

**REMEDIAL INVESTIGATION REPORT FOR  
THE 3163 BUFFALO AVENUE SITE  
NIAGARA FALLS, NEW YORK**

**Volume II: Appendices A, B, C, D, E, and F**

**November 1990**

**Prepared for:**

**NEW YORK STATE DEPARTMENT  
OF ENVIRONMENTAL CONSERVATION  
50 Wolf Road  
Albany, New York 12233**



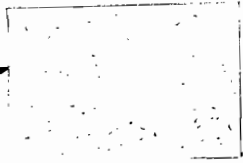
**ecology and environment, inc.**

**BUFFALO CORPORATE CENTER**  
368 PLEASANTVIEW DRIVE, LANCASTER, NEW YORK 14086, TEL. 716/684-8060  
International Specialists in the Environment

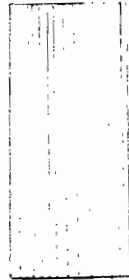
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APPENDIX A

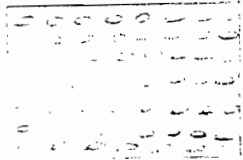
WELL LOGS



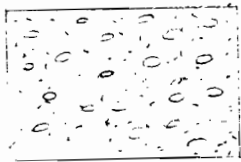
COARSE SILICA SAND PACK



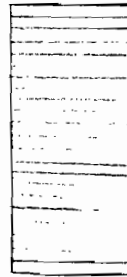
2" to 4" INTERNAL DIAMETER  
304 STAINLESS STEEL CASING



LINEAR, PELLETIZED BENTONITE SEAL



5% BENTONITE GROUT



STAINLESS STEEL WELL SCREEN:  
A ZONE AND 15 WELLS:  
0.010 SLOT SIZE

C, C-D, AND F ZONE WELLS HAVE A 6" BLACK IRON STEEL CASING WHICH EXTENDS TO 11.0' BELOW GROUND SURFACE

DATE  
STARTED 11-3-89  
FINISHED 11-3-89  
SHEET 1 OF 1



E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

HOLE NUMBER SC1  
SURFACE ELEVATION 569.56  
GROUNDWATER DEPTH \_\_\_\_\_

PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave  
Niagara Falls, N.Y.

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE				FIELD IDENTIFICATION OF SOILS	NOTES			
				0	6	6	12	Cl	SI	Sd	Gr					
				12	18	18	24									
0-5																
5-10	2 in. S.S w/ S.S screen Slot size 0.010 in															
10-15																
15-20																
20-25																
25-30																
30-35																
35-40																
40-45																
45-50																
50-55																
55-60																
60-65																
65-70																
70-75																
75-80																
80-85																
85-90																
90-95																
95-100																

B.R. at 6.0'

0.0' - 1.5' - Fill, large pebble to cobble, angular dolomite roadbed, in a matrix of dark brown to black, coarse sand to silt cinder rich, Minor roots and organics, minor assorted brick and cement Fragments and industrial debris

1.5' - 2.0' - Fill, as above, decreasing matrix component

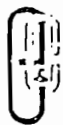
2.0' - 3.4' - Fill, light brown to black, structureless, grain size pebble to clay saturated with volatile organics

5.0' - 6.0' - Clay, tight, stiff, variegated dark gray to black, similar to Pleistocene lake bottom sediments

Sampling done w/ Laskey sampler  
5 ft x 3 in SAMPLER RUN  
0' - 5.0' recovery  
3.4' = 68%

SAMPLER RUN  
5.0' - 6.0'  
Recovery  
1.0' = 100%

DATE 11-7-89  
 STARTED 11-7-89  
 FINISHED 11-13-89  
 SHEET 1 OF 2



E + E DRILLING AND TESTING CO., INC.  
 SUBSURFACE LOG

**Draft**  
 HOLE NUMBER SC 1 E  
 SURFACE ELEVATION 569.46  
 GROUNDWATER DEPTH \_\_\_\_\_

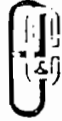
PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave  
Niagara Falls, N.Y.

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER		PROFILE	FIELD IDENTIFICATION OF SOILS	NOTES
				0-6	6-12			
				0	6			
				6	12			
				12	18			
				18	24			
							<p>0.0' - 1.5' - Fill, large pebble to cobble, angular dolomite roadbed, in a matrix of dark brown to black, coarse sand to silt cinder rich, Minor roots and organics, minor assorted brick and cement fragments and industrial debris</p> <p>1.5' - 2.0' - Fill, as above, decreasing matrix component</p> <p>2.0' - 3.4' - Fill, light brown to black, structureless, grain size pebble to clay saturated with volatile organics</p> <p>5.0' - 6.3' - Clay, tight, stiff, variegated dark gray to black, similar to Pleistocene lake bottom sediments</p> <p>6.3' - 17.15' - Dolomite, - B Aquifer, overall, Fine grained, micritic, medium gray to dark gray, minor cavities with calcite sphalerite fillings, dark gray when fresh</p> <p>6.3' - 9.3' - weathered zone, highly fractured, shattered, composed of angular cobble to pebble size fragments, minor</p>	<p>B.R. at 6.3'</p> <p>SAMPLERUN1 0' - 5.0' recovery 3.4' = 68%</p> <p>SAMPLERUN2 5.0' - 6.0' Recovery 1.3' = 100%</p>

CLASSIFICATION/BY John Doerr

DATE  
 STARTED 11-7-89  
 FINISHED 11-13-89  
 SHEET 2 OF 2



E + E DRILLING AND TESTING CO., INC.  
 SUBSURFACE LOG

**Draft**  
 HOLE NUMBER SC1B  
 SURFACE ELEVATION 569.46  
 GROUNDWATER DEPTH \_\_\_\_\_

PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave  
Niagara Falls N.Y.

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE				FIELD IDENTIFICATION OF SOILS	NOTES	
				0	6	6	12	Cl	Sl	Sd	Gr			
				12	18	18	24							
0														
1														
2														
3														
4														
5														
6														
7														
8														
9														
10														
11														
12														
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38														
39														
40														

cavities ~1mm in size  
 9.3'-11.3' - competent, Fresh  
 Horizontal bedding plane fractures  
 11.3-13.1 Fracture zone,  
 60° dipping fractures, showing  
 weathering along fractures,  
 shattered by coring  
 13.1-17.15. Very competent  
 otherwise as before  
 open fractures noted at  
 9.8', 10.0', 15.4', 15.6', 15.7', 16.3',  
 and 1

Draft

DATE  
STARTED 11-13-89  
FINISHED 11-14-89  
SHEET 1 OF 1



E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

HOLE NUMBER SC24  
SURFACE ELEVATION 569.59  
GROUNDWATER DEPTH \_\_\_\_\_

PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave  
Niagara Falls N.Y.

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE				FIELD IDENTIFICATION OF SOILS	NOTES
				0	6	6	12	Cl	Sl	Sd	Gr		
				12	18	18	24						
0.0' - 1.9'												0.0' - 1.9' - Fill, angular pebble to cobble dolomite roadbed with ~ 50% dark brown to black matrix composed of coarse sand to silt size cinder. Minor organics, roots grass. Minor assorted industrial debris. Very soft slight oily sheen.	SAMPLE RUN 0.0' - 5.0' Recovery 1.9' = 38%
1.9' - 3.2'												5.0' - 6.6' - Sandy clay, - possible Fill, moderate rounded pebbles, saturated	SAMPLE RUN 5.0' - 10.0' Recovery 3.2' = 64%
3.2' - 6.6'												6.6' - 8.0' Clay, red, plastic structureless, saturated	
6.6' - 8.0'												8.0' - 8.2' Sandy Clay, saturated, massive, gray	
8.0' - 8.2'													

BR at 8.2'

640088

recycled paper  
recycled paper

CLASSIFICATION/BY John Doerr

DATE: 11-15-89  
 STARTED 11-15-89  
 FINISHED 11-16-89  
 SHEET 1 OF       



E + E DRILLING AND TESTING CO., INC.  
 SUBSURFACE LOG

**Draft** 2  
 HOLE NUMBER SC 2  
 SURFACE ELEVATION 569.39  
 GROUNDWATER DEPTH       

PROJECT Solvent Chemical  
JD 1030

LOCATION 316.3 Buffalo Ave  
Niagara Falls N.Y.

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE	FIELD IDENTIFICATION OF SOILS	NOTES			
				0	6	6	12						
				12	18	18	24						
								CI	SI	Sd	Gr		
INFORMATION PERTAINING TO 0-8.2' FROM SC-2A													
0-1.9'	<p>2.96 in. NX CORE</p>								0.0'-1.9'- Fill, angular pebble to cobble dolomite roadbed with ~ 50% dark brown to black matrix composed of coarse sand to silt size cinder. Minor organics, roots grass. Minor assorted industrial debris. Very soft slight oily sheen.	Recover 1.9' = 38'			
1.9'-5.0'									5.0'-6.6' - Sandy clay, - possible Fill, moderate rounded pebbles, saturated	Recover 3.2' = 64%			
5.0'-6.6'									6.6'-8.0' Clay, red, plastic structureless, saturated				
6.6'-8.0'									8.0'-8.2' Sandy Clay, Saturated, massive, gray				
8.0'-8.2'									8.2-20.0 - Lockport dolomite, B Zone, medium gray to dark gray, dark gray when fresh micritic, fine grained, minor small vugs	Recovery 10.0' = 100%			
8.2-10.2'									8.2'-10.2' weathered zone, highly fractured, numerous cobble sized fragments	Cone Run 2 20' = 150'			
10.2-12.2'									10.2'-12.2' - Competent, massive occasional steeply dipping ~75°, Fine, fractures				
12.2-13.4'									12.2-13.4 - Finely bedded, open weathered bedding plane fractures				
13.4-15.0'									13.4'-15.0' - Competant, massive,				

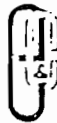
40088

CLASSIFICATION/BY John Doerr



Draft

DATE STARTED 11-15-89  
FINISHED 11-16-89  
SHEET 2 OF 2



E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

HOLE NUMBER SC-21  
SURFACE ELEVATION 569.39  
GROUNDWATER DEPTH \_\_\_\_\_

PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave  
Niagara Falls Ny

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE	FIELD IDENTIFICATION OF SOILS	NOTES
				0	6	6	12			
				12	18	18	24			
							Cl   Sl   Sd   Gr			
									15.0'-15.5' - Open bedding plane fractures, weathered vuggy	CORERUN 2 15.0' - 20.0'
								15.5' - 16.8' - Highly Fractured, weathered zone.		
									16.8' - 20.0' - Competent, massive, minor bedding plane fractures	

640088

CLASSIFICATION/BY John Doerr

Draft

DATE 11-22-89  
STARTED 11-22-89  
FINISHED 11-22-89  
SHEET 1 OF 1

E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

HOLE NUMBER SC31  
SURFACE ELEVATION 570.35  
GROUNDWATER DEPTH

PROJECT Solvent Chemical  
JD 1030

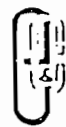
LOCATION 3163 Buffalo Ave  
Niagara Falls NY

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE	FIELD IDENTIFICATION OF SOILS	NOTES
				0	6	6	12			
				12	18	18	24			
							Cl SI Sd Gr			
0-5		SS	1	13	25			0.0'-0.7' Silty loam, dark brown to black, some organic debris, minor fill	Sampling via split spoon	
		SS	2	6	4			0.7'-0.8' Sand, tan, clean, minor sandstone fragments	SAMPLER RUN 0.0'-2.0' RECOVERY = 1.2' = 60%	
		SS	3	4	5			0.8'-1.2' Fill, brick debris in a matrix of silt and sand	SAMPLER RUN 2.0'-4.0' Recovery 1.4' = 70%	
		SS	4	5	7			2.0-3.4' Fill, brick fragments mixed with silt and sand, moist to saturated	SAMPLER RUN 4.0'-6.0' Recovery 2.0' = 100%	
		SS	5	6	20			4.0'-5.0' silt and sand moist	SAMPLER RUN 6.0'-8.0' Recovery 1.8' = 90%	
				11	18			5.0-6.0' silty clay - tan saturated	SAMPLER RUN 8.0'-10.0' Recovery 0.5' = 25%	
				14	40			6.0-7.8' silty sand grading downward to tan to red highly plastic clay, saturated		
								8.0-8.5' Clay, red, moist		
10	2 in. s.s. w/ s.s. screen slot size 0.010 in							B.R. at 9.6'		

640088

CLASSIFICATION/BY R. Meyers

DATE 11-27-89  
 STARTED 11-27-89  
 FINISHED 11-28-89  
 SHEET 1 OF 2



E + E DRILLING AND TESTING CO., INC.  
 SUBSURFACE LOG

**Draft**  
 HOLE NUMBER 2031  
 SURFACE ELEVATION 570.41  
 GROUNDWATER DEPTH 13.3

PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave  
Niagara Falls NY

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE	FIELD IDENTIFICATION OF SOILS	NOTES
				0	6	6	12			
				12	18	18	24			
							CI SI Sd Gr	INFORMATION PERTAINING TO 0-8.5' FROM SC-3A		
0		SS	1	13	25			0.0'-0.7' Silty loam, dark brown to black, some organic debris, minor fill	Sampling via split spoon	
5		2	6	8			0.7'-0.8' Sand, tan, clean, minor sandstone fragments	SAMPLE RUN 0.0' - 2.0' Recovery = 1.2' = 60%		
10		3	4	5			0.8'-1.2' Fill, brick debris in a matrix of silt and sand	SAMPLE RUN 2.0' - 4.0' Recovery 1.4' = 70%		
15		4	5	7			2.0-3.4' Fill, brick fragments mixed with silt and sand, moist to saturated	SAMPLE RUN 4.0' - 6.0' Recovery 2.0' = 100%		
20		5	11	18			4.0'-5.0' silt and sand moist	SAMPLERUN 6.0' - 8.0' Recovery 1.8' = 90%		
								5.0-6.0' silty clay - tan saturated	SAMPLE RUN 8.0' - 10.0' Recovery 0.5' = 25%	
								6.0-7.8' silty sand grading downward to tan to red highly plastic clay, saturated	CORERUN: 11.2 - 16.2 Recovery 4.65' = 93%	
								8.0-8.5' Clay, red, moist		
								8.5-13.5' interbedded weathered dolomite and red clay		
								9.6'-20.0' Lockport dolomite B zone, medium gray to dark gray, micritic, fine grained, minor small vugs		
								13.0'-13.3' High angle ~70° fracture with secondary mineralization		

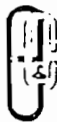
B.R at 9.6'

2.96 in. NX CORE

CLASSIFICATION/BY R. Watt

Draft

DATE  
STARTED 11-27-89  
FINISHED 11-28-89  
SHEET 2 OF 2



E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

HOLE NUMBER 5C-3E  
SURFACE ELEVATION 570.41  
GROUNDWATER DEPTH \_\_\_\_\_

PROJECT Solvent Chemical  
JD 1030

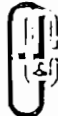
LOCATION 3163 Buffalo Ave  
Niagara Falls NY

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER		PROFILE Cl   Sl   Sd   Gr	FIELD IDENTIFICATION OF SOILS	NOTES
				0	6			
				12	18			
				6	12		15.5'-18.0' - Heavily Fractured zone, horizontal, to sub-horizontal, some along stylolites	
				18	24		15.5'-15.9' Vuggy zone with secondary mineralization (calcite)	CORE RUN 2
							18.0'-19.4' - Competent, massive	16.2 - 21.2'
							19.4'-20.0' Heavily Fractured No pieces > 1"	Recovery 4.3 = 90%
							20.0'-21.0' - Competent, massive	
							21.0'-21.2' - Bedding plane fracture, open, weathered	

640088

CLASSIFICATION/BY R. Watt

DATE  
STARTED 12-1-89  
FINISHED 12-1-89  
SHEET 1 OF 1



E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

HOLE NUMBER SC-41  
SURFACE ELEVATION 568.60  
GROUNDWATER DEPTH \_\_\_\_\_

PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave  
Niagara Falls N.Y.

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE	FIELD IDENTIFICATION OF SOILS	NOTES
				0	6	6	12			
				12	18	18	24			
				Cl	Sl	Sd	Gr			
0-5									0.0'-1.0' Fill, cobble to pebble size angular dolomite roadbed in a matrix of black silt and sand - moist	Sampling via Laskey Sampler
5-10	2 in. s.s. w/ s.s. screen slot size 0.010 in.							BR at 8.0'	3.0'-3.5' - Fill, as above, with increasing gray clay component - saturated	SAMPLE RUN 1: 0'-3.0' RECOVERY: 33%
10-15									3.5'-4.0' clay, gray interbedded with sand, silt and gravel, grading into saturated highly plastic red clay	SAMPLE RUN 2: 3.0'-8.0' RECOVERY: 20%

640088

CLASSIFICATION/BY R. Meyers  
A-12

DATE 11-30-89  
 STARTED 11-30-89  
 FINISHED 12-1-89  
 SHEET 1 OF 1



E + E DRILLING AND TESTING CO., INC.  
 SUBSURFACE LOG

Draft-4E  
 HOLE NUMBER 316-4E  
 SURFACE ELEVATION 568.78  
 GROUNDWATER DEPTH

PROJECT Solvent Chemical  
 JD 1030

LOCATION 3163 Buffalo Ave  
 Niagara Falls N.Y.

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE				FIELD IDENTIFICATION OF SOILS	NOTES
				0	6	12	18	Cl	SI	Sd	Gr		
				6	12	18	24	INFORMATION PERTAINING TO 0-4.0' FROM SC-4A.					
0-5	4" SS											0.0'-1.0' Fill, cobble to pebble size angular dolomite roadbed in a matrix of black silt and sand - moist	Sampling via Lasker Sampler!
5-10												3.0'-3.5' Fill, as above, with increasing gray clay component - saturated	SAMPLE RUN 1: 0'-3.0' RECOVERY = 33%
10-15												3.5'-4.0' clay, gray interbedded with sand, silt and gravel, grading into saturated highly plastic red clay	SAMPLER RUN 2: 3.0'-8.0' RECOVERY = 20%
15-20	2.96 in. NX CORE											8.0'-22.5' Lockport dolomite medium gray to dark gray, dark gray when fresh, micritic, fine grained, minor vugs.	CORE RUN 1: 11.0'-22.5' Recovery 9.0' = 90%
20-25												8.0'-11.0' weathered zone dolomite rubble	
25-30												11.0'-11.6' competent, massive	
30-35												11.6'-16.7' core loss	
35-40												16.7'-17.2' competent, massive	
40-45												17.2'-17.7' many horizontal (bedding plane) fractures	
45-50												17.7'-22.5' competent, massive, minor vugs with secondary mineralization (calcite)	

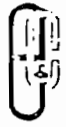
BR at 8.0'

CLASSIFICATION/BY R Meyers

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Draft

DATE STARTED 11-20-89  
FINISHED 11-20-89  
SHEET \_\_\_ OF \_\_\_



E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

HOLE NUMBER SC-57  
SURFACE ELEVATION 570.58  
GROUNDWATER DEPTH

PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave  
Niagara Falls, N.Y.

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE	FIELD IDENTIFICATION OF SOILS	NOTES			
				0	6	12	18						
				6	12	18	24						
								Cl	Sl	Sd	Gr		
0-5		SS	1	7	10				0.0'-0.4' Silty Loam, dark brown to black, minor organics, grass, roots	Sampling via split spoon			
5-6		SS	2	8	7				0.4'-0.7' Silty Clay, medium brown, plastic with yellow dark brown and black nodules glass and angular rock fragments	SAMPLER RUN 1: 0.0'-2.0'			
6-7		SS	3	5	6				2.0'-2.5' silty clay, as above	RECOVERY = 0.71 = 35%			
7-8		SS	4	5	6				2.5'-2.6' - Fill, gravel in black silty matrix;	SAMPLE RUN 2: 2.0'-4.6'			
8-10		SS	5	7	7				4.0-4.3' Clay, mottled brown to black, plastic, saturated	RECOVERY 0.6' = 30%			
10-12.5				9	18				6.0'-7.5' - Clay, yellow Brown to medium brown, finely laminated, yellow brown silt partings, occasional sub rounded pebbles, lacustrine/Fluvial origin.	SAMPLER RUN 3: 4.0'-6.0'			
12.5-12.5							BR at 12.5'		7.5-8.0' Clay, red, massive saturated, plastic	RECOVERY 0.3' = 15%			
12.5-12.5									8.0-12.5' - Interbedded dolomite and clay, no sample	SAMPLER RUN 4: 6.0'-8.0'			
12.5-12.5										RECOVERY 2.0' = 100%			
12.5-12.5										SAMPLE RUN 5: 8.0'-10.0'			
12.5-12.5										RECOVERY 0' = 0%			

640088

CLASSIFICATION/BY J. Doerr

Draft-51

HOLE NUMBER 50-51  
SURFACE ELEVATION 570.6  
GROUNDWATER DEPTH 1

E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

DATE 11-20-89  
STARTED 11-20-89  
FINISHED 11-21-89  
SHEET 1 OF 2

PROJECT Solvent Chemical  
JD 1030

LOCATION 3/63 Buffalo Ave  
Niagara Falls, N.Y.

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE	FIELD IDENTIFICATION OF SOILS	NOTES
				0	6	6	12			
				12	18	18	24			
							CI SI Sd Gr	INFORMATION PERTAINING TO 0-12.5' FROM SC-SA.		
0-5	4" SS	SS	1	7	10			0.0'-0.4' Silty Loam, dark brown to black, minor organics, grass, roots	Sampling via split Spoon	
		SS	2	8	7			0.4'-0.7' Silty Clay, medium brown, plastic with yellow dark brown and black nodules glass and angular rock fragments	SAMPLER RUN 0.0' - 2.0' Recovery = 0.7' = 35%	
		SS	3	5	6			2.0'-2.5' silty Clay, as above	SAMPLER RUN 2.0' - 4.0' Recovery = 0.6' = 30%	
		SS	4	5	6			4.0-4.3' Clay, mottled brown to black, plastic, saturated	SAMPLER RUN 4.0' - 6.0' Recovery = 0.3' = 15%	
		SS	5	6	11			6.0' - 7.5' - Clay, yellow Brown to medium brown, finely laminated, yellow brown silt partings, occasional sub rounded pebbles, lacustrine/Fluvial origin.	SAMPLER RUN 6.0' - 8.0' Recovery = 2.0' = 100%	
				7	7			7.5-8.0' Clay, red, massive saturated, plastic	SAMPLER RUN 8.0' - 10.0' Recovery = 0' = 0%	
				9	18			8.0-12.5' - Interbedded dolomite and clay, no sample		
								12.5-21.0' - Lock port dolomite medium gray to dark gray, dark gray when fresh micritic, Fine grained		
								BR at 12.5'		

2.96 in. NX CORE

10088

CLASSIFICATION/BY J. Doerr

A-15

revised paper

ecology and environment



DATE 11-20-89  
STARTED 11-20-89  
FINISHED 11-21-89  
SHEET 2 OF 2

E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

HOLE NUMBER SC-58  
SURFACE ELEVATION 570.62  
GROUNDWATER DEPTH

PROJECT Solvent Chemical  
JD 1030

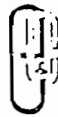
LOCATION 3163 Buffalo Ave  
Niagara Falls NY

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE				FIELD IDENTIFICATION OF SOILS	NOTES	
				0	6	6	12	Cl	SI	Sd	Gr			
				0	6	6	12							
				12	18	18	24							

12.5' - 14.1' - competent, massive, vuggy, increasing in size and number downward, size ranges from ~1mm to ~1cm  
 14.1' - 14.4' Fracture zone,  
 14.4' - 15.2' Heavily weathered zone, 3 bedding fractures  
 15.2' - 19.4' - Heavily Fractured, weathered  
 19.4' - 21.0' - competent massive

CORERUN:  
12.5' - 21.0'

DATE  
 STARTED 11-5-89  
 FINISHED 12-8-89  
 SHEET 1 OF 3



E + E DRILLING AND TESTING CO., INC.  
 SUBSURFACE LOG

HOLE NUMBER SC-5C  
 SURFACE ELEVATION 570.62  
 GROUNDWATER DEPTH \_\_\_\_\_

PROJECT Solvent Chemical  
JD 1030

LOCATION 3/63 Buffalo Ave  
Niagara Falls, N.Y.

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE	FIELD IDENTIFICATION OF SOILS	NOTES
				G		12				
				0	6	12	18			
							CI SI Sd Gr	INFORMATION PERTAINING TO 0-12.5' FROM SC-5A INFORMATION PERTAINING TO 12.5-19' FROM SC-5B		
5	6" Steel	SS	1	7	10			0.0'-0.4' Silty Loam, dark brown to black, minor organics, grass, roots	Sampling via Split Spoon	
		SS	2	5	10				SAMPLER RUN 0.0'-2.0' Recovery = 0.7' = 35%	
		SS	3	5	6			0.4'-0.7' Silty Clay, medium brown, plastic with yellow dark brown and black nodules glass and angular rock fragments	SAMPLER RUN 2.0'-4.0' Recovery 0.6' = 30%	
		SS	4	5	6			2.0'-2.5' Silty Clay, as above		
		SS	5	6	11			2.5'-2.6' Fill, gravel in black silty matrix.	SAMPLER RUN 4.0'-6.0' Recovery 0.3 = 15%	
10	4" Stainless Steel			9	18			4.0-4.3' Clay, mottled brown to black, plastic, saturated	SAMPLER RUN 6.0'-8.0' Recovery 2.0' = 100%	
10.9								6.0'-7.5' Clay, yellow Brown to medium brown, finely laminated, yellow brown silt partings, occasional sub rounded pebbles, lacustrine/fluvial origin.		
15								7.5-8.0' Clay, red, massive saturated, plastic		
20								8.0-12.5' Interbedded dolomite and clay, no sample	SAMPLER RUN 8.0'-10.0' Recovery 0' = 0%	
26.5								12.5-21.0' Lockport dolomite medium gray to dark gray, dark gray when fresh micritic, fine grained		
30										
35										
36.5										
40										

BR at 12.5'

2.96" NX

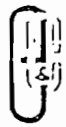
36.5'

CLASSIFICATION BY J Doerr

Draft 51

HOLE NUMBER SC-51  
SURFACE ELEVATION 570.6  
GROUNDWATER DEPTH \_\_\_\_\_

DATE \_\_\_\_\_  
STARTED 11-5-89  
FINISHED 12-8-89  
SHEET 2 OF 3



E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

PROJECT Solvent Chemical  
JD 1030

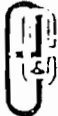
LOCATION 3163 Buffalo Ave  
Niagara Falls NY

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE	FIELD IDENTIFICATION OF SOILS	NOTES
				0	6	12	18			
				6	12	18	24			
							Cl SI Sd Gr			
								12.5' - 14.1' - competent, massive, vuggy, increasing in size and number downward, size ranges from ~1mm to ~1cm		
								14.1' - 14.4' Fracture zone,		
								14.4' - 15.2' Heavily weathered zone, 3 bedding fractures		
								15.2' - 19.0' - Heavily Fractured, weathered		
								19.0' - 19.85' - Highly Fractured dolomite (rubble): mechanical fractures from drilling process. Fragments show large vugs up to 1" in width, partially filled with calcite.	Core run from 19.0' - 26.5' Recovery 7.2' = 96%	
								19.85' - 20.35' - Same dolomite but without vugs. Contains two bedding plane fractures		
								20.35' - 20.5' - Fracture zone.	Lost drill water return	
								20.5' - 23.25' - Competent dolostone with bedding plane fractures, some along stylolites, Contains few small vugs (2-5mm) partially filled with calcite.		
								23.25' - 23.70' - Same dolomite with vertical stress fractures and secondary mineralization with calcite.		
								23.70' - 24.0' - Highly Fractured zone with small vugs and calcite. Believe Fractured by core barrel movement.		
								24.0' - 24.9' - Same dolomite with single vertical fracture showing secondary mineralization with calcite. Some large, up to 1/2", vugs filled with calcite and 2 bedding plane (horiz.) fractures @ stylolites.		
								24.9' - 25.4' - Highly Fractured zone with calcite.		

40088

CLASSIFICATION/BY J Doerr / R. Meyers

DATE  
 STARTED 11-5-89  
 FINISHED 12-8-89  
 SHEET 3 OF 3



E + E DRILLING AND TESTING CO., INC.  
 SUBSURFACE LOG

HOLE NUMBER SC-5C  
 SURFACE ELEVATION 570.6  
 GROUNDWATER DEPTH 17.62 EGS

PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave.  
Niagara Falls, NY

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE	FIELD IDENTIFICATION OF SOILS	NOTES
				0	6	6	12			
				12	18	18	24			
							Cl   Sl   Sd   Gr	25.4'-26.5' Single piece of dolomite with 1 vug (1/2") filled with calcite		
								26.5'-34.5' Competent dolomite with few vugs and stress fractures filled with calcite containing the following fractures:	CORE RUN Feb 26.5'-36.5' Recovery 10' = 100% RQD = 81%	
								26.6' and 27.2' clean mechanical breaks along bedding planes.		
								27.85' horizontal bedding plane fracture with stylolite.		
								28.0' 45° fracture with secondary calcite	Lost drill water return @ 32.75'	
								28.3' horizontal fracture with secondary calcite.		
								28.8' horizontal fracture along bedding plane.		
								29.0' and 29.1' fractures along thick stylolites (horizontal)		
								29.65' horizontal fracture at stylolite		
								30.65' and 31.0' same type fracture		
								31.35' and 31.9' same type of fracture but more open, no mineralization.		
								32.4' to 32.5' large open horizontal fracture with stylolite and secondary mineralization		
								Area of water loss		
								32.7' horizontal fracture with stylolite and secondary mineralization.		
								33.25' to 33.70' area with 4 horizontal fractures with stylolites and secondary mineralization.		
								33.70' to 36.5' Dolostone is more competent with many more small vugs, but no stylolites. Secondary mineralization with calcite throughout. Also has 3 mechanical fractures at 33.95', 35.2', and 36.35'.		

640088

CLASSIFICATION/BY R. Meyers

DATE  
 STARTED 12-12-89  
 FINISHED 12-14-89  
 SHEET 2 OF 4



E + E DRILLING AND TESTING CO., INC.  
 SUBSURFACE LOG

HOLE NUMBER Draft 501  
 SURFACE ELEVATION 571.38  
 GROUNDWATER DEPTH \_\_\_\_\_

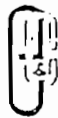
PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave  
Niagara Falls NY

WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE Cl   Sl   Sd   Gr	FIELD IDENTIFICATION OF SOILS	NOTES
			0-6	6-12	12-18	18-24			
2.96' CNX							12.5' - 14.1' - competent, massive, vuggy, increasing in size and number downward, size ranges from ~1mm to ~1cm		
51.5'							14.1' - 14.4' Fracture zone, 14.4' - 15.2' Heavily weathered zone, 3 bedding fractures 15.2' - 19.0' Heavily Fractured, weathered 19.0' - 19.85' - Highly Fractured dolomite (rubble): mechanical fractures from drilling process. Fragments show large vugs up to 1" in width, partially filled with calcite. 19.85' - 20.35' - Same dolomite but without vugs. Contains two bedding plane fractures 20.35' - 20.5' - Fracture zone. 20.5' - 23.25' - Competent dolostone with bedding plane fractures, some along stylolites, contains few small vugs (2-5mm) partially filled with calcite. 23.25' - 23.70' - Same dolomite with vertical stress fractures and secondary mineralization with calcite. 23.70' - 24.0' - Highly fractured zone with small vugs and calcite. Believe fractured by core barrel movement. 24.0' - 24.9' - Same dolomite with single vertical fracture showing secondary mineralization with calcite. Some large, up to 1/2", vugs filled with calcite and 2 bedding plane (horiz.) fractures @ stylolites. 24.9' - 25.4' - Highly fractured zone with calcite.	Core run from 19.0' - 26.5' Recovery 7.2' = 96%	

CLASSIFICATION BY J. Doerr / R. Meyers

DATE  
 STARTED 12-12-89  
 FINISHED 12-14-89  
 SHEET 3 OF 4



E + E DRILLING AND TESTING CO., INC.  
 SUBSURFACE LOG

**Draft**  
 HOLE NUMBER SC 3CD  
 SURFACE ELEVATION 571.38  
 GROUNDWATER DEPTH \_\_\_\_\_

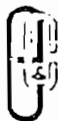
PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave.  
Niagara Falls, NY

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER		PROFILE Cl   Sl   Sd   Gr	FIELD IDENTIFICATION OF SOILS	NOTES
				0	6			
				12	18			
				6	12		25.4'-26.5' Single piece of dolomite with 1 vug (1/2") Filled with calcite	
				18	24		26.5'-34.5' Competent dolomite with few vugs and stress fractures filled with calcite containing the following fractures: 26.6' and 27.2' clean mechanical breaks along bedding planes. 27.85' horizontal bedding plane fracture with stylolite. 28.0' 45° fracture with secondary calcite 28.3' horizontal fracture with secondary calcite. 28.8' horizontal fracture along bedding plane. 29.0' and 29.1' fractures along thick stylolites (horizontal) 29.65' horizontal fracture at stylolite 30.65' and 31.0' same type fracture 31.35' and 31.7' same type of fracture but more open, i.e., no mineralization. 32.4' to 32.5' Large open horizontal fracture with stylolite and secondary mineralization Area of water loss 32.7' horizontal fracture with stylolite and secondary mineralization. 33.25' to 33.70' Area with 4 horizontal fractures with stylolites and secondary mineralization. 33.70' to 36.5' Dolostone is more competent with many more small vugs, but no stylolites. Secondary mineralization with calcite throughout. Also has 3 mechanical fractures at 33.95', 35.2', and 36.35'.	CORE RUN FROM 26.5'-36.5' Recovery 10' = 100% RQD = 81%  Lost drill water return @ 32.75'

CLASSIFICATION/BY R. Meyers

DATE  
STARTED 12-12-89  
FINISHED 12-14-89  
SHEET 4 OF 4



E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

HOLE NUMBER SC-50  
SURFACE ELEVATION 571.38  
GROUNDWATER DEPTH \_\_\_\_\_

PROJECT Solvent Chemical  
JD 1030

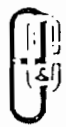
LOCATION 3163 Buffalo Ave.  
Niagara Falls, NY

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE	FIELD IDENTIFICATION OF SOILS	NOTES			
				0	6	6	12						
				12	18	18	24						
								Cl	Sl	Sd	Gr		
										Dolomite with numerous vugs from 34.5' to 35.5' which are partially filled with calcite.			Core Run from 34.5' to 39.5'
										35.5' to 39.5' Dolomite is tighter with fewer, larger vugs completely filled with calcite.			Recovery 5.0' = 100% RQD = 54%
										34.5' to 39.5 The following fractures occur:			
										34.6', 34.75', 34.95', 35.15', 35.95', 36.25' and 36.55' which are bedding plane (horizontal) fractures.			
										36.7' to 36.8' Large open fracture with massive secondary calcite.			
										37.03', 37.90', 38.20' Tight, clean horizontal bedding plane fractures			
										38.73' Horizontal fracture along stylolite			
										38.73' to 39.5' Three clean, horizontal bedding plane fractures.			
										39.5' Horizontal fracture along stylolite.			
										Competent dolomite which is highly fractured. The vast majority of the fractures are tight, clean, mechanical breaks along bedding planes or stylolites with the following exceptions:			Core run from 39.5' to 51.5' Recovery 12' = 100% RQD = 68%
										40.1' Small open fracture with stylolite			
										40.8' to 41.0' Fracture zone with secondary calcite			Area of water loss.
										41.6' to 42.5' Highly fractured zone with secondary calcite. Contains four obviously water bearing fractures, all horizontal.			
										42.9' Horizontal fracture along vuggy zone.			

640088

CLASSIFICATION/BY R. Meyers

DATE STARTED 12-12-89  
 FINISHED 12-14-89  
 SHEET 1 OF 4



E + E DRILLING AND TESTING CO., INC.  
 SUBSURFACE LOG

HOLE NUMBER SC-5C1  
 SURFACE ELEVATION 571.38  
 GROUNDWATER DEPTH \_\_\_\_\_

PROJECT Solvent Chemical  
JD 1030

LOCATION 3123 Buffalo Ave.  
Niagara Falls, NY

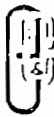
DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER		PROFILE				FIELD IDENTIFICATION OF SOILS	NOTES	
				0-6	6-12	CI	SI	Sd	Gr			
0-5	6" Black Steel	SS	1	7	10					0.0'-0.4' Silty Loam, dark brown to black, minor organics, grass, roots	Sampling via Split Spoon	
5-6		SS	2	8	7							
6-7		SS	3	5	6							0.4'-0.7' Silty Clay, medium brown, plastic with yellow dark brown and black nodules glass and angular rock fragments
7-8		SS	4	5	6							
8-9		SS	5	7	7							
9-10	4" stainless steel			9	18					2.0'-2.5' silty clay, as above	SAMPLE RUN 2.0'-4.0' Recovery 0.6' = 30%	
10-11										2.5'-2.6' - Fill, gravel in black silty matrix.	SAMPLE RUN 4.0'-6.0' Recovery 0.3 = 15%	
11-12										4.0-4.3' Clay, mottled brown to black, plastic, saturated	SAMPLE RUN 6.0'-8.0' Recovery 2.0' = 100%	
12-13										6.0'-7.5' - Clay, yellow Brown to medium brown, finely laminated, yellow brown silt partings, occasional sub rounded pebbles, lacustrine/Fluvial origin.	SAMPLE RUN 8.0'-10.0' Recovery 0' = 0%	
13-14										7.5-8.0' Clay, red, massive saturated, plastic		
14-15										8.0-12.5' - Interbedded dolomite and clay, no sample.		
15-16										12.5-21.0 - Lock port dolomite medium gray to dark gray, dark gray when fresh, micritic, Fine grained		
16-17												
17-18												
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640088

CLASSIFICATION/BY J. Doerr



DATE  
 STARTED 1-2-90  
 FINISHED 1-3-90  
 SHEET 1 OF 6



E + E DRILLING AND TESTING CO., INC.  
 SUBSURFACE LOG

**Draft**  
 HOLE NUMBER SC-5F  
 SURFACE ELEVATION 570.64  
 GROUNDWATER DEPTH 1

PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave.  
Niagara Falls, NY

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE				FIELD IDENTIFICATION OF SOILS <small>INFORMATION PERTAINING TO 0'-12.5' FROM SC-5A          INFORMATION PERTAINING TO 12.5'-19' FROM SC-5B          INFORMATION PERTAINING TO 19'-30.5' FROM SC-5C          INFORMATION PERTAINING TO 30.5'-49.5' FROM SC-5D.</small>	NOTES
				0		6		Cl	SI	Sd	Gr		
				6	12	12	18						
0.0	6" Black Steel	SS	1	7	10					0.0'-0.4' Silty Loam, dark brown to black, minor organics, grass, roots	Sampling via split spoon		
0.5				5	10								
1.0		SS	2	8	7							0.4'-0.7' silty clay, medium brown, plastic with yellow dark brown and black nodules glass and angular rock fragments	
1.5				6	9								
2.0		SS	3	5	6								
2.5				7	6					2.0'-2.5' silty clay, as above			
3.0	SS	4	5	6									
3.5				6	11					2.5'-2.6' - Fill, gravel in black silty matrix.			
4.0	SS	5	7	7									
4.5				9	18					4.0-4.3' Clay, mottled brown to black, plastic, saturated			
5.0													
6.0	4" Stainless Steel									6.0'-7.5' - Clay, yellow Brown to medium brown, finely laminated, yellow brown silt partings, occasional sub rounded pebbles, lacustrine/fluvial origin.	SAMPLER RUN 2 2.0' - 4.0' Recovery 0.6' = 30%		
6.5													
7.0													
7.5													
8.0													
8.5													
9.0													
9.5													
10.0													
10.5													
11.0	2" STAINLESS STEEL									7.5-8.0' Clay, red, massive saturated, plastic	SAMPLER RUN 3 4.0' - 6.0' Recovery 0.3 = 15%		
11.5													
12.0													
12.5													
13.0													
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BR at 12.5'

CLASSIFICATION/BY J. Doerr

DATE

STARTED 1-2-90

FINISHED 1-3-90

SHEET 2 OF 6



E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

HOLE NUMBER SC-5F  
SURFACE ELEVATION 570.69  
GROUNDWATER DEPTH

Draft

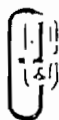
PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave  
Niagara Falls NY

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE Cl Si Sd Gr	FIELD IDENTIFICATION OF SOILS	NOTES
				0-6	6-12	12-18	18-24			
				10	10	10	10			
0								12.5' - 14.1' - competent, massive, vuggy, increasing in size and number downward, size ranges from ~1mm to ~1cm		
5								14.1' - 14.4' Fracture zone, 14.4' - 15.2' Heavily weathered zone, 3 bedding fractures		
10								15.2' - 19.0' - Heavily Fractured, weathered		
15								19.0' - 19.85' - Highly Fractured dolomite (rubble): mechanical fractures from drilling process. Fragments show large vugs up to 1" in width, partially filled with calcite.	Core run from 19.0' - 26.5' Recovery 7.2' = 96%	
20								19.85' - 20.35' - Same dolomite but without vugs. Contains two bedding plane fractures		
25								20.35' - 20.5' - Fracture zone.	Lost drill water return.	
30								20.5' - 23.25' - Competent dolostone with bedding plane fractures, some along stylolites, Contains few small vugs (2-5mm) partially filled with calcite.		
35								23.25' - 23.70' - Same dolomite with vertical stress fractures and secondary mineralization with calcite.		
40								23.70' - 24.0' - Highly Fractured zone with small vugs and calcite. Believe Fractured by core barrel movement.		
45								24.0' - 24.9' - Same dolomite with single vertical fracture showing secondary mineralization with calcite. Some large, up to 1/2", vugs filled with calcite and 2 bedding plane (horiz.) fractures @ stylolites.		
50								24.9' - 25.4' - Highly Fractured zone with calcite.		

CLASSIFICATION BY J. Doerr / R. Meyers

DATE  
 STARTED 1-2-90  
 FINISHED 1-3-90  
 SHEET 3 OF 6



E + E DRILLING AND TESTING CO., INC.  
 SUBSURFACE LOG

HOLE NUMBER SG-5F  
 SURFACE ELEVATION 570.69  
 GROUNDWATER DEPTH \_\_\_\_\_

PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave.  
Niagara Falls, NY

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE Cl   Sl   Sd   Gr	FIELD IDENTIFICATION OF SOILS	NOTES
				0	6	6	12			
				12	18	18	24			
95	2.96 in. NX									
98.0'								25.4' - 26.5' Single piece of dolomite with 1 vug (1/2") filled with calcite	CORE RUN FOR 26.5' - 36.5' Recovery 10' = 100% RQD = 81%  Lost drill water return @ 32.75'	
								26.5' - 34.5' Competent dolomite with few vugs and stress fractures filled with calcite containing the following fractures:		
								26.6' and 27.2' clean mechanical breaks along bedding planes.		
								27.85' Horizontal bedding plane fracture with stylolite.		
								28.0' 45° fracture with secondary calcite		
								28.3' Horizontal fracture with secondary calcite.		
								28.8' Horizontal fracture along bedding plane.		
								29.0' and 29.1' fractures along thick stylolites (horizontal)		
								29.65' Horizontal fracture at stylolite		
								30.65' and 31.0' same type fracture		
								31.35' and 31.7' same type of fracture but more open, no mineralization.		
								32.4' to 32.5' Large open horizontal fracture with stylolite and secondary mineralization		
								Area of water loss		
								32.7' Horizontal fracture with stylolite and secondary mineralization.		
								33.25' to 33.70' Area with 4 horizontal fractures with stylolites and secondary mineralization.		
								33.70' to 36.5' Dolomite is more competent with many more small vugs, but no stylolites. Secondary mineralization with calcite throughout. Also has 3 mechanical fractures at 33.95', 35.2', and 36.35'.		

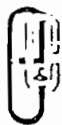
CLASSIFICATION/BY R. Meyers

DATE

STARTED 1-2-90

FINISHED 1-3-90

SHEET 4 OF 6



E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

Draft

HOLE NUMBER SC-5F

SURFACE ELEVATION 570.64

GROUNDWATER DEPTH

PROJECT Solvent Chemical  
JD 030

LOCATION 3103 Buffalo Ave.  
Niagara Falls, NY

WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE	FIELD IDENTIFICATION OF SOILS	NOTES
			0	6	6	12			
			12	18	18	24			
						Cl Si Sd Gr	<p>Dolomite with numerous vugs from 34.5' to 35.5' which are partially filled with calcite.</p> <p>35.5' to 39.5' Dolomite is tighter with fewer, larger vugs completely filled with calcite.</p> <p>34.5' to 39.5' The following fractures occur:</p> <p>34.6', 34.75', 34.95', 35.15', 35.95', 36.25' and 36.55' which are bedding plane (horizontal) fractures.</p> <p>36.7' to 36.8' Large open fracture with massive secondary calcite.</p> <p>37.03', 37.90', 38.20' Tight, clean, horizontal bedding plane fractures.</p> <p>38.73' Horizontal fracture along stylolite.</p> <p>38.73' to 39.5' Three clean, horizontal bedding plane fractures.</p> <p>39.5' Horizontal fracture along stylolite.</p> <p>Competent dolomite which is highly fractured. The vast majority of the fractures are tight, clean, mechanical breaks along bedding planes or stylolites with the following exceptions:</p> <p>40.1' Small open fracture with stylolite.</p> <p>40.8' to 41.0' Fracture zone with secondary calcite.</p> <p>41.6' to 42.5' Highly fractured zone with secondary calcite. Contains four obviously water bearing fractures, all horizontal.</p> <p>42.9' Horizontal fracture along vuggy zone.</p>	<p>Core Run from 34.5' to 39.5'</p> <p>Recovery 5.0' = 100%</p> <p>RQD = 54%</p> <p>Core run from 39.5' to 51.5'</p> <p>Recovery 12' = 100%</p> <p>RQD = 68%</p> <p>Area of water loss.</p>	

CLASSIFICATION/BY

R. Meyers

A-27

DATE  
 STARTED 1-2-90  
 FINISHED 1-3-90  
 SHEET 5 OF 6



E + E DRILLING AND TESTING CO., INC.  
 SUBSURFACE LOG

HOLE NUMBER SC-5F  
 SURFACE ELEVATION 570.64  
 GROUNDWATER DEPTH \_\_\_\_\_


PROJECT Solvent Chemical  
JD1030

LOCATION 3103 Buffalo Ave.  
Niagara Falls, NY

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE Cl   Sl   Sd   Gr	FIELD IDENTIFICATION OF SOILS	NOTES
				0	6	6	12			
				12	18	18	24			
								49.5' to 58.1' Highly Fractured dolostone with core segments ranging from 0.08' to 0.4'. The Fractures are horizontal bedding plane fractures with some secondary calcite. They are fairly tight with very few stylolites. The core has some small vugs (partially calcite filled) randomly spread out over the length of the core. In addition to the horizontal fractures, a single vertical fracture is present from 55.85' to 56.25'.	Core Run From 49.5' to 58.1' Recovery 8.6' = 100% RQD = 17%	
								58.1' to 64.3' The dolostone appears more weathered with more solution features such as vugs and stress fractures filled with calcite and gypsum. Most fractures in this zone are tight horizontal with some secondary mineralization. Large open fractures are present at 61.0', 61.3', 61.9', 62.3', and 63.7'.	CORE RUN From 58.1' to 68.1' Recovery 10' = 100% RQD = 51%	
								64.1' to 64.3' Area of calcite/gypsum		
								64.3' to 68.1' Dolostone is less weathered with very few vugs or solution features. The fractures are horizontal and tighter than above and most are at stylolites. One open fracture is at 65.85'		

640088

CLASSIFICATION/BY R Meyers

DATE STARTED <u>1-2-90</u> FINISHED <u>1-3-90</u> SHEET <u>6</u> OF <u>6</u>	 <b>E + E DRILLING AND TESTING CO., INC.</b> <b>SUBSURFACE LOG</b>	HOLE NUMBER <u>SC-SF</u> SURFACE ELEVATION <u>570.64</u> GROUNDWATER DEPTH _____
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PROJECT <u>Solvent Chemical</u> <u>JD 1030</u>	LOCATION <u>3103 Buffalo Ave.</u> <u>Niagara Falls, NY</u>
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DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE				FIELD IDENTIFICATION OF SOILS	NOTES		
				0	6	6	12	Cl	Sl	Sd	Gr				
				12	18	18	24								
													68.1' to 78.1' Lockport dolostone with comparatively few small vugs. Entire core is highly fractured with horizontal (bedding) fractures along stylolites and areas with secondary calcite/gypsum. Also a single vertical stress fracture from 73.8' to 74.1'.	CORE RUN from 68.1' to 78.1' Recovery 10' = 100% RQD = 15% Slight color.	
													78.1' to 87.6' Same dolostone with few large (up to 2") open vugs and calcite/gypsum filled vugs. Core is highly fractured horizontally as above, with more open fractures at 78.6', 80.1', 82.25', 82.4', 82.7', 83.15', 84.9', and 86.6'.	CORE RUN from 78.1' to 87.6' Recovery 9.6' = 100% RQD = 25%	
													87.6' to 98.0' Same dolostone with few large vugs filled with loose calcite crystals or massive calcite/gypsum. Core loss from 87.65' to 87.85'. The core is highly fractured from 87.6' to 94.5' with few tight bedding plane fractures from 94.5' to 98.0'. The fractures from 87.6' to 94.5' include numerous large open fractures with secondary mineralization (calcite/gypsum) and thick stylolites.	CORE RUN from 87.6' to 98.0' Recovery 10.2' = 98% RQD = 39%	

DATE 11-28-89  
STARTED 11-28-89  
FINISHED 11-28-89  
SHEET 1 OF 1

E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

HOLE NUMBER SC 6A  
SURFACE ELEVATION 570.83  
GROUNDWATER DEPTH

PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave  
Niagara Falls N.Y.

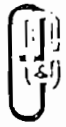
DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER		PROFILE	FIELD IDENTIFICATION OF SOILS	NOTES
				0-6	6-12			
				12-18	18-24	Cl SI Sd Gr		
5	<p>2 in. S.S. w/ SS. Screen slot size 0.010 in.</p>						<p>0.0'-0.7' Fill, gravel, medium to dark gray, 0.7'-1.7' Fill, medium sand to gravel, dark gray to black, moderate brick fragments and industrial debris</p>	<p>Sampled via Laskey Sampler SAMPLE RUN 0.0' - 5.0' RECOVERY = 2.5' = 50%</p>
10						BR at 9.6'	<p>1.7'-2.5' Fill, silt to gravel, black, some industrial debris, moist</p>	<p>SAMPLE RUN 2 5.0' - 10.0' Recovery 1:1 = 24%</p>
15							<p>5.0'-6.1 Sandy silt, black, fine grained, some light gray to red clay, minor brick fragments</p>	
20								
25								
30								
35								
40								
45								
50								

640088

CLASSIFICATION/BY R. Watt  
A-30

Draft 61

DATE 11-29-89  
STARTED 11-29-89  
FINISHED 11-30-89  
SHEET 1 OF 1



E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

HOLE NUMBER 61  
SURFACE ELEVATION 570.8'  
GROUNDWATER DEPTH

PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave  
Niagara Falls N.Y.

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER				PROFILE	FIELD IDENTIFICATION OF SOILS	NOTES
				0-6	6-12	12-18	18-24			
				CI	SI	Sd	Gr			
									INFORMATION PERTAINING TO 0-6.1' FROM SC-6A.	
0-5								0.0'-0.7' Fill, gravel, medium to dark gray,	Sampled via Haskey Sampler	
5-10								0.7'-1.7' Fill, medium sand to gravel, dark gray to black, moderate brick fragments and industrial debris		SAMPLE RUN 1: 0.0' - 5.1' RECOVERY = 2.5' = 50%
10-15									1.7'-2.5' Fill, silt to gravel, black, some industrial debris, moist	SAMPLER RUN 2: 5.0' - 10.0' RECOVERY 1:1 = 24%
15-20									5.0'-6.1' Sandy silt, black, fine grained, some light gray to red clay, minor brick fragments	CORE RUN 1: 10.0' - 20.0' Recover 8.7' = 87%
20-25									9.6'-20.0' Lockport dolomite medium gray to dark gray, dark gray when fresh, micritic, fine grained minor vugs	
25-30									9.6'-12.0' - weathered zone	
30-35									12.0'-17.0' - Highly Fractured some high angle (65°-75°) with 2 <sup>nd</sup> degree calcitic mineralization	
35-40									some subhorizontal with thin mineralized stylolite 2 <sup>nd</sup> degree mineralization	
40-45									17.0'-19.5' - competent, massive	
45-50									19.5'-20.0' - Highly Fractured vuggy	

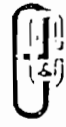
40088

CLASSIFICATION/BY



Draft

DATE  
STARTED 12-4-89  
FINISHED 12-4-89  
SHEET 1 OF 1



E + E DRILLING AND TESTING CO., INC.  
SUBSURFACE LOG

HOLE NUMBER SC 7A  
SURFACE ELEVATION 569.15  
GROUNDWATER DEPTH \_\_\_\_\_

PROJECT Solvent Chemical  
JD 1030

LOCATION 3163 Buffalo Ave  
Niagara Falls N.Y.

DEPTH - FT	WELL DIAGRAM	SAMPLE TYPE	SAMPLE NO.	BLOWS ON SAMPLER		PROFILE				FIELD IDENTIFICATION OF SOILS	NOTES		
				0	6	6	12	CI	SI			Sd	Gr
				12	18	18	24						
5		SS	1	25	35					0.0'-0.5' Fill, angular, pebble cobble dolomite roadbed with dark brown silty loam matrix	Sampled with split spoon. SAMPLER RUN 1 0.0' - 2.0' RECOVERY = 1.0' = 50%		
		SS	2	4	6								
		SS	3	9	1								
		SS	4	25	35								
10	2 in. s.s. w/ s.s. screen slot size 0.010 in			42	50					0.5'-1.0' - Fill, as above, with concrete and brick fragments some industrial debris	SAMPLER RUN 2 2.0' - 4.0' Recovery 1.2' = 60%		
										2.0'-3.2' - Fill, angular dolomite roadbed, black silty sand matrix.	SAMPLER RUN 3 4.0' - 6.0' Recovery 0.8' = 40%		
										4.0'-4.8' - Fill, as above, with some gray clay.	SAMPLER RUN 4 6.0' - 8.0' Recovery 1.5' = 75%		
										6.0'-7.5' - Fill, gravel, with sand silt matrix, black			

B.R. at 7.5'

640088

CLASSIFICATION BY R. Meyers

A-32

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APPENDIX B  
ANALYTICAL DATA

TABLE B-1

DATA SUMMARY FORM: V O L A T I L E S

Site Name: 3163 BUFFALO AVENUE

WATER SAMPLES

(ug/L) (PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor)

CRQL	COMPOUND	Sample No. Dilution Factor	MW-1A	MW-1B	MW-BL1	MW-TB1	MW-2B	MW-2A	MW-3A	MW-6A
10	Chloromethane									
10	Bromomethane									
10	*Vinyl Chloride									
10	Chloroethane									
5	*Methylene Chloride									
10	Acetone									
5	Carbon Disulfide									
5	*1,1-Dichloroethene									
5	1,1-Dichloroethane									
5	*Total-1,2-Dichloroethene									
5	Chloroform									
5	*1,2-Dichloroethane									
10	*2-Butanone									
5	*1,1,1-Trichloroethane									
5	*Carbon Tetrachloride									
10	Vinyl Acetate									
5	Bromodichloromethane									

SEE NARRATIVE FOR CODE DEFINITIONS

\*Action Level Exists

CRDL = Contract Required Detection Limit

E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED

J = BELOW QUANTIFIABLE RANGE; ESTIMATED

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DATA SUMMARY FORM: VOLATILES 1

Site Name: 3163 BUFFALO AVENUE

WATER SAMPLES  
(ug/L) (PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation first:  
(CRQL \* Dilution Factor)

CRQL	COMPOUND	MW-6B	MW-3B	MW-5B	MW-5BD	MW-4A	MW-4B	MW-4BD	MW-PL2
10	Chloromethane								
10	Bromomethane								
10	*Vinyl Chloride	2 J*	23 J*			800			
10	Chloroethane								
5	*Methylene Chloride	1 J*							
10	Acetone								
5	Carbon Disulfide								
5	*1,1-Dichloroethene								
5	1,1-Dichloroethane	4 J*							
5	*Total-1,2-Dichloroethene	2 J*	160			2100			
5	Chloroform								
5	*1,2-Dichloroethane								
10	*2-Butanone								
5	*1,1,1-Trichloroethane								
5	*Carbon Tetrachloride								
10	Vinyl Acetate								
5	Bromodichloromethane								

CRDL = Contract Required Detection Limit      \*Action Level Exists      SEE NARRATIVE FOR CODE DEFINITIONS

\* E = EXCEEDS QUANTIFIABLE RANGE ; ESTIMATED  
J = BELOW QUANTIFIABLE RANGE ; ESTIMATED

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DATA SUMMARY FORM: VOLATILES

Site Name: 3163 BUFFALO AVENUE  
 WATER SAMPLES  
 (ug/L)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
 (CRQL \* Dilution Factor)

CRQL	COMPOUND	MW-7A	MW-5A	MW-TB3	MW-TB4	MW-5C	MW-5CD	MW-TB5	MW-5F
10	Chloromethane	65398	65399	65400	65401	65738	65739	65740	66069
10	Bromomethane								
10	*Vinyl Chloride					74 J*			980
10	Chloroethane								
5	*Methylene Chloride								
10	Acetone								
5	Carbon Disulfide								
5	*1,1-Dichloroethene								
5	1,1-Dichloroethane								
5	*Total-1,2-Dichloroethene		110			820	630		2-100
5	Chloroform								
5	*1,2-Dichloroethane								
10	*2-Butanone								
5	*1,1,1-Trichloroethane								
5	*Carbon Tetrachloride								
10	Vinyl Acetate								
5	Bromodichloromethane								

CRDL = Contract Required Detection Limit      \*Action Level Exists      SEE NARRATIVE FOR CODE DEFINITIONS

\* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
 J = BELOW QUANTIFIABLE RANGE; ESTIMATED

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DATA SUMMARY FORM: VOLATILES 2

Site Name: 3163 BUFFALO AVENUE

WATER SAMPLES (ug/L) (PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limits (CRQL \* Dilution Factor)

CRQL	COMPOUND	Sample No. Dilution Factor Location	MW-1A	MW-1B	MW-BL1	MW-TB1	MW-2B	MW-2A	MW-3A	MW-6A
5	*1,2-Dichloropropane		64993	64994	64995	64996	65191	65192	65193	65194
5	Cis-1,3-Dichloropropene									
5	Trichloroethene							60		3 J
5	Dibromochloromethane									
5	1,1,2-Trichloroethane							100,000		4 J
5	*Benzene		540,000 E*	310,000 E*						
5	Trans-1,3-Dichloropropene									
5	Bromoform									
10	4-Methyl-2-pentanone							380		
10	2-Hexanone									
5	*Tetrachloroethene					87		570		
5	1,1,2,2-Tetrachloroethane									
5	*Toluene					58		960		
5	*Chlorobenzene					43,000		180,000	800	110
5	*Ethylbenzene									
5	*Styrene									
5	*Total Xylenes									

CRDL = Contract Required Detection Limit \*Action Level Exists SEE NARRATIVE FOR CODE DEFINITIONS

E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
 J = BELOW QUANTIFIABLE RANGE; ESTIMATED  
 revised 12/88

**TABLA B-1**

DATA SUMMARY FORM: VOLATILES 2

Site Name: 3163 BUFFALO AVENUE

WATER SAMPLES  
(ug/L)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limits  
(CRQL \* Dilution Factor)

CRQL	COMPOUND	Sample No. Dilution Factor Location	MW-6B	MW-3B	MW-5B	MW-5BD	MW-4A	MW-4B	MW-4BD	MW-BL2
5	*1,2-Dichloropropane									
5	Cis-1,3-Dichloropropene									
5	Trichloroethene			27 J*						
5	Dibromochloromethane									
5	1,1,2-Trichloroethane									
5	*Benzene	300			350	320	140			
5	Trans-1,3-Dichloropropene									
5	Bromoform									
10	4-Methyl-2-pentanone									
10	2-Hexanone									
5	*Tetrachloroethene			45 J*						
5	1,1,2,2-Tetrachloroethane									
5	*Toluene			95						
5	*Chlorobenzene	1800		13,000	2100	1900	910			92
5	*Ethylbenzene			29 J*						
5	*Styrene									
5	*Total Xylenes			150						

CRDL = Contract Required Detection Limit

\*Action Level Exists

SEE NARRATIVE FOR CODE DEFINITIONS

\* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
J = BELOW QUANTIFIABLE RANGE; ESTIMATED

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Site Name: 3163 BUFFALO AVENUE WATER SAMPLES (ug/L)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_ To calculate sample quantitation limits (CRDL \* Dilution Factor)

CRDL	COMPOUND	Sample No. Dilution Factor Location	MW-7A	MW-5A	MW-TB3	MW-TB4	MW-5C	MW-5CD	MW-TB5	MW-5F
5	*1,2-Dichloropropane									
5	Cis-1,3-Dichloropropene									
5	Trichloroethene			150						
5	Dibromochloromethane									
5	1,1,2-Trichloroethane									
5	*Benzene		18,000 E*				920	1200		
5	Trans-1,3-Dichloropropene									
5	Bromoform									
10	4-Methyl-2-pentanone									
10	2-Hexanone									
5	*Tetrachloroethene			820 E*			320			
5	1,1,2,2-Tetrachloroethane									
5	*Toluene			1 J*				98 J*		
5	*Chlorobenzene		39,000 E*				4300	7200	1 J*	58 J*
5	*Ethylbenzene									
5	*Styrene									
5	*Total Xylenes									

CRDL = Contract Required Detection Limit \*Action Level Exists SEE NARRATIVE FOR CODE DEFINITIONS

\* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
 J = BELOW QUANTIFIABLE RANGE; ESTIMATED

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DATA SUMMARY FORM: B N A S 1

Site Name: 3163 BUFFALO AVENUE

WATER SAMPLES  
(ug/L)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CROL \* Dilution Factor)

CROL	Sample No. Dilution Factor Location	MW-1A	MW-1B	MW-BLI	MW-TBI	MW-2B	MW-2A	MW-3A	MW-6A
10	Phenol	64993	64994	64995	64996	65191	65192	65193	65194
10	bis(2-Chloroethyl)ether	1000	330				550		
10	2-Chlorophenol	300	270						
10	*1,3-Dichlorobenzene	2200	3900			20,000	4800	22 J*	
10	*1,4-Dichlorobenzene	7400	12,000 E*			75,000	20,000 E*	39	7 J*
10	Benzyl Alcohol								
10	1,2-Dichlorobenzene	5800 E*	10,000 E*			120,000 E*	19,000 E*	430 E*	2 J*
10	2-Methylphenol								
10	bis(2-Chloroisopropyl)ether								
10	4-Methylphenol								
10	N-Nitroso-di-n-propylamine								
10	Hexachloroethane								
10	Nitrobenzene								
10	Isophorone								
10	2-Nitrophenol								
10	2,4-Dimethylphenol						65 J*		
50	Benzoic Acid								
10	bis(2-Chloroethoxy)methane								
30	2,4-Dichlorophenol	12 J*	20 J*						
10	1,2,4-Trichlorobenzene	1800	4900 E*			47,000	6300	18 J*	
10	Naphthalene	24 J*	25 J*				16 J		
10	4-Chloroaniline								

CRDL = Contract Required Detection Limit \*Action Level Exists SEE NARRATIVE FOR CODE DEFINITIONS

\* E - EXCEEDS QUANTIFIABLE RANGE ; ESTIMATED

J - BELOW QUANTIFIABLE RANGE ; ESTIMATED

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Site Name: 3163 BUFFALO AVENUE WATER SAMPLES (ug/L) (PPM)

Case #: Sampling Date(s):

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor)

CRQL	Sample No. Dilution Factor Location	MW-6B	MW-3B	MW-5B	MW-5BD	MW-4A	MW-4B	MW-4BD	MW-BL2
COMPOUND									
10	Phenol			3 J*			8 J*		
10	bis(2-Chloroethyl)ether								
10	2-Chlorophenol			6 J*	9 J*		14 J*		
10	*1,3-Dichlorobenzene	65	1800	220	280	550	680	830 E*	
10	*1,4-Dichlorobenzene	94	4900	450 E*	580	2700	2400 E*	2600 E*	9 J*
10	Benzyl Alcohol								
10	1,2-Dichlorobenzene	83	12,000 E*	950 E*	1300 E*	16,000 E*	3100 E*	3400 E*	31
10	2-Methylphenol								
10	bis(2-Chloroisopropyl)ether								
10	4-Methylphenol								
10	N-Nitroso-di-n-propylamine								
10	Hexachloroethane								
10	Nitrobenzene								
10	Isophorone								
10	2-Nitrophenol								
10	2,4-Dimethylphenol								
50	Benzoic Acid								
10	bis(2-Chloroethoxy)methane								
10	2,4-Dichlorophenol								
10	1,2,4-Trichlorobenzene	120	4200	350	420	24000	620	680 E*	52
10	Naphthalene								
10	4-Chloroaniline								

CRDL = Contract Required Detection Limit \*Action Level Exists SEE NARRATIVE FOR CODE DEFINITIONS

\* E = EXCEEDS QUANTIFIABLE RANGE ; ESTIMATED

J = BELOW QUANTIFIABLE RANGE ; ESTIMATED

Site Name: 3163 BUFFALO AVENUE

WATER SAMPLES  
(ug/L)(PB)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor)

CRQL	COMPOUND	MW-7A	MW-5A	MW-TB3	MW-TB4	MW-5C	MW-5CD	MW-TBS	MW-5F
	Sample No. Dilution Factor Location								
10	Phenol	150 J*		NA ↓	NA ↓				
10	bis(2-Chloroethyl)ether								
10	2-Chlorophenol	52 J*							
10	*1,3-Dichlorobenzene	2300	2 J*			750	810		4 J*
10	*1,4-Dichlorobenzene	21,000 E*	25			1900 E*	2100 E*		14
10	Benzyl Alcohol								
10	1,2-Dichlorobenzene	25,000	30			3900 E*	4700 E*		39
10	2-Methylphenol								
10	bis(2-Chloroisopropyl)ether								
10	4-Methylphenol								
10	N-Nitroso-di-n-propylamine								
10	Hexachloroethane								
10	Nitrobenzene								
10	Isophorone								
10	2-Nitrophenol								
10	2,4-Dimethylphenol								
50	Benzoic Acid								
30	bis(2-Chloroethoxy)methane								
30	2,4-Dichlorophenol	62 J*							
30	1,2,4-Trichlorobenzene	10,000 E*	11			2800 E*	1500		6 J*
30	Naphthalene	38 J*							
30	4-Chloroaniline								

CRDL = Contract Required Detection Limit \*Action Level Exists SEE NARRATIVE FOR CODE DEFINITIONS

\* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
J = BELOW QUANTIFIABLE RANGE; ESTIMATED

Site Name: 3163 BUFFALO AVENUE

WATER SAMPLES  
(ug/L)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limits  
(CRQL \* Dilution Factor)

CRQL	COMPOUND	MW-1A	MW-1B	MW-BL1	MW-TB1	MW-2B	MW-2A	MW-3A	MW-6A
	Sample No. Dilution Factor Location	64993	64994	64995	64996	65191	65192	65193	65194
10	Hexachlorobutadiene								
10	4-Chloro-3-methylphenol								
10	2-Methylnaphthalene	8 J*	26 J*				16 J*		
10	Hexachlorocyclopentadiene								
10	2,4,6-Trichlorophenol		13 J*						
50	2,4,5-Trichlorophenol								
10	2-Chloronaphthalene								
50	2-Nitroaniline								
10	Dimethylphthalate								
10	Acenaphthylene								
10	2,6-Dinitrotoluene								
50	3-Nitroaniline								
10	Acenaphthene		5 J*						
50	2,4-Dinitrophenol								
50	4-Nitrophenol								
10	Dibenzofuran								
10	2,4-Dinitrotoluene		7 J*						
10	Diethylphthalate								
10	4-Chlorophenyl:phenylether								
10	Fluorene								
50	4-Nitroaniline		8 J*						
50	4,6-Dinitro-2-methylphenol								

CRDL = Contract Required Detection Limit

\*Action Level Exists

SEE NARRATIVE FOR CODE DEFINITIONS

\* E = EXCEEDS QUANTIFIABLE RANGE ; ESTIMATED

J = BELOW QUANTIFIABLE RANGE ; ESTIMATED

Site Name: 3163 BUFFALO AVENUE

WATER SAMPLES  
(ug/L) (PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limits  
(CRQL \* Dilution Factor)

CRQL	COMPOUND	MW-6B	MW-3B	MW-5B	MW-5BD	MW-4A	MW-4B	MW-4BD	MW-BL2
10	Hexachlorobutadiene								
10	4-Chloro-3-methylphenol								
10	2-Methylnaphthalene								
10	Hexachlorocyclopentadiene								
10	2,4,6-Trichlorophenol								
50	2,4,5-Trichlorophenol	65195	65196	65392	65393	65394	65395	65396	65397
10	2-Chloronaphthalene								
50	2-Nitroaniline								
10	Dimethylphthalate								
10	Acenaphthylene								
10	2,6-Dinitrotoluene								
50	3-Nitroaniline								
10	Acenaphthene								
50	2,4-Dinitrophenol								
50	4-Nitrophenol								
10	Dibenzofuran								
10	2,4-Dinitrotoluene								
10	Diethylphthalate								
10	4-Chlorophenyl-phenylether								
10	Fluorene								
50	4-Nitroaniline								
50	4,6-Dinitro-2-methylphenol								

CRDL = Contract Required Detection Limit

\*Action Level Exists

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Site Name: 3163 BUFFALO AVENUE

WATER SAMPLES  
(ug/L)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limits  
(CRQL \* Dilution Factor)

CRQL	COMPOUND	MW-7A	MW-5A	MW-TB3	MW-TB4	MW-5C	MW-5CD	MW-TBS	MW-5E
	Dilution Factor Location	65398	65399	65400	65401	65738	65739	65740	66069
10	Hexachlorobutadiene								
10	4-Chloro-3-methylphenol								
10	2-Methylnaphthalene								
10	Hexachlorocyclopentadiene								
10	2,4,6-Trichlorophenol								
50	2,4,5-Trichlorophenol								
10	2-Chloronaphthalene								
50	2-Nitroaniline								
10	Dimethylphthalate								
10	Acenaphthylene								
10	2,6-Dinitrotoluene								
50	3-Nitroaniline								
10	Acenaphthene								
50	2,4-Dinitrophenol								
50	4-Nitrophenol								
10	Dibenzofuran								
10	2,4-Dinitrotoluene								
10	Diethylphthalate								
10	4-Chlorophenyl-phenylether								
10	Fluorene								
50	4-Nitroaniline								
50	4,6-Dinitro-2-methylphenol								

CRDL = Contract Required Detection Limit      \*Action Level Exists      SEE NARRATIVE FOR CODE DEFINITIONS

Site Name: 3163 BUFFALO AVENUE WATER SAMPLES  
(ug/L)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor)

CRDL	COMPOUND	MW-1A	MW-1B	MW-BL1	MW-TB1	MW-2B	MW-2A	MW-3A	MW-6A
10	N-Nitrosodiphenylamine								
10	4-Bromophenyl-phenylether								
10	*Hexachlorobenzene		92 J*			160 J*	37 J*		
50	*Pentachlorophenol								
10	Phenanthrene	24 J*	47 J*			77 J*	12 J*		
10	Anthracene	4 J*							
10	Di-n-butylphthalate								
10	Fluoranthene	15 J*	40 J*			56 J*	9 J*		
10	Pyrene	10 J*	26 J*			42 J*	7 J*		
10	Butylbenzylphthalate								
20	3,3-Dichlorobenzidine								
10	Benzo(a)anthracene								
10	Chrysene								
10	bis(2-Ethylhexyl)phthalate								
10	Di-n-octylphthalate								
10	Benzo(b)fluoranthene		18 J*						
30	Benzo(k)fluoranthene								
30	Benzo(a)pyrene		7 J*						
30	Indeno(1,2,3-cd)pyrene								
30	Dibenz(a,h)anthracene								
30	Benzo(g,h,i)perylene								

CRDL = Contract Required Detection Limit \*Action Level Exists SEE NARRATIVE FOR CODE DEFINITIONS

\* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED

J = BELOW QUANTIFIABLE RANGE; ESTIMATED

Site Name: 3163 BUFFALO AVENUE  
 WATER SAMPLES  
 (ug/L)(PPL)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
 (CRQL \* Dilution Factor)

CRDL	COMPOUND	Sample No. Dilution Factor Location	MW-6B	MW-3B	MW-5B	MW-5BD	MW-4A	MW-4B	MW-4BD	MW-BL2
10	N-Nitrosodiphenylamine									
10	4-Bromophenyl-phenylether									
10	*Hexachlorobenzene									
50	*Pentachlorophenol									
10	Phenanthrene									
10	Anthracene									
10	Di-n-butylphthalate									
10	Fluoranthene									
10	Pyrene									
10	Butylbenzylphthalate									
20	3,3-Dichlorobenzidine									
10	Benzo(a)anthracene									
10	Chrysene									
10	bis(2-Ethylhexyl)phthalate		6 J							
10	Di-n-octylphthalate									
10	Benzo(b)fluoranthene									
10	Benzo(k)fluoranthene									
10	Benzo(a)pyrene									
10	Indeno(1,2,3-cd)pyrene									
10	Dibenz(a,h)anthracene									
10	Benzo(g,h,i)perylene									

CRDL = Contract Required Detection Limit \*Action Level Exists SEE NARRATIVE FOR CODE DEFINITIONS

\* E = EXCEEDS QUANTIFIABLE RANGE ; ESTIMATED

J = BELOW QUANTIFIABLE RANGE ; ESTIMATED

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Site Name: 3163 BUFFALO AVENUE

WATER SAMPLES  
(ug/L)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor)

CRDL	COMPOUND	MW-7A	MW-5A	MW-TB3	MW-TB4	MW-5C	MW-5CD	MW-TB5	MW-5F
10	N-Nitrosodiphenylamine								
10	4-Bromophenyl-phenylether								
10	*Hexachlorobenzene								
50	*Pentachlorophenol								
10	Phenanthrene	19 J'							
10	Anthracene								
10	Di-n-butylphthalate								
10	Fluoranthene								
10	Pyrene								
10	Butylbenzylphthalate								
20	3,3-Dichlorobenzidine								
10	Benzo(a)anthracene								
10	Chrysene								
10	bis(2-Ethylhexyl)phthalate								
10	Di-n-octylphthalate								12
10	Benzo(b)fluoranthene								
10	Benzo(k)fluoranthene								
10	Benzo(a)pyrene								
10	Indeno(1,2,3-cd)pyrene								
10	Dibenz(a,h)anthracene								
10	Benzo(g,h,i)perylene								

CRDL = Contract Required Detection Limit \*Action Level Exists SEE NARRATIVE FOR CODE DEFINITIONS

\* E=EXCEEDS QUANTIFIABLE RANGE; ESTIMATE  
J' HIGH QUANTIFIABLE RANGE; ESTIMATE

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DATA SUMMARY FORM: P E S T I C I D E S A N D P C B S

TAB: B-1

Site Name: 3163 BUFFALO AVENUE

WATER SAMPLES  
(ug/L)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CROL \* Dilution Factor)

CRQL	COMPOUND	Sample No. Dilution Factor Location	MW-1A	MW-1B	MW-BLI	MW-TBI	MW-2B	MW-2A	MW-3A	MW-6A
0.05	alpha-BHC									
0.05	beta-BHC									
0.05	delta-BHC									
0.05	*Gamma-BHC (Lindane)									
0.05	*Heptachlor									
0.05	Aldrin									
0.05	Heptachlor Epoxide									
0.05	Endosulfan I									
0.10	Dieldrin									
0.10	4,4'-DDE									
0.10	*Endrin									
0.10	Endosulfan II									
0.10	4,4'-DDD									
0.10	Endosulfan Sulfate									
0.10	4,4'-DDT									
0.5	*Methoxychlor									
0.10	Endrin ketone									
0.5	*Alpha-Chlordane									
0.5	*Gamma-Chlordane									
1.0	*Toxaphene									
0.5	*Aroclor-1016									
0.5	*Aroclor-1221									
0.5	*Aroclor-1232									
0.5	*Aroclor-1242									
0.5	*Aroclor-1248									
1.0	*Aroclor-1254									
1.0	*Aroclor-1260									

CRDL = Contract Required Detection Limit  
 \*E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
 J = BELOW QUANTIFIABLE RANGE; ESTIMATED  
 \*Action Level Exists  
 SEE NARRATIVE FOR CODE DEFINITIONS  
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DATA SUMMARY FORM: P E S T I C I D E S A N D P C B S

TAB : B-1

Site Name: 3163 BUFFALO AVENUE

WATER SAMPLES (ug/L)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor)

CRQL	Sample No. Dilution Factor Location	MW-6B	MW-3B	MW-5B	MW-5BD	MW-4A	MW-4B	MW-4BD	MW-BL2
0.05	alpha-BHC					.17 J	.25	2.8	
0.05	beta-BHC			.070		.70	.86	84	
0.05	delta-BHC			.050			.40 J*	1.0	
0.05	*Gamma-BHC (Lindane)	65195	65196	.016 J*			.92	.42 J*	65397
0.05	*Heptachlor								
0.05	Aldrin								
0.05	Heptachlor Epoxide								
0.05	Endosulfan I								
0.10	Dieldrin								
0.10	4,4'-DDE								
0.10	*Endrin								
0.10	Endosulfan II								
0.10	4,4'-DDD								
0.10	Endosulfan Sulfate								
0.10	4,4'-DDT								
0.5	*Methoxychlor								
0.10	Endrin ketone								
0.5	*Alpha-Chlordane								
0.5	*Gamma-Chlordane								
1.0	*Toxaphene								
0.5	*Aroclor-1016								
0.5	*Aroclor-1221								
0.5	*Aroclor-1232								
0.5	*Aroclor-1242								
0.5	*Aroclor-1248								
1.0	*Aroclor-1254								
1.0	*Aroclor-1260								

CRDL = Contract Required Detection Limit \*Action Level Exists SEE NARRATIVE FOR CODE DEFINITIONS

\* E = EXCEEDS, QUANTIFIABLE RANGE; ESTIMATED

J = BELOW QUANTIFIABLE RANGE; ESTIMATED

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DATA SUMMARY FORM: PESTICIDES AND PCB'S

Site Name: 3163 BUFFALO AVENUE WATER SAMPLES (ug/L)(PPB)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor)

CRQL	Sample No. Dilution Factor Location	MW-7A	MW-5A	MW-TB3	MW-TB4	MW-5C	MW-5CD	MW-TBS	MW-5F
COMPOUND		65398	65399	65400	65401	65738	65739	65740	66069
0.05	alpha-BHC						.29		.023 J*
0.05	beta-BHC						.083		
0.05	delta-BHC								
0.05	*Gamma-BHC (Lindane)						.073		
0.05	*Heptachlor								
0.05	Aldrin								
0.05	Heptachlor Epoxide								
0.05	Endosulfan I								
0.10	Dieldrin								
0.10	4,4'-DDE								
0.10	*Endrin								
0.10	Endosulfan II								
0.10	4,4'-DDD								
0.10	Endosulfan Sulfate								
0.10	4,4'-DDT								
0.5	*Methoxychlor								
0.10	Endrin ketone								
0.5	*Alpha-Chlordane								
0.5	*Gamma-Chlordane								
1.0	*Toxaphene								
0.5	*Aroclor-1016								
0.5	*Aroclor-1221								
0.5	*Aroclor-1232								
0.5	*Aroclor-1242								
0.5	*Aroclor-1248								
1.0	*Aroclor-1254								
1.0	*Aroclor-1260								

CRDL = Contract Required Detection Limit \*Action Level Exists  
 \* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
 J = BELOW QUANTIFIABLE RANGE; ESTIMATED

SEE NARRATIVE FOR CODE DEFINITIONS  
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TABLE B-1  
DATA SUMMARY FORM: I N O R G A N I C S

Site Name: 3163 BUFFALO AVENUE

WATER SAMPLES  
(ug/L)(PPB)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

Due to dilution, sample quantitation limit is affected  
See dilution table for specifics.

CRDL	ANALYTE	MW-1A	MW-1B	MW-BL1	MW-TB1	MW-2B	MW-2A	MW-3A	MW-6A	MW-6B
	Sample No.									
	Dilution Factor									
	Location	64993	64994	64995	64996	65191	65192	65193	65194	65195
					NA					
					↓					
200	Aluminum	59600	3540				120,000	64400	33700	5150
60	Antimony									
10	*Arsenic	265	11.8			14.5	38.0	16.5	15.9	9.4
200	Barium	821					8350	649	1280	
5	Beryllium									
5	*Cadmium	21.5	8.4				39.8	21.0	6.0	
5000	Calcium	619,000	588,000			348,000	1,070,000	840,000	758,000	312,000
10	*Chromium	863	51.1				323	242	12.3	21.1
50	Cobalt	653					131			
25	Copper	272	61.4				721	235	254	
100	Iron	186,000	20,000	108		1290	348,000	152,000	73400	11,100
5	*Lead	904	63.2				885	322	477	53.9
5000	Magnesium	113,000	64,400			26,800	40,100	158,000	72,000	53,700
15	Manganese	3140	814			365	7130	4440	1780	393
0.2	Mercury	216	11.9				2.9	5.4		.29
40	*Nickel	1280					419	178	115	
5000	Potassium	18,200	14,800			11,600	40,200	9320	31,800	10,000
5	Selenium									
10	Silver									
5000	Sodium	160,000	23,300			166,000	736,000	15900	31,400	253,000
10	Thallium									
50	Vanadium	87.1					353	133	71.9	
20	Zinc	1610	3230	85.8		74.3	3580	2320	882	394
10	*Cyanide	24.2	84.0			298	256	11.0	34.0	180

\*Action Level Exists

CRDL = Contract Required Detection Limit

SEE NARRATIVE FOR CODE DEFINITIONS

TABLE B-1

DATA SUMMARY FORM: I N O R G A N I C S

Site Name: 3163 BUFFALO AVENUE

WATER SAMPLES  
(ug/L)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

\*Due to dilution, sample quantitation limit is affected  
See dilution table for specifics.

CRDL	Sample No.	MW-3B	MW-5B	MW-5BD	MW-4A	MW-4B	MW-4BD	MW-8L2	MW-7A	MW-5A
	Dilution Factor	65196	65392	65393	65394	65395	65396	65397	65398	65399
	ANALYTE									
200	Aluminum	471	9770	9160	74200	5620	5230		59300	12600
60	Antimony									
10	*Arsenic	14.8	13.5	11.0	18.5				69.7	13.2
200	Barium	97.7			734				1400	1500
5	Beryllium									
5	*Cadmium		10.5	10.7	37.0				46.2	39.6
5000	Calcium	249,000	1,090,000	940,000	1,360,000	400,000	353,000		496,000	1,290,000
10	*Chromium	46.4	38.2	63.7	128	38.5	54.5		252	260
50	Cobalt	13.0			62.1				60.9	112
25	Copper		40.1	28.2	229		17.1		861	531
100	Iron	7640	27500	27,800	151,000	10100	9990		280,000	292,000
5	*Lead	14.7	524	485	481	51.0	52.0		3430	424
5000	Magnesium	22,600	435,000	302,000	283,000	453,000	46,200		135,000	245,000
15	Manganese	363	2530	2170	7270	472	490		4030	7230
0.2	Mercury	.24			.92				3.7	2.1
40	*Nickel			37.1	182	230			271	339
5000	Potassium	16,000	14100	12900	13,100	11700	12,600		164,000	11000
5	Selenium									
10	Silver									
5000	Sodium	235,000	121,000	109,000	67500	113000	127,000		159,000	21700
10	Thallium									
50	Vanadium				155	367	10.0		164	255
20	Zinc	408	1490	1420	5170	30.0	10.0		8690	2320
10	*Cyanide	1890		21.0			10.0		30.0	

CRDL = Contract Required Detection Limit      \*Action Level Exists      SEE NARRATIVE FOR CODE DEFINITIONS

TABLE B-1  
DATA SUMMARY FORM: I N O R G A N I C S

Site Name: 3163 BUFFALO AVENUE  
Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

WATER SAMPLES  
(ug/L) (PPb)

+Due to dilution, sample quantitation limit is affected  
See dilution table for specifics.

CRDL	Sample No. Dilution Factor	ANALYTE	MW-TB3	MW-TB4	MW-5C	MW-5CD	MW-TBS	MW-5F
			65400	65401	65738	65739	65740	66069
200		Aluminum	NA	NA	2240	982	NA	1680
60		Antimony			35.5			
10		*Arsenic						
200		Barium						
5		Beryllium						
5		*Cadmium						
5000		Calcium		121,000		630,000		563000
10		*Chromium		33.5		24.3		88.0
50		Cobalt						
25		Copper						
100		Iron		7290		3730		2650
5		*Lead		67.0		86.0		21.9
5000		Magnesium		30,700		144,000		88100
15		Manganese		218		584		93.2
0.2		Mercury		.43				
40		*Nickel						
5000		Potassium		11800		11200		10200
5		Selenium						
10		Silver						
5000		Sodium		1380000		255000		196000
10		Thallium						
50		Vanadium						
20		Zinc		486		400		177
10		*Cyanide		2540		825		

CRDL = Contract Required Detection Limit      \*Action Level Exists      SEE NARRATIVE FOR CODE DEFINITIONS      Draft      revised 12/88

DATA SUMMARY FORM: V O L A T I L E S 1

Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
(ug/Kg)(Ppb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / (100 - % moisture/100)

CRQL	COMPOUND	Sample No. Dilution Factor % Moisture Location	SB-1B2	SB-1BC	SB-2A2	SB-2AC	SB-5B2	SB-5BC	DU-5BC
10	Chloromethane								
10	Bromomethane								
10	Vinyl Chloride								
10	Chloroethane								
5	Methylene Chloride								
10	Acetone								
5	Carbon Disulfide								
5	1,1-Dichloroethene								
5	1,1-Dichloroethane								
5	Total-1,2-Dichloroethene								
5	Chloroform								
5	1,2-Dichloroethane								
10	2-Butanone								
5	1,1,1-Trichloroethane								
5	Carbon Tetrachloride								
10	Vinyl Acetate								
5	Bromodichloromethane								
		56049	56050	57225	57308	58105	58106	58107	

CRQL = Contract Required Detection Limit

SEE NARRATIVE FOR CODE DEFINITIONS

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TABLE 6-2  
DATA SUMMARY FORM: VOLATILES 1

Site Name: 3163 BUFFALO AVENUE SOIL SAMPLES  
(ug/Kg)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_  
To calculate sample quantitation limit:  
(CROL \* Dilution Factor) / ((100 - % moisture) / 100)

CRQL	COMPOUND	Sample No. Dilution Factor % Moisture Location	SB-3A2	SB-3AC	SB-6A2	SB-6AC	SB-4AC	SB-7A2	SB-7AC	SB-4A2
10	Chloromethane									
10	Bromomethane									
10	Vinyl Chloride									
10	Chloroethane									
5	Methylene Chloride									
10	Acetone									
5	Carbon Disulfide									
5	1,1-Dichloroethene									
5	1,1-Dichloroethane									
5	Total-1,2-Dichloroethene									
5	Chloroform									
5	1,2-Dichloroethane									
10	2-Butanone									
5	1,1,1-Trichloroethane									
5	Carbon Tetrachloride									
10	Vinyl Acetate									
5	Bromodichloromethane									
		58161	58162	58250	58251	58517	58563	58564	61038	

CRDL = Contract Required Detection Limit

SEE NARRATIVE FOR CODE DEFINITIONS

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TABL B-2  
DATA SUMMARY FORM: VOLATILES 1

Site Name: 363 BUFFALO AVENUE SOIL SAMPLES  
(ug/Kg)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_  
To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / ((100 - % moisture)/100)

CRQL	COMPOUND	Sample No. Dilution Factor % Moisture Location	SE-01	SB-E	SB-D	SB-C	SB-A	SB-B	SB-J	SB-JD
10	Chloromethane									
10	Bromomethane									
10	Vinyl Chloride									
10	Chloroethane									
5	Methylene Chloride									
10	Acetone									
5	Carbon Disulfide		200							
5	1,1-Dichloroethene									
5	1,1-Dichloroethane									
5	Total-1,2-Dichloroethene									
5	Chloroform									
5	1,2-Dichloroethane									
10	2-Butanone									
5	1,1,1-Trichloroethane									
5	Carbon Tetrachloride									
10	Vinyl Acetate									
5	Bromodichloromethane									
			61318	68845	69002	69003	69004	69005	69006	69006

CRDL = Contract Required Detection Limit  
SEE NARRATIVE FOR CODE DEFINITIONS  
revised 12/88

DATA SUMMARY FORM: VOLATILES 1

Site Name: 3163 BUFFALO AVENUE SOIL SAMPLES  
(ug/Kg)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / (100 - % moisture)/100)

CRQL	COMPOUND	SB-I	SB-F	SB-H	SB-G	SE-2		
	Sample No.	69181	69183	69184	69185	67818		
	Dilution Factor							
	% Moisture							
	Location							
10	Chloromethane							
10	Bromomethane							
10	Vinyl Chloride							
10	Chloroethane							
5	Methylene Chloride							
10	Acetone							
5	Carbon Disulfide	96,000	780,000	290,000	2,200,000			
5	1,1-Dichloroethane							
5	1,1-Dichloroethane							
5	Total-1,2-Dichloroethene							
5	Chloroform							
5	1,2-Dichloroethane							
10	2-Butanone							
5	1,1,1-Trichloroethane							
5	Carbon Tetrachloride							
10	Vinyl Acetate							
5	Bromodichloromethane							

CRDL = Contract Required Detection Limit

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Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
(ug/Kg)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / ((100 - % moisture)/100)

CRQL	COMPOUND	Sample No. Dilution Factor % Moisture Location	SB-102	SB-10C	SB-2A2	SB-2AC	SB-5B2	SB-50C	DU-50C
5	1,2-Dichloropropane								
5	Cis-1,3-Dichloropropene								
5	Trichloroethene								
5	Dibromochloromethane								
5	1,1,2-Trichloroethane								
5	Benzene		68,000	120,000	56,000	50,000			
5	Trans-1,3-Dichloropropene								
5	Bromoform								
10	4-Methyl-2-pentanone								
10	2-Hexanone								
5	Tetrachloroethene								
5	1,1,2,2-Tetrachloroethane								
5	Toluene								
5	Chlorobenzene		430,000 E*	280,000	90,000	490,000	280 J*	290 J*	
5	Ethylbenzene								
5	Styrene								
5	Total Xylenes								

SEE NARRATIVE FOR CODE DEFINITIONS

CRQL = Contract Required Quantitation Limit

\* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
J = BELOW QUANTIFIABLE RANGE; ESTIMATED

revised 12/86

TABU B-2  
DATA SUMMARY FORM: VOLATILES 2

Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
(ug/Kg)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / ((100 - % moisture)/100)

CRQL	COMPOUND	Sample No. Dilution Factor % Moisture Location	SB-3A2	SB-3AC	SB-6A2	SB-6AC	SB-4AC	SB-7A2	SB-7AC	SB-4A2
5	1,2-Dichloropropane									
5	Cis-1,3-Dichloropropene									
5	Trichloroethene									
5	Dibromochloromethane									
5	1,1,2-Trichloroethane									
5	Benzene		58161	58162	58250	58251	58517	58563	58564	61038
5	Trans-1,3-Dichloropropene									
5	Bromoform									
10	4-Methyl-2-pentanone									
10	2-Hexanone									
5	Tetrachloroethene									
5	1,1,2,2-Tetrachloroethane									
5	Toluene									
5	Chlorobenzene				560 J*				420,000	45
5	Ethylbenzene									
5	Styrene									
5	Total Xylenes				1100 J*	210 J*				4 J*

SEE NARRATIVE FOR CODE DEFINITIONS

CRQL = Contract Required Quantitation Limit

\* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
J = BELOW QUANTIFIABLE RANGE; ESTIMATED

revised 12/86

Site Name: 3163 BUFFALO AVENUE SOIL SAMPLES (ug/Kg)(PPb)

Case #: Sampling Date(s):

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / ((100 - % moisture)/100)

CRQL	COMPOUND	Sample No. Dilution Factor % Moisture Location	SE-OI	SB-E	SB-D	SB-C	SB-A	SB-B	SB-J	SB-JD
5	1,2-Dichloropropane									
5	Cis-1,3-Dichloropropene									
5	Trichloroethene									
5	Dibromochloromethane									
5	1,1,2-Trichloroethane									
5	Benzene	4100 E*	34000 J*	68845	69002	69003	69004	69005	69006	69007
5	Trans-1,3-Dichloropropene									
5	Bromoform									
10	4-Methyl-2-pentanone									
10	2-Hexanone									
5	Tetrachloroethene									
5	1,1,2,2-Tetrachloroethane									
5	Toluene									
5	Chlorobenzene	25000	1500000	56000 J*	75000 J*	34000 J*	100000 J*			
5	Ethylbenzene									
5	Styrene									
5	Total Xylenes									

CRQL = Contract Required Quantitation Limit SEE NARRATIVE FOR CODE DEFINITIONS

\* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
J = BELOW QUANTIFIABLE RANGE; ESTIMATED

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Draft

Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
(ug/Kg)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / ((100 - % moisture)/100)

CRQL	COMPOUND	SB-I	SB-F	SB-H	SB-G	SB-E-2
5	1,2-Dichloropropane					
5	Cis-1,3-Dichloropropene					
5	Trichloroethene					
5	Dibromochloromethane					
5	1,1,2-Trichloroethane	69181	69183	69184	69185	69818
5	Benzene					
5	Trans-1,3-Dichloropropene					
5	Bromoforn					
10	4-Methyl-2-pentanone					
10	2-Hexanone					
5	Tetrachloroethene					
5	1,1,2,2-Tetrachloroethane					
5	Toluene					
5	Chlorobenzene		19,000 J	24,000 J		
5	Ethylbenzene					
5	Styrene					
5	Total Xylenes					

SEE NARRATIVE FOR CODE DEFINITIONS

CRQL = Contract Required Quantitation Limit

\* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
J = BELOW QUANTIFIABLE RANGE; ESTIMATED

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Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
(ug/Kg)(PPB)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / (100 - % moisture/100)

CRQL	Sample No. Dilution Factor % Moisture Location	SB-1B2	SB-1B3	SB-2A2	SB-2A3	SB-5B2	SB-5B3	DU-5B3
330	Phenol	6100	2200 J*					
330	bis(2-Chloroethyl)ether							
330	2-Chlorophenol	850 J*						
330	1,3-Dichlorobenzene	110,000	39,000	7700				
330	1,4-Dichlorobenzene	310,000 E*	150,000	24,000			51 J*	
330	Benzyl Alcohol							
330	1,2-Dichlorobenzene	250,000 E*	98,000	16,000				
330	2-Methylphenol							
330	bis(2-Chloroisopropyl)ether							
330	4-Methylphenol							
330	N-Nitroso-di-n-propylamine							
330	Hexachloroethane							
330	Nitrobenzene							
330	Isophorone							
330	2-Nitrophenol							
330	2,4-Dimethylphenol							
1600	Benzoic Acid							
330	bis(2-Chloroethoxy)methane							
330	2,4-Dichlorophenol							
330	1,2,4-Trichlorobenzene	160,000	72,000	27,000				
330	Naphthalene	2600		420 J*				
330	4-Chloroaniline							

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CRQL = Contract Required Quantitation Limit

\* E=EXCEEDS, QUANTIFIABLE RANGE; ESTIMATED

J=BELOW QUANTIFIABLE RANGE; ESTIMATED



Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
(ug/Kg)(Ppb)

Case #: Sampling Date(s):

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / ((100 - % moisture)/100)

CRQL	Sample No. Dilution Factor % Moisture Location	SB-3A2	SB-3AC	SB-6A2	SB-6AC	SB-4AC	SB-7A2	SB-7AC	SB-4A2
COMPOUND									
330	Phenol		210 J*	180 J*	190 J*				
330	bis(2-Chloroethyl)ether								
330	2-Chlorophenol								
330	1,3-Dichlorobenzene		60 J*	740 J*	180 J*	600 J*	73000	36000 J*	2500 J*
330	1,4-Dichlorobenzene		160 J*	770 J*	200 J*	2700 J*	850,000	500,000	6200
330	Benzyl Alcohol								
330	1,2-Dichlorobenzene	120 J*	1200	440 J*	110 J*	32000	1,500,000	650,000	1600 J*
330	2-Methylphenol								
330	bis(2-Chloroisopropyl)ether								
330	4-Methylphenol		75 J*	62 J*					
330	N-Nitroso-di-n-propylamine								
330	Hexachloroethane								
330	Nitrobenzene								
330	Isophorone	3100							
330	2-Nitrophenol								
330	2,4-Dimethylphenol		64 J*						
1500	Benzoic Acid								
330	bis(2-Chloroethoxy)methane								
330	2,4-Dichlorophenol								
330	1,2,4-Trichlorobenzene		200 J*	1700	480 J*	340,000	2,100,000	730,000	51,000
330	Naphthalene			510 J*	100 J*	920	930		
330	4-Chloroaniline								

CRQL = Contract Required Quantitation Limit  
 \* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
 J = BELOW QUANTIFIABLE RANGE; ESTIMATED  
 SEE NARRATIVE FOR CODE DEFINITIONS  
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TABLE B-2  
DATA SUMMARY FORM: B N A S 1

Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
(ug/Kg(PPb))

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / ((100 - % moisture)/100)

CRQL	Sample No. Dilution Factor % Moisture Location	SE-01	SB-E	SB-D	SB-C	SB-A	SB-B	SB-J	SB-JD
330	Phenol								
330	bis(2-Chloroethyl)ether								
330	2-Chlorophenol								
330	1,3-Dichlorobenzene		170,000		2200	46,000 J*	41,000 J*	160,000	140,000 J*
330	1,4-Dichlorobenzene	470,000	1,900,000		4100	97,000	79,000	942,000	930,000
330	Benzyl Alcohol								
330	1,2-Dichlorobenzene	840,000	2,200,000		9400	360,000	300,000	2,004,000	200,000
330	2-Methylphenol				640				
330	bis(2-Chloroisopropyl)ether								
330	4-Methylphenol				920				
330	N-Nitroso-di-n-propylamine								
330	Hexachloroethane								
330	Nitrobenzene								
330	Isophorone								
330	2-Nitrophenol								
330	2,4-Dimethylphenol				640				
1600	Benzoic Acid								
330	bis(2-Chloroethoxy)methane								
330	2,4-Dichlorophenol								
330	1,2,4-Trichlorobenzene	2,700,000	2,300,000		4200	340,000	470,000	2,150,000	1,900,000
330	Naphthalene				170 J*				
330	4-Chloroaniline								

CRQL = Contract Required Quantitation Limit SEE NARRATIVE FOR CODE DEFINITIONS

\* EXCEEDED QUANTIFIABLE RANGE; ESTIMATED  
 \* EXCEEDED QUANTIFIABLE RANGE; ESTIMATED

Site Name: 363 BUFFALO AVENUE

SOIL SAMPLES  
(ug/Kg)(Ppb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CROL \* Dilution Factor) / (100 - % moisture)

CROL	COMPOUND	Sample No.		SB-F	SB-H	SB-G	SE-2
		Dilution Factor	% Moisture				
330	Phenol						
330	bis(2-Chloroethyl)ether						
330	2-Chlorophenol						
330	1,3-Dichlorobenzene	1700 J*		240,000	54,000 J*	79,000	15,000
330	1,4-Dichlorobenzene	140,000		910,000	330,000	240,000	39,000
330	Benzyl Alcohol						
330	1,2-Dichlorobenzene	270,000		2,400,000 E*	640,000	380,000	52,000
330	2-Methylphenol						
330	bis(2-Chloroisopropyl)ether						
330	4-Methylphenol						
330	N-Nitroso-di-n-propylamine						
330	Hexachloroethane						
330	Nitrobenzene						
330	Isophorone						
330	2-Nitrophenol						
330	2,4-Dimethylphenol						
1600	Benzoic Acid						
330	bis(2-Chloroethoxy)methane						
330	2,4-Dichlorophenol						
330	1,2,4-Trichlorobenzene	1900,000		2,800,000 E*	4,300,000 E*	1,100,000 E*	120,000
330	Naphthalene						840 J*
330	4-Chlorophenol						

CROL = Contract Required Quantitation Limit

\* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED

J = BELOW QUANTIFIABLE RANGE; ESTIMATED

SEE NARRATIVE FOR CODE DEFINITION

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TABLE B-2  
DATA SUMMARY FORM: B N A S 2

Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
(ug/Kg)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / ((100 - % moisture)/100)

CRQL	Sample No. Dilution Factor % Moisture Location	SB-1B2	SB-1B3	SB-2A2	SB-2AC	SB-5B2	SB-5B3	DU-5B3	ecology and environment
COMPOUND									
330	Hexachlorobutadiene	99 J*							
330	4-Chloro-3-methylphenol								
330	2-Methylnaphthalene	2300		400 J*	430 J*				
330	Hexachlorocyclopentadiene								
330	2,4,6-Trichlorophenol								
1600	2,4,5-Trichlorophenol								
330	2-Chloronaphthalene								
1600	2-Nitroaniline								
330	Dimethylphthalate								
330	Acenaphthylene	71 J*		83 J*					
330	2,6-Dinitrotoluene								
1600	3-Nitroaniline								
330	Acenaphthene								
1600	2,4-Dinitrophenol	2300		580 J*	85 J*		85 J*		
1600	4-Nitrophenol								
330	Dibenzofuran	2100		550 J*	99 J*				
330	2,4-Dinitrotoluene								
330	Diethylphthalate								
330	4-Chlorophenyl-phenylether								
330	Fluorene	2100		170 J*	100 J*		91 J*		
1600	4-Nitroaniline								
1600	4,6-Dinitro-2-methylphenol								

SEE NARRATIVE FOR CODE DEFINITIONS

CRQL = Contract Required Quantitation Limit

E = EXCEEDS QUANTITABLE RANGE; ESTIMATED  
J = BELOW QUANTITABLE RANGE; ESTIMATED

Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
(ug/Kg)(ppb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / ((100 - % moisture)/100)

CRQL	COMPOUND	Sample No. Dilution Factor % Moisture Location	SB-3A2	SB-3AC	SB-6A2	SB-6AC	SB-4AC	SB-7A2	SB-7AC	SB-4A2
330	Hexachlorobutadiene									
330	4-Chloro-3-methylphenol									
330	2-Methylnaphthalene			180 J*						
330	Hexachlorocyclopentadiene									
330	2,4,6-Trichlorophenol									
1600	2,4,5-Trichlorophenol									
330	2-Chloronaphthalene									
1600	2-Nitroaniline									
330	Dimethylphthalate									
330	Acenaphthylene			44 J*						
330	2,6-Dinitrotoluene									
1600	3-Nitroaniline			340 J			1100 J*			
330	Acenaphthene									
1600	2,4-Dinitrophenol									
1600	4-Nitrophenol									
330	Dibenzofuran			320 J*		56 J*	220 J*			
330	2,4-Dinitrotoluene									
330	Diethylphthalate		490 J*	460 J*		54 J	690 J*			
330	4-Chlorophenyl-phenylether									
330	Fluorene						410 J*			
1600	4-Nitroaniline									
1600	4,6-Dinitro-2-methylphenol									

SEE NARRATIVE FOR CODE DEFINITIONS

CRQL = Contract Required Quantitation Limit

\* E = EXCEEDS QUANTITABLE RANGE ; ESTIMATED  
J = BELOW QUANTITABLE RANGE ; ESTIMATED

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Draft

Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
(ug/Kg)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CROL \* Dilution Factor) / ((100 - % moisture)/100)

CROL	COMPOUND	Sample No. Dilution Factor % Moisture Location	SE - 01	SB - E	SB - D	SB - C	SB - A	SB - B	SB - J	SB - JD
330	Hexachlorobutadiene									
330	4-Chloro-3-methylphenol									
330	2-Methylnaphthalene	3000								
330	Hexachlorocyclopentadiene									
330	2,4,6-Trichlorophenol									
1600	2,4,5-Trichlorophenol				69003	69004	69005	69006		69007
330	2-Chloronaphthalene									
1600	2-Nitroaniline									
330	Dimethylphthalate									
330	Acenaphthylene									
330	2,6-Dinitrotoluene									
1600	3-Nitroaniline									
330	Acenaphthene	270 J*			80 J*					
1600	2,4-Dinitrophenol									
1600	4-Nitrophenol									
330	Dibenzofuran	140 J*			75 J*					
330	2,4-Dinitrotoluene									
330	Diethylphthalate				190 J*					
330	4-Chlorophenylphenylether									
330	Fluorene	140 J*			54 J*					
1600	4-Nitroaniline									
1600	4,6-Dinitro-2-methylphenol									

SEE NARRATIVE FOR CODE DEFINITION

CROL = Contract Required Quantitation Limit

\* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED.  
 B = BELOW QUANTIFIABLE RANGE; ESTIMATED

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TABLL B-2  
 DATA SUMMARY FORM: B N A S 2

Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
 (ug/Kg)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
 (CROL \* Dilution Factor) / (100 - % moisture)/100

ORQL	Sample No. Dilution Factor % Moisture Location	SB-I	SB-F	SB-H	SB-G	SE-2			
	COMPOUND								
330	Hexachlorobutadiene								
330	4-Chloro-3-methylphenol								
330	2-Methylnaphthalene				160 J*				
330	Hexachlorocyclopentadiene								
330	2,4,6-Trichlorophenol								
1600	2,4,5-Trichlorophenol								
330	2-Chloronaphthalene								
1600	2-Nitroaniline								
330	Dimethylphthalate								
330	Acenaphthylene								
330	2,6-Dinitrotoluene								
1600	3-Nitroaniline								
330	Acenaphthene					370 J*			
1600	2,4-Dinitrophenol								
1600	4-Nitrophenol								
330	Dibenzofuran								
330	2,4-Dinitrotoluene								
330	Diethylphthalate					60 J*			
330	4-Chlorophenyl-phenylether								
330	Fluorene					350 J*			
1600	4-Nitroaniline								
1600	4,6-Dinitro-2-methylphenol								

SEE NARRATIVE FOR CODE DEFINITIONS

CROL = Contract Required Quantitation Limit

\* E = EXCEPTS QUANTIFIABLE VALUES; ESTIMATED  
 J = BELOW QUANTIFIABLE VALUES; ESTIMATED

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Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
(ug/Kg)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / (100 - % moisture)/100

CRQL	COMPOUND	SB-1B2	SB-1BC	SB-2A2	SB-2AC	SB-5B2	SB-5BC	DU-5BC
330	N-Nitrosodiphenylamine							
330	4-Bromophenyl-phenylether							
330	Hexachlorobenzene	1600		1900 J*	1300 J*			
1600	Pentachlorophenol							
330	Phenanthrene	19000	2400 J*	4600	670 J*	540 J*	77 J*	
330	Anthracene	4700	590 J*	1500 J*	140 J*	91 J*		
330	Di-n-butylphthalate							
330	Fluoranthene	17000	2200 J*	5400	760 J*	600 J*	110 J*	
330	Pyrene	12,000	2000 J*	5000	650 J*	240 J*	100 J*	
330	Butylbenzylphthalate							
1600	3,3-Dichlorobenzidine							
330	Benzo(a)anthracene	7700		3000 J*	320 J*		73 J*	
330	Chrysene	8500		2800 J*	350 J*	770 J*	88 J*	
330	bis(2-Ethylhexyl)phthalate							
330	Di-n-octylphthalate							
330	Benzo(b)fluoranthene	9280	2000 J*	5500	610 J*	33,000 E*	120 J*	
330	Benzo(k)fluoranthene							
330	Benzo(a)pyrene	5100		3200 J*	320 J*	13,000	73 J*	
330	Indeno(1,2,3-cd)pyrene	3200		1900 J*	230 J*	7,000	59 J*	
330	Dibenz(a,h)anthracene	1300		580 J*		5300		
330	Benzo(g,h,i)perylene	2900		2000 J*	240 J*	8200	69 J*	

CRQL = Contract Required Quantitation Limit SEE NARRATIVE FOR CODE DEFINITIONS

\* E-EXCEPTS QUANTIFIABLE VALUES ESTABLISHED  
J=BLIND QUANTIFIABLE VALUES ESTABLISHED

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Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
(ug/Kg)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / ((100 - % moisture)/100)

CRQL	COMPOUND	SB-3A2	SB-3AC	SB-6A2	SB-6AC	SB-4AC	SB-7A2	SB-7AC	SB-4A2
	Sample No.	58161	58162	58251	58251	58517	58563	58564	61038
	Dilution Factor								
	% Moisture								
	Location								
	COMPOUND								
330	N-Nitrosodiphenylamine								
330	4-Bromophenyl-phenylether								
330	Hexachlorobenzene			170 J*	48 J*				
1600	Pentachlorophenol								
330	Phenanthrene	130 J*	160 J*	3300	430 J*	4800	660 J*		
330	Anthracene			1000	93 J*	1200 J			
330	Di-n-butylphthalate				70 J*				
330	Fluoranthene	110 J*	180 J*	4100	710 J*	9400	730 J*		
330	Pyrene		160 J*	4300	280 J*	7000			
330	Butylbenzylphthalate								
1600	3,3-Dichlorobenzidine								
330	Benzo(a)anthracene	250 J*			450 J*	3900			
330	Chrysene	290 J*	350 J		580 J*	5000			
330	bis(2-Ethylhexyl)phthalate								
330	Di-n-octylphthalate								
330	Benzo(b)fluoranthene	370 J*	6100	3800	14,000 E*	6000			
330	Benzo(k)fluoranthene								
330	Benzo(a)pyrene	190 J*	3600	2200	6300	2600 J*	1200 J*		
330	Indeno(1,2,3-cd)pyrene	65 J*	2000	1500	2900	1700 J*			
330	Dibenz(a,h)anthracene	180 J*	3500	420 J*	2300	450 J*			
330	Benzo(g,h,i)perylene	140 J*	3800	1500	5200	1500 J	1700 J*		

CRQL = Contract Required Quantitation Limit

SEE NARRATIVE FOR CODE DEFINITIONS

\* E = EXCEEDS QUANTIFIABLE RANGE; J = JUDGMENT  
J = JUDGMENT QUANTIFIABLE RANGE; E = EXCEEDS

Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
(ug/Kg) (PPB)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / ((100 - % moisture)/100)

CRQL	Sample No. Dilution Factor % Moisture Location	SE-O1	SB-E	SB-D	SB-C	SB-A	SB-B	SB-J	SB-JD
COMPOUND									
330	N-Nitrosodiphenylamine								
330	4-Bromophenyl-phenylether								
330	Hexachlorobenzene								
1600	Pentachlorophenol								
330	Phenanthrene	730 J*			840	1400 J*	3200 J*		
330	Anthracene				100 J*				
330	Di-n-butylphthalate								
330	Fluoranthene								
330	Pyrene	1100 J*							
330	Butylbenzylphthalate								
1600	3,3-Dichlorobenzidine								
330	Benzo(a)anthracene								
330	Chrysene				560				
330	bis(2-Ethylhexyl)phthalate				870				
330	Di-n-octylphthalate								
330	Benzo(b)fluoranthene	1300 J*			1700				
330	Benzo(k)fluoranthene								
330	Benzo(a)pyrene	590 J*			650				
330	Indeno(1,2,3-cd)pyrene	570 J*			1000				
330	Dibenz(a,h)anthracene				290 J*				
330	Benzo(g,h,i)perylene	470 J*			1100				

SEE NARRATIVE FOR CODE DEFINITIONS

CRQL = Contract Required Quantitation Limit

\* E = EXCEPTED QUANTIFIABLE VALUE; ESTIMATED

J = BELOW QUANTIFIABLE VALUE; ESTIMATED

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Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES (ug/Kg)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CROL \* Dilution Factor) / ((100 - % moisture)

CROL	COMPOUND	Sample No. Dilution Factor % Moisture Location	SB-I	SB-F	SB-H	SB-G	SE-2
330	N-Nitrosodiphenylamine		64181	69183	69184	69185	69818
330	4-Bromophenyl phenylether						86 J*
330	Hexachlorobenzene			12,000 J*			1500 J*
1600	Pentachlorophenol						2400 J*
330	Phenanthrene						430 J*
330	Anthracene						2400 J*
330	Di-n-butylphthalate						2000 J*
330	Fluoranthene						
330	Pyrene						
330	Butylbenzylphthalate						
1600	3,3-Dichlorobenzidine						
330	Benzo(a)anthracene						
330	Chrysene						1300 J*
330	bis(2-Ethylhexyl)phthalate						
330	Di-n-octylphthalate						220 J*
330	Benzo(b)fluoranthene						2500 J*
330	Benzo(k)fluoranthene						
330	Benzo(a)pyrene						1200 J*
330	Indeno(1,2,3-cd)pyrene						1100 J*
330	Dibenz(a,h)anthracene						290
330	Benzo(g,h,i)perylene						990

CROL = Contract Required Quantitation Limit

SEE NARRATIVE FOR CODE DEFINITION

\* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
J = BELOW QUANTIFIABLE RANGE; ESTIMATED

DATA SUMMARY FORM: PESTICIDES AND PCBS

Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES (ug/Kg)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / ((100 - % moisture)/100)

CRQL	COMPOUND	Sample No. Dilution Factor % Moisture Location	SB-1B2	SB-1B2C	SB-2A2	SB-2AC	SB-5B2	SB-5B2C	DU-5B2C
8	alpha-BHC								
8	beta-BHC								
8	delta-BHC								
8	Gamma-BHC (Lindane)								
8	Heptachlor								
8	Aldrin								
8	Heptachlor Epoxide								
8	Endosulfan I								
16	Dieldrin								
16	4,4'-DDE								
16	Endrin								
16	Endosulfan II								
16	4,4'-DDD								
16	Endosulfan Sulfate								
16	4,4'-DDT								
80	Methoxychlor								
16	Endrin ketone								
80	Alpha-Chlordane								
80	Gamma-Chlordane								
160	Toxaphene								
80	Aroclor-1016								
80	Aroclor-1221								
80	Aroclor-1232								
80	Aroclor-1242								
80	Aroclor-1248								
160	Aroclor-1254								
160	Aroclor-1260								
		56049	56050	57225	57308	58105	58106	58107	
									7.8 J

recycled paper

SEE NARRATIVE FOR CODE DEFINITIONS  
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CRQL = Contract Required Quantitation Limit

\* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED

T = BELOW QUANTIFIABLE RANGE; ESTIMATED

DATA SUMMARY FORM: PESTICIDES AND PCBs

TABLE B-2

Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES (ug/Kg)(PPb)

Case #: Sampling Date(s):

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / ((100 - % moisture)/100)

CRQL	Sample No. Dilution Factor % Moisture Location	SB-3A2	SB-3AC	SB-6A2	SB-6AC	SB-7A2	SB-7AC	SB-4A2
8	alpha-BHC							
8	beta-BHC							
8	delta-BHC							
8	Gamma-BHC (Lindane)							
8	Heptachlor							
8	Aldrin							
8	Heptachlor Epoxide							
8	Endosulfan I							
16	Dieldrin							
16	4,4'-DDE							
16	Endrin							
16	Endosulfan II							
16	4,4'-DDD							
16	Endosulfan Sulfate							
16	4,4'-DDT							
80	Methoxychlor							
16	Endrin ketone							
80	Alpha-Chlordane							
80	Gamma-Chlordane							
160	Toxaphene							
80	Aroclor-1018							
80	Aroclor-1221							
80	Aroclor-1232							
80	Aroclor-1242							
80	Aroclor-1248							
160	Aroclor-1254							
160	Aroclor-1260							

74 J\*  
300  
40 J\*

1600 J\*  
480 J\*

58564  
61038

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CRQL = Contract Required Quantitation Limit

\* EXCEPT WHERE QUANTITIES ARE LARGE, ESTIMATED  
TERMINAL QUANTITIES HAVE BEEN CALCULATED

DATA SUMMARY FORM: PESTICIDES AND PCB'S

Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES (ug/Kg)(PPD)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_ To calculate sample quantitation limit: (CRQL \* Dilution Factor) / ((100 - % moisture)/100)

CRQL	COMPOUND	Sample No. Dilution Factor % Moisture Location	SE-01	SB-E	SB-D	SB-C	SB-A	SB-B	SB-J	SB-JD
8	alpha-BHC									
8	beta-BHC									
8	delta-BHC									
8	Gamma-BHC (Lindane)									
8	Heptachlor									
8	Aldrin									
8	Heptachlor Epoxide									
8	Endosulfan I									
16	Dieldrin									
16	4,4'-DDE									
16	Endrin									
16	Endosulfan II									
16	4,4'-DDD									
16	Endosulfan Sulfate									
16	4,4'-DDT									
80	Methoxychlor									
16	Endrin ketone									
80	Alpha-Chlordane									
80	Gamma-Chlordane									
160	Toxaphene									
80	Aroclor-1018									
80	Aroclor-1221									
80	Aroclor-1232									
80	Aroclor-1242									
80	Aroclor-1248									
160	Aroclor-1254									
160	Aroclor-1260									
				451 J*						

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CRQL = Contract Required Quantitation Limit  
\* EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
J- BELOW QUANTIFIABLE RANGE; ESTIMATE P

TABLE B-2  
DATA SUMMARY FORM: PESTICIDES AND PCBs

Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
(ug/Kg)(PPb)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor) / ((100 - % moisture)/100)

LOI	Sample No. Dilution Factor % Moisture Location	SB-I	SB-F	SB-H	SB-G	SE-2	ecology and environment
COMPOUND							
3	alpha-BHC						
3	beta-BHC						
3	delta-BHC						
3	Gamma-BHC (Lindane)						
3	Heptachlor						
3	Aldrin						
3	Heptachlor Epoxide						
3	Endosulfan I						
16	Dieldrin						
16	4,4'-DDE						
16	Endrin						
16	Endosulfan II						
16	4,4'-DDD						
16	Endosulfan Sulfate						
16	4,4'-DDT						
80	Methoxychlor						
16	Endrin ketone						
80	Alpha-Chlordane						
80	Gamma-Chlordane						
160	Toxaphene						
80	Aroclor-1016						
80	Aroclor-1221						
80	Aroclor-1232						
80	Aroclor-1242						
80	Aroclor-1248						
160	Aroclor-1254						
160	Aroclor-1260						
		69181	69183	69184	69185	69818	

SEE NARRATIVE FOR CODE DEFINITIONS  
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CRQL = Contract Required Quantitation Limit

TABLE B-2  
DATA SUMMARY FORM: I N O R G A N I C S

Site Name: 3163 BUFFALO AVENUE SOIL SAMPLES  
(mg/Kg)(PPM)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

+Due to dilution, sample quantitation limit is affected.  
See dilution table for specifics.

CRDL	ANALYTE	SB-1B2	SB-1BC	SB-2A2	SB-2AC	SB-5B2	SB-5BC	DU-5BC	SB-3A2
40	Aluminum	19900	6750	10,000	6310	8290	7180	9910	3820
12	Antimony								
2	Arsenic	5.3	4.8	23.6	13.3	4.4	3.8	4.6	3.1
40	Barium	326	188	10,200	6340	97.1	84.7	150	51.7
1	Beryllium				.48				
1	Cadmium	3.6	4.6	5.6	2.9	1.4	1.7	2.0	
1000	Calcium	68,700	52,200	70,800	46,600	16,400	74,300	30,700	6,730
2	Chromium	112	217	37.2	28.7	14.8	14	25.1	21.5
10	Cobalt	29.9		12.9					
5	Copper	60.9	142	1870	162	19.4	28.4	24.6	23.1
20	Iron	34,600	38,800	18,300	14,400	13,400	17,600	16,300	7,690
1	*Lead	5.47	3.180	634	225	51.2	60.9	82.9	12.5
1000	Magnesium	5260	18,000	13,800	17,400	4520	10,600	7740	1210
3	Manganese	651	406	364	309	410	395	577	95
0.2	Mercury	443	111	4.4	15.5	.68	.52		.92
8	Nickel	435	210	98.3	55.9	14.8		18.1	
1000	Potassium								
1	Selenium								
2	Silver								
1000	Sodium	5260		1420					
2	Thallium								
10	Vanadium	30.1	13.7	33.4	17.3	13.9	18.5	14.5	
4	Zinc	769	394	1140	488	129	93.6	225	362
2	Cyanide			82.1	25.5			4.8	

CRDL = Contract Required Detection Limit \*Action Level Exists SEE NARRATIVE FOR CODE DEFINITIONS



TABLE B-2

DATA SUMMARY FORM: I N O R G A N I C S

Site Name: 3163 BUFFALO AVENUE

SOIL SAMPLES  
(mg/Kg)(ppm)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

+Due to dilution, sample quantitation limit is affected.  
See dilution table for specifics.

CRDL	Sample No. Dilution Factor % Solids Location	SB-3AC	SB-6AZ	SB-6AC	SB-7A2	SB-7AC	SB-4AZ	SE-01	SB-D
40	Aluminum	4810	8480	5110	2080	4960	9440	8230	30100
12	Antimony								
2	Arsenic	3.0	6.6	6.3	4.9	3.7	2.5	23.4	13.6
40	Barium	81.4	1940	2200	513	104	96.7	21100	623
1	Beryllium			.47					
1	Cadmium	1.9	11.8	4.5	74.7	3.2	1.8	38.6	12.7
1000	Calcium	24700	93800	55900	34200	25300	53700	35700	61100
2	Chromium	30.0	44.8	30.6	71.9	186	14.2	92.3	19.0
10	Cobalt							28.5	
5	Copper	31.9	643	333	643	116	29.8	8860	339
20	Iron	12900	21500	15100	120000	20200	14100	51800	31200
1	*Lead	185	621	386	1080	286	24.2	18700	1360
1000	Magnesium	7540	11300	13800	713	1690	11800	18200	10400
3	Manganese	285	307	248		266	244	654	629
0.2	Mercury	2.9	0.44	38.6	.26	.36	6.4	5.8	23.3
8	Nickel		79.4	36.2	36.6	13.7	13.7	242	37.2
1000	Potassium								4590
1	Selenium							6.2	
2	Silver							5.4	
1000	Sodium								
2	Thallium		.52	.49					
10	Vanadium	12.1	26.6	15.9	42.7	13.9	19.4	54.9	53.3
4	Zinc	267	1690	1070	1990	952	186	34700	7900
2	Cyanide		19.4	3.5			3.9		

CRDL = Contract Required Detection Limit

\*Action Level Exists

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TABLE B-2  
DATA SUMMARY FORM: I N O R G A N I C S

Site Name: 3163 BUFFALO AVENUE SOIL SAMPLES  
(mg/Kg)(ppm)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_  
+Due to dilution, sample quantitation limit is affected.  
See dilution table for specifics.

CRDL	Sample No. Dilution Factor % Solids Location	SB-C	SB-A	SB-B	SB-J	SB-JD	SB-I	SB-F	SB-H	SB-G
40	Aluminum	8930	9500	21500	7140	10700	9970	16300	8980	13100
12	Antimony								75.3	
2	Arsenic	26.8	13.1	104	14.8	20.8	14.7	13.9	39.1	19.4
40	Barium	31.5	5230	19200	370	545	848	869	428	958
1	Beryllium									
1	Cadmium	14.4	17.2	8.7	3.7	7.3	55.0	21.0	74.8	12.6
1000	Calcium	23,800	80500	184000	49600	37300	48700	79600	23400	143000
2	Chromium	33.6	91.8	1530	182	356	81.4	54.6	80.4	200
10	Cobalt		18.4	61.6						
5	Copper	285	1020	352	248	510	877	518	8180	468
20	Iron	34,600	94100	56300	20000	26,400	40200	35500	69900	45600
1	*Lead	1110	1920	752	1180	2790	7240	779	31,000	1520
1000	Magnesium	5130	21400	49600	16600	13,600	17300	25400	11700	69700
3	Manganese	376	770	1480	298	305	591	1080	495	903
0.2	Mercury	19.2	23.4	22.5	2.9	5.7	2.0	1.4	3.9	1.6
8	Nickel	27.4	108	128	31.3	40.7	41.4	42.7	54.4	46.7
1000	Potassium			4900			1070	1560		
1	Selenium								13.4	
2	Silver								16.4	
1000	Sodium									
2	Thallium									
10	Vanadium	33.7	34.5	47.6	22.7	32.1	24.3	38.0	31.4	32.6
4	Zinc	5890	10200	1820	4110	12100	26500	18200	56,900	76,000
2	Cyanide		5.6	1.8	3.2	6.1	1.5			

CRDL = Contract Required Detection Limit \*Action Level Exists SEE NARRATIVE FOR CODE DEFINITIONS



DATA SUMMARY FORM: VOLATILES

Site Name: 3163 BUFFALO AVENUE

NAPL SAMPLES  
(ug/L)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor)

CRQL	COMPOUND	Sample No. Dilution Factor	Location	5B-1B NA	MW-3B														
10	Chloromethane																		
10	Bromomethane																		
10	*Vinyl Chloride																		
10	Chloroethane																		
5	*Methylene Chloride																		
10	Acetone																		
5	Carbon Disulfide																		
5	*1,1-Dichloroethene																		
5	1,1-Dichloroethane																		
5	*Total-1,2-Dichloroethene																		
5	Chloroform																		
5	*1,2-Dichloroethane																		
10	*2-Butanone																		
5	*1,1,1-Trichloroethane																		
5	*Carbon Tetrachloride																		
10	Vinyl Acetate																		
5	Bromodichloromethane																		

SEE NARRATIVE FOR CODE DEFINITIONS

\*Action Level Exists

CRDL = Contract Required Detection Limit

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\*E = EXCEEDS QUANTIFIABLE RANGE, ESTIMATED

J = BELOW QUANTIFIABLE RANGE, ESTIMATED

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DATA SUMMARY FORM: VOLATILES 2

Site Name: 3163 BUFFALO AVENUE

NAPL SAMPLES (ug/L)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limits (CRQL \* Dilution Factor)

CRQL	COMPOUND	Sample No. Dilution Factor Location	SB-1B-NA	MW-2B														
5	*1,2-Dichloropropane		56354	69384														
5	Cis-1,3-Dichloropropene																	
5	Trichloroethene																	
5	Dibromochloromethane																	
5	1,1,2-Trichloroethane																	
5	*Benzene		160,000	120,000														
5	Trans-1,3-Dichloropropene																	
5	Bromolorm																	
10	4-Methyl-2-pentanone																	
10	2-Hexanone																	
5	*Tetrachloroethene			210,000														
5	1,1,2,2-Tetrachloroethane																	
5	*Toluene			67,000														
5	*Chlorobenzene		290,000	110,000														
5	*Ethylbenzene			16,000	J*													
5	*Styrene																	
5	*Total Xylenes			36,000	J*													

CRDL = Contract Required Detection Limit \*Action Level Exists SEE NARRATIVE FOR CODE DEFINITIONS

J = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
 J = BELOW QUANTIFIABLE RANGE; ESTIMATED

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DATA SUMMARY FORM: B N A S 1

Site Name: 3163 BUFFALO AVENUE

NAPL SAMPLES (ug/L)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limits (CRQL \* Dilution Factor)

CRQL	COMPOUND	Sample No. Dilution Factor Location	SB-1B-NA	MW-2B						
10	Phenol		2900							
10	bis(2-Chloroethyl)ether									
10	2-Chlorophenol		470	J*						
10	*1,3-Dichlorobenzene		11,000		45,200					
10	*1,4-Dichlorobenzene		37,000	E*	188,000					
10	Benzyl Alcohol									
10	1,2-Dichlorobenzene		28,000		287,000					
10	2-Methylphenol									
10	bis(2-Chloroisopropyl)ether									
10	4-Methylphenol									
10	N-Nitroso-di-n-propylamine									
10	Hexachloroethane									
10	Nitrobenzene									
10	Isophorone									
10	2-Nitrophenol									
10	2,4-Dimethylphenol									
50	Benzoic Acid									
10	bis(2-Chloroethoxy)methane									
10	2,4-Dichlorophenol									
10	1,2,4-Trichlorobenzene		14,000		123,000					
10	Naphthalene		83	J*						
10	4-Chloroaniline									

CRDL = Contract Required Detection Limit \*Action Level Exists SEE NARRATIVE FOR CODE DEFINITIONS

\* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED

J = BELOW QUANTIFIABLE RANGE, ESTIMATED

Site Name: 3163 BUFFALO AVENUE

NAPL SAMPLES (ug/L)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limits (CROL \* Dilution Factor)

CROL	COMPOUND	Sample No. Dilution Factor Location	SB-10-NA 56354	MW-2-B 69384						
10	Hexachlorobutadiene		72	J*						
10	4-Chloro-3-methylphenol									
10	2-Methylnaphthalene									
10	Hexachlorocyclopentadiene									
10	2,4,6-Trichlorophenol									
50	2,4,5-Trichlorophenol									
10	2-Chloronaphthalene									
50	2-Nitroaniline									
10	Dimethylphthalate									
10	Acenaphthylene									
10	2,6-Dinitrotoluene									
50	3-Nitroaniline									
10	Acenaphthene									
50	2,4-Dinitrophenol									
50	4-Nitrophenol									
10	Dibenzofuran									
10	2,4-Dinitrotoluene									
10	Diethylphthalate									
10	4-Chlorophenyl-phenylether									
10	Fluorene									
50	4-Nitroaniline									
50	4,6-Dinitro-2-methylphenol									

CROL = Contract Required Detection Limit \*Action Level Exists SEE NARRATIVE FOR CODE DEFINITIONS

\* E = EXCEEDS QUANTIFIABLE RANGE; ESTIMATED

J = BELOW QUANTIFIABLE RANGE; ESTIMATED

Site Name: 3163 BUFFALO AVENUE

NAPL SAMPLES  
(ug/L)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CROL \* Dilution Factor)

CRDL	COMPOUND	Sample No. Dilution Factor Location	SB-1B-NA	MW-2B																
10	N-Nitrosodiphenylamine																			
10	4-Bromophenyl-phenylether																			
10	*Hexachlorobenzene																			
50	*Pentachlorophenol																			
10	Phenanthrene		90	J*																
10	Anthracene																			
10	Di-n-butylphthalate																			
10	Fluoranthene		94	J*																
10	Pyrene		76	J*																
10	Butylbenzylphthalate																			
20	3,3-Dichlorobenzidine																			
10	Benzo(a)anthracene																			
10	Chrysene																			
10	bis(2-Ethylhexyl)phthalate																			
10	Di-n-octylphthalate																			
10	Benzo(b)fluoranthene		76	J*																
10	Benzo(k)fluoranthene																			
10	Benzo(a)pyrene																			
10	Indeno(1,2,3-cd)pyrene																			
10	Dibenz(a,h)anthracene																			
10	Benzo(g,h,i)perylene																			

CRDL = Contract Required Detection Limit \*Action Level Exists SEE NARRATIVE FOR CODE DEFINITIONS

\* E EXCEEDS QUANTIFIABLE RANGE; ESTIMATED  
 J: BELOW QUANTIFIABLE RANGE; ESTIMATED

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DATA SUMMARY FORM: PESTICIDES AND PCB'S

Site Name: 3163 BUFFALO AVENUE

NAPL SAMPLES  
(ug/L)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

To calculate sample quantitation limit:  
(CRQL \* Dilution Factor)

CRQL	Sample No. Dilution Factor Location	COMPOUND	SB-1B-NA	MW-2B															
0.05		alpha-BHC																	
0.05		beta-BHC																	
0.05		delta-BHC																	
0.05		*Gamma-BHC (Lindane)																	
0.05		*Heptachlor																	
0.05		Aldrin																	
0.05		Heptachlor Epoxide																	
0.05		Endosulfan I																	
0.10		Dieldrin																	
0.10		4,4'-DDE																	
0.10		*Endrin																	
0.10		Endosulfan II																	
0.10		4,4'-DDD																	
0.10		Endosulfan Sulfate																	
0.10		4,4'-DDT																	
0.5		*Methoxychlor																	
0.10		Endrin ketone																	
0.5		*Alpha-Chlordane																	
0.5		*Gamma-Chlordane																	
1.0		*Toxaphene																	
0.5		*Aroclor-1016																	
0.5		*Aroclor-1221																	
0.5		*Aroclor-1232																	
0.5		*Aroclor-1242																	
0.5		*Aroclor-1248																	
1.0		*Aroclor-1254																	
1.0		*Aroclor-1260																	

recycled paper

recycled paper

CRDL = Contract Required Detection Limit \*Action Level Exists  
 \*BEFORE QUANTIFIABLE RANGE, ESTIMATED  
 \*BELOW QUANTIFIABLE RANGE, ESTIMATED  
 SEE NARRATIVE FOR CODE DEFINITIONS  
 revised 12/88  
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TABLE B-3  
DATA SUMMARY FORM: INORGANICS

Site Name: 3163 BUFFALO AVENUE

NAPL SAMPLES  
(ug/L)

Case #: \_\_\_\_\_ Sampling Date(s): \_\_\_\_\_

\*Due to dilution, sample quantitation limit is affected.  
See dilution table for specifics.

CRDL	ANALYTE	Sample No. Dilution Factor	Location	Sampling Date(s)	Result	Notes
200	Aluminum					
60	Antimony					
10	*Arsenic					
200	Barium					
5	Beryllium					
5	*Cadmium					
5000	Calcium					
10	*Chromium					
50	Cobalt					
25	Copper					
100	Iron					
5	*Lead					
5000	Magnesium					
15	Manganese					
0.2	Mercury					
40	*Nickel					
5000	Potassium					
5	Selenium					
10	Silver					
5000	Sodium					
10	Thallium					
50	Vanadium					
20	Zinc					
10	*Cyanide					

SB-1B-NA MW-2B  
Not run for metals  
NR  
↓

CRDL = Contract Required Detection Limit      \*Action Level Exists      SEE NARRATIVE FOR CODE DEFINITIONS

Table B-4  
Tentatively Identified Compounds (TIC's)  
Soil Samples

Compound	R.F. Value (min)	SB-4A2	SB-E	SB-D	SB-C	SB-A	SB-B	SB-J	SB-JD	SB-F	SB-H	SB-G
<u>Volatiles:</u>												
Benzene, trichloro isomer	28.98 - 29.57	1700 J										
Benzene, trimethyl	22.79	4.8 J										
<u>SEMIVOLATILES:</u>												
Anthracene, methyl					240 J							
Benzene, Bis-methyl	11.22 - 11.34											
Benzene, 1,3,9,6 -	13.96 -		65,000 J		240 J							
Tetrachloro, isomer	15.13 - 15.34				240 J			13,000 J		160,000	340,000 J	
Benzene, 1,3,9,6 -	15.96											
Tetrachloro, isomer	16.19		29,000 J		690 J			110,000 J	73,000 J	280,000	730,000 J	24,000 J
Benzene, 1,2,4,3 -	12.93 - 13.32		270,000 J	83,000 J	830 J	49,000 J	82,000 J	350,000 J	320,000 J	320,000 J	1,400,000 J	130,000 J
Benzene, trichloro ethyl	16.59											
Benzene 1,2,4 trichloro 3 methyl	15.29											
Benzene, 1,2,4 trichloro, isomer	16.49				1700 J							
1,3 Benzene, 9,6 -	15.44											
fluoranthene	15.44											
2,3,4,5,6 Penta - chloro ethyl	22.09 - 22.14											
phenyl 2-(4H-pyridyl)	25.06											

Tentatively Identified Compounds

Soil Samples

Compound	RI	SB 1B2	SB 1BC	SB 1BA2	SB 2AC	SB 5B2	SB 5BC	PU-5BC	SB 3A2	SB 3AC	SB 6A2	SB 6AC	SB 7A2	SB 7AC	SB 7AC	
Valentiles																
enzane, tetrachloro	28.916	4800J														
enzane, trichloro	28.98-															
enzane, dichloro	29.57															
enzane, trimethyl	22.79															
benzocyclobutene																
anthracene, dimethyl																
enzane,	11.22-															
benzocyclobutene	11.34															
enzane, chloro	8.42	2400J														
methyl isomer																
enzane, chloro	8.59	1100J														
nethyl isomer																
benzene,	12.32	2500J														
chloromethyl																
enzane, tetra	13.96															
chloro isomer																
enzane, tetra	15.13-															
chloro isomer	15.34															
enzane, tetra	15.96-															
chloro isomer	16.19															
enzane, trichloro	12.98-															
isomer	12.32															
enzane, trichloro	16.59															
ethyl																
enzane, 1,2,4																
trichloro 3-methyl	15.29															
benzocyclobutene																
enzane,	16.49															
trichloro																
3-Dibenzocyclobutene	15.44															
enzane,	22.09-															
1,2,4,5,6-Penta-	22.14															
chloro ethylene																
isomer																
(4-Hydroxycyclopent-1-en-3-yl)acetylene	29.06															
benzocyclobutene	23.84															
trichloro (C <sub>10</sub> H <sub>10</sub> Cl <sub>3</sub> )	27.94															
benzocyclobutene,																
trichloro isomer																

Table B-4  
Tentatively Identified Compounds (TIC's)  
Water Samples

Compound	R.F. Value (min)	GW-1A	GW-1B	GW-2B	GW-2A	GW-3A	GW-6A	GW-6B	GW-3B	GW-5B	GW-5B D	GW-4A	GW-4B	GW-4B D	
Nonathylene	27.15	64993	64994	65191	65192	6519	65194	65195	65196	65392	65393	65394	65395	65396	
Bromochlorobenzene	28.05														
Tetrahydrothiophene	16.50							80J						170J	
SEMIVOLATILES:															
Chloromethane	8.95		33J			120J									
Benzene, iso	9.10		23J			80J									
Chloromethane	20.18		74J												
Benzene, iso	16.93	83J	500J	1800J	380J										
Chloromethane	17.78	92J	520J	2500J	430J										
Benzene, iso	17.82														
Trichlorobenzene															
Chloromethane	13.40							40J							
Chloromethane	14.73														
Chloromethane	14.80	510J	1400J	9800J	1100J				760J	93J	76J	380J	110J	110J	
Chloromethane	14.98	160J	73J		200J										
Chloromethane	14.59				380J										
Chloromethane	21.64				270J										
Chloromethane	21.81														
Chloromethane	17.41	180J													
Chloromethane	14.39				100J										
Chloromethane	4.87			1400J											
Chloromethane	16.46												1100J		
Chloromethane	25.64														
1-octene tetrahydrothiophene															

Table B-4  
Tentatively Identified Compounds (TIC)  
Water Samples

Compound	RF Value (min)	GW-7A	GW-5A	GW-5C	GW-5D	GW-5F
Bromochlorobenzene, is.	27.15	65398	65399	65738	65739	
Bromochlorobenzene, is.	28.05	70 J				
Tetrahydrothiophene	15.96 16.50	100 J		350 J	650 J	
SEMI-AROMATIC:						
Chloromethylbenzene, is.	8.95					
Chloromethylbenzene, is.	9.10					
Pentachlorobenzene	20.18					
Tetrahydrobenzene	16.93	220 J				
Tetrahydrobenzene, is.	16.96					
Tetrahydrobenzene, is.	17.78	340 J		100 J		
Tetrahydrobenzene, is.	17.82					
AROMATIC:						
Fluorobenzene, is.	13.40					
Fluorobenzene, is.	14.73	2300 J		710 J	410 J	
Chlorobenzene, is.	14.80					
Chlorobenzene, is.	14.58					
Chlorobenzene, is.	14.59					
Chlorotoluene	21.65					
Chlorotoluene	21.81					
Chlorotoluene	17.41					
Chlorotoluene	14.39					
Tetrahydrothiophene	4.87			880 J	2600 J	
Tetrahydrothiophene	16.46					
Tetrahydrothiophene	25.64		15 J			
Tetrahydrothiophene	12.99			330 J		

Table B-4  
Tentatively Identified Compounds  
Sediment Samples

Draft

Compound	RI Value (min)	SE-1 61318	SE-2	SE-1 DL 61318 DL
<u>VOLATILES:</u>				
Benzene, chloromethyl		960J		
<u>SEMIVOLATILES:</u>				
Benzene, pentachloro	19.23 - 19.48	39,000J		92,000J
Benzene, dichloro methyl	12.22	10,000J		
Benzene, tetrachloro isomer	15.88 - 16.32	17,000J	2000J	1,000,000J
Benzene, tetrachloro isomer	16.71 - 17.18	33,000J	3400J	1,200,000J
Benzene, trichloro isomer	17.40	10,000J		
Benzene, trichloro isomer	13.70 - 14.20	110,000J	4000J	880,000J
Benzene, trichloro chloromethyl isomer	18.47	33,000		
Benzene, trichloro chloromethyl isomer	19.20	17,000		
Benzene, trichloromethyl is.	15.45 - 15.67	36,000J		92,000J
Benzene, trichloromethyl is.	15.90	39,000J		
1,1 Biphenyl dichloro	20.93	8500J		
1,1 Biphenyl dichloro	22.12	6900J		
1 Biphenyl trichloro	24.47	4900J		
3,4,5,6 Pentachloro toluene	21.81	9,200J		



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## LABORATORY REPORT

FOR

SOLVENT CHEMICAL - 3163 BUFFALO AVENUE

JOB #: 9000.352RE: JD-1030SAMPLE DATE: 02/06/90PO#:DATE RECEIVED: 02/06/90SAMPLED BY: E & E INC.SAMPLE TYPE: WaterDELIVERED BY: E & E INC.E & E Lab # 90:

64993

64994

64995

Customer #:BA-GW-  
1ABA-GW-  
1BBA-GW-  
BL-1

Results in: MG/L

Total Organic Carbon            400            340            &lt;1.0

Total Organic Halogens        349            240            &lt;1.0

Analytical References: "Test Methods for Evaluating Solid  
Waste, Physical/Chemical Methods,"  
SW-846, Third Edition, U.S.EPA, 1986.





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## LABORATORY REPORT

FOR

SOLVENT CHEMICAL - 3163 BUFFALO AVENUE

JOB #: 9000.366RE: JD-1030SAMPLE DATE: 02/07/90PO#:DATE RECEIVED: 02/07/90SAMPLED BY: E & E INC.SAMPLE TYPE: WaterDELIVERED BY: E & E INC.E & E Lab # 90:

65191

65192

65193

Customer #:BA-GW-  
2BBA-GW-  
2ABA-GW-  
3A

Results in: MG/L

Total Organic Carbon

4.0

180

78

Total Organic Halogens

350

230

0.7

Analytical References:

"Test Methods for Evaluating Solid  
Waste, Physical/Chemical Methods,"  
SW-846, Third Edition, U.S.EPA, 1986.



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## LABORATORY REPORT

FOR

SOLVENT CHEMICAL - 3163 BUFFALO AVENUE

JOB #: 9000.384RE: JD-1030SAMPLE DATE: 02/08/90PO#:DATE RECEIVED: 02/08/90SAMPLED BY: E & E INC.SAMPLE TYPE: WaterDELIVERED BY: E & E INC.E & E Lab # 90:

65395

65396

65397

Customer #:BA-GW-  
4BBA-GW-  
4B  
DUPBA-GW-  
BL2

Results in: MG/L

Total Organic Carbon

29

50

&lt;1.0

Total Organic Halogens

240

580

0.023

Analytical References:

"Test Methods for Evaluating Solid Waste, Physical/Chemical Methods," SW-846, Third Edition, U.S.EPA, 1986.



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LABORATORY REPORT

FOR

SOLVENT CHEMICAL - 3163 BUFFALO AVENUE

JOB #: 9000.384

RE: JD-1030

SAMPLE DATE: 02/08/90

PO#:

DATE RECEIVED: 02/08/90

SAMPLED BY: E & E INC.

SAMPLE TYPE: Water

DELIVERED BY: E & E INC.

---

<u>E &amp; E Lab # 90:</u>	65392	65393	65394
<u>Customer #:</u>	BA-GW-5B	BA-GW-5B DUP	BA-GW-4A

---

Results in: MG/L

Total Organic Carbon	42	110	140
Total Organic Halogens	170	120	1400

Analytical References:

"Test Methods for Evaluating Solid Waste, Physical/Chemical Methods," SW-846, Third Edition, U.S.EPA, 1986.



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LABORATORY REPORT

FOR

SOLVENT CHEMICAL - 3163 BUFFALO AVENUE

JOB #: 9000.384

RE: JD-1030

SAMPLE DATE: 02/08/90

PO#:

DATE RECEIVED: 02/08/90

SAMPLED BY: E & E INC.

SAMPLE TYPE: Water

DELIVERED BY: E & E INC.

---

E & E Lab # 90:

65398

65399

Customer #:

BA-GW-  
7A

BA-GW-  
5A

---

Results in: MG/L

Total Organic Carbon

280

360

Total Organic Halogens

1,800

1,500

Analytical References:

"Test Methods for Evaluating Solid Waste, Physical/Chemical Methods," SW-846, Third Edition, U.S.EPA, 1986.



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LABORATORY REPORT

FOR

SOLVENT CHEMICAL - 3163 BUFFALO AVENUE

JOB #: 9000.402

RE: JD-1030

SAMPLE DATE: 02/09/90

PO#:

DATE RECEIVED: 02/09/90

SAMPLED BY: E & E INC.

SAMPLE TYPE: Water

DELIVERED BY: E & E INC.

---

E & E Lab # 90:

65738

65739

Customer #:

BA-GW-  
5C

BA-GW-  
5C-D

---

Results in: MG/L

Total Organic Carbon

70

30

Total Organic Halogens

2,100

1,300

Analytical References:

"Test Methods for Evaluating Solid Waste, Physical/Chemical Methods," SW-846, Third Edition, U.S.EPA, 1986.



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## LABORATORY REPORT

FOR

SOLVENT CHEMICAL - 3163 BUFFALO AVENUE

JOB #: 9000.425RE: JD-1030SAMPLE DATE: 02/13/90PO#:DATE RECEIVED: 02/13/90SAMPLED BY: E & E INC.SAMPLE TYPE: WaterDELIVERED BY: E & E INC.

---

E & E Lab # 90: 66069Customer #: BA-GW-  
5F

---

Results in: MG/L

Total Organic Carbon 36

Total Organic Halogens 1,100

Analytical References: "Test Methods for Evaluating Solid Waste, Physical/Chemical Methods," SW-846, Third Edition, U.S.EPA, 1986.



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## LABORATORY REPORT

FOR

SOLVENT CHEMICAL - 3163 BUFFALO AVENUE

JOB #: 9000.366RE: JD-1030SAMPLE DATE: 02/07/90PO#:DATE RECEIVED: 02/07/90SAMPLED BY: E & E INC.SAMPLE TYPE: WaterDELIVERED BY: E & E INC.E & E Lab # 90:

65194

65195

65196

Customer #:BA-GW-  
6ABA-GW-  
6BBA-GW-  
3B

Results in: MG/L

Total Organic Carbon

17

50

42

Total Organic Halogens

0.3

0.74

320

Analytical References:

"Test Methods for Evaluating Solid Waste, Physical/Chemical Methods,"  
SW-846, Third Edition, U.S.EPA, 1986.

Table B-1  
 COMPARISON OF TOTAL ORGANICS  
 TO TOTAL ORGANIC HALOGENS

Monitoring Well	TOX ( $\mu\text{g}/\text{l}$ )	Total Organics ( $\mu\text{g}/\text{l}$ )	TOX/Total Organics*
1A	349,000	559,622	.624
2A	230,000	335,667	.685
3A	700	1,520	.460
4A	1,400,000	47,581	29.4
5A	1,500,000	1,164	1,288.
6A	300	148	2.03
7A	1,800,000	118,651	15.2
1B	240,000	344,357	.697
2B	350,000	322,393	1.09
3B	320,000	37,189	8.60
4B	240,000	8,037	29.86
4B (Dup)	580,000	7,878	73.62
5B	170,000	4,542	37.42
5B (Dup)	120,000	4,920	24.39
6B	740	2,525	.293
5C	2,100,000	18,404	114.
5CD	1,300,000	21,898	59.37
5F	1,100,000	3,513	313.

[IL]JD1900:D3048/4085/34

\*Total organics: total detected TCL compounds plus detected TICs.



APPENDIX C

SLUG TEST DATA

Table C-1

**SLUG TESTING SUMMARY TABLE**  
**3163 BUFFALO AVENUE**  
**TESTING DATES: MARCH 16, 1990 - MARCH 21, 1990**

Well	Aq. Type	Screen (ft)	Water Level (ft)	Bouwer-Rice Method	
				Hydr. Cond. <sub>2</sub> (gal/day/ft <sup>2</sup> )	Transmissivity (gal/day/ft <sup>2</sup> )
<b>Shallow Wells</b>					
MW1A	Unconfined	4.0 - 6.0	5.16	1.4	2.81
MW2A	Unconfined	4.0 - 8.2	7.06	1.6	8.05
MW3A	Unconfined	4.5 - 8.5	6.12	1.2	7.12
MW4A	Unconfined	4.0 - 8.0	4.2	0.4	1.97
MW5A	Unconfined	6.0 - 12.5	7.87	2.5	18.96
MW6A	Unconfined	4.0 - 9.6	5.93	1.26	7.08
MW7A	Unconfined	3.5 - 7.5	4.49	18.63	93.17
<b>Intermediate Wells</b>					
MW1B	Unconfined	6.3 - 17.15	11.57	38.45	221.9
MW2B	Unconfined	8.2 - 20.0	9.24	3.75	45.02
MW3B	Unconfined	9.6 - 21.2	16.05	0.8	9.34
MW4B	Unconfined	8.0 - 22.5	10.62	-- NOT ENOUGH DATA --	
MW5B	Unconfined	12.5 - 21.0	18.04	1.04	8.82
MW6B	Unconfined	9.6 - 20.0	9.6	8.72	90.69
<b>Deep Wells</b>					
MW5C	Unconfined	26.5 - 36.5	19.32	19.17	191.72
MW5CD	Unconfined	39.5 - 51.5	23.83	24.77	297.24
MW5F	Unconfined	77.0 - 98.0	17.92	11.31	237.51

02[IL]JD1900:D3048/1262/22

**Average Transmissivities:**

Shallow Wells: 19.88 gpd/ft (heavily weighted by T value for SC7A)  
Intermediate Wells: 75.15 gpd/ft (heavily weighted by T value for SC1B)  
Deep Wells: 242.16 gpd/ft

----- SOLVEX -----

----- PAGE 1 -----

## DATA SET: SOLVEX

CLIENT: SOLVENT CHEMICAL	DATE: 2/13/90
LOCATION: BUFFALO AVE.	WELL NO.: SC1A
COUNTY: ERIE	WELL DEPTH: 6.00 ft
PROJECT: Bail-down Test	WATER TABLE: 5.150 ft
AQUIFER: Unknown	THICKNESS: 2.00 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.167 ft
SCREEN TOP: 4.000 ft	SCREEN BASE: 6.00 ft
INITIAL HEAD: 0.680 ft	TRANS. RATIO: 1.0000

## MODEL PARAMETERS:

TRANSMISSIVITY: .000261 square ft/min

CONDUCTIVITY: .000130 ft/min

MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer &amp; Rice)

No.	TIME (mins)	Head, H (ft) DATA
1	1.000E-04	0.730
2	0.100	0.700
3	0.700	0.680
4	0.800	0.650
5	0.950	0.640
6	1.15	0.620
7	1.60	0.600
8	1.75	0.590
9	2.10	0.560
10	2.80	0.530
11	3.10	0.500
12	3.80	0.480
13	4.10	0.460
14	4.80	0.430
15	5.10	0.410
16	5.80	0.400
17	6.10	0.380
18	6.80	0.350
19	7.10	0.340
20	7.80	0.330
21	8.10	0.260
22	8.80	0.290
23	9.10	0.280

C-3

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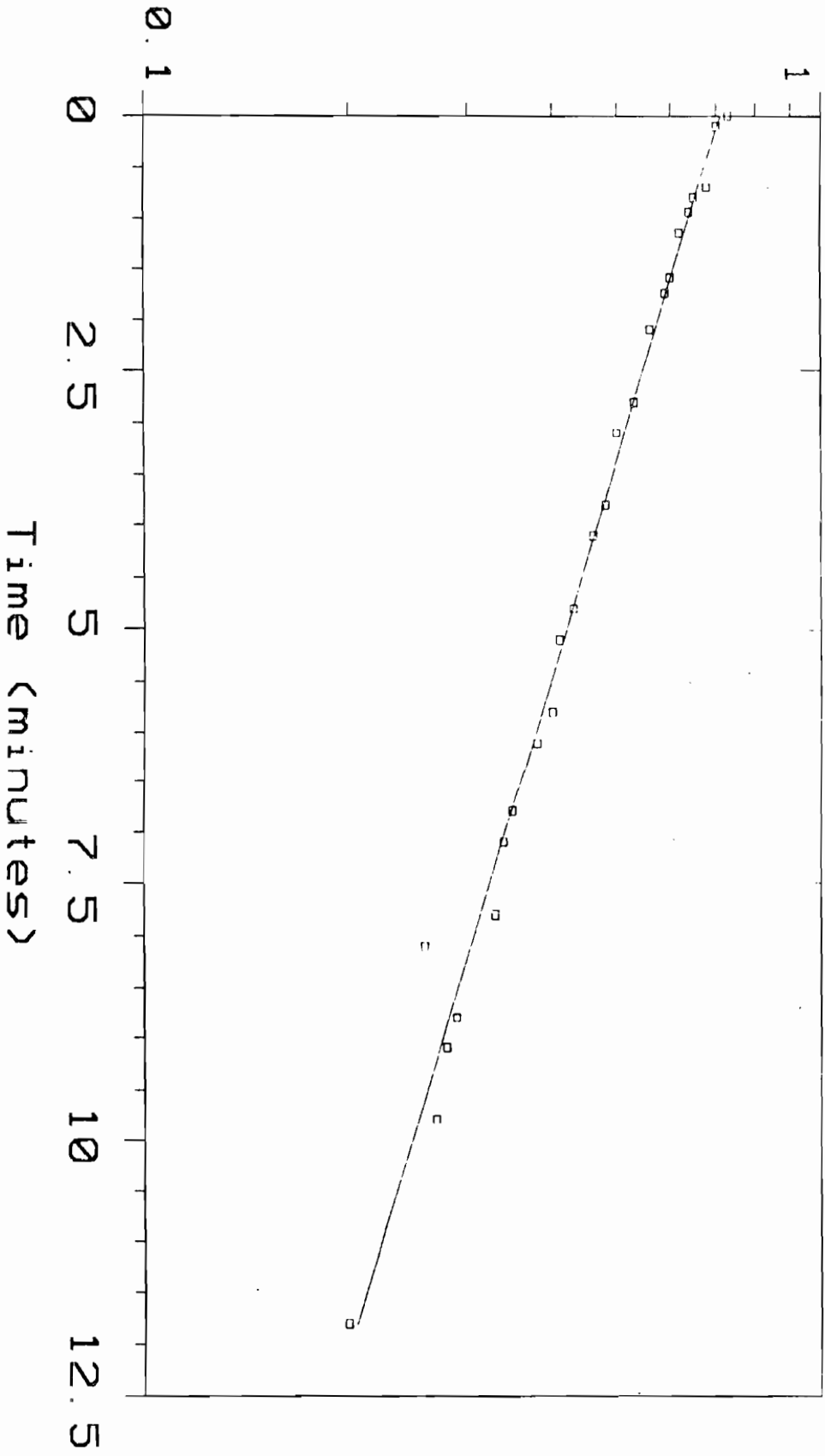
----- SOLVEX -----

----- PAGE 2 -----

No.	TIME (mins)	Head, H (ft) DATA
24	9.80	0.270
25	11.80	0.200

CURRENT RESOLUTION MARIIX NOT AVAILABLE

Head (feet)



C-5

SC1B

PAGE 1

## DATA SET: SC1B

CLIENT: SOLVENT CHEMICAL	DATE: 3/11/93
LOCATION: BUFFALO AVE.	WELL NO.: SC1B
COUNTY: ERIE	WELL DEPTH: 17.50 ft
PROJECT: Slug test	WATER TABLE: 11.570 ft
AQUIFER: Unknown	THICKNESS: 3.74 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.157 ft
SCREEN TOP: 6.000 ft	SCREEN BASE: 17.50 ft
INITIAL HEAD: 6.760 ft	TRANS. RATIO: 1.0000

## MODEL PARAMETERS:

TRANSMISSIVITY: .0206 square ft/min

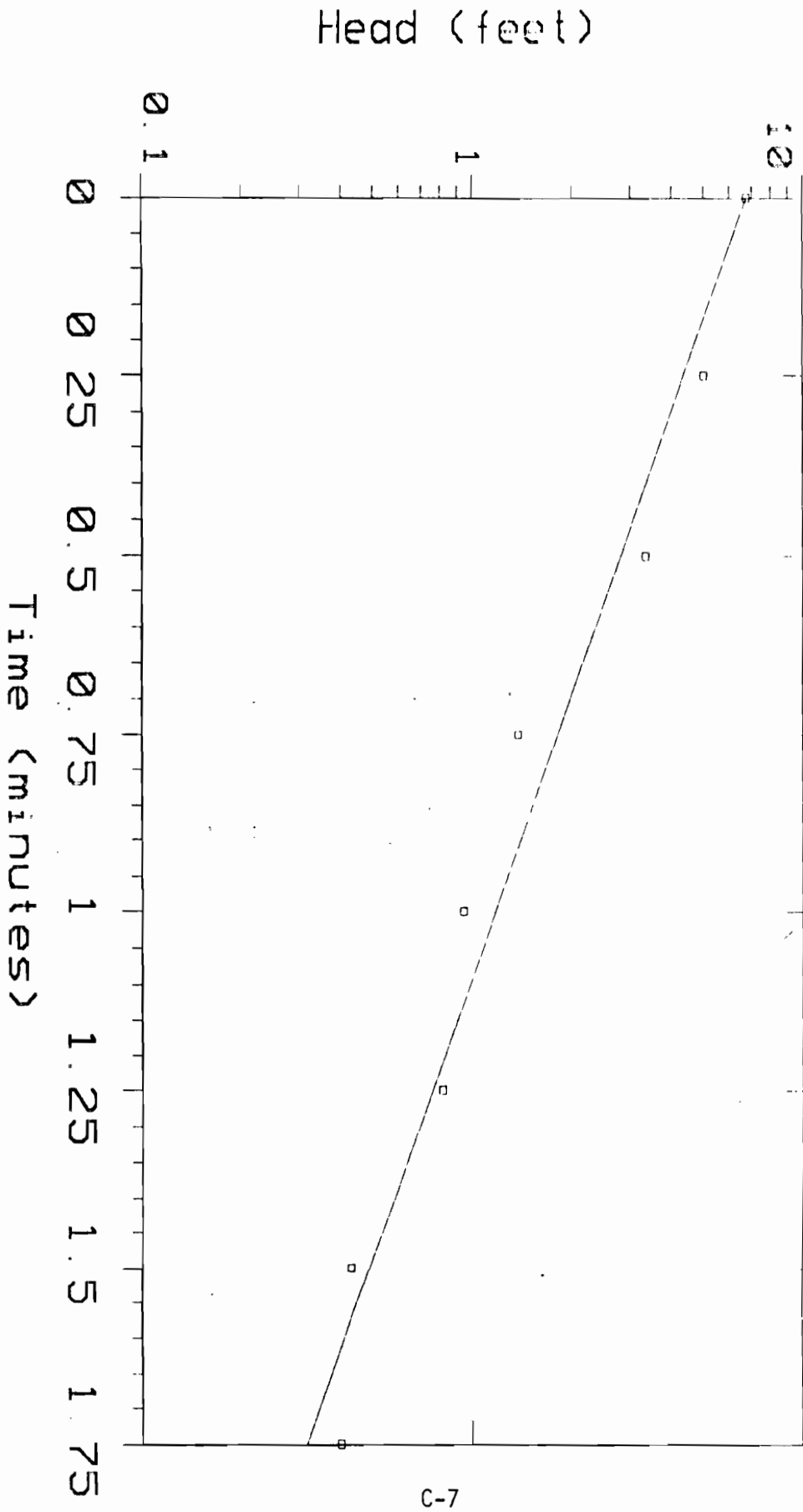
CONDUCTIVITY: .00357 ft/min

MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer &amp; Rice)

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
1	1.000E-04	6.76	6.73	0.332
2	0.250	5.03	4.35	13.47
3	0.500	3.34	2.81	15.84
4	0.750	1.38	1.81	-31.55
5	1.00	0.940	1.17	-24.73
6	1.25	0.810	0.757	6.51
7	1.50	0.430	0.489	-13.73
8	1.75	0.400	0.315	21.03

CURRENT RESOLUTION MATRIX NOT AVAILABLE

# SC1B



## DATA SET: SC2A

CLIENT: SOLVENT CHEMICAL	DATE: 3/13/90
LOCATION: BUFFALO AVE.	WELL NO.: SC2A
COUNTY: ERIE	WELL DEPTH: 8.00 ft
PROJECT: Slug Test	WATER TABLE: 7.060 ft
AQUIFER: Unknown	THICKNESS: 5.00 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.167 ft
SCREEN TOP: 4.000 ft	SCREEN BASE: 8.10 ft
INITIAL HEAD: 1.450 ft	TRANS. RATIO: 1.0000

## MODEL PARAMETERS:

TRANSMISSIVITY: .000748 square ft/min

CONDUCTIVITY: .000149 ft/min

MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer & Rice)

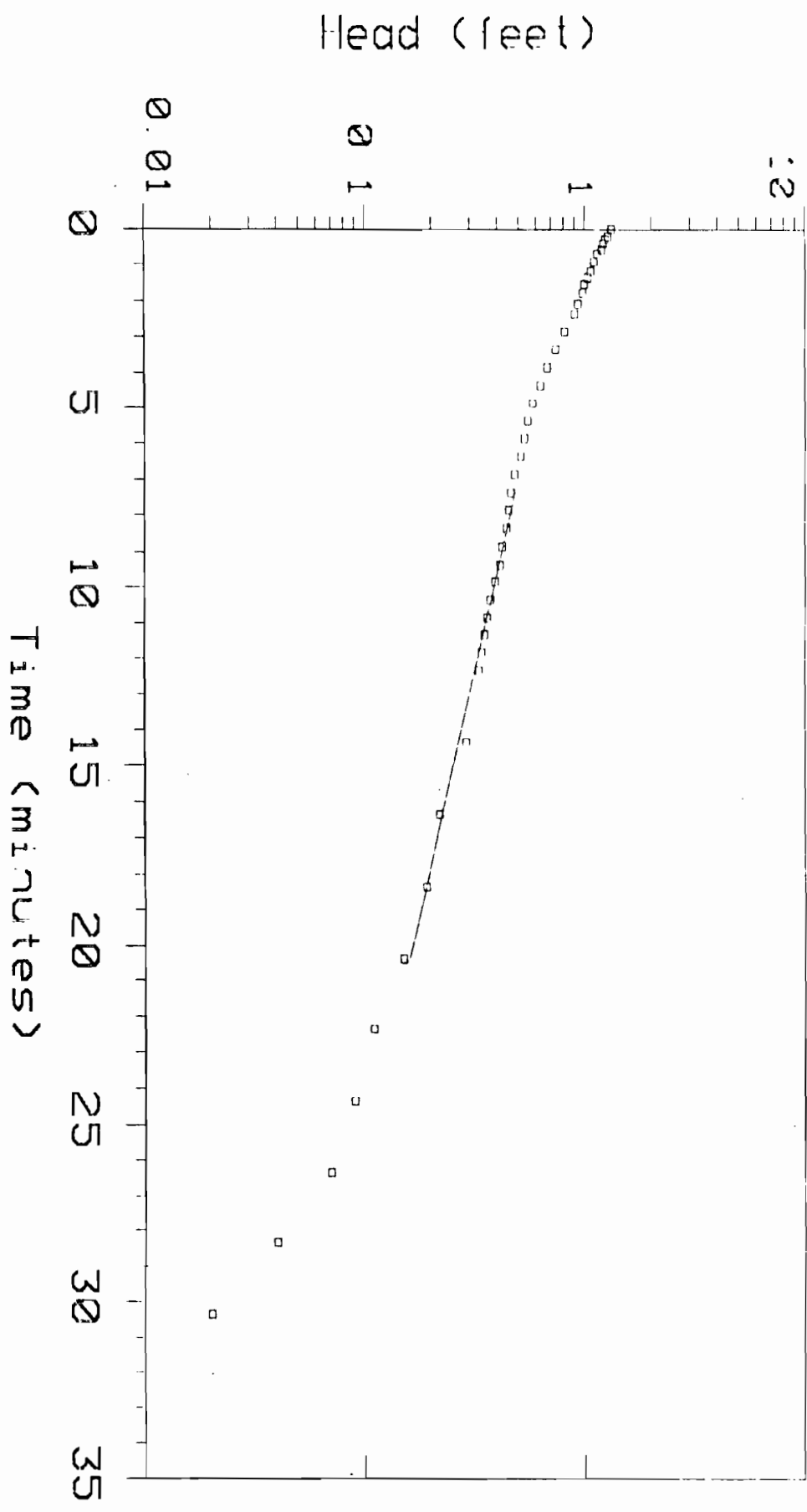
No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
1	1.000E-04	1.32		
2	0.200	1.28		
3	0.280	1.24		
4	0.410	1.21		
5	0.580	1.19		
6	0.700	1.14		
7	0.920	1.10		
8	1.20	1.06		
9	1.35	1.03		
10	1.53	1.00		
11	1.77	0.980		
12	2.07	0.930		
13	2.35	0.900		
14	2.85	0.810		
15	3.35	0.740		
16	3.85	0.680		
17	4.35	0.630		
18	4.85	0.580		
19	5.35	0.550		
20	5.85	0.530		
21	6.35	0.510		
22	6.85	0.480		
23	7.35	0.460	0.482	-4.96



No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
24	7.85	0.450	0.462	-2.83
25	8.35	0.440	0.443	-0.725
26	8.85	0.420	0.425	-1.20
27	9.35	0.410	0.407	0.641
28	9.85	0.390	0.390	-0.101
29	10.35	0.370	0.374	-1.12
30	10.85	0.360	0.358	0.396
31	11.35	0.350	0.343	1.81
32	11.85	0.340	0.329	3.13
33	12.35	0.330	0.315	4.35
34	14.35	0.290	0.266	8.13
35	16.35	0.220	0.224	-2.10
36	18.35	0.190	0.189	0.259
37	20.35	0.150	0.159	-6.33
38	22.35	0.110		
39	24.35	0.0900		
40	26.35	0.0700		
41	28.35	0.0400		
42	30.35	0.0200		

CURRENT RESOLUTION MATRIX NOT AVAILABLE

# SC2A



DATA SET: SC2B

CLIENT: SOLVENT CHEMICAL	DATE: 3/16/90
LOCATION: BUFFALO AVE.	WELL NO.: SC2B
COUNTY: ERIE	WELL DEPTH: 22.00 ft
PROJECT: Bail-down Test	WATER TABLE: 9.240 ft
AQUIFER: Unknown	THICKNESS: 12.00 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.167 ft
SCREEN TOP: 8.200 ft	SCREEN BASE: 20.00 ft
INITIAL HEAD: 0.430 ft	TRANS. RATIO: 1.0000

MODEL PARAMETERS:

TRANSMISSIVITY: .00418 square ft/min

CONDUCTIVITY: .000348 ft/min

MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer & Rice)

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
1	1.000E-04	0.970	0.202	79.10
2	0.250	0.200		
3	0.500	0.180		
4	0.750	0.160		
5	1.00	0.160		
6	1.25	0.150		
7	1.50	0.140		
8	1.75	0.140		
9	2.00	0.140		
10	2.50	0.130		
11	3.00	0.130		
12	3.50	0.120		
13	4.00	0.120		
14	4.50	0.120		
15	5.00	0.110		
16	5.50	0.100		
17	6.00	0.100		
18	6.50	0.0900		
19	7.00	0.0900		
20	7.50	0.0900		
21	8.00	0.0900		
22	8.50	0.0800		
23	9.00	0.0800		

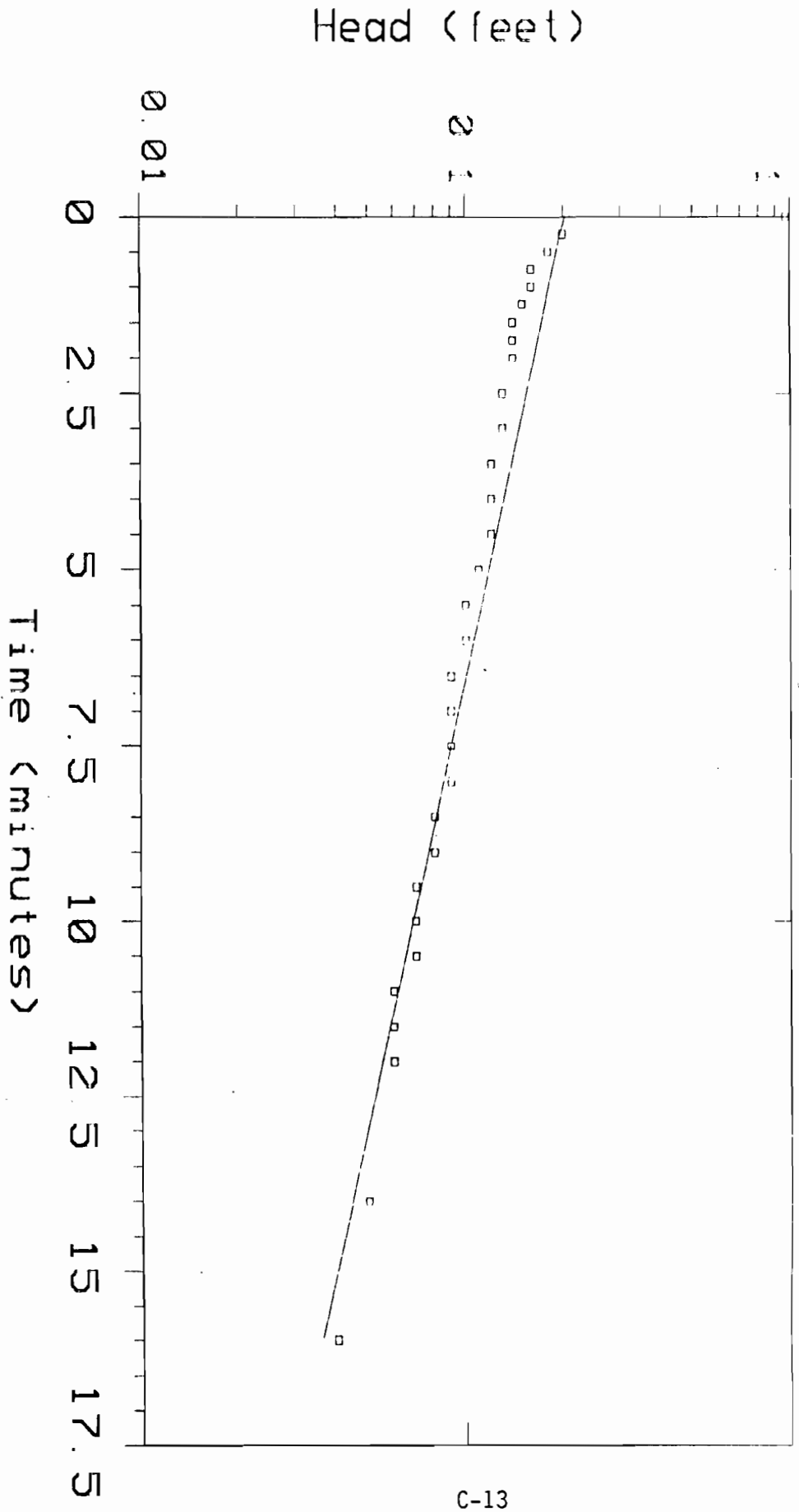
SCHE

PAGE 1

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
24	9.50	0.0700		
25	10.00	0.0700		
26	10.50	0.0700		
27	11.00	0.0600		
28	11.50	0.0600		
29	12.00	0.0600		
30	14.00	0.0500		
31	16.00	0.0400		

CURRENT RESOLUTION MATRIX NOT AVAILABLE

SC2B



----- SC3A -----

----- PAGE 1 -----

## DATA SET: SC3A

CLIENT: SOLVENT CHEMICAL	DATE: 3/19/90
LOCATION: BUFFALO AVE.	WELL NO.: SC3A
COUNTY: ERIE	WELL DEPTH: 9.60 ft
PROJECT: Bail-down Test	WATER TABLE: 6.120 ft
AQUIFER: Unknown	THICKNESS: 6.00 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.167 ft
SCREEN TOP: 4.500 ft	SCREEN BASE: 9.60 ft
INITIAL HEAD: 2.780 ft	TRANS. RATIO: 1.0000

## MODEL PARAMETERS:

TRANSMISSIVITY: .000661 square ft/min

CONDUCTIVITY: .000110 ft/min

MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer &amp; Rice)

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
1	1.000E-04	2.78		
2	0.250	0.290		
3	0.500	0.760		
4	0.750	0.900		
5	1.00	0.960		
6	1.25	1.06		
7	1.50	1.14		
8	1.75	1.19		
9	2.00	1.25		
10	2.50	1.36		
11	3.00	1.51		
12	3.50	1.90		
13	4.00	2.12		
14	4.50	2.27		
15	5.00	2.38		
16	5.50	2.45		
17	6.00	2.52		
18	6.50	2.56		
19	7.00	2.59		
20	7.50	2.63		
21	8.00	2.66		
22	8.50	2.67		

C-14

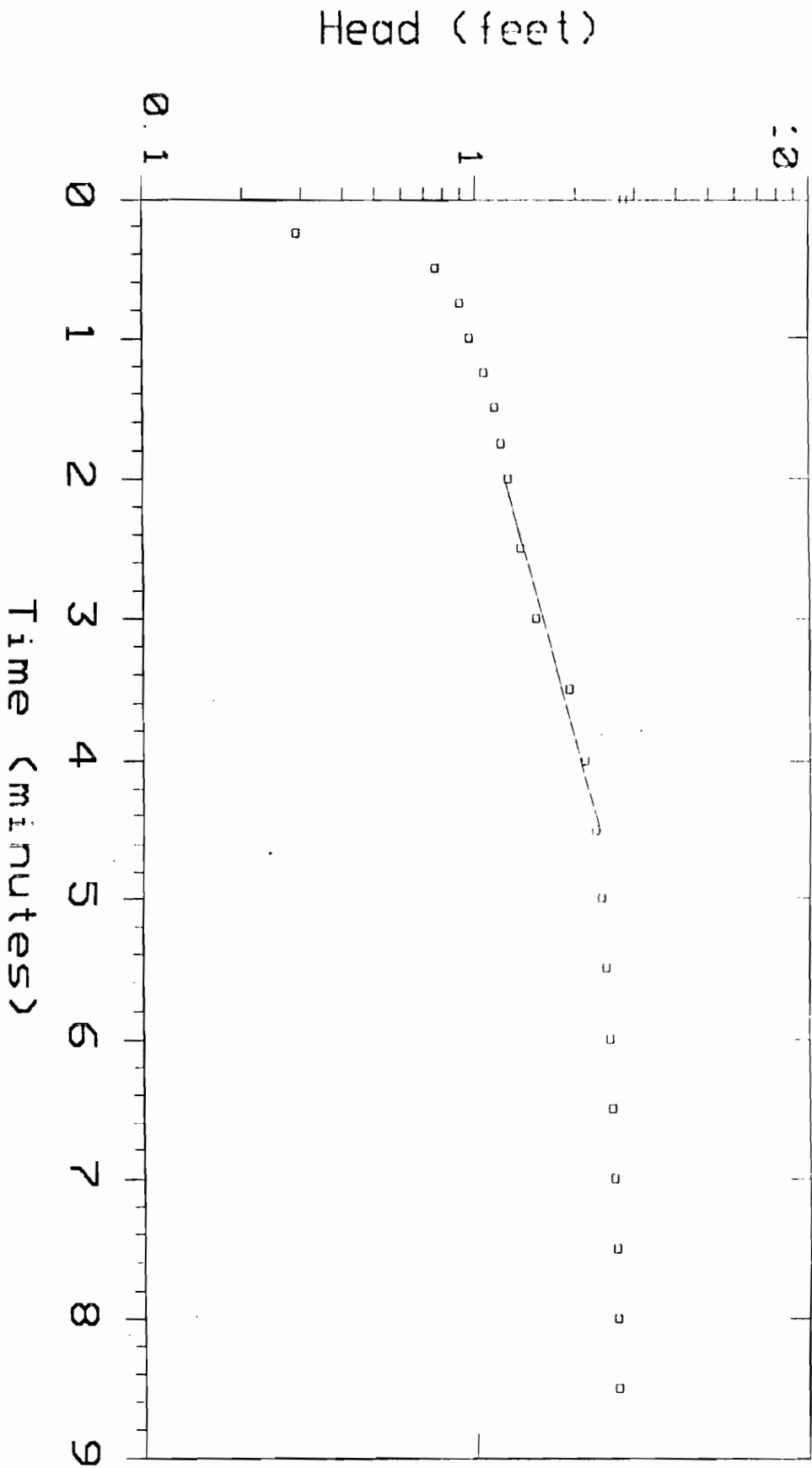
CURRENT RESOLUTION MATRIIX NOT AVAILABLE

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Ecology & Environment

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SC3A





----- SC3B -----

----- PAGE 1 -----

## DATA SET: SC3B

CLIENT: SOLVENT CHEMICAL	DATE: 3/20/90
LOCATION: BUFFALO AVE.	WELL NO.: SC3B
COUNTY: ERIE	WELL DEPTH: 21.20 ft
PROJECT: Bail-down Test	WATER TABLE: 11.050 ft
AQUIFER: Unknown	THICKNESS: 11.60 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.167 ft
SCREEN TOP: 9.600 ft	SCREEN BASE: 21.20 ft
INITIAL HEAD: 0.300 ft	TRANS. RATIO: 1.0000

## MODEL PARAMETERS:

TRANSMISSIVITY: .000867 square ft/min

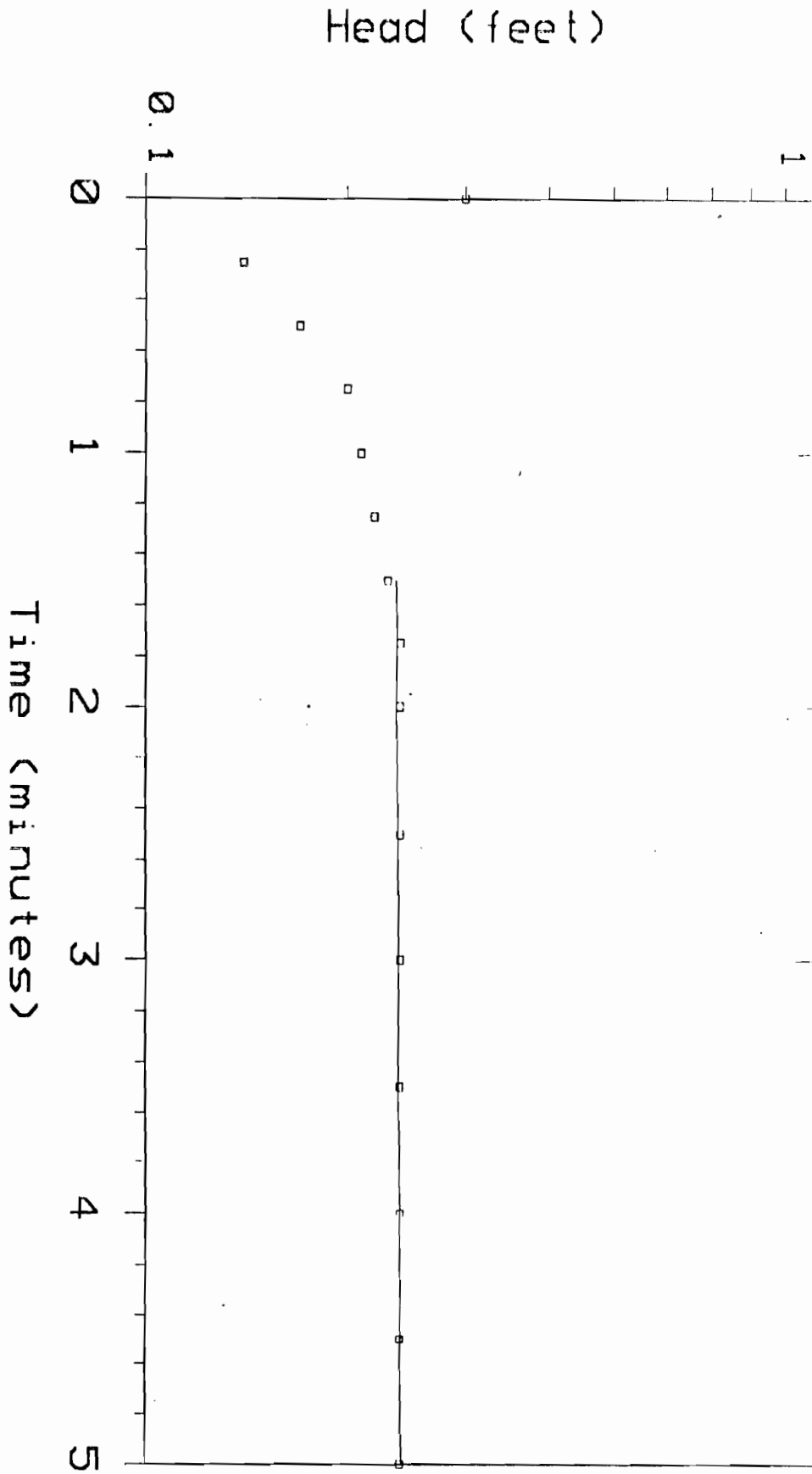
CONDUCTIVITY: 7.47E-5 ft/min

MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer &amp; Rice)

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
1	1.000E-04	0.300		
2	0.250	0.140		
3	0.500	0.170		
4	0.750	0.200		
5	1.00	0.210		
6	1.25	0.220		
7	1.50	0.230		
8	1.75	0.240		
9	2.00	0.240		
10	2.50	0.240		
11	3.00	0.240		
12	3.50	0.240		
13	4.00	0.240		
14	4.50	0.240		
15	5.00	0.240		

CURRENT RESOLUTION MARIIX NOT AVAILABLE

SC3B



SC4A

PAGE 1

## DATA SET: SC4A

CLIENT: SOLVENT CHEMICAL	DATE: 3/19/80
LOCATION: BUFFALO AVE.	WELL NO.: SC4A
COUNTY: ERIE	WELL DEPTH: 8.00 ft
PROJECT: Bail-down Test	WATER TABLE: 4.200 ft
AQUIFER: Unknown	THICKNESS: 5.00 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.167 ft
SCREEN TOP: 3.000 ft	SCREEN BASE: 8.00 ft
INITIAL HEAD: 3.230 ft	TRANS. RATIO: 1.0000

## MODEL PARAMETERS:

TRANSMISSIVITY: .000183 square ft/min

CONDUCTIVITY: 3.67E-5 ft/min

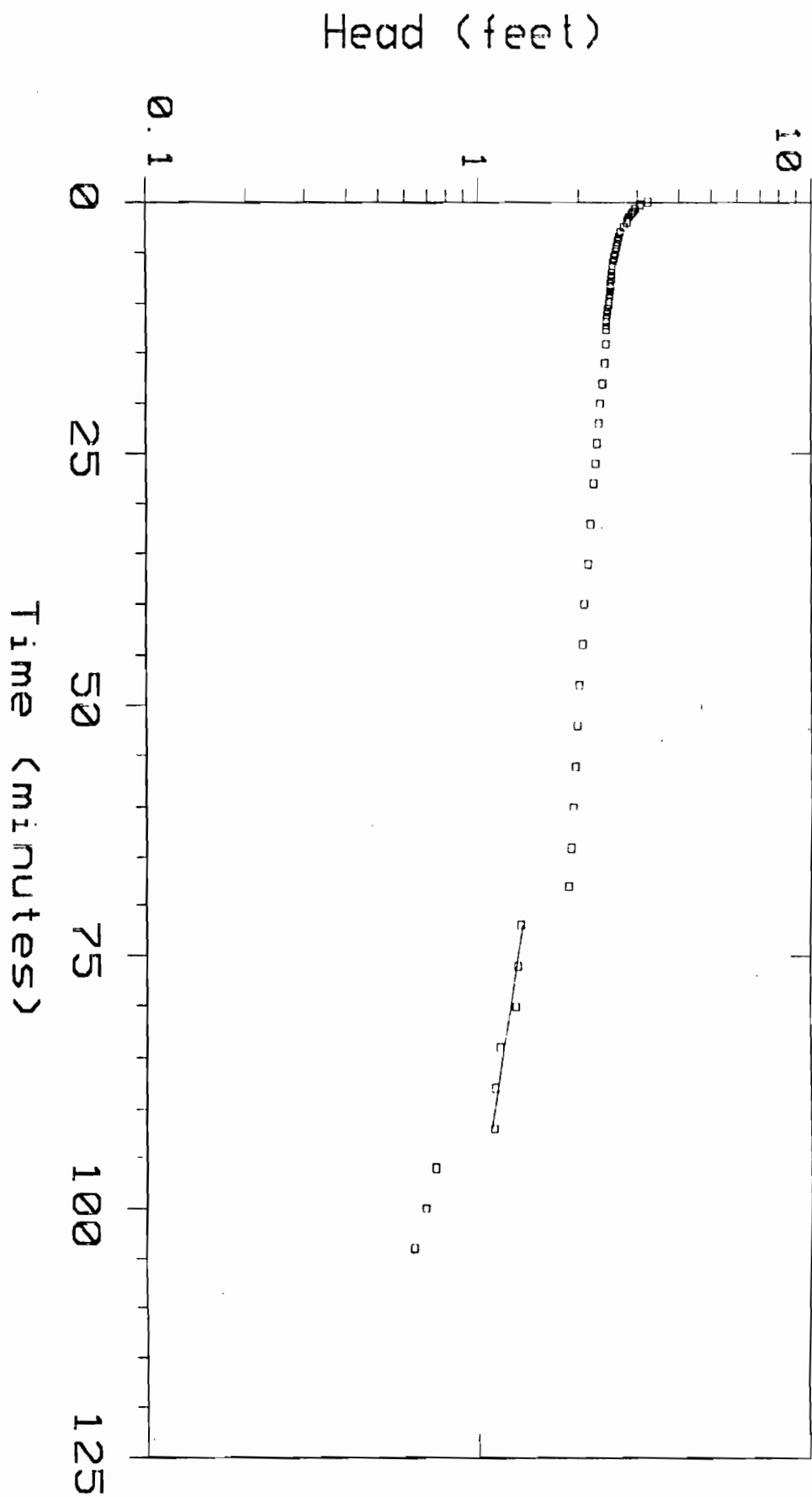
MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer & Rice)

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
1	1.000E-04	3.23		
2	0.250	3.06		
3	0.500	3.06		
4	0.750	2.96		
5	1.00	2.91		
6	1.25	2.88		
7	1.50	2.84		
8	1.75	2.81		
9	2.00	2.80		
10	2.50	2.74		
11	3.00	2.68		
12	3.50	2.65		
13	4.00	2.63		
14	4.50	2.61		
15	5.00	2.59		
16	5.50	2.57		
17	6.00	2.55		
18	6.50	2.54		
19	7.00	2.53		
20	7.50	2.52		
21	8.00	2.50		
22	8.50	2.50		
23	9.00	2.49		

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
24	9.50	2.48		
25	10.00	2.47		
26	10.50	2.45		
27	11.00	2.44		
28	11.50	2.43		
29	12.00	2.42		
30	12.50	2.42		
31	14.00	2.41		
32	16.00	2.39		
33	18.00	2.36		
34	20.00	2.32		
35	22.00	2.30		
36	24.00	2.28		
37	26.00	2.25		
38	28.00	2.22		
39	32.00	2.17		
40	36.00	2.13		
41	40.00	2.08		
42	44.00	2.05		
43	48.00	2.01		
44	52.00	1.98		
45	56.00	1.95		
46	60.00	1.92		
47	64.00	1.89		
48	68.00	1.86		
49	72.00	1.84		
50	76.00	1.81		
51	80.00	1.79		
52	84.00	1.76		
53	88.00	1.74		
54	92.00	1.71		
55	96.00	0.740		
56	100.0	0.690		
57	104.0	0.640		

CURRENT RESOLUTION MATRIIX NOT AVAILABLE

# SC4A



SC4B

PAGE 1

## DATA SET: SC4B

CLIENT: SOLVENT CHEMICAL	DATE: 8/13/80
LOCATION: BUFFALO AVE.	WELL NO.: SC4B
COUNTY: ERIE	WELL DEPTH: 22.50 ft
PROJECT: Bail-down Test	WATER TABLE: 10.620 ft
AQUIFER: Unknown	THICKNESS: 14.50 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.167 ft
SCREEN TOP: 9.000 ft	SCREEN BASE: 22.50 ft
INITIAL HEAD: 0.020 ft	TRANS. RATIO: 1.0000

## MODEL PARAMETERS:

TRANSMISSIVITY: UNKNOWN

CONDUCTIVITY: -1.00E-9 ft/min

MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer &amp; Rice)

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
1	1.000E-04	0.0200	0.0200	4.1575E-05
2	0.500	0.0200		
3	0.750	0.0200		

CURRENT RESOLUTION MATRIX NOT AVAILABLE

SC5A

PAGE 1

## DATA SET: SC5A

CLIENT: SOLVENT CHEMICAL	DATE: 8/15/80
LOCATION: BUFFALO AVE.	WELL NO.: SC5A
COUNTY: ERIE	WELL DEPTH: 12.50 ft
PROJECT: Bail-down Test	WATER TABLE: 7.870 ft
AQUIFER: Unknown	THICKNESS: 7.50 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.167 ft
SCREEN TOP: 5.000 ft	SCREEN BASE: 10.50 ft
INITIAL HEAD: 2.890 ft	TRANS. RATIO: 1.0000

## MODEL PARAMETERS:

TRANSMISSIVITY: .00176 square ft/min

CONDUCTIVITY: .000235 ft/min

MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer & Rice)

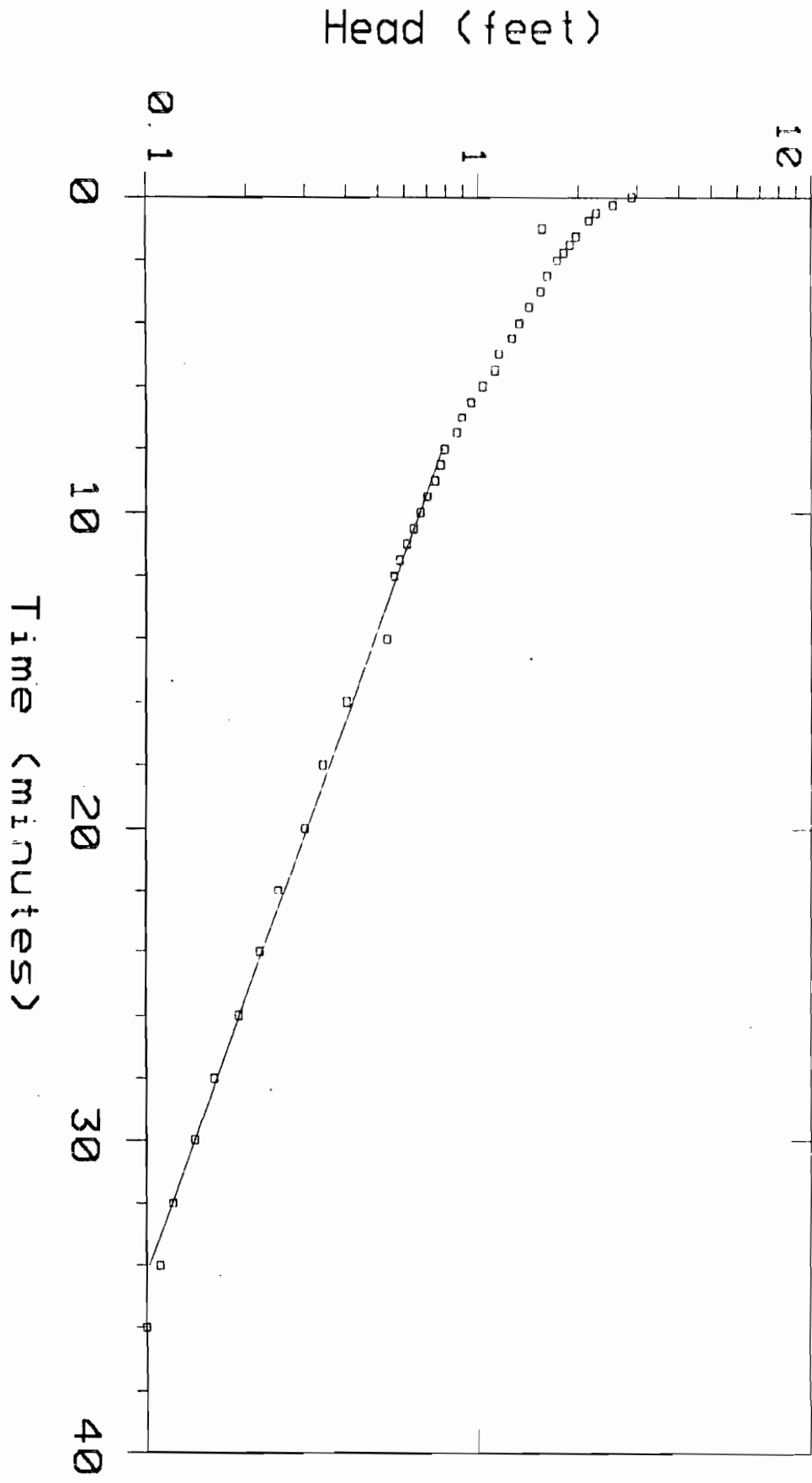
No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
1	1.000E-04	2.89		
2	0.250	2.54		
3	0.500	2.26		
4	0.750	2.15		
5	1.00	1.56		
6	1.25	1.97		
7	1.50	1.89		
8	1.75	1.81		
9	2.00	1.73		
10	2.50	1.61		
11	3.00	1.54		
12	3.50	1.42		
13	4.00	1.33		
14	4.50	1.26		
15	5.00	1.15		
16	5.50	1.12		
17	6.00	1.03		
18	6.50	0.950		
19	7.00	0.890		
20	7.50	0.860		
21	8.00	0.790		
22	8.50	0.770		
23	9.00	0.740		

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
24	9.50	0.700		
25	10.00	0.670		
26	10.50	0.640		
27	11.00	0.610		
28	11.50	0.580		
29	12.00	0.560		
30	14.00	0.530		
31	16.00	0.400		
32	18.00	0.340		
33	20.00	0.300		
34	22.00	0.250		
35	24.00	0.220		
36	26.00	0.190		
37	28.00	0.160		
38	30.00	0.140		
39	32.00	0.120		
40	34.00	0.110		
41	36.00	0.100		

CURRENT RESOLUTION MATRIIX NOT AVAILABLE



SC5A



SC5B

PAGE 1

## DATA SET: SC5B

CLIENT: SOLVENT CHEMICAL	DATE: 3/20/90
LOCATION: BUFFALO AVE.	WELL NO.: SC5B
COUNTY: ERIE	WELL DEPTH: 21.00 ft
PROJECT: Slug Test	WATER TABLE: 18.040 ft
AQUIFER: Unknown	THICKNESS: 8.50 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.167 ft
SCREEN TOP: 12.500 ft	SCREEN BASE: 11.00 ft
INITIAL HEAD: 0.620 ft	TRANS. RATIO: 1.0000

## MODEL PARAMETERS:

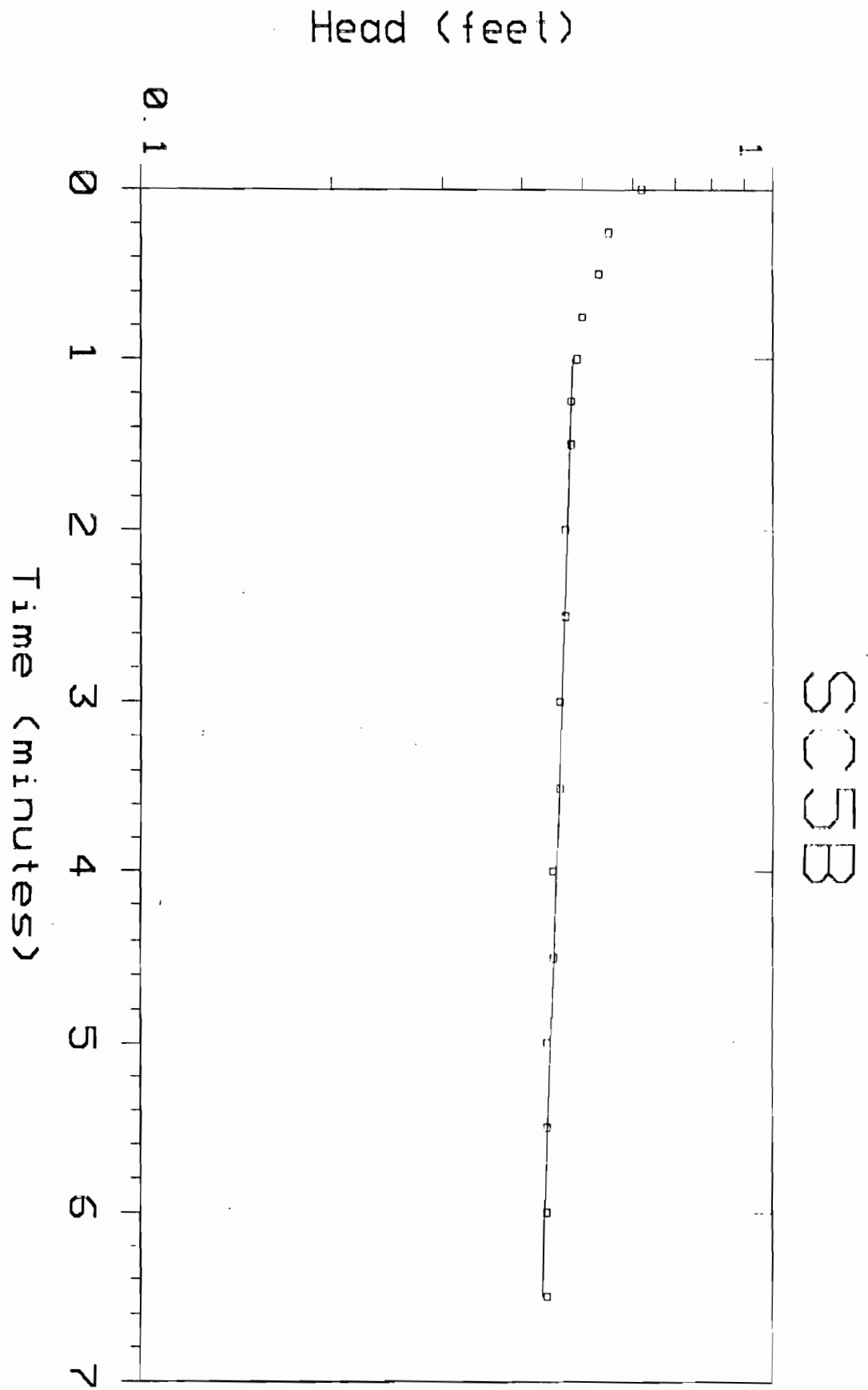
TRANSMISSIVITY: .000819 square ft/min

CONDUCTIVITY: 9.63E-5 ft/min

MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer &amp; Rice)

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
1	1.000E-04	0.620		
2	0.250	0.550		
3	0.500	0.530		
4	0.750	0.500		
5	1.00	0.490		
6	1.25	0.480		
7	1.50	0.480		
8	2.00	0.470		
9	2.50	0.470		
10	3.00	0.460		
11	3.50	0.460		
12	4.00	0.450		
13	4.50	0.450		
14	5.00	0.440		
15	5.50	0.440		
16	6.00	0.440		
17	6.50	0.440		

CURRENT RESOLUTION MARIIX NOT AVAILABLE



SC5C

PAGE 1

## DATA SET: SC5C

CLIENT: SOLVENT CHEMICAL	DATE: 3/16/90
LOCATION: BUFFALO AVE.	WELL NO.: SC5C
COUNTY: ERIE	WELL DEPTH: 36.50 ft
PROJECT: Slug test	WATER TABLE: 19.320 ft
AQUIFER: Unknown	THICKNESS: 10.00 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.00 ft
SCREEN TOP: 26.500 ft	SCREEN BASE: 36.50 ft
INITIAL HEAD: 1.140 ft	TRANS. RATIO: 1.0000

## MODEL PARAMETERS:

TRANSMISSIVITY: .0178 square ft/min

CONDUCTIVITY: .00178 ft/min

MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer & Rice)

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
1	1.000E-04	1.14		
2	0.0800	1.00		
3	0.200	0.980		
4	0.300	0.910		
5	0.470	0.880		
6	0.580	0.810		
7	0.670	0.760		
8	0.830	0.710		
9	0.950	0.670		
10	1.11	0.600		
11	1.30	0.570		
12	1.47	0.530		
13	1.58	0.480		
14	1.73	0.460		
15	1.83	0.430		
16	2.30	0.420	0.446	-6.24
17	2.83	0.300	0.321	-7.15
18	3.50	0.230	0.212	7.68
19	3.83	0.160	0.173	-8.19
20	4.50	0.130	0.114	12.03
21	4.83	0.0800	0.0932	-16.52
22	5.50	0.0700	0.0615	12.02
23	5.83	0.0500	0.0502	-0.406

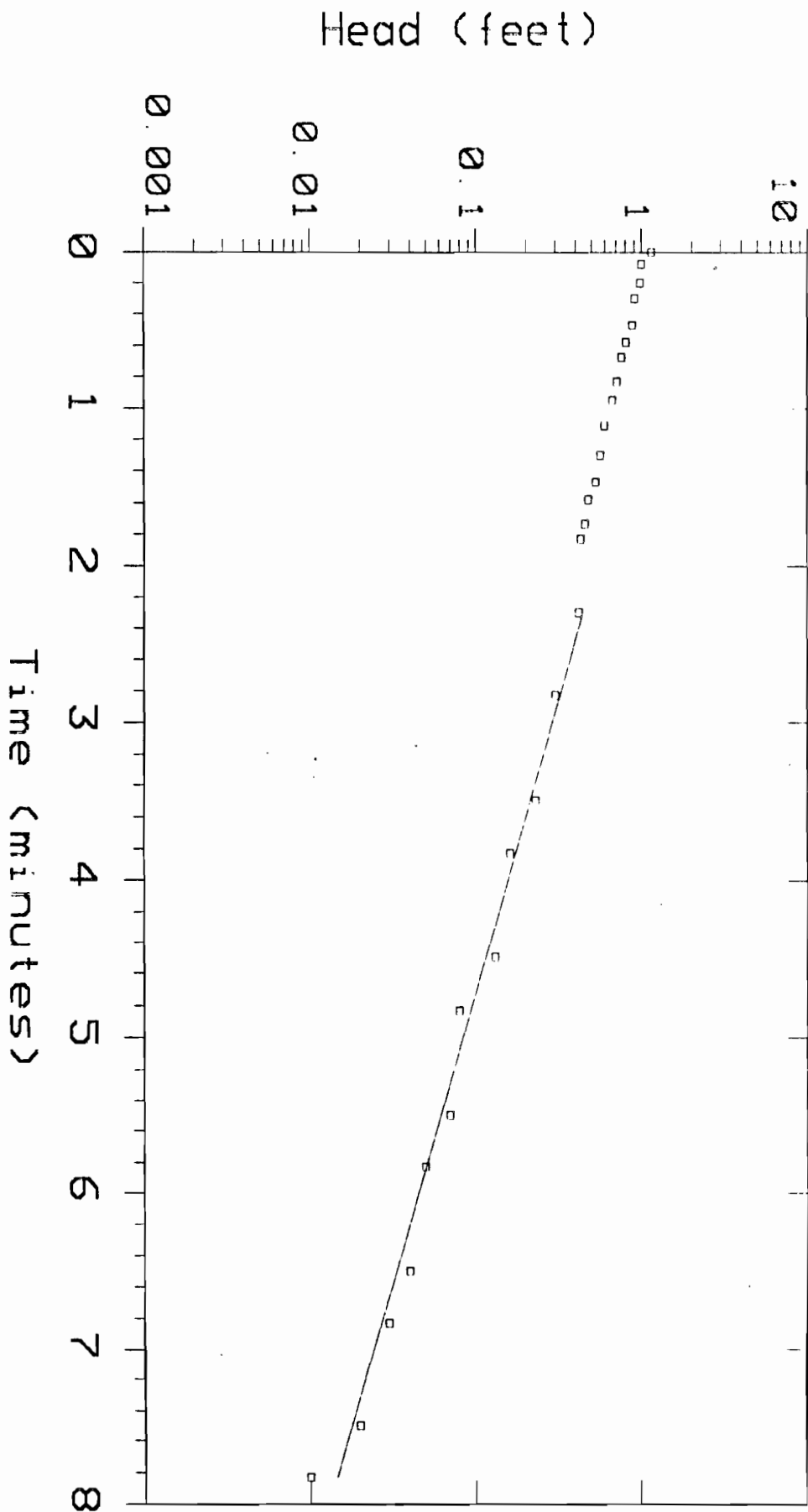
No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
24	6.50	0.0400	0.0331	17.04
25	6.83	0.0300	0.0270	9.83
26	7.50	0.0200	0.0178	10.91
27	7.83	0.0100	0.0145	-45.59

CURRENT RESOLUTION MATRIX NOT AVAILABLE

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SC5C



SC5CD

PAGE 1

## DATA SET: SC5CD

CLIENT: SOLVENT CHEMICAL	DATE: 3/20/90
LOCATION: BUFFALO AVE.	WELL NO.: SC5CD
COUNTY: ERIE	WELL DEPTH: 42.90 ft
PROJECT: Slug test	WATER TABLE: 23.830 ft
AQUIFER: Unknown	THICKNESS: 30.40 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.157 ft
SCREEN TOP: 12.500 ft	SCREEN BASE: 42.90 ft
INITIAL HEAD: 8.180 ft	TRANS. RATIO: 1.0000

## MODEL PARAMETERS:

TRANSMISSIVITY: .0701 square ft/min

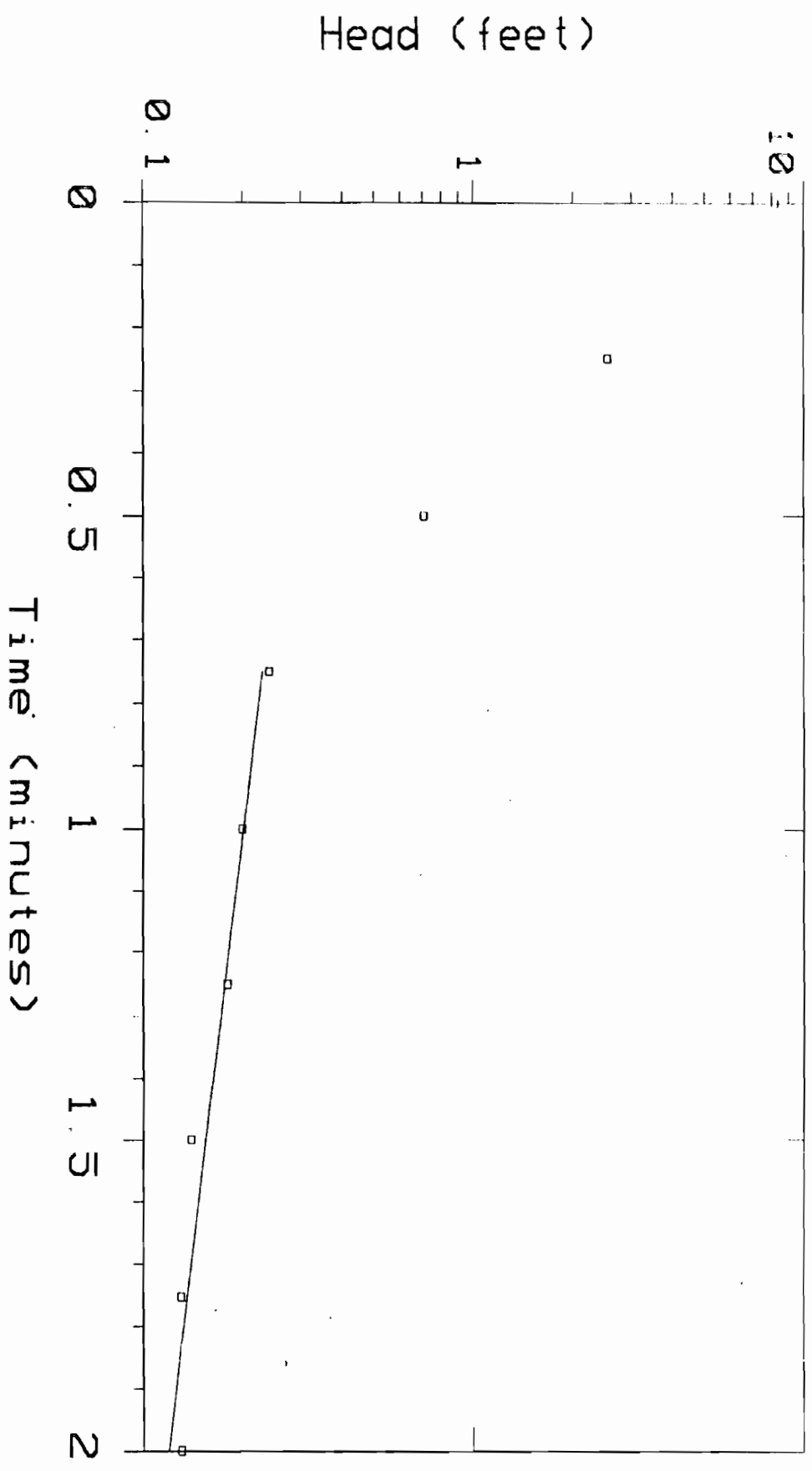
CONDUCTIVITY: .00230 ft/min

MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer &amp; Rice)

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
1	1.000E-04	8.18		
2	0.250	2.55		
3	0.500	0.710		
4	0.750	0.240	0.229	4.23
5	1.00	0.200	0.201	-0.735
6	1.25	0.180	0.176	1.88
7	1.50	0.140	0.154	-10.58
8	1.75	0.130	0.135	-4.39
9	2.00	0.130	0.118	8.48

CURRENT RESOLUTION MATRIIX NOT AVAILABLE

SC50D





SC5F

PAGE 1

## DATA SET: SC5F

CLIENT: SOLVENT CHEMICAL	DATE: 3/20/90
LOCATION: BUFFALO AVE.	WELL NO.: SC5F
COUNTY: ERIE	WELL DEPTH: 98.00 ft
PROJECT: Slug test	WATER TABLE: 17.920 ft
AQUIFER: Unknown	THICKNESS: 98.00 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.167 ft
SCREEN TOP: 12.500 ft	SCREEN BASE: 98.00 ft
INITIAL HEAD: 1.670 ft	TRANS. RATIO: 1.0000

## MODEL PARAMETERS:

TRANSMISSIVITY: .0902 square ft/min

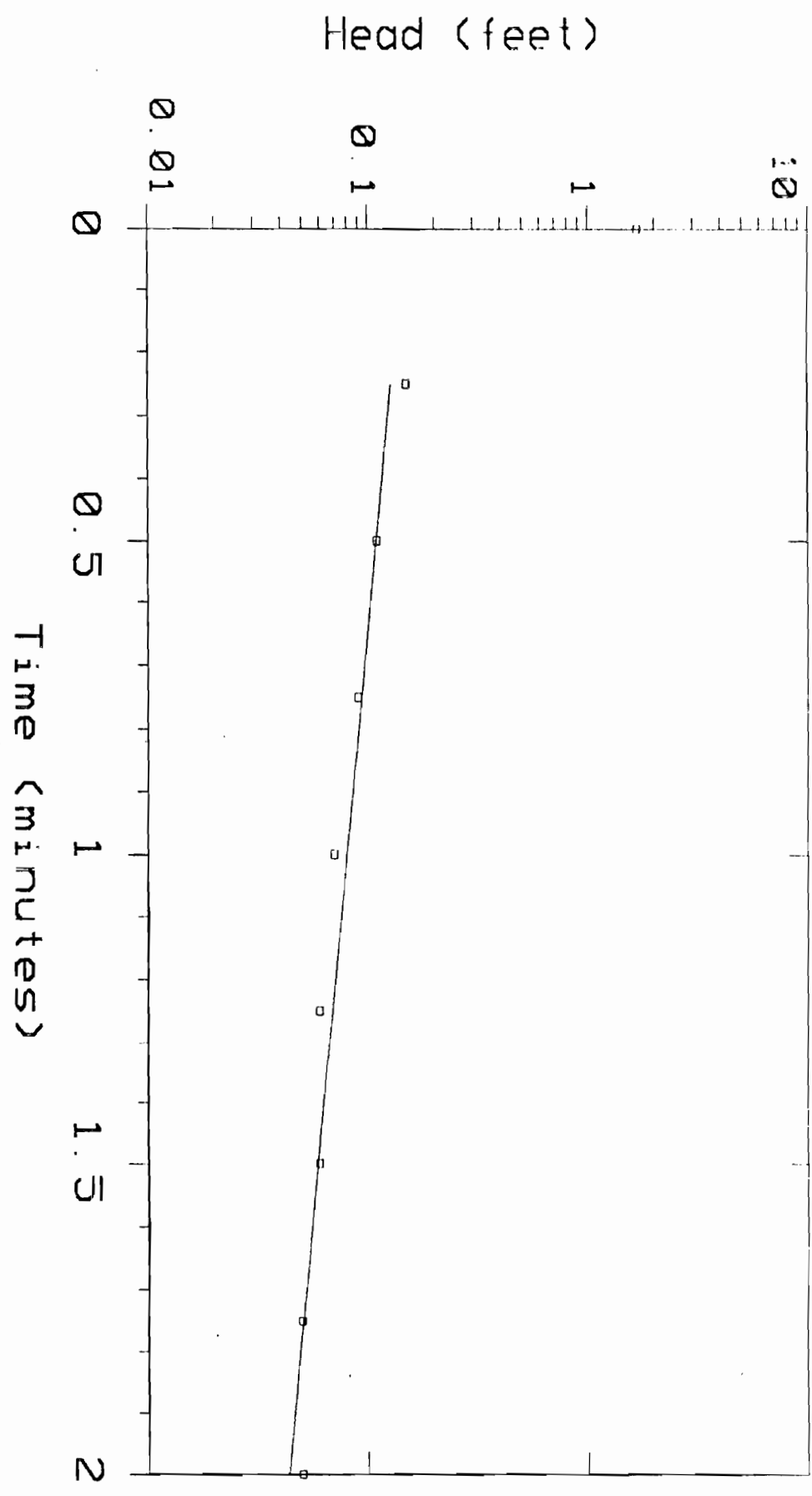
CONDUCTIVITY: .00105 ft/min

MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer &amp; Rice)

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
1	1.000E-04	1.67		
2	0.250	0.150	0.127	14.77
3	0.500	0.110	0.109	0.445
4	0.750	0.0900	0.0938	-4.22
5	1.00	0.0700	0.0803	-14.78
6	1.25	0.0600	0.0688	-14.71
7	1.50	0.0600	0.0589	1.73
8	1.75	0.0500	0.0505	-1.00
9	2.00	0.0500	0.0432	13.43

CURRENT RESOLUTION MARIIX NOT AVAILABLE

SCSF



----- SC6A

----- PAGE 1

## DATA SET: SC6A

CLIENT: SOLVENT CHEMICAL	DATE: 3/19/90
LOCATION: BUFFALO AVE.	WELL NO.: SC6A
COUNTY: ERIE	WELL DEPTH: 9.60 ft
PROJECT: Slug test	WATER TABLE: 5.830 ft
AQUIFER: Unknown	THICKNESS: 5.60 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.167 ft
SCREEN TOP: 4.000 ft	SCREEN BASE: 9.60 ft
INITIAL HEAD: 3.110 ft	TRANS. RATIO: 1.0000

## MODEL PARAMETERS:

TRANSMISSIVITY: .000657 square ft/min

CONDUCTIVITY: .000117 ft/min

MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer &amp; Rice)

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
1	1.000E-04	3.11		
2	0.250	3.03		
3	0.500	2.94		
4	0.750	2.88		
5	1.00	2.85		
6	1.25	2.81		
7	1.50	2.78		
8	1.75	2.76		
9	2.00	2.73		
10	2.50	2.69		
11	3.00	2.65		
12	3.50	2.59		
13	4.00	2.55		
14	4.50	2.52		
15	5.00	2.49		
16	5.50	2.46		
17	6.00	2.42		
18	6.50	2.39		
19	7.00	2.36		
20	7.50	2.33		
21	8.00	2.28		
22	8.50	2.25		
23	9.00	2.22		

No.	TIME (mins)	Head. H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
24	9.50	2.20		
25	10.00	2.17		
26	10.50	2.13		
27	11.00	2.12		
28	11.50	2.09		
29	12.00	2.07		
30	12.50	2.04		
31	13.00	2.02		
32	15.00	1.94		
33	17.00	1.83		
34	19.00	1.75		
35	21.00	1.66		
36	23.00	1.58		
37	25.00	1.52		
38	27.00	1.46		
39	29.00	1.40		
40	31.00	1.20		
41	33.00	1.06		
42	35.00	0.930		
43	37.00	0.840		
44	39.00	0.750		
45	41.00	0.680		
46	43.00	0.600		
47	45.00	0.550		
48	47.00	0.490		
49	49.00	0.440		
50	51.00	0.400		
51	53.00	0.370		
52	55.00	0.330		
53	57.00	0.310		
54	59.00	0.280		
55	61.00	0.260		
56	63.00	0.230		
57	65.00	0.210		
58	67.00	0.180		

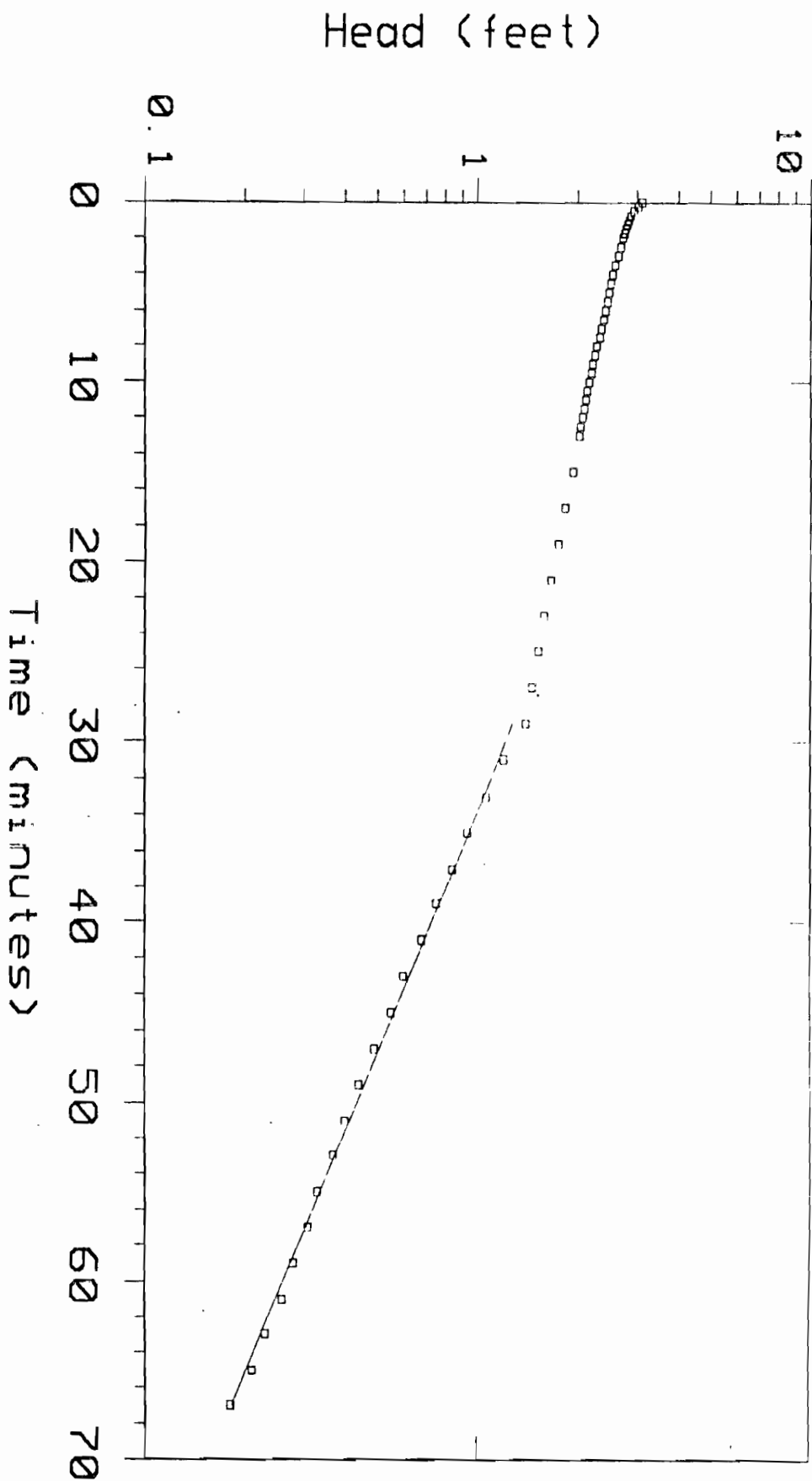
CURRENT RESOLUTION MATRIIX NOT AVAILABLE

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Ecology &amp; Environment

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505A



SC6B

PAGE 1

## DATA SET: SC6B

CLIENT: SOLVENT CHEMICAL	DATE: 01/20/90
LOCATION: BUFFALO AVE.	WELL NO.: SC6B
COUNTY: ERIE	WELL DEPTH: 20.00 ft
PROJECT: Slug test	WATER TABLE: 9.600 ft
AQUIFER: Unknown	THICKNESS: 11.40 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.167 ft
SCREEN TOP: 9.600 ft	SCREEN BASE: 11.00 ft
INITIAL HEAD: 0.690 ft	TRANS. RATIO: 1.0000

## MODEL PARAMETERS:

TRANSMISSIVITY: .00842 square ft/min

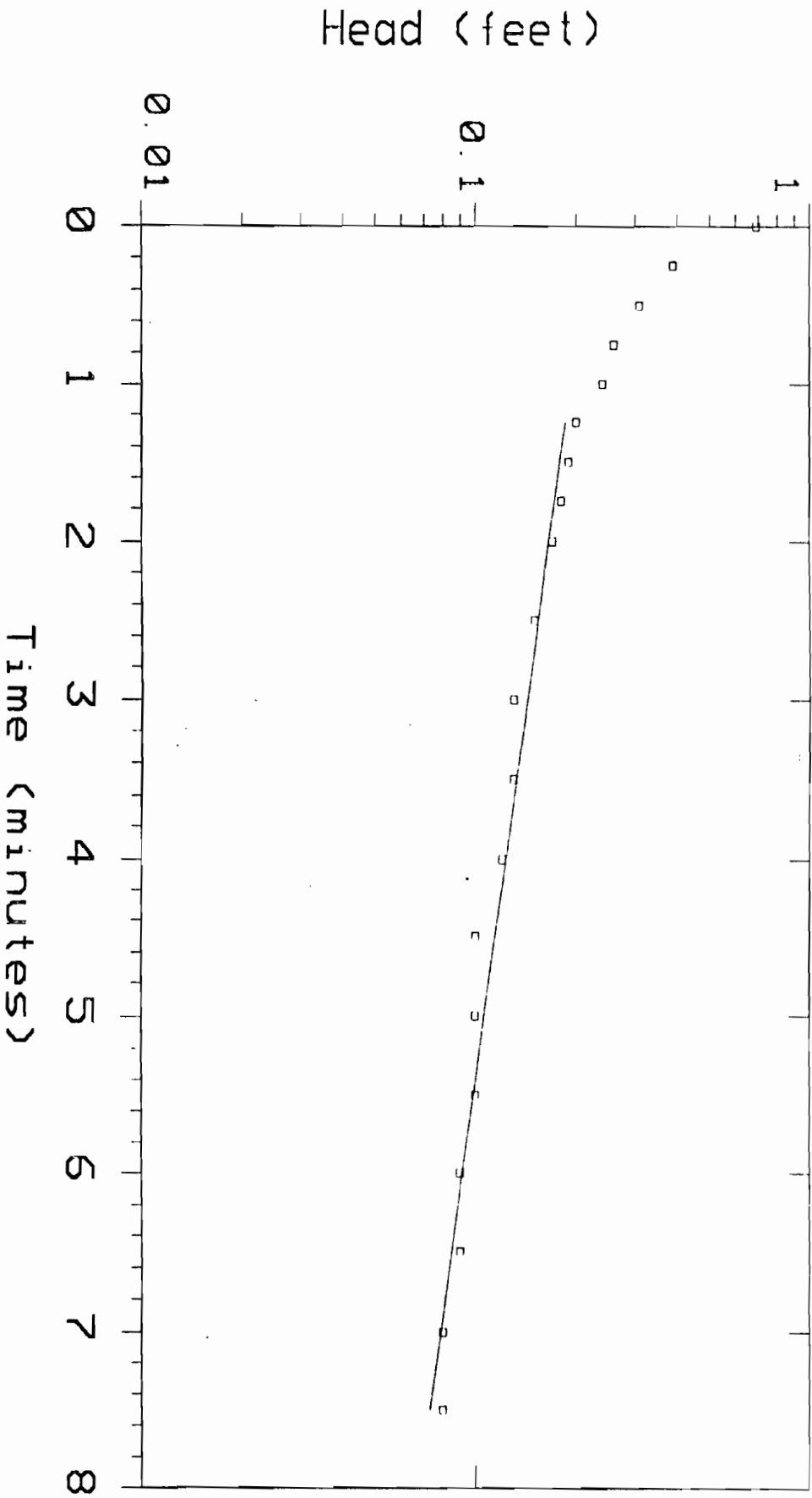
CONDUCTIVITY: .000810 ft/min

MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer &amp; Rice)

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
1	1.000E-04	0.690		
2	0.250	0.390		
3	0.500	0.310		
4	0.750	0.260		
5	1.00	0.240		
6	1.25	0.200		
7	1.50	0.190		
8	1.75	0.180		
9	2.00	0.170		
10	2.50	0.150		
11	3.00	0.130		
12	3.50	0.130		
13	4.00	0.120		
14	4.50	0.100		
15	5.00	0.100		
16	5.50	0.100		
17	6.00	0.0900		
18	6.50	0.0900		
19	7.00	0.0800		
20	7.50	0.0800		

CURRENT RESOLUTION MATRIIX NOT AVAILABLE

# SC6B



----- SC7A

----- PAGE 1

## DATA SET: SC7A

CLIENT: SOLVENT CHEMICAL	DATE: 3/19/90
LOCATION: BUFFALO AVE.	WELL NO: SC7A
COUNTY: ERIE	WELL DEPTH: 7.50 ft
PROJECT: Slug test	WATER TABLE: 4.490 ft
AQUIFER: Unknown	THICKNESS: 5.00 ft
INTAKE RADIUS: 0.667 ft	CASING RADIUS: 0.187 ft
SCREEN TOP: 2.500 ft	SCREEN BASE: 7.50 ft
INITIAL HEAD: 2.900 ft	TRANS. RATIO: 1.0000

## MODEL PARAMETERS:

TRANSMISSIVITY: .00865 square ft/min

CONDUCTIVITY: .00173 ft/min

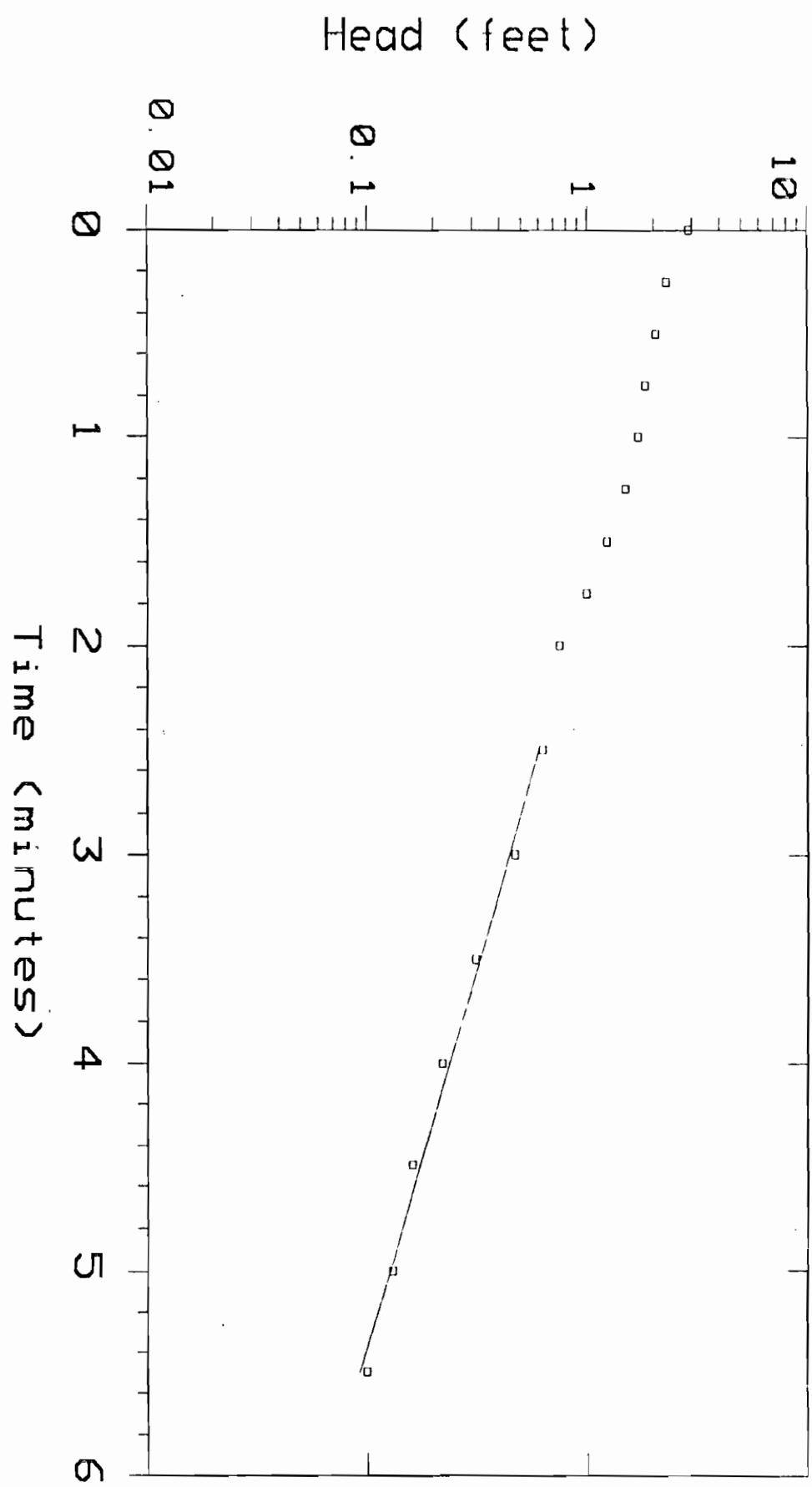
MODEL TYPE: UNCONFINED PARTIALLY PENETRATED AQUIFER (Bouwer &amp; Rice)

No.	TIME (mins)	Head, H (ft)		DIFFERENCE (percent)
		DATA	SYNTHETIC	
1	1.000E-04	2.90		
2	0.250	2.29		
3	0.500	2.05		
4	0.750	1.84		
5	1.00	1.70		
6	1.25	1.49		
7	1.50	1.23		
8	1.75	0.990		
9	2.00	0.750		
10	2.50	0.630		
11	3.00	0.470		
12	3.50	0.310		
13	4.00	0.220		
14	4.50	0.160		
15	5.00	0.130		
16	5.50	0.100		

CURRENT RESOLUTION MARIIX NOT AVAILABLE



# SC7A



APPENDIX D

LOADING DATA

3163 BUFFALO AVENUE LOADING DATA  
A-ZONE (BEST CASE)

Contaminant	Western Boundary*		Northern Boundary**	
	February (#/D)	May (#/D)	February (#/D)	May (#/D)
Vinyl chloride	--	--	$2.34 \times 10^{-5}$	$2.6 \times 10^{-5}$
Total-1,2-dichloroethene	--	--	$8.65 \times 10^{-5}$	$9.58 \times 10^{-5}$
1,2-Dichloroethane	--	--	--	--
Trichloroethene	--	--	$3.4 \times 10^{-5}$	$3.8 \times 10^{-5}$
Benzene	0.0742	0.083	$4.1 \times 10^{-6}$	$4.55 \times 10^{-6}$
Tetrachloroethene	--	--	$1.84 \times 10^{-4}$	$2.04 \times 10^{-4}$
Chlorobenzene	0.042	0.046	$2.7 \times 10^{-5}$	$3 \times 10^{-5}$
1,3-Dichlorobenzene	$2.69 \times 10^{-3}$	$2.98 \times 10^{-3}$	$1.65 \times 10^{-5}$	$1.79 \times 10^{-4}$
1,4-Dichlorobenzene	0.023	0.026	$8.46 \times 10^{-5}$	$8.86 \times 10^{-4}$
1,2-Dichlorobenzene	0.027	0.03	$4.76 \times 10^{-4}$	$5.28 \times 10^{-4}$
1,2,4-Trichlorobenzene	0.0109	0.0121	$7.06 \times 10^{-4}$	$7.84 \times 10^{-4}$
Arsenic	$7.73 \times 10^{-5}$	$8.59 \times 10^{-5}$	$3.49 \times 10^{-6}$	$3.88 \times 10^{-6}$
Barium	$1.58 \times 10^{-3}$	$1.76 \times 10^{-3}$	$3.51 \times 10^{-4}$	$3.96 \times 10^{-4}$
Cadmium	$5.17 \times 10^{-5}$	$5.75 \times 10^{-5}$	$9.94 \times 10^{-6}$	$1.11 \times 10^{-5}$
Chromium	$2.79 \times 10^{-4}$	$3.1 \times 10^{-4}$	$6.18 \times 10^{-5}$	$6.82 \times 10^{-5}$
Lead	$3.79 \times 10^{-3}$	$4.18 \times 10^{-3}$	$1.09 \times 10^{-4}$	$1.22 \times 10^{-4}$
Manganese	$4.64 \times 10^{-3}$	$5.16 \times 10^{-3}$	$1.83 \times 10^{-3}$	$2.04 \times 10^{-3}$
Zinc	$9.47 \times 10^{-3}$	0.0105	$5.24 \times 10^{-4}$	$5.82 \times 10^{-4}$
Cyanide	$3.46 \times 10^{-4}$	$3.84 \times 10^{-5}$	$1.51 \times 10^{-4}$	$1.68 \times 10^{-4}$

[IL]JD1900:D3048/1229/23

\*Western wells = SC1A, SC7A  
\*\*Northern wells = SC5A, SC4A

3163 BUFFALO AVENUE LOADING DATA  
A-ZONE (WORST CASE)

Contaminant	Western Boundary*		Northern Boundary**	
	February (#/D)	May (#/D)	February (#/D)	May (#/D)
Vinyl chloride	--	--	$4.06 \times 10^{-5}$	$4.5 \times 10^{-5}$
Total-1,2-dichloroethene	--	--	$1.56 \times 10^{-4}$	$1.23 \times 10^{-3}$
1,2-Dichloroethane	--	--	--	--
Trichloroethene	--	--	$6.6 \times 10^{-5}$	$7.4 \times 10^{-5}$
Benzene	0.0783	0.0874	$7.11 \times 10^{-6}$	$7.88 \times 10^{-6}$
Tetrachloroethene	--	--	$3.57 \times 10^{-4}$	$3.96 \times 10^{-4}$
Chlorobenzene	0.0506	0.0557	$4.7 \times 10^{-5}$	$5 \times 10^{-5}$
1,3-Dichlorobenzene	$3.21 \times 10^{-3}$	0.028	$2.89 \times 10^{-5}$	$3.1 \times 10^{-4}$
1,4-Dichlorobenzene	0.028	0.0387	$1.48 \times 10^{-4}$	$1.97 \times 10^{-4}$
1,2-Dichlorobenzene	0.0331	0.037	$8.26 \times 10^{-4}$	$9.17 \times 10^{-4}$
1,2,4-Trichlorobenzene	0.0132	0.0146	$1.22 \times 10^{-3}$	$1.36 \times 10^{-3}$
Arsenic	$9.31 \times 10^{-5}$	$1.03 \times 10^{-4}$	$6.65 \times 10^{-6}$	$7.67 \times 10^{-6}$
Barium	$1.9 \times 10^{-3}$	$2.11 \times 10^{-3}$	$6.88 \times 10^{-4}$	$3.1 \times 10^{-4}$
Cadmium	$6.22 \times 10^{-5}$	$6.72 \times 10^{-4}$	$1.74 \times 10^{-4}$	$3.16 \times 10^{-4}$
Chromium	$3.36 \times 10^{-4}$	$3.7 \times 10^{-4}$	$1.2 \times 10^{-4}$	$1.31 \times 10^{-4}$
Lead	$4.54 \times 10^{-3}$	$5.04 \times 10^{-3}$	$2.08 \times 10^{-4}$	$2.33 \times 10^{-4}$
Manganese	$5.55 \times 10^{-3}$	$6.18 \times 10^{-3}$	$3.51 \times 10^{-3}$	$3.9 \times 10^{-3}$
Zinc	0.115	0.0127	$1 \times 10^{-3}$	$1.13 \times 10^{-3}$
Cyanide	$4.14 \times 10^{-4}$	$4.58 \times 10^{-4}$	$2.62 \times 10^{-4}$	$2.91 \times 10^{-4}$

[IL]JD1900:D3048/1229/23

\*Western wells = SC1A, SC7A

\*\*Northern wells = SC5A, SC4A

3163 BUFFALO AVENUE LOADING DATA  
A-ZONE

Loading Totals	Western Boundary*		Northern Boundary**	
	February (#/D)	May (#/D)	February (#/D)	May (#/D)
<b>VOLATILE ORGANICS</b>				
Best case	0.1162	0.129	$3.59 \times 10^{-4}$	$3.98 \times 10^{-4}$
Worst case	0.1289	0.1431	$6.74 \times 10^{-4}$	$1.87 \times 10^{-3}$
<b>SEMI-VOLATILE ORGANICS</b>				
Best case	0.0636	0.0711	$1.28 \times 10^{-3}$	$2.37 \times 10^{-3}$
Worst case	0.0775	0.1183	$2.22 \times 10^{-3}$	$2.78 \times 10^{-3}$
<b>METALS</b>				
Best case	0.02	0.022	$3.04 \times 10^{-3}$	$3.39 \times 10^{-3}$
Worst case	0.128	0.0276	$5.97 \times 10^{-3}$	$6.32 \times 10^{-3}$
<b>TOTAL LOADING</b>				
Best case	0.1998	0.2221	$4.68 \times 10^{-3}$	$6.16 \times 10^{-3}$
Worst case	0.3344	0.289	$8.86 \times 10^{-3}$	0.011

[IL]JD1900:D3048/1229/23

\*Western wells = SC1A, SC7A  
\*\*Northern wells = SC5A, SC4A

3163 BUFFALO AVENUE LOADING DATA  
B-ZONE (BEST CASE)

Contaminant	Western Boundary*		Northern Boundary**	
	February (#/D)	May (#/D)	February (#/D)	May (#/D)
Vinyl chloride	--	--	--	--
Total-1,2-dichloroethene	--	--	--	--
1,2-Dichloroethane	--	--	--	--
Trichloroethene	--	--	--	--
Benzene	8.97	12.5	$2.6 \times 10^{-4}$	$3.6 \times 10^{-4}$
Tetrachloroethene	--	--	--	--
Chlorobenzene	--	--	$1.56 \times 10^{-3}$	$2.18 \times 10^{-3}$
1,3-Dichlorobenzene	0.113	0.158	0.0304	0.0425
1,4-Dichlorobenzene	0.347	0.485	0.1068	0.1494
1,2-Dichlorobenzene	0.289	0.404	0.1382	0.1933
1,2,4-Trichlorobenzene	0.142	0.199	0.2776	0.0388
Arsenic	$3.4 \times 10^{-4}$	$4.76 \times 10^{-4}$	$9.29 \times 10^{-6}$	$1.3 \times 10^{-5}$
Barium	--	--	--	--
Cadmium	$2.43 \times 10^{-4}$	$3.4 \times 10^{-4}$	$7.8 \times 10^{-6}$	$1.09 \times 10^{-5}$
Chromium	$1.47 \times 10^{-3}$	$2.06 \times 10^{-3}$	$1.73 \times 10^{-3}$	$2.4 \times 10^{-3}$
Lead	$3.86 \times 10^{-3}$	$5.4 \times 10^{-3}$	$2.65 \times 10^{-3}$	$3.7 \times 10^{-3}$
Manganese	0.024	0.034	0.228	0.032
Zinc	0.093	0.13	$2.43 \times 10^{-3}$	$3.4 \times 10^{-3}$
Cyanide	$2.43 \times 10^{-3}$	$3.4 \times 10^{-3}$	--	--

[IL]JD1900:D3048/1229/23

\*Western wells = SC1B

\*\*Northern wells = SC4B, SC5B

3163 BUFFALO AVENUE LOADING DATA  
B-ZONE (WORST CASE)

Contaminant	Western Boundary*		Northern Boundary**	
	February (#/D)	May (#/D)	February (#/D)	May (#/D)
Vinyl chloride	--	--	--	--
Total-1,2-dichloroethene	--	--	--	--
1,2-Dichloroethane	--	--	--	--
Trichloroethene	--	--	--	--
Benzene	8.97	12.5	$1.21 \times 10^{-3}$	$1.69 \times 10^{-3}$
Tetrachloroethene	--	--	--	--
Chlorobenzene	--	--	$7.28 \times 10^{-3}$	0.0102
1,3-Dichlorobenzene	0.113	0.158	0.031	0.0433
1,4-Dichlorobenzene	0.347	0.485	0.1081	0.1512
1,2-Dichlorobenzene	0.289	0.404	0.1408	0.1969
1,2,4-Trichlorobenzene	0.142	0.199	0.0287	0.0401
Arsenic	$3.4 \times 10^{-4}$	$4.76 \times 10^{-4}$	$4.33 \times 10^{-5}$	$6.06 \times 10^{-5}$
Barium	--	--	--	--
Cadmium	$2.43 \times 10^{-4}$	$3.4 \times 10^{-4}$	$3.64 \times 10^{-5}$	$5.09 \times 10^{-5}$
Chromium	$1.47 \times 10^{-3}$	$2.06 \times 10^{-3}$	$1.823 \times 10^{-3}$	$2.55 \times 10^{-3}$
Lead	$3.86 \times 10^{-3}$	$5.4 \times 10^{-3}$	$4.07 \times 10^{-3}$	$5.69 \times 10^{-3}$
Manganese	0.024	0.034	0.0294	0.0414
Zinc	0.093	0.13	$6.46 \times 10^{-3}$	$9.03 \times 10^{-3}$
Cyanide	$2.43 \times 10^{-3}$	$3.4 \times 10^{-3}$	--	--

[IL]JD1900:D3048/1229/23

\*Western wells = SC1B (same values as best case)

\*\*Northern wells = SC4B, SC5B

3163 BUFFALO AVENUE LOADING DATA  
B-ZONE

Loading Totals	Western Boundary*		Northern Boundary**	
	February (#/D)	May (#/D)	February (#/D)	May (#/D)
<b>VOLATILE ORGANICS</b>				
Best case	8.97	12.5	$1.82 \times 10^{-3}$	$2.54 \times 10^{-3}$
Worst case	8.97	12.5	$8.49 \times 10^{-3}$	0.0119
<b>SEMI-VOLATILE ORGANICS</b>				
Best case	0.891	1.246	0.553	0.424
Worst case	0.891	1.246	0.3086	0.4315
<b>METALS</b>				
Best case	0.125	0.176	0.235	0.0415
Worst case	0.125	0.176	0.0418	0.0588
<b>TOTAL LOADING</b>				
Best case	10.0	13.92	0.79	0.468
Worst case	10.0	13.92	0.3589	0.5022

[IL]JD1900:D3048/1229/23

\*Western wells = SC1B  
\*\*Northern wells = SC4B, SC5B



3163 BUFFALO AVENUE LOADING DATA

Contaminant	Zone*	Best Case (#/D)	Worst Case (#/D)	Month W.L. Taken
Vinyl chloride	C	1.13 x 10 <sup>-5</sup>	1.18 x 10 <sup>-5</sup>	March
Total-1,2-dichloroethene	C	0.013	0.0136	March
1,2-Dichloroethane	C	--	--	March
Trichloroethene	C	3.97 x 10 <sup>-3</sup>	4.5 x 10 <sup>-3</sup>	March
Benzene	C	0.147	0.153	March
Tetrachloroethene	C	5.09 x 10 <sup>-3</sup>	5.33 x 10 <sup>-3</sup>	March
Chlorobenzene	C	0.0684	0.072	March
1,3-Dichlorobenzene	C	0.1192	0.124	March
1,4-Dichlorobenzene	C	0.0302	0.032	March
1,2-Dichlorobenzene	C	0.062	0.065	March
1,2,4-Trichlorobenzene	C	0.0446	0.047	March
Arsenic	C	5.64 x 10 <sup>-4</sup>	5.9 x 10 <sup>-4</sup>	March
Barium	C	--	--	March
Cadmium	C	--	--	March
Chromium	C	5.3 x 10 <sup>-4</sup>	5.58 x 10 <sup>-4</sup>	March
Lead	C	1.07 x 10 <sup>-3</sup>	1.12 x 10 <sup>-3</sup>	March
Manganese	C	3.46 x 10 <sup>-3</sup>	3.63 x 10 <sup>-3</sup>	March
Zinc	C	7.73 x 10 <sup>-3</sup>	8.09 x 10 <sup>-3</sup>	March
Cyanide	C	0.04	0.042	March

[IL]JDI900:D3048/1229/23

\*C Zone = well SC5C; C-D Zone = well SC5C-D; F Zone = well SC5F; all are northern boundary wells.

## 3163 BUFFALO AVENUE LOADING DATA

Contaminant	Zone*	Best Case (#/D)	Worst Case (#/D)	Month W.L. Taken
Vinyl chloride	C-D	--	--	May
Total-1,2-dichloroethene	C-D	0.0154	0.0346	May
1,2-Dichloroethane	C-D	--	--	May
Trichloroethene	C-D	--	--	May
Benzene	C-D	0.0295	0.067	May
Tetrachloroethene	C-D	--	--	May
Chlorobenzene	C-D	0.175	0.395	May
1,3-Dichlorobenzene	C-D	0.0195	0.044	May
1,4-Dichlorobenzene	C-D	0.051	0.115	May
1,2-Dichlorobenzene	C-D	0.115	0.258	May
1,2,4-Trichlorobenzene	C-D	0.0364	0.082	May
Arsenic	C-D	--	--	May
Barium	C-D	--	--	May
Cadmium	C-D	--	--	May
Chromium	C-D	$5.91 \times 10^{-4}$	$1.33 \times 10^{-3}$	May
Lead	C-D	$2.09 \times 10^{-3}$	$4.7 \times 10^{-3}$	May
Manganese	C-D	0.014	0.032	May
Zinc	C-D	$9.77 \times 10^{-3}$	0.022	May
Cyanide	C-D	0.02	0.045	May

[IL]JD1900:D3048/1229/23

\*C Zone = well SC5C; C-D Zone = well SC5C-D; F Zone = well SC5F; all are northern boundary wells.

## 3163 BUFFALO AVENUE LOADING DATA

Contaminant	Zone*	Best Case (#/D)	Worst Case (#/D)	Month W.L. Taken
Vinyl chloride	F	0.646	3.42	May
Total-1,2-dichloroethene	F	0.158	0.836	May
1,2-Dichloroethane	F	--	--	May
Trichloroethene	F	--	--	May
Benzene	F	--	--	May
Tetrachloroethene	F	--	--	May
Chlorobenzene	F	--	--	May
1,3-Dichlorobenzene	F	--	--	May
1,4-Dichlorobenzene	F	$9.06 \times 10^{-4}$	$4.8 \times 10^{-3}$	May
1,2-Dichlorobenzene	F	$2.57 \times 10^{-3}$	0.0136	May
1,2,4-Trichlorobenzene	F	--	--	May
Arsenic	F	--	--	May
Barium	F	--	--	May
Cadmium	F	--	--	May
Chromium	F	0.578	3.06	May
Lead	F	$1.43 \times 10^{-3}$	$7.57 \times 10^{-3}$	May
Manganese	F	$6.04 \times 10^{-3}$	0.032	May
Zinc	F	0.117	0.062	May
Cyanide	F	--	--	May

[IL]JD1900:D3048/1229/23

\*C Zone = well SC5C; C-D Zone = well SC5C-D; F Zone = well SC5F; all are northern boundary wells.

3163 BUFFALO AVENUE LOADING DATA

Loading Totals	C Zone	C-D Zone	F Zone
<b>VOLATILE ORGANICS</b>			
Best case	0.2375	0.2199	0.804
Worst case	0.2484	0.4966	4.256
<b>SEMI-VOLATILE ORGANICS</b>			
Best case	0.256	0.2219	3.476 x 10 <sup>-3</sup>
Worst case	0.268	0.499	0.0184
<b>METALS</b>			
Best case	0.0533	0.0446	0.7024
Worst case	0.056	0.105	3.16
<b>TOTAL LOADING</b>			
Best case	0.5468	0.4864	1.51
Worst case	0.5724	1.1	7.43

[IL]JDI900:D3048/1229/23

3163 BUFFALO AVENUE LOADING DATA

(values given in lb/day)  
(all worst case)

	Western Boundary*	Northern Boundary**
<b>A ZONE</b>		
PESTICIDE		
α BHC	1.9 x 10 <sup>-5</sup>	9.5 x 10 <sup>-6</sup>
β BHC	--	3.9 x 10 <sup>-5</sup>
δ BHC	3.9 x 10 <sup>-6</sup>	--
γ BHC	--	--
<b>B ZONE</b>		
PESTICIDE		
α BHC	0.011	0.155
β BHC	--	--
δ BHC	--	7.75 x 10 <sup>-5</sup>
γ BHC	--	--
<b>C ZONE</b>		
PESTICIDE		
α BHC	--	--
β BHC	--	--
δ BHC	--	--
γ BHC	--	--

[IL]JD1900:D3048/1229/23

\*Western wells = SC1A; SC1B.  
\*\*Northern wells = SC4A; SC4B; SC5B; SC5C; SC5C; SC5C; SC5C-D; SC5F.

3163 BUFFALO AVENUE LOADING DATA

(values given in lb/day)  
(all worst case)

C-D ZONE	Well SC-D
PESTICIDE	
α BHC	0.016
β BHC	4.55 x 10 <sup>-3</sup>
δ BHC	--
γ BHC	4 x 10 <sup>-3</sup>
F ZONE	Well 5F
PESTICIDE	
α BHC	8 x 10 <sup>-3</sup>
β BHC	--
δ BHC	--
γ BHC	--

[IL]JD1900:D3048/1229/23

**APPENDIX E**  
**TOXICOLOGICAL PROFILES**

## ARSENIC

Arsenic is a naturally occurring element usually found combined with one or more other elements such as oxygen, chlorine, and sulfur. Arsenic combined with these elements is referred to as inorganic arsenic, while oxygen combined with carbon and hydrogen is referred to as organic arsenic. The organic forms are usually less toxic than the inorganic forms (ATSDR 1989). Arsenic is widely distributed in the environment from natural sources, but higher concentrations have been found associated with the following situations: waste chemical disposal sites, smelting of copper and other metals, fossil fuel combustion, and pesticide use. Average 24-hour ambient air arsenic levels in the United States (U.S.), based on National Air Sampling Network data, ranged from 2.6 ng/m<sup>3</sup> to 10.9 ng/m<sup>3</sup> (Akland 1983). The natural arsenic content of soils varies between 0.1 and 80 ppm, with an average of 5 to 6 ppm (Walsh and Keenly 1975). Over 90% of all surface water contains 10 µg/L arsenic or less. The average concentration of arsenic in U.S. drinking water supplies is about 2 µg/L (Greathouse and Craun 1978).

### Environmental Chemistry and Fate

Arsenic may be released to the atmosphere as a gas or vapor, or adsorbed to particulate matter and transported to other media by dry or wet deposition (ATSDR 1989). Trivalent arsenic may undergo oxidation in the air; therefore, atmospheric arsenic is usually a mixture of the trivalent and pentavalent forms. Most airborne arsenic is usually adsorbed on small-diameter particulate matter. Photolysis is not considered to be an important fate process for arsenic.

Arsenic in surface water can undergo a complex pattern of transformations: oxidation-reduction, ligand exchange, biotransformation, precipitation, and adsorption (EPA 1978). As a consequence of these reactions, arsenic is extremely mobile in aquatic systems, and river-borne arsenic is capable of being transported great distances. Factors most strongly influencing the rates of these reactions include: Eh, pH, metal sulfide and sulfide ion concentrations, iron concentration, presence of phosphorus minerals, temperature, salinity, and distribution and composition of biota (EPA 1979).



Sorption onto clays, iron oxides, manganese compounds, and organic matter is an important fate mechanism in surface water, with sediment serving as a reservoir for most of the arsenic entering surface water. Sediment-bound trivalent and pentavalent arsenic, methylated by aerobic and anaerobic microorganisms, may be released back into the water column.

Soluble forms of arsenic adsorb to soil and travel with the soil matter. Shifts in oxidation state may occur in either direction, depending on the particular characteristics of the soil and groundwater. Volatilization of methylated arsenicals from groundwater is possible.

Arsenic in soil is predominantly found in an insoluble, adsorbed form. Clay with high anion-exchange capacity strongly adsorbs pentavalent arsenic. Other important adsorption processes include complexation and chelation by organic material, iron, or calcium. Leaching of arsenic is usually important in the top 30 centimeters of soil, but may also be important at greater depth in sandy soils. Arsenate predominates in aerobic soils; arsenite is the predominant form in slightly reduced soils; and arsine, methylated arsenicals and elemental arsenic predominate in very reduced conditions (e.g., swamps and bogs)(ATSDR 1987).

As noted above, microorganisms may reduce and methylate arsenicals in water and soil, resulting in volatilization and emission to the air. The volatilization rate is heavily dependent on whether the soil is oxygenated or anaerobic, the pH, and the microbe types and concentrations in soils.

In aquatic systems, bioconcentration of arsenic primarily occurs in algae and lower invertebrates, but biomagnification does not appear to be significant (EPA 1979).

Plants may accumulate arsenic via root uptake, with uptake being dependent on the species, soil arsenic concentration, and soil characteristics.

### Toxicokinetics

Exposure to arsenic compounds may occur orally and through inhalation. Little is known about the dermal absorption route. Inorganic arsenic compounds are generally well absorbed, widely distributed and

promptly excreted. The rates of these processes depend on the chemical and physical form, particularly the water solubility, of the arsenic compound. Exposure of animal species to either trivalent or pentavalent arsenic leads to the initial accumulation of arsenic in liver, kidney, lung, spleen, aorta, skin, hair, and upper gastrointestinal tract (EPA 1984a). These tissues are cleared rapidly except for skin and hair, where the sulfhydryl groups of keratin may promote tight arsenic (III) binding (ATSDR 1989).

Metabolism of arsenic occurs predominantly in the liver by methylation. The major metabolite is dimethylarsinic acid, which is excreted in the urine.

### **Noncarcinogenic Effects**

At high doses, arsenic compounds have been shown to produce acute and chronic toxic effects, including irreversible systemic damage. The trivalent compounds are the most toxic and tend to accumulate in the body. Animal studies have shown that chronic arsenic exposure may cause body weight changes, decreased blood hemoglobin, liver damage, and kidney damage.

There is evidence that arsenic is an essential element enhancing growth and development in certain animal species, and it has been suggested that arsenic may be an essential element for humans (NAS 1980). Whether or not arsenic is an essential element is the subject of continuing research.

Teratogenic effects of arsenic compounds at relatively high exposure levels have been demonstrated in a number of animal species (ATSDR 1989). Generally, these effects have been observed following parenteral (injection) administration; whereas, administration at lower doses by the more relevant oral route has not resulted in any significant reproductive or developmental effects.

### **Mutagenicity and Carcinogenicity**

Arsenic has been shown to be mutagenic in several assay systems and to induce chromosomal aberrations in vivo and in vitro. Animal carcinogenicity studies have reported conflicting results. Several studies have reported an increased incidence of bronchogenic carcinomas in rats

exposed intratracheally to an arsenic-containing pesticide. Reasons for inconsistent carcinogenicity findings in animals may include inappropriate selection of an animal model, and use of flawed study designs. In humans, epidemiologic studies and case reports have documented that arsenic is associated with tumors of the skin, lungs, genital organs, and visual organs (EPA 1987, ATSDR 1989).

EPA has classified arsenic in Group A, a human carcinogen, based on extensive evidence of human carcinogenicity through inhalation and ingestion exposure (EPA 1990a,b).

### Quantitative Indices of Toxicity

Quantitative indices of toxicity for arsenic are presented in Table 1.

Using the absolute-risk linear model, EPA derived the upper-bound 95% confidence limit slope factor (SF) based on human exposure data. The upper-bound estimate for the inhalation route is  $50.0 \text{ (mg/kg/day)}^{-1}$ .

### Standards and Criteria

Standards and criteria relevant to arsenic are listed in Table 2. The current MCL for arsenic under the National Interim Drinking Water Regulations is 50 ug/L. The NAS Drinking Water Committee has analyzed the toxicology of arsenic (NAS 1983a). Based upon this evaluation, NAS recommended the retention of the MCL, pending resolution of the question whether arsenic is an essential element in the human diet.

Consistent with the NAS recommendations, EPA has proposed that the MCL remain at the current MCL of 50 ug/L. In its determination, EPA stated that the MCL was below concentrations at which noncarcinogenic toxicity had been demonstrated and was within the concentration range which might be, based on further investigation, essential for humans (EPA 1985, 1987).

### References

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Table 1A  
 NONCARCINOGENIC INDICES  
 OF TOXICITY FOR ARSENIC

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<u>Oral Route</u>	
Chronic Reference Dose (RfD) mg/kg/day	1 x 10 <sup>-3</sup>
Subchronic RfD (RfDs) mg/kg/day	1 x 10 <sup>-3</sup>
Confidence Level	NS
Critical Effect	keratosis; hyperpigmentation; cancer
Test Species for Critical Effect	Human
RfD Basis	Inhalation
RfD Source	HEAST - EPA 1990b
Uncertainty Factor	
- Chronic RfD	1
- Subchronic RfD	1

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NS = Not specified.

Table 1B  
 CARCINOGENIC TOXICITY INDICES  
 FOR ARSENIC

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<u>Oral Route</u>	
Slope Factor (SF) (mg/kg-day) <sup>-1</sup>	NA
Weight of Evidence Classification	A
Type of Cancer	Skin
Test Species	Human populations
SF Basis	Water
SF Source	IRIS - EPA 1990a
 <u>Inhalation Route</u>	
Slope Factor (mg/kg-day) <sup>-1</sup>	50.0
Weight of Evidence Classification	A
Type of Cancer	Respiratory tract
Test Species	Human populations
SF Basis	Occupational air
SF Source	IRIS - EPA 1990a

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**Table 2**  
**STANDARDS AND CRITERIA**  
**FOR ARSENIC**

Standard or Criterion	Value	Reference
<u>Drinking Water</u>		
U.S. Primary Drinking Water Standards		
MCL (a)	0.05 mg/L	EPA 1990a
MCLG (b)	0.05 mg/L	EPA 1990a
<u>Surface Water</u>		
U.S. EPA Ambient Water Quality Criteria		
Ingesting Water and Organisms	$2.2 \times 10^{-6}$ mg/L	EPA 1990a
Ingesting Organisms Only	$1.75 \times 10^{-5}$ mg/L	EPA 1990a
<u>Occupational Air Concentrations</u>		
OSHA PEL TWA	10 $\mu\text{g}/\text{m}^3$	OSHA 1989
ACGIH TWA	0.2 $\text{mg}/\text{m}^3$	ACGIH 1989

(a) Maximum contaminant level  
(b) Maximum contaminant level goal

## BARIUM

Barium (Ba) is a naturally occurring silvery-white metal used industrially commercially in the form of Ba compounds, primarily as barite ( $\text{BaSO}_4$ ). Barite is used extensively as a weighting agent in oil and gas drilling fluids. Other major uses for barite are in the production of barium chemicals and in the glass, paint, and rubber industries. Barium concentrations have been monitored in various environmental media. In soils, barium concentrations range from 100 to 3,000 mg/kg, the median being about 500 ppm (Adriano 1986). Concentrations of barium in drinking water supplies generally range from  $<0.6$   $\mu\text{g/L}$  to approximately 10  $\mu\text{g/L}$ .

### Environmental Chemistry and Fate

In soils, barium is not expected to be very mobile because it forms water-insoluble salts and because it does not form soluble complexes with humic and fulvic materials (EPA 1985a). Under acidic conditions, however, some of the water-insoluble barium compounds may become more soluble and partitioning to groundwater may occur.

In aquatic media, barium is likely to be present as suspended particulate matter or sediments. In the absence of any other possible removal mechanism, the residence time of barium in aquatic systems would be several hundred years (EPA 1985a).

### Noncarcinogenic Effects

Acute exposure to barium in animals and humans results in a variety of cardiac, gastrointestinal, and neuromuscular effects (EPA 1985a).

The role of barium in the development of hypertension in experimental animals and humans is open to question. In deriving an oral reference dose (RfD), EPA identified a no-observed-adverse-effect level (NOAEL) reported in a study by Perry *et al.* (1983), in which mice chronically received  $5.1 \times 10^{-1}$  mg/kg/day of barium (EPA 1990b). EPA applied an uncertainty factor to the NOAEL to derive an oral RfD of  $5.0 \times 10^{-2}$  mg/kg/day (EPA 1990b). EPA has also derived an inhalation RfD of  $1.4 \times 10^{-4}$  mg/kg/day by application of an uncertainty factor of 1,000 to a subchronic no-observed-effect-level (NOEL) of 0.14 mg/kg/day identified in a study in rats (EPA 1990b).



### Reproduction and Development

No adequate mammalian studies on the potential reproductive or developmental effects of barium have been identified (EPA 1987).

### Mutagenicity and Carcinogenicity

No adequate studies on the mutagenicity and carcinogenicity of barium have been identified (EPA 1987). Based on the absence of data, EPA has placed barium in carcinogenicity category D, not classified (EPA 1987).

### Quantitative Indices of Toxicity

Quantitative indices of toxicity for barium are presented in Table 1.

Based upon the studies by Perry et al. (1983), EPA has derived an oral reference dose (RfD) and RfDs of  $5 \times 10^{-2}$  mg/kg/day. An inhalation RfD was calculated from the experiments by Tamsenko et al. (1977). The RfD is  $1 \times 10^{-4}$  mg/kg/day for chronic and  $1 \times 10^{-3}$  mg/kg/day subchronic.

### Standards and Criteria

Standards and criteria relevant to barium are listed in Table 2.

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Occupational Safety and Health Administration (OSHA), 1989, 29 CFR Part 1910, Air Contaminants, Final Rule, 54 Federal Register 2329-2984, January 19, 1989.

Table 1  
INDICES OF TOXICITY  
FOR BARIUM

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<u>Oral Route</u>	
Chronic Reference Dose (RfD) mg/kg/day	$5 \times 10^{-2}$
Subchronic RfD (RfDs) mg/kg/day	$5 \times 10^{-2}$
Confidence Level	Medium
Critical Effect	Fetotoxicity; increased blood pressure
Test Species for Critical Effect	Rat
RfD Basis	Drinking water
RfD Source	HEAST, IRIS (EPA 1990a,b)
Uncertainty Factor	
- Chronic RfD	100
- Subchronic RfD	100
 <u>Inhalation Route</u>	
Chronic RfD, mg/kg/day	$1 \times 10^{-4}$
Subchronic RfDs, mg/kg/day	$1 \times 10^{-3}$
Confidence Level	Medium
Critical Effect	Fetotoxicity; increased blood pressure
Test Species for Critical Effect	Rat
RfD Basis	Drinking water
RfD Source	HEAST, EPA 1990b
Uncertainty Factor	
- Chronic RfD	1,000
- Subchronic RfD	100

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Table 2  
STANDARDS AND CRITERIA FOR BARIUM

Standard or Criterion	Value	Reference
<u>Drinking Water</u>		
National Primary Drinking Water Regulations		
MCL (a)	1.0 mg/L (5.0 mg/L proposed)	EPA 1989
MCLG (proposed) (b)	1.5 mg/L	EPA 1989
<u>Occupational Exposure Limits (Air)</u>		
Barium-Soluble Compounds as Ba		
OSHA PEL TWA	0.5 mg/m <sup>3</sup>	OSHA 1989
ACGIH TWA	0.5 mg/m <sup>3</sup>	ACGIH 1989

a MCL = Maximum contaminant level.

b MCLG = Maximum contaminant level goal.

**BENZENE**

Benzene has a long history of industrial use. Most notably, benzene has found extensive use as a solvent and as a starting material for the synthesis of other chemicals. Currently, benzene is used as an octane enhancing additive to gasoline to replace alkyl lead compounds. Benzene has been detected in drinking water, food, and air. Based upon federal drinking water surveys, concentrations of benzene in approximately 1.3% of all groundwater systems are estimated to exceed 0.5 µg/L. The highest level reported in groundwater was 80 µg/L. Benzene has been reported in urban and suburban air at levels less than 30 µg/m<sup>3</sup> (10 ppb). Benzene also has been reported in indoor air at levels higher than in outdoor air.

**Environmental Chemistry and Fate**

The relevant physical and chemical properties and environmental fate of benzene (CAS No. 71-43-2) are summarized below (Howard 1989; MacKay and Shiu 1981):

Chemical Formula	C <sub>6</sub> H <sub>6</sub>
Molecular Weight	78.1 g/mole
Physical State at 20°C	Liquid
Water Solubility	1.79 E+03 mg/L (25°C)
Liquid Density at 20°C	.88 g/ml
Vapor Pressure	95.2 mm Hg (25°C)
Henry's Law Constant	5.43 x 10 <sup>-3</sup> atm·m <sup>3</sup> /mole
Octanol-Water Partition Coefficient (Log K <sub>ow</sub> )	2.13
Organic Carbon Partition Coefficient (K <sub>oc</sub> )	83 ml/g
Bioconcentration Factor (BCF)	5.2

Benzene has a high water solubility and vapor pressure. As a consequence of these two properties, benzene can be characterized as a highly mobile chemical. For benzene released to air, some rainwater washout is anticipated. After deposition in water or soil, volatilization is expected to return some portion back to the atmosphere. Based

upon benzene's high Henry's Law Constant, volatilization following release to water will result in substantial loss to the atmosphere.

Benzene's half-life in surface water has been estimated to range from 1 to 6 days, depending on the water temperature, depth, turbulence, and air movement over the water surface. Its half-life in air has been estimated to be 6 days or longer, with the major degradation mechanism being photooxidation.

Due to its high water solubility and high vapor pressure, transport to sediments is not expected to be a major surface-water fate process.

Benzene released to soil can be transported to air via volatilization, to surface water via runoff, and to groundwater via leaching. The first two pathways predominate in surficial soil, whereas the third pathway predominates at lower soil depths.

Benzene, like other low-molecular-weight organics, is subject to biooxidation in subsurface soil and groundwater. No estimates of the half-life in these media were found. Frequently, the availability of other nutrients such as nitrogen, phosphorus, sulfur, trace minerals, and oxygen is a rate-limiting factor in this process.

According to criteria developed by Kenaga (1980), benzene, with a  $K_{oc}$  of 83, would be considered mobile in soils. Other factors which influence soil mobility include soil type, amount of rainfall, depth to groundwater, and extent of degradation (ATSDR 1989).

Benzene is rapidly degraded in the atmosphere via reaction with the hydroxy radical. In soils and water, biodegradation is also an important process. In contrast to water, the biodegradation process in soil has not been well characterized. In this medium benzene metabolism proceeds through the formation of cis-dihydrodiols and with further oxidation, to catechols which are substrates for ring fission (Gibson, 1988; Hopper, 1978).

### Toxicokinetics

Benzene is readily absorbed via inhalation and ingestion, but poorly absorbed through skin (NIOSH 1974). If benzene behaves like other low-molecular-weight organics, it will be absorbed almost completely via ingestion. However, in humans, only about 50% of an inhaled dose is absorbed (Nomiyama and Nomiyama 1974a, b). Benzene is highly

lipid soluble and distributes preferentially to adipose tissue, bone marrow, and the liver (EPA 1983).

Metabolism occurs via oxidation to phenol, followed by conjugation, preferentially with sulfate, but also to glucuronic acid. The metabolites are excreted in the urine (EPA 1985a). Elimination of benzene from the body is biphasic. The initial rapid phase is due to exhalation of unchanged benzene, with a half-life of about 5 hours, while the remainder is excreted much more slowly as metabolites in the urine (Nomiyama and Nomiyama 1974a, b).

### **Noncarcinogenic Effects**

The main effects of brief exposure to high levels of benzene are drowsiness, dizziness, and headaches. These symptoms should disappear after exposure stops.

The best-known and longest-recognized toxic effect of benzene in humans is depression of bone marrow function. Benzene-exposed individuals have been found to display anemia, leucopenia, and/or thrombocytopenia (Kalf 1987; EPA 1987; ATSDR 1989). When simultaneous depression of all three cell types (pancytopenia) is accompanied by bone marrow necrosis, the syndrome is called aplastic anemia.

Benzene and its metabolites localize in the bone marrow, and it is widely agreed that the metabolites are primarily responsible for various hematotoxic effects.

The potential mechanisms for the development of pancytopenia in humans include the destruction of bone marrow cells, the impairment of the differentiation of these cells, or the destruction of more mature hematopoietic cell precursors and circulating cells (Goldstein 1977).

This conclusion is based upon studies which demonstrate that chemicals which alter benzene metabolism also alter benzene toxicity. For example, coadministration of agents such as toluene and phenobarbitol alleviate benzene toxicity, while agents such as ethanol increase benzene toxicity.

Specific benzene metabolites for which hematotoxic effects have been demonstrated experimentally include: benzene oxide, hydroquinone, phenol, catechol and trans, trans-mucondialdehyde.

### Carcinogenicity and Mutagenicity

Excess leukemia mortality, particularly acute myelogenous and monocytic leukemia, has been demonstrated among humans occupationally exposed to benzene (see for example, Aksoy 1985; Rinsky et al. 1981; Ott et al. 1981). According to its weight-of-evidence carcinogenicity criteria, EPA has classified benzene in Category A, "human carcinogen," based on sufficient evidence from epidemiological studies on man (EPA 1987). In addition to this definitive human evidence, several long-term bioassays have demonstrated increased incidences of tumors and leukemia following administration in animals.

Benzene has been tested extensively for genotoxic properties. Benzene was not mutagenic in several bacterial and yeast systems. Equivocal results have been reported for clastogenic results in vitro; several investigators have reported positive results in mouse micro-nucleus assays, as well as chromosomal observations in rabbits. Many investigators have reported significant increases in chromosomal aberrations in symptomatic and asymptomatic workers with either a current or past history of exposure to benzene (ATSDR 1989).

### Quantitative Indices of Toxicity

Table 1 summarizes the quantitative indices of toxicity for benzene.

Both carcinogenic and noncarcinogenic quantitative indices of toxicity have been derived for benzene. EPA has derived a slope factor (SF) for benzene from epidemiologic data from studies of workers occupationally exposed to benzene (Rinsky et al. 1981; Ott et al. 1978). The SF of  $0.029 \text{ (mg/kg/day)}^{-1}$  adopted by the EPA is an average value derived from the application of several mathematical carcinogenesis models (EPA 1990a, b).

EPA has derived an oral acceptable daily intake of 0.0007 mg/kg/day (EPA 1987) using data from Wolf et al. (1956) and leukopenia as the severest noncarcinogenic toxicological end point of concern.

### Standard and Criteria

Standards and criteria applicable to benzene are summarized in Table 2.



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Table 1B  
INDICES OF TOXICITY  
FOR BENZENE

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<u>Oral Route</u>	
Slope Factor (SF) (mg/kg-day) <sup>-1</sup>	2.9 x 10 <sup>-2</sup>
Weight of Evidence Classification	A
Type of Cancer	Leukemia
Test Species	Human
SF Basis	Occupational
SF Source	IRIS - EPA 1990a
<u>Inhalation Route</u>	
Slope Factor (mg/kg-day) <sup>-1</sup>	2.9 x 10 <sup>-2</sup>
Weight of Evidence Classification	A
Type of Cancer	Leukemia
Test Species	Human
SF Basis	Occupational
SF Source	HEAST; EPA 1985a, 1987a

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Table 2  
STANDARDS AND CRITERIA FOR BENZENE

Standard or Criterion	Value	Reference
<u>Slope Factor (SF)</u>	$2.9 \times 10^{-2} (\text{mg/kg/day})^{-1}$	EPA 1990a
<u>Drinking Water</u>		
National Primary Drinking Water Regulations		
MCL (a)	0.005 mg/L	EPA 1990a
MCLG (b)	0	EPA 1990a
<u>Surface Water</u>		
EPA Ambient Water Quality Criteria		
Drinking Water Only	0*	EPA 1986
<u>Occupational Air Concentrations</u>		
OSHA PEL TWA	$3.0 \text{ mg/m}^3$ (1.0 ppm)	OSHA 1989
OSHA STEL	$15 \text{ mg/m}^3$ (5 ppm)	OSHA 1989
ACGIH TLV TWA	$30 \text{ mg/m}^3$ (10 ppm)	ACGIH 1989

\*Recommended concentration in drinking water is 0; concentration of 0.67  $\mu\text{g/L}$  corresponds to the  $10^{-6}$  lifetime cancer risk value.

a MCL = Maximum contaminant level.

b MCLG = Maximum contaminant level goal.

## CADMIUM

Cadmium is a naturally occurring mineral distributed in trace amounts in the earth's crust. Many of the lead and zinc ores contain small amounts of cadmium and cadmium is produced as a by-product of lead and zinc smelting. The principal uses of cadmium are in the fabrication of alloys and solders, metal plating, as pigments, as stabilizers in plastic materials and in batteries.

Background concentrations of cadmium in surface waters are usually less than 1  $\mu\text{g/L}$ . Surface waters contain more than 2  $\mu\text{g/L}$  of cadmium have probably been contaminated by discharges of industrial wastes or by leaching from areas of landfill or from soils to which sewage sludge has been added (WHO 1984). Drinking water normally contains 1  $\mu\text{g/L}$  or less of cadmium. Higher levels of cadmium (up to 5  $\mu\text{g/L}$  in tap water may be associated with plated plumbing fittings, silver-based solders, and galvanized iron piping materials (CEC 1978). Cadmium has been detected in ambient air at concentrations less than 0.001  $\mu\text{g/m}^3$  in rural areas to 0.5  $\mu\text{g/m}^3$  in urban and industrialized areas (WHO 1984). Most food stuffs contain trace amounts of cadmium (less than 0.1 mg/kg wet weight). Typical dietary intakes range from 15 to 60  $\mu\text{g}$  of cadmium per day.

### Environmental Chemistry and Fate

The primary sources of atmospheric cadmium are combustion of coal and petroleum products. Cadmium from these sources is primarily adsorbed on small, highly respirable particles, which can be transported over large distances and transferred to other environmental compartments via wet and dry deposition. Cadmium adsorbed to small particulates is more persistent in the atmosphere than that adsorbed to larger particulates. Photochemical reactions are apparently not involved in the environmental fate of cadmium (ATSDR 1989).

Relative to other metals, cadmium is mobile in surface water. In natural waters, cadmium exists as a hydrated ion, metal-inorganic complexes with carbonate, hydroxyl, chlorine or sulfate anions; or as metal-organic complexes with humic acids (ATSDR 1989).

Because it exists only as the divalent cation, aqueous cadmium is not strongly influenced by the redox potential of water. However, under

reducing conditions forming sulfide, cadmium will precipitate in sediments as cadmium sulfide. The concentration of aqueous cadmium is usually inversely related to the pH value and the amount of organic material present (ATSDR 1989). Humic acid substances account for most of the organic complexes, with solubility dependent on the nature of the humic substance. Sorption by clays and iron oxides is important in reducing aquatic cadmium concentrations.

Cadmium concentrations are typically low in groundwater due to several factors. These factors include sorption by mineral matter and clay, binding to humic substances, precipitation as cadmium sulfide in the presence of sulfide, and precipitation as cadmium carbonate at high pHs.

In soil, cadmium may occur as free cadmium compounds or as the divalent ion dissolved in soil moisture. As a consequence of cation exchange, cadmium may be bound to soil minerals or organic constituents. The aerobic nature of topsoils tends to reduce the amount of cadmium bound to sulfide. High soil acidity favors release of the divalent cadmium cation and facilitates uptake by plants.

Cadmium is not reduced or methylated by microorganisms. However, the biological production of sulfide results in cadmium precipitation. Cadmium is strongly accumulated by all organisms, with typical concentrations in freshwater and marine organisms being hundreds to thousands of times higher than in water. Bioaccumulation of cadmium is strongly correlated with soil cation-exchange capacity (CEC), increasing with decreasing CEC. Bioconcentration in aquatic life is greatest for bottom feeders (e.g., mollusks and crustaceans), followed by fish and aquatic plants (ATSDR 1989). Bioaccumulation due to the use of cadmium-containing fertilizers on food crops has been noted in beef and poultry.

### **Toxicokinetics**

Cadmium ingested in food and water is poorly absorbed from the gastrointestinal tract with absorption efficiencies ranging from 1 to 6% depending on the chemical form and dose level of the cadmium and on the age, sex, and gastrointestinal contents of the receptor organism. Absorption of cadmium via the inhalation route depends on the size of the particles carrying the cadmium and how deeply they penetrate into

the lungs. No information on inhalation absorption was found for humans. However, values from 30 to 60% have been reported for animals.

Once absorbed, cadmium distributes to most tissues but it is preferentially accumulated in the kidneys and liver. Excretion is primarily via the urine and is very slow with a half-life in humans estimated to range from 17 to 38 years. The long half-life leads to a marked tendency for cadmium to accumulate in exposed organisms. Cadmium accumulation in the renal cortex leads to the characteristic renal injury that is the most common toxicological consequence of chronic exposure.

### **Noncarcinogenic Effects**

Acute and chronic exposure to cadmium in animals and humans results in renal dysfunction, hypertension, anemia, and altered liver microsomal activity. The kidneys are considered to be the critical target organ in humans chronically exposed to cadmium by ingestion. The early clinical signs of renal injury include proteinuria, glucosuria, and aminoaciduria.

To calculate a drinking water equivalent level (DWEL), USEPA used renal dysfunction as an endpoint, and the most widely accepted estimate for the critical (threshold) concentration of cadmium in the renal cortex--200  $\mu\text{g/g}$ . Using a 4.5% absorption of the daily dose and 0.01% excretion in the total body burden per day, USEPA calculated an lowest observed adverse effect level (LOAEL) of 352  $\mu\text{g/day}$  for renal effects in humans. Incorporating an uncertainty factor of 10, USEPA has developed a reference intake of 35  $\mu\text{g/day}$ . Adjusting the intake for consumption of 2 liters of water per day, USEPA has derived a DWEL of 18  $\mu\text{g/L}$  (EPA 1989).

Embryotoxic and teratogenic effects have been demonstrated in many mammalian species following parenteral administration of high doses of cadmium. In contrast, there is little evidence of these effects at lower doses by either of the more relevant inhalation or oral exposure routes (USEPA 1981; USPHS 1987).

### **Carcinogenicity and Mutagenicity**

Cadmium chloride aerosol administered by inhalation for 18 months produced lung tumors in rats. In contrast, all cancer bioassays in



which cadmium has been administered orally have been negative. Recent epidemiological studies indicated that workers chronically exposed to cadmium are at risk of elevated lung cancer mortality. According to its weight-of-evidence carcinogenicity criteria, USEPA has classified cadmium in Group B1 (probable human carcinogen) for inhalation based on the epidemiological data (USEPA 1986).

While the Agency has concluded that cadmium is a carcinogen by the inhalation route, USEPA has classified cadmium in Group D, inadequate evidence for carcinogenicity for the oral route of exposure, because of the negative results reported for cancer bioassays in which cadmium was administered orally (USEPA 1989). Consistent with this categorization, USEPA has proposed that the maximum contaminant level (MCL) for cadmium be set based upon noncarcinogenic toxicological endpoints (EPA 1989).

#### **Quantitative Indices of Toxicity**

Quantitative indices of toxicity for cadmium are presented in Table 1.

EPA has derived a reference dose (RfD) based upon the assumption that absorption of cadmium is 2.5% for food and 5% for water. Thus the toxicokinetic model predicts a NOAEL of .005 and .01  $\mu\text{g}/\text{kg}/\text{day}$  for water and food respectively. With a UF value equal to 10, an RfD of .005  $\mu\text{g}/\text{kg}/\text{day}$  for cadmium in water and an RfD of .001  $\mu\text{g}/\text{kg}/\text{day}$  for cadmium in food was established.

#### **Standards and Criteria**

Standards and criteria applicable to cadmium are summarized in Table 2.

The current MCL for cadmium, under the National Interim Primary Drinking Water Regulations, is 10  $\mu\text{g}/\text{L}$ . This level was designed to prevent renal dysfunction, and was based on a critical value of cadmium in the kidney cortex of 200  $\mu\text{g}/\text{g}$ , and assumptions on gastrointestinal absorption, excretion of the absorbed dose, daily excretion of the total body burden, and daily dietary cadmium intakes. The World Health Organization (WHO) guideline for drinking water is 5  $\mu\text{g}/\text{L}$ . This value was based on a value for provisional tolerable weekly cadmium intake, assuming that 25% of the total cadmium intake was attributable to drinking

water. USEPA has proposed a maximum contaminant level goal (MCLG) of 5 µg/L based upon the WHO guidelines and the National Academy of Science (NAS) recommendations (USEPA 1989).

The WHO's provisional tolerable weekly intake criteria of 0.0067 to 0.0083 mg/kg/week is used to evaluate dietary intake of cadmium by the Food and Drug Administration (FDA). Standards for cadmium concentrations in food are not currently available and cadmium in food surveys are not routinely conducted by Federal regulatory agencies. The FDA's contaminants team is proposing to develop criteria for evaluating inorganic contaminants in shellfish (FDA 1989).

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Table 1A  
 NONCARCINOGENIC INDICES OF TOXICITY  
 FOR CADMIUM

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<u>Oral Route</u>	
Chronic Reference Dose (RfD) mg/kg/day	
- Food	$1 \times 10^{-3}$
- Water	$5 \times 10^{-4}$
Subchronic RfD mg/kg/day	ND
Confidence Level	High
Critical Effect	Significant proteinuria
Test Species for Critical Effect	Human - renal cortex tissue
RfD Basis	Food and Water
RfD Source	IRIS - EPA 1990a
Uncertainty Factor	
- Chronic RfD	10
- Subchronic RfD	ND
Modifying Factor	
- Chronic RfD	1
- Subchronic RfD	ND

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ND = Not determined.

Table 1B  
INDICES OF TOXICITY  
FOR CADMIUM

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<u>Inhalation Route</u>	
Slope Factor (mg/kg-day) <sup>-1</sup>	6.1
Weight of Evidence Classification	B1
Type of Cancer	Respiratory tract
Test Species	Human
SF Basis	Occupational
SF Source	HEAST - EPA 1990b

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Table 2  
STANDARDS AND CRITERIA  
For Cadmium

Standard or Criterion	Value	Reference
<u>Drinking Water</u>		
National Primary Drinking Water Regulations	0.005 mg/L (p)	EPA 1989
Maximum Contaminant Level	0.010 mg/L	EPA 1989
Proposed Revised MCL	0.005 mg/L	EPA 1989
Maximum Contaminant Level Goal	0.005 mg/L (p)	EPA 1989
<u>Surface Water</u>		
EPA Ambient Water Quality Criteria		
Water Consumption Only	0.010 mg/L	EPA 1990a
<u>Occupational Air Concentrations</u>		
OSHA PEL TWA: Dust	0.2 mg/m <sup>3</sup> (f)	OSHA 1989
Fumes	0.1 mg/m <sup>3</sup> (f)	OSHA 1989
All forms	0.001/0.005 mg/m <sup>3</sup> (p)	OSHA 1989
ACGIH TWA	0.05 mg/m <sup>3</sup>	ACGIH 1989

(f) = final standard  
(p) = proposed standard

**CHLOROBENZENE**

Chlorobenzene is used as a chemical manufacturing intermediate and as a heat transfer medium. Industrial uses of chlorobenzene include in metal cleaning operations and as an organic solvent. Releases of chlorobenzene to the environment have occurred near industrial areas. The EPA reports that a maximum of 0.1% of groundwater sources of drinking water contain chlorobenzene levels between 0.5 and 5 µg/L. Chlorobenzene has been identified as a contaminant in air at very low levels (less than 1 ppb) in urban and suburban areas.

**Environmental Transport and Fate**

The relevant physical and chemical properties and environmental fate of chlorobenzene (CAS No. 108-90-7) are summarized below (EPA 1986a):

Molecular Formula	$C_6H_5Cl$
Molecular Weight	113 g/mole
Physical State at 20°C	Liquid
Water Solubility	466 mg/L (25°C)
Density	1.10 g/ml
Vapor Pressure	11.7 mm Hg (25°C)
Henry's Law Constant	$3.7 \times 10^{-3}$ atm·m <sup>3</sup> /mole (20°C)
Octanol-Water Partition Coefficient (Log $K_{ow}$ )	2.84
Organic Carbon Partition Coefficient ( $K_{oc}$ )	330 mL/g
Bioconcentration Factor (BCF)	10

Chlorobenzene's moderate water solubility, vapor pressure, and Henry's Law Constant indicate that volatilization is probably a major loss mechanism of chlorobenzene from surface water and surficial soils. A half-life range of 0.5 to 9 hours has been estimated for surface waters, depending on water temperature, depth, turbulence, and air movement across the water surface. A half-life in air has been estimated to be 3.5 days (EPA 1986).

Chlorobenzene's moderate water solubility, vapor pressure,  $K_{oc}$ , and

log  $K_{ow}$  values suggest that this compound will have a low to moderate mobility in the subsurface. Biodegradation of chlorobenzene in the subsurface is possible; however, limited data suggest that it is a slow process for this compound. Accordingly, chlorobenzene is estimated to have a soil half-life of several months.

Chlorobenzene has a moderate BCF and reportedly bioaccumulates in fish, aquatic invertebrates, and algae.

### Toxicokinetics

No specific data were found in the available literature on the absorption of chlorobenzene. However, based on the physical properties of the compound and extrapolation from related compounds, chlorobenzene would be expected to be nearly completely absorbed via the oral route and about 60% absorbed via inhalation (Astrand 1975; Dallas et al. 1983). In rats, chlorobenzene absorbed by inhalation was preferentially found in the adipose tissue, liver, and kidneys (Sullivan et al. 1983). Metabolism occurs via oxidation to p-chlorophenol, p-chlorocatechol, and p-chlorophenyl-mercapturic acid (Williams et al. 1975). At lower doses, chlorobenzene is excreted mainly as metabolites, including conjugation products, in the urine. However, as these pathways become saturated at higher doses, excretion of unchanged chlorobenzene in exhaled air increases (Sullivan et al. 1983).

### Noncarcinogenic Effects

Data on toxic effects in humans is sparse and is derived from incidents of accidental poisoning or occupational exposure in which the dose or even the causative agent may be unclear. Adverse effects reported include CNS depression, eye and respiratory irritation, and decreased blood cell counts.

In animals (mainly rats), increases in liver/body weight and kidney/body weight ratios, decreased body weight gains, and histopathological lesions in the liver, kidney, and lymphoid tissue have been reported (Monsanto 1980; Knapp et al. 1971; Hazelton 1967b; NTP 1985; Battelle 1978).

No data were found in the available literature on the reproductive effects of chlorobenzene.



Two studies in rats and rabbits in which pregnant females were exposed to chlorobenzene during the major organ-forming period failed to produce any evidence of teratogenicity, although some maternal toxicity (increased liver/body weight ratios) was noted at higher doses (John et al. 1984; Hayes et al. 1982).

Chlorobenzene has been shown to be mutagenic in some experimental systems, but not in others (EPA 1987).

### **Carcinogenicity and Mutagenicity**

Chlorobenzene was tested in the NTP bioassay program (NTP 1985). The results indicated a borderline significant increase in liver tumors in high-dose male rats. No increased tumor incidence in female rats or male or female mice was detected. According to its weight-of-evidence carcinogenicity criteria, EPA has placed chlorobenzene in Category D, "not classified."

### **Standards and Criteria**

Standards and criteria applicable to chlorobenzene are listed in Table 1.

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Table 1

**STANDARDS AND CRITERIA  
FOR CHLOROBENZENE**

Standard or Criterion	Value	Reference
<u>Reference Dose</u>		
Oral	$3 \times 10^{-2}$ (mg/kg/day)	EPA 1990
Inhalation	$5 \times 10^{-3}$ (mg/kg/day)	EPA 1990
<u>Surface Water</u>		
EPA Ambient Water Quality Criteria		
Water Ingestion Only	488 $\mu\text{g/L}$	EPA 1986
Freshwater Acute Aquatic Toxicity	488 $\mu\text{g/L}$	EPA 1986
<u>Occupational Air Concentrations</u>		
OSHA PEL TWA	350 $\text{mg/m}^3$ (75 ppm)	OSHA 1989
ACGIH TWA	350 $\text{mg/m}^3$ (75 ppm)	ACGIH 1989

## CHROMIUM

Chromium is a naturally occurring element that is used mainly for making steel and other alloys. Chromium compounds are also used in refractory brick for the metallurgical industry and in the chemical industry for metal finishing, manufacture of pigments, leather tanning, wood treatment, and water treatment. Larger amounts of chromium are emitted into the environment from the mining of ores containing chromium and the use of chromium compounds in industrial processes than from natural processes. The atmospheric chromium concentration in the United States is typically less than  $0.01 \mu\text{g}/\text{m}^3$  in rural areas and  $0.01$  to  $0.03 \mu\text{g}/\text{m}^3$  in urban areas (Fishbein 1984). The concentration of chromium in United States river waters usually ranges between  $1$  and  $30 \mu\text{g}/\text{L}$ . Chromium concentrations in soils are reported to range from  $5$  to  $1,500 \text{ mg}/\text{kg}$  (Carey 1982).

### Environmental Chemistry and Fate

The dominant chromium species in water and sediment are hexavalent chromium, or Cr(VI), and trivalent chromium, or Cr(III), both of which will partition between water and sediment. The principal chromium species which will enter into chromium equilibrium in natural water is  $\text{Cr}(\text{OH})_3$ . In soil, Cr(III) is the dominant species. Cr(VI) is soluble in water, existing in solution as a complex anion. Cr(III) may be soluble as an organic complex (Towill *et al.* 1978), depending on the availability of suitable complexing agents. Cr(VI) is reduced readily by organic matter to Cr(III), while Cr(III) may be oxidized slowly to Cr(VI) in ambient water (EPA 1985a). Cr(III) is likely to be precipitated as insoluble  $\text{Cr}(\text{OH})_3$  and deposited in sediment. In the pH range of natural water (5.0 to 9.0) and in the presence of chlorides, Cr(III) compounds are likely to precipitate as  $\text{Cr}(\text{OH})_3$ . Higher pH levels are likely to increase  $\text{Cr}(\text{OH})_3$  formation. Most of the Cr(III) present in surface water is present as particulate matter in sediment.

Most chromium is present in soil in a mineral state, either adsorbed or precipitated (EPA 1985a). Organic matter present in soil is likely to reduce any soluble chromate to insoluble chromium trioxide ( $\text{Cr}_2\text{O}_3$ ). The most common form of chromium in soil is Cr(III) as  $\text{Cr}_2\text{O}_3 \cdot n\text{H}_2\text{O}$  (EPA 1985a).

Chromium may be deposited in surface water and sediment by surface runoff, atmospheric deposition of chromium-containing particulate matter, and emissions from specific chromium sources (Versar 1979). Cr(VI), due to its solubility, is likely to be transported by water. Cr(III) may also be transported by water as suspended particulate matter or as bedload sediment.

Flooding of soils and the resulting anaerobic decomposition of plant matter may increase dissolution of  $\text{Cr}_2\text{O}_3\text{-nH}_2\text{O}$  in soil (Towill et al. 1978). Chromium is also transported from soil by runoff and may be transported to the atmosphere by aerosol formation (John et al. 1973).

No data were found to indicate that photolysis, volatilization, or sorption are important factors in the environmental fate and transport of chromium (EPA 1985a).

### Toxicokinetics

Absorption of chromium and chromium compounds is associated with the inhalation, oral, and dermal routes of exposure. Orally administered chromium in humans resulted in approximately 0.4% of the  $^{51}\text{Cr}$  (from  $^{51}\text{CrCl}_3$ ) and approximately 10.6% of the  $^{51}\text{Cr}$  (from  $\text{Na}_2\text{CrO}_4$ ) being absorbed (Donaldson and Barreras 1966). Dermal absorption of chromium is dependent on the chemical species, as well as on physical factors.

Chromium may be distributed throughout the body, with the highest concentrations found in the kidneys, lymph nodes, lungs, spleen, liver, and blood.

Chromium is metabolized by reduction of Cr(VI) to Cr(III) (ATSDR 1987). In vitro studies have shown that Cr(VI) is readily reduced to Cr(III) by gastric juices or glutathione (GSH) (Kitagawa et al. 1982; Levis et al. 1978).

Excretion of chromium occurs in multiple stages: a rapid phase representing clearance from the blood, and at least two slower phases representing clearance from tissues. Urinary excretion is the primary route of elimination (Cavalleri and Minoia 1985; Mertz et al. 1965).

### Noncarcinogenic Effects

Following inhalation, Cr(VI) is a respiratory tract irritant. Nasal irritation and slight transient effects on pulmonary function

occur at the lowest-observed-adverse-effect level (LOAEL). Ulceration and perforation of the nasal passages have been observed at concentrations of 20 to 46  $\mu\text{g}/\text{m}^3$  (ATSDR 1989). In vitro studies of chromium-induced toxicity following inhalation have indicated that the immune system may be affected. Depression of some indices of the immune system were reported at the higher concentrations, and stimulation was reported at lower concentrations. Hypersensitivity reactions have been reported in humans following inhalation exposure to chromium compounds (Glaser 1985; ATSDR 1989; Moller 1986).

Occupational exposure to chromium compounds by inhalation have resulted in changes in the kidney and liver. Reports of kidney damage, but not liver damage, following oral and dermal exposure to Cr(VI) compounds have been cited (ATSDR 1989).

### **Carcinogenicity and Mutagenicity**

Based on positive animal studies and positive epidemiological studies demonstrating excess cancer mortality in humans among Cr(VI)-exposed workers, and according to its weight-of-evidence carcinogenicity criteria, EPA has placed Cr(VI) in Category A, "human carcinogen" (EPA 1985a). However, since chromium has not been shown to be carcinogenic by the oral route, EPA has concluded that chromium in drinking water should be regulated based upon noncarcinogenic chronic toxicity data (EPA 1985a, b).

Cr(VI) has demonstrated consistently positive mutagenic activity in a number of bacterial systems. Both Cr(III) and Cr(VI) have been shown to interact with DNA in bacterial assays. Cr(VI) has inhibited DNA synthesis and increased unscheduled DNA synthesis in mammalian cells in culture. Both valences have been demonstrated to produce clastogenic effects in mammalian cells. Increased frequencies of chromosomal aberrations have also been observed in occupationally exposed workers.

### **Quantitative Indices of Toxicity**

Table 1 summarizes the quantitative indices of toxicity for chromium (III) and chromium (VI).

There are two chromium valences of principal concern in soil and drinking water: Cr(III) and Cr(VI). Cr(VI) is much more toxic than

Cr(III) and has been shown to produce liver and kidney damage, internal hemorrhages, and respiratory disorders. EPA has developed an adjusted acceptable daily intake (AADI) for total chromium based upon the effects of Cr(VI) for the following reasons (EPA 1985a):

- o The two valences are in dynamic equilibrium in aqueous media;
- o An AADI based upon Cr(VI), the more toxic of the two valences, will be more conservative (health-protective); and
- o Reduction of Cr(VI) to Cr(III) in the stomach following oral intake is incomplete and, relative to Cr(III), there is greater Cr(VI) absorption and tissue accumulation.

EPA derived the provisional AADI for total chromium based on a no-observed-adverse-effect level (NOAEL) reported in a study in which rats were exposed to Cr(VI) in drinking water for one year. Based on a NOAEL of 2.41 mg/kg/day, an uncertainty factor of 500, and standard intake and physiological assumptions, EPA derived an oral reference dose (RfD) of  $5.0 \times 10^{-3}$  mg/kg/day (EPA 1987, 1990a,b).

### Standards and Criteria

Standards and criteria applicable to chromium are summarized in Table 2.

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Table 1A  
 INDICES OF TOXICITY  
 FOR CHROMIUM (III)

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<u>Oral Route</u>	
Chronic Reference Dose (RfD) mg/kg/day	1.0
Subchronic RfD (RfDs) mg/kg/day	10
Confidence Level	Low
Critical Effect	Hepatotoxicity
Test Species for Critical Effect	Rat
RfD Basis	Diet
RfD Source	IRIS - EPA 1990a
Uncertainty Factor	
- Chronic RfD	100
- Subchronic RfD	100
Modifying Factor	
- Chronic RfD	10
- Subchronic RfD	NS

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Table 1B  
 INDICES OF TOXICITY  
 FOR CHROMIUM (VI)

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<u>Oral Route</u>	
Chronic Reference Dose (RfD) mg/kg/day	$5 \times 10^{-3}$
Subchronic RfD (RfDs) mg/kg/day	$2 \times 10^{-2}$
Confidence Level	Low
Critical Effect	Cancer - not defined
Test Species for Critical Effect	Rat
RfD Basis	Water
RfD Source	IRIS - EPA 1990a
Uncertainty Factor	
- Chronic RfD	500
- Subchronic RfD	100
Modifying Factor	
- Chronic RfD	1
- Subchronic RfD	NS

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Table 1C  
CARCINOGENIC INDICES OF TOXICITY  
FOR CHROMIUM (VI)

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Inhalation Route

Slope Factor (mg/kg-day) <sup>-1</sup>	41.0
Weight of Evidence Classification	A
Type of Cancer	Lung
Test Species	Rat
SF Basis	Occupational Air
SF Source	IRIS

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Table 2  
STANDARDS AND CRITERIA  
FOR CHROMIUM

Standard or Criterion	Value	Reference
<u>Drinking Water</u>		
National Primary Drinking Water Regulations		
MCL (a) present	0.05 mg/L	EPA 1989a
proposed	0.1 mg/L	EPA 1989a
MCLG (b) proposed	0.1 mg/L	EPA 1989a
<u>Surface Water</u>		
EPA Ambient Water Quality Criteria		
Water and Fish Consumption		
Cr(III)	179 mg/L	ATSDR 1989
Cr(VI)	50 µg/L	ATSDR 1989
<u>Occupational Air Concentrations</u>		
OSHA PEL TWA		
Chromates	0.1 mg/m <sup>3</sup> (ceiling)	OSHA 1989
ACGIH TLV TWA		
Chromium Metal	0.5 mg/m <sup>3</sup>	ACGIH 1989
Cr(II) and Cr(III)	0.5 mg/m <sup>3</sup>	ACGIH 1989
Water-Soluble Cr(VI) Compounds	0.05 mg/m <sup>3</sup>	ACGIH 1989
Certain Water-Insoluble Cr(VI) Compounds	0.05 mg/m <sup>3</sup>	ACGIH 1989
Chromite Ore Processing	0.05 mg/m <sup>3</sup>	ACGIH 1989

a MCL = Maximum contaminant level.

## CYANIDE

## Environmental Chemistry and Fate

The relevant physical and chemical properties and environmental fate for three representative cyanides (Clement Associates, Inc. 1985) are summarized below.

	<u>Hydrogen Cyanide</u>	<u>Potassium Cyanide</u>	<u>Sodium Cyanide</u>
CAS #	74-90-8	151-50-8	143-33-9
Chemical Formula	HCN	KCN	NaCN
Molecular Weight (g/mole)	27	65	49
Physical State at 20°C	Liquid		
Water Solubility	$1.0 \times 10^6$	$5.0 \times 10^5$	$8.2 \times 10^5$
Density at 20°C	.70	--	--
Vapor Pressure	$6.2 \times 10^2$	--	--
Henry's Law Constant	$1.2 \times 10^{-4}$	--	--
Log $K_{ow}$	$-2.5 \times 10^{-1}$	--	--
$K_{oc}$	0.66	-0.44	--
Bioconcentration Factor	0	0	0

Cyanides (CNs) are naturally occurring substances found in a number of foods and plants and are produced by certain bacteria, fungi, and algae. Minute amounts of cyanocobalamin (vitamin B-12) are a human dietary requirement (ATSDR 1989).

Cyanides have industrial uses as intermediates in a variety of processes. Widespread use contributes to the numerous sources from which cyanide is released to the environment.

Hydrogen cyanide (HCN) gas and soluble cyanide salts are the most mobile forms of cyanide in the environment (EPA 1984). Residence times in soil and water are variable and are influenced by such factors as pH, wind speed and cyanide concentration (Callahan *et al.* 1979).

A pH less than 9.2 is considered more conducive to environmental transport. Volatilization is considered a major fate process from surface water and surficial soils. Half-life estimates for atmospheric residence have been reported at approximately 334 days (Fritz *et al.* 1982). The long residence time results in widespread distribution

before removal by wet and dry deposition.

A number of common plants may bioaccumulate large quantities of cyanogenic glycoside. Hydrolyzation may occur upon ingestion, forming free HCN, resulting in toxicity.

In aquatic environments, hydrogen cyanide is not expected to undergo direct photolysis, to chemically hydrolyze, to adsorb significantly to suspended sediments, or to bioaccumulate significantly in aquatic organisms (Callahan et al. 1979). Biodegradation will occur unless cyanide concentrations are at levels toxic to microorganisms. High concentrations of cyanides in subsurface soils (i.e., landfills, spills) may leach into groundwater (EPA 1984).

### Toxicokinetics

Cyanides are readily absorbed via the inhalation, oral, and dermal routes of exposure. Inhalation of HCN is reportedly the most rapid route of entry resulting in the most rapid onset of toxic effects (ATSDR 1989).

Following absorption, cyanide is distributed throughout the body. Detoxification and excretion occur through the formation of thiocyanate, which is excreted in the urine. Small amounts of cyanide are also released through the lungs (ATSDR 1989).

Cyanide exerts its acute toxic effects through interference with the electron transport chain, preventing the utilization of oxygen by cells (ATSDR 1989).

### Noncarcinogenic Effects

Cyanide is acutely lethal at high concentrations (Dudley et al. 1942). Inhalation of 270 ppm HCN ( $300 \text{ mg/m}^3$ ) results in nearly immediate death, whereas inhalation of 135 ppm ( $150 \text{ mg/m}^3$ ) is fatal after 30 minutes of exposure. The human oral and dermal  $\text{LD}_{50}$ s have been estimated to be 1.52 mg/kg (EPA 1987) and 100 mg/kg (Reiders 1971), respectively. Symptoms of acute exposure to HCN include tachycardia accompanied by palpitation, vertigo, buzzing in the ears, headache, epigastric burning, vomiting, general weakness, tremor, sensory obtusion, dyspnea, and loss of consciousness (Carmelo 1955). The severity and rapidity of the onset of effects depends on the route, dose, and dura-

tion of exposure and the CN compound administered.

Neurotoxicity is the major target organ effect in cyanide poisoning. The nervous system is sensitive to cyanide toxicity partly because of its high metabolic oxygen demands. Symptoms of chronic exposure to CN in humans and laboratory animals include enlargement of the thyroid gland (Vanderlaan and Bissel 1946). The enlarged thyroid is thought to result from an iodine imbalance caused by thiocyanate, cyanide's principal metabolite.

Evidence of possible developmental effects following inhalation exposure to concentrations of CN below fetotoxic doses is not conclusive.

### **Carcinogenicity and Mutagenicity**

Cyanides have not been associated with carcinogenic effects in animals or humans. Mutagenicity studies have been negative or only slightly positive (ATSDR 1989).

### **Quantitative Indices of Toxicity**

The reference dose (RFD) for cyanide was based upon a dietary rat study by Howard and Hanzel (1955). This study provided the highest NOAEL, 10.8 mg/kg/day, and was chosen for the derivation of an RFD for Cyanide of 1.5 mg/day or .02 mg/kg/day. Quantitative indices of toxicity are summarized in Table 1.

### **Standards and Criteria**

Standards and criteria relevant to the cyanides are listed in Table 2.

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Table 1  
INDICES OF TOXICITY  
FOR CYANIDE

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<u>Oral Route</u>	
Chronic Reference Dose (RfD) mg/kg/day	$2 \times 10^{-2}$
Subchronic RfD (RfDs) mg/kg/day	$2 \times 10^{-2}$
Confidence Level	Medium
Critical Effect	Myelin degeneration, weight loss, thyroid effects
Test Species for Critical Effect	Rat
RfD Basis	DIET
RfD Source	IRIS - EPA 1990a
Uncertainty Factor	
- Chronic RfD	100
- Subchronic RfD	100
Modifying Factor	
- Chronic RfD	5
- Subchronic RfD	5

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Table 2  
STANDARDS AND CRITERIA  
For Cyanide

Standard or Criterion	Value	Reference
<u>Drinking Water</u>		
Lifetime drinking water health advisory	0.154 mg/L	EPA 1987
<u>Surface Water</u>		
EPA Ambient Water Quality Criteria Aquatic Organisms and Drinking Water	200 ug/L	EPA 1986
<u>Occupational Air Concentrations</u>		
OSHA PEL TWA	5 mg/m <sup>3</sup>	OSHA 1989
ACGIH	5 mg/m <sup>3</sup>	ACGIH 1989

## 1,2- and 1,3-DICHLOROBENZENES

Industrial uses of 1,2-dichlorobenzene (1,2-DCB) and 1,3-dichlorobenzene (1,3-DCB) include use as intermediates in chemical manufacturing, in metal polishing agents, and in industrial odor control. Environmental releases have resulted in the detection of 1,2 DCB and 1,3-DCB in various media. Surface water concentrations of 0.13 to 0.4 ppb have been reported.

### Environmental Transport and Fate

The relevant physical and chemical properties and environmental fate of 1,2-DCB (CAS No. 95-50-1) and 1,3-DCB (CAS No. 541-73-1) are presented below (Howard 1989; MacKay and Shiu 1981):

	<u>1,2-DCB</u>	<u>1,3-DCB</u>
Chemical Formula	$C_6H_4Cl_2$	$C_6H_4Cl_2$
Molecular Weight (g/mole)	147.0	147.0
Physical State at 20°C	Liquid	Liquid
Water Solubility (mg/L at 25°C)	156	111
Density at 20°C	1-3	1-3
Vapor Pressure (mm Hg at 25°C)	1.47	2.35
Henry's Law Constant ( $atm \cdot m^3/mole$ )	$1.20 \times 10^{-3}$	$1.8 \times 10^{-3}$
Octanol-Water Partition Coefficient ( $\log K_{ow}$ )	3.38	3.6
Organic Carbon Partition Coefficient ( $K_{oc}$ )	1,700	1,700
Bioconcentration Factor (BCF)	5	5

The  $\log K_{ow}$ s, high  $K_{oc}$ s, and low vapor pressures indicate that adsorption to soils is the major fate process of DCB isomers in soils. Adsorption to sediments will dominate the transport and fate of isomers discharged into aquatic media. Both isomers are heavier than water, causing DCB to sink in standing water.

The  $\log K_{ow}$ s suggest that DCB isomers will bioaccumulate. Biodegradation is not likely to be a significant degradation pathway for DCB isomers, based upon data which indicate that chlorobenzene is resistant

to biodegradation and that resistance increases with increasing chlorination of the benzene ring (ATSDR 1989). Very little information is available on the meta-isomer (1,3-DCB). Therefore, unless otherwise noted, the properties of this isomer will be assumed to be identical to those of the ortho-isomer (1,2-DCB).

### **Toxicokinetics**

The DCB isomers exhibit low water solubilities and high lipid solubilities (Neely *et al.* 1974). The absorption characteristics are assumed to be similar to those of benzene and the low-molecular-weight chlorinated aliphatics. As such, approximately 100% of an orally administered dose of DCB is absorbed, while approximately 30% of any DCB isomer inhaled over a period of one to several hours is absorbed and retained (Astrand 1975). In laboratory animals, following absorption, DCBs are primarily distributed to adipose tissue, and subsequently to the liver and kidneys (EPA 1985). Metabolism proceeds by oxidation of the parent compound, forming principally phenols and catechols. Further metabolism to form conjugates of glucuronic or sulfuric acids occurs as the major elimination pathway. Hawkins *et al.* (1980) studied the kinetics of excretion of  $^{14}\text{C}$ -p-DCB in rats. Most of the  $^{14}\text{C}$  activity (91 to 97%) was eliminated in the urine within 5 days after cessation of exposure, while small amounts were found in the feces and expired air. About 50 to 60% was excreted in the bile during the first 2 days, suggesting reabsorption in the enterohepatic circulation.

### **Noncarcinogenic Effects**

The principal toxic effects of 1,2-DCB in humans and other animals following acute and longer-term exposures include central nervous system (CNS) depression, blood dyscrasias, and lung, kidney, and liver damage. Similar data are not available for 1,3-DCB. However, EPA has determined that short-term assessments developed for 1,2-DCB should apply to 1,3-DCB (EPA 1985a, b).

### **Carcinogenicity and Mutagenicity**

The few studies available on the carcinogenic potential of DCBs have been negative or insufficient to clearly classify any DCB isomer as

carcinogenic. Preliminary results of a National Toxicology Program (NTP) stomach tube (gavage) bioassay indicate that 1,2-DCB was not carcinogenic under the conditions of the experiment. Pending receipt of the final NTP report for 1,2-DCB, and according to its weight-of-evidence carcinogenicity criteria, EPA has placed 1,2-DCB in Group D, "not classified" (EPA 1987d).

In general, DCBs have shown little or no mutagenic activity in a range of bacterial systems. However, several studies with mold and plant cultures treated with DCBs have reported mutations and chromosomal alterations (EPA 1987d).

### **Quantitative Indices of Toxicity**

EPA is in the process of establishing an enforceable MCL for 1,2-DCB, but not for 1,3-DCB. As a first step in the process, EPA has issued a proposed MCLG for 1,2-DCB based upon a no-observed-adverse-effect level (NOAEL) reported in a subchronic gavage study in mice and rats. Based upon a NOAEL of 125 mg/kg/day and an uncertainty factor of 100, EPA has derived a proposed MCLG for 1,2-DCB of 0.6 mg/L (EPA 1985b).

In the absence of sufficient data, EPA has not developed, and is not in the process of developing, a drinking water standard for 1,3-DCB.

### **Standards and Criteria**

Standards and criteria applicable to 1,2-DCB are presented in Table 1, and those applicable to 1,3-DCB are presented in Table 2.

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**Table 1**  
**STANDARDS AND CRITERIA FOR 1,2-DICHLOROBENZENE**

Standard or Criterion	Value	Reference
<u>Reference Dose</u>		
Oral	$9 \times 10^{-2}$ mg/kg/day	EPA 1990a
Inhalation	$4 \times 10^{-2}$ mg/kg/day	EPA 1990a
<u>Drinking Water</u>		
U.S. Primary Drinking Water Standards		
MCL (proposed)	0.6 mg/L	EPA 1989
MCLG (proposed)	0.6 mg/L	EPA 1989
<u>Surface Water</u>		
EPA Ambient Water Quality Criteria		
Adjusted for Drinking Water Only	470 $\mu$ g/L	EPA 1986
Aquatic Organisms and Drinking Water	400 $\mu$ g/L	EPA 1986
<u>Occupational Air Concentrations</u>		
OSHA PEL TWA	300 mg/m <sup>3</sup> (50 ppm) (Ceiling)	OSHA 1989
ACGIH TWA	300 mg/m <sup>3</sup> (50 ppm) (Ceiling)	ACGIH 1989

Table 2  
STANDARDS AND CRITERIA FOR 1,3-DICHLOROBENZENE

Standard or Criterion	Value	Reference
EPA Ambient Water Quality Criteria		
Ingestion of Aquatic Organisms and Water	400 µg/L	EPA 1986

**1,4-DICHLOROBENZENE (p-DCB)**

p-DCB is commonly used as an insecticide, and as a component of deodorant blocks used in restrooms. It is estimated that 70 million pounds of p-DCB are used each year in the manufacture of these two types of products. It is also used in the manufacture of certain resins; smaller amounts are used in the pharmaceutical industry and as a general insecticide in farm operations.

Home use of products containing p-DCB can result in release of large amounts to the indoor air, resulting in indoor air concentrations which are much higher than outdoor concentrations. A survey of select chemicals in indoor air, undertaken by the EPA Total Exposure Assessment Methodology (TEAM) Study (Wallace et al. 1986), reported mean personal air concentrations of 5.5 to 18  $\mu\text{g}/\text{m}^3$ . By contrast, outdoor mean concentrations ranged from 0.53 to 4.2  $\mu\text{g}/\text{m}^3$ . The TEAM Study by Wallace et al. (1986) reported on the combined occurrence of m- and p-DCB in drinking water in selected cities in New Jersey, North Carolina, and North Dakota. These DCB's were detected in  $\geq 3\%$  of the drinking water samples, with the reported concentration ranging from  $<1 \mu\text{g}/\text{L}$  to  $100 \mu\text{g}/\text{L}$ .

**Environmental Chemistry and Fate**

The relevant physical and chemical properties and environmental fate of p-DCB (CAS No. 106-16-7) are presented below (Howard 1989; MacKay and Shiu 1981):

Chemical Formula	$\text{C}_6\text{H}_4\text{Cl}_2$
Molecular Weight (g/mole)	147.0
Physical State at 20°C	Liquid
Water Solubility (mg/L at 25° C)	80
Density at 20°C	1.3
Vapor Pressure (mmHg at 25° C)	1.8
Henry's Law Constant ( $\text{atm}\cdot\text{m}^3/\text{mole}$ )	$2.89 \times 10^{-3}$
Log $K_{ow}$	3.6
$K_{oc}$	1,700
BCF	1.31

Little is known about the environmental fate of p-DCB. The high

log octanol/water partition coefficient suggests that adsorption to organic matter in sediments and soil is probably an important environmental fate process. Atmospheric transport of p-DCB occurs following volatilization. The estimated half-life for evaporation of p-DCB from surface waters is 9 hours or less (ATSDR 1989). The residence time for p-DCB in the atmosphere is estimated at more than 38 days. p-DCB appears to be fairly resistant to biodegradation and persistent in the environment. Bioaccumulation is estimated to be an important fate process. Sorption, bioaccumulation, and volatilization with subsequent atmospheric oxidation are likely to be competing processes, in which the dominant fate is determined by local environmental conditions (ATSDR 1989).

### **Toxicokinetics**

The dichlorobenzenes exhibit a low water solubility and are highly lipid soluble (Neely et al. 1974; EPA 1985). This property enables p-DCB to cross most of the barrier membranes, including those in skin, brain, and placenta. Quantitative absorption studies are not available and it is assumed that p-DCB possesses similar absorption characteristics to benzene and the lower molecular weight chlorinated aliphatics. As such, it is expected to be 100% absorbed when administered orally and about 30% absorbed via inhalation when exposure persists from 1 to 3 hours.

Laboratory animal experiments show that p-DCB absorbed following oral or inhalation exposure is distributed mainly to adipose tissue, with some distribution to the liver and kidney, and minor amounts to other organs. Absorbed p-DCB is metabolized mainly by oxidation to dichlorophenol, conjugated with glucuronic or sulfonic acid, and rapidly eliminated, mainly in the urine (ATSDR 1989).

### **Noncarcinogenic Effects**

The major target organs of p-DCB are the CNS, liver, and kidneys. The adverse effects on these tissues are reported in most of the available literature (ATSDR 1989).

CNS effects have been reported following inhalation of relatively high doses. Acute poisoning is characterized by signs of disturbance of

the CNS, including hyperexcitability, restlessness, and muscle spasms or tremors. The most frequent cause of death is respiratory depression (EPA 1987a).

Liver effects have been reported in animal studies following short-term, intermediate, and chronic exposure by the oral and inhalation routes. Findings typically include liver degeneration or necrosis, sometimes coincident with porphyria. Effects on liver enzymes have also been reported. In case studies in humans, cirrhoses and subacute or acute yellow atrophy of the liver have been diagnosed following inhalation exposure (EPA 1987; ATSDR 1987).

Adverse renal effects have generally been observed in the same sub-chronic and chronic studies in which hepatotoxicity has occurred. Reported effects ranged in severity from increased kidney weights to degeneration, mineralization, and hyperplasia of renal tissue (ATSDR 1987).

#### **Carcinogenicity and Mutagenicity**

No human data are available regarding the carcinogenicity of p-DCB. Positive studies for carcinogenicity have been obtained using animal models following oral administration, but not following long-term inhalation exposure. In the NTP (1987) 2-year carcinogenesis bioassay of p-DCB, there was clear evidence of carcinogenicity in male rats and in mice of both sexes.

The p-DCB was not mutagenic in microbial or mammalian systems (ATSDR 1989; Anderson 1976). The positive results in carcinogenicity testing of p-DCB combined with negative results in mutagenicity testing suggest that p-DCB may act as a tumor promoter rather than as an initiator in the carcinogenic process. Further studies are needed (ATSDR 1989).

#### **Quantitative Indices of Toxicity**

EPA has categorized p-DCB in group B-2: probable human carcinogen. EPA has derived an oral carcinogenic potency factor of  $2.4 \times 10^{-2}$  (mg/kg/day)<sup>-1</sup> based on an analysis of the NTP (1986) carcinogenicity data. A reference dose of  $1 \times 10^{-2}$  mg/kg/day has been calculated for p-DCB (EPA 1990b).

## Standards and Criteria

Standards and criteria relevant to 1,4-dichlorobenzene are listed in Table 1.

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Table 1  
STANDARDS AND CRITERIA  
For  
1,4-Dichlorobenzene

Standard or Criterion	Value	Reference
<u>Reference Dose</u>		
Inhalation	$1 \times 10^{-2}$ mg/kg/day	EPA 1990b
<u>Carcinogenic Potency Factor</u>		
Oral	$2.4 \times 10^{-2}$ (mg/kg/day) <sup>-1</sup>	EPA 1990b
<u>Drinking Water</u>		
U.S. Primary Drinking Water Standards		
MCL	0.075 mg/L	EPA 1987b
MCLG	0.075 mg/L (Op)	EPA 1987b
<u>Surface Water</u>		
U.S. EPA Ambient Water Quality Criteria		
Aquatic Organisms and Water Consumption	0.4 mg/L	EPA 1986
Adjusted for Water Consumption Only	0.47 mg/L	EPA 1986
<u>Occupational Air Concentrations</u>		
OSHA PEL TWA	$450 \text{ mg/m}^3$ (75 ppm)	OSHA 1989
ACGIH TWA	$450 \text{ mg/m}^3$ (75 ppm)	ACGIH 1989

MCL: Maximum Contaminant Level  
MCLG: Maximum Contaminant Level Goal  
P: Proposed

## 1,2-DICHLOROETHANE (ETHYLENE DICHLORIDE OR EDC)

EDC is a clear, volatile, and flammable synthetic organic liquid that is not found naturally in the environment. EDC is used primarily to make vinyl chloride and other solvents that remove grease, glue, and dirt. EDC is widely used and has been detected in ambient urban and rural air, and in indoor air samples of residences located near hazardous waste disposal sites. It has also been identified in surface water, groundwater, and drinking water. The average background concentration of EDC in ambient air at mid-latitudes has been reported to be 40 ppt (168 ng/m<sup>3</sup>). In urban areas concentrations ranged from 0.1 to 1.5 ppb (Singh et al. 1982). Concentrations of EDC in domestic surface waters used as drinking water sources have been reported to range from trace amounts to 4.8 µg/L (Brown et al. 1984). Concentrations in domestic groundwater supplies used for drinking water have been reported at concentrations up to 400 µg/L (Brown et al. 1984). No information was found on the concentrations of EDC in soil. It is expected that the lack of available soil monitoring data is due at least in part to rapid partitioning of EDC released from soils into ambient air and water.

### Environmental Chemistry and Fate

The relevant physical and chemical properties and environmental fate of 1,2-dichloroethane (CAS No. 107-06-2) are summarized below (Howard 1989; McKay and Shiu 1981):

Chemical Formula	C <sub>2</sub> H <sub>4</sub> Cl <sub>2</sub>
Physical State at 20°C	Liquid
Molecular Weight	99.0 g/mole
Density at 20°C	1.25
Water Solubility	8.52 x 10 <sup>3</sup> mg/L (25° C)
Vapor Pressure	78.7 mmHg (25° C)
Henry's Law Constant	1.1 x 10 <sup>-4</sup> atm-m <sup>3</sup> /mole (25°C)
Octanol-Water Partition Coefficient (Log K <sub>ow</sub> )	1.48
Organic Carbon Partition	

Coefficient ( $K_{oc}$ )	14 ml/g
Bioconcentration Factor (BCF)	1.2

Based on its high water solubility, high vapor pressure, and high Henry's Law Constant, volatilization would be expected to be the predominant removal mechanism for EDC from surface water and surficial soils. Its half-life in surface water has been estimated to range from 1 to 6 days, and its half-life in air has been estimated to be as short as 1.3 days due to photooxidation.

This compound is quite mobile in the subsurface. Its high vapor pressure, high vapor density, and high water solubility make vapor phase transport of this compound through the unsaturated zone quite likely. Its low octanol/water and organic carbon distribution coefficients indicate that it is only weakly adsorbed to soil particles and consequently only slightly retarded in its movement in groundwater.

Based on its low BCF, EDC is not expected to accumulate significantly in aquatic organisms.

### Toxicokinetics

EDC is absorbed by humans and laboratory animals following inhalation, ingestion, and dermal exposure. Reitz *et al.* (1980, 1982) found that  $^{14}\text{C}$ -EDC in corn oil given orally to rats (150 mg/kg) was completely absorbed by virtue of a complete recovery of radioactivity in exhaled air, urine, and carcass. Absorption following inhalation and dermal application were also significant routes of EDC entry into the body (EPA 1985).

Distribution studies have been conducted *in vivo* in rats. Forty-eight hours after a single oral dose, the highest EDC concentrations occurred in the liver and kidneys. EDC was also found in the forestomach, stomach, and spleen (Reitz *et al.* 1980).

Yllner (1971) reported on excretory pathways of EDC in mice following intraperitoneal injection. Eleven to 46% of the dose was excreted unchanged via the lungs, 5 to 13% of the dose was metabolized to carbon dioxide and water, and 50 to 73% of the dose was excreted as urinary metabolites.

### Noncarcinogenic Effects

EDC exhibits a moderate level of acute toxicity, as evidenced by median lethal doses of 489, 680, and 860 mg/kg for the mouse, rat, and rabbit, respectively (NIOSH 1983).

At relatively high doses, EDC produces central nervous system (CNS) depression as well as damage to the liver, kidneys, and adrenals. Symptoms of CNS depression typically include headache, dizziness, nausea, and general weakness. Effects on the liver include necrosis and epithelial cell damage, and effects on the kidneys include degeneration of the proximal tubule (EPA 1987).

In a multi-generation study in which male and female mice received doses of EDC in drinking water ranging from 0 to 50 mg/kg/day for 25 weeks, no reproductive or developmental effects were observed (Lane et al. 1982).

### Carcinogenicity and Mutagenicity

In an NCI-sponsored bioassay, EDC administered by stomach tube (gavage) was shown to produce statistically significant increased tumor incidences in both  $B_6C_3F_1$  mice and Osborne-Mendel rats (NCI 1978).

Male mice received EDC doses of 97 or 195 mg/kg/day and female mice 149 or 299 mg/kg/day for 78 weeks. Male and female rats received 47 or 95 mg/kg/day for 78 weeks.

Statistically significant increased tumor incidences of alveolar/bronchiolar adenomas were reported in both sexes of mice (NCI 1978). In addition, statistically significant increases in mammary adenocarcinomas and endometrial polyps or sarcomas were reported in female mice only (NCI 1978). Statistically increased incidence of squamous cell carcinomas of the forestomach and circulatory system hemangiosarcomas and mammary gland adenocarcinomas were reported in male and female rats, respectively (NCI 1978).

EDC has been shown to induce gene mutations in bacteria, plants, Drosophila melanogaster, and cultured Chinese hamster ovary cells (EPA 1985). In addition, EDC has been reported to cause meiotic chromosomal disjunction in Drosophila. Based upon these data and weight-of-evidence criteria, EPA has determined that EDC is a mutagen that may have the potential for causing adverse effects in humans (EPA 1985).

Based upon the above data, EPA has categorized EDC in group B<sub>2</sub>, "probable human carcinogen," according to its carcinogenicity guidelines (EPA 1987).

### Quantitative Indices of Toxicity

Due to insufficient data, EPA has not derived a reference dose for noncarcinogenic effects (EPA 1987, 1990a,b).

In a National Cancer Institute (NCI) report, EDC has been shown to elicit statistically significant increased tumor incidences in B<sub>6</sub>C<sub>3</sub>F<sub>1</sub> mice and Osborne-Mendel rats (NCI 1978). Based on these data, EPA has categorized EDC in group B<sub>2</sub>, "probable human carcinogen" (EPA 1987). Applying the linearized multistage model to the hemangiosarcoma incidence data in male rats, EPA has derived a 95% upper-bound estimate of the oral slope factor (SF) of  $9.1 \times 10^{-2} \text{ (mg/kg/day)}^{-1}$  (EPA 1990a). The inhalation SF is  $9.1 \times 10^{-2} \text{ (mg/kg/day)}^{-1}$  (EPA 1990a). Quantitative indices of toxicity are summarized in Table 1.

### Toxicity to Wildlife and Domestic Animals

EDC is one of the least toxic of the chlorinated ethanes to aquatic life. Acute toxicity has been reported in both fresh- and saltwater species, at concentrations greater than 118 mg/L, and chronic toxicity was observed at 20 mg/L (EPA 1984).

### Standards and Criteria

Standards and criteria relevant to 1,2-Dichloroethane are listed in Table 2.

### References

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**Table 1**  
**INDICES OF TOXICITY**  
**For 1,2-Dichloroethane**

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<u>Oral Route</u>	
Slope Factor (SF) (mg/kg-day) <sup>-1</sup>	9.1 x 10 <sup>-2</sup>
Weight of Evidence Classification	B2
Type of Cancer	Hemangiosarcoma
Test Species	Rat - National Cancer Institute Sponsored Bioassay
SF Basis	Gavage
SF Source	IRIS - EPA 1990a
<u>Inhalation Route</u>	
Slope Factor (mg/kg-day) <sup>-1</sup>	9.1 x 10 <sup>-2</sup>
Weight of Evidence Classification	B2
Type of Cancer	Hemangiosarcoma
Test Species	Rat
SF Basis	Oral exposure
SF Source	IRIS, EPA 1990a

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**Table 2**  
**STANDARDS AND CRITERIA**  
**For 1,2-Dichloroethane**

Standard or Criterion	Value	Reference
<u>Drinking Water</u>		
National Primary Drinking Water Regulations		
MCL (a)	0.005 mg/L	EPA 1987
MCLG (b)	0	EPA 1987
<u>Surface Water</u>		
EPA Ambient Water Quality Criteria		
Aquatic Organisms and Drinking Water	0 (0.94 ug/L)*	EPA, 1990a
<u>Occupational Air Concentrations</u>		
OSHA PEL TWA	4 mg/m <sup>3</sup>	OSHA 1989
OSHA STEL	8 mg/m <sup>3</sup>	OSHA 1989
ACGIH TLV TWA	40 mg/m <sup>3</sup> (10 ppm)	ACGIH 1989

\* The concentration given in parentheses for potential carcinogens corresponds to a risk of  $10^{-6}$ .

a MCL = Maximum contaminant level

b MCLG = Maximum contaminant level goal

**CIS-1,2-DICHLOROETHENE (cis-1,2-DCE)**

The dichloroethylenes are synthetic organic chemicals with no known natural sources. Cis-1,2-DCE's major use is as a captive intermediate in the manufacture of other chlorinated organic chemicals.

Environmentally, cis-1,2-DCE can also occur as the degradation product of trichloroethylene and tetrachloroethylene.

Monitoring studies have been undertaken to determine the concentration of cis-1,2-DCE in various media. Results indicate that cis-1,2-DCE is a relatively rare contaminant of groundwater. It was reported that the 1,2-dichloroethylenes have been usually found to co-occur with trichloroethylene.

**Environmental Chemistry and Fate**

The relevant physical and chemical properties (EPA 1986) and environmental fate of cis-1,2-DCE (CAS #540-59-0) are summarized below (Howard 1989; MacKay and Shiu 1981):

Chemical Formula	$C_2Cl_2H_2$
Molecular Weight	97.0 g/mole
Physical state at 20°C	Liquid
Water Solubility	3.500 mg/L (25°C)
Density	
Vapor Pressure	$2.00 \times 10^2$ mmHg (25°C)
Henry's Law Constant	$7.58 \times 10^{-3}$ atm-m <sup>3</sup> /mol (25°C)
Octanol-Water Partition Coefficient (Log $K_{ow}$ )	1.86
Organic Carbon Partition Coefficient ( $K_{oc}$ )	49 ml/g
Bioconcentration Factor (BCF)	1.6

The relatively high vapor pressure and high water solubility of cis-1,2-DCE indicate that its predominant loss mechanism from surface water and surface soils is likely to be volatilization. The half-life in surface water has been estimated to range from 1 to 6 days, depending on temperature, water turbulence, and air movement across the water surface. The evaporative loss from surface soils is expected to be in the

same range as the surface water half-life. The half-life of cis-1,2-DCE in air has been estimated to be less than 2 days (EPA 1987). Cis-1,2-DCE has been identified as a biodegradation product of trichloroethene and tetrachloroethene in groundwater.

### **Toxicokinetics**

Cis-1,2-DCE is a neutral, low-molecular weight, lipid soluble material which would be expected to be readily absorbed following exposure by any route (e.g., oral, inhalation, dermal) (EPA 1984). Distribution and elimination patterns for cis-1,2-DCE are predicted to be similar to 1,1-dichloroethene (1,1-DCE). As such, the highest concentrations are expected to be found in the liver and kidneys (McKenna *et al.* 1978). Elimination is expected to occur rapidly, with most of a single dose being excreted in the urine within 24 to 72 hours after exposure (Jaeger *et al.* 1977).

### **Noncarcinogenic Effects**

At high concentrations, the dichloroethylenes, like other chlorinated ethylenes, possess anesthetic properties. In fact, cis-1,2-DCE was used as an anesthetic agent, with some success prior to introduction of newer anesthetic gases (Irish 1963). In laboratory studies, the cis/trans DCE isomer mixture was reported to have an LC<sub>50</sub> of 770 mg/kg for rats (NIOSH 1978). No information was found in the available literature on the effects of long-term exposures to cis-1,2-DCE (EPA 1987).

### **Carcinogenicity and Mutagenicity**

No information was found in the literature on the carcinogenic potential of cis-1,2-DCE. Several studies have failed to detect any mutagenic activity on the part of cis-1,2-DCE, with or without microsomal activation (EPA 1987). Cis-1,2-DCE has been placed in EPA's weight-of-evidence of carcinogenicity category Group D: not classified. This category is for agents with inadequate evidence of carcinogenicity.

### **Quantitative Indices of Toxicity and Standards and Criteria**

Standards and criteria relevant to cis-1,2-DCE are listed in Table 1.

## References

- American Conference of Governmental Industrial Hygienists (ACGIH), 1989, Threshold Limit Values and Biological Exposure Indices for 1989-1990.
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TABLE 1  
STANDARDS AND CRITERIA  
FOR CIS-1,2-DICHLOROETHENE

Standard or Criterion	Value	Reference
<u>Reference Dose</u>		
Acceptable Daily Intake	0.01 mg/kg/day	EPA 1987
<u>Drinking Water</u>		
National Primary Drinking Water Regulations		
MCLG (Proposed)	0.07 mg/L	EPA 1986
<u>Occupational Air Concentrations</u>		
OSHA PEL TWA	790 mg/m <sup>3</sup> (200 ppm)	OSHA 1989
ACGIH TLV TWA	790 mg/m <sup>3</sup>	ACGIH 1989

**HEXACHLOROCYCLOHEXANE (HCH)**

HCH is a synthetic chemical that exists in eight chemical forms called isomers. These isomers may be identified as alpha-, beta-, delta-, and gamma-HCH. Gamma-HCH, commonly called Lindane, has been used as an insecticide and as a human pharmaceutical for the treatment of lice and scabies.

Technical-grade HCH, a mixture of several isomers, typically contains approximately 40% Lindane as well as the alpha-, beta-, delta-, and gamma-forms of HCH. Technical-grade HCH does not occur as a natural product and is no longer available in the U.S. for insecticide use. Also, Lindane has not been produced in the U.S. since 1977. Lindane is available in emulsifiable and flowable concentrates, soluble concentrates/liquids, wettable powders, dusts, ready-to-use liquids, pressurized liquids and impregnated materials, oil base and aerosol sprays, granules, and as a smoke generator (Berg 1988; EPA 1985a). Gamma-HCH is also sold separately or in combination with fungicides, fertilizers, other insecticides, or wood preservatives (Hayes 1982).

**Environmental Chemistry and Fate**

Physical and chemical properties relevant to the environmental fate and transport of HCH are summarized below (ATSDR 1989):

<u>Property</u>	<u><math>\alpha</math>-HCH</u>	<u><math>\beta</math>-HCH</u>	<u><math>\gamma</math>-HCH</u>	<u><math>\delta</math>-HCH</u>
CAS#	391-84-6	319-85-7	58-98-9	319-86-8
Chemical Formula	$C_6H_6Cl_6$	$C_6H_6Cl_6$	$C_6H_6Cl_6$	$C_6H_6Cl_6$
Molecular Weight	290.85	290.85	290.85	290.85
Physical State @ 20°C	monoclonic prism	crystalline solid	crystalline solid; monoclonic prism	fine plates
Water solubility	69.5 ppm	5 ppm	17 ppm	10 ppm
Vapor pressure @ 20°C (mmHg)	.02	.005	$4 \times 10^{-6}$	.02

Henry's Law Constant @ 25°C (ATM-m <sup>3</sup> /mol)	6.0x10 <sup>-6</sup>	4.5x10 <sup>-7</sup>	8.0x10 <sup>-6</sup>	2.1x10 <sup>-7</sup>
Organic Carbon Partition Coefficient [K <sub>oc</sub> (ml/g)]	3,800	3,800	1,080	6,600
Octanol/Water Partition Coefficient [Log K <sub>ow</sub> ]	3.81	3.78	3.61	4.14
Bioconcentration Factor (BCF) (L/kg)	130	130	130	130

HCH is a white solid with some tendency to volatilize into the air. The vapor is colorless and almost odorless (ATSDR 1989).

Lindane and  $\alpha$ -,  $\beta$ -, and  $\delta$ -HCH can be found in the air as a vapor or attached to small particles such as soil and dust.

#### Transport Processes

Gamma-HCH present in soil can lead to groundwater, sorbed to soil particles, or volatilize into the atmosphere. Based upon its moderate K<sub>oc</sub> value and a water solubility of 17 ppm (Hollified 1979), gamma-HCH is expected to leach slowly to groundwater. Gamma-HCH that has sorbed to the soil can be released to the atmosphere by wind erosion of surface soil particles. It is believed that windblown dust contributes substantially to the occurrence and long distance transport of pesticides in the atmosphere (ATSDR 1989). This is supported by monitoring studies performed by Stanley *et al.* (1971) which found that many pesticides are present in the atmosphere attached to particulates. Dust entrainment can lead to the transport of the pesticides from agricultural areas to the urban areas. Gamma-HCH can also enter the atmosphere via volatilization from treated agricultural soils and plant foliage (Lewis and Lee 1976). The widespread global distribution of HCH isomers is indicative of persistence of gamma-HCH in the air (ATSDR 1989).

### Degradation Process

Gamma-HCH can be present in the air as vapor or sorbed to particulate matter. Photodegradation and other degradation processes are not prominent in the removal of gamma-HCH from the air as compared to wash-out with precipitation or dry deposition. However, gamma-HCH undergoes photodegradation to form two isomers of tetrachlorohexene and pentachlorohexane in propanol solution when irradiated with ultraviolet light from a low pressure mercury lamp (Hamada et al. 1981). Similar transformations of gamma-HCH and other isomers are thought to occur to some extent in the atmosphere. Biodegradation is believed to be the significant degradative process for gamma-HCH in aquatic systems. Zoetemann et al. (1980) estimated river, lake, and groundwater half-lives for gamma-HCH from degradation data in these bodies to be 3-30 days, and >300 days, respectively. Gamma-HCH in soil and sediment is degraded primarily by biotransformation. Numerous studies conclude that gamma-HCH is transformed to tetrachlorohexene, tri-, tetra-, and pentachlorinated benzenes, penta-, and tetra-cyclohexanes, other isomers of HCH, and other related chemicals (Callahan et al. 1979).

### Environmental Concentrations

Air. Air monitoring during the years 1970-1972 in 10 states across the U.S. reported a mean gamma-HCH concentration in ambient air of 0.9 ng/m<sup>3</sup> for all years and all states combined (Kutz et al. 1976). Gamma-HCH was detected in 67.7% of all 2,479 samples, with a maximum concentration of 11.7 ng/m<sup>3</sup> reported (ATSDR 1989). A mean alpha-HCH concentration in ambient air of 1.2 ng/m<sup>3</sup> was also reported in positive samples (Kutz et al. 1976). Atlas and Giam (1981) measured the concentration of organic pollutants, including gamma-HCH, in the atmosphere and precipitation in the remote marine atmosphere. A total of 17 atmospheric samples and 16 rain samples were analyzed and yielded mean gamma-HCH concentrations of 0.015 ng/m<sup>3</sup> and 0.51 ng/l, respectively. A global distribution and atmospheric transport of chlorinated hydrocarbons study performed by Tanabe et al. (1982) confirmed the widespread distribution of HCH isomers. HCH residues were detected in all 79 air



and water samples collected. The range of concentrations were 1.1 to 2.0 ng/m<sup>3</sup> in air and 3.1 to 7.3 ng/l in water.

**Water.** Surface water concentrations of gamma-HCH have been measured in many areas across the U.S. Much of the available monitoring studies were conducted in the early to mid-1970s. A recent study in 1980-1981 was performed at the Niagara River near its entry into Lake Ontario. This study showed gamma-HCH detected in 99% of all samples at a mean concentration of 2.1 parts per trillion (ppt) (Kuntz and Warry 1983). Gamma-HCH has been detected in groundwater at a median concentration of 16 ppt in Chesterfield County, South Carolina and 163 ppt in Hampton, South Carolina (Sandhu et al. 1978). Gamma-HCH has also been detected in drinking water from Cincinnati, Ohio (Keith et al. 1976), Hampton, South Carolina (Sandhu et al. 1978), and Oahu, Hawaii (Bevenue et al. 1972) at mean concentrations of 0.01 ppt, 10 ppt, and 0.2 ppt, respectively. In addition, rain and snow water in Portland, Oregon in 1982, gamma-HCH was detected at mean concentrations of 0.45 to 11 ppt (Pankow et al. 1984).

**Soil.** The United States Soils Monitoring Program showed that gamma-HCH was not detected in cropland soils or crops in 37 states during the summer and fall of 1971 (Carey et al. 1978). The minimum detection levels were 0.002 to 0.03 ppm. Gamma-HCH concentrations in the surface soils of five western Alabama counties were less than 0.1 ppm (Albright et al. 1974). Most of the sediment monitoring data for gamma-HCH are derived from the Great Lakes. The average concentration for the Niagara River was 2 ppb (Kuntz and Warry 1983), the Lake Ontario sediments contained 2.4 ppb (Oliver and Charlton 1984) and Lake Michigan sediments contained less than 2.5 ppt to 0.15 ppt (Schacht et al. 1974).

#### **Human Exposure and Body Burdens**

The most probable route of non-medicinal human exposure to gamma-HCH is ingestion of food or water containing the pesticide. The average daily dietary intake has been estimated to be 4 µg/kg/day (Duggan et al. 1983). Gamma-HCH can travel through the food chain. Human exposures can result from the ingestion of plants, animals, animal products, milk,

and water containing pesticide (ATSDR 1989). The National Human Adipose Tissue Survey (NHATS) in 1982 showed that beta-HCH detected in 87% of 46 composite samples at <math>19-570 \mu\text{g/g}</math> concentrations (Stanley 1986). These concentrations were detected most often in post-mortem samples collected from southern U.S. citizens.

Human body burdens of HCH may be influenced by factors such as age, dietary habits, and residence. Saxena et al. (1981a) found that non-vegetarian women between the ages of 26 and 34 who live in a rural area tend to show higher body levels of gamma-HCH than other women. The higher levels of gamma-HCH in women at an older child-bearing age suggest that a longer life span may cause a greater accumulation of pesticide in the body (ATSDR 1989). In addition, studies indicate that gamma-HCH is also present in human milk at an average level of 0.006 ppm in Alberta, Canada (Currie et al. 1979). Takahaski et al. (1981) demonstrated that of 50 breast milk donors in Oahu, Hawaii, 82% of the samples showed HCH at a mean level of 81 ppb as extractable lipid with a range of 0-480 ppb.

### Toxicokinetics

Absorption of HCH following oral exposure have lead to many accidental poisonings in humans (Berry et al. 1987; Nantel et al. 1977; Harris et al. 1969; Khare et al. 1977; Munk and Nantel 1977; Powell 1980; Starr and Clifford 1972). Information concerning the rate of absorption from the gastrointestinal tract (G.I. tract) is inferred from studies conducted in mice and rats.

Gamma-HCH is readily absorbed from the G.I. tract (Aldaya et al. 1981; Turner and Shanks 1980) and then absorbed from the intestine into the blood. Turner and Shanks (1980) showed that only a small amount of gamma-HCH actually entered the lymphatic system from the intestine. The rapid absorption and excretion of gamma-HCH may be attributed to its unusually high solubility in water.

Evidence exists that humans absorb gamma-HCH vapor or dusts via inhalation. Occupational studies have shown adverse health effects, including hematological abnormalities and neurological effects, as a result of worker exposure to gamma-HCH in workplace air (Brassow et al. 1981; Czelgledi-Janko and Arar 1970; Kashyap 1986; Samuels and Milby

1971). However, no specific studies have quantified the rate or extent of absorption of the HCH isomers following inhalation exposure.

The ready absorption of gamma-HCH through human skin has been demonstrated in several studies that examined the usage of antiscabies lotion (Feldmann and Maibach 1974; Grinsburg et al. 1977; Lange et al. 1981). The maximum levels of gamma-HCH reached in scabies patient were greater than those reported for normal volunteers. Studies involving topical application of gamma-HCH to the forearm indicated that at least 9% of an applied dose is readily absorbed and that maximum absorption occurs 2-3 days after application (Feldmann and Maibach 1974).

### Distribution

HCH residues are quite lipophilic and are distributed throughout the body tissues in proportion to their lipid content. The overall distribution of gamma-HCH is greatest in fat followed by brain, kidney, muscle, lungs, heart, spleen, liver, and blood (Srinivasan and Radhakrishnamurty 1983b).

### Metabolism

Gamma-HCH appears to be rapidly transformed by hepatic enzymes to its primary metabolites, chlorophenols, and chlorobenzenes (Chadwick et al. 1978a; Chadwick and Freal 1972a; Engst et al. 1979; Kujawa et al. 1977). Other urinary metabolites identified include other trichlorophenols, dichlorophenols, tetrachlorophenols, and dihydroxy-chlorobenzenes. Following occupational exposure, pentachlorophenol has also been identified as a urinary metabolite in humans (Engst et al. 1979). In vitro studies have established that an epoxide forms during the metabolism of pentachlorocyclohexane, a primary metabolite formed by dehydrogenation of gamma-HCH, which may be responsible for the mutagenic and carcinogenic effects of gamma-HCH (Fitzloff and Pan 1984).

### Excretion

The major route of excretion of HCH in humans appears to be in the urine, but some excretion also occurs via breast milk and semen (ATSDR 1989). Very little HCH is eliminated in the exhaled air (Ahdaya et al. 1981; Chadwick et al. 1985) and in the feces (Chadwick et al. 1985).

### Noncarcinogenic Effects

The major noncarcinogenic effects of HCH appear to involve the nervous system, the liver, the blood, and reproduction and development of offspring.

### Neurological Effects

In humans, the nervous system appears to be one of the primary target systems for HCH toxicity. Most of the information is from case reports of acute gamma-HCH poisoning. Seizures and convulsions have been observed in individuals who have accidentally or intentionally ingested gamma-HCH as insecticide pellets, liquid scabicide, or contaminated food (Davies et al. 1983; Harris et al. 1969; Munk and Nantel 1977; Nantel et al. 1977; Powell 1980; Starr and Clifford 1972). These symptoms were observed in rats following a single intragastric administration of approximately 60-150 mg/kg (Tilson et al. 1987; Tusell et al. 1987).

Paresthesia of the face and extremities, headache, and vertigo have been reported in a group of 45 workers occupationally exposed to technical-grade HCH for prolonged periods of time (Kashyap 1986). Abnormal EEG patterns have been reported in 16 of 37 workers following exposure to gamma-HCH for 0.5 to 2 years in a fertilizer plant (Czegledi-Janko and Arar 1970). However, exposure concentrations were not reported; these EEG changes were found to correlate with blood levels of gamma-HCH.

### Hepatic Effects

In humans, technical-grade HCH exposure caused significant increases in the blood levels of the enzymes lactate dehydrogenase, leucine aminopeptidase, and gamma-glutamyl transpeptidase in 19 individuals in a HCH formulating plant (Kashyap 1986). Experimentally, gamma-HCH was reported to increase liver microsomal activity in Osborne-Mendel rats exposed to 6.2 and 13.3  $\mu\text{g}/\text{kg}/\text{day}$  and in CF1 and B6C3F1 strain mice exposed to 16.1 and 34.6  $\mu\text{g}/\text{kg}/\text{day}$  in the diet for 3 days (Oesch et al. 1982). Similar effects were also shown in experiments with alpha-HCH (a mixture of the alpha, beta, gamma and delta-HCH

isomers, was reported to cause fatty degeneration and necrosis of the liver in rats exposed to 2.5 µg/kg/day for 33-61 weeks (Fitzhugh et al. 1950); these effects were more apparent at 40 µg/kg/day. Technical-grade HCH was also reported to cause liver cancer in mice following exposure to 65 µg/kg/day in the diet for 2-8 months (Thakore et al. 1981; Karnick et al. 1981) and exposure to 0.3-13 µg/kg/day for 32-80 weeks (Kashyap et al. 1979; Munir et al. 1983).

### **Immunological Effects**

A significant increase (approximately 18%) in the level of immunoglobulin M (IgM) was noted in 19 workers occupationally exposed to technical-grade HCH during pesticide formulation (Kashyap 1986).

Some evidence of possible immunotoxic effects of gamma-HCH is available from animal studies. Immunosuppression was reported in rats exposed by garage to 6.25 and 25 µg of gamma-HCH/kg/day for five weeks (Dewan et al. 1980) and in rabbits exposed by capsule five times each week to 1.5, 6 and 12 µg/kg/day for 5-6 weeks (Desi et al. 1978).

### **Hematological Effects**

Hematological effects have been reported in humans following acute or long-term inhalation exposure to gamma-HCH. Hypochronic anemia was discovered in a 2.5-year-old boy who was exposed to gamma-HCH in a home due to operation of an HCH vaporizer. Air concentrations measured in the basement and living room of the house were 2.4 to 5.5 µg/m<sup>3</sup> (Morgan et al. 1980). Other abnormalities have been reported as result of chronic human occupational exposure including instances of leukopenia, leukocytosis, granulocytopenia, granulocytosis, eosinophilia, monocytosis, and thrombocytopenia (Brassow et al. 1981; Jedlicka et al. 1958; Samuels and Milby 1971). Aplastic anemia and bone marrow hyperplasia have been reported in a woman as a result of excessive dermal exposure to gamma-HCH when bathing her dog in a 2% solution once a week for years (Woodliff et al. 1966). In addition, reduced hemoglobin and hematocrit values and near complete absence of red blood cell precursors in bone marrow were reported in a 2-year-old boy exposed to a dog that was dipped regularly in mange containing 12% gamma-HCH (Vodopick 1975).

## Standards and Guidelines

Standards and guidelines pertinent to HCH are summarized in Table 1.

## Reproductive and Developmental Effects

Statistically significant increases in levels of serum luteinizing hormone (LH) were reported in a group of 54 men after occupational exposure to gamma-HCH for approximately 8 years in a Lindane-producing factory (Tomczak *et al.* 1981). Ovarian and uterine weight increases and atrophy were also associated with exposure to HCH. A 13-week exposure study to 0.5  $\mu\text{g}$  beta-HCH/kg/day in rats fed 12.5  $\mu\text{g}$ /kg/day displayed these effects (Van Velsen *et al.* 1986). Exposure to 12.5  $\mu\text{g}$ -HCH/kg/day resulted in severed effects in male rats including degeneration of somniferous tubules and disruption of spermatogenesis (Van Velsen *et al.* 1986).

Gamma-HCH has not been reported to cause developmental effects in animals.

## Genotoxic Effects

No increase in the frequency of chromosome aberrations was observed in humans exposed to HCH primarily by inhalation in a pesticide production factory following an exposure of 8 hours/day for at least 6 months (Kiraly *et al.* 1979). No other studies were located pertaining to genotoxicity of HCH to humans (ATSDR 1989). In animals, ingestion of tech-grade HCH was reported to induce dominant lethal mutations in mice (Lakkad *et al.* 1982). In rats, exposure to alpha-HCH was reported to result in mitotic disturbances including an increased mitotic rate and frequency of polyploid hepatic cells (Hitachi *et al.* 1975).

## Carcinogenic Effects

No studies were located regarding the carcinogenicity in humans following exposure to gamma-, alpha-, beta-, delta-isomers of HCH, or technical-grade HCH by any route. Alpha-, beta-, gamma-, and technical-grade HCH have been found to be carcinogenic in rats and/or mice following long-term exposure (Hanada 1973; Ito *et al.* 1973, 1975, 1976;

Kashyap et al. 1979; Munir et al. 1983; NCI 1977; Thakore et al. 1981; Thorpe and Walker 1973; Tsukada et al. 1979; Wolff et al. 1987).

Hepatocellular carcinoma is the most frequently reported tumor type, although in many studies the liver was the only organ under investigation (ATSDR 1989). The available animal data suggest that liver cancer may be of potential concern to individuals exposed to HCH for prolonged periods of time.

Table 1  
STANDARDS AND CRITERIA  
FOR HCH

Standard or Criterion	Value	Reference
<u>Reference Dose</u>		
Oral	$3 \times 10^{-4}$ mg/kg/day	EPA 1990
<u>Carcinogenicity Potency Factor</u> (mg/kg/day) <sup>-1</sup>		
Inhalation	$\alpha$ -HCH            6.3 $\beta$ -HCH             1.8 technical-HCH    1.8	EPA 1990
<u>Drinking Water</u>		
National Primary Drinking Water Regulations		
Maximum Contaminant Level (MCL) [Lindane]	.004 mg/L	EPA 1975 (40 CFR 141)
MCL (proposed) [Lindane]	.0002 mg/L	EPA 1985c
<u>Food</u>		
EPA Tolerances	Range:	
	.01 ppm	EPA 1974
	7.0 ppm (pecans)	(40 CFR 180.133)
	1 or 3 ppm (fruits and vegetables)	
<u>Ambient Water (Aquatic Organisms) [Lindane]</u>		
Fresh Water:	Acute            2.0 $\mu$ g/L	EPA 1986
	Chronic        0.8 $\mu$ g/L	
	(24 hr. avg.)	
Marine:	Acute            16 $\mu$ g/L	
	Chronic        --	
	(24 hr. avg.)	

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Table 1 (Cont.)

Standard or Criterion	Value	Reference
<u>Ambient Air</u>		
No Ambient air quality criteria established		
<u>Occupational Air Concentrations [Lindane]</u>		
ACGIH TLV TWA (a)	.5 mg/m <sup>3</sup> (skin)	ACGIH 1986
OSHA PEL TWA (b)	.5 mg/m <sup>3</sup> (skin)	OSHA 1989
NIOSH REL TWA (c)	.5 mg/m <sup>3</sup>	NIOSH 1985
NIOSH IDLH (d)	1,000 mg/m <sup>3</sup>	NIOSH 1985

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- a: American Conference of Governmental Industrial Hygienists, Threshold Limit Value, Time Weighed Average.
- b: Occupational Safety and Health Administration, Permissible Exposure Limit, Time Weighted Average.
- c: National Institute for Occupational Safety and Health, Recommended Exposure Limit, Time Weighted Average.
- d: National Institute for Occupational Safety and Health, Immediately Dangerous to Life or Health Level.

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## LEAD

Lead (Pb) is a naturally occurring metal which is used in such processes as the manufacture of storage batteries and production of ammunition, and in miscellaneous metal products (e.g., sheet lead, solder, and pipes) and various chemicals, including gasoline additives. Lead has been monitored in the ambient air, surface waters, ground waters, and soils. Levels of lead in ambient air range from  $7.5 \times 10^{-5} \mu\text{g}/\text{m}^3$  in remote areas to more than  $10 \mu\text{g}/\text{m}^3$  near stationary sources such as smelters. Levels of lead in surface waters throughout the United States typically range between 5 and 30  $\mu\text{g}/\text{L}$ , although levels as high as 890  $\mu\text{g}/\text{L}$  have been monitored (EPA 1986a). The lead content of soil ranges from 10 to 30  $\mu\text{g}/\text{gm}$  in soils originating from crustal rock to 10,000  $\mu\text{g}/\text{g}$  in soils adjacent to roadways (EPA 1986a).

### Environmental Chemistry and Fate

Lead and its inorganic compounds exist in the atmosphere mainly as particulates that can be removed from the atmosphere by either wet or dry deposition. Particle size and atmospheric turbulence are the primary factors determining the half life of particulate lead in the atmosphere. Lead also readily undergoes photolysis in the atmosphere.

The movement of lead in aquatic environments is influenced by lead speciation. In water with high concentrations of dissolved organics, complexation is an important mechanism for retaining lead in solution. In waters without substantial dissolved organics, lead can become adsorbed to suspended particulates and eventually settle out.

Sorption processes exert dominant effects on the distribution of lead in aquatic and terrestrial environments. Adsorption to inorganic solids, organic materials, hydrous iron, and manganese oxides controls the mobility of lead in soils and sediments.

The dominant sorption mechanisms are dependent on geological setting, pH, Eh, availability of ligands and chemical composition. Additional factors operative in sediments include dissolved and particulate ion concentrations and salinity. The solubilities of lead carbonate, sulfate, and sulfide are low. Over most of the pH range, lead carbonate and lead sulfate control solubility. Lead is strongly complexed to organic materials present in aquatic systems and soil. Lead in soils is

not easily taken up by plants, so the availability of lead to terrestrial organisms by this route appears to be limited (EPA 1982).

Bioaccumulation of lead has been demonstrated for a variety of organisms, with bioconcentration factors typically ranging from 42 to 1,700 (EPA 1986).

### Toxicokinetics

The rate and degree of absorption of chemicals are largely related to their solubility in body tissues and fluids. Each lead compound has unique solubility characteristics, resulting in compound-specific differences in absorption.

Prior to absorption of airborne lead by the lungs, some fraction of inhaled lead must be deposited in the respiratory tract. The rate of deposition of airborne particulate lead in adults ranges from 30 to 50% and is modified by such factors as particle size and deposition rate (EPA 1986). Relatively little is known about deposition of airborne lead in children. Once deposited in the lower respiratory tract, lead is almost completely absorbed (EPA 1986). Particulate lead deposited in the upper respiratory tract is cleared from the tract by ciliary action but may then be swallowed.

The primary site of lead absorption in children is the gastrointestinal tract (Hammond 1982). For dietary lead, absorption is approximately 50% in children and 5 to 15% in adults (Goyer 1986). The physical and chemical properties of the ingested lead and a number of dietary factors such as nutritional status and the composition of the diet affect the extent and rate of gastrointestinal absorption of lead.

Dermal absorption of inorganic lead compounds is much less significant than absorption due to inhalation or oral exposure (Goyer 1986).

The distribution of lead in the body is initially dependent on the rate of delivery by the bloodstream to various organs and tissues. However, lead may subsequently redistribute based upon its relative affinity with various tissues. In humans, roughly 95% of the total body burden of lead is found in the bones (ATSDR 1989).

In humans, any dietary lead not absorbed by the gastrointestinal tract is eliminated in the feces. Airborne lead that has been swallowed and not absorbed is also eliminated in the feces. Absorbed lead is

excreted either by the kidneys or into the gastrointestinal tract via the bile (ATSDR 1989).

### Noncarcinogenic Effects

When toxicological information is considered in assessing the non-carcinogenic effects of substances, the data are evaluated in order to identify an exposure level below which no adverse effects are observed. Historically, the observed threshold, or no-effect level, for lead-induced toxic effects has continued to decline as increasingly sophisticated experimental and clinical measures are employed to detect the more subtle effects. These include alterations in physiological functions at blood lead (PbB) levels below the currently accepted maximum safe level for exposure to children, the segment of the population currently regarded to be at highest risk of lead-induced effects (EPA 1985a, ATSDR 1989).

The most serious effects associated with markedly elevated PbB levels are severe neurotoxic effects that include irreversible brain damage. For most adults, such damage does not occur until PbB levels exceed 100 to 120 micrograms per deciliter ( $\mu\text{g}/\text{dl}$ ). At these PbB levels, severe gastrointestinal symptoms and effects on several other organ systems are often found (Kehoe *et al.* 1961a). Precise thresholds for occurrence of overt neurological and gastrointestinal signs and symptoms of lead exposure in cases of subencephalopathic lead intoxication have yet to be established, but such effects have been observed in chronic occupationally exposed adults at PbB levels as low as 40 to 50  $\mu\text{g}/\text{dl}$  (ATSDR 1988).

Toward the lower range of PbB levels associated with overt lead intoxication, less severe but important signs of impairment in normal physiological functioning in several organ systems are evident among apparently asymptomatic lead-exposed adults (EPA 1985c). These include:

- o Slowed nerve conduction velocities, indicative of peripheral nerve dysfunction (at PbB levels as low as 30 to 40  $\mu\text{g}/\text{dl}$ ) (Seppalainen *et al.* 1975);
- o Altered testicular function (at PbB levels of 40 to 50  $\mu\text{g}/\text{dl}$ ) (Lancranjan *et al.* 1975); and
- o Reduced hemoglobin production (at approximately 50  $\mu\text{g}/\text{dl}$ )

(Zielhuis 1975).

EPA has concluded that all of the above effects point toward a generalized impairment of normal physiological functioning of several different organ systems at adult PbB levels exceeding 30 to 40  $\mu\text{g}/\text{dl}$ . Evidence of impaired heme synthesis in blood occurs at even lower levels.

The second National Health and Nutrition Examination Survey (NHANES-II) examined a representative sample of the U.S. population and found that PbB levels were related to ambient environmental exposures. A clinically relevant relationship between blood lead and blood pressure was seen. Younger men (aged 24 to 55 years) with diastolic high blood pressure had significantly higher blood lead levels than those with normal pressures (Harlan et al. 1985). Additional analyses of the same data by Pirkle et al. (1985) found a statistically significant correlation between PbB levels and diastolic blood pressure in white males, ages 40 to 50, with no threshold apparent in the range of 6 to 30  $\mu\text{g}/\text{dl}$ . Of particular concern is the finding of a 2 mm Hg increase in diastolic pressure per incremental PbB level increase of 0.5  $\mu\text{g}/\text{dl}$ . Possible increases in the risk of more severe medical events (stroke, heart attack, death) associated with lead-induced increases in blood pressure are also estimated in one of the recently published studies (EPA 1986).

Children represent a sensitive subpopulation with regard to lead toxicity. As with adults, lead affects many different organ systems and biochemical/physiological processes across a wide range of exposure levels. Effective PbB levels for producing encephalopathy or death in children are lower than in adults, starting at approximately 80 to 100  $\mu\text{g}/\text{dl}$ . Permanent mental retardation and other marked neurological deficits are among lasting neurological sequelae typically seen in cases of nonfatal childhood lead encephalopathy. Other overt neurological signs and symptoms of subencephalopathic lead intoxication, such as peripheral neuropathies (functional and/or pathological changes in the peripheral nervous system), have been detected in some children at PbB levels as low as 40 to 60  $\mu\text{g}/\text{dl}$ . Chronic kidney disease is most evident at PbB levels above 100  $\mu\text{g}/\text{dl}$ . Moreover, colic and other overt gastrointestinal symptoms occur in children, at PbB levels as low as 60  $\mu\text{g}/\text{dl}$ . Frank anemia is also evident at 70  $\mu\text{g}/\text{dl}$ , representing an extreme manifesta-

tion of reduced hemoglobin synthesis at PbB levels as low as 40 µg/dl. All these effects are widely accepted as adverse health effects, and are reflective of the widespread marked impact of lead on the normal physiological functioning of many different organ systems (EPA 1984d, 1985c, ATSDR 1989).

Other studies demonstrate additional important health effects occurring in children non-overtly intoxicated with lead at similar or lower PbB levels than those indicated above. Among the most important and controversial of these electrophysiological and neuropsychological effects are indications of peripheral nerve dysfunction, evidenced by slowed nerve conduction velocities (NCV) found in children with PbB levels lower than 30 µg/dl (Otto et al. 1981, 1985). EPA has concluded that while none of these studies on central nervous system (CNS) effects can be regarded individually as conclusively proving significant cognitive (IQ) or behavioral effects occurring below 30 µg/dl, they clearly indicate likely associations between neuropsychologic deficits at PbB levels as low as 30 to 50 µg/dl. The magnitude of average observed IQ deficits is approximately 5 points at mean PbB levels of 50 to 70 µg/dl and about 4 points at mean levels of 30 to 50 µg/dl (EPA 1985b). In testimony given to the Subcommittee on Health and the Environment (COEC 1988), Dr. Herbert Needleman reported findings that children with elevated lead levels in shed baby teeth had lower IQ scores, poorer auditory and language functioning scores, and difficulties with attention. Infants with blood levels greater than 10 µg/dl had IQ scores 4-7 points below those with blood lead levels below 3 µg/dl.

Many different impacts (representing potentially impaired functioning and depleted reserve capacities of many different tissues and organs) have been noted at PbB levels below 30 µg/dl.

At PbB levels around 10 to 15 µg/dl, initial signs of detectable heme synthesis impairment occur in many different organ systems, indications of increasing degrees of pyrimidine metabolism interference, signs of altered nervous system activity, and interference in vitamin-D metabolism. EPA has stated that on the basis of these data, these effects might be viewed as sufficiently adverse to warrant avoidance as PbB levels exceed 10 to 15 µg/dl (EPA 1988c).

## Reproduction and Development

There is a paucity of exposure data on which to evaluate the effects of lead on reproduction and development in humans. Early studies of pregnant women exposed to high levels of lead indicated toxic, but not teratogenic, effects on the fetus. One recently reported study hints at birth anomalies which may be associated with exposure to low lead levels (mean cord blood level of 15  $\mu\text{g}/\text{dl}$ ) among women in the general population. However, the significance of these studies has been questioned because of the absence of reported statistically significant associations between cord blood levels and specific types of minor anomalies or any major anomalies. Evidence from recent studies indicates that the length of gestation is reduced as prenatal lead exposure increases, even at PbB levels below 15  $\mu\text{g}/\text{dl}$  (McMichael et al. 1986; Dietrich et al. 1986, 1987). Based on risk estimates of McMichael et al. (1986), the risk of preterm delivery increases by at least four-fold as either cord blood or maternal blood lead level at delivery increases from  $\leq 8$  to  $>14$   $\mu\text{g}/\text{dl}$ .

A review of the literature regarding neurobehavioral effects of prenatal lead exposure suggests that neurobehavioral deficits are associated with prenatal internal exposure levels, at maternal or cord blood lead concentrations, of  $\approx 10$  to 15  $\mu\text{g}/\text{dl}$  (ATSDR 1988; 1987; EPA 1986a) and possibly even lower. There are no reliable data pointing to adverse effects in human offspring following lead exposure to fathers (ATSDR 1989). However, the contribution of adversely affected sperm is not ruled out.

EPA has concluded that the current collective human data, regarding lead's effects on reproduction and in utero development, are insufficient for accurate estimation of exposure-effect or no-effect levels (EPA 1985b). In the absence of sufficient data, it has been suggested that it would be prudent to avoid lead exposures resulting in PbB levels exceeding 25 to 30  $\mu\text{g}/\text{dl}$  to pregnant women and women of child-bearing age in general. This conclusion was based on the known equilibration between maternal and fetal blood lead concentrations and growing evidence of deleterious effects in young children as PbB levels approach 25 to 30  $\mu\text{g}/\text{dl}$ . Industrial lead exposure of men, in which PbB levels of 40 to 50  $\mu\text{g}/\text{dl}$  are seen, also appears to result in altered testicular

function (EPA 1985b).

### **Carcinogenicity and Mutagenicity**

Several studies have reported renal tumors in Wistar rats following ingestion of high doses of a lead salt (lead acetate) (Azar 1973). Lead subacetate (another lead salt) produced benign tumors (renal carcinomas or adenomas) in Swiss mice and in several strains of rats, but not in golden hamsters. Gliomas (CNS tumors) were also observed in many of these studies (Azar 1973; Zawirska and Medras 1972; ATSDR 1989).

There have been a number of epidemiological studies which have assessed the mortality of lead-exposed workers. In some of the studies, no excess cancer mortality was observed. In one study, nonstatistically significant excess cancer mortality of the respiratory system and cancer of the digestive organs and peritoneum was reported, which on evaluation by other statistical techniques by another investigator was reported to achieve statistical significance. Another study has reported increased mortality from renal cancer among a group of lead smelting workers. However, this excess mortality, based on only six cases, did not achieve statistical significance. On review of all of these studies, EPA concluded that the absence of good lead exposure documentation made it difficult to assess the contribution of lead to the observed results.

The International Agency for Research on Cancer (IARC) has classified lead in Group 3, inadequate evidence for carcinogenicity in humans, sufficient evidence for carcinogenicity in animals (for some salts). EPA has classified lead in category B<sub>2</sub> (sufficient evidence in animals, insufficient evidence in humans) according to EPA's Guidelines for Carcinogen Risk Assessment (EPA 1985b, 1986b). However, the agency noted that the doses inducing kidney tumors in positive rat studies were beyond the human lethal dose, and several epidemiological studies have not shown an association between lead exposure and elevated cancer in occupationally exposed workers. Nevertheless, EPA has recently proposed to set a maximum contaminant level goal (MCLG) for lead in drinking water based on both carcinogenic and noncarcinogenic endpoints (EPA 1988).

Lead is a cellular poison. Consequently, short-term mutagenicity assays result in significant cellular toxicity prior to expression of

mutagenicity.

### Quantitative Indices of Toxicity

EPA derived an oral reference dose of  $1.4 \times 10^{-3}$  mg/kg/day for lead based on its determination that a PbB concentration of 15 µg/dl represents a level of concern for the most sensitive human subpopulation-- infants (EPA 1985).

The current EPA drinking water maximum contaminant level (MCL) for lead of 50 µg/L was designed to limit PbB levels in 99.5% of the population to below 30 µg/dl.

EPA has recently proposed (EPA 1988) a new MCL for lead of 0.005 mg/L for finished drinking water entering a distribution system, supplemented by requirements for additional corrosion control or educational measures if either the average lead concentration in the mornings first draw of water at the tap exceeds 0.010 mg/L or if 5% of the samples exceed 0.020 mg/L. These primary drinking water standards were proposed with the intent of achieving a large reduction in the number of people with blood lead levels above the range of concern of 10 to 15 µg/dl and above 25 µg/dl, the level at which the Centers for Disease Control (CDC) recommends medical intervention.

### Standards and Criteria

Standards and criteria applicable to lead are summarized in Table 1.

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Table 1  
STANDARDS AND CRITERIA  
FOR LEAD

Standard or Criterion	Value	Reference
<u>Reference Dose (Withdrawn, but not replaced)</u>		
Oral	$1.4 \times 10^{-3}$ (mg/kg/day)	EPA 1986b
Inhalation	$4.3 \times 10^{-4}$ (mg/kg/day)	EPA 1986b
<u>Drinking Water</u>		
National Primary Drinking Water Regulations		
MCL (a)	50(5p) $\mu\text{g/L}$	EPA 1988
MCLG (b)	0.02 mg/L (p)	EPA 1988
<u>Surface Water</u>		
EPA Ambient Water Quality Criteria		
Aquatic Organisms and Drinking Water	50 $\mu\text{g/L}$	EPA 1990a
Drinking Water only	50 $\mu\text{g/L}$	EPA 1986
<u>Occupational Air Concentrations</u>		
Lead inorganic dusts and fumes		
OSHA PEL TWA	$0.05 \text{ mg/m}^3$	OSHA 1989
ACGIH TLV TWA	$0.15 \text{ mg/m}^3$	ACGIH 1989

p proposed (53 Federal Register 31517-31578)

a MCL = Maximum contaminant level.

b MCLG = Maximum contaminant level goal.

## MANGANESE

Manganese is a naturally occurring element, and is an essential element for both animals and man. Background concentrations vary depending on geological and other influences. Freshwater may contain from 1 to 2,000  $\mu\text{g/L}$ , and higher concentrations have been detected in waters impacted by industrial discharges. Ambient air concentration for nonindustrialized and industrialized areas of  $0.05 \mu\text{g/m}^3$  and  $0.3 \mu\text{g/m}^3$  have been reported (WHO 1984).

The manganese content of food varies considerably. Low concentrations are found in dairy products (0.0 to 1.9 mg/kg), meats (0.0 to 0.8 mg/kg), and fish (0.0 to 0.1 mg/kg). Higher concentrations have been found in grains and cereals (1.2 to 30.8 mg/kg), nuts (0.4 to 35.1 mg/kg), and vegetables (0.2 to 12.7 mg/kg). Tea leaves contained the highest concentration, and a cup of tea can contain from 1.4 to 3.6 mg (WHO 1984).

### Environmental Transport and Fate

Manganese (elemental CAS #7439-96-5) can exist in the oxidation states from -3 to +7. Its inorganic chemistry is dominated by compounds in the +2, +4, and +7 states (USEPA 1984). The principle sources of manganese in the atmosphere are natural including dust, volcanic emissions, and forest fires. Anthropogenic sources of manganese include industrial emissions and combustion of fossil fuels (Lantzy and MacKenzie 1979). Manganese in the atmosphere is generally present in particulate form (USEPA 1982). The atmospheric residence time is estimated at 7 days (Cupitt 1980) with removal occurring by wet or dry deposition.

In aquatic systems, the fate of manganese is influenced by chemical and microbiological reactions. In most natural systems manganese is expected to be predominantly adsorbed to sediments and suspended particulates, in the form of  $\text{MnO}_2$  and/or  $\text{Mn}_3\text{O}_4$ . Although manganese may undergo chemical speciation due to chemical and microbiological reactions, the residence time of aquatic manganese may be a few hundred years (USEPA 1984).

In the soil, manganese speciation may occur through chemical and microbiological interactions. Soil pH and the oxidation-reduction

potential will influence speciation. It has been suggested that in water-logged acidic soils, manganese passes freely into solution and may leach to groundwater (USEPA 1982).

### Toxicokinetics

Manganese is an essential element and is a co-factor for a number of enzymatic reactions particularly those involved in phosphorylation and cholesterol and fatty acid synthesis. Absorption of manganese from the gastrointestinal tract is controlled by homeostatic mechanisms. Extent of absorption is dependent upon availability, concentration in the diet, interactions with other metals or other dietary constituents, and age (USEPA 1982). Limited quantitative data indicate that under normal conditions gastrointestinal absorption of manganese is low, averaging approximately 3% of the ingested manganese. It is expected to be absorbed as  $Mn^{2+}$  (EPA 1984; Mena et al. 1969). Following inhalation exposure, manganese absorption into the bloodstream occurs only if particles are sufficiently small to be able to reach the alveoli (WHO 1980). Larger particles are removed by mucociliary clearance.

Absorption through the skin is not expected to occur to any great extent (Rodier 1955).

Manganese is reportedly distributed in the plasma by a transferrin type of B-globulin (WHO 1981). Absorbed manganese is concentrated in the liver and may form complexes with bile components (WHO 1981). Elimination is almost exclusively through the feces.

### Noncarcinogenic Effects

In humans, manganese dusts and compounds have relatively low oral and dermal toxicity, but may cause a variety of toxic effects after inhalation exposure (WHO 1981). One of two common syndromes associated with inhalation exposure (manganism) involves the central nervous system (CNS). Chronic manganese exposure may result in a psychiatric disorder with characteristic symptoms including irritability, difficulty in walking, and speech disturbances. If the condition persists, a Parkinson-like syndrome may develop (Mena et al. 1967).

The second condition associated with acute manganese inhalation exposure (generally due to manganese dioxide in mining or manufacturing)

is manganese pneumonitis. Men working in industrial plants with high concentrations of manganese dust show an incidence of respiratory disease 30 times greater than normal (Goyer 1986).

### **Carcinogenicity**

There is no epidemiological information relating manganese exposure to cancer occurrence in humans. Results of studies with divalent manganese are suggestive of carcinogenic activity in laboratory animals (USEPA 1984). More information is needed before a more definitive conclusion can be made about the carcinogenicity of manganese and its compounds (USEPA 1984; EPA 1982; Goyer 1986). Therefore, it is classified by USEPA in category D - not classifiable as to carcinogenicity.

### **Quantitative Indices of Toxicity**

The quantitative indices of toxicity for manganese are summarized in Table 1.

EPA has derived an inhalation reference dose (RfD) of  $3 \times 10^{-4}$  mg/kg/day based upon studies conducted by Saric et al. (1977). Oral reference doses were calculated from experiments performed by Laskey et al. (1982); the RfD chronic is  $2 \times 10^{-1}$  mg/kg/day and the subchronic, RfD is  $5 \times 10^{-1}$  mg/kg/day.

### **Standards and Criteria**

Relevant standards and criteria for manganese are summarized in Table 2.

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Table 1  
INDICES OF TOXICITY  
FOR MANGANESE

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<u>Oral Route</u>	
Chronic Reference Dose (RfD) mg/kg/day	$2 \times 10^{-1}$
Subchronic RfD (RfDs) mg/kg/day	$5 \times 10^{-1}$
Confidence Level	NS
Critical Effect	CNS; reproductive
Test Species for Critical Effect	Rat
RfD Basis	Drinking water
RfD Source	IRIS - EPA 1990a [AND/OR] HEAST - EPA 1990b
Uncertainty Factor	
- Chronic RfD	100
- Subchronic RfD	100

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<u>Inhalation Route</u>	
Chronic Reference Dose (RfD) mg/kg/day	$3 \times 10^{-4}$
Subchronic RfD (RfDs) mg/kg/day	$3 \times 10^{-4}$
Confidence Level	NS
Critical Effect	CNS
Test Species for Critical Effect	Human
RfD Basis	Occupational air
RfD Source	IRIS - EPA 1990a [AND/OR] HEAST - EPA 1990b
Uncertainty Factor	
- Chronic RfD	100
- Subchronic RfD	100

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NS = Not specified.

Table 2  
STANDARDS AND CRITERIA  
For Manganese

Standard or Criterion	Value	Reference
<u>Surface Water</u>		
USEPA Ambient Water Quality Criteria		
Ingestion of Fish and Water	50 mg/L	USEPA 1986
<u>Occupation Exposure Limits (Air)</u>		
OSHA PEL TWA:		
Dusts	5.0 mg/m <sup>3</sup> (ceiling)	OSHA 1989
Fumes	1.0 mg/m <sup>3</sup>	OSHA 1989
ACGIH TWA:		
Dusts	5.0 mg/m <sup>3</sup>	ACGIH 1989
Fumes	1.0 mg/m <sup>3</sup>	ACGIH 1989

**TETRACHLOROETHENE (PERCHLOROETHYLENE)**

Tetrachloroethene, also known as perchloroethylene (PCE), is a synthetic organic chemical with no natural resources. It is used in industry in a variety of processes, as a solvent for many organic substances, as a cleaning agent, as a metal degreaser, as an intermediate in the synthesis of certain fluorocarbons, and in the textile industry. It is not consumed during its various uses and may be released to the environment. Air monitoring data for the United States has demonstrated mean PCE concentrations of 160 ppt in rural and remote areas, 790 ppt in urban and suburban areas, and 1,300 ppt in areas near emission sources (ATSDR 1988). PCE has been detected in many drinking water sources, with median concentrations of 0.3 and 3.0 µg/L in surface and groundwaters, respectively (ATSDR 1988).

**Environmental Chemistry and Fate**

The relevant physical and chemical properties and environmental transport and fate of PCE (CAS No. 127-18-4) are summarized below (Howard 1989; Clement Associates, Inc. 1985; MacKay and Shiu 1981):

Chemical Formula	$C_2Cl_4$
Molecular Weight	165.8 g/mole
Physical State at 20°C	Liquid
Water Solubility	1,500 mg/L (25°C)
Density at 20°C	1.63 g/ml
Vapor Pressure	18.5 mm Hg (25°C)
Henry's Law Constant	2.3 E-04 (25 °C)
Octanol-Water Partition Coefficient (Log $K_{ow}$ )	2.88
Organic Carbon Partition Coefficient ( $K_{oc}$ )	364 mL/g
Bioconcentration Factor (BCF)	31

PCE's moderate water solubility, vapor pressure, and Henry's Law constant indicate that volatilization is the major loss mechanism from surface soil and surface water. The half-life in surface water has been estimated to range from 1 to 30 days. Its moderate  $K_{oc}$  and moderate

water solubility indicate that transport from unsaturated zone soils to groundwater via rain infiltration is an important pathway. In soils and groundwater, PCE is biodegraded in a series of monodechlorinations to trichloroethene, cis- and trans-1,2-dichloroethylene, vinylidene chloride (1,1-dichloroethene), and vinyl chloride (chloroethene). PCE's half-life in the subsurface has been estimated to range from as little as 2 days to about 9 months (Smith and Dragun 1984). Once PCE reaches the groundwater, its moderate  $K_{oc}$  indicates that transport may be moderately retarded by high organic carbon sediments. Finally, PCE is subject to low bioconcentration in aquatic species.

### Toxicokinetics

PCE is absorbed completely by rats following oral exposure (Schumann et al. 1980). However, only 25% absorption occurred in human volunteers following a 4-hour inhalation exposure. Animal studies indicate that once in the bloodstream, PCE concentrates in adipose tissue and the brain (Monster 1979). Metabolism occurs via oxidation, possibly through an epoxide intermediate, to trichloroethanol and trichloroacetic acid (Ikeda 1977). PCE is eliminated unchanged via the lungs, with an estimated half-life of 65 to 70 hours; its metabolites are eliminated via the urine with a half-life of 144 hours (Ikeda and Imamura 1973).

### Noncarcinogenic Effects

In animals, the most characteristic effect following acute high-level exposure to PCE is on the central nervous system (CNS). At increasing concentrations, CNS depression, dizziness, unconsciousness, respiratory and cardiac arrest, and finally death occur (EPA 1985; ATSDR 1989). Short-term, subchronic, high-level exposure effects are manifested principally as damage to the liver and kidney. Liver damage progresses from congestion and cloudy swelling to fatty degeneration and cell death. Kidney damage progresses from increased organ rate, cloudy swelling of the tubular cell walls, sloughing of cells, and cell death. Longer-term exposure in animals results principally in the liver and kidney damage described above. In humans, like animals, the principal effects consequent to high short-term or chronic exposure are CNS and liver and kidney dysfunction.

Mild reproductive toxicity was exhibited by  $B_6C_3F_1$  mice and Sprague-Dawley rats following inhalation exposure to PCE for 7 hours per day from days 6 to 15 of gestation, the period of highest susceptibility to teratogenic effects (Schwetz et al. 1975). Pregnant rats exposed to 300 ppm ( $2,000 \text{ mg/m}^3$ ) had mild reductions in body weight and twice the number of resorptions per implantation compared to controls, while mice exhibited a significant increase in liver weight and decreased fetal weight compared to controls (Schwetz et al. 1975). Following prenatal exposure, significant subcutaneous edema, delayed skull hardening, and presence of split sternebrae were observed in mouse pups. Differences in neurotransmitter levels and some alterations in behavioral tests were noted in offspring of rats exposed to 900 ppm ( $3,600 \text{ mg/m}^3$ ), but not in those exposed to 100 ppm ( $400 \text{ mg/m}^3$ ). As the more significant findings occurred at concentrations which may have been maternally toxic, the relevance of these findings to reproductive risks at lower nonmaternally toxic concentrations (e.g., 100 ppm) can be questioned.

#### **Carcinogenicity and Mutagenicity**

The National Cancer Institute (NCI) concluded that commercially used PCE-containing stabilizers acted as a liver carcinogen in  $B_6C_3F_1$  mice administered 386 to 1,072 mg/kg by stomach tube (gavage) for 78 weeks (NCI 1977, as cited in EPA 1987). Because of high mortality rates, NCI made no conclusion as to carcinogenicity of PCE in Osborne-Mendel rats (NCI 1976, as cited in EPA 1987).

In a National Toxicity Program (NTP) inhalation bioassay (NTP 1985, as cited in EPA 1987),  $B_6C_3F_1$  mice and Osborne-Mendel rats were exposed to PCE in concentrations of 0, 200 ( $1,340 \text{ mg/m}^3$ ), and 400 ppm ( $2,680 \text{ mg/m}^3$ ), and 0, 100 ( $680 \text{ mg/m}^3$ ), and 200 ppm ( $1,360 \text{ mg/m}^3$ ), respectively. PCE induced hepatocellular carcinomas in both sexes of mice. Male rats exhibited a significantly increased incidence of mononuclear cell leukemia and an increased incidence of combined kidney tubular adenomas and carcinomas. Classification of PCE as a carcinogen in rats is controversial. EPA's Science Advisory Board (SAB) has questioned the relevance of mononuclear leukemia to man (a resistant species to this type of leukemia) and the validity of combining the adenomas and carcinomas to achieve statistical significance (EPA 1987).

Based upon these data, and according to its weight-of-evidence carcinogenicity criteria, EPA has placed PCE in Category B2, "probable human carcinogen" (EPA 1987). However, EPA's SAB has recommended classifying PCE in Category C, "possible human carcinogen" (EPA 1987).

PCE has been evaluated for its ability to cause gene mutation, chromosomal aberrations, unscheduled DNA synthesis, and mitotic recombination. In general, these responses have been weak and were observed at high concentrations that were cytotoxic (EPA 1985). Additionally, no dose-dependent relationships were demonstrated in these studies (EPA 1985).

### Quantitative Indices of Toxicity

Quantitative indices of toxicity for tetrachloroethene are presented in Table 1. Using the linearized multistage carcinogenesis model, EPA derived the upper-bound 95% confidence limit slope factor (SF) based on animal data. The upper-bound estimate for the oral route is  $5.1 \times 10^{-2} \text{ (mg/kg/day)}^{-1}$ ; the corresponding estimate for the inhalation route is  $3.3 \times 10^{-3} \text{ (mg/kg/day)}^{-1}$  (EPA 1990a).

EPA has derived a reference dose (RfD) based upon a study (Buben and O'Flaherty 1985) in which Swiss-Cox mice received PCE in corn oil by gavage at doses ranging from 0 to 2,000 mg/kg/day 5 days per week for 6 weeks (EPA 1987). Based upon findings of slight increases in liver weight, EPA defined 20 mg/kg/day as a no-observed-adverse-effect level (NOAEL). Based upon findings of significantly increased liver weights, EPA defined 100 mg/kg/day as a lowest-observed-adverse-effect level (LOAEL) (EPA 1990a). Using the NOAEL of 20 mg/kg/day scaled to correspond to average daily doses over a 6-week period and dividing by an uncertainty factor of 1,000, EPA derived an RfD of  $1 \times 10^{-2} \text{ mg/kg/day}$  (EPA 1990b).

PCE is the most acutely toxic to aquatic life of the chloroethylenes but is only moderately toxic relative to other types of compounds. The trout was reportedly the most sensitive to PCE exposure with an  $LC_{50}$  value of 4,800  $\mu\text{g/L}$ . The EPA (1984) reported the lowest values known to cause toxicity in aquatic organisms; however, the aquatic species tested was not specified. Freshwater species toxicity values of 5,280 and 840  $\mu\text{g/L}$  were reported for acute and chronic

exposure, respectively.

## Standards and Criteria

Standards and criteria applicable to PCE are summarized in Table 2.

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Table 1  
 INDICES OF TOXICITY  
 FOR TETRACHLOROETHENE

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Oral Route

Chronic Reference Dose (RfD) mg/kg/day	$1 \times 10^{-2}$
Subchronic RfD (RfDs) mg/kg/day	$1 \times 10^{-1}$
Confidence Level	Medium
Critical Effect	Hepatotoxicity - mice Weight gain - rats
Test Species for Critical Effect	mice/rat
RfD Basis	Oral
RfD Source	IRIS - EPA 1990a
Uncertainty Factor	
- Chronic RfD	1000
- Subchronic RfD	100
Modifying Factor	
- Chronic RfD	1
- Subchronic RfD	NS

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NS = Not specified.

Table 2  
Standards and Criteria  
for Tetrachloroethene

Standard or Criterion	Value	Reference
<u>Drinking Water</u>		
National Primary Drinking Water Regulations		
Proposed MCL (a)	0.005 mg/L	EPA 1990a,b
Proposed MCLG (b)	0	EPA 1990a,b
<u>Surface Water</u>		
EPA Ambient Water Quality Criteria		
Aquatic Organisms and Drinking Water	0*	EPA 1986
Drinking Water Only	0	EPA 1986
<u>Occupational Air Concentrations</u>		
OSHA PEL TWA	670 mg/m <sup>3</sup> (100 ppm)	OSHA 1989
ACGIH TWA	335 mg/m <sup>3</sup> (50ppm)	ACGIH 1989

\*Potential carcinogens have a criterion value of 0. A concentration of 0.8 µg/L corresponds to a risk of 10<sup>-6</sup>, as presented in water quality criteria documents.

a MCL = Maximum contaminant level.

b MCLG = Maximum contaminant level goal.

## POLYCYCLIC AROMATIC HYDROCARBONS

### Introduction

Polycyclic aromatic hydrocarbons (PAHs) contain only carbon and hydrogen and consist of two or more fused benzene rings in linear, angular or cluster arrangements. PAHs are formed wherever natural or synthetic chemicals are exposed to high temperature; they may also be synthesized by some plants and bacteria.

The following sections address specific aspects of PAH-related information useful in evaluating potential human health and environmental risks of PAHs.

### Sources and Concentrations of PAHs in the Environment

As discussed above, there are both natural (e.g., forest fires and synthesis by plants and microorganisms, etc.) and anthropogenic sources of PAHs. Currently, primary anthropogenic sources accounting for greater than 90% of total air emissions are fossil fuel combustion, by electrical power plants, refuse burning, and agricultural burning (NAS 1972). PAHs are also emitted from automobiles, buses, and trucks and are typically localized along highways. Those emitted by power plants or jet engine exhausts may be spread over long distances.

Many of the current combustion processes and certain industrial processes (e.g., coke production and petroleum refining) have led to the widespread presence of PAHs in ambient air. The concentrations of PAHs in air vary greatly depending on the relation to sources. Savicki *et al.* (1965) examined benzo(a)pyrene (BaP) concentrations in air for 131 urban and suburban areas in the United States. In urban areas concentrations ranged from 0.1 to 61 ng/m<sup>3</sup>. In nonurban areas concentrations ranged from 0.01 to 1.9 ng/m<sup>3</sup>. Gordon (1976) reported the geometric mean of the sum of 15 PAH airborne concentrations to be 10.9 ng/m<sup>3</sup>, only 4.2% of which was due to BaP.

Typical concentrations with no known sources of BaP in soils of the world range from 100 to 1,000 µg/kg (Edwards 1983). Where quantified, total PAH concentrations were reported to be 10 times the value of BaP alone (Edwards 1983). Measured concentrations ranged from 0.4 µg/kg in protected remote areas to 650,000 µg/kg in highly polluted areas

(Edwards 1983).

PAHs in surface waters originate from fallout of particulate matter, absorption of atmospheric gases, and polluted water (IARC 1983). Except near major sources, concentrations of PAHs in water are typically on the order of 0.0003  $\mu\text{g/L}$ . A survey of PAH concentrations found in water is given in Table 1.

#### Environmental Transport and Fate

Relevant physical and chemical properties of selected PAHs are summarized in Table 2. In general, most priority pollutant PAHs can be characterized as having low vapor pressure, low water solubility, low Henry's Law constants, high octanol-water partition coefficients (log  $K_{ow}$ ) and high organic carbon partition coefficients ( $K_{oc}$ ). High  $K_{oc}$ s indicate that most PAHs are strongly sorbed to organic matter in the soils. Combined with low water solubilities, rates of transport of most PAHs from the unsaturated zone via infiltration to the saturated zone will be extremely low. Low vapor pressures, low Henry's Law constants and low  $K_{oc}$ , indicate that most PAHs will not readily volatilize from surface water nor from surface soils.

The exceptions to this generalization are acenaphthene, fluorene, fluoranthene, fluorene, naphthalene and pyrene with solubilities greater than 100  $\mu\text{g/L}$ . Although these compounds have relatively high  $K_{oc}$ s ( $10^3$  or greater) relative to other PAHs, their high solubility indicates that they are relatively mobile and may be observed in groundwater.

Typically, although they are regarded as persistent in the environment, PAHs are degradable by soil microorganisms. Degradation rates and degree of degradation are influenced by environmental factors, microbial flora and physicochemical properties of the PAHs themselves. Important environmental factors include temperature, pH, oxygen status, populations present, and the relative proportions of bacteria, fungi, and actinomycetes (Sims and Overcash 1983). Physico-chemical properties include chemical structure, concentration, and lipophilicity.

## Human Exposure Levels

Because of the ubiquitous presence of PAHs in the environment, it is important to evaluate baseline, non-site-related human exposures. For the purpose of this discussion, PAH exposure data have been divided into four categories consistent with the breakdown of available data: benzo(a)pyrene, naphthalene, the anthracene group, and the acenaphthene group.

BaP has been the most studied PAH. Table 3 presents typical dietary consumption, BaP concentrations in food, and estimated BaP daily intakes (USEPA 1982). As the table displays, BaP is found in numerous dietary items including charcoaled beef, oil, fruits, grains, and vegetables corresponding to an estimated 0.05  $\mu\text{g}/\text{day}$  intake in the diet.

Table 4 presents an estimated distribution of the U.S. population exposed to ranges of BaP concentrations in ambient air (USEPA 1982). The table indicates that nearly half the U.S. population is exposed to concentrations less than 1  $\text{ng}/\text{m}^3$  and nearly 99.5% of the population is exposed to less than 5  $\text{ng}/\text{m}^3$ . Using a breathing rate of 0.875  $\text{m}^3/\text{day}$ , assuming 24-hour exposure and 100% absorption, daily intakes of 20  $\text{ng}/\text{day}$  and 100  $\text{ng}/\text{day}$  were predicted for those exposed to the respective BaP concentrations.

Cigarette smoke is a major source of PAH exposure. Tables 5 and 6 show that, with the exception of acenaphthene and fluorene for which data were not available, the priority pollutant PAHs are found in significant concentrations in mainstream cigarette smoke. Assuming 25 cigarettes smoked per day, mean intake estimates range from 0.05  $\mu\text{g}/\text{day}$  for benzo(g,h,i)perylene to 38  $\mu\text{g}/\text{day}$  for naphthalene, with an intake of 1  $\mu\text{g}/\text{day}$  for BaP. Although the data are far less extensive, side-stream smoke and concentration in smoke-polluted rooms represent significant sources of PAH intake for the nonsmoker and smoker alike.

Table 7 summarizes the typical human intakes of BaP, naphthalene, the anthracene group and the acenaphthalene group of PAHs (USEPA 1982; IARC 1983). In descending order of intake, naphthalene was followed by individual members of the anthracene group, benzo(a)pyrene, and individual members of the acenaphthene group. Not counting exposure to side-stream smoke or exposures in the "other" category, intakes for naphthalene were 41.2 and 3.2  $\mu\text{g}/\text{day}$  for smokers and nonsmokers. Values for

individual members of the anthracene group were 7 and 1  $\mu\text{g}/\text{day}$ , respectively. Intakes of benzo(a)pyrene were 2.1 and 1.1  $\mu\text{g}/\text{day}$ , respectively.

## Toxicology of PAHs

### Noncarcinogenic Effects

Very little attention has been paid to noncarcinogenic effects of PAHs. It is known, however, that rapidly proliferating tissues (e.g., bone marrow, lymphoid organs, testes, etc.) appear to be the preferred targets for PAH-induced cytotoxicity.

Acute and chronic exposure to various PAHs classified as carcinogens has resulted in destruction of specific hematopoietic and lymphoid elements, ovotoxicity, anti-spermatogenic effects, adrenal necrosis and changes in the intestinal and respiratory epithelia. This tissue damage occurs at doses expected to induce carcinomas and malignancy risks predominate in evaluating PAH toxicity. For PAHs classified as noncarcinogenic, very little is known about toxic responses or mechanisms.

### Carcinogenicity and Mutagenicity

EPA has issued final carcinogenicity risk assessment guidelines (51 FR 33992-34012, September 24, 1986). These guidelines established weight-of-evidence criteria for evaluating and categorizing chemicals as to their potential carcinogenicity. According to this categorization scheme, five of the 15 priority pollutant PAHs have been placed in category B<sub>2</sub> (probable human carcinogens) sufficient evidence in animals, inadequate data for humans. A sixth (indeno (1,2,3-cd) perylene) has been placed in category C, possible human carcinogen based on limited evidence of carcinogenicity in animals in the absence of human data (EPA 1986A). Table 8 contains EPA's most current categorization of priority pollutant PAHs (EPA 1986). Following its risk assessment guidelines, EPA typically performs quantitative risk assessments for groups A or B, and in some cases, depending on the quality of the data, for group C. In order to be reasonably conservative (health-protective), estimated potencies will be used for all six PAHs categorized as B<sub>2</sub> or C carcinogens.

To date, EPA has estimated a carcinogenicity slope factor (SF) for carcinogenic PAHs using data for a single PAH, benzo(a)pyrene (BaP). This limited effort does not take into account the clearly documented differences in quantitative dose-response relationships for the other PAHs. Thorslund et al. (1986) have evaluated the relative potency estimates for the other five carcinogenic PAHs to BaP. Using a series of sophisticated statistical procedures, these authors have derived estimated relative potencies for the five other "carcinogenic" PAHs relative to BaP. For the potency estimation, the authors used only bioassays from individual laboratories in which BaP and other PAHs were tested in common. Table 9 summarizes the results of the procedures.

### Standards and Criteria

Relevant standards and criteria for PAHs are summarized in Table 10.

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Table 1.  
 CONCENTRATIONS OF 15 PRIORITY POLLUTANTS POLYCYCLIC  
 AROMATIC HYDROCARBONS IN WATER

Compound	Concentration Range (ng/l)			
	Surface Water	Tap Water	Rainfall	Groundwater
Acenaphthene	N.D.	N.D.	N.D.	N.D.
Anthracene	1,000	1.1-59.7	N.D.	N.D.
Benzo(a)anthracene	1.9-30.6	0.4-10.7	3.2-12.3	0-1.3
Benzo(b)fluoranthene	0-320	0.6-45	4.4-840	0.5-9.0
Benzo(b)fluoranthene	0-400	0.9-8.0	1.6-450	0.2-3.5
Benzo(g,h,i)perylene	1.0-11.2	N.D.	N.D.	N.D.
Benzo(a)pyrene	0-13,000	0-1,000	10-1,000	0.1-6
Chrysene	7.6-62.0	N.D.	N.D.	N.D.
Dibenzo(a,h)anthracene	N.D.	N.D.	N.D.	N.D.
Fluoranthene	4.7-1,200	7.2-132.6	5.6-1,460	3.5-100
Fluorene	300	4-16	N.D.	N.D.
Indeno(1,2,3-cd)pyrene	0-350	0.3-75	0-1,020	0.2-5
Naphthalene	N.D.	N.D.	N.D.	N.D.
Phenanthrene	0-1,300	24-90	N.D.	N.D.
Pyrene	2.0-530	N.D.	5.8-27.8	1.6-2.5

Source: Commission of European Communities (1979) as cited in IARC (1983).

Table 2  
PHYSICAL AND CHEMICAL PROPERTIES OF SELECTED PAHS

Compound	Molecular Weight (g/mole)	Cas No.	Vapor Pressure (mmHg) at 20°C	Water Solubility (mg/l)	Henry's Law Constant		Log Kow	Koc (ml/g)	Source	
					Source	Constant				
Acenaphthene	154.2	83-32-9	1.55 x 10 <sup>-3</sup>	3.42	A	9.2 x 10 <sup>-5</sup>	3.98	A	4.6 x 10 <sup>3</sup>	A
Acenaphthylene	154.2	208-96-8	2.90 x 10 <sup>-2</sup>	3.93	A	1.45 x 10 <sup>-3</sup>	3.72	A	2.5 x 10 <sup>3</sup>	A
Anthracene	178.2	120-12-7	1.70 (25°C) x 10 <sup>-5</sup>	4.5 x 10 <sup>-2</sup>	A	8.6 x 10 <sup>-5</sup>	4.45	A	1.4 x 10 <sup>4</sup>	A
Benzo(a)anthracene	228.3	56-55-3	2.2 x 10 <sup>-8</sup>	1.2 x 10 <sup>-3</sup>	B	1.0 x 10 <sup>-6</sup>	5.61	A	2.0 x 10 <sup>5</sup>	A
Benzo(b)fluoranthene	252.3	205-99-2	5.0 x 10 <sup>-7</sup>	1.4 x 10 <sup>-2</sup>	A	1.22 x 10 <sup>-5</sup>	6.06	A	5.5 x 10 <sup>5</sup>	A
Benzo(k)fluoranthene	252.3	207-08-9	5.1 x 10 <sup>-7</sup>	4.3 x 10 <sup>-3</sup>	A	5.0 x 10 <sup>-7</sup>	6.06	A	5.5 x 10 <sup>5</sup>	A
Benzo(g,h,i)perylene	276.3	191-24-2	1.0 (25°C) x 10 <sup>-10</sup>	7.0 x 10 <sup>-3</sup>	A	5.34 x 10 <sup>-8</sup>	6.51	A	1.6 x 10 <sup>6</sup>	A
Benzo(a)pyrene	252.3	50-32-8	5.6 x 10 <sup>-9</sup>	3.8 x 10 <sup>-3</sup>	A	4.9 x 10 <sup>-7</sup>	6.06	A	5.5 x 10 <sup>6</sup>	A
Chrysene	228.3	218-01-9	6.3 x 10 <sup>-9</sup>	1.8 x 10 <sup>-3</sup>	B	1.05 x 10 <sup>-6</sup>	5.61	A	2.0 x 10 <sup>5</sup>	A
Dibenzo(a,h)-anthracene	278.4	53-70-3	1.1 x 10 <sup>-10</sup>	5.0 x 10 <sup>-4</sup>	B	7.33 x 10 <sup>-8</sup>	6.83	A	3.3 x 10 <sup>6</sup>	A
Fluoranthene	202.3	206-44-0	5.0 (25°C) x 10 <sup>-6</sup>	2.6 x 10 <sup>-1</sup>	A	6.46 x 10 <sup>-6</sup>	4.90	A	3.8 x 10 <sup>4</sup>	A
Fluorene	166.2	86-73-7	7.1 x 10 <sup>-4</sup>	1.83	B	6.42 x 10 <sup>-5</sup>	4.18	A	7.3 x 10 <sup>3</sup>	A
Indeno(1,2,3-cd)-perylene	276.3	193-39-5	1.0 x 10 <sup>-10</sup>	5.3 x 10 <sup>-4</sup>	A	6.95 x 10 <sup>-8</sup>	6.51	A	1.6 x 10 <sup>6</sup>	A
2-Methylnaphthalene	142.2	91-57-6	ND	ND	ND	ND	ND	ND	ND	ND
Naphthalene	128.2	91-20-3	8.7 x 10 <sup>-2</sup>	3.17 x 10 <sup>1</sup>	A	4.6 x 10 <sup>-4</sup>	3.37	A	9.3 x 10 <sup>2</sup>	A
Phenanthrene	178.2	85-01-3	9.6 (25°C) x 10 <sup>-4</sup>	1.3	A	2.26 x 10 <sup>-4</sup>	4.46	A	1.44 x 10 <sup>4</sup>	A
Pyrene	202	129-00-3	2.5 (25°C) x 10 <sup>-6</sup>	1.47 x 10 <sup>-1</sup>	B	5.04 x 10 <sup>-6</sup>	4.88	A	3.8 x 10 <sup>4</sup>	A

Key: ND = No Data

Sources:

A: Mabey, W. et al, 1982, Aquatic Fate Process Data for Organic Priority Pollutants (EPA 440/4-81-014).  
 B: ATSDR, 1990, Toxicological Profile for Polycyclic Aromatic Hydrocarbons. Draft for Public Comment.

Table 3  
 LEVELS OF BENZO(A)PYRENE IN FOODS  
 AND ESTIMATED HUMAN INTAKES

Food	Consumption (g/day)		Benzo(a)pyrene Contamination (ug/kg)		Intake (ug/day)	
	Typical	Maximum	Typical	Maximum	Typical	Maximum
Charcoal broiled beef	--	--	--	--	--	--
Hamburger	10	ND	ND	2.6	ND	0.03
Steak	3	86	5	50	0.02	4.3
Smoked Pork**	1	27	2	55	0.002	1.5
Smoked Sausage***	1.5	30	ND	4	0.006	0.12
Smoked Fish?	0.1	14	1	37	0.0001	0.5
Oil	18	ND	1	8	0.02	0.14
Fruits	205	ND	0.02	6	0.004	1.2
Grains	256	ND	ND	0.3	ND	0.008
Vegetables - Total	248	ND	0.01	0.1	0.002	0.2
- Leafy	40	ND	ND	7.5	NA	0.3
Total					0.05	

Source: USEPA 1982.

Key:

ND = No data available.

\* = Consumption of beef - 86 g/day, 15% charcoal broiled - 80% hamburger, 20% steak. Worst case maximum 86 g consumption of charcoal-broiled steak.

\*\* = Consumption of pork - 27 g/day, 5% smoked. Worst case maximum, 27 g/day smoked.

\*\*\* = Consumption of sausage - 30 g/day, 5% smoked. Worst case maximum, 30 g/day smoked.

? = Consumption of fish - 14 g/day, 1% smoked. Worst case maximum 14 g/day smoked.

**Table 4**  
**ESTIMATED DISTRIBUTION OF THE SIZE OF THE U.S. POPULATION**  
**EXPOSED TO RANGES OF BENZO(a)PYRENE (BaP) IN AMBIENT AIR\***

	BaP Concentration (ng/m <sup>3</sup> )				Total
	<0.5	0.5 - 1.0	1.0 - 5.0	>5.0	
Population (1,000's) Exposed	73,294	26,731	102,132	1,059	203,216
Percentage	36.1	13.2	50.3	0.5	100.1**

\*Monitoring data were not available for areas representing 50% of the population. Upper 95% confidence limits of natural average concentrations were used to assign exposures to these populations. The levels were as follows:

Urban standard metropolitan statistical area (SMSA):	1.3 ng/m <sup>3</sup>
Urban non-SMSA:	1.4 ng/m <sup>3</sup>
Rural:	0.23 ng/m <sup>3</sup>

\*\*Does not sum to 100% due to independent rounding.

Table 5  
DAILY INTAKE FROM CIGARETTE SMOKE FOR 15  
PRIORITY POLLUTANT PAHs\*

Compound	Concentration Range (ug/100 Cigarettes)	Intake Range 1,2 (ug/day)	1 Mean Intake (ug/day)
Acenaphthene	ND	ND	
Anthracene	2.3 - 23.5	0.6 - 5.9	3.3
Benzo (a) anthracene	0.4 - 7.6	0.1 - 1.9	1.0
Benzo (b) fluoranthene	0.3 - 2.2	0.1 - 0.6	0.4
Benzo (k) fluoranthene	0.6 - 1.2	0.2 - 0.3	0.3
Benzo (g,h,i) perylene	0.1 - 0.4	0.03 - 0.1	0.05
Benzo (a) pyrene	0.5 - 7.8	0.1 - 2.0	1.0
Chrysene	0.6 - 9.6	0.2 - 2.4	1.3
Dibenzo (a,h) anthracene	0.4	0.1	0.1
Fluoranthene	1 - 27.2	0.03 - 6.8	3.4
Fluorene	present	ND	ND
Indeno (1,2,3k-cd) pyrene	present	ND	ND
Naphthalene	300**	75	38
Phenanthrene	8.5 - 62.4	2.1 - 15.6	8.8
Pyrene	5-27	1.3 - 6.8	4.1

Key:

ND = No data.

\*Unless otherwise footnoted, value from Table 1 (IARC 1983).

\*\*USEPA 1982.

1Assuming 10% absorption and 25 cigarettes smoked per day.

2Rounded to the nearest tenth.

Table 6  
 CONCENTRATION OF THE 15 PRIORITY POLYCYCLIC  
 AROMATIC HYDROCARBONS IN CIGARETTE SMOKE\*

Compound	Cigarette Side Stream Smoke (ug/100 cigarettes)	Cigarette Smoke-Polluted Rooms (ng/m <sup>3</sup> )
Acenaphthene	ND	ND
Anthracene	ND	ND
Benzo (a) anthracene	ND	ND
Benzo (b) fluoranthene	ND	ND
Benzo (k) fluoranthene	ND	ND
Benzo (g,h,i) perylene	9.8	5.9 - 17
Benzo (a) pyrene	2.5 - 19.9	2.8 - 760
Chrysene	ND	ND
Dibenzo (a,h) anthracene	ND	ND
Fluoranthene	126	99
Fluorene	ND	ND
Indeno (1,2,3-cd) pyrene	ND	ND
Naphthalene	ND	830**
Phenanthrene	ND	ND
Pyrene	34 - 101	2-66

Key:

ND = No data.

\*Unless otherwise footnoted, value from Table 1 (IARC 1983).

\*\*USEPA 1982.

ESTIMATED DAILY INTAKE OF 15 PRIORITY POLLUTANT POLYCYCLIC AROMATIC HYDROCARBONS (PAHS) (ug/day)<sup>a</sup>

Route of Exposure	Benzo(a)pyrene	Naphthalene	Individual PAHs in the Anthracene Group <sup>c</sup>	Individual PAHs in the Acenaphthalene Group
Typical Diet	0.005	0.4 <sup>f</sup>	1 <sup>i</sup>	<0.05
Drinking Water	0.0006	2.8	1 <sup>i</sup>	<0.005
Ambient Air	---	0.001-0.007 <sup>g</sup>	1 <sup>i</sup>	0.002-6.4 <sup>j</sup>
Urban	0.02-2	N.D.	1 <sup>i</sup>	---
Suburban	0.002-0.2	N.D.	1 <sup>i</sup>	---
Smoking <sup>b</sup>	1.0	38 <sup>k</sup>	3.3-8.8 <sup>l</sup>	0.05-1.3 <sup>l</sup>
Sidestream Smoke	---	145 <sup>h</sup>	---	---
Charcoal Meats <sup>e</sup>	N.D.	N.D.	---	N.D. <sup>e</sup>
Other		140 (mothballs)	21 (fluoranthene)	---
Total				
Smoker <sup>m</sup>	2.1	41.2	7	0.9
Nonsmoker <sup>m</sup>	1.1	3.2	1	0.2

Key:

- N.D. = No data found.
- <sup>a</sup>Unless otherwise footnoted, all data except smoking taken from USEPA 1982. Smoking data taken from IARC (1983).
- <sup>b</sup>Consistent with estimates in USEPA 1982, smokers are assumed to smoke an average of 25 cigarettes per day. USEPA (1982) reported that roughly 25% to 30% of smokers consume more cigarettes than this average. Consequently, those smokers would be subject to higher exposure.
- <sup>c</sup>Compounds in the anthracene group are anthracene, acenaphthene, fluoranthene, fluorene, phenanthrene, and pyrene.
- <sup>d</sup>Compounds in the acenaphthylene group are acenaphthylene, benzo(a)anthracene, chrysene, dibenzo(a,h)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(g,h,i)perylene, and indeno(1,2,3-cd)pyrene.
- <sup>e</sup>Unless otherwise specified, intakes for charcoaled meats included in estimated intakes for a typical diet.
- <sup>f</sup>Estimate for naphthalene based upon fish consumption only. No data were found for other foods.
- <sup>g</sup>Urban/rural breakdown not available. Data applies to all ambient air other than a nearby source.
- <sup>h</sup>Maximum concentration for a 2-hour exposure in a smoke-filled room, using a concentration in air of 83 ug/m<sup>3</sup> (USEPA 1982) and assuming a respiratory flow of 0.875 m<sup>3</sup>/hr.
- <sup>i</sup>Total for dietary, ambient air, and drinking water exposure. Exposure to fluorene could be as high as 21 ug/day.
- <sup>j</sup>Levels of benzo(g,h,i)perylene are somewhat higher (USEPA 1982).
- <sup>k</sup>Benzo(a)anthracene identified in cigarette smoke (USEPA 1982).
- <sup>l</sup>Data taken from Table 5.
- <sup>m</sup>Totals calculated using mean of ranges. For acenaphthalene group, categories with less than notations, the mean of 0 and the upper limit are used in calculating totals. No sidestream smoke exposures are included in nonsmoking totals.

Table 8

**EPA CARCINOGENICITY CATEGORIZATION FOR  
ORAL AND INHALATION ROUTES OF EXPOSURE FOR THE  
15 PRIORITY POLLUTANTS POLYCYCLIC AROMATIC HYDROCARBONS\***

Compound	EPA Carcinogenicity Classifications*	
	Inhalation	Oral
Acenaphthene	D	D
Anthracene	D	D
Benzo (a) anthracene	B2	B2
Benzo (b) fluoranthene	B2	B2
Benzo (k) fluoranthene	D	D
Benzo (g,h,i) perylene	D	D
Benzo (a) pyrene	B2	B2
Chrysene	B2	B2
Dibenzo (a,h) anthracene	B2	B2
Fluoranthene	D	D
Fluorene	D	D
Indeno (1,2,3-cd) pyrene	C	C
Naphthalene	D**	D**
Phenanthrene	D	D
Pyrene	D	D

\*Unless otherwise footnoted, classification taken from USEPA (1986a).

\*\*Classification from USEPA (1984).



**Table 9**  
**RELATIVE POTENCY ESTIMATES AND SLOPE**  
**FACTORS (SFS) DERIVED**  
**FOR POLYCYCLIC AROMATIC HYDROCARBONS**  
**CATEGORIZED IN GROUP A, B, OR C ACCORDING**  
**TO EPA'S WEIGHT-OF-EVIDENCE CRITERIA**

Compound	Relative Potency Estimates	Oral* SFs	Inhalation* SFs
Benzo(a)pyrene	1	11.5	6.1
Benzo(a)anthracene	0.145	1.7	0.89
Benzo(b)fluoranthene	0.140	1.6	0.85
Chrysene	0.0044	0.05	0.026
Dibenzo(a,h)anthracene	2.82	32.4	17.2
Indeno(1,2,3-cd)perylene	0.232	2.7	1.4

\*SFs derived by obtaining the product of the relative potency estimates and the EPA SF for benzo(a)pyrene.

Source: Thorslund et al. (1986).

**Table 10**  
**RELEVANT STANDARDS AND CRITERIA FOR**  
**POLYCYCLIC AROMATIC HYDROCARBONS (PAHs)**

Standard or Criterion	Value	Reference
Slope Factors (SFs)		
Inhalation**	6.11 (mg/kg/day) <sup>-1</sup>	USEPA 1986b
Oral**	11.5 (mg/kg/day) <sup>-1</sup>	
Water Quality Criteria for Drinking Water	2.8 ng/L	USEPA 1986c
Occupational Exposure Limit		
OSHA (PEL)	0.2 mg/m <sup>3</sup> *	OSHA 1989

\*As coal tar pitch volatiles.

\*\*For benzo(a)pyrene.

**TRICHLOROETHENE (TCE)**

Trichloroethene (TCE) is a widely used industrial solvent, particularly in metal degreasing, which consumes the large majority of its production. Other miscellaneous applications include use in dry cleaning, as a low temperature heat exchange fluid, and in textile processing.

**Environmental Chemistry and Fate**

The relevant physical and chemical properties and environmental fate of TCE (CAS No. 79-01-6) are summarized below (Howard 1989; MacKay and Shiu 1981):

Chemical Formula	$C_2HCl_3$
Molecular Weight	131.5 g/mole
Physical state at 20°C	Liquid
Water Solubility	1,100 mg/L (25°C)
Density at 20°C	1.46 g/ml
Vapor Pressure	57.9 mm Hg (25°C)
Henry's Law Constant	$1.2E-03 \text{ atm}\cdot\text{m}^3/\text{mole}$
Octanol-Water Partition Coefficient (Log $K_{ow}$ )	2.38
Organic Carbon Partition Coefficient ( $K_{oc}$ )	126 mL/g
Bioconcentration Factor (BCF)	10.6

TCE's high vapor pressure and Henry's Law Constant indicate that volatilization will be the predominant removal mechanism for this compound from surface water and surficial soil. Its half-life in surface water has been estimated to range from 1 to 90 days, depending on water temperature, depth, turbulence, and movement of air across the water surface. In air, its half-life has been estimated to be 4 days or more, with its major degradation mechanism being photooxidation.

TCE's high water solubility and low to moderate log  $K_{ow}$  and  $K_{oc}$  indicate that TCE will be moderately to highly mobile in the subsurface. Since TCE is denser than water as a liquid and denser than air as a vapor, it has a marked tendency to sink through the unsaturated and saturated zones. TCE is biodegraded in the subsurface by reductive dehalogenation to dichloroethenes and vinyl chloride. Its half-life in

groundwater has been estimated to range from minimal at low concentrations to 4 to 9 months at higher concentrations (Smith and Dragun 1984).

TCE is moderately bioconcentrated in aquatic organisms.

### **Toxicokinetics**

TCE was absorbed almost completely (97%) following oral administration in rats (Dekant *et al.* 1984). No information was found on absorption following inhalation exposure; however, only 25% of an inhalation dose of tetrachloroethene was absorbed by human volunteers. TCE distributes throughout the body, but the highest levels are found in the adipose tissues, kidney, lung, adrenals, vas deferens, epididymis, brain, and liver. TCE is metabolized oxidatively to trichloroethylene oxide, trichloroacetaldehyde, trichloroacetic acid, monochloroacetic acid, trichloroethanol, and trichloroethanol glucuronide (EPA 1985b). TCE is eliminated from the body by exhalation of the original compound and urinary excretion of the metabolites (EPA 1987b).

### **Noncarcinogenic Effects**

TCE elicits a low level of acute toxicity, as reflected by the median lethal dose (LD<sub>50</sub>) of 4,920 mg/kg in rats, 3,200 mg/kg in mice, and 2,800 mg/kg in dogs. At near-lethal doses, mild changes in biochemical indices of liver function have been reported. Following short-duration inhalation exposure to high airborne concentrations, central nervous system (CNS) effects predominate. Chronic exposure of laboratory animals to TCE vapor has resulted in mild kidney and liver damage.

TCE exhibits no embryotoxic or teratogenic toxicity below maternally toxic levels (EPA 1985). Illustrative of this finding is the absence of significant results in a study by Schwetz *et al.* (1975) of the effects on offspring of mice and rats exposed to 300 ppm TCE (1,614 mg/m<sup>3</sup>) for 7 hours/day during the period of highest susceptibility to teratogenic effects (during days 6 to 15 of gestation).

### **Carcinogenicity and Mutagenicity**

There has been a series of animal bioassays evaluating the carcinogenicity of TCE following ingestion. The first of these studies showed a dose-related increased incidence of hepatocellular carcinomas in

$B_6C_3F_1$  mice receiving technical-grade TCE by stomach tube (gavage) (NCI 1976). Osborne-Mendel rats, exposed according to the same protocols as the  $B_6C_3F_1$  mice, showed no increases in tumors (NCI 1976). The finding of an increased incidence of hepatocellular carcinomas was confirmed in subsequent gavage studies, in which  $B_6C_3F_1$  mice received purified TCE (NTP 1982, 1986). Also in these studies, an increased incidence of kidney adenocarcinomas was reported in a high-dose group of male Fischer 344 rats (NTP 1982, 1986). Maltoni et al. (1986) reported an increased incidence of leukemia in male Sprague-Dawley rats receiving TCE by gavage.

Other oral studies tend to confirm the results or show deficiencies which limit their usefulness (EPA 1987a). A number of inhalation studies have also been performed. Of these, Maltoni et al. (1986) show a statistically significant increased incidence of hepatomas in male Swiss mice and male and female  $B_6C_3F_1$  mice, as well as hepatomas and lung tumors in  $B_6C_3F_1$  mice. Male Sprague-Dawley rats exposed under similar experimental protocols showed a statistically increased incidence in kidney tumors and Leydig cell tumors, as well as an increased incidence of leukemia (Maltoni et al. 1986). In another inhalation study, female Han:NMRI mice (but not Han:Wist rats) and Syrian hamsters exposed to TCE by inhalation showed an increased incidence of lymphomas (Henschler et al. 1980). However, viruses and/or immunosuppression may have enhanced susceptibility (Henschler et al. 1980; EPA 1987a). Fukuda et al. (1983) reported an increased incidence of lung tumors in ICR mice, but not in Sprague-Dawley rats, and Bell et al. (1978) reported an increased incidence of hepatocellular carcinomas in  $B_6C_3F_1$  mice, but not in Charles River rats exposed by inhalation to TCE.

There have been three cohort studies of TCE-exposed workers (Shindell and Ulrich 1985; Axelson et al. 1978; Tola et al. 1980), one malignant lymphoma case-control study (Hardell et al. 1981), and two surveys of liver cancer cases for TCE exposure (Paddle 1985; Novotna et al. 1978). EPA has concluded that these studies suffered from one or more deficiencies that diminish their usefulness and/or diminish their sensitivity to detect a human carcinogenic response: small sample size, absence of analyses by tumor site, problems with exposure definition, and problems with duration of exposure (EPA 1987a). Consequently, EPA

has judged the epidemiologic data to be inadequate for evaluating the carcinogenic potential of TCE (EPA 1987a).

Based upon the positive animal data and the absence of adequate human data, and according to its weight-of-evidence carcinogenicity criteria, EPA has placed TCE in Category B2, "probable human carcinogen," for both the oral and inhalation routes of exposure (EPA 1987a; 1990a).

Commercial TCE containing stabilizers (e.g., dioxane, etc.) has been reported to be weakly mutagenic in a variety of in vitro and in vivo assays representing a wide evolutionary range of organisms (EPA 1987a). Based on these data, EPA has concluded that commercial TCE may have the potential to cause weak or borderline increases above the spontaneous level of mutagenic effects in exposed human tissues (EPA 1987a).

#### Quantitative Indices of Toxicity

Based on the results of the NTP study (1983), EPA has derived slope factors for the oral and inhalation routes of exposure. The upper-bound estimate for the oral route, based upon the gavage data, is  $1.1 \times 10^{-2}$  (mg/kg/day)<sup>-1</sup> (EPA 1985; 1990a). The corresponding estimate for the inhalation data is  $1.3 \times 10^{-3}$  (EPA 1990a). Quantitative indices of toxicity are summarized in Table 1.

#### Standards and Criteria

Standards and criteria applicable to TCE are summarized in Table 2.

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Table 1

**INDICES OF TOXICITY  
FOR TRICHLOROETHENE**

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<u>Oral Route</u>	
Slope Factor (SF) (mg/kg-day) <sup>-1</sup>	1.1 x 10 <sup>-2</sup>
Weight of Evidence Classification	B2
Type of Cancer	Hepatocellular Carcinoma Malignant lymphoma
Test Species	Mice
SF Basis	Oral, inhalation
SF Source	IRIS - EPA 1990a
<u>Inhalation Route</u>	
Slope Factor (mg/kg-day) <sup>-1</sup>	1.3 x 10 <sup>-2</sup>
Weight of Evidence Classification	B2
Type of Cancer	Hepatocellular
Test Species	Mice
SF Basis	Oral exposure
SF Source	IRIS

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TABLE 2  
STANDARDS AND CRITERIA  
FOR TRICHLOROETHENE

Standard or Criterion	Value	Reference
<u>Drinking Water</u>		
National Primary Drinking Water Regulations		
MCL (a)	0.005 mg/L	EPA 1987
MCLG (b)	0	EPA 1987
<u>Surface Water</u>		
EPA Ambient Water Quality Criteria		
Aquatic Organisms and Drinking Water	0 (2.7 µg/L)*	EPA 1986
Adjusted for Drinking Water Only	0 (2.8 µg/L)*	EPA 1986
<u>Occupational Air Concentrations</u>		
OSHA PEL TWA	270 mg/m <sup>3</sup> (50 ppm)	OSHA 1989
OSHA STEL	1,080 mg/m <sup>3</sup> (200 ppm)	OSHA 1989
ACGIH TLV TWA	270 mg/m <sup>3</sup> (50 ppm)	ACGIH 1989

\* The concentration value given in parentheses corresponds to a risk of 10<sup>-6</sup>.

a MCL = Maximum contaminant level.

b MCLG = Maximum contaminant level goal.

**1,2,4-TRICHLOROBENZENE**

1,2,4-Trichlorobenzene (TCB) is the most widely used as a dye carrier in the textile industry. The pesticide industry consumes approximately 28% of produced TCB, in the manufacture of pesticides. In addition, 18% of TCB production is used in dielectric liquids and transformer oils. Miscellaneous uses include degreasing agents, septic tank and drain cleaner formulations, wood preservation, and abrasive formulations used in the manufacture of grinding wheels (EPA 1980).

**Environmental Chemistry and Fate**

The relevant physical chemical properties and environmental fate of TCB (CAS No. 120-82-1) are summarized below (Howard 1989; Weast 1983; MacKay and Shiu 1981; Mabey et al. 1982; U.S. Air Force 1989).

Molecular Formula	$C_6H_3Cl_3$
Molecular Weight	181.5
Physical State at 20°C	Liquid
Water Solubility (mg/L at 25°C)	4.88 E+01
Liquid Density (g/ml)	1.45
Vapor Pressure (mmHg at 25°C)	2.9E-01
Henry's Law Constant (atm-m <sup>3</sup> /mole)	4.33E-03
Octanol-Water Partition Coefficient (Log $K_{ow}$ )	4.02
Organic Carbon Partition Coefficient ( $K_{oc}$ )	9,200
Bioconcentration Factor (BCF)	630

The Log  $K_{ow}$ s, moderate  $K_{oc}$ , and low vapor pressure, indicate that absorption to soils and leaching to groundwater will copredominate for TCB in soils. Similarly, absorption to sediments and solvation will copredominate in groundwater and surface water. TCB has a higher density than water, causing TCB to sink in aqueous media.

The Log  $K_{ow}$  suggests that TCB will bioaccumulate. 1,2,4-TCB is not expected to be rapidly biodegraded in the environment. In most soil-groundwater systems, the concentration of microorganisms capable of biodegrading 1,2,4-TCB is expected to be low and drop off sharply with increasing depth.

### Toxicokinetics

In male rats and female rats at least 89% and 99% of 10 mg of radiolabelled TCB was absorbed from the gastrointestinal tract (Lingg et al. 1982). Kociba et al. (1981) has shown that TCB is absorbed in the respiratory tract following inhalation by rats, rabbits, and beagle dogs. Unfortunately, the latter study was not designed to provide quantitative absorption rate data.

### Noncarcinogenic Toxicity

Several animal studies have been reported on the subchronic toxicity of 1,2,4-TCB. 1,2,4-TCB inhalation studies of 1.5 to 6 months duration in rats, rabbits, dogs, and monkeys showed no major irreversible toxic effects, but reported transient histological changes and increased liver weight (Kociba et al. 1981; Carlson and Tardiff 1976). Increased uroporphyrins were also reported (Kociba et al. 1981; Watanabe et al. 1978).

### Reproduction and Development

A multigenerational study conducted indicated no adverse reproductive effects in rats receiving 400 mg/L of 1,2,4-TCB in drinking water (Robinson et al. 1981). Two studies indicated no teratogenic effects in rats ingesting 300 mg/kg/day 1,2,4-TCB (Ruddick et al. 1983) but reduced embryonic development was observed at a maternally toxic dose of 360 mg/kg (Kitchin et al. 1983).

### Carcinogenicity and Mutagenicity

Neither oral nor inhalation carcinogenicity bioassays could be located in the available literature. No oral or human carcinogenicity data could be located in the available literature.

Negative results were obtained in a Salmonella typhimurium reverse mutation assay in five strains without rat liver S-9 metabolic activation (Schoeny et al. 1979; Lawelor et al. 1979). However, this test system is insensitive to chlorinated compounds (Rinkus and Legator 1980).

According to its weight-of-evidence carcinogenicity guidelines, EPA has categorized 1,2,4-TCB in Group D - not classified (EPA 1990b).

### Quantitative Indices of Toxicity

Table 1 summarizes the quantitative indices of toxicity for 1,2,4-TCB.

### Standards and Criteria

Standards and criteria applicable to 1,2,4-TCB are summarized in Table 2.

### References

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Table 1A  
 INDICES OF TOXICITY  
 FOR 1,2,4-TRICHLOROBENZENE

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<u>Oral Route</u>	
Chronic Reference Dose (RfD) mg/kg/day	2.0E-02
Subchronic RfD (RfDs) mg/kg/day	2.0E-01
Confidence Level	NS
Critical Effect	Increased liver-to-body weight ratio
Test Species for Critical Effect	--
RfD Basis	Gavage
RfD Source	HEAST - EPA 1990b
Uncertainty Factor	
- Chronic RfD	1000
- Subchronic RfD	100

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<u>Inhalation Route</u>	
Chronic Reference Dose (RfD) mg/kg/day	3.0E-03
Subchronic RfD (RfDs) mg/kg/day	3.0E-02
Confidence Level	NS
Critical Effect	Increased uroporphyrin
Test Species for Critical Effect	--
RfD Basis	Inhalation
RfD Source	HEAST - EPA 1990b
Uncertainty Factor	
- Chronic RfD	1000
- Subchronic RfD	100

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NS = Not specified.



**Table 1B**  
**INDICES OF TOXICITY**  
**FOR 1,2,4-TRICHLOROBENZENE**

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<u>Oral Route</u>	
Slope Factor (SF) (mg/kg-day) <sup>-1</sup>	None
Weight of Evidence Classification	D
Type of Cancer	NA
Test Species	NA
SF Basis	NA
SF Source	IRIS - EPA 1990a
<u>Inhalation Route</u>	
Slope Factor (mg/kg-day) <sup>-1</sup>	None
Weight of Evidence Classification	D
Type of Cancer	NA
Test Species	NA
SF Basis	NA
SF Source	NA

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NA = Not applicable

**Table 2**  
**Standards and Criteria**  
**for 1,2,4-Trichlorobenzene**

Standard or Criterion	Value	Reference
<u>Drinking Water</u>		
National Primary Drinking Water Regulations		
Proposed MCL (a)	None	EPA 1990a
Proposed MCLG (b)	None	EPA 1990a
<u>Surface Water</u>		
EPA Ambient Water Quality Criteria		
Aquatic Organisms and Drinking Water	None	EPA 1990
Drinking Water Only	None	EPA 1990
<u>Occupational Air Concentrations</u>		
OSHA PEL Ceiling	40 mg/m <sup>3</sup> (5 ppm)	OSHA 1989
ACGIH TWA	40 mg/m <sup>3</sup> (5 ppm)	ACGIH 1989

\*Potential carcinogens have a criterion value of 0. A concentration of 0.8 µg/L corresponds to a risk of 10<sup>-6</sup>, as presented in water quality criteria documents.

a MCL = Maximum contaminant level.

b MCLG = Maximum contaminant level goal.

**TRANS-1,2-DICHLOROETHENE (t-1,2-DCE)**

Trans-1,2-dichloroethene (t-1,2-DCE) is widely used in industry. Applications include use as a solvent for fats, phenols, and camphor, as a refrigerant, as an additive to dye and lacquer solutions, as a low-temperature solvent for heat sensitive substances, and in rubber manufacturing, organic synthesis, and medicine. T-1,2-DCE is a synthetic chemical with no known natural sources. Monitoring studies have found that t-1,2-DCE is a relatively rare contaminant in groundwater, but levels as high as 40 µg/L have been reported. The majority of detected concentrations were below 0.5 µg/L (EPA 1985).

**Environmental Chemistry and Fate**

The relevant physical and chemical properties and environmental fate of t-1,2-DCE (CAS No. 156-60-5) are summarized below (Howard 1989; MacKay and Shiu 1981)):

Chemical Formula	$C_2H_2Cl_2$
Molecular Weight	97 g/mole
Physical State at 20°C	Liquid
Water Solubility	6,300 mg/L (25°C)
Density at 20°C	1.26
Vapor Pressure	340 mm Hg (25°C)
Henry's Law Constant	$6.5 \times 10^{-3} \text{ atm}\cdot\text{m}^3/\text{mole}$ (25°C)
Octanol-Water Partition Coefficient (Log $K_{ow}$ )	0.48
Organic Carbon Partition Coefficient ( $K_{oc}$ )	59 mL/g
Bioconcentration Factor (BCF)	1.6

The relatively high vapor pressure of t-1,2-DCE indicates that its predominant loss mechanism from surface water and surface soils is likely to be volatilization. Its half-life in surface water has been estimated to range from 1 to 6 days, depending on temperature, water turbulence, and air movement across the water surface. No half-life estimate in soils was found; however, evaporative loss from surface soils would probably have a half-life in the same range as the surface

water half-life. The half-life of t-1,2-DCE in air has been estimated to be <2 days.

Bulk t-1,2-DCE is denser than water, and t-1,2-DCE vapors are denser than air. Therefore, the compound has a tendency to sink through surface and groundwater when present as a bulk liquid and through the unsaturated zone as a vapor.

The low  $K_{oc}$  value indicates that t-1,2-DCE is only poorly adsorbed by soil particles and therefore only slightly retarded in its movement through the subsurface. In subsurface soils and groundwater, biologically mediated reductive dehalogenation to vinyl chloride is probably the primary fate (Smith and Dragun 1984). However, no half-life estimate has been found.

### **Toxicokinetics**

T-1,2-DCE is a low-molecular-weight, lipid-soluble material which should be readily absorbed by any route of exposure (oral, inhalation, dermal) (EPA 1984). No specific information on the distribution, metabolism, and excretion of t-1,2-DCE was found; however, if it behaves like the cis-isomer, rat studies indicate that it will concentrate in the liver and kidneys, undergo metabolism by liver microsomal enzymes, and most of a single dose will be excreted in the urine in a few days (EPA 1987).

### **Noncarcinogenic Effects**

At high concentrations over short-term periods, t-1,2-DCE, like other chlorinated ethylenes, possesses anesthetic properties. An oral median lethal dose ( $LD_{50}$ ) of 1,300 ppb for rats has been reported (Freundt et al. 1977). After prolonged exposure, severe pneumonic infiltration, changes in blood chemistry and cell counts, and liver degeneration with lipid accumulation have been observed (Freundt et al. 1977). No information on reproductive or developmental effects was found in the available literature.

### **Carcinogenic Effects**

No information was found in the literature on the carcinogenic potential of t-1,2-DCE. Several studies have failed to detect any mutagenic activity, with or without microsomal activation (EPA 1987).

### Quantitative Indices of Toxicity

No lifetime toxicity data were available for t-1,2-DCE. The oral reference dose (RfD) was derived based on data from a 90-day feeding study in which 1,2-DCE was administered to mice in drinking water (Barnes et al. 1985). Using a no-observed-adverse-effect level (NOAEL) of 0.1 mg/kg, EPA derived an oral RfD of 0.02 mg/kg/day (EPA 1990a). EPA has not derived an inhalation RfD for t-1,2-DCE.

The EPA confidence in the database is rated low because of the lack of chronic studies and the lack of data on reproductive and developmental toxicity. Quantitative indices of toxicity are summarized in Table 1.

### Standards and Criteria

Standards and criteria applicable to t-1,2-DCE are listed in Table 2.

### References

- American Conference for Governmental Industrial Hygienists (ACGIH), 1989, Threshold Limit Values and Biological Exposure Indices for 1989-1990.
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Table 1  
 INDICES OF TOXICITY  
 FOR TRANS-1,2-DICHLOROETHENE

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Oral Route

Chronic Reference Dose (RfD) mg/kg/day	0.02
Subchronic RfD mg/kg/day	0.2
Confidence Level	Low
Critical Effect	Increased serum alkaline phosphatase
Test Species for Critical Effect	Mice
RfD Basis	Drinking water
RfD Source	IRIS
Uncertainty Factor	
- Chronic RfD	1,000
- Subchronic RfD	100
Modifying Factor	
- Chronic RfD	1
- Subchronic RfD	1

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NS = Not specified.

**Table 2**  
**STANDARDS AND CRITERIA**  
**FOR TRANS-1,2-DICHLOROETHENE**

Standard or Criterion	Value	Reference
<u>Drinking Water</u>		
National Primary Drinking Water Regulations		
Proposed MCL (a)	0.1 mg/L	EPA 1989
Proposed MCLG (b)	0.1 mg/L	EPA 1989
<u>Surface Water</u>		
EPA Ambient Water Quality Criteria	Insufficient data to develop	EPA 1986
<u>Occupational Air Concentrations</u>		
OSHA PEL TWA	790 mg/m <sup>3</sup> (200 ppm)	OSHA 1989
ACGIH TLV TWA	790 mg/m <sup>3</sup>	ACGIH 1989

a MCL = Maximum contaminant level.

b MCLG = Maximum contaminant level goal.



## VINYL CHLORIDE

Vinyl chloride and polyvinyl chloride (PVC) are used as raw materials in the plastics, rubber, paper, glass, and automotive industries. In addition, vinyl chloride and PVC are used in the manufacture of electrical wire insulation and cables, piping, industrial and household equipment, medical supplies, food packaging materials, and building and construction materials. Man-made sources are responsible for all of the vinyl chloride found in the environment. Air in rural/remote and urban/suburban areas of the United States (U.S.) typically contain no detectable amounts of vinyl chloride (Stephens et al. 1986). In areas near polyvinyl chloride and vinyl chloride manufacturers, the concentration of vinyl chloride in air typically ranges from trace levels to 105  $\mu\text{g}/\text{m}^3$  (ATSDR 1989).

Vinyl chloride has been identified in some surface, ground and drinking waters in the U.S. In a 1982 EPA groundwater supply survey, vinyl chloride was identified in less than 1% of groundwater supplies. The maximum concentration detected was 8.4  $\mu\text{g}/\text{L}$  (Westrick et al. 1984).

### Environmental Transport and Fate

The relevant physical and chemical properties and environmental fate of vinyl chloride (CAS No. 75-01-4) are summarized below (Howard 1989; McKay and Shiu 1981).

Chemical Formula	$\text{CH}_2\text{CHCl}$
Molecular Weight	63 g/mole
Physical State at 20°C	gas
Water Solubility	2,760 mg/L (25° C)
Vapor Density	2.15
Vapor Pressure	2,660 mm Hg (25° C)
Henry's Law Constant	$6.9\text{E}-01 \text{ atm}\cdot\text{m}^3/\text{mole } (^\circ\text{C})$
Octanol-water Partition Coefficient (Log Kow)	1.38
Organic Carbon Partition Coefficient (Koc)	57
Bioconcentration Factor (BCF)	0.91

Vinyl chloride has a high water solubility and vapor pressure. As a consequence of these two properties, vinyl chloride can be characterized as a highly mobile chemical. For vinyl chloride released to air, some rainwater washout is anticipated. After deposition in water or soil, volatilization is expected to return some portion back to the atmosphere. Based on vinyl chloride's high Henry's Law Constant, following release to water, volatilization will result in substantial loss to the atmosphere. Vinyl chloride's half-life in a typical pond, river, and lake has been estimated, to be 43.3, 8.7, and 34.7 hours, respectively (EPA 1985). In waters containing photosensitizers, such as humic materials, photodegradation may be fairly rapid (ATSDR 1989). The half-life for vinyl chloride in air, based on reaction with photochemically generated hydroxyl radicals, has been estimated to range from 1.5 to 1.8 days (EPA 1985).

Due to its high water solubility, high vapor pressure and moderate Koc, adsorption to sediments is not expected to be a major surface water fate process.

Vinyl chloride released to soil can be transported to air via volatilization, to surface water via runoff and groundwater via leaching. The first two pathways predominate in surficial soil, whereas the latter predominates at lower soil depths (EPA 1985).

According to criteria developed by Kenaga (1980), vinyl chloride with a Koc of 57, would be considered to be mobile in soil.

Based on data in aquatic media, chemical reaction of vinyl chloride in soil does not appear to be a significant fate process, and it appears that vinyl chloride in soil would be resistant to biodegradation under aerobic conditions (ATSDR 1989). In soils, vinyl chloride is a biodegradation product of trans-1,2-dichloroethene, tetrachloroethene, 1,1,1-trichloroethane, and trichloroethane.

### **Toxicokinetics**

Vinyl chloride is readily absorbed via inhalation and ingestion, but poorly absorbed through intact skin (ATSDR 1989). Vinyl chloride metabolism occurs primarily in the liver, with toxicity apparently attributable to enzymatic conversion to reactive polar intermediates. Three alternative metabolic pathways have been postulated for vinyl

chloride following oral or inhalation exposure (Hefner 1975b). At low concentrations, sequential oxidation to 2-chloroethanol, 2-chloroacetaldehyde and 2-chloroacetic acid involving alcohol dehydrogenase appears to predominate. When this dehydrogenase becomes saturated, 2-chloroethanol may be oxidized by catalase in the presence of hydrogen peroxide. The third pathway involves oxidation of vinyl chloride by a mixed function oxidase to form 2-chloroethylene oxide which spontaneously rearranges to 2-chloroacetaldehyde.

At low oral doses (less than 1 ug/kg) of vinyl chloride the metabolites are excreted primarily in the urine, whereas, at high oral doses (greater than 100 ug/kg), most of the chemical is expired as vinyl chloride (EPA 1985).

### **Noncarcinogenic Effects**

Studies on humans working in vinyl chloride plants suggest that noncarcinogenic systemic effects may occur above an airborne concentration of 50 ppm. At levels upwards to and exceeding 1000 ppm, workers reported dizziness, headaches and/or euphoria. Long-term exposure at these levels have resulted in numerous pulmonary, gastrointestinal, cardiovascular and central nervous system effects. Data are insufficient to characterize effects at low air concentration.

### **Reproduction and Developmental Effects**

Infante et al. (1976a,b) reported an association between human exposure to vinyl chloride and birth defects and fetal loss, but this association was contradicted by Edmonds et al. (1975) and Hatch et al. (1981). Inhalation exposure of rats and rabbits to vinyl chloride concentrations as high as 2,500 ppm ( $6,500 \text{ mg/m}^3$ ) on days 6 to 15 (rats) and 6 to 18 (rabbits) of gestation, and mice to vinyl chloride levels as high as 500 ppm ( $2,300 \text{ mg/m}^3$ ) on days 6 to 15 of gestation, did not induce teratogenic effects but did increase skeletal variants in the high dosed group of mice (John et al. 1977). A developmental effects study with vinyl chloride in rats exposed by inhalation to 600 or 6,000 ppm ( $2,160$  or  $21,160 \text{ mg/m}^3$ ) 4 hours daily on days 9 through 21 of gestation was negative for teratogenicity and inconclusive for fetotoxicity (Radike et al. 1977).

### Carcinogenicity and Mutagenicity

Elevated angiosarcoma mortality among workers occupationally exposed to vinyl chloride have unequivocally shown vinyl chloride to be a human carcinogen. EPA has placed vinyl chloride according to its carcinogenicity weight-of-evidence criteria in category A, "human carcinogen" (EPA 1990a). In addition to definitive human evidence, several long-term bioassays have demonstrated increased incidence of tumors in mice, rats, and hamsters.

Studies of vinyl chloride effects in occupationally exposed workers showed increases in the number of chromosome aberrations in peripheral lymphocytes particularly at exposure concentrations above the current 1 ppm Occupational Safety and Health Administration Standard. Numerous in vitro assays have shown vinyl chloride metabolites, but not the parent compound, to be mutagenic.

### Quantitative Indices of Toxicity

EPA has derived an estimated oral slope factor (SF) of 2.3 (mg/kg/day)<sup>-1</sup> (EPA 1987, EPA 1990a) based upon the incidence of lung and liver tumors in male and female rats exposed dietarily to vinyl chloride for a lifetime (Feron et al. 1981). EPA has also derived an inhalation SF (EPA 1990a) of 2.95 X 10<sup>-1</sup> (mg/kg/day)<sup>-1</sup> based upon the incidence of liver angiosarcomas in male and female rats exposed for up to 1 year to concentrations of up to 30,000 ppm (Maltoni et al. 1980). EPA has not derived a noncarcinogenic reference dose for either oral or inhalation exposure to vinyl chloride (EPA 1990a,b). Table 1 summarizes the quantitative indices of toxicity for vinyl chloride.

### Standards and Criteria

Standards and criteria applicable to vinyl chloride are listed in Table 2.

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Table 1  
INDICES OF TOXICITY  
FOR VINYL CHLORIDE

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<u>Oral Route</u>	
Slope Factor (SF) (mg/kg-day) <sup>-1</sup>	2.3
Weight of Evidence Classification	A
Type of Cancer	Lung
Test Species	Rat
SF Basis	Diet
SF Source	HEAST - EPA 1989b
<u>Inhalation Route</u>	
Slope Factor (mg/kg-day) <sup>-1</sup>	2.95 x 10 <sup>-1</sup>
Weight of Evidence Classification	A
Type of Cancer	Liver
Test Species	Rat
SF Basis	1 year inhalation study
SF Source	HEAST; EPA 1989b

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TABLE 2  
STANDARDS AND CRITERIA FOR VINYL CHLORIDE

Standard or Criterion	Value	Reference
<u>U.S. Primary Drinking Water Standards</u>		
MCL	0.002 mg/L	EPA 1990a
MCLG	0	EPA 1990a
<u>Surface Water</u>		
EPA Ambient Water Quality Criteria Human Health	0.002 mg/L (1 X 10 <sup>-6</sup> risk)	EPA 1986b
<u>Occupational Air Concentrations</u>		
OSH PEL TWA	2 mg/m <sup>3</sup> (1 ppm)	OSHA 1989
ACGIH TLV TWA	10 mg/m <sup>3</sup> (5 ppm)	ACGIH 1989

MCL: Maximum Contaminant Level.  
MCLG: Maximum Contaminant Level Goal.



ZINC CAS No.: 7440-66-6

Zinc is a naturally occurring element usually found in the form of zinc sulfide ores. Smelting or electrolytic processing are the two most common methods for extracting zinc from the ore. Zinc is commonly used as a protective coating of other metals and in alloys such as bronze and brass. Zinc is also used in organic chemical extractions and reductions.

Zinc may be released to the atmosphere as dust and fumes from zinc production facilities, lead smelters, brass works, automobile emissions, fuel combustion, incineration, and soil erosion (ATSDR 1988). Zinc concentrations in the ambient air in urban areas have been found to range from 0.1 to 1.7  $\mu\text{g}/\text{m}^3$ . Near smelting operations, concentrations as high as 15.7  $\mu\text{g}/\text{m}^3$  were reported (ATSDR 1988).

Urban runoff, mine drainage, and municipal and industrial effluents are concentrated sources of zinc released to water. The maximum, minimum, and average concentrations in 1,577 raw surface waters in the United States were 1.183, 0.002, and 0.064 mg/L, respectively. There was a 76.5% frequency of detection. The maximum, minimum, and average concentrations in 380 finished waters in the United States were 2.01, 0.003, and 0.08 mg/L, respectively (NRC 1977). Higher mean concentrations of zinc in finished water may be due to water treatment and/or transport through pipe systems.

Data pertaining to ambient concentrations of zinc in soil are limited. Zinc is generally found in soils at concentrations between 10 mg/kg and 300 mg/kg with a mean of approximately 50 mg/kg (USEPA 1980). The major source of zinc for the general population is food (EPA 1987). Meat products contain relatively high concentrations (6 mg/kg in potatoes and 8 mg/kg in grains). An average zinc intake in man is on the order of 0.14 to 0.21 mg/kg/day (NRC 1977).

#### ENVIRONMENTAL CHEMISTRY AND FATE

Zinc is a metal belonging to Group IIB of the periodic table. Its natural valence states are 0 and 2+. Zinc forms a variety of inorganic compounds and a number of compounds with organic ligands. The element is amphoteric and forms both acidic and basic salts.

In the atmosphere zinc is expected to be present as dust and fumes

from zinc production facilities, lead smelts, brass works, vehicular emissions, fuel combustion, incineration, and soil erosion. Atmosphere is found preferentially in the smaller diameter particles (<3 um in aerodynamic diameter). Smaller particles have longer atmospheric residence times than larger particles; however, no estimate of the atmospheric half-life is available.

Zinc introduced into the aquatic environment is partitioned into sediments through sorption onto hydrous iron and manganese oxides, clay minerals and organic material; a small part may be partitioned into the aquatic phase through speciation into soluble zinc compounds. Sorption of zinc is probably the dominant fate of zinc in the aquatic environment.

Information regarding the fate of zinc in soil is inadequate. However, zinc is likely to be strongly sorbed onto soil. Soil conditions not amenable for the sorption of zinc may lead to the leaching of zinc. The tendency of zinc to be sorbed is affected by the pH and salinity of soils. Decrease of pH (<7) and increase of soil salinity favors desorption (U.S. EPA, 1980).

The bioconcentration factors (BCFs) for zinc in aquatic organisms have been determined by several investigators (U.S. EPA, 1980). BCFs for zinc in edible portions of aquatic organisms have been found to vary from 43 in soft-shell clam, Mya arenaria, to 16,700 in oyster, Crassostrea virginica (U.S. EPA, 1980).

### Toxicokinetics

Zinc is an essential element and its uptake from the gastrointestinal tract depends in part on the zinc status of the organism. Zinc levels in the body are rigidly controlled by a homeostatic mechanism. Zinc is absorbed and excreted from the gastrointestinal tract. Apparent absorption ranges from 20% to 80% with a mean of about 50%. Many dietary factors, including protein, phytate, and fiber content, seem to influence zinc absorption. In one study in which human volunteers ingested zinc added to bread during baking, zinc absorption was reported to range from 12% to 39%, with an average of 25%.

No quantitative studies of zinc absorption via inhalation were found. However, increased serum and plasma zinc levels have been observed following inhalation of zinc oxide fumes. An indeterminate amount of this absorption may have resulted from ingestion of zinc bearing particles following ciliary clearance from the respiratory tract.

### Noncarcinogenic Effects

Zinc is an essential trace element in human and animal nutrition. In the body it is found in high concentrations in male reproductive organs, pancreatic islets, muscle, kidney, liver, and bone. It is essential for the activity of some enzymes (U.S. EPA, 1980). The human recommended daily allowance (RDA) of zinc for adults is 15 mg (NAS, 1980). Zinc appears to be toxic only at levels at least an order of magnitude greater than the recommended daily allowance (RDA); toxicity appears to result from an overload of the homeostatic mechanism for absorption and excretion of zinc.

Acute toxicity resulting from the ingestion of food stored in galvanized containers has produced symptoms including severe diarrhea, abdominal cramping, nausea, and vomiting.

There are a variety of reports in the literature concerning subchronic exposure to zinc, including both experimental toxicological and therapeutic studies. Most commonly no adverse effects of zinc were noted. Subchronic occupational exposure in humans has resulted almost exclusively from inhalation of zinc fumes or dusts and has been associated with a condition called "metal fume fever." This condition has also been associated with chronic exposure and is characterized by flu-like symptoms including throat irritation, body aches, weakness, fatigue, and general malaise followed by fever and chills. The syndrome commonly runs its course in 24 to 48 hours. "Immunity" to the syndrome quickly develops among workers, but it is also quickly lost on cessation of exposure so that the syndrome most commonly re-occurs after several days away from work.

No teratogenic effects were observed in one experimental study in rats. However, reduced copper concentrations in the liver and other organs of the fetuses were found. There are a small number (3) of case

reports of premature deliveries, including one stillborn fetus, in pregnant women who took zinc dietary supplements during the third trimester. Zinc supplementation in animals has resulted in increased fetal resorption. Zinc supplementation for pregnant women has been recommended, but because of the known interaction between zinc and copper, excessive zinc supplementation for prolonged times could have an adverse effect on the fetus. It is also well-documented that zinc deficiency during pregnancy may have an adverse effect on the fetus (NRC, 1978).

No reports of teratogenicity or fetotoxicity in man or animals associated with inhalation of zinc or its compounds have been found in the available literature.

#### **Carcinogenicity and Mutagenicity**

No pertinent reports associating zinc with cancer in humans were found in the available literature, nor were any reports of bioassays of zinc or its compounds available. Accordingly, zinc and its compounds have been placed in EPA's carcinogenicity weight-of-evidence category D - Not classified.

No data were found concerning mutagenicity of zinc or its compounds.

#### **Quantitative Indices of Toxicity**

Table 1 summarizes the quantitative indices of toxicity for zinc.

EPA has derived an oral reference dose (RfD) of  $2 \times 10^{-1}$  mg/kg/day based upon studies presented by Pories et al. (1967) and Prasad et al. (1975).

#### **STANDARDS AND CRITERIA**

Table 2 summarizes relevant standards and criteria for zinc.

Standards for zinc in food are not currently available and zinc in food surveys are not routinely conducted by US regulatory agencies. The Food and Drug Administration's (FDA's) contaminants team are proposing to develop criteria for evaluating inorganic contaminants in shellfish (FDA 1989).

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**Table 1**  
**INDICES OF TOXICITY**  
**FOR ZINC**

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Oral Route

Chronic Reference Dose (RfD)	$2 \times 10^{-1}$
Subchronic RfD (RfDs)	$2 \times 10^{-1}$
Confidence Level	NS
Critical Effect	Anemia
Test Species for Critical Effect	Human
RfD Basis	Therapeutic dosage
RfD Source	HEAST
Uncertainty Factor	
- Chronic RfD	10
- Subchronic RfD	10

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Table 2  
STANDARDS AND CRITERIA  
FOR ZINC

Standard or Criterion	Value	Reference
<u>Drinking Water</u>		
National Secondary Drinking Water Regulations		
SMCL (a)	5 mg/L	EPA 1989
<u>Surface Water</u>		
EPA Ambient Water Quality Criteria		
Water Ingestion	5 mg/L	ATSDR 1988
<u>Occupational Air Concentrations</u>		
OSHA PEL TWA		
• Zinc chloride fume	1 mg/m <sup>3</sup>	OSHA 1989
• Zinc oxide fume	5 mg/m <sup>3</sup>	OSHA 1989
• Zinc oxide dust	10 mg/m <sup>3</sup>	OSHA 1989
ACGIH TWA		
• Zinc chloride fume	0.01 mg/m <sup>3</sup>	ACGIH 1989
• Zinc chromates	0.01 mg/m <sup>3</sup>	ACGIH 1989
• Zinc oxide fume	5 mg/m <sup>3</sup>	ACGIH 1989
• Zinc oxide dust	10 mg/m <sup>3</sup>	ACGIH 1989

(a) SMCL is a secondary contaminant level based on aesthetics rather than adverse health effects.

## CARBON DISULFIDE

Carbon disulfide ( $CS_2$ , also known as carbon bisulfide and dithiocarbonic anhydride) is a colorless, highly flammable, and highly volatile liquid used to manufacture rayon, cellophane and carbon tetrachloride. Applications developed during the 19th century still prevail in the manufacture of flotation agents, herbicides, insecticides, ammonium thiocyanate, sodium thiocyanate, xanthogenates, electric vacuum tubes, etc. Carbon disulfide can be utilized as a direct reactant, a chemical intermediate, and as a solvent (Timmerman 1978). As a solvent, it is used by industry for phosphorus, sulfur, selenium, bromine, iodine, fats, resins, and rubber (CMR 1983; Berg 1981; Windholz 1983).

Carbon disulfide exists in the atmosphere at concentrations typically around 0.02 ppb (Brimblecombe 1986). Carbon disulfide is emitted from a wide variety of man-made and natural sources (Hoshika *et al.* 1975; Graedel 1978; Beard and Guenzi 1983; Aneja *et al.*, 1982; Steudler and Peterson 1984; Farwell *et al.* 1979; Adams *et al.* 1979; Lovelock 1974). The major anthropogenic source of carbon disulfide emissions are viscose rayon manufacturing sites (U.S. Environmental Protection Agency [EPA] 1980); however, the largest global sources are natural (Khalil and Rasmussen 1984). Based on average atmospheric concentrations in rural and urban areas of the United States (Brodzinsky and Singh 1982), the average daily intakes of carbon disulfide by inhalation in these areas are 0.0026 and 0.0041 mg, respectively.

## ENVIRONMENTAL CHEMISTRY AND FATE

Physical and chemical properties relevant to the environmental fate and transport of carbon disulfide (CAS#: 75-15-0) are summarized below.

<u>Property</u>	<u>Value</u>	<u>Source</u>
Chemical formula:	$CS_2$	
Molecular weight:	76.13	
Water solubility (at 20°C):	2,940 mg/l	Windholz 1983
Log $K_{ow}$ (calculated):	2.16	Leo <i>et al.</i> 1971
$K_{oc}$ (calculated):	54	EPA 1986a



Density (at 20/4°C):	1.2632	Windholz 1983
Vapor pressure (at 20°C):	297 mmHg	Timmerman 1978
Henry's Law Constant:	$1.23 \times 10^{-2}$ atm · m <sup>3</sup> /mol	EPA 1986b
Bioconcentration Factor (BCF):	≤26	EPA 1986a

### TRANSPORT

Carbon disulfide's high vapor pressure and Henry's Law Constant indicates that it will rapidly evaporate from surface waters and soils. Its relatively high water solubility and low  $K_{oc}$  indicate that it will be highly mobile in the subsurface. Its low BCF indicates that it will not accumulate significantly in aquatic organisms.

### FATE

Carbon disulfide appears to be stable in aqueous media and soil under environmentally relevant conditions. It does not undergo hydrolysis, oxidation, or photolysis under these conditions (EPA 1986a). Since carbon disulfide has been used as a soil fumigant because of its toxicity to bacteria, it is unlikely to undergo significant biodegradation in soil or water (EPA 1986a).

In the atmosphere, carbon disulfide appears to be susceptible to oxidation by hydroxide radicals, atomic oxygen, and ozone. The half-life of carbon disulfide in these processes has been estimated to range from three to 150 days. Khalil and Rasmussen (1984) estimated the overall lifetime of atmospheric carbon disulfide to be about 13 days.

### ENVIRONMENTAL CONCENTRATIONS

#### Air

Khalil and Rasmussen (1984) estimated emissions of carbon disulfide to the atmosphere in the northern hemisphere as follows:

<u>Source</u>	<u>Metric tons/year</u>
oceans	300,000
soils	640,000
marshes	50,000
volcanoes	10,000
chemical industries	260,000
sulfur recovery	70,000

Brodzinsky and Singh (1982) measured carbon disulfide at 61 rural/remote and 88 urban/suburban sites in the United States; the average concentrations found were 41 and 65 parts per trillion (ppt), respectively. Using these concentrations and assuming an average adult breathing rate of  $20\text{m}^3/\text{day}$ , the average daily exposure to carbon disulfide in the U.S. would be 0.0026 and 0.0041 mg in rural and urban areas, respectively (EPA 1986a).

Air concentrations of carbon disulfide of 16 to  $40\text{mg}/\text{m}^3$  have been reported at viscose rayon plants (Westburg et al. 1984; Ciccollelm and Vincent 1984).

### **Water**

Carbon disulfide has been identified, but not quantified in drinking waters of Miami, Florida, Cincinnati, Ohio (EPA 1975), and New Orleans, Louisiana (EPA 1974), and in sewage treatment plant effluents. It has also been found in Lake Ontario at levels ranging from a trace to 3.9 ppm as well as in the Niagara River waters at levels of 0 to 25 ppb (Kaiser et al. 1983). Lovelock (1974) reported levels of carbon disulfide in the open Atlantic Ocean, in mud at the sea bottom, in the Atlantic Ocean off Ireland and in stagnant bay waters of 0.52, 29.5, 0.78, and 5.4 ng/l, respectively. Lovelock speculates that anaerobic conditions on the sea floor are the source of the carbon disulfide.

### **Human Exposure**

Carbon disulfide has been detected in waters, ambient air, grains that were fumigated with this compound, and the mother's milk of rayon

factory employees. Women from Bayonne, New Jersey, Jersey City, New Jersey, Bridgeville, Pennsylvania and Baton Rouge, Louisiana were identified as having carbon disulfide in their breast milk. Seventeen rayon factory employees who were breast-feeding were found to have concentrations of 6.8 to 12.3  $\mu\text{g}/100\text{ ml}$ . Contamination of milk was still detected before and after work hours, and even after 23 to 57 days away from the job (Cai and Bao 1981).

## PHARMACOKINETICS

### Absorption

In humans, carbon disulfide is absorbed substantially from the lungs. Herrman et al. (1982) reported that volunteers exposed to 6 to 108 mg carbon disulfide/ $\text{m}^3$  for 30 minutes retained an average of 52.4% of the inhaled chemical in the lungs (retention was 48.7% during the last 5 minutes of exposure). It has been reported that retention of carbon disulfide is approximately 70 to 80% initially and then declines to a steady state of 15 to 45% within 1 to 2 hours (Beauchamp et al. 1983; World Health Organization (WHO) 1981).

### Distribution

Studies on rats and mice indicate that carbon disulfide and its metabolites are distributed rapidly to body fat and highly perfused tissues (McKenna and DiStefano 1977; Bergman et al. 1984). Carbon disulfide is eliminated more rapidly from the tissues than are its metabolites, but neither appears to bioaccumulate in any tissue (McKenna and DiStefano 1977). Danielsson et al. (1984) also showed that carbon disulfide and its metabolites can cross the placenta throughout gestation, with metabolites accumulating in the bone, liver and neuroepithelium of the fetus.

### Metabolism

Studies on animals and humans suggest that carbon disulfide is metabolized by reaction with amino acids or reduced glutathione to form thiocarbamates or conjugated glutathione, and by reactions catalyzed by cytochrome P450 to form reactive sulfur. Reactive sulfur may further

react to form thiourea, carbonyl sulfide, or monothiocarbamate (McKenna and DiStefano 1977; Van Doorn et al. 1981; Soucek and Madlo 1953; Chengelis and Neal 1980, 1981; Bond and DeMatteis 1969; Freundt and Dreher 1969; Hunter and Neal 1975; Freundt and Kuttner 1969; Freundt et al. 1976; Mack et al. 1974; DeMatteis and Seawright 1973; Dalvi et al. 1974, 1975; Dalvi and Neal 1978; DeMatteis 1974; Zatignani and Neal 1975).

### **Excretion**

In humans, 10-30% of absorbed carbon disulfide is excreted unchanged in expired air (Soucek 1957; McKee et al., 1943; Demus 1964) and <1% is excreted unchanged in urine (Soucek 1957; Soucek and Pavelkova 1953; McKee et al. 1943). The remaining carbon disulfide is excreted primarily as metabolites in the urine (Beauchamp et al. 1983). Urinary excretion appears to be proportional to the exposure concentration.

### **TOXICITY**

#### **Carcinogenic Effects**

Exposure to carbon disulfide has not been proven to be carcinogenic. It has been reported, however, that there is an association between lymphocytic leukemia and multiple solvent exposure (Checkoway et al. 1984; Wilcosky et al. 1984). The cases of 11 hourly male workers from the U.S. rubber industry whose deaths were attributed to lymphocytic leukemia were studied and compared with exposure records from the plants. It was found that of the 24 solvents to which workers were exposed, carbon disulfide and carbon tetrachloride were strongly associated with leukemia (McMichael et al. 1976).

#### **Mutagenicity**

Mutagenicity studies with carbon disulfide have yielded primarily negative results. Genotoxicity has been reported in foreign literature, but omission of experimental details precludes interpretation of these studies (Beauchamp et al. 1983).

### Teratogenicity

Administration of carbon disulfide to CD rats and New Zealand white rabbits by gavage resulted in adverse maternal and developmental effects including decreased average fetal body weight, maternal toxicity (abnormal posture, rigidity, or paralysis of hind limbs, ataxia, lethargy, and rough or erect coat), a significant dose-related reduction in maternal weight gain and elevated liver weights (Jones-Price et al. 1984a,b). Salnikova and Chirkova (1974) observed "slight embryotoxicity" in rats exposed to 4.2 ppm throughout gestation; however, failure to report daily exposures makes these results difficult to interpret. In these studies, congenital malformations such as hydrocephalus, club foot, and tail deformation were observed in the F<sub>1</sub> generation at 100 and 200 mg/m<sup>3</sup>. The incidence and severity of these malformations increased in the F<sub>2</sub> generation. It is suggested that carbon disulfide interferes with hormonal regulation of prenatal development, which may result in permanent alteration of the xenobiotic metabolizing function of the body and impaired future ability to detoxify carbon disulfide (EPA 1986).

### Other Reproductive Effects

Based on studies in rats and human occupational exposure, it has been suggested that inhaled carbon disulfide may reduce male reproductive performance (reduced sperm count) (Zenick et al. 1984a,b; Tepe and Zenick 1984; Cirila et al. 1978; Lancrajan et al. 1969; Lancrajan 1972).

In a study in which "mongrel" rats were administered distilled CS<sub>2</sub> dissolved in peanut oil intraperitoneally, rats treated with 25 mg/kg of CS<sub>2</sub> every second day for 60 days had thickened vascular walls in the testis and a reduced number of spermatozoa in the tubular lumen. In rats dosed with 25 mg/kg CS<sub>2</sub> (120 days, every other day), advanced regressive lesions were found in all parts of the testis (Gondzik 1971).

### NONCARCINOGENIC EFFECTS

Studies show that repeated ingestion of CS<sub>2</sub> causes neurologic and hematologic damage. Occupational exposure to inhaled carbon disulfide is associated with cardiovascular, neurological, immunological, and ocular disorders (Beauchamp et al. 1983).

### Neurological Effects

Case reports from occupational exposures suggest that carbon disulfide at concentrations of 144 to 321 ppm may cause psychosis, polyneuritis (absent or weakened Achilles or patellar reflexes), tremors, weakness of limbs, myopathy, and vertigo (Vigliani 1954). Epidemiological studies have also linked exposure to psychological disorders (Hanninen 1971; Tuttle et al. 1976).

### Cardiovascular Effects

Occupational exposure to carbon disulfide at concentrations of 10 to 161 ppm has been associated with an increased risk of mortality from coronary heart disease (National Institute of Occupational Safety and Health [NIOSH] 1978).

### Ophthalmological Effects

A majority of occupational exposure reports indicate ocular damage due to carbon disulfide exposure, but the exact nature of the damage is widely disputed (EPA 1986).

## STANDARDS AND CRITERIA

Standards and criteria relevant to carbon disulfide are listed in Table 1.

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**Table 1**  
**STANDARDS AND CRITERIA FOR CARBON DISULFIDE**

Standard or Criterion	Value	Reference
<b><u>Reference Dose</u></b>		
Oral	0.1 mg/kg/day	EPA 1990a,b
Inhalation	0.01 mg/kg/day	EPA 1990a,b
<b><u>Drinking Water</u></b>		
National Primary Drinking Water Regulations		
MCL(a)	Not established	
MCLG(b)	Not established	
DWEL(c)	0.7 mg/L	Calculated from reference dose
<b><u>Surface Water</u></b>		
EPA Ambient Water Quality Criteria	None established	
<b><u>Occupational Air Concentrations</u></b>		
OSHA PEL TWA	12 mg/m <sup>3</sup> (Skin)	OSHA 1989
OSHA PEL STEL	36 mg/m <sup>3</sup>	OSHA 1989
ACGIH TLV TWA	31 mg/m <sup>3</sup> (Skin)	ACGIH 1990
NIOSH REL TWA	3 mg/m <sup>3</sup> (Skin)	ACGIH 1990
NIOSH REL STEL	30 mg/m <sup>3</sup>	ACGIH 1990

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**Key:**

- (a) MCL = Maximum Contaminant Level
- (b) MCLG = Maximum Contaminant Level Goal
- (c) DWEL = Drinking Water Equivalent Level

APPENDIX F

CONTAMINANT MIGRATION ESTIMATION METHODS

This appendix briefly describes the models, assumptions, and input data used to estimate chemical concentrations in surface water or ambient air to which workers or recreational fishermen may be exposed.

#### River Dispersion and Volatilization Loss Model

The model for mixing and dilution in a river described in Thomann and Mueller (1987) was applied to estimate exposure concentrations in the Niagara River, as well as the volatilization loss to the ambient air for volatile chemicals.

The discharge from the Falls Street tunnel into the lower Niagara River was modeled as a point source to the river. The contaminants assumed to be entering the river at this point source are the groundwater contaminants detected in the vicinity of the site, which are assumed to be captured by the Falls Street tunnel and to flow without degradation through the length of the tunnel.

Two downstream locations were chosen as points where river concentrations were modeled: the Whirlpool (for fishermen exposure), and the mouth of the Niagara River at Lake Ontario (for residential drinking water exposure).

The inorganics were modeled as conservative (non-decaying) dissolved pollutants. At any downstream location, the in-stream concentration is given by:

$$s = \frac{W}{Q}$$

where

$s$  = concentration (assuming complete mixing) in mg/L

$W$  = total mass loading rate of a given inorganic (metal or cyanide), in mg/day

$Q$  = river flow in L/day

In applying this model, the annual average diverted Niagara River flow of 100,000 cfs, or  $2.45 \times 10^{11}$  L/day, was used for  $Q$ . (The actual average annual flow of the river is 200,000 cfs. However, up to 100,000 cfs is diverted around the Niagara Falls area for public and industrial

use. Thus, the flow model uses 100,000 cfs because of the diversion.) The mass loadings  $W$  were calculated as described elsewhere.

The river concentrations of organic compounds due to the point discharge from the tunnel were modeled using the model for nonconservative pollutants from a point source:

$$S_1 = S_{10} e^{-K_{11} X_1/U_1}$$

where

$S_1$  = concentration of non conservative pollutant due to point source, mg/L

$S_{10}$  = maximum concentration in river immediately after complete mixing with point discharge, mg/L

$K_{11}$  = volatilization rate, day<sup>-1</sup>

$X_1$  = distance downstream from the point discharge, feet

$U_1$  = average river velocity, in feet/day

where

$$S_{10} = \frac{W_1}{Q_1}$$

where

$W_1$  = pollutant mass loading from point source, in mg/day

$Q_1$  = average river flow in reach just downstream from point source, in L/day. ( $Q_1 = Q$ )

The average river velocity was calculated as follows:

$$U_1 = \frac{Q}{A}$$

where

$$A = B \cdot H$$

where

$A$  = average river channel effective cross-sectional area, ft<sup>2</sup>

$B$  = average river channel width, ft

$H$  = average river channel depth, ft



The volatilization rate  $K_{11}$  was calculated using the chemicals' Henry's Law constants and the estimated stream reaeration coefficient (see Thomann and Mueller, pp. 532-536).

Table F-1 summarizes the site-specific input parameter values assumed in applying the river dispersion model.

Volatilization rates of organic compounds from the river surface were calculated at the assumed exposure locations, and were then used as vapor source strengths as inputs to appropriate air dispersion models in order to ultimately calculate ambient air exposure concentrations of vapors for nearby fishermen or workers. The total river volatilization rate was calculated as follows (see Thomann and Mueller, p. 532):

$$Q = k_1 \cdot A_s \cdot f_d \cdot C_T$$

where

$Q$  = total mass loss rate from river due to volatilization, mg/s

$k_1$  = volatilization rate in m/s

$A_s$  = river/air<sub>2</sub> interface surface area over which volatilization occurs, m<sup>2</sup>

$f_d$  = fraction of contaminant dissolved in water column, assumed to be 1.0

$C_T$  = total concentration of contaminant in water column, mg/m<sup>3</sup>

In the above equation,

$$k_1 = K \cdot H \cdot \frac{1 \text{ day}}{86,400s}$$

where

$K$  = volatilization rate in day<sup>-1</sup>

=  $K_{11}$  above

$H$  = average river depth, in m

Also,  $C_T$  is calculated as  $S_1$ , using the point source model above. The river surface area  $A_s$  over which volatilization occurs was taken to be the area of an appropriate section of the river at each exposure location.

Table F-1

**SUMMARY OF SITE-SPECIFIC INPUTS  
USED IN THE RIVER DISPERSION AND  
VOLATILIZATION MODEL**

Symbol	Parameter	Units	Value	Source
Q	Average river flow	L/day	2.45E+11	Annual average flow for Niagara River is 100,000 cfs
B	Average river width	ft	400	At Whirlpool; Estimated from site map
H	Average river depth	ft	16.7	Report on Niagara River
A	Average river cross-sectional area	ft <sup>2</sup>	6,680	Calculated as B·H
U <sub>1</sub>	Average river velocity	ft/day	2.59E+06	Site-specific estimate of 30 ft/s near the Whirlpool
<b>Point Source Model</b>				
W <sub>1</sub>	Mass loading	mg/day	--	Calculated based on groundwater concentrations
x <sub>1</sub>	Distance downstream from point	ft	13,800	Estimated distance from Falls Treet tunnel outfall to Whirlpool
K <sub>11</sub>	Volatilization rate for point source	day <sup>-1</sup>	--	Calculated from Henry's Law constant and river re-aeration coefficient (see Thomann and Mueller 1987, p. 532); chemical-specific value

02[IL]JD1900:D3048/1258/14

Source: Ecology and Environment, Inc. 1990.

**Near Field Air Dispersion: "Box Model"**

For scenarios where the receptor is at the source or very close to the source, within approximately 100 meters downwind, the near field "box model" of GRI (1988) Section 1.2.2 was applied. This model is as follows:

$$C_a = Q / (H_b W_b \cdot U_m)$$

where

$C_a$  = On-site air concentration ( $\text{mg}/\text{m}^3$ )

$Q$  = Compound source strength or emission rate from earth or water surface ( $\text{mg}/\text{s}$ )

$H_b$  = Height of box at downwind edge (m) (depends on downwind distance  $x$  to receptor)

$W_b$  = Crosswind width of box = crosswind dimension of land or water area (m) emitting volatile organic vapors

$U_m$  = Average wind speed throughout box (m/s)

$$= 0.22 U_{10} \ln (2.5 H_b)$$

where

$U_{10}$  = Wind speed at 10 m elevation (m/s)

In applying the above model,  $Q$  was calculated as the mass flux from volatilization from the river surface, as described previously.

Table F-2 summarizes the site-specific input parameter values assumed in applying this model.

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Table F-2

SUMMARY OF SITE-SPECIFIC INPUTS  
USED IN THE "BOX" MODEL FOR  
NEAR FIELD AIR DISPERSION

Symbol	Parameter	Units	Value	Source
$A_c$	Area of contamination	$m^2$	59,830	Area of section of Niagara River just upstream of Falls Street tunnel outfall, contributing to worker exposures: 1,400' x 460'
$H_b$	Height of box	m	6.1	GRI (1988), Exhibit 1.2.2-2
x	Downwind distance to receptor from upwind edge of source	m	100	For nearby receptors (workers)
$W_b$	Crosswind width of box	m	140.2	One-half the width of the Niagara River near Prospect Point (460 ft.)
$U_{10}$	Wind speed at 10m height	m/s	5.5	Annual average for Buffalo, N.Y.

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Source: Ecology and Environment, Inc., 1990.

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