a total indicator score per compound. This summing of the IS values assumes all environmental media to be equally important. The compounds were then ranked numerically based upon decreasing indicator scores. The top-scoring compounds (based on IS values) were then re-evaluated based upon water solubility , vapor pressure, Henry's law constant, and soil water partition coefficient (K_{OC}, as a general indicator of mobility) to determine the final "indicator compounds". This re-evaluation considers the IS values, but selectively eliminates those compounds which are degradation products of others, have similar physical/compound properties, or have comparable half-lives in the various environmental media. The remaining compounds, generally those with the higher IS values, are the final indicator compounds. Table 3-1 lists the compounds at the site and the justification for their selection/non-selection as indicator compounds.

In a CERCLA risk assessment, the potential exposure point concentrations are expressed only in terms of the indicator compound concentrations during the exposure assessment. However, a more comprehensive and conservative approach is to use the concentrations of similar compounds to represent the effect of chemical group, i.e. the total mass of a chemical group is used as the mass of the indicator compound representing that group. This conservative assumption allows for exposures to entire chemical families to be incorporated in the risk calculations.

TABLE 3-1
INDICATOR JUSTIFICATION

CHEMICAL	*SELECTED*	*JUSTIFICATION*
Arsenic	No	Not found in soils; average concentration below MCL
Barium	No	Average concentration below MCL
Benzene	No	Average concentration below MCL; infrequently detected; ranked low among PCs
Chloroform	No	Average concentration below MCL (total trihalomethanes); infrequently detected
Dibromochloromethane	No	Ranked low among NCs; infrequently detected
1,1-Dichloroethane	Yes	Present in all media; selected to represent ethane compounds
1,1-Dichloroethene	No	Infrequently detected
1,2-Dichloroethane	No	Ranked low among NCs; infrequently detected
1,2-Dichloroethene (trans)	No	Degradation product of trichloroethene
Ethylbenzene	No	Ranked low among NCs; infrequently detected
Heptachlor epoxide	No	Ranked low among PCs; infrequently detected
PCBs	Yes	Present in all media; ranked high among PCs
Toluene	Yes	Present in water and subsurface soil; selected to represent aromatic compounds
1,1,1-Trichloroethane	No	Ranked low among NCs
Trichloroethene	Yes	Ranked high among PCs and NCs; average concentration exceeds MCL
1,1,2,2-Tetrachloroethane	No	Ranked low among NCs and PCs; infrequently detected
Vinyl Chloride	Yes	Class A carcinogen; ranks high among PCs; average concentration exceeds MCL
Xylene	No	Ranked low among NCs; infrequently detected
Zinc	No	Ranked low among NCs; infrequently detected
Phenol	No	Ranked low among NCs; infrequently detected
2-Butanone	No	Ranked low among NCs; infrequently detected
Chlorobenzene	No	Ranked low among NCs; infrequently detected
Lead	No	Average concentration meets MCL
Methylene Chloride	No	Ranked low among NCs; infrequently detected

PC - Potential Carcinogen
NC - Noncarcinogen
MCL - Maximum Contaminant Level (under the Safe Drinking Water Act)

The noncarcinogenic indicator compounds chosen for the Route 8 Landfill are toluene and 1,1-dichloroethane. Toluene represents a family of aromatic compounds, while 1,1-dichloroethane represents the family of ethane compounds. The carcinogenic indicator compounds chosen are trichloroethene, vinyl chloride, and polychlorinated biphenyls (PCBs). Trichloroethene represents the alkene family of compounds. Vinyl chloride was chosen for analysis as a compound of special concern because of its designation as a known human carcinogen. All of the indicator compounds are considered volatile organics except PCBs.

SECTION

4

SECTION 4

EXPOSURE EVALUATION

4.1 Exposure Evaluation

The purpose of an exposure evaluation is to determine the potential for intake of each indicator compound by potentially exposed populations. This involves characterization of the major pathways of contaminant transport leading from the sources on the site to the points of exposure. An exposure pathway is considered complete if it consists of the following four elements:

1) a source of contaminant;
2) a contaminant release pathway;
3) a mode of transport; and
4) human contact at exposure points.

Populations at risk are defined, including environmental populations. Concentrations of the indicator chemicals are determined in each medium to which a population may be exposed. Finally, the intakes of indicator chemicals are calculated.

The Route 8 Landfill exposure evaluation considers the migration of indicator compounds from the site to potentially exposed populations using the following process:

- identifying source areas;
- evaluating important fate and transport processes for the indicator compounds;

- establishing potential exposure pathways for each medium;
- determining exposures to potentially affected populations;
- analyzing concentrations of the indicator compounds at the identified exposure points; and
- calculating potential doses and resultant intakes of the indicator compounds.

4.2 Source Areas

The source areas at the Route 8 Landfill site are described in detail in Section 1. The primary potential routes of indicator compound migration at the site are the following:

- the glacial overburden ground water system, which discharges to the land surface via the Gifford Road Spring and the marsh area, and also to the underlying bedrock aquifer; and

- the bedrock aquifer, in which ground water flows northwesterly, out into the Susquehanna River Valley.

4.3 Fate and Transport Processes

The second step in the analysis of potential exposure is to evaluate the fate and transport processes for the indicator compounds in a qualitative manner. This is done so that the potential for releases from on-site and off-site sources can be considered in the exposure analysis. From this analysis any significant inter-media transport routes can be identified.

Examples of the environmental fates of the Route 8 Landfill indicator compounds include sorption onto soils and sediments, volatilization into the atmosphere, photochemical degradation, and bioaccumulation. Table 4-1 summarizes the physical and

Table 4-1.
PHYSICAL AND CHEMICAL PROPERTIES OF THE INDICATOR CHEMICALS
Amphenol Route 8 Landfill

	Toluene	1,1-Dichloro-ethane	Trichloro-ethene	Vinyl chloride	PCBs
Molecular Weight, g/mol	92	99	131	62.5	328-376
Melting Point, °C	-95	-97.4	-87	-153.8	
Boiling Point, °C	110.6	57.3	86.7	-13.37	385-420
Density, g/mL	0.867	1.174	1.46	0.912	1.3-1.8
PARTITION COEFFICIENT					
Water Solubility, ppm (25 °C)	5.35E+02	5.50E+03	1.10E+03	2.70E+03	3.10E-02
Octanol-Water, log Kow			2.38	1.23	6.04
Sediment-Water, Koc	300	30	126	8.2	5.30E+05
Microorganism-Water, Kb [(ug/g)/(mg/L)]			97	5.7	1.30E+06
VOLATILIZATION COEFFICIENTS					
Henry's Law Constant atm-m3/mol	6.37E-03	4.31E-03	9.10E-03	8.14E-02	7.40E-01
Vapor Pressure, mmHg (25 ° C)	28.1	182	57.9	2660	0.0000405
Reaeration Rate Ratio KvC/Kvo			0.55	0.68	0.35

KEY:
1.00E-03 = 0.001
NAV - not applicable to volatilization calculations
NA - not applicable

References:
Verschueren, K., 1983
Mabey, W.R., et al, 1982

chemical properties of the indicator compounds as defined for the Route 8 Landfill. Physical and chemical constants such as solubility and octanol/water partition coefficient are tabulated so that their importance in affecting fate and mobility of the compounds can be evaluated.

4.3.1 Fate of Indicator Compounds

4.3.1.1 Volatile Organics

The processes influencing the fate of the indicator compounds are evaluated in Table 4-2. Volatilization is the primary fate process for toluene, 1,1-dichloroethane, trichloroethene, and vinyl chloride from surface waters and is estimated in this analysis. However, volatilization of toluene, trichloroethene, and vinyl chloride from marsh area soils is not a factor because these compounds have not been detected in the surface soils; current contamination by these compounds only exists at a depth of about six feet and below. Emission rates for these chemicals through this depth of cover would be very slow, and would represent a relatively insignificant fate process. Only low levels of 1,1-dichloroethane were detected in any surface soils. Volatilization represents the only significant pathway for these compounds, and appears to be important only for surface waters at the Route 8 Landfill.

Of special consideration at the Site are potential degradation processes for the VOCs formed in ground water. Specific degradation products of TCE such as vinyl chloride may ultimately pose a greater risk than the original compound; for example, vinyl chloride is a Class A (known human) carcinogen. While reduction dehalogenation of TCE in the immediate area of the Route 8 Landfill is assumed to have occurred, evidenced both by the presence of vinyl chloride and past disposal of refuse,

Table 4-2.
Relative Importance of Processes Influencing Fate of the Indicator Chemicals at the Amphenol Route 8 Landfill

INDICATOR CHEMICAL	SORPTION	VOLATILIZATION	BIODEGRADATION	PHOTOLYSIS	HYDROLYSIS	BIOACCUMULATION	OXIDATION
Toluene	?	+	?	-	-	-	-
1,1-Dichloroethane	-	+	?	-	-	-	-
Trichloroethene	-	+	?	-	-	-	?
Vinyl Chloride	-	+	?	-	-	-	-
Polychlorinated Biphenyls (PCBs)	+	?	+	?	-	+	?

KEY:
+ Could be important fate process
- Not likely to be an important fate process
? Importance of fate process uncertain or unknown

Reference:
Mills,W.B., et al, 1982.
Callahan, M.A., et al, 1979.

continuing degradation is not expected to occur during transport through the aquifer. TCE, DCE, and VC are therefore expected to migrate through the aquifer at their current relative levels. A discussion of evidence supporting this follows.

The principal VOCs detected in the site ground water are 1,2-dichloroethene (DCE), toluene, 1,1,1-trichloroethane (TCA), vinyl chloride (VC), ethylbenzene, 1,1 dichloroethane (DCA), and trichloroethene (TCE). The ethylbenzene and toluene are non-chlorinated aromatic VOCs which are often used as industrial solvents, but are also present in refined petroleum hydrocarbon products and so may be related to disposal of waste oils. The other compounds are chlorinated aliphatic (or straight chain) hydrocarbons, of which TCE and TCA are very commonly used as degreasers. The other aliphatic compounds have two potential origins:

- impurities in either TCE or TCA; or
- transformation of TCE and/or TCA by reductive dehalogenation in the environment.

The process of reductive dehalogenation occurs when anaerobic microbes, present in the subsurface, biodegrade TCE by "stripping off" chlorine atoms. The breakdown sequence for TCE is:

```
                cis 1,2-DCE
TCE -----> trans 1,2-DCE -----> VC
                1,1-DCE
```

Thus, it can be seen that the DCE compounds are the first intermediate step, and VC is the next. Beyond VC, some limited laboratory data indicate that further breakdown may occur, possibly yielding ethylene or CO_2, and water.

It is interesting to note that the reductive dehalogenation process occurs only by co-metabolism in the presence of a carbon source other than the TCE molecule. The most frequently cited carbon source is also associated with the presence of methanogenic microbes, which are almost always associated with refuse disposal in a landfill. It is ERM's experience that reductive dehalogenation is less of a concern at waste sites without a substantial alternate carbon source, i.e. where refuse disposal has not occurred. The Amphenol Hill Site appears to be a good example, where TCE presently constitutes up to 50 percent of the residual total alkenes, and no VC production has occurred.

4.3.1.2 PCBs

For PCBs, sorption onto soils and sediments is the principal fate process (Table 4-2). Bioaccumulation and biodegradation could be important fate processes for PCBs at the Route 8 site. Volatilization can occur, depending upon exact chemical composition, but is generally a less important fate mechanism for PCBs. At this site, photolysis and hydrolysis fate processes are not environmentally significant pathways for any of the indicator compounds. A more detailed analysis of the environmental fate and transport characteristics of the indicator compounds is given in Appendix B.

4.3.2 Transport of Indicator Compounds

4.3.2.1 Glacial Overburden

The indicator compounds present in the glacial overburden are the volatile compounds and PCBs. Ground water flow in the glacial till and downgradient glacial outwash is a principal migration pathway at the site. Since hydrolysis, sorption, oxidation, and biodegradation are not significant fate process for VOCs, the ground water pathway offers little retardation of these compounds. Sorption and possibly biodegradation are, however, significant fate processes for PCBs. These factors, combined with the low solubility of PCBs in water, greatly retard migration of this compound.

VOCs have migrated vertically through the full thickness of the glacial overburden, and laterally northwestward in the till and outwash until a thick sequence of glaciolacustrine silty clay is encountered. However, ground water flow is expected to be principally within the outwash which is of much higher hydraulic conductivity than the silty clay. The VOC concentrations detected in the till and outwash are in the tens of thousands of parts per billion in the central plume area.

PCBs have migrated principally laterally in a permeable sand outwash lens and sandy till beneath the site, in oil floating on the shallow water table. Some PCBs in oil have migrated vertically into the glacial till beneath the landfill source area, but this migration is limited in depth and extent.

4.3.2.2 Regional Bedrock Aquifer

At the Route 8 Landfill site, VOCs have migrated vertically into the bedrock aquifer via recharging ground water from the

overlying glacial deposits. Concentrations directly downgradient from the source area are in the tens of thousands of parts per billion. Flow in the bedrock is principally through joints and fractures, and the plume is intercepted by an east-west trending fracture zone which supplies water to the Unalam production well. This limits the extent of downgradient migration in the flow system such that the limit of the plume has been defined as the area of the Village Maintenance Shop Well. Concentrations at the Unalam well are in the one to two thousands of parts per billion range. The pumping of the Unalam well, and the relatively flat-bedded nature of the bedrock, have limited vertical migration in the aquifer.

4.3.2.3 Soils

The soils investigation has indicated that both VOCs and PCBs are present in the subsurface soils within, around, and directly beneath the landfill source areas. Downgradient, PCBs, oil and VOCs are present in the soils of the brown sandy till and glacial outwash sand units. The PCB concentrations range from less than 10 to 13,300 ppm, and the VOCs in the thousands of parts per billion. Seasonally the water table rises into these soils. All other subsurface occurrences of VOCs and PCBs are evaluated in relation to ground water flow in the saturated zones.

4.3.2.4 Surface Water

On the south border of the Route 8 Landfill Site, the K-Mart Drain discharges water from the Hill Site with approximately 100 ppb of VOCs to an intermittent drainage way. Downstream near Gifford Road, discharges from the Route 8 Landfill area and Gifford Road Spring increase the VOC concentrations to up to 400 to 500 ppb. North of Gifford Road, in the seasonal marsh area, concentrations of VOCs detected ranged from 7 ppb to 393 ppb. In

the Unalam Tributary, no VOCs were detected upstream of the Unalam cooling water outfall, while 366 ppb were detected downstream of it.

For the most part, PCBs were not detected in the surface waters, except where oil discharges were present. The PCBs in these samples were associated with oil discharges, and ranged from 0.0034 ppm in the marsh area to 0.0042 ppm at the Gifford Road Spring.

4.3.2.5 Drainageway Sediment

Except for one marsh area sediment sample with 32 ppb, no VOCs were detected in any drainageway sediments. However, PCBs were detected in two samples, one in the marsh area at 6.68 ppm and one at the Gifford Road Spring at 2.5 ppm.

4.3.2.6 Air

Because most of the volatile compounds migrate in the subsurface, transport of site-related compounds in the air is very limited. The main potential for air-related exposures is at the surface water bodies in which VOCs have been shown to be present. These VOCs can volatilize from these surface waters into the air. The only other potential air source would be via volatilization from ground water withdrawals. The only withdrawal related to the site is at the Unalam Well, where cooling water is withdrawn. Since the cooling water is used in a closed process in the plant, this potential emission is evaluated at the Unalam Tributary, to which the cooling water discharges.

4.4 Exposure Pathways for Each Medium

Exposure pathways are determined by integrating information from the RI with knowledge about potentially exposed populations and their likely behavior. The mode of potential exposure to the population, such as inhalation, ingestion, or dermal contact, is identified as part of an exposure scenario. Evaluations can then be made regarding the present potential impact on public health, including identification of the potential points of exposure. A summary of the site exposure pathway analysis is presented in Table 4-3.

4.4.1 Identification of Points of Exposure

The potential points of exposure to compounds associated with the Route 8 Landfill are as follows:

1. Surface water exposure at the Gifford Road Spring, the marshes, and the Unalam cooling water discharge.

2. Soils/sediments exposure at the Gifford Road Spring and marsh;

3. Ground water exposure at a hypothetical potable well on River Road; and

4. Exposure to volatiles in the air in the site vicinity.

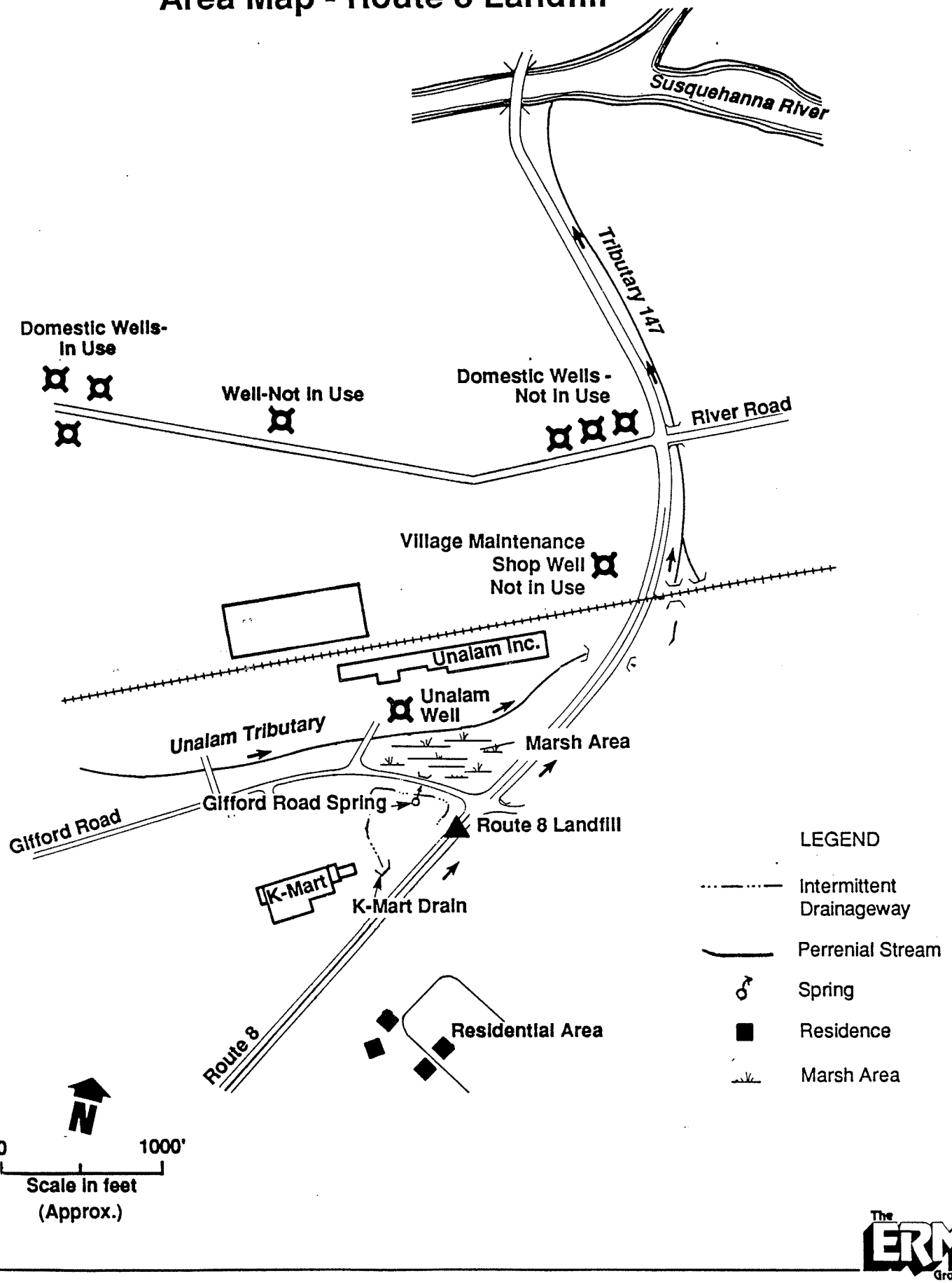
A schematic of the Route 8 Landfill area, with potential exposure points, is shown in Figure 4-1.

TABLE 4-3
EXPOSURE PATHWAY ANALYSIS
ROUTE 8 LANDFILL

MEDIA	*PATHWAY*	*EXPOSURE MECHANISM*	*TYPE OF EXPOSURE*	*EXPOSURE POINT*	*SELECTED FOR ANALYSIS*
Air	From Contaminated Soil	Volatilization	Inhalation	Gifford Road Spring Marsh Area	No - volatiles not present in surface soils
		Fugitive Dust	Inhalation	Gifford Road Spring Marsh Area	No - areas not amenable to soil erosion; highly vegetated
	From Contaminated Surface Water	Volatilization	Inhalation	Gifford Road Spring Marsh Area Unalam Cooling Water Discharge	Yes Yes Yes
Ground Water	Infiltration Through Contaminated Soil	Residential/Industrial Use - Regional Bedrock Aquifer	Ingestion Dermal Contact Inhalation	Hypothetical Potable Well at River Road (no present users)	Yes Yes Yes
		Residential/Industrial Use - Glacial Overburden	Ingestion Dermal Contact Inhalation	No Exposure Point Available	No - no current users, not a viable potable water supply
Surface Water	Runoff	Casual Contact	Dermal Contact	Downgradient of Site	No - no surface runoff occurs
	Groundwater Discharge	Casual Contact	Dermal Contact	Gifford Road Spring Marsh Area Unalam Cooling Water Discharge	Yes Yes Yes
Soils/Sediments	Contaminated Soil	Casual Contact	Dermal Contact	Gifford Road Spring Marsh Area Downslope of Landfill Source Areas	Yes Yes No - all contamination has migrated in subsurface; no surface contamination present
		Pica*	Ingestion	Gifford Road Spring Marsh Area Downslope of Landfill Source Areas	Yes Yes No - all contamination has migrated in subsurface; no surface contamination present

*Pica is defined in this context as the compulsive ingestion of soil

Figure 4-1
Area Map - Route 8 Landfill



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4.5 Determining Exposures to Potentially Affected Populations

The next step in the quantitative determination of the potential exposures is identifying the potential populations which may receive exposure at the exposure points. The additional demographic information needed for this analysis is presented in Section One of this report. Potential receptors include adults and children from numerous residential areas traveling through the indicated areas, most notably to and from the K-Mart shopping area. Such travelers and passers-by have been witnessed during field investigations.

A summary of the potential site related exposures to affected populations analyzed in this assessment is presented in Table 4-4. The potential exposures analyzed include dermal contact with soils and surface water by adults and children who may travel through or by the site on the way to and from the K-Mart Shopping area inhalation by adults and children due to volatilization of chemicals into the air from surface water, and residential drinking water ingestion, dermal contact while bathing, and inhalation via bathing for all age groups at a hypothetical well. Since no actual ground water related exposures are present, this latter exposure is based on hypothetical ground water use for a future potable supply well located at River Road. Additionally, adults may be subject to inhalation exposure to chemicals through volatilization from ground water at the Unalam discharge point. Pica behavior is also assessed, although it represents a highly unlikely scenario. The site-related exposures are discussed separately for media as follows.

Table 4-4.
Amphenol Route 8 Landfill
Routes of Exposures Used to Calculate Intakes
Existing Site Conditions

Exposed Population	Routes of Exposure		
	Dermal	Ingestion	Inhalation
Children ages 2-6	Incidental Soil Contact Bathing	Drinking Water Pica	Via Bathing
Children ages 6-12	Incidental Soil Contact Surface Water Contact Bathing	Drinking Water	Via Surface Water Volatilization Bathing
Adults	Incidental Soil Contact Surface Water Contact Bathing	Drinking Water	Via Surface Water Volatilization Bathing

4.5.1 Glacial Overburden

The glacial overburden is not used for water supply, and generally does not have sufficient yield to serve as a potable water supply. There are no users of the glacial overburden and, therefore, no exposed populations. Water from the glacial overburden, however, discharges seasonally from the Gifford Road Spring and other seasonal seeps south and north of Gifford Road. Potentially exposed populations could include downwind K-Mart patrons and passersby; these exposures are evaluated in this and were discussed previously.

4.5.2 Regional Bedrock Aquifer

The existence of a limited number of private wells northwest of the site on River Road and the potential for future users of this aquifer provide the potential for an exposed population. Unconsolidated valley fill deposits overlie the bedrock aquifer in a downgradient direction. Figure 4-1 illustrates the locations of existing wells downgradient between the Route 8 Landfill Site and the Susquehanna River. The Unalam production well is used for process cooling and is not used by plant personnel for consumption or for other purposes. The next nearest downgradient wells are located along River Road. The four wells which lie most directly along the flow path from the site are not currently in use. Three domestic wells currently in use are located peripheral to the flow direction (Figure 4-1). For the purpose of the exposure analysis, potential future users of wells in the immediate downgradient area are considered to represent the nearest potential receptors, although such a scenario is highly unlikely due to the availability of public water in that area.

4.5.3 Soils/Sediments

Two classes of site "soils" are considered here: surface and subsurface soils at and immediately downslope of the landfill source areas, and soils/sediments in site-related drainageways and marshes. In regard to the first, all site-related compounds have migrated in the subsurface, with no potential for surficial exposures present. With regard to the drainageway related sediments, the only potential exposures are at the Gifford Road Spring and in the marsh to the north of Gifford Road. Potential exposures at those locations would be to occasional passersby, and are evaluated in this assessment.

Pica behavior (defined in this assessment as the persistent and compulsive craving and ingestion of any non-food item, specifically soil) is also evaluated in this assessment for children ages 2-6. The occurrence and characteristics of this behavior are currently highly controversial, with no consensus from the risk community yet available pertaining to the appropriateness of including it in an assessment such as this one. Areas of surface soil contamination at the Route 8 Landfill are nonresidential, not adjacent to residential areas, and access is expected to be difficult for this age group which is normally well-supervised. It is evaluated in this assessment to represent a worst case scenario both in terms of the occurrence of this behavior and as a severe upper-limit estimation of potential incidental ingestion of soils and household dust.

4.5.4 Surface Water

Some low parts per billion levels of VOCs and PCBs are present in the discharges of the Gifford Road Spring and related seasonal seepages which discharge to the marsh. In addition, the site investigation has shown that the discharge of the Unalam cooling

water to the Unalam tributary contributes VOCs to that stream. Populations at potential risk include occasional passersby at the Gifford Road Spring, marshes, and other seeps, and are evaluated in this assessment.

4.5.5 Air

Volatilization of VOCs from the site-related surface water may occur at the Gifford Road Spring, marshes, and Unalam Tributary discharge. Populations potentially exposed include workers, passersby, and residents in downwind directions.

4.6 Analysis of Potential Exposure Point Concentrations

The exposure point concentrations are determined once the exposure scenarios and potentially affected populations are identified. If the transport of compounds associated with the site is under steady-state conditions, monitoring data are adequate to determine potential exposure concentrations. If no data are available or if conditions are transient (such as a migrating plume in ground water), models are used to predict concentrations. Ground water transport through advection and dispersion is described in Section 1 of this report. Transport in ground water and other media, such as surface water and the atmosphere, may require modeling assessments to determine long-term potential exposures. Many factors, including the fate and transport processes reviewed previously, must be considered when selecting the most appropriate model.

The maximum and mean concentrations for each indicator compound in each medium at each exposure point have been determined for the Route 8 Landfill. The values calculated are presented in Table 4-5. The analysis for each exposure point is presented as follows.

Table 4-5
Potential Exposures for Ambient Conditions
Amphenol Route 8 Landfill
Exposure Point Concentrations
Children Ages 2-6

Exposure Media	Exposure Area	Route of Exposure	Indicator Compound	Maximum Concentration	Mean Concentration
Soil/Sediment	Gifford Road Spring	Dermal Contact (mg/kg)	Toluene	NA	NA
			1,1-Dichloroethane	NA	NA
			Trichloroethene	NA	NA
			Vinyl chloride	NA	NA
			PCBs	2.50E+00	1.17E+00
		Pica Ingestion (mg/kg)	Toluene	NA	NA
			1,1-Dichloroethane	NA	NA
			Trichloroethene	NA	NA
			Vinyl chloride	NA	NA
			PCBs	2.50E+00	1.17E+00
	Marsh Area	Dermal Contact (mg/kg)	Toluene	NA	NA
			1,1-Dichloroethane	NA	NA
			Trichloroethene	NA	NA
			Vinyl chloride	NA	NA
			PCBs	6.68E+00	1.19E+00
		Pica Ingestion (mg/kg)	Toluene	NA	NA
			1,1-Dichloroethane	NA	NA
			Trichloroethene	NA	NA
			Vinyl chloride	NA	NA
			PCBs	6.68E+00	1.19E+00
Ground Water	Potable Well	Dermal Contact Bathing (mg/L)	Toluene	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05
			Trichloroethene	9.35E-05	3.00E-05
			Vinyl chloride	7.50E-05	7.50E-05
			PCBs	ND	ND
		Ingestion (mg/L)	Toluene	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05
			Trichloroethene	9.35E-05	3.00E-05
			Vinyl chloride	7.50E-05	7.50E-05
			PCBs	ND	ND
		Inhalation (mg/L)	Toluene	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05
			Trichloroethene	9.35E-05	3.00E-05
			Vinyl chloride	7.50E-05	7.50E-05
			PCBs	ND	ND

KEY
ND - Not detected
NA - Not analyzed

Table 4-5 (Continued)
Potential Exposures for Ambient Conditions
Amphenol Route 8 Landfill
Exposure Point Concentrations
Children Ages 6-12

Exposure Media	Exposure Area	Route of Exposure	Indicator Compound	Maximum Concentration	Mean Concentration
Surface Water	Gifford Road Spring	Dermal Contact (mg/L)	Toluene	ND	ND
			1,1-Dichloroethane	1.54E-01	2.78E-02
			Trichloroethene	5.60E-02	4.10E-03
			Vinyl chloride	2.00E-02	7.70E-03
			PCBs	4.20E-03	4.20E-03
		Inhalation (mg/m3)	Toluene	ND	ND
			1,1-Dichloroethane	1.50E-04	9.03E-06
			Trichloroethene	5.07E-05	1.24E-06
			Vinyl chloride	2.48E-05	3.19E-06
			PCBs	2.32E-06	7.75E-07
	Marsh Area	Dermal Contact (mg/L)	Toluene	1.00E-02	6.90E-04
			1,1-Dichloroethane	2.30E-01	2.39E-02
			Trichloroethene	2.43E-01	1.23E-02
			Vinyl chloride	3.20E-02	6.55E-03
			PCBs	3.40E-03	1.18E-03
		Inhalation (mg/m3)	Toluene	1.90E-03	4.36E-05
			1,1-Dichloroethane	4.00E-02	1.39E-03
			Trichloroethene	3.93E-02	6.62E-04
			Vinyl chloride	7.09E-03	4.84E-04
			PCBs	3.36E-04	3.89E-05
	Unalam Cooling Discharge	Dermal Contact (mg/L)	Toluene	4.00E-01	2.81E-02
			1,1-Dichloroethane	1.90E-01	5.20E-02
			Trichloroethene	1.90E+00	3.40E-01
			Vinyl chloride	3.70E-01	3.10E-01
			PCBs	ND	ND
		Inhalation (mg/m3)	Toluene	8.43E-06	1.97E-07
			1,1-Dichloroethane	3.67E-06	3.35E-07
			Trichloroethene	3.41E-05	2.03E-06
			Vinyl chloride	9.11E-06	2.54E-06
			PCBs	ND	ND
Soil/Sediment	Gifford Road Spring	Dermal Contact (mg/kg)	Toluene	NA	NA
			1,1-Dichloroethane	NA	NA
			Trichloroethene	NA	NA
			Vinyl chloride	NA	NA
			PCBs	2.50E+00	1.17E+00
	Marsh Area	Dermal Contact (mg/kg)	Toluene	NA	NA
			1,1-Dichloroethane	NA	NA
			Trichloroethene	NA	NA
			Vinyl chloride	NA	NA
			PCBs	6.68E+00	1.19E+00
Ground Water	Potable Well	Dermal Contact Bathing (mg/L)	Toluene	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05
			Trichloroethene	9.35E-05	3.00E-05
			Vinyl chloride	7.50E-05	7.50E-05
			PCBs	ND	ND
		Ingestion (mg/L)	Toluene	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05
			Trichloroethene	9.35E-05	3.00E-05
			Vinyl chloride	7.50E-05	7.50E-05
			PCBs	ND	ND
		Inhalation (mg/L)	Toluene	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05
			Trichloroethene	9.35E-05	3.00E-05
			Vinyl chloride	7.50E-05	7.50E-05
			PCBs	ND	ND

KEY
ND - Not detected
NA - Not analyzed

Table 4-5 (Continued)
Potential Exposures for Ambient Conditions
Amphenol Route 8 Landfill
Exposure Point Concentrations
Adults

Exposure Media	Exposure Area	Route of Exposure	Indicator Compound	Maximum Concentration	Mean Concentration
Surface Water	Gifford Road Spring	Dermal Contact (mg/L)	Toluene	ND	ND
			1,1-Dichloroethane	1.54E-01	2.78E-02
			Trichloroethene	5.60E-02	4.10E-03
			Vinyl chloride	2.00E-02	7.70E-03
			PCBs	4.20E-03	4.20E-03
		Inhalation (mg/m3)	Toluene	ND	ND
			1,1-Dichloroethane	1.50E-04	9.03E-06
			Trichloroethene	5.07E-05	1.24E-06
			Vinyl chloride	2.48E-05	3.19E-06
			PCBs	2.32E-06	7.75E-07
	Marsh Area	Dermal Contact (mg/L)	Toluene	1.00E-02	6.90E-04
			1,1-Dichloroethane	2.30E-01	2.39E-02
			Trichloroethene	2.43E-01	1.23E-02
			Vinyl chloride	3.20E-02	6.55E-03
			PCBs	3.40E-03	1.18E-03
		Inhalation (mg/m3)	Toluene	1.90E-03	4.36E-05
			1,1-Dichloroethane	4.00E-02	1.39E-03
			Trichloroethene	3.93E-02	6.62E-04
			Vinyl chloride	7.09E-03	4.84E-04
			PCBs	3.36E-04	3.89E-05
	Unalam Cooling Discharge	Dermal Contact (mg/L)	Toluene	4.00E-01	2.81E-02
			1,1-Dichloroethane	1.90E-01	5.20E-02
			Trichloroethene	1.90E+00	3.40E-01
			Vinyl chloride	3.70E-01	3.10E-01
			PCBs	ND	ND
		Inhalation (mg/m3)	Toluene	8.43E-06	1.97E-07
			1,1-Dichloroethane	3.67E-06	3.35E-07
			Trichloroethene	3.41E-05	2.03E-06
			Vinyl chloride	9.11E-06	2.54E-06
			PCBs	ND	ND
Soil/Sediment	Gifford Road Spring	Dermal Contact (mg/kg)	Toluene	NA	NA
			1,1-Dichloroethane	NA	NA
			Trichloroethene	NA	NA
			Vinyl chloride	NA	NA
			PCBs	2.50E+00	1.17E+00
	Marsh Area	Dermal Contact (mg/kg)	Toluene	NA	NA
			1,1-Dichloroethane	NA	NA
			Trichloroethene	NA	NA
			Vinyl chloride	NA	NA
			PCBs	6.68E+00	1.19E+00
Ground Water	Potable Well	Dermal Contact Bathing (mg/L)	Toluene	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05
			Trichloroethene	9.35E-05	3.00E-05
			Vinyl chloride	7.50E-05	7.50E-05
			PCBs	ND	ND
		Ingestion (mg/L)	Toluene	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05
			Trichloroethene	9.35E-05	3.00E-05
			Vinyl chloride	7.50E-05	7.50E-05
			PCBs	ND	ND
		Inhalation (mg/L)	Toluene	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05
			Trichloroethene	9.35E-05	3.00E-05
			Vinyl chloride	7.50E-05	7.50E-05
			PCBs	ND	ND

KEY
ND - Not detected
NA - Not analyzed

4.6.1 Glacial Overburden

The glacial overburden is the medium through which the compounds of concern have migrated to potential exposure points at the Gifford Road Spring, other seasonal seeps, and marshes. Since these discharges are actually surface water exposure points, the glacial overburden is not considered further. Rather, the discharges from it are considered to be surface water exposures. Exposure point concentrations have been determined using the results of hydrogeologic study analyses. The highest VOC concentrations discharging from the glacial overburden were 486 ppb near the Gifford Road Spring and 393 ppb in the marsh north of Gifford Road. PCBs of 4.2 and 3.4 ppb were detected at the spring and marsh, respectively. The indicator compound concentrations used are developed under the surface water discussion in Section 4.6.4.

4.6.2 Bedrock Aquifer

The regional bedrock aquifer is the aquifer of primary concern. Samples were taken by the New York State Department of Health at the accessible off-gradient domestic wells on River Road. Those samples contained no site-related compounds. The migration of volatile organics in the bedrock flow system has been defined by the ERM analyses from on-site and off-site monitoring wells. The data indicate that the VOC plume is mostly captured by pumping at the Unalam cooling water well. Along its downgradient axis, the plume is reduced to 14 ppb total VOCs at the Village Maintenance Shop Well. Potential exposure point concentrations for hypothetical future wells using the bedrock aquifer at River Road have been calculated using the results of the RI analyses.

Concentrations at potential downgradient exposure points on River Road were calculated based on application of an assumed first order decay with distance. More complex computer models (such as the VHS model) were considered but not applied because EPA does not accept them as valid in bedrock where flow is anistropic along zones of fracturing. Thus, a simple dilution/reduction between two wells in the contamination plume was determined for each indicator compound at the Route 8 Landfill site.

The two wells within the plume used to determine this simple dilution factor were R8-33 and the Village Maintenance Shop Well (VMSW) (see Figure 1-12). The factor between these two wells on the axis of the VOC plume is calculated as follows:

$$di = \frac{\text{concentration of component i at Well R8-33}}{\text{concentration of component i at VMSW}}$$

The intervening distance between these wells is approximately 600 feet. The distance from the Village Maintenance Shop Well to the nearest potential receptor on River Road is approximately 800 feet. Therefore, the amount of dilution expected between the VWSW and the receptor location would be di x (800/600). The calculated dilution factors and resulting concentrations are presented in Table 4-6. The potential maximum concentrations at the River Road wells, based on assumed linear reduction, are not detectable for toluene (representing aromatic compounds), 0.06 ppb for 1,1-dichloroethane (alkane compounds), 0.094 ppb for trichloroethene (alkene compounds), 0.075 ppb for vinyl chloride, and not detectable for PCBs. These estimates do not take into account additional dilution by recharge from the prolific valley train glacial aquifer in the Susquehanna River Valley. Therefore, the actual potential exposure concentrations at River Road would be even lower.

Table 4-6
Amphenol Route 8 Landfill
Dilution Calculations to River Road Well in Bedrock Aquifer

Bedrock Aquifer Concentrations

Indicator	R8-33 Well Concentrations		Village Maintenance Shop Well Concentrations		Projected Concentrations at a Well on River Road	
	Maximum (mg/L)	Mean (mg/L)	Maximum (mg/L)	Mean (mg/L)	Maximum (mg/L)	Mean (mg/L)
Toluene	ND	ND	ND	ND	ND	ND
1,1-Dichloroethane	5.00E-02	1.29E-02	2.00E-03	8.75E-04	6.00E-05	4.38E-05
Trichloroethene	6.50E-01	1.56E-01	9.00E-03	2.50E-03	9.35E-05	3.00E-05
Vinyl Chloride	1.00E-02	NC*	1.00E-03	NC*	7.50E-05	NC*
PCBs	ND	ND	ND	ND	ND	ND

Calculated Dilution Factors

Indicator	Dilution Factor Between R8-33 and VMSW		Dilution Factor Between VMSW and River Road	
	Maximum	Mean	Maximum	Mean
Toluene	N/A	N/A	N/A	N/A
1,1-Dichloroethane	25	15	33.3	20
Trichloroethene	72.2	62.4	96.3	83.2
Vinyl Chloride	10	NC*	13.3	NC*
PCBs	N/A	N/A	N/A	N/A

KEY:
ND - Not detected
N/A - Not applicable
NC* - Not calculated because only one analysis was performed.

The potential exposures from ground water use are ingestion of drinking water, dermal exposure due to bathing, and inhalation of volatiles while bathing.

4.6.3 Soils/Sediments

Potential for direct contact with PCBs in the surface soils/sediments at the site is limited to two areas, the Gifford Road Spring where the maximum detected was 2.5 ppm, and in the marsh where the maximum detected was 6.68 ppm. The Gifford Road Spring sample results and the GRS4 sample results dated 7/19/84 presented in Table 1-6 were used to determine the maximum and mean concentrations for that area. Sample results from locations GRN1, GRN2, GRN4, GRN5, GRS4 (results from 9/12/85 only), GRS6, and GRS7, presented in Table 1-6, were used to determine the maximum and mean concentrations for the marshes. When the indicator compounds were not detected in a sample, one half the detection limit of 1000 ug/kg was assumed present to determine the mean concentration (i.e. 500 ug/kg was added for an ND). This assumption is conservative, as it accounts for the probability that the compound may actually be present in the media but below the detection limit in some of the samples.

Emissions of PCBs from the soils/sediments were not calculated. The low Henry's law constant for that family of compounds and low media concentrations would result in exposure levels that are orders of magnitude smaller than those due to direct contact with the soils/sediments. The VOC analysis results for soil/sediment samples were much lower than the corresponding surface water concentrations, and so were not considered.

Dermal exposures to soils and sediments is limited to the occasional passerby who is estimated to have approximately 1 hour of contact with the PCB-containing soil 10 days per year.

4.6.4 Surface Waters

The surface water data considered are those from the Gifford Road Spring, the marsh, and the Unalam Tributary at the cooling water discharge. The surface water exposure point concentrations were determined using data from the RI, as presented in Table 1-5.

For the VOCs at the Gifford Road Spring, five samples were available for consideration: the four results labelled Gifford Road Spring, and the GRS4 result dated 7/19/84. The indicator maximum concentrations were not detected for toluene, 154 ppb for 1,1-dichloroethane, 56 ppb for trichloroethene, and 20 ppb for vinyl chloride. Analysis for PCBs was conducted once for the Gifford Road Spring samples; that concentration was 4.2 ppb. When VOCs were reported as not detected (ND), one half the detection limit, or 0.5 ppb, was assumed present to determine mean concentrations for indicator compounds that were detected in at least one sample.

The sample results from locations GRN1, GRN2, GRN3, GRN4, GRN5, GRS4, GRS6, and GRS7 (presented in Table 1-5) were used to calculate the maximum and mean exposure point concentrations for the marsh area. For the VOCs, the maximum concentrations were 10 ppb for toluene, 230 ppb for 1,1-dichloroethane, 243 ppb for trichloroethene, and 32 ppb for vinyl chloride. The maximum concentration for PCBs was 3.4 ppb. When calculating mean exposure point concentrations, one half the detection limit was assumed present when a compound was reported as ND. This value is 0.5 ppb for VOCs, and 1.0 ppb for PCBs.

The concentrations of indicators at the Unalam cooling water discharge were determined from the Unalam Well monitoring data (presented in Table 1-7). This data was used because no direct

sampling data was available for the discharge. The maximum exposure concentrations are 400 ppb for toluene, 190 ppb for 1,1-dichloroethane, 1900 ppb for trichloroethene, and 370 ppb for vinyl chloride.

The potential for exposure to other surface water due to dermal contact is limited to the Gifford Road Spring and marsh areas. It is assumed that an occasional passerby may be exposed for a 1-hour event approximately 10 days a year. Inhalation exposures due to volatilization of indicator compounds have also been analyzed. These potential exposures are discussed further under Section 4.6.6.

4.6.5 Air

Potential exposure point concentrations via air were estimated for the Gifford Road Spring, the marshes, and the Unalam cooling water discharge. Air emissions were calculated using methodology presented in the Superfund Exposure Assessment Manual (US EPA, 1988) for estimating volatilization releases of low solubility compounds from waterbodies. The calculations for emission rates from Gifford Road Spring, the marsh, and the Unalam cooling water discharge are presented in Table 4-7. The three surface waters were modeled as area sources into a "box" over the Gifford Road/marsh area (Schlesinger, et al., 1987). While the accuracy of box models in predicting near-source contaminant concentrations in air is not established, these models represent the best tools currently available. Of the wide range of box models in use, the one selected is judged to be one of the most applicable due to its use of site-specific physical parameters to estimate box size. The calculations of the volatilized air concentrations are presented in Table 4-8.

Table 4-7
Amphenol Route 8 Landfill
Surface Water Emission Calculations

Gifford Road Spring

Indicator Compound	Maximum concentration (mg/L)	Mean concentration (mg/L)	Overall Mass transfer coef. Ki (cm/sec)	Area of source (cm2)	Subchronic Emissions Rate (g/sec)	Chronic Emissions Rate (g/sec)
Toluene	ND	ND	6.66E-03	1.17E+06	ND	ND
1,1-Dichloroethane*	0.154	0.0278	6.11E-03	1.17E+06	1.10E-03	1.99E-04
Trichloroethene	0.056	0.0041	5.67E-03	1.17E+06	3.71E-04	2.72E-05
Vinyl chloride	0.02	0.0077	7.78E-03	1.17E+06	1.82E-04	7.01E-05
PCBs	0.0042	0.0042	3.47E-03	1.17E+06	1.71E-05	1.71E-05

Marsh Area

Indicator Compound	Maximum concentration (mg/L)	Mean concentration (mg/L)	Overall Mass transfer coef. Ki (cm/sec)	Area of source (cm2)	Subchronic Emissions Rate (g/sec)	Chronic Emissions Rate (g/sec)
Toluene	0.01	0.00069	6.66E-03	2.09E+08	1.39E-02	9.60E-04
1,1-Dichloroethane*	0.23	0.0239	6.11E-03	2.09E+08	2.94E-01	3.05E-02
Trichloroethene	0.243	0.0123	5.67E-03	2.09E+08	2.88E-01	1.46E-02
Vinyl chloride	0.032	0.00655	7.78E-03	2.09E+08	5.20E-02	1.07E-02
PCBs	0.0034	0.00118	3.47E-03	2.09E+08	2.47E-03	8.56E-04

Unalam Cooling Water Discharge

Indicator Compound	Maximum concentration (mg/L)	Mean concentration (mg/L)	Overall Mass transfer coef. Ki (cm/sec)	Area of source (cm2)	Subchronic Emissions Rate (g/sec)	Chronic Emissions Rate (g/sec)
Toluene	0.4	0.0281	6.66E-03	2.32E+04	6.18E-05	4.34E-06
1,1-Dichloroethane*	0.19	0.052	6.11E-03	2.32E+04	2.69E-05	7.37E-06
Trichloroethene	1.9	0.34	5.67E-03	2.32E+04	2.50E-04	4.47E-05
Vinyl chloride	0.37	0.31	7.78E-03	2.32E+04	6.68E-05	5.60E-05
PCBs	<0.001	<0.001	3.47E-03	2.32E+04	N/A	N/A

The equation used to calculate emissions rates: Ei=(Ki*Concentration*A)/1.0E+6
Ki's were taken from Lyman, 1982.
*The Ki for 1,1-Dichloroethane was not available and was estimated from the value for ethyl bromine.

KEY:
ND - Not detected
N/A - Not applicable

Table 4-8
Amphenol Route 8 Landfill
Air Concentrations from Surface Water Emissions

Gifford Road Spring

Indicator Compound	Subchronic Emission Rate (g/sec)	Chronic Emission Rate (g/sec)	Modeled Height of Box (m)	Width of Box (m)	Subchronic Windspeed (m/sec)	Chronic Windspeed (m/sec)	**Subchronic Concentration (g/m3)**	**Chronic Concentration (g/m3)**
Toluene	ND	ND	56.42	130	1	3	ND	ND
1,1-Dichloroethane	1.10E-03	1.99E-04	56.42	130	1	3	1.50E-07	9.03E-09
Trichloroethene	3.71E-04	2.72E-05	56.42	130	1	3	5.07E-08	1.24E-09
Vinyl chloride	1.82E-04	7.01E-05	56.42	130	1	3	2.48E-08	3.19E-09
PCBs	1.71E-05	1.71E-05	56.42	130	1	3	2.32E-09	7.75E-10

Marsh Area

Indicator Compound	Subchronic Emission Rate (g/sec)	Chronic Emission Rate (g/sec)	Modeled Height of Box (m)	Width of Box (m)	Subchronic Windspeed (m/sec)	Chronic Windspeed (m/sec)	**Subchronic Concentration (g/m3)**	**Chronic Concentration (g/m3)**
Toluene	1.39E-02	9.60E-04	56.42	130	1	3	1.90E-06	4.36E-08
1,1-Dichloroethane	2.94E-01	3.05E-02	56.42	130	1	3	4.00E-05	1.39E-06
Trichloroethene	2.88E-01	1.46E-02	56.42	130	1	3	3.93E-05	6.62E-07
Vinyl chloride	5.20E-02	1.07E-02	56.42	130	1	3	7.09E-06	4.84E-07
PCBs	2.47E-03	8.56E-04	56.42	130	1	3	3.36E-07	3.89E-08

Unalam Cooling Water Discharge

Indicator Compound	Subchronic Emission Rate (g/sec)	Chronic Emission Rate (g/sec)	Modeled Height of Box (m)	Width of Box (m)	Subchronic Windspeed (m/sec)	Chronic Windspeed (m/sec)	**Subchronic Concentration (g/m3)**	**Chronic Concentration (g/m3)**
Toluene	6.18E-05	4.34E-06	56.42	130	1	3	8.43E-09	1.97E-10
1,1-Dichloroethane	2.69E-05	7.37E-06	56.42	130	1	3	3.67E-09	3.35E-10
Trichloroethene	2.50E-04	4.47E-05	56.42	130	1	3	3.41E-08	2.03E-09
Vinyl chloride	6.68E-05	5.60E-05	56.42	130	1	3	9.11E-09	2.54E-09
PCBs	N/A	N/A	56.42	130	1	3	N/A	N/A

The equation used to calculate air concentrations is as follows:

$Xi=Ei/(H*W*\mu m)$

Where:
Xi - Concentration of contaminant in air
Ei - Emission rate
H - Height of box
W - Width of box
μm - Average wind speed through the box

KEY:
ND - Not Detected
N/A - Not Applicable

The inhalation exposures to compounds that volatilized into the air were analyzed for occasional passersby, assuming inhalation of air for 1-hour, 10 times a year.

4.7 Exposure and Daily Intake Calculations

4.7.1 Methods

The resultant doses and potential intakes of potentially exposed populations are calculated once the exposure concentrations in all media have been determined. Dose is defined as the amount of chemical contacting body boundaries (skin, lungs, or gastrointestinal tract), and intake is the amount of chemical absorbed by the body.

For the Route 8 Landfill assessment, three potential routes of exposure are applicable: ingestion, inhalation of volatilized compounds, and dermal exposure. Calculations were made for each exposure mechanism in accordance with available, applicable guidance. Both doses and daily intakes are expressed in terms of mass per unit of body mass per day, by dividing daily exposures by an average assumed body mass of 70 kg for adults, 29 kg for children aged 6-12, and 16 kg for children aged 2-6. Parameters used to calculate doses and intakes are given in Table 4-9.

Both subchronic and chronic intakes have been calculated. The Subchronic Daily Intake (SDI) is the projected human intake averaged over the short-term period (10 to 90 days), and is calculated by multiplying peak concentrations by the human intake and body weight factors. Chronic Daily Intake (CDI) is the projected human intake over the long-term period (70 years), and is calculated by multiplying average concentrations by the human intake and body weight factors. Table 4-10 lists the

TABLE 4-9
AMPHENOL ROUTE 8
STANDARD PARAMETERS FOR CALCULATION OF DOSAGE AND INTAKE

		Adult	Child Age 6-12	Child Age 2-6	Uncertainty
PHYSICAL CHARACTERISTICS					
Average Body Weight	(a)	70 kg	29 kg	16 kg	
Average Skin Surface Area	(a)	18,150 cm2	10,470 cm2	6980 cm2	
ACTIVITY CHARACTERISTICS					
Amount of Water Ingested Daily	(f)	2 liters	2 liters	2 liters	2 liters is for adult male; consumption for other populations is less
Soil Ingested (Pica)	(f)			0.1 g	Forthcoming study results should refine
Frequency of Soil Contact	(d)	1 hr/d for 10 d/yr	1 hr/d for 10 d/yr	1 hr/d for 10 d/yr	Adjusted for site conditions
Percentage of Skin Surface Area Contacted by Soils	(d)	20%	20%	20%	Forearms and hands are exposed
Skin Absorption Rate of Compounds in Soil	(c)	6%	12%	12%	Absorption over a 12-hr period, should not be adjusted at this time
Frequency of Surface Water Contact (Casual)	(d)	10 hrs/year	10 hrs/year		Based on assumption that all outdoor activities are surface water recreation
Duration of Surface Water Contact (Casual)	(d)	1 hr/day	1 hr/day		Adjusted for site conditions Adjusted for site conditions
Percentage of Skin Surface Area Immersed	(d)	18%	16%		Lower legs and hands are exposed
Length of Time Spent Bathing	(b)	20 min	20 min	20 min	Current activity data suggest 5 min.; probable overestimate
Absorption Rate of Inhaled Air	(d)	100	100	100	
Aveolar (Inhalation Rate)	(d)	0.83 m3/hr	0.46 m3/hr	0.25 m3/hr	Valid for volatiles only
Length of Additional Exposure After Bathing	(b)	10 min	10 min	10 min	
Percentage of Skin Surface Area Immersed While Bathing	(g)	100%	100%	100%	
Volume of Showerstall	(b)	3 m3	3 m3	3 m3	
Volume of Bathroom	(b)	10 m3	10 m3	10 m3	
Volume of Water Used While Showering	(b)	200 liters	200 liters	200 liters	
MATERIAL CHARACTERISTICS					
Dust Adherence	(e)	0.51 mg/cm2			
Soil Matrix Effect	(c)	15%	15%	15%	Based on TCDD; will depend upon properties of contaminant
Mass Flux Rate (water-based)	(g)	0.5 mg/cm2/hr			Chemical-specific permeability data is available for very few chemicals

a - Anderson, E., Browne, N., Duletsky, S., Warn, T., "Development of Statistical Distributions or Ranges of Standard Factors Used in Exposure Assessments", PB 85-242667/AS, US EPA, Office of Health and Environmental Assessment, 1984.
b - K.G. Symms, "An Approximation of the Inhalation Exposure to Volatile Synthetic Organic Chemicals from Showering with Contaminated Household Water ", Paper presented at the Symposium of American College of Toxicologists, Nov. 15, 1986
c - J.K. Hawley, "Assessment of Health Risk from Exposure to Contaminated Soil",. Risk Analysis, Vol. 5, No. 4, 1985
d - ERM Staff Professional Judgement
e - Lepow, M.L., Bruckman, L., Gillette, M., Markowitz, S., Robino, R., Kapish, J., "Investigations into Sources of Lead in the Environment of Urban Children", Environmental Research 10:415-426, 1975, and
Lepow, M.L., Bruckman, M., Robino, L., Markowitz, S., Gillette, R., Kapish, J., "Role of Airborne Lead in Increased Body Burden of Lead in Hartford Children", Environmental Health Perspectives 6:99-101, 1974
f - Superfund Public Health Evaluation Manual
g - Superfund Exposure Assessment Manual
h - Kimbrough R, Falk H, Stemr P, Fries G. 1984. "Health implications of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) contamination of residential soil", Journal of Toxicology and Environmental Health 14:47-93.

Table 4-10
Amphenol Route 8 Landfill
Characteristics of Subchronic/Chronic Exposure Scenarios
Existing Site Conditions

Route of Exposure	Media	Activity	Population	Subchronic Exposure Characteristics	Chronic Exposure Characteristics
Dermal	Soil/Sediments	Casual Contact	Children ages 2-6 Children ages 6-12 Adult	1 exposure event (hands only) per day at highest concentration	1 exposure event (hands only) per day, 10 days per year, at average concentration
	Surface Water	Casual Contact	Children ages 6-12 Adult	1 hour per day at highest concentration	1 hour of exposure (18% of body), 10 hours per year, at average concentration
	Ground Water	Bathing (hypothetical)	Children ages 2-6 Children ages 6-12 Adult	1, 20-minute exposure per day at the highest concentration	1, 20-minute exposure event, 365 days per year, at average concentration
Ingestion	Ground Water	Drinking Water (hypothetical)	Children ages 2-6 Children ages 6-12 Adult	2 liters per day at highest concentration; 75% of ingestion occurs at home	2 liters per day, 365 days per year, at average concentration; 75% of ingestion occurs at home
	Soil/Sediments	Casual Contact (Pica soil ingestion)	Children age 2-6	1 hour of exposure per day at highest concentration	1 hour of exposure per day, 10 days per year at average concentration
Inhalation	Surface Water Emission	Gifford Road Area	Children age 2-6 Children age 6-12 Adult	1 hour of exposure per day within a 162m x130m area at the site at the highest emission rate	10, 1-hour exposures within a 162m x130m area at the site per year at the average emission rate
	Ground Water	Showering/ Bathing (hypothetical)	Children age 2-6 Children age 6-12 Adult	30 minutes per day at highest concentration	30 minutes of exposure per day, 365 days per year, at average concentration

characteristics used to calculate the subchronic and chronic doses and intakes.

The calculation methods used to determine dermal, ingestion, and inhalation intakes were presented in Section 2; sample calculations are given in Appendix C. The resultant intakes are then used in the risk characterization process. For carcinogens, the CDI values are used to assess carcinogenic risk. For noncarcinogens the SDI and CDI values are used to evaluate subchronic and chronic effects, respectively.

The subchronic and chronic intakes calculated for the Route 8 Landfill are presented in Table 4-11.

Table 4-11
Potential Exposures for Ambient Conditions
Amphenol Route 8 Landfill
Exposure Point Concentrations and Calculated Intakes
Children Ages 2-6

Exposure Media	Exposure Area	Route of Exposure	Indicator Compound	Maximum Concentration	Mean Concentration	Subchronic Intakes (mg/kg/day)	Lifetime Adjusted Chronic Daily Intakes (mg/kg/day)
Soil/Sediment	Gifford Road Spring	Dermal Contact (mg/kg)	Toluene	NA	NA	NA	NA
			1,1-Dichloroethane	NA	NA	NA	NA
			Trichloroethene	NA	NA	NA	NA
			Vinyl chloride	NA	NA	NA	NA
			PCBs	2.50E+00	1.17E+00	2.01E-06	1.51E-09
		Pica Ingestion (mg/kg)	Toluene	NA	NA	NA	NA
			1,1-Dichloroethane	NA	NA	NA	NA
			Trichloroethene	NA	NA	NA	NA
			Vinyl chloride	NA	NA	NA	NA
			PCBs	2.50E+00	1.17E+00	1.56E-05	1.18E-08
	Marsh Area	Dermal Contact (mg/kg)	Toluene	NA	NA	NA	NA
			1,1-Dichloroethane	NA	NA	NA	NA
			Trichloroethene	NA	NA	NA	NA
			Vinyl chloride	NA	NA	NA	NA
			PCBs	6.68E+00	1.19E+00	5.36E-06	1.54E-09
		Pica Ingestion (mg/kg)	Toluene	NA	NA	NA	NA
			1,1-Dichloroethane	NA	NA	NA	NA
			Trichloroethene	NA	NA	NA	NA
			Vinyl chloride	NA	NA	NA	NA
			PCBs	6.68E+00	1.19E+00	4.17E-05	1.20E-08
Ground Water	Potable Well	Dermal Contact Bathing (mg/L)	Toluene	ND	ND	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05	4.32E-09	1.86E-10
			Trichloroethene	9.35E-05	3.00E-05	6.73E-09	1.27E-10
			Vinyl chloride	7.50E-05	7.50E-05	5.40E-09	3.18E-10
			PCBs	ND	ND	ND	ND
		Ingestion (mg/L)	Toluene	ND	ND	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05	5.64E-06	2.42E-07
			Trichloroethene	9.35E-05	3.00E-05	8.79E-06	1.66E-07
			Vinyl chloride	7.50E-05	7.50E-05	7.05E-06	4.15E-07
			PCBs	ND	ND	ND	ND
		Inhalation (mg/L)	Toluene	ND	ND	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05	1.32E-05	5.67E-07
			Trichloroethene	9.35E-05	3.00E-05	2.06E-05	3.88E-07
			Vinyl chloride	7.50E-05	7.50E-05	1.65E-05	9.71E-07
			PCBs	ND	ND	ND	ND

KEY
ND - Not detected
NA - Not analyzed

Table 4-11 (Continued)
Potential Exposures for Ambient Conditions
Exposure Point Concentrations and Calculated Intakes
Children Ages 6-12

Exposure Media	Exposure Area	Route of Exposure	Indicator Compound	Maximum Concentration	Mean Concentration	Subchronic Intakes (mg/kg/day)	Lifetime Adjusted Chronic Daily Intakes (mg/kg/day)
Surface Water	Gifford Road Spring	Dermal Contact (mg/L)	Toluene	ND	ND	ND	ND
			1,1-Dichloroethane	1.54E-01	2.78E-02	4.99E-06	2.19E-09
			Trichloroethene	5.60E-02	4.10E-03	1.81E-06	3.22E-10
			Vinyl chloride	2.00E-02	7.70E-03	6.48E-07	6.05E-10
			PCBs	4.20E-03	4.20E-03	1.36E-07	3.30E-10
		Inhalation (mg/m3)	Toluene	ND	ND	ND	ND
			1,1-Dichloroethane	1.50E-04	9.03E-06	2.38E-06	3.47E-10
			Trichloroethene	5.07E-05	1.24E-06	8.05E-07	4.76E-11
			Vinyl chloride	2.48E-05	3.19E-06	3.94E-07	1.22E-10
			PCBs	2.32E-06	7.75E-07	3.68E-08	2.97E-11
	Marsh Area	Dermal Contact (mg/L)	Toluene	1.00E-02	6.90E-04	3.24E-07	5.42E-11
			1,1-Dichloroethane	2.30E-01	2.39E-02	7.45E-06	1.88E-09
			Trichloroethene	2.43E-01	1.23E-02	7.87E-06	9.67E-10
			Vinyl chloride	3.20E-02	6.55E-03	1.04E-06	5.15E-10
			PCBs	3.40E-03	1.18E-03	1.10E-07	9.28E-11
		Inhalation (mg/m3)	Toluene	1.90E-03	4.36E-05	3.02E-05	1.67E-09
			1,1-Dichloroethane	4.00E-02	1.39E-03	6.35E-04	5.34E-08
			Trichloroethene	3.93E-02	6.62E-04	6.24E-04	2.54E-08
			Vinyl chloride	7.09E-03	4.84E-04	1.13E-04	1.86E-08
			PCBs	3.36E-04	3.89E-05	5.33E-06	1.49E-09
	Unalam Cooling Discharge	Dermal Contact (mg/L)	Toluene	4.00E-01	2.81E-02	1.30E-05	2.21E-09
			1,1-Dichloroethane	1.90E-01	5.20E-02	6.15E-06	4.09E-09
			Trichloroethene	1.90E+00	3.40E-01	6.15E-05	2.67E-08
			Vinyl chloride	3.70E-01	3.10E-01	1.20E-05	2.44E-08
			PCBs	ND	ND	ND	ND
		Inhalation (mg/m3)	Toluene	8.43E-06	1.97E-07	1.34E-07	7.56E-12
			1,1-Dichloroethane	3.67E-06	3.35E-07	5.83E-08	1.29E-11
			Trichloroethene	3.41E-05	2.03E-06	5.41E-07	7.79E-11
			Vinyl chloride	9.11E-06	2.54E-06	1.45E-07	9.75E-11
			PCBs	ND	ND	ND	ND
Soil/Sediment	Gifford Road Spring	Dermal Contact (mg/kg)	Toluene	NA	NA	NA	NA
			1,1-Dichloroethane	NA	NA	NA	NA
			Trichloroethene	NA	NA	NA	NA
			Vinyl chloride	NA	NA	NA	NA
			PCBs	2.50E+00	1.17E+00	1.64E-06	1.86E-09
	Marsh Area	Dermal Contact (mg/kg)	Toluene	NA	NA	NA	NA
			1,1-Dichloroethane	NA	NA	NA	NA
			Trichloroethene	NA	NA	NA	NA
			Vinyl chloride	NA	NA	NA	NA
			PCBs	6.68E+00	1.19E+00	4.39E-06	1.89E-09
Ground Water	Potable Well	Dermal Contact Bathing (mg/L)	Toluene	ND	ND	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05	3.60E-09	2.32E-10
			Trichloroethene	9.35E-05	3.00E-05	5.61E-09	1.59E-10
			Vinyl chloride	7.50E-05	7.50E-05	4.50E-09	3.97E-10
			PCBs	ND	ND	ND	ND
		Ingestion (mg/L)	Toluene	ND	ND	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05	3.12E-06	2.01E-07
			Trichloroethene	9.35E-05	3.00E-05	4.86E-06	1.38E-07
			Vinyl chloride	7.50E-05	7.50E-05	3.90E-06	3.44E-07
			PCBs	ND	ND	ND	ND
		Inhalation (mg/L)	Toluene	ND	ND	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05	1.38E-05	8.89E-07
			Trichloroethene	9.35E-05	3.00E-05	2.15E-05	6.09E-07
			Vinyl chloride	7.50E-05	7.50E-05	1.73E-05	1.52E-06
			PCBs	ND	ND	ND	ND

KEY
ND - Not detected
NA - Not analyzed

Table 4-11 (Continued)
Potential Exposures for Ambient Conditions
Exposure Point Concentrations and Calculated Intakes
Adults

Exposure Media	Exposure Area	Route of Exposure	Indicator Compound	Maximum Concentration	Mean Concentration	Subchronic Intakes (mg/kg)	Lifetime Adjusted Chronic Daily Intakes (mg/kg)
Surface Water	Gifford Road Spring	Dermal Contact (mg/L)	Toluene	ND	ND	ND	ND
			1,1-Dichloroethane	1.54E-01	2.78E-02	3.64E-06	1.54E-08
			Trichloroethene	5.60E-02	4.10E-03	1.32E-06	2.27E-09
			Vinyl chloride	2.00E-02	7.70E-03	4.73E-07	4.26E-09
			PCBs	4.20E-03	4.20E-03	9.94E-08	2.32E-09
		Inhalation (mg/m3)	Toluene	ND	ND	ND	ND
			1,1-Dichloroethane	1.50E-04	9.03E-06	1.78E-06	2.50E-09
			Trichloroethene	5.07E-05	1.24E-06	6.01E-07	3.44E-10
			Vinyl chloride	2.48E-05	3.19E-06	2.94E-07	8.84E-10
			PCBs	2.32E-06	7.75E-07	2.75E-08	2.15E-10
	Marsh Area	Dermal Contact (mg/L)	Toluene	1.00E-02	6.90E-04	2.37E-07	3.81E-10
			1,1-Dichloroethane	2.30E-01	2.39E-02	5.44E-06	1.32E-08
			Trichloroethene	2.43E-01	1.23E-02	5.75E-06	6.80E-09
			Vinyl chloride	3.20E-02	6.55E-03	7.57E-07	3.62E-09
			PCBs	3.40E-03	1.18E-03	8.04E-08	6.52E-10
		Inhalation (mg/m3)	Toluene	1.90E-03	4.36E-05	2.25E-05	1.21E-08
			1,1-Dichloroethane	4.00E-02	1.39E-03	4.74E-04	3.85E-07
			Trichloroethene	3.93E-02	6.62E-04	4.66E-04	1.83E-07
			Vinyl chloride	7.09E-03	4.84E-04	8.41E-05	1.34E-07
			PCBs	3.36E-04	3.89E-05	3.98E-06	1.08E-08
	Unalam Cooling Discharge	Dermal Contact (mg/L)	Toluene	4.00E-01	2.81E-02	9.46E-06	1.55E-08
			1,1-Dichloroethane	1.90E-01	5.20E-02	4.49E-06	2.87E-08
			Trichloroethene	1.90E+00	3.40E-01	4.49E-05	1.88E-07
			Vinyl chloride	3.70E-01	3.10E-01	8.75E-06	1.71E-07
			PCBs	ND	ND	ND	ND
		Inhalation (mg/m3)	Toluene	8.43E-06	1.97E-07	9.99E-08	5.46E-11
			1,1-Dichloroethane	3.67E-06	3.35E-07	4.35E-08	9.28E-11
			Trichloroethene	3.41E-05	2.03E-06	4.04E-07	5.62E-10
			Vinyl chloride	9.11E-06	2.54E-06	1.08E-07	7.04E-10
			PCBs	ND	ND	ND	ND
Soil/Sediment	Gifford Road Spring	Dermal Contact (mg/kg)	Toluene	NA	NA	NA	NA
			1,1-Dichloroethane	NA	NA	NA	NA
			Trichloroethene	NA	NA	NA	NA
			Vinyl chloride	NA	NA	NA	NA
			PCBs	2.50E+00	1.17E+00	5.96E-07	6.52E-09
	Marsh Area	Dermal Contact (mg/kg)	Toluene	NA	NA	NA	NA
			1,1-Dichloroethane	NA	NA	NA	NA
			Trichloroethene	NA	NA	NA	NA
			Vinyl chloride	NA	NA	NA	NA
			PCBs	6.68E+00	1.19E+00	1.59E-06	6.63E-09
Ground Water	Potable Well	Dermal Contact Bathing (mg/L)	Toluene	ND	ND	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05	2.58E-09	1.61E-09
			Trichloroethene	9.35E-05	3.00E-05	4.02E-09	1.10E-09
			Vinyl chloride	7.50E-05	7.50E-05	3.23E-09	2.75E-09
			PCBs	ND	ND	ND	ND
		Ingestion (mg/L)	Toluene	ND	ND	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05	1.28E-06	7.99E-07
			Trichloroethene	9.35E-05	3.00E-05	2.00E-06	5.48E-07
			Vinyl chloride	7.50E-05	7.50E-05	1.61E-06	1.37E-06
			PCBs	ND	ND	ND	ND
		Inhalation (mg/L)	Toluene	ND	ND	ND	ND
			1,1-Dichloroethane	6.00E-05	4.38E-05	1.02E-05	6.35E-06
			Trichloroethene	9.35E-05	3.00E-05	1.59E-05	4.35E-06
			Vinyl chloride	7.50E-05	7.50E-05	1.28E-05	1.09E-05
			PCBs	ND	ND	ND	ND

KEY
ND - Not detected
NA - Not analyzed

SECTION

5

SECTION 5

TOXICITY EVALUATION

5.1 Evaluation Process

The toxicity evaluation is conducted as part of an endangerment assessment to quantitatively and qualitatively assess the potential for adverse human health effects from exposure to the indicator compounds. The quantitiative portion of the evaluation entails identifying the relevant indices of toxicity against which calculated intakes can be compared in the risk characterization. The qualitative aspect of the evaluation includes a summary of the pertinent toxicology data for each compound as well as US EPA and IARC weight-of-evidence classifications that describe each compound's potential for human carcinogenicity. The background and methodology for the toxicity evaluation is included in Section 2.

The weight-of-evidence carcinogenicity classification followed by a brief summary of the adverse health effects associated with each indicator chemical are presented in the following sections. A detailed toxicology profile for each indicator compound is included in Appendix E.

A summary of acceptable intake levels and cancer potency values for the indicator chemicals are presented in Table 5-1.

Table 5-1
Summary of Toxicological Information
For the Indicator Chemicals
Amphenol Route 8 Landfill

Indicator Chemical	Oral* AIS mg/kg/day	Oral* RFD mg/kg/day	Inhalation* AIS mg/kg/day	Inhalation* RFD mg/kg/day	Oral** CPF 1/mg/kg/day	Inhalation** CPF 1/mg/kg/day	EPA Carcinogen Classification	Reference
Toluene	0.4	0.3 (IRIS)	1.0	1.0	NA	NA	D	PHRED
1,1-Dichloroethane	1.2	0.12	1.38	0.138	NA	NA	C	SPHEM
Trichloroethene	NA	NA	NA	NA	0.011	0.013	B2	IRIS
Vinyl Chloride	NA	NA	NA	NA	2.3	0.295	A	ATSDR+
PCBs	NA	NA	NA	NA	7.7	7.7	B2	ATSDR++

*Noncarcinogenic effects
**Carcinogenic effects

AIS - Acceptabe Intake Subchronic Exposures
RFD - Reference Dose
CPF - Carcinogenic Potency Factor

NA - Not Available

PHRED - Public Health Risk Evaluation Database 9/88.
SPHEM - Superfund Public Health Evaluation Manual 10/86.
IRIS - EPA's On-Line Integrated Risk Information System accessed 1/20/89.
ATSDR+ - Agency for Toxic Substances and Disease Registry, draft toxicological profile for vinyl chloride 1/88.
ATSDR++ - Agency for Toxic Substances and Disease Registry, draft toxicological profile for selected PCBs 11/87.

5.2 Identification of Carcinogens

Evidence of possible carcinogenicity in humans comes primarily from long-term animal tests and epidemiological investigations. Results from these studies are supplemented with information from short-term tests, pharmacokinetic studies, comparative metabolism studies, structure activity relationships, and other relevant information sources.

When judging qualitative evidence of carcinogenicity, EPA as well as the IARC have adopted a policy of "weight-of-evidence", meaning that the quality and adequacy of all relevant data on responses induced by a possible carcinogen using different procedures will be considered. There are three major steps in determining the weight-of-evidence for carcinogenicity:

1. characterization of the evidence from human studies and from animal studies individually,

2. combination of the two types of studies into a final indication of overall weight-of-evidence for possible human carcinogencity, and

3. evaluation of all supportive information to determine if the overall weight-of-evidence should be modified.

Further details concerning the classification systems of the EPA and the IARC, and use of this data in the risk assessment process, are presented in Appendix D.

5.3 Determination of Carcinogenic Potency

The second phase in carcinogen assessment involves the quantification of risk. Experimental studies of carcinogenic effects utilizing the low exposure levels usually encountered in the environment usually are not feasible. Therefore, various mathematical models have to be used for extrapolation from the high doses used in animal bioassays to the very much lower dosages of interest in connection with exposure to ambient environmental concentrations. Since the resolution power of animal studies, for example, is not adequate for precise elaboration of the dose-response curve, extrapolating from a high-dose to a low-dose introduces a level of uncertainty which may amount to orders of magnitude. Given the recognized differences in carcinogenic response between species, and between strains of the same species, it is clear that additional uncertainties will be introduced when quantitative extrapolations, for example between rodents and humans, are made. Among various proposed models for quantitative extrapolation, EPA recommends the use of a linearized multistage model, "unless there is evidence on carcinogenesis mechanisms or other biological evidence that indicates the greater suitability of an alternative extrapolation model, or there is statistical or biological evidence that excludes the use of the linearized multistage model" (Federal Register, 1984). The carcinogenic potency of a compound is often expressed in terms of a potency factor which represents the upper 95 percent confidence limit on the probability of response per unit intake (mg/kg, etc.) of a compound over a 70-year lifetime. EPA's Carcinogen Assessment Group (CAG) has evaluated more than fifty-four compounds as suspected human carcinogens and developed relative carcinogenic potency factors for each compound. CPFs are expressed in risk units; that is the excess lifetime risk associated with exposure

to 1 mg/kg body weight per day, every day for a lifetime of 70 years. The toxicity information presented herein relies primarily on the information provided in the Superfund Public Health Manual (USEPA, 1986a), and US EPA on-line Integrated Risk Information System (IRIS).

The level of evidence for carcinogenicity for the Route 8 Landfill indicator compounds is discussed in detail in Appendix D. A brief summary of that discussion is given below.

There is a significant controversy in the international scientific community surrounding the classification of trichloroethene. EPA has classified trichloroethene as a probable human (Class B2) carcinogen. However, EPA's interpretation of mouse liver tumors observed in long-term studies and the appropriateness of the use of the linearized multistage model for calculation of carcinogenic potency have not been widely accepted by the scientific community. IARC has determined that there is insufficient evidence to classify trichloroethene with regard to carcinogenicity at this time. In this Risk Assessment, ERM has regarded trichloroethene according to EPA's classification and has included it in the carcinogenic risk assessment. However, this classification of trichloroethene is a matter of debate and may ultimately result in an over-estimation of carcinogenic risks.

Both EPA and IARC have classified vinyl chloride as a known human carcinogen. EPA's carcinogen potency factor for vinyl chloride is 2.3 $(mg/kg/day)^{-1}$ by the oral route and 0.295 $(mg/kg/day)^{-1}$ by the inhalation route.

Both EPA and IARC have classified PCBs as probable human carcinogens. No evidence exists to indicate that PCBs have caused

cancer in humans. EPA's carcinogenic potency factor for this group of compounds is 7.7 $(mg/kg/day)^{-1}$.

Toluene is considered a noncarcinogen by the EPA and IARC based on either "no evidence" or "inadequate evidence" of carcinogenicity in animal studies. 1,1-Dichloroethane is not considered a carcinogen by EPA and by IARC.

5.4 Noncarcinogenic Effects

A full discussion of noncarcinogenic effects of the indicator compounds is provided in Appendix E. A brief summary of these effects is given here to provide an understanding of the types of effects that have been documented. Most of the effects observed result from exposures to levels much higher than OSHA or ACGIH occupational exposure limits. The OSHA exposure limit for 1,1-Dichloroethane is 400 mg/m^3, while animal experiments are usually conducted at much higher concentrations. For example, one experiment was conducted using repeated daily exposures of rats to 1000 mg/m^3 of 1,1-dichloroethane. The resulting doses the animals received is much higher than the dose to humans because of the animals' lesser body weight. Overall, the effects discussed would occur over a range of acute and chronic doses which are much higher than those associated with releases from the Route 8 Landfill:

- Toluene: There is no conclusive evidence that toluene is carcinogenic or mutagenic in animals or humans based on EPA studies. Acute exposure to toluene produces central nervous system depression and narcosis in humans. However, inhalation exposure to quantities sufficient to produce unconsciousness fail to produce residual organ damage. Chronic inhalation exposure to toluene at relatively high

concentrations produces cerebellar degeneration and an irreversible encephalopathy in mammals.

- 1,1-Dichloroethane: Although limited toxicological testing of 1,1-dichloroethane has been conducted, the literature indicates that 1,1-dichloroethane is one of the least toxic of the chlorinated ethanes. A National Cancer Institute (NCI) bioassay on 1,1-dichloroethane was limited due to poor survival of test animals. (The early mortality appeared to be related to a high incidence of pneumonia.) However, marginal tumorigenic effects were seen. 1,1-Dichloroethane was not found to be mutagenic using the Ames assay method. 1,1-Dichloroethane causes central nervous system depression when inhaled at high concentrations, and evidence suggests that the compound is hepatotoxic in humans. Kidney and liver damage was seen in animals exposed to high levels (8,000 ppm).

- Trichloroethene: TCE has a low acute toxicity in mammals. In humans higher concentrations of this volatile substance have anesthetic and analgesic properties and are known to occasionally elicit cardiac arrythmias. Chronic exposure has been reported to induce damage to the nervous system leading to incoordination sleep disturbances and psychotic episodes.

- Vinyl Chloride: The acute toxicity of vinyl chloride is low, and short-term human exposure to high concentrations mainly causes depression of the central nervous system. Chronic exposure to vinyl chloride has been associated with liver and kidney damage, thickening of the skin and changes in the circulation and bone structure of the fingers. There is also evidence in experimental animals as well as in

humans suggesting that vinyl chloride may cause toxic effects to the fetus and development defects.

- PCBs: PCBs are a mixture of many different congeners. They can be absorbed through the skin, gastrointestinal tract, and the lungs. The acute toxicity of PCBs is low, but chronic exposure can cause chloracne (a long-lasting, disfiguring skin disease), liver damage, reproductive disorders, and neurologic disease. PCBs are carcinogenic in rats and mice and are considered by EPA to be probable human carcinogens. PCBs have caused toxic effects to the fetus, but they are not believed to cause birth defects.

SECTION

6

SECTION 6

RISK CHARACTERIZATION

6.1 Introduction

This section assesses the potential risks to human health and the environment associated with exposure to the various indicator compounds, under existing conditions associated with the Route 8 Landfill. The potential risks of exposure to carcinogens and noncarcinogens were assessed separately by comparing:

- the current exposure point intakes calculated in Section 5 to acceptable intakes for noncarcinogens;

- the calculated potential carcinogenic risks to acceptable risk levels for potential carcinogens;

6.2 Noncarcinogenic Risk

The Hazard Index method is used for assessing the overall potential for noncarcinogenic effects posed by the indicator compounds. This approach assumes that multiple sub-threshold exposures could result in an adverse effect and that the magnitude of the adverse effect will be proportional to the sum of the ratios of the sub-threshold exposures to acceptable exposures. This can be expressed as:

$$\text{Hazard Index} = E_1/AL_1 + E_2/AL_2 + \ldots + E_i AL_i$$

where E_i = Exposure level (or intake) for the i^{th} toxicant
AL_i = Acceptable level (or intake) for i^{th} toxicant

For a single compound, there may be a potential adverse health effect when the hazard index exceeds one. For multiple compound exposures, if the sum of the hazard indices exceeds one, the exposures may result in a potential adverse health effect, even if no single compound exceeds its acceptable level. However, the assumption of additivity should only be made for compounds that produce the same toxic effect by the same mechanisms of action. Table 6-1 presents the calculated hazard index for each route of exposure. All resulting hazard indices are well below the EPA guideline of one. Since the sum of all the hazard indices for each single age group is also below one, analysis by toxic effect is not necessary for the Route 8 Landfill Site.

6.3 Calculation of Carcinogenic Risk

For potential carcinogens, risks are estimated as probabilities. The carcinogenic potency factor, which is the upper 95 percent confidence limit of the probability of a carcinogenic response per unit intake over a lifetime of exposure, converts estimated CDIs directly to incremental risk values. In general, because only relatively low CDIs are likely to result from environmental exposures, it is assumed in the EPA methodology that the exposure will be in the linear portion of the dose-response curve. Based on this assumption, the slope of the dose-response curve is equivalent to the carcinogenic potency factor, and the risk is directly related to the CDI at low levels of exposure. The low-dose carcinogenic risk equation is:

Table 6-1
Potential Exposures for Ambient Conditions
Amphenol Route 8 Landfill
Calculated Hazard Indicies
Children Ages 2-6

Exposure Media	Exposure Area	Route of Exposure	Indicator Compound	Subchronic Intakes (mg/kg/day)	AIS (mg/kg/day)	Hazard Index Subchronic	Lifetime Adjusted Chronic Daily Intakes (mg/kg/day)	AIC (mg/kg/day)	Contribution to Lifetime Adjusted Hazard Index
Soil/Sediment	Gifford Road Spring	Dermal Contact (mg/kg)	Toluene	NA	4.30E-01	N/A	NA	3.00E-01	N/A
			1,1-Dichloroethane	NA	1.20E+00	N/A	NA	1.20E-01	N/A
			Trichloroethene	NA	N/A	N/A	NA	N/A	N/A
			Vinyl chloride	NA	N/A	N/A	NA	N/A	N/A
			PCBs	2.01E-06	N/A	N/A	1.51E-09	N/A	N/A
		PICA Ingestion (mg/kg)	Toluene	NA	4.30E-01	N/A	NA	3.00E-01	N/A
			1,1-Dichloroethane	NA	1.20E+00	N/A	NA	1.20E-01	N/A
			Trichloroethene	NA	N/A	N/A	NA	N/A	N/A
			Vinyl chloride	NA	N/A	N/A	NA	N/A	N/A
			PCBs	1.56E-05	N/A	N/A	1.18E-08	N/A	N/A
	Marsh Area	Dermal Contact (mg/kg)	Toluene	NA	4.30E-01	N/A	NA	3.00E-01	N/A
			1,1-Dichloroethane	NA	1.20E+00	N/A	NA	1.20E-01	N/A
			Trichloroethene	NA	N/A	N/A	NA	N/A	N/A
			Vinyl chloride	NA	N/A	N/A	NA	N/A	N/A
			PCBs	5.36E-06	N/A	N/A	1.54E-09	N/A	N/A
		PICA Ingestion (mg/kg)	Toluene	NA	4.30E-01	N/A	NA	3.00E-01	N/A
			1,1-Dichloroethane	NA	1.20E+00	N/A	NA	1.20E-01	N/A
			Trichloroethene	NA	N/A	N/A	NA	N/A	N/A
			Vinyl chloride	NA	N/A	N/A	NA	N/A	N/A
			PCBs	4.17E-05	N/A	N/A	1.20E-08	N/A	N/A
Ground Water	Potable Well	Dermal Contact Bathing (mg/L)	Toluene	ND	4.30E-01	N/A	ND	3.00E-01	N/A
			1,1-Dichloroethane	4.32E-09	1.20E+00	3.60E-09	1.86E-10	1.20E-01	1.55E-09
			Trichloroethene	6.73E-09	N/A	N/A	1.27E-10	N/A	N/A
			Vinyl chloride	5.40E-09	N/A	N/A	3.18E-10	N/A	N/A
			PCBs	ND	N/A	N/A	ND	N/A	N/A
		Ingestion (mg/L)	Toluene	ND	4.30E-01	N/A	ND	3.00E-01	N/A
			1,1-Dichloroethane	5.64E-06	1.20E+00	4.70E-06	2.42E-07	1.20E-01	2.02E-06
			Trichloroethene	8.79E-06	N/A	N/A	1.66E-07	N/A	N/A
			Vinyl chloride	7.05E-06	N/A	N/A	4.15E-07	N/A	N/A
			PCBs	ND	N/A	N/A	ND	N/A	N/A
		Inhalation (mg/L)	Toluene	ND	1.00E+00	N/A	ND	1.00E+00	N/A
			1,1-Dichloroethane	1.32E-05	1.38E+00	9.57E-06	5.67E-07	1.38E-01	4.11E-06
			Trichloroethene	2.06E-05	N/A	N/A	3.88E-07	N/A	N/A
			Vinyl chloride	1.65E-05	N/A	N/A	9.71E-07	N/A	N/A
			PCBs	ND	N/A	N/A	ND	N/A	N/A

Total subchronic hazard index = 1.43E-05

Total contribution to lifetime chronic hazard index = 6.13E-06

KEY
ND - Not detected
NA - Not analyzed
NC - Not calculated because only one analysis was performed
N/A - Not applicable/available
AIS - Acceptable intake subchronic
AIC - Acceptable intake chronic

Table 6-1 (Continued)
Potential Exposures for Ambient Conditions
Amphenol Route 8 Landfill
Calculated Hazard Indicies
Children Ages 6-12

Exposure Media	Exposure Area	Route of Exposure	Indicator Compound	Subchronic Intakes (mg/kg/day)	AIS (mg/kg/day)	Hazard Index Subchronic	Lifetime Adjusted Chronic Daily Intakes (mg/kg/day)	AIC (mg/kg/day)	Contribution to Lifetime Adjusted Hazard Index
Surface Water	Gifford Road Spring	Dermal Contact (mg/L)	Toluene	ND	4.30E-01	N/A	ND	3.00E-01	N/A
			1,1-Dichloroethane	4.99E-06	1.20E+00	4.16E-06	2.19E-09	1.20E-01	1.82E-08
			Trichloroethene	1.81E-06	N/A	N/A	3.22E-10	N/A	N/A
			Vinyl chloride	6.48E-07	N/A	N/A	6.05E-10	N/A	N/A
			PCBs	1.36E-07	N/A	N/A	3.30E-10	N/A	N/A
		Inhalation (mg/m3)	Toluene	ND	1.00E+00	N/A	ND	1.00E+00	N/A
			1,1-Dichloroethane	2.38E-06	1.38E+00	1.73E-06	3.47E-10	1.38E-01	2.51E-09
			Trichloroethene	8.05E-07	N/A	N/A	4.76E-11	N/A	N/A
			Vinyl chloride	3.94E-07	N/A	N/A	1.22E-10	N/A	N/A
			PCBs	3.68E-08	N/A	N/A	2.97E-11	N/A	N/A
	Marsh Area	Dermal Contact (mg/L)	Toluene	3.24E-07	4.30E-01	7.53E-07	5.42E-11	3.00E-01	1.81E-10
			1,1-Dichloroethane	7.45E-06	1.20E+00	6.21E-06	1.88E-09	1.20E-01	1.57E-08
			Trichloroethene	7.87E-06	N/A	N/A	9.67E-10	N/A	N/A
			Vinyl chloride	1.04E-06	N/A	N/A	5.15E-10	N/A	N/A
			PCBs	1.10E-07	N/A	N/A	9.28E-11	N/A	N/A
		Inhalation (mg/m3)	Toluene	3.02E-05	1.00E+00	3.02E-05	1.67E-09	1.00E+00	1.67E-09
			1,1-Dichloroethane	6.35E-04	1.38E+00	4.60E-04	5.34E-08	1.38E-01	3.87E-07
			Trichloroethene	6.24E-04	N/A	N/A	2.54E-08	N/A	N/A
			Vinyl chloride	1.13E-04	N/A	N/A	1.86E-08	N/A	N/A
			PCBs	5.33E-06	N/A	N/A	1.49E-09	N/A	N/A
	Unalam Cooling Discharge	Dermal Contact (mg/L)	Toluene	1.30E-05	4.30E-01	3.01E-05	2.21E-09	3.00E-01	7.36E-09
			1,1-Dichloroethane	6.15E-06	1.20E+00	5.13E-06	4.09E-09	1.20E-01	3.41E-08
			Trichloroethene	6.15E-05	N/A	N/A	2.67E-08	N/A	N/A
			Vinyl chloride	1.20E-05	N/A	N/A	2.44E-08	N/A	N/A
			PCBs	ND	N/A	N/A	ND	N/A	N/A
		Inhalation (mg/m3)	Toluene	1.34E-07	1.00E+00	1.34E-07	7.56E-12	1.00E+00	7.56E-12
			1,1-Dichloroethane	5.83E-08	1.38E+00	4.22E-08	1.29E-11	1.38E-01	9.32E-11
			Trichloroethene	5.41E-07	N/A	N/A	7.79E-11	N/A	N/A
			Vinyl chloride	1.45E-07	N/A	N/A	9.75E-11	N/A	N/A
			PCBs	ND	N/A	N/A	ND	N/A	N/A
Soil/Sediment	Gifford Road Spring	Dermal Contact (mg/kg)	Toluene	NA	4.30E-01	N/A	NA	3.00E-01	N/A
			1,1-Dichloroethane	NA	1.20E+00	N/A	NA	1.20E-01	N/A
			Trichloroethene	NA	N/A	N/A	NA	N/A	N/A
			Vinyl chloride	NA	N/A	N/A	NA	N/A	N/A
			PCBs	1.64E-06	N/A	N/A	1.86E-09	N/A	N/A
	Marsh Area	Dermal Contact (mg/kg)	Toluene	NA	4.30E-01	N/A	NA	3.00E-01	N/A
			1,1-Dichloroethane	NA	1.20E+00	N/A	NA	1.20E-01	N/A
			Trichloroethene	NA	N/A	N/A	NA	N/A	N/A
			Vinyl chloride	NA	N/A	N/A	NA	N/A	N/A
			PCBs	4.39E-06	N/A	N/A	1.89E-09	N/A	N/A
Ground Water	Potable Well	Dermal Contact Bathing (mg/L)	Toluene	ND	4.30E-01	N/A	ND	3.00E-01	N/A
			1,1-Dichloroethane	3.60E-09	1.20E+00	3.00E-09	2.32E-10	1.20E-01	1.93E-09
			Trichloroethene	5.61E-09	N/A	N/A	1.59E-10	N/A	N/A
			Vinyl chloride	4.50E-09	N/A	N/A	3.97E-10	N/A	N/A
			PCBs	ND	N/A	N/A	ND	N/A	N/A
		Ingestion (mg/L)	Toluene	ND	4.30E-01	N/A	ND	3.00E-01	N/A
			1,1-Dichloroethane	3.12E-06	1.20E+00	2.60E-06	2.01E-07	1.20E-01	1.67E-06
			Trichloroethene	4.86E-06	N/A	N/A	1.38E-07	N/A	N/A
			Vinyl chloride	3.90E-06	N/A	N/A	3.44E-07	N/A	N/A
			PCBs	ND	N/A	N/A	ND	N/A	N/A
		Inhalation (mg/L)	Toluene	ND	1.00E+00	N/A	ND	1.00E+00	N/A
			1,1-Dichloroethane	1.38E-05	1.38E+00	1.00E-05	8.89E-07	1.38E-01	6.44E-06
			Trichloroethene	2.15E-05	N/A	N/A	6.09E-07	N/A	N/A
			Vinyl chloride	1.73E-05	N/A	N/A	1.52E-06	N/A	N/A
			PCBs	ND	N/A	N/A	ND	N/A	N/A

Total subchronic hazard index = 5.51E-04

Total contribution to lifetime chronic hazard index = 8.58E-06

KEY
ND - Not detected
NA - Not analyzed
N/A - Not applicable/available

AIS - Acceptable intake subchronic
AIC - Acceptable intake chronic

Table 6-1 (Continued)
Potential Exposures for Ambient Conditions
Amphenol Route 8 Landfill
Calculated Hazard Indicies
Adults

Exposure Media	Exposure Area	Route of Exposure	Indicator Compound	Subchronic Intakes (mg/kg/day)	AIS (mg/kg/day)	Hazard Index Subchronic	Lifetime Adjusted Chronic Daily Intakes (mg/kg/day)	AIC (mg/kg/day)	Contribution to Lifetime Adjusted Hazard Index
Surface Water	Gifford Road Spring	Dermal Contact (mg/L)	Toluene	ND	4.30E-01	N/A	ND	3.00E-01	N/A
			1,1-Dichloroethane	3.64E-06	1.20E+00	3.04E-06	1.54E-08	1.20E-01	1.28E-07
			Trichloroethene	1.32E-06	N/A	N/A	2.27E-09	N/A	N/A
			Vinyl chloride	4.73E-07	N/A	N/A	4.26E-09	N/A	N/A
			PCBs	9.94E-08	N/A	N/A	2.32E-09	N/A	N/A
		Inhalation (mg/m3)	Toluene	ND	1.00E+00	N/A	ND	1.00E+00	N/A
			1,1-Dichloroethane	1.78E-06	1.38E+00	1.29E-06	2.50E-09	1.38E-01	1.81E-08
			Trichloroethene	6.01E-07	N/A	N/A	3.44E-10	N/A	N/A
			Vinyl chloride	2.94E-07	N/A	N/A	8.84E-10	N/A	N/A
			PCBs	2.75E-08	N/A	N/A	2.15E-10	N/A	N/A
	Marsh Area	Dermal Contact (mg/L)	Toluene	2.37E-07	4.30E-01	5.50E-07	3.81E-10	3.00E-01	1.27E-09
			1,1-Dichloroethane	5.44E-06	1.20E+00	4.53E-06	1.32E-08	1.20E-01	1.10E-07
			Trichloroethene	5.75E-06	N/A	N/A	6.80E-09	N/A	N/A
			Vinyl chloride	7.57E-07	N/A	N/A	3.62E-09	N/A	N/A
			PCBs	8.04E-08	N/A	N/A	6.52E-10	N/A	N/A
		Inhalation (mg/m3)	Toluene	2.25E-05	1.00E+00	2.25E-05	1.21E-08	1.00E+00	1.21E-08
			1,1-Dichloroethane	4.74E-04	1.38E+00	3.44E-04	3.85E-07	1.38E-01	2.79E-06
			Trichloroethene	4.66E-04	N/A	N/A	1.83E-07	N/A	N/A
			Vinyl chloride	8.41E-05	N/A	N/A	1.34E-07	N/A	N/A
			PCBs	3.98E-06	N/A	N/A	1.08E-08	N/A	N/A
	Unalam Cooling Discharge	Dermal Contact (mg/L)	Toluene	9.46E-06	4.30E-01	2.20E-05	1.55E-08	3.00E-01	5.18E-08
			1,1-Dichloroethane	4.49E-06	1.20E+00	3.75E-06	2.87E-08	1.20E-01	2.40E-07
			Trichloroethene	4.49E-05	N/A	N/A	1.88E-07	N/A	N/A
			Vinyl chloride	8.75E-06	N/A	N/A	1.71E-07	N/A	N/A
			PCBs	ND	N/A	N/A	ND	N/A	N/A
		Inhalation (mg/m3)	Toluene	9.99E-08	1.00E+00	9.99E-08	5.46E-11	1.00E+00	5.46E-11
			1,1-Dichloroethane	4.35E-08	1.38E+00	3.15E-08	9.28E-11	1.38E-01	6.73E-10
			Trichloroethene	4.04E-07	N/A	N/A	5.62E-10	N/A	N/A
			Vinyl chloride	1.08E-07	N/A	N/A	7.04E-10	N/A	N/A
			PCBs	ND	N/A	N/A	ND	N/A	N/A
Soil/Sediment	Gifford Road Spring	Dermal Contact (mg/kg)	Toluene	NA	4.30E-01	N/A	NA	3.00E-01	N/A
			1,1-Dichloroethane	NA	1.20E+00	N/A	NA	1.20E-01	N/A
			Trichloroethene	NA	N/A	N/A	NA	N/A	N/A
			Vinyl chloride	NA	N/A	N/A	NA	N/A	N/A
			PCBs	5.96E-07	N/A	N/A	6.52E-09	N/A	N/A
	Marsh Area	Dermal Contact (mg/kg)	Toluene	NA	4.30E-01	N/A	NA	3.00E-01	N/A
			1,1-Dichloroethane	NA	1.20E+00	N/A	NA	1.20E-01	N/A
			Trichloroethene	NA	N/A	N/A	NA	N/A	N/A
			Vinyl chloride	NA	N/A	N/A	NA	N/A	N/A
			PCBs	1.59E-06	N/A	N/A	6.63E-09	N/A	N/A
Ground Water	Potable Well	Dermal Contact Bathing (mg/L)	Toluene	ND	4.30E-01	N/A	ND	3.00E-01	N/A
			1,1-Dichloroethane	2.58E-09	1.20E+00	2.15E-09	1.61E-09	1.20E-01	1.34E-08
			Trichloroethene	4.02E-09	N/A	N/A	1.10E-09	N/A	N/A
			Vinyl chloride	3.23E-09	N/A	N/A	2.75E-09	N/A	N/A
			PCBs	ND	N/A	N/A	ND	N/A	N/A
		Ingestion (mg/L)	Toluene	ND	4.30E-01	N/A	ND	3.00E-01	N/A
			1,1-Dichloroethane	1.28E-06	1.20E+00	1.07E-06	7.99E-07	1.20E-01	6.66E-06
			Trichloroethene	2.00E-06	N/A	N/A	5.48E-07	N/A	N/A
			Vinyl chloride	1.61E-06	N/A	N/A	1.37E-06	N/A	N/A
			PCBs	ND	N/A	N/A	ND	N/A	N/A
		Inhalation (mg/L)	Toluene	ND	1.00E+00	N/A	ND	1.00E+00	N/A
			1,1-Dichloroethane	1.02E-05	1.38E+00	7.39E-06	6.35E-06	1.38E-01	4.60E-05
			Trichloroethene	1.59E-05	N/A	N/A	4.35E-06	N/A	N/A
			Vinyl chloride	1.28E-05	N/A	N/A	1.09E-05	N/A	N/A
			PCBs	ND	N/A	N/A	ND	N/A	N/A

Total subchronic hazard index = 4.10E-04

Total contribution to lifetime chronic hazard index = 5.60E-05

Total Lifetime Chronic Hazard Index = 7.08E+05

KEY

ND - Not detected
NA - Not analyzed
N/A - Not applicable/available

AIS - Acceptable intake subchronic
AIC - Acceptable intake chronic

Risk = CDI x Carcinogenic Potency Factor

Table 6-2 presents the calculated potential carcinogenic risks associated with each route of exposure. Under the Superfund program, the EPA guideline for acceptable total lifetime carcinogenic site risk lies within the range of 1×10^{-7} to 1×10^{-4}. Specifically related to ground water cleanup goals, the 1×10^{-6} risk level has been applied as a guideline by the EPA. It can be seen that almost all Route 8 Landfill-related potential contributions to lifetime carcinogenic risk are within the potentially acceptable EPA range.

Potential site-related contributions to lifetime carcinogenic risk within the acceptable range, but exceeding 1×10^{-6} occur only at one location:

- a hypothetical future well on River Road, due to potential exposure to vinyl chloride

The potential for exposure by the ground water route is very low, due to the availability of public water and non-usage of the aquifer downgradient of the site. Taken along with the high degree of conservatism in the EPA risk calculation process, the Route 8 Landfill site risk levels as calculated are overestimated; thus the actual site-related risks are expected to be low.

6.4 Summary and Conclusions

Summaries of site-related non-carcinogenic effects and carcinogenic risk levels are shown in Tables 6-1 and 6-2. No noncarcinogenic adverse health effects are associated with the site. Conservative carcinogenic risk assessment indicates that

Table 6-2
Potential Exposures for Ambient Conditions
Amphenol Route 8 Landfill
Calculation of Risk From Potential Carcinogens
Children Ages 2-6

Exposure Media	Exposure Area	Route of Exposure	Indicator Compound	Lifetime Adjusted Chronic Daily Intakes (mg/kg/day)	Carcinogen Potency Factor (mg/kg/day)-1	Contribution to Lifetime Risk
Soil/Sediment	Gifford Road Spring	Dermal Contact (mg/kg)	Trichloroethene	NA	1.10E-02	N/A
			Vinyl chloride	NA	2.30E+00	N/A
			PCBs	1.51E-09	7.70E+00	1E-08
		Pica Ingestion (mg/kg)	Trichloroethene	NA	1.10E-02	N/A
			Vinyl chloride	NA	2.30E+00	N/A
			PCBs	1.18E-08	7.70E+00	9E-08
	Marsh Area	Dermal Contact (mg/kg)	Trichloroethene	NA	1.10E-02	N/A
			Vinyl chloride	NA	2.30E+00	N/A
			PCBs	1.54E-09	7.70E+00	1E-08
		Pica Ingestion (mg/kg)	Trichloroethene	NA	1.10E-02	N/A
			Vinyl chloride	NA	2.30E+00	N/A
			PCBs	1.20E-08	7.70E+00	9E-08
Ground Water	Potable Well	Dermal Contact Bathing (mg/L)	Trichloroethene	1.27E-10	1.10E-02	1E-12
			Vinyl chloride	3.18E-10	2.30E+00	7E-10
			PCBs	ND	7.70E+00	N/A
		Ingestion (mg/L)	Trichloroethene	1.66E-07	1.10E-02	2E-09
			Vinyl chloride	4.15E-07	2.30E+00	1E-06
			PCBs	ND	7.70E+00	N/A
		Inhalation (mg/L)	Trichloroethene	3.88E-07	1.30E-02	5E-09
			Vinyl chloride	9.71E-07	2.95E-01	3E-07
			PCBs	ND	7.70E+00	N/A

KEY
ND - Not detected
NA - Not analyzed
NC - Not calculated because only one analysis was performed
N/A - Not applicable/available

Table 6-2 (Continued)
Potential Exposures for Ambient Conditions
Amphenol Route 8 Landfill
Calculation of Risk From Potential Carcinogens
Children Ages 6-12

Exposure Media	Exposure Area	Route of Exposure	Indicator Compound	Lifetime Adjusted Chronic Daily Intakes (mg/kg/day)	Carcinogen Potency Factor (mg/kg/day)-1	Contribution to Lifetime Risk
Surface Water	Gifford Road Spring	Dermal Contact (mg/L)	Trichloroethene	3.22E-10	1.10E-02	4E-12
			Vinyl chloride	6.05E-10	2.30E+00	1E-09
			PCBs	3.30E-10	7.70E+00	3E-09
		Inhalation (mg/m3)	Trichloroethene	4.76E-11	1.30E-02	6E-13
			Vinyl chloride	1.22E-10	2.95E-01	4E-11
			PCBs	2.97E-11	7.70E+00	2E-10
	Marsh Area	Dermal Contact (mg/L)	Trichloroethene	9.67E-10	1.10E-02	1E-11
			Vinyl chloride	5.15E-10	2.30E+00	1E-09
			PCBs	9.28E-11	7.70E+00	7E-10
		Inhalation (mg/m3)	Trichloroethene	2.54E-08	1.30E-02	3E-10
			Vinyl chloride	1.86E-08	2.95E-01	5E-09
			PCBs	1.49E-09	7.70E+00	1E-08
	Unalam Cooling Discharge	Dermal Contact (mg/L)	Trichloroethene	2.67E-08	1.10E-02	3E-10
			Vinyl chloride	2.44E-08	2.30E+00	6E-08
			PCBs	ND	7.70E+00	N/A
		Inhalation (mg/m3)	Trichloroethene	7.79E-11	1.30E-02	1E-12
			Vinyl chloride	9.75E-11	2.95E-01	3E-11
			PCBs	ND	7.70E+00	N/A
Soil/Sediment	Gifford Road Spring	Dermal Contact (mg/kg)	Trichloroethene	NA	1.10E-02	N/A
			Vinyl chloride	NA	2.30E+00	N/A
			PCBs	1.86E-09	7.70E+00	1E-08
	Marsh Area	Dermal Contact (mg/kg)	Trichloroethene	NA	1.10E-02	N/A
			Vinyl chloride	NA	2.30E+00	N/A
			PCBs	1.89E-09	7.70E+00	1E-08
Ground Water	Potable Well	Dermal Contact Bathing (mg/L)	Trichloroethene	1.59E-10	1.10E-02	2E-12
			Vinyl chloride	3.97E-10	2.30E+00	9E-10
			PCBs	ND	7.70E+00	N/A
		Ingestion (mg/L)	Trichloroethene	1.38E-07	1.10E-02	2E-09
			Vinyl chloride	3.44E-07	2.30E+00	8E-07
			PCBs	ND	7.70E+00	N/A
		Inhalation (mg/L)	Trichloroethene	6.09E-07	1.30E-02	8E-09
			Vinyl chloride	1.52E-06	2.95E-01	4E-07
			PCBs	ND	7.70E+00	N/A

KEY
ND - Not detected
NA - Not analyzed
NC - Not calculated because only one analysis was performed
N/A - Not applicable/available

Table 6-2 (Continued)
Potential Exposures for Ambient Conditions
Amphenol Route 8 Landfill
Calculation of Risk From Potential Carcinogens
Adults

Exposure Media	Exposure Area	Route of Exposure	Indicator Compound	Lifetime Adjusted Chronic Daily Intakes (mg/kg/day)	Carcinogen Potency Factor (mg/kg/day)-1	Contribution to Lifetime Risk
Surface Water	Gifford Road Spring	Dermal Contact (mg/L)	Trichloroethene	2.27E-09	1.10E-02	2E-11
			Vinyl chloride	4.26E-09	2.30E+00	1E-08
			PCBs	2.32E-09	7.70E+00	2E-08
		Inhalation (mg/m3)	Trichloroethene	3.44E-10	1.30E-02	4E-12
			Vinyl chloride	8.84E-10	2.95E-01	3E-10
			PCBs	2.15E-10	7.70E+00	2E-09
	Marsh Area	Dermal Contact (mg/L)	Trichloroethene	6.80E-09	1.10E-02	7E-11
			Vinyl chloride	3.62E-09	2.30E+00	8E-09
			PCBs	6.52E-10	7.70E+00	5E-09
		Inhalation (mg/m3)	Trichloroethene	1.83E-07	1.30E-02	2E-09
			Vinyl chloride	1.34E-07	2.95E-01	4E-08
			PCBs	1.08E-08	7.70E+00	8E-08
	Unalam Cooling Discharge	Dermal Contact (mg/L)	Trichloroethene	1.88E-07	1.10E-02	2E-09
			Vinyl chloride	1.71E-07	2.30E+00	4E-07
			PCBs	ND	7.70E+00	N/A
		Inhalation (mg/m3)	Trichloroethene	5.62E-10	1.30E-02	7E-12
			Vinyl chloride	7.04E-10	2.95E-01	2E-10
			PCBs	ND	7.70E+00	N/A
Soil/Sediment	Gifford Road Spring	Dermal Contact (mg/kg)	Trichloroethene	NA	1.10E-02	N/A
			Vinyl chloride	NA	2.30E+00	N/A
			PCBs	6.52E-09	7.70E+00	5E-08
	Marsh Area	Dermal Contact (mg/kg)	Trichloroethene	NA	1.10E-02	N/A
			Vinyl chloride	NA	2.30E+00	N/A
			PCBs	6.63E-09	7.70E+00	5E-08
Ground Water	Potable Well	Dermal Contact Bathing (mg/L)	Trichloroethene	1.10E-09	1.10E-02	1E-11
			Vinyl chloride	2.75E-09	2.30E+00	6E-09
			PCBs	ND	7.70E+00	N/A
		Ingestion (mg/L)	Trichloroethene	5.48E-07	1.10E-02	6E-09
			Vinyl chloride	1.37E-06	2.30E+00	3E-06
			PCBs	ND	7.70E+00	N/A
		Inhalation (mg/L)	Trichloroethene	4.35E-06	1.30E-02	6E-08
			Vinyl chloride	1.09E-05	2.95E-01	3E-06
			PCBs	ND	7.70E+00	N/A
					Total Lifetime Carcinogenic Risk:	1E-05

KEY
ND - Not detected
NA - Not analyzed
NC - Not calculated because only one analysis was performed
N/A - Not applicable/available

all potential site related exposures contribute below the potentially acceptable EPA guideline range of 1×10^{-7} to 1×10^{-4} to lifetime carcinogenic risk. Site-related potential exposure exceeding the 1×10^{-6} value is limited to a hypothetical potential exposure at a future well on River Road.

Based on the above findings, the following conclusions are drawn:

1) The remaining overall site-related carcinogenic risks are very low, and are within the potentially acceptable range.

2) The only potential site-related risk which needs be addressed in the remedial feasibility study is related to vinyl chloride in the bedrock aquifer.

3) The risks estimated were based on extremely conservative assumptions regarding the amount of human contact with site-related contaminants, and are expected to overestimate risk even for the very low percentage of the area's population which may be exposed to site-related constituents.

4) The Hill Site Risk Assessment (ERM, 1986) addressed the risk resulting from potential exposure to the constituents present in the K-Mart drain. The comprehensive evaluation of remedial alternatives for the Route 8 Site will consider the K-Mart drain because of the physical proximity of the drain to the site. The total lifetime risk from the K-Mart drain was determined to be 9×10^{-7}. This does not significantly affect the level of calculated risk associated with potential exposures at the Route 8 Site.

ACRONYMS

ACGIH	American Conference of Governmental Industrial Hygenists
AIC	Acceptable Intakes for Chronic Exposures
AIS	Acceptable Intakes for Subchronic Exposures
CAG	Carcinogen Assessment Group - USEPA
CDI	Chronic Daily Intake
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act (syn: Superfund)
CNS	Central Nervous System
CT	Concentration Times the Toxicity Constant for Each Medium
EPA	Environmental Protection Agency
ERM	Environmental Resources Management, Inc.
FDA	Food and Drug Administration
FS	Feasibility Study
IARC	International Agency for Research on Cancer of the World Health Organization
ICRP	International Committee for Radiologic Protection
IS	Indicator Score
MCL	Maximum Concentration Level
NAAQS	National Ambient Air Quality Standards
NC	Noncarcinogen
NCP	National Oil and Hazardous Substance Pollution Contingency Program
NIC	National Cancer Institute
NPL	National Priority List
NTP	National Toxicology Program
NYSDEC	New York State Department of Environmental Conservation
OERR	Office of Emergency and Remedial Response - USEPA
OSHA	Occupational Safety and Health Administration
PC	Potential Carcinogen
PCBs	Polychlorinated Biphenyls
ppb	Parts per billion
ppm	Parts per million
RA	Risk Assessment
RI	Remedial Investigation
SDI	Subchronic Daily Intake
TLV	Threshold Limit Value
VOCs	Volatile Organic Compounds

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APPENDICES

APPENDIX A

WORKSHEETS

WORKSHEET 1.
SCORING FOR INDICATOR CHEMICAL SELECTION:
CONCENTRATIONS AND Koc VALUES
IN VARIOUS ENVIRONMENTAL MEDIA.

Site: Amphenol Route 8
Date Prepared: August 18, 1987
Prepared by: M. Wulff
Verified by: DMW
Date: 9/30/87

		GROUND WATER (mg/L)			SURFACE WATER (mg/L)			SURFACE SOIL (mg/kg)			SUBSURFACE SOIL (mg/kg)		
CHEMICAL	Koc VALUE	MINIMUM	MAXIMUM	REPRES	MINIMUM	MAXIMUM	REPRES	MINIMUM	MAXIMUM	REPRES	MINIMUM	MAXIMUM	REPRES
Benzene	8.30E+01	ND	4.00E-01	4.49E-03							3.00E-02	2.60E+02	2.46E+01
Toluene	3.00E+02	ND	3.50E+01	4.47E+00	ND	1.10E-02	8.30E-04				ND	7.10E+00	8.90E-01
Ethylbenzene	1.10E+03	ND	5.00E+00	3.97E-01	ND	5.60E-02	2.87E-03				ND	2.70E-02	7.80E-04
Chlorobenzene	3.30E+02												
1,1-Dichloroethene	6.50E+01	ND	6.90E-01	2.85E-02	ND	3.00E-03	1.30E-04						
1,1-Dichloroethane	3.00E+01	ND	1.50E+00	4.15E-01	ND	2.00E-01	6.75E-02	ND	3.20E-02	8.00E-03	ND	1.50E-01	1.36E-02
trans-1,2-Dichloroethene	5.90E+01	ND	3.60E+01	5.81E+00	ND	2.80E-01	4.32E-02				ND	7.10E+00	1.05E+00
Chloroform	3.10E+01	ND	1.50E+00	6.72E-02	ND	1.10E-02	8.30E-04						
Trichloroethene	1.26E+02	ND	1.40E+01	7.32E-01	ND	5.10E-02	4.35E-03				ND	5.70E+00	1.01E+00
Methylene Chloride	8.80E+00	ND	1.40E+00	5.51E-02									
Vinyl Chloride	5.70E+01	ND	1.10E+01	9.64E-01	ND	7.00E-02	1.04E-02						
1,1,1-Trichloroethane	1.52E+02	ND	5.60E+00	6.50E-01	ND	1.54E-01	1.36E-02				ND	4.10E-01	3.73E-02
1,2-Dichloroethane	1.40E+01	ND	2.90E+00	4.68E-02	ND	3.00E-03	3.50E-04						
Chloroethane U	3.00E+01	ND	2.70E-02	3.40E-04	ND	4.70E-02	5.04E-03						
1,1,2,2-Tetrachloroethane	3.64E+02	ND	1.00E-01	1.16E-03									
Dibromochloromethane		ND	6.00E-02	8.50E-04									
Acetone	2.20E+00	ND	4.80E-02	5.05E-01									
2-Butanone	4.50E+00	ND	5.80E-03	6.11E-02									
4-Methyl-2-pentanone U		ND	1.80E-03	1.89E-02									
Xylenes	2.40E+02	ND	3.40E+00	4.53E-02	1.30E-01	1.30E-01	5.65E-03				ND	8.20E+01	7.47E+00
PCBs	5.30E+05	ND	2.32E+00	4.47E-02	ND	4.20E-03	6.02E-04	ND	6.68E+00	4.17E-01	ND	1.33E+04	2.91E+02
Arsenic		ND	3.10E-02	1.00E-02	2.70E-02	2.70E-02	2.70E-02						
Barium		ND	3.00E-01	1.70E-01	2.00E-01	2.00E-01	2.00E-01						
Calcium U		1.99E+01	7.28E+01	4.87E+01	2.57E+01	2.57E+01	2.57E+01						
Iron U		ND	5.75E+00	1.92E+00	2.31E+01	2.31E+01	2.31E+01						
Lead		ND	1.50E-01	5.00E-02									
Magnesiuum U		3.24E+00	3.34E+01	2.02E+01	2.21E+01	2.21E+01	2.21E+01						
Manganese U		ND	1.55E+01	5.17E+00	5.94E+00	5.94E+00	5.94E+00						
Potassium U		3.85E+00	5.99E+00	5.02E+00	3.00E+00	3.00E+00	3.00E+00						
Sodium U		1.18E+01	5.82E+01	3.68E+01	3.19E+01	3.19E+01	3.19E+01						
Zinc		ND	3.00E-02	5.00E-03	ND								
Naphthalene U		ND	2.00E-01	8.00E-02	4.00E-02	4.00E-02	4.00E-02						
Heptachlor Epoxide	2.20E+02	ND	8.00E-04	2.70E-04									
Phenols	1.42E+01	ND	1.90E-02	4.75E-03									

U= unknown, no toxicity constants for the Environmental Media available

WORKSHEET 2
TOXICITY INFORMATION

Site: Amphenol Route 8
Date Prepared: August 17, 1987
Prepared by: M. Wulff
Verified by: TAS
Date: 9/16/87

CHEMICAL	TOXICOLOGIC CLASS	EPA RATING VALUE ORAL	EPA RATING VALUE INHALATION	WATER T.C.	SOIL T.C.	AIR T.C.
Acetone	NC					
Arsenic and compounds	PC	A	A	4.07E+00	2.03E-04	4.07E+01
	NC	9	9	1.80E+01	9.00E-04	1.80E+02
Barium and compounds	NC	10	10	4.08E+00	2.04E-04	4.08E+01
Benzene	PC	A	A	7.71E-03	3.86E-07	7.71E-02
	NC	5	10	1.17E-01	5.85E-06	1.18E+02
2-Butanone	NC	10	10	7.75E-03	3.87E-07	7.75E-02
Chlorobenzene	NC	4	1	1.43E-01	7.14E-06	2.79E-01
Chlorodibromomethane	NC	6	6	1.82E+00	9.09E-05	1.82E+01
Chloroform	PC	B2	B2	5.63E-02	2.81E-06	5.63E-01
1,1-Dichloroethane	NC	7	7	2.58E-02	1.29E-06	2.58E-01
1,2-Dichloroethane (EDC)	PC	B2	B2	5.86E-02	2.93E-06	5.86E-01
	NC	10	8	1.76E-02	8.80E-07	1.10E+00
1,1-Dichloroethene	PC	C	C	1.23E-01	6.14E-06	1.23E+00
	NC	7	5	3.71E-01	1.86E-05	5.65E+00
1,2-Dichloroethene (trans)	NC	5	5	5.29E-02	2.65E-06	5.29E-01
Dichloromethane	PC	B2	B2	NA	NA	NA
	NC	10	10	9.20E-04	4.60E-08	6.20E-03
Ethylbenzene	NC	4	4	1.10E-02	5.52E-07	1.10E-01
Heptachlor Epoxide	PC	B2	B2	8.28E+00	4.14E-04	8.28E+01
Lead and compounds (inorganic)	NC	10	10	8.93E-01	4.46E-05	8.93E+00
PCBs	PC	B2	B2	5.71E-01	2.86E-05	5.71E+00
Phenol	NC	3	10	1.00E-01	5.02E-06	2.49E+00
Toluene	NC	7	7	5.20E-03	2.60E-07	5.20E-02
1,1,2,2-Tetrachloroethane	PC	C	C	4.74E-02	2.37E-06	4.74E-01
1,1,1-Trichloroethane	NC	2	2	7.33E-04	3.67E-08	7.33E-03
Trichloroethene	PC	B2	B2	4.29E-03	2.14E-07	4.29E-02
	NC	5	4	1.05E+00	5.26E-05	2.96E+01
Vinyl chloride	PC	A	A	4.29E-03	2.14E-07	4.29E-02
	NC	10	10	8.77E-02	4.39E-06	8.77E-01
Xylene (mixed)	NC	10	4	4.40E-03	2.20E-07	5.70E-01
Zinc and compounds	NC	8	8	1.07E-01	5.33E-06	1.07E+00

WORKSHEET 3.
SCORING FOR INDICATOR CHEMICAL SELECTION:
CALCULATION OF CT AND IS VALUES FOR CARCINOGENIC EFFECTS.

NAME OF SITE: AMPHENOL ROUTE 8
DATE PREPARED: AUGUST 18,1987
ANALYST: M. WULFF
VERIFIED BY: SCL
DATE: 9/30/87

	GROUND WATER CT		SURFACE WATER CT		SURFACE SOIL CT		SUBSURFACE SOIL CT		IS VALUE		TENATIVE RANK	
CHEMICAL	MAX	REPRES	MAX	REPRES	MAX	REPRES	MAX	REPRES	MAX	REPRES	MAX	REPRES
Benzene	3.08E-03	3.46E-05					1.00E-04	9.50E-06	3.18E-03	4.41E-05	10	10
1,1-Dichloroethene	8.49E-02	3.51E-03	3.69E-04	1.60E-05					8.52E-02	3.52E-03	4	6
1,2-Dichloroethane	1.70E-01	2.74E-03	1.76E-04	3.96E-03					1.70E-01	6.70E-03	3	3
Chloroform	8.45E-02	3.78E-03	6.19E-04	4.67E-05					8.51E-02	3.83E-03	5	5
1,1,2,2-Tetrachloroethane	4.74E-03	5.50E-05							4.74E-03	5.50E-05	9	9
Trichloroethene	6.01E-02	3.14E-03	2.19E-04	1.87E-05			1.22E-06	2.16E-07	6.03E-02	3.16E-03	6	7
Vinyl Chloride	4.72E-02	4.14E-03	3.00E-04	4.46E-05					4.75E-02	4.18E-03	7	4
PCBs	1.32E+00	2.55E-02	2.40E-03	3.44E-04	1.91E-04	1.19E-05	3.80E-01	8.32E-03	1.71E+00	3.42E-02	1	2
Arsenic	1.26E-01	4.07E-02	1.10E-01	1.10E-01					2.36E-01	1.51E-01	2	1
Heptachlor Epoxide	6.62E-03	2.20E-03							6.62E-03	2.20E-03	8	8

WORKSHEET 4.
SCORING FOR INDICATOR CHEMICAL SELECTION:
CALCULATION OF CT AND IS VALUES FOR NONCARCINOGENIC EFFECTS.

NAME OF SITE: AMPHENOL ROUTE 8
DATE PREPARED:AUGUST 20, 1987
ANALYST: M. WULFF
VERIFIED BY: SCL
DATE: 9/30/87

	GROUND WATER CT		SURFACE WATER CT		SURFACE SOILS CT		SUBSURFACE SOILS CT		IS VALUE		TENATIVE RANK	
CHEMICAL	MAX	REPRES	MAX	REPRES	MAX	REPRES	MAX	REPRES	MAX	REPRES	MAX	REPRES
Arsenic	5.58E-01	1.80E-01	4.86E-01	4.86E-01					1.04E+00	6.66E-01	4	2
Benzene	4.68E-02	5.25E-04							4.68E-02	5.25E-04	12	16
Toluene	1.82E-01	2.32E-02	5.72E-05	4.32E-06			6.76E-05	6.40E-06	1.82E-01	2.33E-02	7	7
Ethylbenzene	5.50E-02	4.37E-03	6.16E-04	3.16E-05			3.92E-06	4.91E-07	5.56E-02	4.40E-03	10	11
Chlorobenzene							1.93E-07	5.57E-09	1.93E-07	5.57E-09	21	21
1,1-Dichloroethene	2.56E-01	1.06E-02	1.11E-03	4.82E-05					2.57E-01	1.06E-02	6	9
1,1-Dichloroethane	3.87E-02	1.07E-02	5.16E-03	1.74E-03	6.27E-08	1.57E-08	2.94E-07	1.75E-08	4.39E-02	1.24E-02	14	8
1,2-Dichloroethane	5.10E-02	8.24E-04	5.28E-05	6.16E-06					5.11E-02	8.30E-04	12	13
1,2-Dichloroethene(trans)	1.90E+00	3.07E-01	1.48E-02	2.29E-03			1.88E-05	2.78E-06	1.92E+00	3.10E-01	3	3
1,1,2,2-Tetrachloroethane	4.55E-02	5.28E-04							4.55E-02	5.28E-04	13	15
Trichloroethene	1.47E+01	5.73E-01	5.36E-02	4.57E-03			3.00E-04	5.31E-05	1.48E+01	5.78E-01	1	4
1,1,1-Trichloroethane	4.10E-03	4.76E-04	1.13E-04	9.97E-06			1.50E-08	1.37E-09	4.22E-03	4.86E-04	16	17
Methylene Chloride	1.47E-03	5.07E-05							1.47E-03	5.07E-05	19	20
Vinyl Chloride	9.65E-01	8.45E-02	6.14E-03	9.12E-04					9.71E-01	8.55E-02	5	5
Dibromochloromethane	1.09E-01	1.55E-03							1.09E-01	1.55E-03	9	12
2-Butanone	4.50E-02	4.74E-04							4.50E-02	4.74E-04	21	19
Xylenes	1.50E-02	4.97E-03	5.72E-04	2.49E-05			1.80E-05	1.64E-06	1.56E-02	5.00E-03	15	10
Barium	1.22E+00	6.94E-01	8.16E-01	8.16E-01					2.04E+00	1.51E+00	2	1
Lead	1.34E-01	4.47E-02							1 34E-01	4.47E-02	8	6
Zinc	3.21E-03	5.35E-04							3.21E-03	5.35E-04	17	14
Phenols	1.90E-03	4.75E-04							1.90E-03	4.75E-04	18	18

NAME OF SITE: AMPHENOL ROUTE 8
DATE PREPARED: AUGUST 20, 1987
ANALYST: M. WULFF
VERIFIED BY: TAS/SCL
DATE: 9/30/87

WORKSHEET 5.
SCORING FOR INDICATOR CHEMICAL SELECTION:
EVALUATION OF EXPOSURE FACTORS AND FINAL CHEMICAL SELECTION.

	IS VALUE		RANKING		WATER SOLUBILITY	VAPOR PRESSURE	HENRY'S LAW CONSTANT		HALF-LIFE (DAYS)				MOBILITY * INDEX	FREQUENCY DETECTED	
CHEMICAL	PC	NC	PC	NC	(mg/L)	(mm HG)	(atm-m3/mole)	Koc	GW	SW	SOIL	AIR			IC
Arsenic	1.51E-01	6.66E-01	1	2		0.00E+00								5 / 12	
Barium		1.51E+00		1										3 / 4	
Benzene	4.41E-05	5.25E-04	10	16	1.75E+03	9.52E+01	5.59E-03	8.30E+01					3.30E+00	6 / 133	
Chloroform	3.83E-03		5		8.20E+03	1.51E+02	2.87E-03	3.10E+01		0.3/30.0		80.0/-	4.60E+00	22 / 125	
Dibromochloromethane		1.55E-03		12										2 / 95	
1,1-Dichloroethane		1.24E-02		8	5.50E+03	1.82E+02	4.31E-03	3.00E+01		1.0/5.0		45.0/-	4.52E+00	100 / 136	+
1,1-Dichloroethene	3.52E-03	1.06E-02	6	9	2.25E+03	6.00E+02	3.40E-02	6.50E+01		1.0/6.0		2.00/-	4.32E+00	18 / 125	
1,2-Dichloroethane	6.70E-03	8.30E-04	3	13	8.52E+03	6.40E+01	9.78E-04	1.40E+01		0.17/-		36.0/127	4.59E+00	27 / 125	
1,2-Dichloroethene(trans)		3.10E-01		3	6.30E+03	3.24E+02	6.56E-03	5.90E+01		1.0/6.0		2.1/-	4.54E+00	113 / 136	
Ethylbenzene		4.40E-03		11	1.52E+02	7.00E+00	6.43E-03	1.10E+03		1.5/7.5		1.46/-	-1.00E-02	45 / 136	
Heptachlor Epoxide	2.20E-03		8		3.50E-01	3.00E-04	4.39E-04	2.20E+02					-6.30E+00	1 / 4	
PCBs	3.42E-02		2		3.10E-02	7.70E-05	1.07E-03	5.30E+05		2.0/12.9		58.0/-	-1.13E+01	125 / 169	+
Toluene		2.33E-02		7	5.35E+02	2.81E+01	6.37E-03	3.00E+02		0.17/-		1.30/-	1.70E+00	69 / 136	+
1,1,1-Trichloroethane		4.86E-04		17	1.50E+03	1.23E+02	1.44E-02	1.52E+02		0.14/7.0		803.0/1752	3.08E+00	78 /136	
Trichloroethene	3.16E-03	5.78E-01	7	4	1.10E+03	5.79E+01	9.10E-03	1.26E+02		1.0/90.0		3.701/-	2.70E+00	64 /136	+
1,1,2,2-Tetrachloroethane	5.50E-05	5.28E-04	9	15	2.90E+03	5.00E+00	3.81E-04	1.18E+02		0.04/-		584.0/-	2.09E+00	4 / 95	
Vinyl Chloride	4.18E-03	8.55E-02	4	5	2.67E+03	2.66E+03	8.19E-02	5.70E+01		1.0/5.0		1.20/-	5.23E+00	77 /125	+
Xylene		5.00E-03		10	1.98E+02	1.00E+01	7.04E-03	2.40E+02		1.5/9.0		0.50/-	9.20E-01	5 / 15	
Zinc		5.35E-04		14		0.00E+00				PERS		4.80 /20.0		2 / 12	
Phenol		4.75E-04		18	9.30E+04	3.41E-01	4.54E-07	1.42E+01		0.62 /9.00		0.62 /9.00	3.35E+00	1 / 7	
2-Butanone		4.74E-04		19										3 / 15	
Chlorobenzene		5.57E-09		21	4.66E+02	1.17E+01	3.72E-03	3.30E+02					1.22E+00	3 / 11	
Lead		4.47E-02		6		0.00E+00				PERS		4.80 / -		4 / 8	
Methylene Chloride		5.07E-05		20	2.00E+04	3.62E+02	2.03E-03	8.80E+00		6.00 /-		53.20 /-	5.92E+00	4 / 30	

* Soil Mobility Index = Log (Water Solubility * Vapor Pressure) / Koc

APPENDIX B

FATE & TRANSPORT PROFILES FOR INDICATOR COMPOUNDS

VINYL CHLORIDE

General:

Vinyl chloride (chloroethene) is a starting material in the manufacture of PVC and other copolymers. It is moderately water soluble and is an extremely volatile unsaturated aliphatic hydrocarbon. Based on its density, vinyl chloride will float, on a water column if its water solubility were exceeded.

Fate and Transport:

Volatilization is the predominant fate and transport process for vinyl chloride in surface water and surface soil environments. The laboratory volatilization half-life of vinyl chloride from water is 26 minutes while the overall half-lives in air and surface water are 1 day and 1-5 days, respectively. Once in the troposphere, vinyl chloride reacts rapidly ($t_{1/2}$ = few hours) to form hydrogen chloride (HCl) and formyl chloride (HCOCl) and, subsequently, carbon monoxide and hydrogen chloride ($t_{1/2}$ = 20 minutes). Studies indicate that volatilization proceeds so rapidly that the slower fate processes (photolysis, hydrolysis, and bioaccumulation) cannot occur. Sorption and biodegradation studies show minimal evidence that these processes occur for vinyl chloride. Vinyl chloride will be transported with ground water in the predominant flow direction. Vinyl chloride does not tend to bioaccumulate as it can be metabolized. A bioconcentration factor of 1.17 has been reported for fish.

Summary:

The predominant transport process for vinyl chloride from surface soils and surface water is volatilization to the atmosphere followed by oxidation in the troposphere. Vinyl chloride can be leached for contaminated subsurface soils to the ground water and be transported with the ground water flow.

References

Verschueren, K., 1983; Weast, R.C., 1974-1975; Mills, W.B., et al., 1982; U.S. EPA, 1985c; Callahan, M.A., et al., 1979; Mabey, W.R., et al., 1982; Vogel, T.M., et al., 1987.

1,1-DICHLOROETHANE

General:

1,1-Dichloroethane is a highly volatile saturated aliphatic hydrocarbon with a sweet chloroform-like odor. The uses of this chemical includes the manufacture of vinyl chloride; chlorinated solvent intermediate; coupling agent in anti-knock gasoline; paint, varnish, and finish removers; metal degreasing; organic synthesis; and ore flotation. It has a relatively high water solubility and a density greater than water. Thus, excess 1,1-dichloroethane would sink in water.

Fate and Transport:

Volatilization of 1,1-dichloroethane in the environment is the most important fate process. It has a laboratory half-life of 22 minutes. In the atmosphere, 1,1-dichloroethane reacts rapidly with hydroxyl radicals to allow little intact compound to reach the stratosphere. The initial photodissociation product is probably chloroacetyl chloride. The overall half-lives for 1,1-dichloroethane in the atmosphere and surface water are 45 and 1-5 days, respectively. Oxidation (calculated $t_{1/2}$ = 1.5 months) sorption, hydrolysis, biodegradation, and bioaccumulation are not considered important fate processes for 1,1-dichloroethane in the environment based upon the available information.

Summary:

The major environmental transport process for 1,1,-dichloroethane is volatilization from soils and/or surface water to the atmosphere. 1,1-Dichloroethane may be redeposited in the hydrosphere through precipitation, dry transfer, and dry fallout of particles from the adsorbed compound.

References:

Mabey, W.R., et al., 1982; Mills, W.B., et al., 1982; Callahan, M.A., et al., 1979; Vershueren, K., 1983; U.S. EPA, 1985g; Perwak, J. et al., 1980.

TRICHLOROETHENE

General:

Trichloroethene (TCE) is ubiquitous in the environment, although it is not naturally occurring. Widely used as a solvent in industrial degreasing of metals, TCE has minor uses in fumigant mixtures, inhalation anesthesia, and decaffeination of coffee. TCE is a highly volatile unsaturated aliphatic hydrocarbon with a relatively high water solubility. From its density, any TCE in excess of its water solubility would sink to the bottom of the water.

Fate and Transport:

Volatilization of TCE in the environment is its most important fate process. Its laboratory half-life is reported to be 21 minutes. Once the compound enters the troposphere, high temperatures and UV radiation promote rapid degradation ($t_{1/2}$ = 4 days) to hydrochloric acid (HCl), dichloroacetyl chloride, phosgene, carbon monoxide, and hexachlorobutadiene. The overall half-life of TCE in surface water and air is 1-90 days and 4 days, respectively. Limited laboratory studies on the adsorption of TCE onto soils and sediments indicate that TCE does not adsorb to a great extent to pure clays (<5 percent adsorption). Thus, adsorption will not be considered as a major fate process. TCE does not significantly bioaccumulate in the environment as seen by bioconcentration factors of 10^{-17} for bluegills, with a half-life in tissue of less than 1 day. Higher mammals, including man, can degrade TCE to chlorinated acetic acids. Under anaerobic conditions, TCE can degrade to carbon dioxide in subsurface environments. However, biodegradation/ biotransformation is considered of minor significance as an environmental fate process.

Summary:

The major environmental transport process for TCE is volatilization from surface water and soils to the atmosphere. In ground water and subsurface soils, TCE will infiltrate and migrate with the ground water flow.

References:

Callahan, M.A. et al., 1979; Mills, W.B. et al., 1982, U.S. EPA, 1985c; Schuller, T.A., 1983; Wilson and Wilson, 1985.

TOLUENE

General:

Toluene is a flammable, colorless liquid with a sour or burnt odor. It is moderately soluble in water and is miscible with most other organic solvents. Toluene occurs naturally as a component of petroleum oil and is produced indirectly in large volumes during gasoline refining and other operations. The main uses for toluene are as a raw material in the production of benzene and other organic solvents, as a solvent (especially for paints, coatings, gums, oils and resins), and as a gasoline additive to elevate octane ratings. This unsaturated, aromatic hydrocarbon will float in water if its water solubility is exceeded.

Fate and Transport:

The major environmental fate process for toluene is volatilization with an estimated half-life of 5.18 hours. Photooxidation is the primary atmospheric fate process for toluene with benzaldehyde as the principal organic product reported. Direct photolysis of toluene in the troposphere is energetically improbable while oxidation and hydrolysis in aquatic systems are probably not important. Little quantifiable information exists in the literature concerning the photolysis, hydrolysis, oxidation, and bioaccumulation of toluene in the environment. Therefore, these processes are considered to be of minor environmental significance. Biodegradtion of toluene does occur in aquatic and soil environments, but at slow rates. Therefore, biodegradation is not considered a significant fate process.

Summary:

The major environmental transport process for toluene is volatilization from soils or surface water (or both) to the atmosphere as well as fugitive dust emissions and dry deposition of toluene and oxidation products to the aquatic and terrestrial environments.

References:

Callahan, M.A., et al., 1979; Mabey, W.R., et al., 1982; Mills, W.B., et al., 1982; Verschueren, K., 1983; U.S. EPA, 1986a; U.S. EPA, 1985a; U.S. EPA, 1985b; Long, S.C., 1986.

POLYCHLORINATED BIPHENYLS

General:

Polychlorinated biphenyls (PCBs) are a class of chlorinated, aromatic hydrocarbons which had widespread use because of their stability and chemical inertness as well as their dielectric properties. PCBs are widely varied in their physical (oil to liquid to resins) and chemical (soluble to insoluble) properties. In general, PCBs as a class are liquid, denser than water, insoluble in water, and non-volatile. Depending on the properties of individual PCBs, they are used as dielectric fluids, fire retardants, and plasticizers.

Fate and Transport:

Biotransformation and biodegradation are important fate processes for the mono-, di-, and tri-chlorinated biphenyls, are of intermediate importance for tetrachlorinated biphenyls, and are of no importance for penta- and higher chlorinated biphenyls which are completely resistant. Lesser chlorinated hydrocarbons are biotransformed in the environment to chlorobenzoic acids and chlorophenylglyoxylic acid. Sorption, volatilization (aerosol distribution followed by fallout with dust or rain and fugitive dust emissions), and bioaccumulation are other important fate processes. PCBs strongly sorb to sediments and/or suspended particles resulting in extremely long half-lives ($t_{1/2}$ = 52.5 days) and making desorption a possibility for years to come. Volatilization of PCBs results from fugitive dust emissions ($t_{1/2}$ = 10.4 hours). PCBs strongly bioaccumulate in the food chain through desorption from sediments and direct uptake by plants and other aquatic species. Experiments with Daphnia magna show a tendency for bioconcentration factors to increase with increasing chlorine content or decreasing water solubility. Photolysis is a minor fate process for PCBs in natural surface waters. PCBs can be partially dechlorinated with shortwave UV light to yield chlorinated biphenylenes and chlorinated dibenzofurans. Photolysis of PCBs requires an oxygen-depleted atmosphere. The photic zone in natural waters is oxygen-rich from photosynthesis and reaeration; thus, photolysis is not likely to occur in most surface water environments. PCBs are fairly stable and resistant to hydrolysis and oxidation.

Summary:

Environmental transport processes for PCBs include volatilization from soils, surface waters, and sediments; adsorption onto soil particles leading to sedimentation; desorption from soil particles and sediments leading to re-solution; bioconcentration in the food chain; biodegradation of lesser chlorinated hydrocarbons; fugitive dust emissions leading to volatilization and precipitation; and to a small extent, photolysis.

References:

Mabey, W.R., et al., 1982; Callahan, M.A., et al., 1979; Verschueren, K., 1983; Mills, W.B., et al., 1982; U.S. EPA, 1985g; Safe, S., 1983; D'Itri and Kamrin, 1983.

APPENDIX C
SAMPLE CALCULATIONS

Appendix C
Calculation of Subchronic and Chronic Exposures

Dermal Exposure

Soils

$DEX = N_e \times Conc \times 1/BW \times Area \times DA \times Abs_S \times SM$

Where:

DEX - Total exposure (mg/kg/day)
N_e - Number of events per day (1/day)
Conc - Concentration of contaminant in soil (expressed as fraction of total weight)
BW - Body weight (kg)
Area - Amount of skin surface area exposed (cm^2)
DA - Dust adherence (mg/cm^2)
Abs_S - Skin absorption rate of compounds in soil (%)
SM - Soil matrix effect (%)

Surface/Ground Water

$DEX = Conc \times 1/BW \times Area \times FR \times E_t \times Abs_K$

Where:

DEX - Total exposure (mg/kg/day)
Conc - Concentration of contaminant in soil (expressed as fraction of total weight)
BW - Body weight (kg)
FR - Mass flux rate of water across the skin surface; water based ($mg/cm^2/hr$)
E_t - Length of exposure (hr/day)
Abs_K - Percent of contaminant absorbed into the bloodstream

Ingestion

$Ing = Conc \times 1/BW \times Amt \times Abs_W$

Where:

Ing - Total exposure (mg/kg/day)
Conc - Concentration of contaminant in water (mg/L) or soil (mg/kg)
BW - Body weight (kg)
Amt - Amount ingested (liters or kilograms per day)
Abs_W - Percent of contaminant absorbed into the bloodstream

Inhalation

$Inh = Conc \times 1/BW \times BR \times E_t \times Abs_a$

Where:

Inh - Total exposure (mg/kg/day)
Conc - Concentration of contaminant in water (mg/L)
BW - Body weight (kg)
BR - Breathing rate (m^3/hr)
E_t - Length of exposure (hr/day)
Abs_a - Percent of contaminant absorbed into the bloodstream

Inhalation While Bathing

$Inh = \{[(AW \times Conc \times E_1 \times BR)/(2 \times SV)] + [(AW \times Conc \times E_2 \times BR)/BV]\} \times Abs_a \times 1/BW$

Where:

Inh - Total exposure (mg/kg/day)
Conc - Concentration of contaminant in water (mg/L)
AW - Amount of Water used during shower (L)
BW - Body weight (kg)
BR - Breathing rate (m^3/hr)
E_1 - Length of exposure in shower (hr)
E_2 - Length of additional exposure in enclosed bathroom (hr)
SV - Shower volume (m^3)

APPENDIX D

EPA AND IARC APPROACHES TO THE CLASSIFICATION OF CARCINOGENS

IARC CLASSIFICATION SYSTEM

The International Agency for Research on Cancer (IARC) initiated a research program in 1971 to evaluate the carcinogenic risk of chemicals to humans. In 1982, IARC developed a system for categorization of carcinogens based on the strength of evidence for carcinogenicity. Although IARC classifies chemicals based on carcinogenic nature, it does not assess the relevance of experimental laboratory animal data to extrapolation of human risk. IARC's system is in sharp contrast to the EPA categorization system which was adapted from the 1982 IARC system and is a basic element of the risk assessment process. The EPA categorization system differs from the IARC system in that it stresses the weight-of-evidence approach which incorporates the balancing of positive and negative studies. During January 1987, IARC revised its categorization system resulting in changes that incorporate some new features of the EPA system, but digress from it in other ways.

The IARC categorization system is based on a definition of chemical carcinogenesis as the induction by chemicals of neoplasms that are not usually observed, of neoplasms that are commonly observed, and/or of more neoplasms than are usually found.

The evidence for carcinogenicity in humans by IARC can be derived from three types of studies:

1. Case reports of individual cancer patients which include a history of exposure to the chemical in question.

2. Descriptive epidemiological studies.

3. Analytical epidemiological studies (case control and cohort).

The degrees of evidence for carcinogenicity in studies of humans by IARC are defined as:

1. Sufficient evidence of carcinogenicity, which indicates that there is a causal relationship between the agent and human cancer.

2. Limited evidence of carcinogenicity, which indicates that a causal interpretation is credible, but that alternative explanations, such as chance, bias, or confounding could not be adequately excluded.

3. Inadequate evidence, which applies to both positive and negative evidence, indicates that one of two conditions prevailed: a) there were few pertinent data, b) the available studies, while showing evidence of association, did not exclude chance, bias, or confounding.

4. "Evidence Suggesting Lack of Carcinogenicity", which applies when several adequate studies were available which do not show evidence of carcinogenicity.

The assessment of evidence of carcinogenicity from studies in experimental animals by IARC are defined as:

1. Sufficient evidence of carcinogenicity, which indicates that there is an increased incidence of malignant tumors or of an appropriate combination of benign and malignant neoplasms: a) in multiple species or strains, b) in multiple experiments, or c) to an unusual degree with regard to incidence, site, type of tumor, or age of onset. Chemicals for which there is sufficient evidence of carcinogenicity in humans are judged by IARC to present a carcinogenic risk to humans.

2. Limited evidence of carcinogenicity, which means that the data suggest a carcinogenic effect but are limited because of some type of inadequacy in experimental design.

3. Inadequate evidence, which indicates that because of major qualitative or quantitative limitations, the study cannot be interpreted as showing either the presence or absence of a carcinogenic effect.

4. "Evidence Suggesting Lack of Carcinogenicity" applies when several adequate studies involving at least two species show that the chemical does not induce cancer.

The new IARC categories are listed below:

IARC Category		IARC Titles
1	Sufficient evidence from epidemiological studies	(Known) human carcinogen
2A	Sufficient animal evidence Evidence of human carcinogenicity or at least limited evidence from epidemiological studies	Probable human carcinogen

IARC Category		IARC Titles
2B	Sufficient animal evidence and inadequate evidence from human studies OR limited evidence from human studies in the absence of sufficient animal evidence	Possible human carcinogen
3	Inadequate animal evidence and inadequate evidence from human studies	Not classifiable
4	Evidence for lack of carcinogenicity	Noncarcinogenic to humans

EPA CLASSIFICATION SYSTEM

EPA (Fed Register, 1986) has made the following modifications of the IARC (IARC, 1982) approach to classifying human and animal studies. For human studies:

1. "The observation of a statistically significant association between an agent and life-threatening benign tumors in humans is included in the evaluations of risk to humans."

2. "A 'no-data available' classification is added."

3. "A "no evidence of carcinogenicity" classification is added. This classification indicates that no association was found between exposure and increased risk of cancer in well-conducted, well-designed, independent analytical epidemiologic studies.

For animal studies:

1. An increased incidence of combined benign and malignant tumors will be considered to provide sufficient evidence of carcinogenicity if the other criteria defining the "sufficient" category of evidence are met.

2. A statement that increased incidence of benign tumors alone provides "limited" evidence of carcinogenicity is added.

3. Under specific circumstances, such as the production of neoplasms that occur with high spontaneous background incidence, the evidence may be decreased to "limited" if warranted by specific information available on the agent.

4. A "no data available" classification has been added.

5. A "no evidence of carcinogenicity" classification is also added.

Agents that are judged to be in the EPA weight-of-evidence stratification Groups A and B are to be regarded as suitable for quantitative risk assessments. The appropriateness of quantifying the risks from agents in Group C, specifically agents that are at the boundary of Group C and D, would be judged on a case-by-case basis. Agents that are judged to be in Groups D and E should generally not be evaluated as carcinogens using quantitative risk assessments.

Evidence of carcinogenicity from human studies comes from three main sources:

1. Case reports of individual cancer patients who were exposed to the agent(s).

2. Descriptive epidemiological studies.

3. Analytical epidemiologic (case control and cohort) studies.

Three criteria must be met before a causal association can be inferred between exposure and cancer in humans:

1. There is no identified bias which can explain the association.

2. The possibility of confounding has been considered and ruled out as explaining the association.

3. The association is unlikely to be due to chance.

   The weight-of-evidence for carcinogenicity from studies in humans can be categorized by:

   a. Sufficient evidence of carcinogenicity, which indicates that there is a causal relationship between the agent and human cancer.

   b. Limited evidence of carcinogenicity, which indicates that a causal interpretation is credible, but that alternative explanations such as change, bias, or confounding, could not be adequately excluded.

   c. Inadequate evidence.

      i. There were few pertinent data, or

      ii. The available studies, while showing evidence of association, did not exclude chance, bias or confounding.

4. No evidence.

5. No data available.

Assessments of weight-of-evidence for carcinogenicity from studies in experimental animals are classified into five groups:

1. Sufficient evidence of carcinogenicity, which indicates an increased incidence of malignant tumors or combined malignant and benign tumors:

   a. In multiple species or strains; or

   b. In multiple experiments (preferably with different routes of administration or using different dose levels); or

   c. To an unusual degree in a single experiment with regard to incidence, site or type of tumor, or age at onset.

2. Limited evidence of carcinogenicity.

   a. Studies involve a single species, strain, or experiment; or

   b. The experiments are restricted by inadequate dose levels, inadequate duration of exposure to the agent, inadequate period of follow-up, poor survival, too few animals, or inadequate reporting; or

   c. An increase in the incidence of benign tumors only.

3. Inadequate evidence.

4. No evidence of carcinogenicity.

5. No data.

The categorization of overall evidence of carcinogenicity is subdivided into five groups.

Group A: Human carcinogens are used only when there is sufficient evidence from epidemiologic studies to support the causal association between exposure to agent(s) and cancer.

Group B: Probable human carcinogens include agents for which the evidence of human carcinogenicity from epidemiologic studies ranges from almost "sufficient" to "inadequate." B1 is reserved for agents for which there is at least limited evidence of carcinogenicity to humans from epidemiologic studies. The agents for which there is inadequate evidence from human studies or no data from epidemiologic studies, but sufficient

evidence exists from animal studies, would usually be classified as B2.

Group C: Possible human carcinogens are used for agents with limited evidence of carcinogenicity in animals in the absence of human data. It includes a wide variety of evidence:

a. Definitive malignant tumor response in a single well-conducted study,

b. Marginal tumor responses in studies having inadequate design or reporting,

c. Benign but not malignant tumors with an agent showing no response in a variety of short-term tests for mutagenicity, and

d. Marginal responses in a tissue known to have a high and variable background rate.

Group D: Not classified is used for agent(s) with inadequate human or animal evidence of carcinogenicity or for which no data are available.

Group E: No evidence of carcinogenicity for humans is used for agents that show no evidence of carcinogenicity in at least two adequate animal studies in different species or in both adequate epidemiologic and animal studies.

The text for the general weight-of-evidence discussion is taken from proposed guidelines for carcinogen risk assessment (Fed. Reg. 1986).

The EPA Carcinogen Assessment Group (CAG) has evaluated more than fifty chemicals as suspect human carcinogens and developed relative carcinogenic potency factors for each chemical. The ranking of potency indices is subjected to the uncertainty of comparing different routes of exposure and a number of different species. These indices are based on estimates of low dose risk using linear multistage extrapolation from the observed range. Thus, these indices are not valid when compared to potencies in the experimental or observational range, especially if linearity does not exist in this range.

APPENDIX E

TOXICOLOGICAL PROFILES FOR INDICATOR CHEMICALS

1,1-DICHLOROETHANE

References: US EPA 1986b,Perwak et al 1981

Summary of Health Effects Data

Limited toxicological testing of 1,1-dichloroethane has been conducted, although the literature indicates that 1,1-dichloroethane is one of the least toxic of the chlorinated ethanes. A National Cancer Institute (NCI) bioassay on 1,1-dichloroethane was limited due to poor survival of test animals, but some marginal tumorigenic effects were seen. Inhalation exposure to high doses (over 16,000 mg/m^3) of 1,1-dichloroethane caused retarded fetal development in rats. 1,1-Dichloroethane was not found to be mutagenic using the Ames assay. 1,1-Dichloroethane causes central nervous system depression when inhaled at high concentrations, and evidence suggests that the compound is hepatotoxic in humans. Kidney and liver damage was seen in animals exposed to high levels of 1,1-dichloroethane. The oral LD_{50} value in the rat is 725 mg/kg.

Pharmacokinetics and Metabolism

1,1-Dichloroethane is adsorbed by humans and laboratory animals through the lungs, gastrointestinal tract, and skin. Proportions of the dose of 1,1-dichloroethane adsorbed in the skin and gastrointestinal tract are unknown. Distribution of 1,1-dichloroethane in the body is rapid with the liver and kidneys contained the highest concentration of the chemical. Successively lower concentrations occur in the forestomach, stomach and spleen. 1,1-Dichloroethane may readily pass the brain/blood barrier where it is metabolized to a chlorinated ethanol compound and subsequently converted to alcohol and aldehyde dehydrogenases. Excretion of 1,1-dichloroethane from the body is mainly through expired air and urine.

Toxic and/or Carcinogenic Studies

1,1-Dichloroethane vapor is a narcotic. Rats exposed to 32,000 ppm for 30 minutes did not survive. The most consistent findings in animals at concentration above 8,000 ppm for up to 7 hours were pathologic changes in the kidney and liver, and at higher concentrations (nearly 64,000 ppm) damaged the lungs as well. Repeated daily exposure of several species of animals to 1,000 ppm resulted in no pathological or hematologic changes. The liquid applied to the intact or abraded skin of rabbits produced slight adema and very slight necrosis after six daily applications. Immediate, moderate conjunctival irritation and swelling, which subsided within a week, were noted after instillation in the eyes of rabbits. There are no reported cases

of human over-exposure by inhalation; however, prolonged, confined, repeated skin contact can produce a slight burn.

TOLUENE **CAS No. 108-88-3**

Physical Properties

Toluene is a colorless liquid with an aromatic odor similar to benzene.

melting point: -95°C
boiling point: 110.4°C
vapor pressure: 36.7mm at 30°C

Sources

Toluene's primary industrial use is in gasoline blending to increase octane ratings. It is also used as a solvent in surface coatings. It is derived from coal tar, and commercial grades may contain very low amounts of benzene.

Summary of Health Effects

Toluene enters the body primarily through the lungs and the gastrointestinal tract by the processes of inhalation and ingestion. Skin contact is another route of exposure. The amount of absorption of toluene solution is only important when the solution is highly concentrated. The toxic effect which immediately follows the inhalation of high levels of toluene (200ppm) is central nervous system (CNS) depression. Exposures to less than 500ppm have not been associated with any long term organ damage. Once exposure to toluene has ceased, recovery from these types of effects is usually complete.

Environmental Concentrations

A 1983 EPA Health Assessment document for toluene names this chemical the most prevalent hydrocarbon in the atmosphere and

is present at levels ranging from 0.12-57ppb. The main sources of toluene at the atmosphere are gasoline usage and automobile exhaust. Toluene contamination of soil, water, food and air is higher in urban areas than rural areas. The Health Assessment document places the range of toluene levels in water from a trace to 10ppb. It is estimated that the general public may receive as much as .75 mg/week of toluene through ingestion of food and water, depending upon if the area is urban or rural.

Toxiological Information

Non-neoplastic Effects

The toxic effect which immediately follows the inhalation of high levels of toluene (200ppm) is central nervous system (CNS) depression. Symptons of CNS depression include impairment of coordination and sluggishness. Inhalation of higher concentrations (200 - 800ppm) increases the severity of these effects and produces other effects and produces other effects such as nausea, headache, and eye and skin irritation. Exposures to less than 500ppm have not been associated with any long term organ damage. Once exposure to toluene has ceased, recovery from these types of effects is usually complete.

However, long term exposure to toluene at relatively high concentrations has caused brain damage in laboratory animals during experimental studies. There have been some reports in the literature of liver and blood damage after human exposures to greater than 500ppm of toluene, but these types of effects are rare.

Carcinogenic Studies

Long term exposure to small amounts of toluene has not caused cancer in laboratory animals during experimental studies. Toluene is not considered as a carcinogen by EPA

Mutagenic Effects and Adverse Effects on Reproduction

Toluene has not been shown to exhibit any mutagenic activity. Toluene does not seem to induce any biologically significant embryotoxic or teratogenic effects in experimental animals.

Ecotoxicology

In the literature, numerous studies on single species exposed to toluene are described, however, there is insufficient information available to characterize the effects of toluene on the higher levels of organization (i.e., community, ecosystem). The toxicological effects to toluene are considered transient in organisms since toluene is not stored in animal tissue and it is rapidly metabolized and excreted (EPS Canada 1984). Toluene has proven toxic to aquatic organisms (i.e., fish) at concentrations at low as 10 mg/L. The volatile nature of toluene and the brevity of it's half-life ($t_{1/2}$ = 5.18 hours) suggest that the incidence of chronic effects in aquatic organisms is suspect.

At the microorganism level, toluene inhibited cell multiplication in bacteria (Psuedomonas aeruginosa) at 29 mg/L, algae (Microcystis aeruginosa) at 105 mg/L, green algae (Scenedesmus quadricauda) at > 400 mg/L, and protozoans (Uronema parduczi and Entosiphon sulcatum) at > 450 mg/L and 456 mg/L, respectively (Verschueren 1983). Marine bacteria (Psuedomonads) exhibited a loss of chemotatic response at 1,000 to 5,000 mg/L.

In studies with plants exposed to toluene it was apparent they are less sensitive than fish to toluene toxicity. At

245 mg/L of toluene, the algae (Chlorella vulgaris) showed a 50% decrease in cell number. Giant Kelp (Macrocystis pyrifera) demonstrated decreases in growth, respiration, and photosynthesis at 10 mg/L of toluene. Corn and bean seedlings and tea and grape plants exposed to an unspecified concentration of toluene vapor, absorbed and metabolized the vapor in their roots, stems, and leaves (EPS: Canada 1984).

In an acute study of an invertebrate cladoceran (Daphnia magna) the 48 hour EC_{50} was determined as 50 mg/L. Additional invertebrate studies examined the effects of toluene upon saltwater species. Ninety-six hour LC_{50}'s reported for crustaceans include: 9.5 mg/L for grass shrimp (Palaemonetes pugio), 4.3 mg/L for bay shrimp (Crangon franciscorum), and 1050 mg/L for the pacific oyster (Crassostrea gigas). In a series of six static bioassays, grass shrimp (P. pugio) were exposed for 24 hours to toluene at various temperatures, salinities and life stages. The results indicated the LC_{50}'s range from 17 to 38 mg/L and temperature, salinity and life stage do not affect toxicity (US EPA 1980). Several terrestrial invertebrates were exposed to toluene, 10 to 15 ppm proved lethal to houseflies and LD_{50}'s for mosquito larvae (4th in star) and grain weevils were 22 mg/L and 96 mg/L, respectively (EPS: Canada 1984).

The acute effects of toluene contamination have been monitored through many single species tests performed in laboratory media. Studies of the anadromous coho salmon (Oncorhynchus Kisutch) indicated toluene decreased the growth rate of coho salmon fry and 9.36 ug/L was the LC_{50} level. Juvenlie coho salmon (O. kisutch) exposed to toluene in artifical seawater experienced no mortalities at 10 mg/L after 96 hours, 90% mortality in 50 mg/L after 24 hours, 100% mortability in 50mg/L after 48 up to 96 hours, and 100% mortability in 100 mg/L after 48 up to 96 hours. Adults of

the closely related pink salmon (Oncorhynchus gorbuscha) were exposed to toluene for 96 hours a three different temperatures. The 96 hour TL_M's were 6.41 mg/L at 4°C, 7.63 mg/L at 8°C, and 8.09 mg/L at 11oC (Verschueren 1983). Although this data suggests that toxicity increases with decreasing temperature, there is insufficient data available to verify this hypothesis.

In a series of softwater, static bioassays, Pickering (1966) determined the 24,48, and 96 hour TL_m's were 46.3 mg/L. 46.3 mg/L, and 34.3 mg/L for fathead minnows (Pimephales promelas), all 24 mg/L for the bluegill (Lepomis macrochirus), all 57.7 mg/L for goldfish (Carassius auratus), and 62.81 mg/L, 60.9 mg/L, and 59.3 mg/L for guppies (Lebistes reticulatus). Using fathead minnows (P. promelas), Pickering (1966) performed the bioassay in hardwater and found the 24, 48, and 96 hour TL_M's were 56 mg/L, 56 mg/L, and 42.3 mg/L. Ninety-six LC_{50}'s of 22.8 mg/L, 70 mg/L and 240 mg/L are listed in the literature for goldfish (L. auratus), dace (unspecified species) and channel catfish (Ictalurus punctatus), respectively (Verschueren 1983, EPS: Canada 1984). In studies using saltwater species, 96 hour LC50 values of 7.3 mg/L and 277 to 485 mg/L were reported for the striped bass (Morone saxatilis) and Sheepshead Minnow (cyprinodon variegatus), respectively (US EPA 1980, Verschueren 1983).

No chronic data is available for freshwater species. At 5 mg/L of toluene, the hatching success and survival of sheepshead minnows (C. variegatus) was affected (US EPA 1980).

According to the Environmental Protection Service of Canada (1984) toluene floating on the water surface poses a threat to avian species. Toluene may cause a loss of insulation by

destroying waxes and other substances which waterproof and trap air in fatheads.

Currently, no Ambient Water Quality Criteria (AWQC) have been established by the US EPA for the protection of aquatic life from toluene contamination.

Regulatory Standards

Toluene is not classified as a carcinogen by either the EPA or IARC. The American Conference of Governmental Industrial Hygienists (ACGIH) has recommended 100ppm as the TLV in an occupational setting.

References

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US EPA. 1986. Quality Criteria for Water 1986. EPA 440/86-001

US EPA. 1983. Health Assessment Document for Toluene, EPA600/8-82-008F.

TRICHLOROETHENE

Synonyms

1,1,2-trichloroethene, acetylene trichloride, ethinyl trichloride, ethylene trichloride, TCE, TRI

Physical Properties

Trichloroethene (TCE) is a colorless, non-flammable liquid with a sweet odor resembling chloroform.

melting point: -84.8°C
boiling point: 87°C
vapor pressure: 58 mm Hg at 20°C

Sources

TCE has a variety of uses as an industrial solvent. Its major use is as a solvent for removing grease from metal parts. It is used as a building block in the manufacture of other chemicals and has had a limited use as a surgical anesthetic and analgesic. Household products which may contain TCE include typewriter correction fluid, paint removers, adhesives, spot removers, cleaning fluids, or rugs, and metal cleaners.

Summary of Health Effects Data

The acute toxicity of trichloroethene is relatively low, mainly causing central nervous system depression at high concentration levels. In experimental animals, kidney and liver toxicity may be induced by chronic exposure at elevated doses. There is evidence that trichlorethene is carcinogenic in rodents at high concentrations, but the significance of these findings with respect to low-level human exposure is controversial. Extensive epidemiological investigations have failed to substantiate an increased carcinogenic risk for humans.

Environmental Concentrations

Trichloroethene (TCE) is a colorless organic solvent widely used in the degreasing of metals. TCE has no natural sources, therefore all inputs of TCE to the environment come from anthropogenic sources. The production of TCE has declined in recent years from the 238.2 million pounds which were manufactured in the United States in 1982.

Utilizing data from 2300 monitoring points, Brodzinsky and Singh (as cited in Technical Resources Inc. 1988) determined

that the mean TCE concentration in air was 30 parts per trillion (ppt) in rural/remote areas, located near emitters. In the northern hemisphere, the average background TCE level in air ranges from 11 to 30 ppt (US EPA, 1985).

The detection of TCE in drinking water supplies, surface waters, the oceans, and aquatic organisms indicate that TCE is widely distributed in the aquatic environment. In an analysis of drinking water from 133 cities using surface water as a source, the average TCE levels ranged from 0.06 to 3.2 ug/L (Technical Resources, Inc. 1988). In a study of 25 cities utilizing ground water for drinking water, the level of TCE detected ranged from 0.11 to 53.0 ug/L (Technical Resources, Inc. 1988). An analysis of the US EPA STORET Data Base indicated that TCE was positively detected at 2603 of 9295 surface water stations.

Data extracted from the US EPA STORET Data Base indicated that TCE was detected in 20 of 338 soil samples. In soils sampled near TCE producers and users, levels of up to 5.6 ug/kg were found.

TCE has also been detected in a variety of foods including dairy products (0.3 to 10 ug/kg), meat (12,000 to 22,000 ug/kg), beverages (0.02 to 60 ug/kg), fruits and vegetables (1.7 to 5 ug/kg), and fresh bread (7 ug/kg). Samples of U.S. margarine were found to contain TCE levels from 440 to 3600 ug/kg (Technical Resources, Inc. 1988).

Toxicokinetics

Trichloroethene can be absorbed by dermal or oral contact, or by inhalation. Absorption by the dermal route is normally not high enough to elicit toxic effects. Pulmonary uptake of the substance is rapid, and distribution occurs to all body tissues with a considerable fraction in adipose (fatty) tissue. It readily crosses the placental barrier. In humans part of the absorbed trichloroethene (about 10%) is expired unchanged in exhaled air. Metabolic conversion in the liver results in urinary excretion of 30-50% as trichloroethanol (partly as a glucuronide) and 10-30% as a glucuronide) and 10-30% as trichloroacetic acid. Estimation of these metabolites in urine may be utilized for the biological monitoring of exposure. After a single exposure, the level of trichloroacetic acid in blood and urine increases for up to 20-40 hrs, whereupon the concentration decreases with a half-life of 70-100 hrs. Although elimination from the tissues occurs at a slow rate, virtually all the trichloroethene from a single high dose is excreted within 48 hours of administration.

Toxicodynamics

Non-neoplastic effects: Trichloroethene has a low acute oral toxicity in mammals with LC_{50} values in the range 5,000-15,000 ppm. In humans, higher concentrations of this volatile solvent have anesthetic as well as analgesic properties and may occasionally elicit cardiac arrhythmias. Chronic exposure to high levels has been reported to induce neurotoxic symptoms like ataxia, sleep disturbances and psychotic episodes as well as neuropathy of the cranial nerves. Humans exposed to extremely high concentrations of trichloroethene have experienced liver and kidney damage similar to the effects noted in animal studies. The induction of irreversible neuropathies may involve decomposition products of trichloroethene like highly toxic dichloroacetylene. This idea is supported by the finding that such effects have not been found consistently in epidemiological studies involving high exposure levels.

Carcinogenicity Studies: There is evidence that trichloroethene, with and without epoxide stabilizers, induces liver tumors in mice upon inhalation or oral administration of high doses. There is limited evidence that this solvent also induces renal tumors associated with toxic nephrosis in male rats, but this assay (NTP, 1982) has been considered inadequate to evaluate the carcinogenic response.

The hepatocarcinogenic action of trichloroethene in mice has been associated with peroxisome induction (caused by the metabolite trichloroacetic acid). Opinions differ as to the significance of these findings with respect to its relevance to man. Further, the suitability of the linearized multistage model used by the USEPA for low-dose extrapolation with respect to this type of rodent carcinogen has been questioned.

A number of epidemiological investigations including occupationally exposed population groups have been carried out to examine the possible carcinogenic action of trichloroethene, but so far no adequate support for a carcinogenic action in humans has been obtained. These studies tend to support the view that the carcinogenic potency factor derived by EPA, which is of the same order of magnitude as for the well established human carcinogens benzene and vinyl chloride, represents a significant overestimation of risk. It appears extremely unlikely that if trichloroethene is a potent human carcinogen it would have escaped detection in the epidemiological surveys already conducted.

Mutagenic Effects and Adverse Effects on Reproduction: Due to the presence of mutagenic impurities and other factors

present in trichloroethene, the results from short-term mutagenicity testing have been ambiguous. The mutagenic activity of trichloroethene must be regarded as low or non-existent.

Trichloroethene does not seem to induce any biologically significant embryotoxic or teratogenic effects in experimental animals.

Ecotoxicology

Although trichloroethene (TCE) contamination is widespread in the aquatic environment, there is a lack of information assessing the effects of TCE on aquatic organisms and aquatic ecosystems. Almost all the studies have been performed using single species exposed in laboratory media, therefore, the effects of TCE on different life stages and higher levels of organization (e.g., ecosystems) need to be determined in order to further assess the ecotoxicity of TCE.

Exposure of the freshwater algae (Phaeodactylum tricornutum) to 8,000 ug/L of TCE caused a decrease in 14 C uptake. The LC50 values for two freshwater invertebrates (Daphnia magna and Daphnia pulex) are 64,000 and 45,000 ug/L of TCE, respectively. No chronic effects were observed in these two freshwater invertebrates when exposed to 10,000 ug/L of TCE (US EPA 1980). Lay et al. (as cited in US EPA 1985b) performed studies on the invertebrate (D. magna) and several species of phytoplankton exposed to TCE in a natural pond. The invertebrates demonstrated a toxic response at the 25 mg/L level and the invertebrate population was eliminated at a concentration of 110 mg/1. At both concentrations of TCE the abundance of phyoplankton increased, but this increase is attributable to a decrease in the abundance of herbivores. Exposure of fathead minnows (Pimephales promelas) to TCE in flow-through and static test systems yielded LC_{50}'s of 40,700 and 66,800 ug/L, respectively. A loss of equilibrium was observed in fathead minnows (P. promelas) exposed to 21,900 ug/L of TCE. The 96-hour LC_{50} for bluegills (Lepomis macrochirus) was attained at a TCE concentration of 44,700 ug/L. In other acute toxicity tests using bluegills (L. macrochirus) exposed to chlorinated hydrocarbons, a correlation between toxicity and degree of chlorination was shown; an increase in chlorine content causes an increase in lethality. For TCE, a bioconcentration factor of 17 was calculated using bluegills (L. macrochirus). Considering the half-life of TCE in tissues is less than one day the occurrence of chronic toxicity in aquatic organisms in questionable (US EPA 1980).

In studies of saltwater species, erratic swimming, uncontrolled movement, and loss of equilibrium were observed

in sheepshead minnows (Cyprinodon variegatus) and grass shrimp (Palaemonetes pugio) exposed to 20,000 and 2,000 ug/L of TCE respectively (US EPA 1980).

Regulatory Standards

On the basis of the long-term studies in rodents, the EPA has classified trichloroethene as a Group B2 carcinogen; probable human carcinogen with an oral carcinogenic potency factor of 0.011 $(mg/kg/day)^{-1}$. IARC considers that only limited evidence is available that trichloroethene is carcinogenic in mice and has classified the substance in Group 3 (non-classifiable as to its carcinogenicity for humans). The position of the IPCS International Task Group concerning the induction of tumors in rodents was that "the significance of these findings needs to be evaluated in the context of further studies on the mechanism of action of trichloroethene." In the European Common Market this solvent is classified as "Harmful" (X).

According to USEPA, a mutagenic potential cannot be ruled out. But USEPA takes the position that if the compound is mutagenic, the available data suggest that the substance would be a very weak, indirect mutagen. IARC has judged available evidence to be inadequate to assess the mutagenicity of trichloroethene.

The current American Conference of Governmental Industrial Hygienists (ACGIH) 8 hrs Time Weighted Average (TWA) Threshold Limit Value (TLV) for trichloroethene is 50 ppm (270 mg/m^3). The ambient water quality criterion for the protection of aquatic life in freshwater has been set at 45 mg/L.

References

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US EPA, Office of Research and Development. 1985. Health Assessment Document for Trichloroethylene: Final Report.

US EPA, 1980, Ambient Water Quality Criteria for Trichloroethylene. EPA-440/5-80-077.

World Health Organization, International Agency for Research on Cancer, IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Suppl. 4, Lyon, October 1982.

POLYCHLORINATED BIPHENYLS (PCBs)

References:Safe, S., 1983; D'Itri, F.M., 1983; EPA 1984d

Summary of Health Effects Data

Humans exposed to PCBs (in the workplace or via accidental contamination of food) reported adverse effects including chloracne (a long-lasting, disfiguring skin disease), impairment of liver function, a variety of neurobehavioral and affective symptoms, menstrual disorders, minor birth abnormalities, and probably increased incidence of cancer. Animals experimentally exposed to PCBs have shown most of the same symptoms, as well as impaired reproduction; pathological changes in the liver, stomach, skin, and other organs; and suppression of immunological functions. PCBs are carcinogenic in rats and mice and, in appropriate circumstances, enhance the effects of other carcinogens. Reproductive and neurobiological effects of PCBs have been reported in rhesus monkeys at the lowest dose level tested, (11 ug/kg body weight/day over several months).

Pharmacokinetics and Metabolism

One of the problems associated with understanding the toxicokinetics of PCBs products is that they are mixtures of many different isomers, each with its own characteristic kinetics or behavior in the animal body. PCBs can be absorbed by dermal or oral contact or by inhalation, although quantitative data seem to be lacking with regard to the latter route of exposure. Dermal absorption of PCB contaminated oils and inhalation of PCBs absorbed onto dust particles are minor routes of absorption and ingestion of PCBs represents the principal mode of entry into the organism. Several studies indicate that PCBs are readily absorbed from the gastrointestinal tract. The rate of metabolic conversion of PCBs is mainly a function of the degree of chlorination, and some isomers are relatively readily metabolized to polar compounds which can be excreted. However, PCB sulphones are formed from some PCB which specifically accumulates in certain tissues, e.g. the lung. A main concern is the high persistence of unchanged bioaccumulated PCBs in fatty tissue from where it is only slowly eliminated.

Toxic and/or Carcinogenic Studies

Whereas the acute toxicity of the PCBs to mammals is relatively low, a diversity of toxic effects is noted upon chronic exposure at low levels involving several target tissues and organs accompanied by generalized effects like anorexia and weight loss. Notable pathological findings involve the liver (hepatomegaly, fatty liver, necrosis), skin (hyperpigmentation, hyperkeratinization, chloracne), immune system (thymus atrophy,

immunosuppression), nervous system (hyperactivity and retarded learning ability in monkeys). PCBs also induce fetotoxicity in several animal species upon low level administration to the mother (monkeys, 1-5 ppm in diet).

PCBs have been demonstrated to induce liver tumors in rats and mice in some studies and EPA has classified these compounds as Group B2 carcinogen. PCBs are among the more potent experimental carcinogens evaluated by the Agency. However, the applicability of the linearized multistage model in this case may be questioned, and the potency factor may represent an appreciable over-estimation of risk. The results from short-term tests have been mainly negative. PCBs have been classified by some authorities as carcinogens of the promoter type.

VINYL CHLORIDE

Synonyms

(mono)chloroethene, (mono)chloroethylene, ethylene monochloride, vinyl C monomer, Trovidur, VC

Physical Properties

Vinyl chloride is a highly flammable, colorless gas with a faintly sweet odor.

melting point: -153.8°C
boiling point: -13.4°C
vapor pressure: 2660 mm Hg at 25°C

Sources

Vinyl chloride's major use is in the manufacture of polyvinyl chloride (PVC), which is used to make pipes, wire and cable coatings, packaging materials, furniture and automobile upholstery, wall coverings, housewares, and automotive parts. Vinyl chloride is used to a lesser extent as a refrigerant gas and in the manufacture of other chlorinated compounds.

References

Environmental Protection Service: Canada. 1985. Vinyl Chloride, Environmental and Technical Information for Problem Spills. Prepared as part of the series: ENVIRO TIPS.

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Klaasen, C.D., Amdur, M.O., and Doull, J. eds., Casarett and Doull's Toxicology. 1986. MacMillan Publishing Company, New York.

Technical Resources. Inc. Toxicological Profile for Vinyl Chloride (Draft). 1988. Prepared By: Technical Resources, Inc. Prepared For: The Agency for Toxic Substances and Disease Registry, U.S. Public Health Service.

USEPA. 1987. Health Advisories for 25 Organics. Washington, D.C.:NTIS PB 87-235578.

USEPA. 1986. Quality Criteria for Water 1986. EPA 440/5-86-001.

Summary of Health Effects Data

Vinyl chloride causes depression of the central nervous system and may cause death due to narcosis. Long-term exposure can lead to a syndrome which includes liver and kidney damage, thickening of the skin, changes in the circulation and bone structure of the digits, and hematologic effects. Vinyl chloride has been proven to cause cancer in exposed workers. An increased incidence of cancers of the livers, brain, lungs, digestive system, and the blood-forming tissues has been noted. Vinyl chloride causes toxic effects to the fetus and may cause developmental defects.

Environmental Concentrations

Vinyl chloride is a synthetic chemical, all inputs of vinyl chloride to the environment come from anthropogenic sources. In 1986, manufacturers in the United States, produced an estimated 8.5 to 8.6 billion pounds of vinyl chloride.

In areas located near a vinyl chloride or polyvinyl chloride (PVC) manufacturer, concentrations of vinyl chloride in the air have ranged from trace levels to 105 ug/m^3; levels above 2600 ug/m^3 have also been reported. Air samples taken in the vicinity of landfills have contained vinyl chloride levels ranging from below the detection limit to 23.64 ug/m^3 (Technical Resources, Inc. 1988).

Concentrations of vinyl chloride as high as 9.8 ug/l have been observed in surface waters, but the low water solubility and high volatility of vinyl chloride limits the levels occurring in surface waters. In finished drinking water, vinyl chloride has been found at levels up to 10 ug/L, but most monitoring efforts have reported non-detectable concentrations (Technical Resources, Inc. 1988). However, it is possible for vinyl chloride to migrate from PVC pipes into drinking water. The concentration of vinyl chloride in the water is directly proportional to the residual level in the pipe. During the 1982 EPA Ground Water Supply Survey, only 0.74% of 945 test sites contained detectable levels of vinyl chloride with a maximum level of 8.4 ug/L (Technical Resources, Inc. 1988). In other studies, vinyl chloride in ground water was found at concentrations of 840 ug/L or below.

No data regarding vinyl chloride concentrations in soil were located in the literature.

The occurrence of vinyl chloride in foodstuffs is a result of its migration from PVC food wrappings. Vinyl chloride has been found in vinegar at levels up to 9.4 ppm, in edible oils at 0.15 to 14.8 ppm, and in butter at 0.05 ppm when the foods were stored in PVC containers (Technical Resources, Inc. 1988).

Toxicokinetics

Vinyl chloride is rapidly absorbed from the lungs and the gastrointestinal tract. Studies have indicated that an average of 42% of an inhalation dose was retained in humans and that vinyl chloride is completely absorbed from the gastrointestinal tract. Significant amounts of vinyl chloride are not absorbed through the skin. In vivo distribution of vinyl chloride is rapid and widespread, but is influenced by metabolism. After metabolism of the chemical was experimentally blocked, highest levels of vinyl chloride were found in fat, with lesser amounts in the blood, liver, kidney, muscle, and spleen. If metabolisms was not blocked, highest levels were found in the liver and kidney. Vinyl chloride may be metabolized by three different pathways. The first pathway, which operates under low concentrations, consists of oxidative transformation of vinyl chloride to 2-chloroethanol, then to 2-chloroacetaldehyde, and finally to 2-chloroacetic acid. When this pathway becomes saturated, 2-chloroethanol may be transformed to a peroxide in the presence of catalase and hydrogen peroxide. A third pathway uses the mixed-function oxidase system to change vinyl chloride into 2-chloroethylene oxide, a highly reactive epoxide intermediate that spontaneously rearranges to form 2-chloroacetaldehyde. Several studies have indicated that saturation of these metabolic pathways occurs at 200 to 250 ppm in the air. Elimination of unchanged vinyl chloride from the lungs is insignificant at low concentrations. Excretion of the metabolites into the urine represents the most important route of elimination.

Toxicodynamics

Non neo-Plastic Effects: The acute toxicity of vinyl chloride is low and is associated with depression of the central nervous system. Dizziness, giddiness, euphoria, ataxia, headache, and narcosis have been caused by acute exposure of workers to 8,000 to 20,000 ppm (0.8 to 2.0%) in the air. Unspecified concentrations of vinyl chloride have caused death in exposed workers due to narcosis. The oral LD_{50} in the rat is 500 mg/kg, and inhalation of 100,000 ppm is lethal to guinea pigs after 30 to 60 minutes of exposure. Chronic, low-level exposure has been associated with subtle signs of neurologic and psychiatric disease. Mild distal axonal neuropathy, suggestive of a dying back syndrome, was seen in 45 of 64 exposed workers.

Chronic exposure to vinyl chloride has also caused damage to the liver and kidney. Such evidence of liver damage as abdominal pain, hepatomegaly, portal hypertension, thrombocytopenia, and cirrhosis have been observed in exposed workers. Adverse kidney effects, such as increased relative weight, increased blood urea nitrogen (BUN), altered urinalysis parameters, and increased intensity of progressive nephrosis compared to the controls, were

seen in rats exposed to 5,000 ppm for 7 hours per day, 5 days per week, for one year.

Vinyl chloride disease is another result of chronic occupational exposure. Symptoms of this disease include severe damage to the liver, scleroderma, acro-osteolysis (dissolution of the ends of the bones in the fingers), thickening of the skin, Raynaud Syndrome (intermittent loss of circulation to the fingers and toes), and hematologic effects. Exposure levels that caused these signs have not been identified in the literature. Animals exposed to vinyl chloride exhibit hepatic effects similar to humans, but the other signs of vinyl chloride disease: acro-osteolysis, Raynaud Syndrome, and scleroderma; have not been reproduced in animals.

Carcinogenicity Studies: Vinyl chloride is a known human carcinogen. Several epidemiological studies have indicated that workers at vinyl chloride manufacturing plants have an increased risk of liver cancer, particularly angiosarcomas. The risk of cancer is greatly increased especially if intermittent high exposures have occurred. Vinyl chloride is also believed to be responsible for an increase of tumors of the brain and CNS, the lung and respiratory tract, the digestive system, the lymphatic system, and the hemopoietic system. Vinyl chloride was experimentally administered to rats, mice,and hamsters in the air. An increased incidence of tumors was observed in all species exposed to 50 ppm or greater.

Mutagenic Effects and Adverse Effects on Reproduction: Vinyl chloride has demonstrated mutagenicity in *in vitro* and *in vivo* mammalian test systems and in the *Salmonella typhimurium* assay.

A study of exposed workers indicated that vinyl chloride causes an exposure- and duration-related decline in sexual function in men and women. Women exposed to vinyl chloride exhibited ovarial dysfunction, benign uterine growths, and prolapsed genital organs. A significant increase in the incidence of fetal loss was associated with exposure to an unspecified level of vinyl chloride. There is inconclusive epidemiological evidence that vinyl chloride causes developmental defects. A study of a Canadian town with a vinyl chloride-manufacturing plant compared to three matched towns without such plants revealed an increased incidence of developmental defects of the musculoskeletal, cardiovascular, central nervous, and urogenital systems of babies born to women in the town with the manufacturing plant. However, there was insufficient data to prove a link between vinyl chloride exposure and an increased incidence of developmental defects.

Ecotoxicology

There is a lack of information regarding the ecological effects of vinyl chloride contamination. Vinyl chloride is a highly volatile compound with a half-life in water of 1 to 5 days. Considering the rapid volatilization rate of vinyl chloride, the incidence of chronic aquatic contamination is suspected to be low. Results of fate modelling studies with vinyl chloride indicate that it should not reside in aquatic ecosystems under natural conditions. The USEPA (1987) has not established criteria for the protection of freshwater and saltwater organisms since no acute or chronic tests have been performed to assess vinyl chloride toxicity to these organisms.

Vinyl chloride elicits an anesthetic response in mammals exposed to concentrations above 500 ppm (EPA: Canada 1985). At higher concentrations, vinyl chloride may act as an asphyxiant to mammals. The toxicity of vinyl chloride to plants varies from 10 to 10,000 ppm in 7-day exposure tests (EPA: Canada 1985).