

Site: Industri Flex
Break: 3.6
Other: 34148

APPENDIX E

Calculations

CLIENT/PROJECT ISRT/GSIP PAGE 1 OF 6
 BY M.A. De Cillis DATE May 29, 1991 PROJECT NO. 06609D
 CHECKED BY J. Gerlach DATE June 5, 1991

DESCRIPTION Ground-Water Discharge and Velocity Calculations

Discharge to Aberjona River

- Use Darcy's Law, $Q = KIA$ (Fetter, Jr. 1980)

$K = 200 \text{ gpd/ft}^2 \text{ to } 280 \text{ gpd/ft}^2$

- from published data for sand with silt (Freeze and Cherry 1979) corroborated by pumping test K of 280 gpd/ft² (Temporary Well TW-3S)

$I = 0.022 \text{ ft/ft to } 0.026 \text{ ft/ft, ave} = 0.024 \text{ ft/ft}$

$$I = \frac{\Delta h}{A} = \frac{h_{PZ-1} - w_1, \text{ Aberjona}}{25 \text{ ft}}$$

$$= \frac{63.59 \text{ ft} - 63.04 \text{ ft}}{25 \text{ ft}} \quad 4/10/90$$

$\checkmark = 0.022 \text{ ft/ft}$

$$I = \frac{63.98 \text{ ft} - 63.32 \text{ ft}}{25 \text{ ft}} \quad 8/13/90$$

$\checkmark = 0.026 \text{ ft/ft}$

$$I_{\text{ave}} = \frac{0.022 \text{ ft/ft} + 0.026 \text{ ft/ft}}{2}$$

$$\checkmark = 0.024 \text{ ft/ft}$$

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 BY M.A. DeCillis DATE May 29, 1991
 CHECKED BY J. Gustach DATE June 5, 1991

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 PROJECT NO. 066091

DESCRIPTION Ground-Water Discharge and Velocity Calculations

$$A = 405 \text{ ft}^2$$

- distance from PZ-1/PZ-2 to Staff Gauge SW-17 = 270 ft
 - approximate depth of Aberjona in vicinity of PZ-1/PZ-2 to SW-17 \approx 1.5 ft
- $$\checkmark (1.5 \text{ ft})(270 \text{ ft}) = 405 \text{ ft}^2$$

$$Q = K I A$$

$$= \left(\frac{200 \text{ gpd}}{\text{ft}^2} \right) \left(0.022 \frac{\text{ft}}{\text{ft}} \right) (405 \text{ ft}^2)$$

$$\checkmark = 1,782 \text{ gpd}$$

$$Q = K I A$$

$$= \left(\frac{280 \text{ gpd}}{\text{ft}^2} \right) \left(0.026 \frac{\text{ft}}{\text{ft}} \right) (405 \text{ ft}^2)$$

$$\checkmark = 2,948 \text{ gpd}$$

$$Q_{\text{ave}} = \frac{1,782 \text{ gpd} + 2,948 \text{ gpd}}{2}$$

$$\checkmark = 2,365 \text{ gpd}$$

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 PROJECT NO. 06609D

DESCRIPTION Ground-Water Discharge and Velocity Calculations

Discharge per linear foot of Aberjona =

$$\frac{Q}{\text{length of reach (270 ft)}}$$

$$\frac{1,782 \text{ gpd}}{270 \text{ linear ft}} = 6.6 \text{ gpd/linear ft} \checkmark$$

$$\frac{2,948 \text{ gpd}}{270 \text{ linear ft}} = 10.9 \text{ gpd/linear ft} \checkmark$$

$$\frac{2,365 \text{ gpd}}{270 \text{ linear ft}} = 8.8 \text{ gpd/linear ft} \checkmark$$

Horizontal Ground-Water Velocities

- use velocity equation from Fetter, Jr. (1980),

i.e.,
$$v = \frac{K}{n_e} \frac{dh}{dl}$$

$$\frac{dh}{dl} = I_h$$

$K = 280 \text{ gpd/ft}^2 \text{ and } 530 \text{ gpd/ft}^2$

- from pumping test K for Temporary Well TW-3S (which is similar to Golder slug test value of 212 gpd/ft^2), and Temporary Well TW-1S and TW-2S, respectively

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 BY M.A. DeCillis DATE MAY 29, 1991
 CHECKED BY J. Geelach DATE JUNE 5, 1991

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 PROJECT NO. 06609D

DESCRIPTION Ground-Water Discharge and Velocity Calculations

$\eta_e = 0.25 \text{ to } 0.30$
 - from Fetter, Jr. (1980)

$I = 0.009 \text{ ft/ft}$
 - from a flow line extending from
 Observation Well DW-22 to
 Observation Well DW-17, i.e.,

$$\frac{dh}{dl} = \frac{73.78 \text{ ft} - 52.22 \text{ ft}}{2,400 \text{ ft}}$$

$$= 0.009 \text{ ft/ft} \checkmark$$

$$V = \frac{K}{\eta_e} I_h$$

$$V = \frac{(280 \text{ gpd/ft}^2)}{(0.25) (7.48 \frac{\text{gal}}{\text{ft}^3})} (0.009) = 1.35 \text{ ft/d} \checkmark$$

$$V = \frac{(280 \text{ gpd/ft}^2)}{(0.30) (7.48 \frac{\text{gal}}{\text{ft}^3})} (0.009) = 1.12 \text{ ft/d} \checkmark$$

$$V = \frac{(530 \text{ gpd/ft}^2)}{(0.25) (7.48 \frac{\text{gal}}{\text{ft}^3})} (0.009) = 2.55 \text{ ft/d} \checkmark$$

$$V = \frac{(530 \text{ gpd/ft}^2)}{(0.30) (7.48 \frac{\text{gal}}{\text{ft}^3})} (0.009) = 2.13 \text{ ft/d} \checkmark$$

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 PROJECT NO. 06609D

DESCRIPTION Ground-Water Discharge and Velocity Calculations

Horizontal Ground-Water Travel Time

$$d = r t \quad ; \quad t = \frac{d}{r}$$

use highest and lowest r (rate) / velocity to calculate lowest and highest travel time

$$t_{\max} = \frac{2,400 \text{ ft}}{1.12 \text{ ft/d}}$$

$$= 2,143 \text{ days or } 5.9 \text{ years } \checkmark$$

$$t_{\min} = \frac{2,400 \text{ ft}}{2.55 \text{ ft/d}}$$

$$= 941 \text{ days or } 2.6 \text{ years } \checkmark$$

Vertical Ground - Water Velocities

- use velocity equation from Fetter, Jr. (1980),

$$\text{i.e., } v = \frac{K}{n_e} \frac{dh}{dl}$$

$$\frac{dh}{dl} = \frac{T}{v}$$

$$K_z = 2 \text{ gpd/ft}^2 \text{ to } 57 \text{ gpd/ft}^2$$

$K = K_z$ (vertical K)

- From pumping test K_z for Temporary Piezometers TW-4S and TW-1D, TW-2D,

CLIENT/PROJECT ISRT/GSIP PAGE 6 OF 6
 BY M. A. DeCillis DATE May 29, 1991 PROJECT NO. 06609D
 CHECKED BY J. Gselach DATE June 5, 1991

DESCRIPTION Ground-Water Discharge and Velocity Calculations

and TW-3D, respectively.

$\eta_e = 0.28$
 - average estimate from Fetter, Jr. (1980)

$I_v = 0.002 \text{ ft/ft to } 0.007 \text{ ft/ft}$

$$I_v = \frac{\Delta h}{\Delta l} = \frac{h_1 - h_2}{\text{distance between well screen midpoints}}$$

$$= \frac{52.19 \text{ ft} - 52.11 \text{ ft}}{39.70 \text{ ft}} \quad \text{OW-33A/OW-33B}$$

$$= 0.002 \text{ ft/ft} \checkmark$$

$$I = \frac{52.58 \text{ ft} - 52.46 \text{ ft}}{17.05 \text{ ft}}$$

$$= 0.007 \text{ ft/ft} \checkmark$$

$$v = \frac{K}{\eta_e} I_v$$

$$= \frac{(2 \text{ gpd/ft}^2)(0.002 \text{ ft/ft})}{(0.28)(7.48 \text{ gal/ft}^3)} = \sqrt{0.002 \text{ ft/d}}$$

$$= \frac{(2 \text{ gpd/ft}^2)(0.007 \text{ ft/ft})}{(0.28)(7.48 \text{ gal/ft}^3)} = \sqrt{0.007 \text{ ft/d}}$$

$$= \frac{(57 \text{ gpd/ft}^2)(0.002 \text{ ft/ft})}{(0.28)(7.48 \text{ gal/ft}^3)} = \sqrt{0.054 \text{ ft/d}}$$

$$= \frac{(57 \text{ gpd/ft}^2)(0.007 \text{ ft/ft})}{(0.28)(7.48 \text{ gal/ft}^3)} = \sqrt{0.191 \text{ ft/d}}$$

APPENDIX F

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APPENDIX G

Risk Assessment Documentation

- Section 1. Summary Tables**
- Section 2. Toxicity Profiles**
- Section 3. Photographs**

APPENDIX G
Risk Assessment Documentation
Section 1. Summary Tables

TABLE G-1A

RAW DATABASE USED FOR CALCULATING DESCRIPTIVE STATISTICS
FOR METAL CONCENTRATIONS IN SEDIMENT

Industri-Plex Superfund Site
Woburn, MA

	SW1/052	SW1/055	SW1/056	SW1/057	SW1/059	SW-5	SW-12	SW-14	SW-17	SW1/018	SW1/019	SW1/020	SW1/021
Aluminum				3520		4190	3340	3480	4890				
Antimony				11.7									
Arsenic	3.5	17.7	11	5	3.9	12.5	9.4	20.6	58.6	16.3	171.6	90.2	45.9
Barium				4		12.9	15.1	11.3	438				
Beryllium				0.13				0.26					
Cadmium				1.2		2.5	2		3.5				
Calcium				595		1160	947	835	1990				
Chromium	6.2	48.6	8.4	6.7	7.7	22.2	18.6	13.5	100	67.2	188.9	99.1	151.5
Cobalt				1.3		2.8	1.5	3.7	4.2				
Copper				0.59		12.2	27.3	16.6	71.8				
Iron				2470		7100	6900	7080	25800				
Lead	4.9	81.2	19.9	9.5	18.4	28.7	41	7.3	116	41.8	138.9	73.9	71.6
Magnesium				822		1980	1640	1610	2520				
Manganese				25.6		91.5	92.7	158	189				
Mercury				0.1				0.3					
Nickel				6.2									
Potassium				527		407	584	575	441				
Selenium				0.51									
Silver				0.82									
Sodium				468				65.1	8.7				
Thallium				0.51									
Tin						14.4	9.6	8.48	5.7				
Vanadium				3.6		11	10.1	9	16.5				
Zinc				8.3		88.5	47.7	78.1	150				

NOTE: All concentrations given are in mg/kg dry weight.

TABLE G-1A

RAW DATABASE USED FOR CALCULATING DESCRIPTIVE STATISTICS
FOR METAL CONCENTRATIONS IN SEDIMENT

Industri-Plex Superfund Site
Woburn, MA

	SW-3	SW-4	SW-8	SW1/040	SW1/042	SW1/043	SW1/044	SW1/045	SW1/048	SW1/047	SW1/049	SW1/050	SW1/051
Aluminum	19900	10200	3430		6440								
Antimony			3.1		7.5								
Arsenic	29.4	4.2	2.65	3.8	1.1	4.5	6.9	4.5	2.2		69.6	6.5	30.4
Barium	26.5	49.5	7.55		7.2								
Beryllium		0.46			0.26								
Cadmium		2.8			0.81								
Calcium	1400	2220	667.5		930								
Chromium	30.4	27.3	6.95	14.8	5.9	6.3	61.7	6.9	13	7.2	51.8	12.4	6.9
Cobalt	4.4	9.9	1.45		1.4								
Copper	44.3	33.1	11.7		4.3								
Iron	31400	16800	4645		1900								
Lead	26.7	28.7	5.95		3.6	16		8.7	10.2	10.1	487	39.5	
Magnesium	3330	5710	1315		286								
Manganese	108	162	49.25		22.4								
Mercury	0.8				0.2								
Nickel	27.7	20			4.5								
Potassium	1120	2160	369		332								
Selenium	2.5				1								
Silver					1.3								
Sodium	479	62.2	133.5		597								
Thallium					0.64								
Tin	1010	12.5	17.75										
Vanadium	30.4	32.5	7.75		5.2								
Zinc	71.3	50.9	16.15		11.8								

NOTE: All concentrations given are in mg/kg dry weight.

TABLE G-1A

RAW DATABASE USED FOR CALCULATING DESCRIPTIVE STATISTICS
FOR METAL CONCENTRATIONS IN SEDIMENT

Industri-Plex Superfund Site
Woburn, MA

	SW-1	SW-2	SW1/024	SW1/025	SW1/026	SW1/027	SW1/028	SW1/029	SW1/030	SW1/031	SW1/032	SW1/033	SW1/034
Aluminum	6530	3620											
Antimony	7.3												
Arsenic	4.8	371	47.7	132.2	13.2	422	301	1150	404	366	145	70	11.2
Barium	25.4	27.4											
Beryllium		0.27											
Cadmium		1.2											
Calcium	1550	676											
Chromium	28.6	646	41.9	284.7	37.8	27.7	785	105	1260	158	37	264	10.8
Cobalt	6.2	3.7											
Copper	38.4	121											
Iron	9140	21600											
Lead	11.6	212	215.5	167.2	20.9	250	817	2680	1210	962	1880	198	128
Magnesium	2800	1040											
Manganese	174	48.4											
Mercury		0.1											
Nickel													
Potassium	1040	287											
Selenium		0.52											
Silver		1.8											
Sodium	119	53.1											
Thallium													
Tin	16.3	8.6											
Vanadium	15.5	11.7											
Zinc	56.3	189											

NOTE: All concentrations given are in mg/kg dry weight.

TABLE G-1A

RAW DATABASE USED FOR CALCULATING DESCRIPTIVE STATISTICS
FOR METAL CONCENTRATIONS IN SEDIMENT

Industri-Plex Superfund Site
Woburn, MA

	SW1/022	SW1/023	SW-6	SW-7	SW-10	SW-15	SW1/001	SW1/002	SW1/003	SW1/004	SW1/005	SW1/006	SW1/007
Aluminum			922	5400	4390	5160							
Antimony				375	16.4	19.8							
Arsenic	33.2	103.4	154	1580	170	511	16.6	3.3	122.9	245.3	259.0	232	211
Barium			48.5	462	25.9	111							
Beryllium				0.89		0.76							
Cadmium			3.4		2.2	2.3							
Calcium			2350	1160	1480	3120							
Chromium	132.3	140.0	95.4	331	32.1	118	234	10700	567.3	1,856.7	143.8	192	600
Cobalt			2.8	11.9	5.9	13.1							
Copper			18.4	216	79.2	171							
Iron			22400	44100	34200	37400							
Lead	119.0	157.0	27.3	4210	315	346	16.3	30.1	165.7	1,210.0	246.7	429	291
Magnesium			626	513	2330	950							
Manganese			394	140	183	807							
Mercury			0.4	9.5		0.3							
Nickel				9		11.2							
Potassium			329	419	713	429							
Selenium				10.7		0.86							
Silver			0.76	13.6		3.7							
Sodium			356	240	84.4	312							
Thallium				38.4									
Tin				221	16.8	157							
Vanadium			4.9	15.8	15.2	12.9							
Zinc			46.7	384	223	437							

NOTE: All concentrations given are in mg/kg dry weight.

TABLE G-1A

RAW DATABASE USED FOR CALCULATING DESCRIPTIVE STATISTICS
FOR METAL CONCENTRATIONS IN SEDIMENT

Industri-Plex Superfund Site
Woburn, MA

	SW1/013	SW1/014	SW1/015	SW1/016	SW1/017
Aluminum					
Antimony					
Arsenic	602.7	73.1	37.2	3,453.3	412.3
Barium					
Beryllium					
Cadmium					
Calcium					
Chromium	159.3	93.8	425.0	724.7	3,123.3
Cobalt					
Copper					
Iron					
Lead	313.3	157.5	164.4	4,700.0	993.7
Magnesium					
Manganese					
Mercury					
Nickel					
Potassium					
Selenium					
Silver					
Sodium					
Thallium					
Tin					
Vanadium					
Zinc					

NOTE: All concentrations given are in mg/kg dry weight.

TABLE G-1A

RAW DATABASE USED FOR CALCULATING DESCRIPTIVE STATISTICS
FOR METAL CONCENTRATIONS IN SEDIMENT

Industri-Plex Superfund Site
Woburn, MA

	SW1/035	SW1/036	SW1/037	SW1/038	SW1/039	SW1/041	SW1/048	SW-9	SW-11	SW-13	SW-16	CORE	SW1/012
Aluminum								15800	14700	6380	10300	3400	
Antimony								41.5					
Arsenic	23.8	7.7	5.1	211	57.2	12.9	1.6	9830	1750	1330	928	9.9	8.4
Barium								171	122	64	175	7.8	
Beryllium								3.7	1.8	1.2	1.1		
Cadmium								51.5	27.4	14.1	23.4		
Calcium								14900	8220	4330	10300	655	
Chromium	25.9	7	29.2	3020	338	22.4	29.6	1090	529	382	140	13.9	109.8
Cobalt								27.8	41.9	14.4	35.9	2.8	
Copper								1160	523	287	357	18.8	
Iron								112000	94100	55100	87500	8640	
Lead	49.9	16.1	11.5	221	155	37.3		611	320	275	354	5.7	29.5
Magnesium								4880	3270	1190	4610	1620	
Manganese								1150	783	488	575	84.3	
Mercury								1.9	1.7	1	0.4		
Nickel								48	44.8	26.1	26.9		
Potassium								2040	1290	1040	801	497	
Selenium								10.9	7.8	6	41.4		
Silver								6.4		2.7			
Sodium								12700	1610	817	69.6	66.2	
Thallium								6.3					
Tin								1230	30	50.4	24.9	9.3	
Vanadium								79.1	47	30.4	159	8.9	
Zinc								7940	5980	2580	1800	62.6	

NOTE: All concentrations given are in mg/kg dry weight.

TABLE G-181

DESCRIPTIVE STATISTICS AND FREQUENCY DISTRIBUTIONS
FOR TOTAL METALS IN SURFACE WATER

Groundwater/Surface Water Investigation Plan
Industri-Plex Superfund Site
Woburn, MA

	Lead	Magnesium	Manganese	Potassium	Sodium	Zinc
Class Width	0.4	784.2	122.8	728.7	9,730.8	17.9
Minimum	2.2	3770	82.7	1830	10600	15.4
Maximum	8.6	11500	1840	11800	148000	276
Mean (geometric)	3.5	5838.3	422.7	4188.8	38738.3	45.1
Mean (arithmetic)	3.8	5842.0	612.7	4544.7	48930.0	68.1
IDL	2	37	1	900	38	8
CRDL	5	5000	15	5000	5000	20
	CLASS WIDTH FREQUENCY					
	2.0 1	37 0	1 0	900 0	38 0	8 0
	2.4 3	801 0	124 2	1827 0	9789 0	28 4
	2.8 0	1585 0	248 3	2354 3	19500 2	44 8
	3.2 3	2329 0	389 1	3081 1	29231 2	62 1
	3.6 1	3083 1	491 2	3808 2	38982 5	80 0
	4.0 0	3857 1	614 1	4535 5	48893 1	98 0
	4.4 3	4821 0	737 0	5282 0	58424 1	116 0
	4.8 0	5385 5	859 2	5989 0	68155 1	134 1
	5.2 1	6149 0	982 1	6716 2	77886 0	152 1
	5.6 0	6913 5	1,104 0	7443 0	87617 1	170 1
	6.0 1	7877 0	1,227 1	8170 0	97348 0	188 0
	6.4 1	8441 1	1,350 1	8897 0	107079 0	206 0
	6.8 0	9205 1	1,472 0	9824 1	118810 0	224 0
	7.2 0	9989 0	1,595 0	10351 0	128641 0	242 0
	7.6 0	10733 0	1,717 0	11078 0	138272 1	260 0
	8.0 0	11497 0	1,840 1	11805 1	148003 1	278 1
		1	1	0	0	0

NOTE: Class widths, with the exception of arsenic, chromium, and lead, are calculated as [Maximum value - Instrument Limit of Detection]/15. For arsenic, chromium, and lead, the first frequency distribution ranges from zero to the Consent Decree Action Level. The second frequency distribution ranges from the Consent Decree Action Level to the maximum concentration detected. For each frequency distribution, only the upper class limit is given. Frequencies cited will fall within the limit value next to it and the next lowest value. For example, if the class width is 50, and the frequency given is 11, next to a class limit of 100, then 11 values in the database fall below 100 but above 50. Also note that the minimum and maximum cited in this table are for detected values only, and that the means are calculated using zero for non-detects, not one-half of the detection limit.

TABLE G-1B1

DESCRIPTIVE STATISTICS AND FREQUENCY DISTRIBUTIONS
FOR TOTAL METALS IN SURFACE WATER

Groundwater/Surface Water Investigation Plan
Industri-Plex Superfund Site
Woburn, MA

	Aluminum	Arsenic	Barium	Calcium	Chromium	Copper	Iron
Class Width	41.3	2.6	4.4	3,665.3	4.0	0.5	325.1
Minimum	34	2	18.7	26400	3.1	7.3	446
Maximum	648	40.6	67.7	56000	62.6	13.5	4880
Mean (geometric)	127.4	6.9	32.2	30941.6	3.9	2.9	1716.6
Mean (arithmetic)	156.7	11.6	32.2	30043.3	9.8	4.6	1939.3
IDL	27	2	2	20	3	6	3
CRDL	200	10	200	6000	10	25	100
	CLASS WIDTH FREQUENCY						
	27.0 0	2.0 4	2.0 0	20 0	3 6	6.0 8	3 0
	68.3 3	4.6 0	6.4 0	3685 0	7 5	6.5 0	328 0
	109.6 2	7.2 1	10.8 0	7350 0	11 0	7.0 0	653 1
	150.9 4	9.8 4	16.2 0	11015 0	15 1	7.5 1	878 1
	192.2 2	12.4 0	19.6 2	14680 0	19 0	8.0 1	1303 3
	233.5 1	15.0 1	24.0 1	18345 1	23 1	8.5 0	1628 0
	274.8 1	17.6 2	28.4 0	22010 0	27 0	9.0 0	1953 5
	316.1 0	20.2 1	32.8 6	25875 0	31 1	9.5 0	2278 0
	357.4 1	22.8 0	37.2 3	29340 6	35 0	10.0 2	2803 1
	398.7 0	25.4 0	41.6 0	33005 3	39 0	10.5 1	2928 1
	440.0 0	28.0 0	46.0 1	36670 2	43 0	11.0 0	3253 1
	481.3 0	30.6 1	50.4 0	40335 1	47 0	11.5 1	3578 1
	522.6 0	33.2 0	54.8 0	44000 1	51 0	12.0 0	3903 0
	563.9 0	35.8 0	59.2 1	47665 0	55 0	12.5 0	4228 0
	605.2 0	38.4 0	63.6 0	51330 0	59 0	13.0 0	4553 0
	646.5 1	41.0 1	68.0 1	54995 0	63 1	13.5 1	4878 0
	0 0	0 0	0 0	0 1	0 0	0 0	0 1

NOTE: Class widths, with the exception of arsenic, chromium, and lead, are calculated as (Maximum value - Instrument Limit of Detection)/15. For arsenic, chromium, and lead, the first frequency distribution ranges from zero to the Consent Decree Action Level. The second frequency distribution ranges from the Consent Decree Action Level to the maximum concentration detected. For each frequency distribution, only the upper class limit is given. Frequencies cited will fall within the limit value next to it and the next lowest value. For example, if the class width is 60, and the frequency given is 11, next to a class limit of 100, then 11 values in the database fall below 100 but above 60. Also note that the minimum and maximum cited in this table are for detected values only, and that the means are calculated using zero for non-detects, not one-half of the detection limit.

TABLE G-1B2

DESCRIPTIVE STATISTICS AND FREQUENCY DISTRIBUTIONS
FOR TOTAL METALS IN SEDIMENT

Groundwater/Surface Water Investigation Plan
Industrial-plex Superfund Site
Woburn, MA

	Cadmium		Calcium		Chromium (*)		Chromium (*)		Cobalt		Copper	
Class Width	3.2		992.4		40.0		873.3		2.8		77.1	
Minimum	0.8		596.0		5.9				1.3		0.6	
Maximum	51.5		14900		10700				41.9		1180	
Mean (geometric)	2.9		1,874.8		83.8				5.7		50.9	
Mean (arithmetic)	7.3		3096.3		3010.1				10.2		168.0	
IDL	3		14		3				3		3	
CRDL	5		5000		10				50		25	
	CLASS LIMITS	FREQUENCY										
	3.0	13	14	0	0	0	600	0	3.0	6	3	1
	6.2	2	1008	8	40	28	1273	4	5.6	4	80	11
	9.4	0	1999	8	80	6	1947	1	8.2	2	167	1
	12.6	0	2991	2	120	7	2620	0	10.8	1	234	2
	15.8	1	3984	1	160	7	3293	2	13.4	2	311	1
	19.0	0	4976	1	200	2	3967	0	16.0	1	389	1
	22.2	0	5968	0	240	1	4640	0	18.6	0	466	0
	25.4	1	6961	0	280	1	5313	0	21.2	0	543	1
	28.6	1	7953	0	320	1	6088	0	23.8	0	620	0
	31.8	0	8946	1	360	2	6860	0	26.4	0	697	0
	35.0	0	9938	0	400	1	7333	0	29.0	1	774	0
	38.2	0	10930	1	440	1	8006	0	31.6	0	851	0
	41.4	0	11923	0	480	0	8880	0	34.2	0	928	0
	44.6	0	12915	0	520	0	9363	0	36.8	1	1005	0
	47.8	0	13908	0	560	2	10028	0	39.4	0	1082	0
		1		1	600	2	10700	0		1		1

NOTE: Class widths, with the exception of arsenic, chromium, and lead, are calculated as (Maximum value - Instrument Limit of Detection)/15. For arsenic, chromium, and lead, the first frequency distribution ranges from zero to the Consent Decree Action Level. The second frequency distribution ranges from the Consent Decree Action Level to the maximum concentration detected. For each frequency distribution, only the upper class limit is given. Frequencies cited will fall within the limit value next to it and the next lowest value. For example, if the class width is 50, and the frequency given is 11, next to a class limit of 100, then 11 values in the database fall below 100 but above 50. Also note that the minimum and maximum cited in this table are for detected values only, and that the means are calculated using zero for non-detects, not one-half of the detection limit. Maximum values were considered outliers in the calculation of class limits for aluminum, arsenic, iron, manganese, nickel, silver, sodium, and zinc.

TABLE G-1B2

DESCRIPTIVE STATISTICS AND FREQUENCY DISTRIBUTIONS
FOR TOTAL METALS IN SEDIMENT

Groundwater/Surface Water Investigation Plan
Industri-Plex Superfund Site
Woburn, MA

	Nickel	Potassium	Selenium	Silver	Sodium	Thallium
Class Width -	1.0	118.6	2.0	0.5	845.6	2.0
Minimum -	4.6	267.0	0.6	0.6	8.7	0.6
Maximum -	48	2160	41.4	13.6	12700	38.4
Mean (geometric) -	4.6	647.6	1.9	1.5	127.7	1.3
Mean (arithmetic) -	11.8	764.4	4.3	1.8	966.6	2.4
IDL -	24	428	2	2	18	4
CRDL -	40	6000	6	10	6000	10
	CLASS LIMITS FREQUENCY					
	24.0 14	428 6	2.0 19	2.0 16	18 3	4.0 17
	25.6 0	544 3	4.6 1	2.8 1	964 14	6.3 1
	27.2 2	659 2	7.2 1	3.6 0	1700 1	8.8 0
	28.8 1	775 1	9.8 1	4.4 1	2566 0	10.9 0
	30.4 0	890 1	12.4 2	5.2 0	3400 0	13.2 0
	32.0 0	1006 0	15.0 0	6.0 0	4246 0	15.6 0
	33.6 0	1121 3	17.6 0	6.8 1	5091 0	17.8 0
	35.2 0	1237 0	20.2 0	7.6 0	5937 0	20.1 0
	36.8 0	1352 1	22.8 0	8.4 0	6782 0	22.4 0
	38.4 0	1468 0	25.4 0	9.2 0	7628 0	24.7 0
	40.0 0	1583 0	28.0 0	10.0 0	8473 0	27.0 0
	41.6 0	1699 0	30.6 0	10.8 0	9319 0	29.3 0
	43.2 0	1814 0	33.2 0	11.6 0	10164 0	31.6 0
	44.8 1	1930 0	35.8 0	12.4 0	11010 0	33.9 0
	46.4 0	2045 1	38.4 0	13.2 0	11855 0	36.2 0
	1 1	1 1	1 1	1 1	1 1	1 1

NOTE: Class widths, with the exception of arsenic, chromium, and lead, are calculated as (Maximum value - Instrument Limit of Detection)/15. For arsenic, chromium, and lead, the first frequency distribution ranges from zero to the Consent Decree Action Level. The second frequency distribution ranges from the Consent Decree Action Level to the maximum concentration detected. For each frequency distribution, only the upper class limit is given. Frequencies cited will fall within the limit value next to it and the next lowest value. For example, if the class width is 50, and the frequency given is 11, next to a class limit of 100, then 11 values in the database fall below 100 but above 50. Also note that the minimum and maximum cited in this table are for detected values only, and that the means are calculated using zero for non-detects, not one-half of the detection limit. Maximum values were considered outliers in the calculation of class limits for aluminum, arsenic, iron, manganese, nickel, silver, sodium, and zinc.

TABLE G-1B2

DESCRIPTIVE STATISTICS AND FREQUENCY DISTRIBUTIONS
FOR TOTAL METALS IN SEDIMENT

Groundwater/Surface Water Investigation Plan
Industrial-Phase Superfund Site
Woburn, MA

	Iron	Lead (*)	Lead (*)	Magnesium	Manganese	Mercury						
Class Width	7486.6	66.7	248.7	379.1	78.6	0.6						
Minimum	1,900.0	3.8		286.0	22.4	0.1						
Maximum	112000	4700		6710	1150	9.5						
Mean (geometric)	18,587.9	79.8		1,046.8	169.9	0.7						
Mean (arithmetic)	32717.6	1533.0		2169.8	295.8	0.9						
IDL	3	2		23	1	0.2						
CRDL	100	5		6000	15	0.2						
	CLASS LIMITS	FREQUENCY										
	3	0	0	4	1000	0	23	0	1	0	0.2	10
	7470	6	67	27	1247	2	402	1	78	4	0.8	5
	14937	1	133	8	1493	0	781	2	154	4	1.4	1
	22404	3	200	8	1740	0	1160	3	231	5	2.0	2
	29671	1	267	5	1967	1	1539	2	307	0	2.6	0
	37338	2	334	5	2234	0	1919	2	384	0	3.2	0
	44805	2	400	2	2480	0	2298	1	461	1	3.8	0
	52272	0	467	1	2727	1	2677	3	537	1	4.4	0
	59739	1	534	1	2974	0	3056	0	614	1	5.0	0
	67206	0	600	0	3220	0	3435	2	690	0	5.6	0
	74673	0	667	1	3467	0	3814	0	767	1	6.2	0
	82140	0	734	0	3714	0	4193	0	844	1	6.8	0
	89607	1	800	0	3960	0	4572	0	920	0	7.4	0
	97074	1	867	1	4207	0	4951	2	997	0	8.0	0
	104541	0	934	0	4454	1	5330	0	1073	0	8.6	0
		1	1,000	2	4701	1		1		1		1
						0						

NOTE: Class widths, with the exception of arsenic, chromium, and lead, are calculated as (Maximum value - Instrument Limit of Detection)/16. For arsenic, chromium, and lead, the first frequency distribution ranges from zero to the Consent Decree Action Level. The second frequency distribution ranges from the Consent Decree Action Level to the maximum concentration detected. For each frequency distribution, only the upper class limit is given. Frequencies cited will fall within the limit value next to it and the next lowest value. For example, if the class width is 50, and the frequency given is 11, next to a class limit of 100, then 11 values in the database fall below 100 but above 50. Also note that the minimum and maximum cited in this table are for detected values only, and that the means are calculated using zero for non-detects, not one-half of the detection limit. Maximum values were considered outliers in the calculation of class limits for aluminum, arsenic, iron, manganese, nickel, silver, sodium, and zinc.

TABLE G-183

DESCRIPTIVE STATISTICS AND FREQUENCY DISTRIBUTIONS
FOR TOTAL METALS IN GROUNDWATER

Groundwater/Surface Water Investigation Plan
Industrial-Plex Superfund Site
Woburn, MA

	Aluminum	Antimony	Arsenic	Barium	Beryllium	Cadmium						
Class Width	2720	8	50	30	0.62	2						
Minimum	44.9	10.4	2	4.8	0.4	1.5						
Maximum	224000	143	2350	505	8.8	27.4						
Mean (geometric)	752.7	4.0	13.8	20.8	1.0	1.4						
Mean (arithmetic)	6,866.3	39.4	194.8	62.5	2.2	12.7						
IDL	27	37	2	2	1	5						
CRDL	200	80	10	200	5	5						
	CLASS LIMITS	FREQUENCY										
	27	0	5	0	0	0	0	0	0	0		
	2747	43	13	3	50	28	30	21	0.5	1		
	5487	10	21	4	100	5	80	13	1	3		
	8187	2	29	6	150	1	90	4	1.5	1		
	10907	1	37	1	200	2	120	3	2	1		
	13627	2	45	4	250	3	150	1	2.5	0		
	16347	1	53	2	300	0	180	2	3	0		
	19067	1	61	4	350	0	210	1	3.5	1		
	21787	0	69	0	400	0	240	3	4	0		
	24507	0	77	0	450	1	270	0	4.5	0		
	27227	0	85	0	500	1	300	2	5	0		
	29947	1	93	0	550	1	330	1	5.5	0		
	32667	0	101	0	600	0	360	0	6	0		
	35387	0	109	1	650	2	390	0	6.5	0		
	38107	0	117	0	700	0	420	0	7	0		
		2		1		2		1		1		
N =		63		26		46		62		8		11

NOTE: Class widths were generally calculated as [Maximum value - Instrument Limit of Detection]/15. For each frequency distribution, only the upper class limit is given. Frequencies cited will fall within the limit value next to it and the next lowest value. For example, if the class width is 50, and the frequency given is 11, next to a class limit of 100, then 11 values in the database fall below 100 but above 50. Also note that the minimum and maximum cited in this table are for detected values only, and that the means are calculated using zero for non-detects (not one-half of the detection limit). Maximum values were considered outliers when calculating the class width for aluminum, arsenic, antimony, copper, iron, lead, magnesium, mercury, sodium, and zinc.

TABLE G-1B2

DESCRIPTIVE STATISTICS AND FREQUENCY DISTRIBUTIONS
FOR TOTAL METALS IN SEDIMENT

Groundwater/Surface Water Investigation Plan
Industri-Plex Superfund Site
Woburn, MA

	Tin	Vanadium	Zinc			
Class Width	60.0	10.4	528.9			
Minimum	5.7	3.6	6.3			
Maximum	1230	159	7940			
Mean (geometric)	20.1	16.4	162.4			
Mean (arithmetic)	149.1	27.2	1050.4			
IDL	17	3	6			
CRDL	--	60	20			
	CLASS LIMITS	FREQUENCY	CLASS LIMITS	FREQUENCY	CLASS LIMITS	FREQUENCY
	17	11	3	0	6.0	0
	97	4	13	9	635.0	16
	177	1	24	4	1064.0	0
	257	1	34	3	1593.0	0
	337	0	45	0	2122.0	1
	417	0	55	1	2651.0	1
	497	0	65	0	3180.0	0
	577	0	76	0	3709.0	0
	657	0	86	1	4238.0	0
	737	0	97	0	4767.0	0
	817	0	107	0	5296.0	0
	897	0	117	0	5825.0	0
	977	0	128	0	6354.0	1
	1057	1	138	0	6883.0	0
	1137	0	149	0	7412.0	0
		1		1		1

NOTE: Class widths, with the exception of arsenic, chromium, and lead, are calculated as (Maximum value - Instrument Limit of Detection)/15. For arsenic, chromium, and lead, the first frequency distribution ranges from zero to the Consent Decree Action Level. The second frequency distribution ranges from the Consent Decree Action Level to the maximum concentration detected. For each frequency distribution, only the upper class limit is given. Frequencies cited will fall within the limit value next to it and the next lowest value. For example, if the class width is 50, and the frequency given is 11, next to a class limit of 100, then 11 values in the database fall below 100 but above 50. Also note that the minimum and maximum cited in this table are for detected values only, and that the means are calculated using zero for non-detects, not one-half of the detection limit. Maximum values were considered outliers in the calculation of class limits for aluminum, arsenic, iron, manganese, nickel, silver, sodium, and zinc.

TABLE G-1B3

DESCRIPTIVE STATISTICS AND FREQUENCY DISTRIBUTIONS
FOR TOTAL METALS IN GROUNDWATER

Groundwater/Surface Water Investigation Plan
Industri-Plex Superfund Site
Woburn, MA

	Magnesium	Manganese	Mercury	Nickel	Potassium	Selenium						
Class Width	10000	530	0.1	20	4050	5						
Minimum	1110	19.3	0.185	10.7	823	1.1						
Maximum	224000	28700	1.8	322	74800	78						
Mean (geometric)	11,347.4	557.8	0.04	4.6	2,883.4	1.4						
Mean (arithmetic)	28,830.6	1,596.6	0.8	57.8	10,365.0	19.3						
IDL	37	1	0.2	12	900	2						
CFDL	5000	15	0.2	40	5000	5						
	CLASS LIMITS	FREQUENCY										
	37	0	1	0	0	0	12	1	900	0	2	2
	10037	38	531	28	0.1	0	32	13	5850	34	7	4
	20037	8	1081	11	0.2	1	62	7	10800	11	12	0
	30037	4	1591	12	0.3	1	72	3	15750	8	17	1
	40037	1	2121	5	0.4	1	92	1	20700	2	22	0
	50037	4	2651	2	0.5	0	112	0	25650	0	27	0
	60037	0	3181	0	0.6	1	132	0	30600	0	32	1
	70037	2	3711	1	0.7	0	152	0	35550	1	37	0
	80037	2	4241	1	0.8	0	172	0	40500	2	42	0
	90037	0	4771	1	0.9	0	192	0	45450	1	47	0
	100037	0	5301	1	1	0	212	1	50400	0	52	1
	110037	1	5831	0	1.1	0	232	0	55350	0	57	0
	120037	0	6361	0	1.2	0	252	0	60300	0	62	0
	130037	1	6891	0	1.3	0	272	1	65250	0	67	0
	140037	0	7421	0	1.4	0	292	0	70200	0	72	0
		4		3		1		1		2		1
N =		65		65		6		28		59		10

NOTE: Class widths were generally calculated as (Maximum value - Instrument Limit of Detection)/15. For each frequency distribution, only the upper class limit is given. Frequencies cited will fall within the limit value next to it and the next lowest value. For example, if the class width is 50, and the frequency given is 11, next to a class limit of 100, then 11 values in the database fall below 100 but above 50. Also note that the minimum and maximum cited in this table are for detected values only, and that the means are calculated using zero for non-detects (not one-half of the detection limit). Maximum values were considered outliers when calculating the class width for aluminum, arsenic, antimony, copper, iron, lead, magnesium, mercury, sodium, and zinc.

TABLE G-1B3

DESCRIPTIVE STATISTICS AND FREQUENCY DISTRIBUTIONS
FOR TOTAL METALS IN GROUNDWATER

Groundwater/Surface Water Investigation Plan
Industri-Plex Superfund Site
Woburn, MA

	Aluminum	Chromium	Cobalt	Copper	Iron	Lead
Class Width	34465.333333	28	10	30	4420	12
Minimum	3810	1.9	7.4	7.1	322	2
Maximum	517000	428	157	1350	226000	298
Mean (geometric)	56,172.2	10.2	3.4	6.3	5,632.8	3.6
Mean (arithmetic)	99,031.7	64.7	26.6	99.2	13,620.9	25.0
IDL	20	3	7	6	3	2
CRDL	5000	10	50	25	100	5
	CLASS LIMITS FREQUENCY					
	20 0	3 3	7 0	0 0	3 0	0 0
	34520 20	31 29	17 10	30 23	4423 26	12 24
	69020 19	59 7	27 7	60 2	8843 14	24 7
	103520 8	87 2	37 2	90 2	13263 8	36 1
	138020 3	115 1	47 2	120 0	17683 7	48 2
	172520 2	143 0	57 4	150 1	22103 2	60 0
	207020 2	171 3	67 0	180 1	26523 3	72 0
	241520 4	199 1	77 0	210 1	30943 1	84 0
	276020 1	227 0	87 0	240 0	35363 1	96 0
	310520 2	255 0	97 0	270 0	39783 0	108 1
	345020 1	283 0	107 0	300 0	44203 0	120 0
	379520 1	311 1	117 0	330 0	48623 1	132 0
	414020 0	339 1	127 0	360 0	53043 1	144 0
	448520 0	367 0	137 0	390 0	57463 0	156 0
	483020 0	395 2	147 0	420 0	61883 0	168 1
	2	1	1	3	2	1
N =	65	51	26	33	65	37

NOTE: Class widths were generally calculated as (Maximum value - Instrument Limit of Detection)/15. For each frequency distribution, only the upper class limit is given. Frequencies cited will fall within the limit value next to it and the next lowest value. For example, if the class width is 50, and the frequency given is 11, next to a class limit of 100, then 11 values in the database fall below 100 but above 50. Also note that the minimum and maximum cited in this table are for detected values only, and that the means are calculated using zero for non-detects (not one-half of the detection limit). Maximum values were considered outliers when calculating the class width for aluminum, arsenic, antimony, copper, iron, lead, magnesium, mercury, sodium, and zinc.

Table G-1C

HABITAT ASSESSMENT SUMMARY

Groundwater/Surface Water Investigation Plan
 Industri-Plex Superfund Site
 Woburn, MA

SAMPLE #	BS-1A	BS-2	BS-3	BS-4	BS-5	BS-6	BS-7	BS-8	BS-9
DEPTH	2 ft.	<0.2 ft	<0.5 ft.	0.5 ft.	0.5-3 ft.	1 ft.	1 ft.	9 ft.	>15 ft.
1. Bottom Substrate		1	13	19	4	3	3		
2. Embeddedness		3	3	18	4	3	3		
3. Low Flow (<=0.15 cms)		1	1	3	7	3	1		
High Flow (>0.15 cms)		8	2	12	3	1	1		
4. Channel Alteration		8	13	11	4	8	8		
5. Bottom Scoring and Deposition		2	7	8	2	6	3		
6. Pool/riffle Ratio		3	1	2	8	4	2		
7. Bank Stability	9	2	5	3	2	8	2	6	7
8. Bank Vegetative	9	8	8	7	4	6	2	6	8
9. Streamside Cover	9	6	8	7	4	6	2	8	7
10. Predominant Land Use	I	FI	FC	FC	I	FI	I	CI	
11. Local Watershed Erosion	M	H	H	M	NN	M	H	M	
12. Local Watershed NPS Pollution	OS	OS	OS	OS	OS	OS	OS	OS	OS
13. Estimated Stream Width (m)		1	2	2	3	2	1	0.8	
14. High Water Mark (m)	SP	1	ST		CH	CH	CH		
15. Canopy Cover	OPO	PO	PO	PS	PO	OPO	PS	PO	
16. Sediment Odors	N	PC	N	SW		An	AnC	N	
17. Sediment Oils		SL	SL	AB	SL	AB	AB	AB	
18. Sediment Deposits		S	S(O/F)	S	S	S	S	S	Sg
19. Underlies black?		Y		Y		Y			
20. Inorganic Substrate									
21. Organic Substrate	MM	DMM	DMM	D	DMM	DMM	DMM	MM	MM
22. Water Odors									
23. Water Surface Oils		SH	SH		SH		SH	NN	
24. Turbidity		SL	CL	CL	SL	CL	CL	CL	

KEY:

AB - Absent	D - Detritus	P - Pond	SI - Silt
An - Anaerobic	DMM - Detritus/Muck-Mud	PC - Petroleum/Chemical	SL - Slight
AnC - Anaerobic/Chemical	FC - Field/Commercial	PO - Partly Open	SLT - Slight
C - Commercial	FI - Field/Industrial	PS - Partly Shaded	SP - Spill
CH - Channelized	H - Heavy	PBSD - Partly Shaded/Shaded	SPS - So
CI - Commercial/Industrial	HWM - High Water Mark	RF - Riffle	ST - Stag
CL - Clear	I - Industrial	RS - Residential	SW, SA - S
CL(OB) - Clear/Olive Brown	M - Moderate	RSC - Residential/Commercial	SW - Sew
CL(RB) - Clear(Rusty Brown)	MM - Muck-Mud	RSW - Residential/Wetland	SW - Slow
CL/SLT - Clear/Slightly Turbid	N - Normal	S - Sand	SY - Sand
CPM - Coarse Plant Material	NN - None	SD - Shaded	S(O/F) - Se
C/G, S/SI - Cobble/Gravel	O - Open	SG - Sludge	VEL - Velo
C/G - Cobble/Gravel	OPO - Open-Partly Open	SgY - Sludge/Yes	VHS - Vel
C/S - Cobble/Sand	OS - Obvious Source	Sh - Sheen	Y - Yes

TABLE G-1B3

DESCRIPTIVE STATISTICS AND FREQUENCY DISTRIBUTIONS
FOR TOTAL METALS IN GROUNDWATER

Groundwater/Surface Water Investigation Plan
Industri-Plex Superfund Site
Woburn, MA

	Silver	Sodium	Thallium	Vanadium	Zinc					
Class Width	1.5	17000	0.5	25	120					
Minimum	0.571	74.1	2.4	2.4	12.4					
Maximum	21.2	757000	10	370	9470					
Mean (geometric)	1.1	29,711.9	1.0	4.5	40.5					
Mean (arithmetic)	7.9	68,743.4	2.6	48.8	669.9					
IDL	3	38	2	6	8					
CRDL	10	5000	10	50	20					
	CLASS LIMITS	FREQUENCY								
	0	0	38	0	0	0	6	0	8	0
	1.5	1	17038	14	0.5	0	31	18	128	33
	3	0	34038	28	1	0	56	5	248	8
	4.5	0	51038	8	1.5	0	81	1	368	3
	6	2	68038	4	2	0	106	0	488	1
	7.5	0	85038	1	2.5	1	131	0	608	0
	9	0	102038	2	3	1	156	1	728	0
	10.5	0	119038	2	3.5	0	181	0	848	0
	12	0	136038	2	4	0	206	1	968	0
	13.5	0	153038	1	4.5	0	231	0	1088	1
	15	0	170038	1	5	0	256	1	1208	0
	16.5	0	187038	0	5.5	0	281	0	1328	0
	18	0	204038	0	6	0	306	0	1448	0
	19.5	0	221038	1	6.5	0	331	0	1568	1
	21	0	238038	1	7	0	356	0	1688	1
		1		2		0		1		4
N		4		65		2		33		63

NOTE: Class widths were generally calculated as (Maximum value - Instrument Limit of Detection)/15. For each frequency distribution, only the upper class limit is given. Frequencies cited will fall within the limit value next to it and the next lowest value. For example, if the class width is 50, and the frequency given is 11, next to a class limit of 100, then 11 values in the database fall below 100 but above 50. Also note that the minimum and maximum cited in this table are for detected values only, and that the means are calculated using zero for non-detects (not one-half of the detection limit). Maximum values were considered outliers when calculating the class width for aluminum, arsenic, antimony, copper, iron, lead, magnesium, mercury, sodium, and zinc.

Table G-1C

HABITAT ASSESSMENT SUMMARY

Groundwater/Surface Water Investigation Plan
 Industri-Plex Superfund Site
 Woburn, MA

SAMPLE #	BS-19	BS-20	BS-21	BS-22	BS-23	BS-24	BS-25	BS-26
DEPTH	1-2 ft.	< 1 ft.	< 1 ft.	1-3 ft.	3 ft.	1-3 ft.	1-3 ft.	7.5 ft.
1. Bottom Substrate	6	12	12					
2. Embeddedness	3	11	4					
3. Low Flow (<=0.15 cms)	10	2	4					
High Flow (>0.15 cms)	5	12	12					
4. Channel Alteration	13	13	4					
5. Bottom Scoring and Deposition	6	5	4					
6. Pool/riffle Ratio	8	10	5					
7. Bank Stability	9	9	7	10	9	7	7	6
8. Bank Vegetative	3	10	4	9	9	8	8	6
9. Streamside Cover	3	10	4	10	9	9	9	8
10. Predominant Land Use	RSC	RSW	RS	I	I	I	I	CI
11. Local Watershed Erosion	M	NN	M	H	M	M	M	M
12. Local Watershed NPS Pollution	OS	OS	OS	OS	OS	SPS	SPS	OS
13. Estimated Stream Width (m)	P	<0.5	2.5	P	P	P	P	P
14. High Water Mark (m)		VHS		SP				
15. Canopy Cover	O	PSSD	O	O		O	O	PO
16. Sediment Odors	N	N	N	N	N			N
17. Sediment Oils	AB	AB	AB	AB	AB			AB
18. Sediment Deposits				S	S	S	S	S
19. Undersides black?		No	Y					
20. Inorganic Substrate	S/SI	C/G,S/SI	C/G,S	MM	M(CPOM)	MM(FPOM)	MM(FPOM)	
21. Organic Substrate	D(CPOM)							MM
22. Water Odors		N	N	N	N	N	N	
23. Water Surface Oils		NN	NN	NN	NN	SH	SH	NN
24. Turbidity		CL		CL/SL	CL/SLT	SLT	SLT	CL

KEY:

AB - Absent

An - Anaerobic

AnC - Anaerobic/Chemical

C - Commercial

CH - Channelized

CI - Commercial/Industrial

CL - Clear

CL(OB) - Clear/Olive Brown

CL(RB) - Clear/Rusty Brown

CL/SLT - Clear/Slightly Turbid

CPM - Coarse Plant Material

C/G,S/SI - Cobbles/Gravel

C/G - Cobble/Gravel

C/S - Cobble/Sand

D - Detritus

DMM - Detritus/Muck-Mud

FC - Field/Commercial

FI - Field/Industrial

H - Heavy

HWM - High Water Mark

I - Industrial

M - Moderate

MM - Muck-Mud

N - Normal

NN - None

O - Open

DPO - Open-Partly Open

OS - Obvious Source

P - Pond

PC - Petroleum/Chemical

PO - Partly Open

PS - Partly Shaded

PSSD - Partly Shaded/Shaded

RF - Riffle

RS - Residential

RSC - Residential/Commercial

RSW - Residential/Wetland

S - Sand

SD - Shaded

Sg - Sludge

SgY - Sludge/Yes

Sh - Sheen

SI - Silt

SL - Slight

SLT - Slight

SP - Spill

SPS - So

ST - Stag

SW,BA - S

SW - Sew

SW - Slow

SY - Sand

S(O)F - Sa

VEL - Velo

VHS - Vel

Y - Yes

Table G-1C

HABITAT ASSESSMENT SUMMARY

Groundwater/Surface Water Investigation Plan
 Industri-Plex Superfund Site
 Woburn, MA

SAMPLE #	BS-10	BS-11	BS-12	BS-13	BS-14	BS-15	BS-16	BS-17	BS-18
DEPTH	<1 ft.	>15 ft.	0.5-3 ft.	>15 ft.	0.5-3 ft.	1 ft.	<0.5 ft.	<1 ft.	1-1.5 ft.
1. Bottom Substrate	18		8		16	4	5	4	6
2. Embeddedness	7		10		15	2	3	2	5
3. Low Flow (<=0.15 cms)	20		7		20	5	0	2	10
High Flow (>0.15 cms)	6		8		11	3	2	3	11
4. Channel Alteration	7		8		10	3	4	4	9
5. Bottom Scoring and Deposition	9		7		7	5	1	3	9
6. Pool/riffle Ratio	6		6		8	5	6	3	9
7. Bank Stability	7		7		9	4	7	3	8
8. Bank Vegetative	8		6		5	8	5	7	8
9. Streamside Cover	8		6		5	6	5	7	8
10. Predominant Land Use	CI	I	I	I	I	I	C	C	RS
11. Local Watershed Erosion	H	M	NN	M	M	H	M	M	NN
12. Local Watershed NPS Pollution	OS	SPS	OS	SPS	OS	OS	OS	OS	OS
13. Estimated Stream Width (m)	3	100	5/RF-3	100	HWM-2.0	3	4	2	3
14. High Water Mark (m)			CH	HWM.0.6	CH	CH	CH	CH	
15. Canopy Cover	PS		PO		PO	PS	SD	PO	SD
16. Sediment Odors	N	N	N		N	CAn	An	PC	N
17. Sediment Oils	AB	SL	SL		AB	AB	AB	AB	AB
18. Sediment Deposits		Sg	S	Sg		S			
19. Undersides black?	Y		Y		Y	Y		Y	No
20. Inorganic Substrate	C/G	SI							C/S
21. Organic Substrate	CPM	MM(FPOM)	D	MM(FPOM)	D	MM(FPOM)	DMM	D	D(CPOM)
22. Water Odors								PC	N
23. Water Surface Oils	NN		SH		NN	NN	SH	SH	NN
24. Turbidity	CL		CL(OB)		CL(RB)	CL			CL

KEY:

AB - Absent	D - Detritus	P - Pond	SI - Silt
An - Anaerobic	DMM - Detritus/Muck-Mud	PC - Petroleum/Chemical	SL - Slight
AnC - Anaerobic/Chemical	FC - Field/Commercial	PO - Partly Open	SLT - Slight
C - Commercial	FI - Field/Industrial	PS - Partly Shaded	SP - Spill
CH - Channelized	H - Heavy	PSSD - Partly Shaded/Shaded	SPS - So
CI - Commercial/Industrial	HWM - High Water Mark	RF - Riffle	ST - Stag
CL - Clear	I - Industrial	RS - Residential	SW,SA - S
CL(OB) - Clear/Olive Brown	M - Moderate	RSC - Residential/Commercial	SW - Sew
CL(RB) - Clear(Rusty Brown)	MM - Muck-Mud	RSW - Residential/Wetland	SW - Slow
CL/SLT - Clear/Slightly Turbid	N - Normal	S - Sand	SY - Sand
CPM - Coarse Plant Material	NN - None	SD - Shaded	S(O)F - Sa
C/G,S/Sl - Cobbles/Gravel	O - Open	Sg - Sludge	VEL - Velo
C/G - Cobble/Gravel	OPO - Open-Partly Open	SgY - Sludge/Yes	VHS - Vel
C/S - Cobble/Sand	OS - Obvious Source	Sh - Sheen	Y - Yes

TABLE G-1E

Pearson's Correlation Matrix for Physical
and Chemical Parameters in Surface Water and Sediment
(log transformed data)

Correlations:	SED_AL	SED_AS	SED_BA	SED_CD	SED_CA	SED_CR
SED_AL	1.0000					
SED_AS	-.0525	1.0000				
SED_BA	.5742*	.4264	1.0000			
SED_CD	-.3221	.6901**	.1496	1.0000		
SED_CA	.7806**	.2163	.1496	.1092	1.0000	
SED_CR	.3336	.6861**	.6856**	.4242	.4543	1.0000
SED_CO	.8180**	.7687**	.7402**	.0501	.8980**	.5978*
SED_CU	.6051*	.3626	.7310**	.3770	.7057**	.8312**
SED_FE	.7544**	.7025**	.8220**	.2379	.8356**	.7177**
SED_PB	.3928	.5160	.7831**	.3447	.4855	.8193**
SED_MG	.9322**	-.2068	.4914	-.2946	.8197**	.2032
SED_MN	.6751*	.2693	.6721*	.1054	.9325**	.4458
SED_HG	-.3972	.6545*	.1528	.2597	-.3256	.4672
SED_K	.9382**	-.1519	.5125	-.3637	.8146**	.2609
SED_NA	-.1462	.1174	.0304	.2616	-.0282	-.1129
SED_V	.9293**	.0865	.6297*	-.1202	.8747**	.3914
SED_ZN	.3467	.7928**	.4747	.5333	.5676*	.7143**
SED_SN	.7274**	.2354	.3859	-.2016	.4455	.3211
SW_AL	.3973	.1663	.4394	-.1731	.3657	.1698
SW_AS	-.0926	.3765	.1557	.4989	.1516	.3932
SW_BA	-.0058	.0526	.0527	.2852	.2960	.2431
SW_CA	-.0061	.2308	.2912	.1392	.2064	.4416
SW_CR	-.0487	.6789**	.5015	.3685	.1906	.6304*
SW_CU	-.0671	.3799	-.1542	.4051	.1167	.0577
SW_FE	-.0474	.2776	.2722	.1875	.1804	.4586
SW_PB	.0537	-.0828	-.0265	.1492	.2017	.1322
SW_MG	-.0164	.2331	.2913	.1412	.1970	.4380
SW_MN	-.0702	.3998	.3093	.2373	.1656	.5200
SW_K	-.0396	.2518	.2823	.1561	.1886	.4216
SW_NA	-.0281	.2431	.2959	.1252	.1890	.4043
SW_ZN	-.2038	-.1462	-.2552	.0025	-.0823	.0180
CONDUCT.	-.0563	.3113	.3416	.2405	.2352	.2660
RICH	.3590	.0015	.3077	-.1708	.3358	.2117
ABUND	.0643	-.0869	.0414	-.1261	.0593	.0823
DI	.4911	.0229	.4190	-.2010	.3894	.3176
HAB	.3901	.1091	.4995	-.1434	.3396	.3355
SO4_SW	-.0385	.3650	.3453	.1984	.1743	.5201
EH_SW	.3163	.2070	.3107	.0033	.3611	.3929
EH_SED	.4316	-.3183	.0274	-.2486	.2707	-.2425
HARDNESS	-.0067	.2285	.2892	.1368	.2041	.4386

N of cases: 18

1-tailed Signif: * - .01 ** - .001

*Note: Sediment metal concentrations were "normalized" to grain size by multiplying by a "dilution" factor (100/percent less than 125 microns).

TABLE G-1D

NUMBER AND TYPES OF BENTHIC MACROINVERTEBRATES
 SAMPLED AT SELECTED BIOLOGICAL SAMPLING STATIONS

Groundwater/Surface Water Investigation Plan
 Industri-Plex Superfund Site
 Woburn, MA

INSECT FAMILY	BIOLOGICAL SAMPLING STATION NO.																												
	1A	1B	1C	2	3	4	6	8	7	8	8	10	11	12	13	14A	14B	15	16	17	18	19	20	21	22	23	24	25	26
EPHEMEROPTERA																													
Caenidae						1																							
PLECOPTERA																													
Nemouridae						1																							
TRICHOPTERA																													
Hydropsychidae						1						10				37						8	4		1				
Limnephilidae					1	1	1																1						
Polycentropodidae					1																								
ODONATA																													
Coenagrionidae								2						1															
Libellulidae					1																								
Aeschnidae										1																			
Gomphidae	1									1												2							
HEMiptera																													
Corixidae																													
Notonectidae																													
Belostomatidae														1															
Hydrometridae						1																							
COLEOPTERA																													
Dytiscidae				8	11		11	13						2						1			7		2				
Gyrinidae																					1								
Eimidae				2		1							1									2	2						
Halipidae						1																	5						
Hydrophilidae					1																								
DIPTERA																													
Chironomidae	14	84	80	125		8	241	285		4		14		155	2	31	75	3	94	2	26	71	75	160	81	31	3	38	8
Ceratopogonidae			1												1														1
Simuliidae				1028		3	20							82		4	8				5	8	10	1	8				
Tipulidae					5				1																				
Empididae																													
Stratiomyidae					1																								
Byrrhidae					1																								
LEPIDOPTERA																													
Pyridae																									1				
ISOPODA																													
Aeidae				82	15	1	8					3			12		1					27	94		28			12	
AMPHIPODA																													
Gammaridae					15						15															1		1	
Talitridae				2		18	5	3																2					2
DECAPODA																													
Cambarinae																													
MOLLUSCA																													
Gastropoda																													
Planorbidae								1															1		1				
Viviparidae																													
Physidae				2																			11						
Ancylidae								1																					
Lymnaeidae								1																					
Pelecypoda																													
Sphaeriidae				38			1																1	1	2		1		
Unionidae																													2
MISCELLANEOUS																													
Oligochaeta		8	3	344			111	58		222		1		3	30	3	28	25				477	788	317	148	88	40	18	77
Nematoda																	1												
Hirudinae				3	2																		2	25	1				
Hydracarina																								2					
Cladocera																													78
Copepoda																													20

Includes pupae identified to family.

TABLE G-1E

Pearson's Correlation Matrix for Physical
and Chemical Parameters in Surface Water and Sediment
(log transformed data)

Correlations:	SED_HG	SED_K	SED_NA	SED_V	SED_ZN	SED_SN
SED_AL	-.3972	.9382**	-.1462	.9293**	.3467	.7274**
SED_AS	.6545*	-.1519	.1174	.0865	.7928**	.2354
SED_BA	.1528	.5125	.0304	.6297*	.4747	.3859
SED_CD	.2597	-.3637	.2616	-.1202	.5333	-.2016
SED_CA	-.3256	.8146**	-.0282	.8747**	.5676*	.4455
SED_CR	.4672	.2609	-.1129	.3914	.7143**	.3211
SED_CO	-.0743	.7795**	.0189	.8854**	.6874**	.5786*
SED_CU	.2315	.5006	.0698	.6968**	.8650**	.6302*
SED_FE	-.0121	.7011**	.0220	.8140**	.6564*	.5470*
SED_PB	.4683	.2845	.0001	.4722	.6926**	.4710
SED_MG	-.6290*	.9563**	-.2361	.9085**	.2182	.4845
SED_MN	-.1846	.7484**	.0035	.7164**	.5289	.3685
SED_HG	1.0000	-.4839	.2460	-.4211	.2789	.0532
SED_K	-.4839	1.0000	-.1469	.8791**	.2506	.5413
SED_NA	.2460	-.1469	1.0000	-.1414	.0512	.1142
SED_V	-.4211	.8791**	-.1414	1.0000	.4823	.6305*
SED_ZN	.2789	.2506	-.1414	.4823	1.0000	.4771
SED_SN	.0532	.5413	.0512	.4823	1.0000	.4771
SW_AL	.2225	.4145	.1142	.6305*	.4771	1.0000
SW_AS	.1297	-.0992	-.2646	.3403	.1504	.3791
SW_BA	-.0558	.1646	-.1826	-.1154	.2336	-.2098
SW_CA	.3299	.1197	-.1432	-.0403	.1424	-.2407
SW_CR	.5862*	-.0006	.1876	-.0701	.1999	-.1142
SW_CU	.1973	-.0522	.2649	-.0430	.3573	.0639
SW_FE	.3654	.0746	-.1250	-.0842	.4550	.1984
SW_PB	-.2811	.2269	-.2648	-.1088	.2109	-.1382
SW_MG	.3341	.1077	-.2648	.0498	.0940	-.2416
SW_MN	.4738	.0363	-.1588	-.0811	.1915	-.1227
SW_K	.3617	.0891	-.1302	-.1259	.2746	-.1065
SW_NA	.3777	.0965	-.1102	-.1035	.1957	-.1263
SW_ZN	-.0769	-.0769	-.0940	-.0944	.1908	-.0983
CONDUCT.	.3193	-.0396	-.3404	-.2267	.0055	-.3009
RICH	-.1844	.3751	.0799	-.0071	.1932	-.0002
ABUND	-.1307	.0901	-.4090	.3888	-.0997	.1800
DI	-.1933	.5242	-.5193	.1012	-.2841	-.0856
HAB	-.0218	.4217	-.4431	.4289	-.0182	.1735
SO4_SW	.4653	.0538	-.4126	.3064	-.0340	.1239
EH_SW	.2133	.3376	-.1098	-.1002	.2821	-.0621
EH_SED	-.3021	.4112	-.0788	.2417	.4617	.3530
HARDNESS	.3289	.1185	.0581	.3057	-.1870	.3945
			-.1469	-.0713	.1977	-.1140

N of cases: 18

1-tailed Signif: * - .01 ** - .001

*Note: Sediment metal concentrations were "normalized" to grain size by multiplying by a "dilution" factor (100/percent less than 125 microns).

TABLE G-1E

Pearson's Correlation Matrix for Physical
and Chemical Parameters in Surface Water and Sediment
(log transformed data)

Correlations:	SED_CO	SED_CU	SED_FE	SED_PB	SED_MG	SED_MN
SED_AL	.8180**	.6051*	.7544**	.3928	.9322**	.6751*
SED_AS	.3626	.7025**	.5160	.7622**	-.2068	.2693
SED_BA	.7402**	.7310**	.8220**	.7831**	.4914	.6721*
SED_CD	.0501	.3770	.2379	.3447	-.2946	.1054
SED_CA	-.8980**	.7057**	.8356**	.4855	.8197**	.9325**
SED_CR	.5978*	.8312**	.7177**	.8193**	.2032	.4458
SED_CO	1.0000	.8344**	.8985**	.6447*	.7373**	.8460**
SED_CU	.8344**	1.0000	.8694**	.8434**	.4546	.6485*
SED_FE	.8985**	.8694**	1.0000	.7890**	.6703*	.7997**
SED_PB	.6447*	.8434**	.7890**	1.0000	.2223	.4733
SED_MG	.7373**	.4546	.6703*	.2223	1.0000	.6992**
SED_MN	.8460**	.6485*	.7997**	.4733	.6992**	1.0000
SED_HG	-.0743	.2315	-.0121	.4683	-.6290*	-.1846
SED_K	.7795**	.5006	.7011**	.2845	.9563**	.7484**
SED_NA	.0189	.0698	.0220	.0001	-.2361	.0035
SED_V	.8854**	.6968**	.8140**	.4722	.9085**	.7164**
SED_ZN	.6874**	.8650**	.6564*	.6926**	.2182	.5289
SED_SN	.5786*	.6302*	.5470*	.4710	.4845	.3685
SW_AL	.4966	.3405	.4589	.4453	.2933	.4617
SW_AS	.0331	.1301	.1867	.1656	-.0070	.2299
SW_BA	.0714	.0891	.0935	.0201	.1266	.3569
SW_CA	.1378	.2147	.1609	.3275	.0072	.2881
SW_CR	.2557	.4505	.4349	.5860*	-.1441	.3972
SW_CU	.1141	.2390	.0758	.0784	-.1287	.2234
SW_FE	.1224	.2084	.1553	.3161	-.0368	.2780
SW_PB	.0061	.0188	.0283	-.0671	.2221	.1369
SW_MG	.1251	.2050	.1560	.3324	-.0016	.2822
SW_MN	.1409	.2675	.2081	.4205	-.0896	.2940
SW_K	.1223	.2006	.1530	.3333	-.0305	.2882
SW_NA	.1321	.1995	.1526	.3459	-.0286	.2867
SW_ZN	-.2347	-.1875	-.3402	-.3257	-.0933	-.1226
CONDUCT.	.1363	.1946	.2109	.3604	-.1167	.2357
RICH	.2184	.1248	.3870	.2900	.3678	.2602
ABUND	-.0665	-.0972	.0775	.1007	.1176	.0055
DI	.3321	.1864	.5354	.3358	.4915	.3908
HAB	.2811	.1825	.5141	.4207	.3908	.4078
SO4_SW	.1523	.2830	.1965	.4259	-.0693	.2792
EH_SW	.3841	.4480	.2708	.4053	.2259	.3551
EH_SED	.1565	-.0012	.2052	.0621	.4115	.2258
HARDNESS	.1352	.2121	.1583	.3269	.0067	.2855

N of cases: 18 1-tailed Signif: * - .01 ** - .001

*Note: Sediment metal concentrations were "normalized" to grain size by multiplying by a "dilution" factor (100/percent less than 125 microns).

TABLE G-1E

Pearson's Correlation Matrix for Physical
and Chemical Parameters in Surface Water and Sediment
(log transformed data)

Correlations:	SW_FE	SW_PB	SW_MG	SW_MN	SW_K	SW_NA
SED_AL	-.0474	.0537	-.0164	-.0702	-.0396	-.0281
SED_AS	.2776	-.0828	.2331	.3998	.2518	.2431
SED_BA	.2722	-.0265	.2913	.3093	.2823	.2959
SED_CD	.1875	.1492	.1412	.2373	.1561	.1252
SED_CA	.1804	.2017	.1970	.1656	.1886	.1890
SED_CR	.4586	.1322	.4380	.5200	.4216	.4043
SED_CO	.1224	.0061	.1251	.1409	.1223	.1321
SED_CU	.2084	.0188	.2050	.2675	.2006	.1995
SED_FE	.1553	.0283	.1560	.2081	.1530	.1526
SED_PB	.3161	-.0671	.3324	.4205	.3333	.3459
SED_MG	-.0368	.2221	-.0016	-.0896	-.0305	-.0286
SED_MN	.2780	.1369	.2822	.2940	.2882	.2867
SED_HG	.3654	-.2811	.3341	.4738	.3617	.3777
SED_K	.0746	.2269	.1077	.0363	.0891	.0965
SED_NA	-.1250	-.2648	-.1588	-.1302	-.1102	-.0940
SED_V	-.1088	.0498	-.0811	-.1259	-.1035	-.0944
SED_ZN	.2109	.0940	.1915	.2746	.1957	.1908
SED_SN	-.1382	-.2416	-.1227	-.1065	-.1263	-.0983
SW_AL	.3137	-.0425	.3056	.3664	.3397	.3876
SW_AS	.5556*	.4212	.5055	.5668*	.4985	.4665
SW_BA	.8322**	.8104**	.8303**	.7786**	.8232**	.7981**
SW_CA	.9930**	.6958**	.9994**	.9716**	.9969**	.9917**
SW_CR	.5533*	.0470	.5017	.6303*	.5277	.5135
SW_CU	.3886	.3241	.3343	.4128	.3753	.3769
SW_FE	1.0000	.6852**	.9919**	.9821**	.9928**	.9845**
SW_PB	.6852**	1.0000	.6882**	.5923*	.6685*	.6433*
SW_MG	.9919**	.6882**	1.0000	.9741**	.9973**	.9925**
SW_MN	.9821**	.5923*	.9741**	1.0000	.9804**	.9754**
SW_K	.9928**	.6685*	.9973**	.9804**	1.0000	.9969**
SW_NA	.9845**	.6433*	.9925**	.9754**	.9969**	1.0000
SW_ZN	.5909*	.7919**	.5860*	.4928	.5619*	.5404
CONDUCT.	.6521*	.3537	.6587*	.6532*	.6736*	.6835**
RICH	.1563	.1334	.1818	.1638	.1605	.1638
ABUND	.2254	.1839	.2399	.2155	.2159	.2067
DI	.1410	.1323	.1713	.1809	.1495	.1491
HAB	.2580	.0854	.2908	.3293	.2783	.2879
SO4_SW	.9854**	.6036*	.9850**	.9890**	.9871**	.9843**
EH_SW	.7645**	.5844*	.7895**	.7355**	.7850**	.7994**
EH_SED	-.0155	.0023	.0408	-.0152	.0456	.0790
HARDNESS	.9927**	.6959**	.9995**	.9713**	.9969**	.9919**

N of cases: 18 1-tailed Signif: * - .01 ** - .001

*Note: Sediment metal concentrations were "normalized" to grain size by multiplying by a "dilution" factor (100/percent less than 125 microns).

TABLE G-1E

Pearson's Correlation Matrix for Physical
and Chemical Parameters in Surface Water and Sediment
(log transformed data)

Correlations:	SW_AL	SW_AS	SW_BA	SW_CA	SW_CR	SW_CU
SED_AL	.3973	-.0926	-.0058	-.0061	-.0487	-.0671
SED_AS	.1663	.3765	.0526	.2308	.6789**	.3799
SED_BA	.4394	.1557	.0527	.2912	.5015	-.1542
SED_CD	-.1731	.4989	.2852	.1392	.3685	.4051
SED_CA	.3657	.1516	.2960	.2064	.1906	.1167
SED_CR	.1698	.3932	.2431	.4416	.6304*	.0577
SED_CO	.4966	.0331	.0714	.1378	.2557	.1141
SED_CU	.3405	.1301	.0891	.2147	.4505	.2390
SED_FE	.4589	.1867	.0935	.1609	.4349	.0758
SED_PB	.4453	.1656	.0201	.3275	.5860*	.0784
SED_MG	.2933	-.0070	.1266	.0072	-.1441	-.1287
SED_MN	.4617	.2299	.3569	.2881	.3972	.2234
SED_HG	.2225	.1297	-.0558	.3299	.5862*	.1973
SED_K	.4145	-.0992	.1646	.1197	-.0006	-.0522
SED_NA	.1694	-.2646	-.1826	-.1432	.1876	.2649
SED_V	.3403	-.1154	-.0403	-.0701	-.0430	-.0842
SED_ZN	.1504	.2336	.1424	.1999	.3573	.4550
SED_SN	.3791	-.2098	-.2407	-.1142	.0639	.1984
SW_AL	1.0000	.0253	.0808	.3077	.3803	.3719
SW_AS	.0253	1.0000	.6028*	.4953	.3788	.3440
SW_BA	.0808	.6028*	1.0000	.8322**	.2536	.4414
SW_CA	.3077	.4953	.8322**	1.0000	.5011	.3430
SW_CR	.3803	.3788	.2536	.5011	1.0000	.2877
SW_CU	.3719	.3440	.4414	.3430	.2877	1.0000
SW_FE	.3137	.5556*	.8322**	.9930**	.5533*	.3886
SW_PB	-.0425	.4212	.8104**	.6958**	.0470	.3241
SW_MG	.3056	.5055	.8303**	.9994**	.5017	.3343
SW_MN	.3664	.5668*	.7786**	.9716**	.6303*	.4128
SW_K	.3397	.4985	.8232**	.9969**	.5277	.3753
SW_NA	.3876	.4665	.7981**	.9917**	.5135	.3769
SW_ZN	-.3040	.3102	.6813**	.5946*	-.0962	.2385
CONDUCT.	.2884	.2613	.4971	.6572*	.3876	.2804
RICH	.2287	.0622	.1546	.1726	.1447	-.2617
ABUND	.1100	.1440	.2359	.2284	.1039	-.2259
DI	.2122	.1673	.1603	.1595	.1530	-.3144
HAB	.3666	.3178	.2034	.2722	.2825	-.2351
SO4_SW	.3370	.5154	.7601**	.9841**	.5982*	.3752
EH_SW	.4028	.1975	.6181*	.7982**	.2529	.5023
EH_SED	.4547	-.0802	.1540	.0332	-.2670	.1235
HARDNESS	.3080	.4952	.8317**	1.0000**	.4980	.3426
N of cases:	18	1-tailed Signif: * - .01 ** - .001				

*Note: Sediment metal concentrations were "normalized" to grain size by multiplying by a "dilution" factor (100/percent less than 125 microns).

TABLE G-1E

Pearson's Correlation Matrix for Physical
and Chemical Parameters in Surface Water and Sediment
(log transformed data)

Correlations:	SO4_SW	EH_SW	EH_SED	HARDNESS
SED_AL	-.0385	.3163	.4316	-.0067
SED_AS	.3650	.2070	-.3183	.2285
SED_BA	.3453	.3107	.0274	.2892
SED_CD	.1984	.0033	-.2486	.1368
SED_CA	.1743	.3611	.2707	.2041
SED_CR	.5201	.3929	-.2425	.4386
SED_CO	.1523	.3841	.1565	.1352
SED_CU	.2830	.4480	-.0012	.2121
SED_FE	.1965	.2708	.2052	.1583
SED_PB	.4259	.4053	.0621	.3269
SED_MG	-.0693	.2259	.4115	.0067
SED_MN	.2792	.3551	.2258	.2855
SED_HG	.4653	.2133	-.3021	.3289
SED_K	.0538	.3376	.4112	.1185
SED_NA	-.1098	-.0788	.0581	-.1469
SED_V	-.1002	.2417	.3057	-.0713
SED_ZN	.2821	.4617	-.1870	.1977
SED_SN	-.0621	.3530	.3945	-.1140
SW_AL	.1370	.4028	.4547	.3080
SW_AS	.5154	.1975	-.0802	.4952
SW_BA	.7601**	.6181*	.1540	.8317**
SW_CA	.9841**	.7982**	.0332	1.0000**
SW_CR	.5982*	.2529	-.2670	.4980
SW_CU	.3752	.5023	.1235	.3426
SW_FE	.9854**	.7645**	-.0155	.9927**
SW_PB	.6036*	.5844*	.0023	.6959**
SW_MG	.9850**	.7895**	.0408	.9995**
SW_MN	.9890**	.7355**	-.0152	.9713**
SW_K	.9871**	.7850**	.0456	.9969**
SW_NA	.9843**	.7994**	.0790	.9919**
SW_ZN	.5245	.5000	-.2750	.5947*
CONDUCT.	.6642*	.5139	.1606	.6571*
RICH	.1128	.0317	.4229	.1738
ABUND	.1762	.0004	.3143	.2307
DI	.1354	-.0081	.3719	.1598
HAB	.2757	.0404	.3924	.2732
SO4_SW	1.0000	.7920**	-.0309	.9839**
EH_SW	.7920**	1.0000	.1878	.7991**
EH_SED	-.0309	.1878	1.0000	.0364
HARDNESS	.9839**	.7991**	.0364	1.0000

N of cases: 18 1-tailed Signif: * - .01 ** - .001

*Note: Sediment metal concentrations were "normalized" to grain size by multiplying by a "dilution" factor (100/percent less than 125 microns).

TABLE G-1E

Pearson's Correlation Matrix for Physical
and Chemical Parameters in Surface Water and Sediment
(log transformed data)

Correlations:	SW_ZN	CONDUCT.	RICH	ABUND	DI	HAB
SED_AL	-.2038	-.0563	.3590	.0643	.4911	.3901
SED_AS	-.1462	.3113	.0015	-.0869	.0229	.1091
SED_BA	-.2552	.3416	.3077	.0414	.4190	.4995
SED_CD	.0025	.2405	-.1708	-.1261	-.2010	-.1434
SED_CA	-.0823	.2352	.3358	.0593	.3894	.3396
SED_CR	.0180	.2660	.2117	.0823	.3176	.3355
SED_CO	-.2347	.1363	.2184	-.0665	.3321	.2811
SED_CU	-.1875	.1946	.1248	-.0972	.1864	.1825
SED_FE	-.3402	.2109	.3870	.0775	.5354	.5141
SED_PB	-.3257	.3604	.2900	.1007	.3358	.4207
SED_MG	-.0933	-.1167	.3678	.1176	.4915	.3908
SED_MN	-.1226	.2357	.2602	.0055	.3908	.4078
SED_HG	-.0769	.3193	-.1844	-.1307	-.1933	-.0218
SED_K	-.0769	-.0396	.3751	.0901	.5242	.4217
SED_NA	-.3404	.0799	-.4090	-.5193	-.4431	-.4126
SED_V	-.2267	-.0071	.3888	.1012	.4289	.3064
SED_ZN	.0055	.1932	-.0997	-.2841	-.0182	-.0340
SW_AL	-.3009	-.0002	.1800	-.0856	.1735	.1239
SW_AS	-.3040	.2884	.2287	.1100	.2122	.3666
SW_BA	.3102	.2613	.0622	.1440	.1673	.3178
SW_CA	.6813**	.4971	.1546	.2359	.1603	.2034
SW_CR	.5946*	.6572*	.1726	.2284	.1595	.2722
SW_CO	-.0962	.3876	.1447	.1039	.1530	.2825
SW_CU	.2385	.2804	-.2617	-.2259	-.3144	-.2351
SW_FE	.5909*	.6521*	.1563	.2254	.1410	.2580
SW_PB	.7919**	.3537	.1334	.1839	.1323	.0854
SW_MG	.5860*	.6587*	.1818	.2399	.1713	.2908
SW_MN	.4928	.6532*	.1638	.2155	.1809	.3293
SW_K	.5619*	.6736*	.1605	.2159	.1495	.2783
SW_NA	.5404	.6835**	.1638	.2067	.1491	.2879
SW_ZN	1.0000	.3202	-.0472	.0806	-.0972	-.1521
CONDUCT.	.3202	1.0000	.4390	.3892	.2347	.2784
RICH	-.0472	.4390	1.0000	.8859**	.8168**	.7080**
ABUND	.0806	.3892	.8859**	1.0000	.5733*	.4953
DI	-.0972	.2347	.8168**	.5733*	1.0000	.9278**
HAB	-.1521	.2784	.7080**	.4953	.9278**	1.0000
SO4_SW	.5245	.6642*	.1328	.1762	.1354	.2757
EH_SW	.5000	.5139	.0317	.0004	-.0081	.0404
EH_SED	-.2750	.1606	.4229	.3143	.3719	.3924
HARDNESS	.5947*	.6571*	.1738	.2307	.1598	.2732

N of cases: 18 1-tailed Signif: * - .01 ** - .001

*Note: Sediment metal concentrations were "normalized" to grain size by multiplying by a "dilution" factor (100/percent less than 125 microns).

APPENDIX G-1F

I Ingestion and Household Use of Groundwater

A. Equation: Ingestion

$$\text{Exposure Concentration (mg/kg/day)} = \frac{(\text{CW}) (\text{IR}) (\text{EF}) (\text{ED}) (\text{RAF})}{(\text{BW}) (\text{AT})}$$

where,

CW	=	Chemical Concentration in Water (mg/liter)
IR	=	Ingestion Rate (liters/day)
EF	=	Exposure Frequency (days/year)
ED	=	Exposure Duration (years)
BW	=	Body Weight (kg)
AT	=	Averaging Time (period over which exposure is averaged -- days)
RAF	=	Relative Absorption Factor (unitless)

1. Example for Arsenic (carcinogenic; adult from Table 4.21):

$$\begin{aligned} \text{Exposure Concentration (mg/kg/day)} &= \frac{(209 \text{ ug/l})(0.001 \text{ mg/ug})(2 \text{ l/day})(365 \text{ days/yr})(70 \text{ yrs})(1)}{(70 \text{ kg})(70 \text{ yr})(365 \text{ days/yr})} \\ &= 5.97\text{E-}03 \text{ mg/kg/day} \end{aligned}$$

2. Example for Arsenic (non-carcinogenic; child from Table 4.21):

$$\begin{aligned} \text{Exposure Concentration (mg/kg/day)} &= \frac{(7.1 \text{ ug/l})(0.001 \text{ mg/ug})(1 \text{ l/hr})(365 \text{ days/yr})(5 \text{ yrs})(1)}{(10 \text{ kg})(5 \text{ yr})(365 \text{ days/yr})} \\ &= 7.1\text{E-}04 \text{ mg/kg/day} \end{aligned}$$

APPENDIX G-1F
Risk Assessment Documentation
Equations and Sample Risk Assessment Calculations

B. Equation: Ingestion of Chemicals in Sediment.

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

where,

CS	=	Chemical Concentration in Soil (mg/kg)
IR	=	Ingestion Rate (mg soil/day)
CF	=	Conversion Factor (10 ⁻⁶ kg/mg)
FI	=	Fraction Ingested from Contaminated Source (unitless)
EF	=	Exposure Frequency (days/years)
ED	=	Exposure Duration (years)
BW	=	Body Weight (kg)
AT	=	Averaging Time (period over which exposure is averaged -- days)

1. Example for Arsenic (carcinogenic; child from Table 4.22):

$$\begin{aligned} \text{Exposure Concentration (mg/kg/day)} &= \frac{(55.7 \text{ mg/kg})(100 \text{ mg/day})(0.000001 \text{ kg/mg})(1)(12 \text{ days/yr})(5 \text{ yr})}{(45 \text{ kg})(70 \text{ yr})(365 \text{ days/yr})} \\ &= 2.91\text{E-}07 \text{ mg/kg/day} \end{aligned}$$

2. Example for Arsenic (carcinogenic; adult from Table 4.23):

$$\begin{aligned} \text{Exposure Concentration (mg/kg/day)} &= \frac{(55.7 \text{ mg/kg})(50 \text{ mg/day})(0.000001 \text{ kg/mg})(1)(12 \text{ days/yr})(10 \text{ yr})}{(70 \text{ kg})(70 \text{ yr})(365 \text{ days/yr})} \\ &= 1.87\text{E-}07 \text{ mg/kg/day} \end{aligned}$$

3. Example for Arsenic (carcinogenic; child from Table 4.22):

$$\begin{aligned} \text{Exposure Concentration (mg/kg/day)} &= \frac{(55.7 \text{ mg/kg})(100 \text{ mg/day})(0.000001 \text{ kg/mg})(1)(12 \text{ days/yr})(5 \text{ yr})}{(45 \text{ kg})(5 \text{ yr})(365 \text{ days/yr})} \\ &= 4.07\text{E-}06 \text{ mg/kg/day} \end{aligned}$$

II Recreational Bathing - Wading or Swimming

A. Equation: Ingestion of Chemicals in Surface Water.

$$\text{Exposure Concentration (mg/kg/day)} = \frac{(CW)(CR)(ET)(EF)(ED)(RAF)}{(BW)(AT)}$$

where,

- CW = Chemical Concentration in Water (mg/liter)
- CR = Contact Rate (liters/hour)
- ET = Exposure Time (hours/event)
- EF = Exposure Frequency (events/year)
- ED = Exposure Duration (years)
- BW = Body Weight (kg)
- AT = Averaging Time (period over which exposure is averaged -- days)
- RAF = Relative Absorption Factor (unitless)

1. Example for Arsenic (carcinogenic; child from Table 4.22):

$$\begin{aligned} \text{Exposure Concentration (mg/kg/day)} &= \frac{(6.4 \text{ ug/l})(0.001 \text{ mg/ug})(0.05 \text{ l/hr})(2.6 \text{ hr/day})(12 \text{ days/yr})(5 \text{ yrs})(1)}{(45 \text{ kg})(70 \text{ yr})(365 \text{ days/yr})} \\ &= 4.34\text{E-}08 \text{ mg/kg/day} \end{aligned}$$

2. Example for Arsenic (carcinogenic; adult from Tables 4.23):

$$\begin{aligned} \text{Exposure Concentration (mg/kg/day)} &= \frac{(6.4 \text{ ug/l})(0.001 \text{ mg/ug})(0.05 \text{ l/hr})(2.6 \text{ hr/day})(12 \text{ days/yr})(10 \text{ yrs})(1)}{(70 \text{ kg})(70 \text{ yr})(365 \text{ days/yr})} \\ &= 5.58\text{E-}08 \text{ mg/kg/day} \end{aligned}$$

3. Example for Arsenic (non-carcinogenic; child from Table 4.22):

$$\begin{aligned} \text{Exposure Concentration (mg/kg/day)} &= \frac{(6.4 \text{ ug/l})(0.001 \text{ mg/ug})(0.05 \text{ l/hr})(2.6 \text{ hr/day})(12 \text{ days/yr})(5 \text{ yrs})(1)}{(45 \text{ kg})(5 \text{ yr})(365 \text{ days/yr})} \\ &= 6.08\text{E-}07 \text{ mg/kg/day} \end{aligned}$$

III Ingestion of Sediment

A. Equation: Ingestion of Chemicals in Soil

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

where,

- CS = Chemical Concentration in Soil (mg/kg)
- IR = Ingestion Rate (mg soil/day)
- CF = Conversion Factor (10⁻⁶ kg/mg)
- FI = Fraction Ingested from Contaminated Source (unitless)
- EF = Exposure Frequency (days/years)
- ED = Exposure Duration (years)
- BW = Body Weight (kg)
- AT = Averaging Time (period over which exposure is averaged -- days)

1. Example for Arsenic (non-carcinogenic; child from Table 4.26):

$$\begin{aligned} \text{Exposure Concentration (mg/kg/day)} &= \frac{(928 \text{ mg/kg})(100 \text{ mg/day})(0.000001 \text{ kg/mg})(1)(30 \text{ days/yr})(5 \text{ yr})}{(45 \text{ kg})(5 \text{ yr})(365 \text{ days/yr})} \\ &= 1.69\text{E-}04 \text{ mg/kg/day} \end{aligned}$$

2. Example for Arsenic (carcinogenic; child from Table 4.26):

$$\begin{aligned} \text{Exposure Concentration (mg/kg/day)} &= \frac{(928 \text{ mg/kg})(100 \text{ mg/day})(0.000001 \text{ kg/mg})(1)(30 \text{ days/yr})(5 \text{ yr})}{(45 \text{ kg})(70 \text{ yr})(365 \text{ days/yr})} \\ &= 1.21\text{E-}05 \text{ mg/kg/day} \end{aligned}$$

C. Equation: Dermal Contact with Chemicals in Water.

$$\text{Exposure Concentration (mg/kg/day)} = \frac{(CW)(SA)(PC)(ET)(EF)(ED)(CF)}{(BW)(AT)}$$

where,

CW	=	Chemical Concentration in Water (mg/liter)
SA	=	Skin Surface Area Available for Contact (cm ²)
PC	=	Chemical-specific Dermal Permeability Constant (cm/hr)
ET	=	Exposure Time (hours/day)
EF	=	Exposure Frequency (days/year)
ED	=	Exposure Duration (years)
CF	=	Volumetric Conversion Factor for Water (1 liter/1000 cm ³)
BW	=	Body Weight (kg)
AT	=	Averaging Time (period over which exposure is averaged -- days)

1. Example for Methylene Chloride (carcinogenic; child from Table 4.24):

$$\begin{aligned} \text{Exposure Concentration} \\ (\text{mg/kg/day}) &= \frac{(7.7 \text{ ug/l})(0.001 \text{ mg/ug})(14850 \text{ cm}^2)(0.008 \text{ cm/hr})(2.6 \text{ hr/day})(12 \text{ days/yr})(5 \text{ yr})(0.001 \text{ l/cm}^3)}{(45 \text{ kg})(70 \text{ yr})(365 \text{ days/yr})} \\ &= 1.30\text{E-}07 \text{ mg/kg/day} \end{aligned}$$

2. Example for Methylene Chloride (carcinogenic; adult from Table 4.25):

$$\begin{aligned} \text{Exposure Concentration} \\ (\text{mg/kg/day}) &= \frac{(7.7 \text{ ug/l})(0.001 \text{ mg/ug})(18150 \text{ cm}^2)(0.008 \text{ cm/hr})(2.6 \text{ hr/day})(12 \text{ days/yr})(10 \text{ yr})(0.001 \text{ l/cm}^3)}{(70 \text{ kg})(70 \text{ yr})(365 \text{ days/yr})} \\ &= 2.04\text{E-}07 \text{ mg/kg/day} \end{aligned}$$

3. Example for Methylene Chloride (carcinogenic; child from Table 4.24):

$$\begin{aligned} \text{Exposure Concentration} \\ (\text{mg/kg/day}) &= \frac{(7.7 \text{ ug/l})(0.001 \text{ mg/ug})(14850 \text{ cm}^2)(0.008 \text{ cm/hr})(2.6 \text{ hr/day})(12 \text{ days/yr})(5 \text{ yr})(0.001 \text{ l/cm}^3)}{(45 \text{ kg})(5 \text{ yr})(365 \text{ days/yr})} \\ &= 1.82\text{E-}06 \text{ mg/kg/day} \end{aligned}$$

1
RISK CHARACTERIZATION
HYPOTHETICAL EXPOSURE PATHWAY: Industrial Exposure

Groundwater/Surface Water Investigation Plan
Industri-Plex Superfund Site
Woburn, MA

CHEMICAL	RISK CHARACTERIZATION FOR INDUSTRIAL GROUNDWATER USE							
	CDI (cancer)		CANCER RISK		CDI (non-cancer)		HAZARD INDEX	
	MAXIMUM OFF-SITE	MEAN OFF-SITE	MAXIMUM OFF-SITE	MEAN OFF-SITE	MAXIMUM OFF-SITE	MEAN OFF-SITE	MAXIMUM OFF-SITE	MEAN OFF-SITE
Acetone	0.00E+00	5.11E-06			0.00E+00	7.16E-05	0.0E+00	7.2E-04
Antimony	1.39E-04	9.59E-05			1.95E-03	1.34E-03	4.9E+00	3.4E+00
Arsenic (3)	1.06E-03	3.63E-05	2.3E-04	8.0E-06	1.49E-02	5.08E-04	1.0E+01	3.6E-01
Barium	2.57E-03	7.72E-05			3.60E-02	1.08E-03	7.2E-01	2.2E-02
Benzene	1.02E-02	4.86E-06	3.0E-04	1.4E-07	1.42E-01	6.81E-05	0.0E+00	0.0E+00
Benzoic acid	0.00E+00	6.36E-05			0.00E+00	8.90E-04	0.0E+00	2.2E-04
Cadmium	1.39E-04	1.72E-05			1.95E-03	2.41E-04	3.9E+00	4.8E-01
Chloroform	1.02E-05	2.65E-06	6.2E-08	1.6E-08	1.42E-04	3.71E-05	1.4E-02	3.7E-03
Chromium (III)	8.14E-04	2.85E-05			1.14E-02	3.98E-04	1.1E-02	4.0E-04
1,1-Dichloroethane	3.05E-05	3.67E-06			4.27E-04	5.14E-05	4.3E-03	5.1E-04
1,1-Dichloroethene	4.07E-05	2.65E-06	2.4E-05	1.6E-06	5.70E-04	3.71E-05	6.3E-02	4.1E-03
trans-1,2-Dichloroethylene	1.42E-04	3.16E-06			1.99E-03	4.43E-05	1.0E-01	2.2E-03
Lead	1.59E-04	1.44E-05			2.22E-03	2.02E-04	1.6E+00	1.4E-01
Mercury	3.00E-06	5.39E-07			4.20E-05	7.55E-06	1.4E-01	2.5E-02
4-Methylphenol (p-cresol)	0.00E+00	1.27E-05			0.00E+00	1.78E-04	0.0E+00	3.6E-03
Methylene Chloride	1.17E-04	6.15E-06	8.8E-07	4.6E-08	1.64E-03	8.60E-05	2.7E-02	1.4E-03
Nickel	4.24E-04	4.85E-05			5.94E-03	6.79E-04	3.0E-01	3.4E-02
Phenol	2.19E-03	1.63E-05			3.06E-02	2.29E-04	5.1E-02	3.8E-04
Toluene	2.04E-05	3.24E-06			2.85E-04	4.54E-05	1.4E-03	2.3E-04
1,1,1-Trichloroethane	1.32E-04	3.25E-06			1.85E-03	4.55E-05	2.1E-02	5.1E-04
Trichloroethene	5.60E-04	4.16E-06	6.2E-06	4.6E-08	7.84E-03	5.83E-05	0.0E+00	0.0E+00
Xylenes (total)	6.61E-05	3.13E-06			9.26E-04	4.38E-05	4.6E-04	2.2E-05
Zinc	4.29E-02	3.51E-04			6.00E-01	4.91E-03	3.0E+00	2.5E-02
	TOTAL		5.6E-04	9.8E-06			2.5E+01	4.5E+00

(file:\TBL4-29X.WR1)

(date:5/21/81)

(project:490 5054)

1. Sample calculations and exposure parameters are given in Appendix G.

2. NOTE: CDI is for ingestion only (1.4 liters/day).

3. Cancer risk from arsenic based on exposure to inorganic arsenic, which is 11% of total. Hazard quotient based on total (organic and inorganic) Arsenic.
arsenic.

IV. Fish Ingestion

A. Equation: Ingestion of Contaminated Fish

$$\text{Exposure Concentration (mg/kg/day)} = \frac{(\text{CF}) (\text{IR}) (\text{FI}) (\text{EF}) (\text{ED})}{(\text{BW}) (\text{AT})}$$

where,

- CF = Contaminant Concentration in Fish (mg/kg)
- IR = Ingestion Rate (kg/meal)
- FI = Fraction Ingested from Contaminated Source (unitless)
- EF = Exposure Frequency (meals/years)
- ED = Exposure Duration (years)
- BW = Body Weight (kg)
- AT = Averaging Time (period over which exposure is averaged -- days)

1. Example for Arsenic (non-carcinogenic; child from Table 4.27):

$$\begin{aligned} \text{Exposure Concentration (mg/kg/day)} &= \frac{(4.2 \text{ ug/l})(0.001 \text{ mg/ug})(4 \text{ l/kg})(0.038 \text{ kg/meal})(0.1)(48 \text{ meals/yr})(5 \text{ yr})}{(45 \text{ kg})(5 \text{ yr})(365 \text{ days/yr})} \\ &= 1.85\text{E-}07 \text{ mg/kg/day} \end{aligned}$$

2. Example for Arsenic (carcinogenic; adult from Table 4.27):

$$\begin{aligned} \text{Exposure Concentration (mg/kg/day)} &= \frac{(4.2 \text{ ug/l})(0.001 \text{ mg/ug})(4 \text{ l/kg})(0.038 \text{ kg/ml})(0.1)(48 \text{ meals/yr})(30 \text{ yr})}{(70 \text{ kg})(70 \text{ yr})(365 \text{ days/yr})} \\ &= 5.11\text{E-}08 \text{ mg/kg/day} \end{aligned}$$

APPENDIX G-1H

ENVIRONMENTAL SCIENCE AND ENGINEERING

To: Steve Clough
Fr: Brad Schwab
Re: Hazard Identification for metals in groundwater
Industriplex site

Date: 5/19/91
Proj: 490-5046

As you requested, I have applied the concentration-toxicity screen outlined in Section 5.9.5 of the Risk Assessment Guidance for Superfund, to determine if this approach would cause us to select more metals for the groundwater exposure route for the Baseline Health Risk Assessment of the Industriplex Site.

The approach requires that a risk factor (R_{ij}) for each chemical detected be calculated by multiplying the concentration of the chemical (C_{ij}) by a toxicity factor (T_{ij} , where T_{ij} equals the cancer slope factor or the reciprocal of the RfD). If the risk factor for a chemical represents a significant fraction of the summed risk (R_j , the sum of all R_{ij} ; in the Guidance, an example significant fraction is 1% [0.01]), then the metal should be considered as a Constituent of Concern in the risk assessment.

The Guidance indicates that it is "conservative" to use the maximum concentration in the calculation. The location of the maximum concentrations at the Industriplex site is, however, not consistent. Therefore, I used both maximum and geometric mean concentrations and made two R_{ij} -calculations.

The R_{ij}/R_j for each metal is presented in the attached table. Using a strict interpretation of the table (i.e., only looking at maximum concentrations) would cause us to add antimony, barium, manganese and zinc. We would also have to add thallium, if one considered the R_{ij}/R_j value calculated from mean concentrations. Interestingly, applying the system would also allow us to drop beryllium, cadmium, mercury, and nickel from the chemicals of potential concern we assessed previously (beryllium would not be eliminated if the R_{ij}/R_j were based on mean concentrations).

I would like to caution you that my review of the Guidance does not indicate that the system is to be used to the exclusion of other considerations. Although EPA suggested you perform this exercise, I would resist using it as a sole means for selection. The metals this method would allow us to eliminate are highly toxic and I would tend to retain them whether the system allows elimination or not. On the other hand, there is discussion in the Guidance on eliminating chemicals based on low frequency of detection (Section 5.9.3), which is an additional, not alternative, consideration in selecting chemicals of potential concern. Thus, while thallium exceeds that R_{ij}/R_j criteria of 0.01, it was only detected in 2 of 65 samples

APPENDIX G-1H

(3%), and would not seem to require further consideration. Additionally, for certain metals mean concentrations in reference wells were equal to or greater than in study area wells. Of the metals that would be selected based on an R_{ij}/R_j -value above 0.01, beryllium, manganese, and thallium have ratios of site-to-reference concentrations of 1.0, 0.6 and 1.0, respectively (I believe you were using a criteria of 2 times upgradient concentration for selection in the original selection process). You may not really be characterizing the risk of the site using these metals in the risk assessment.

In summary, using the system described, but taking other relevant factors, such as frequency of detection and local reference concentration, into account, the metals that would seem appropriate for use in the risk assessment are:

- Antimony
- Arsenic
- Barium
- Cadmium
- Chromium
- Lead
- Mercury
- Nickel
- Zinc

The system resulted in the addition of three metals not previously considered (antimony, barium, zinc), while one metal that was included in the original risk assessment was dropped (beryllium).

APPENDIX G-1H

HAZARD IDENTIFICATION OF TOTAL METALS IN GROUNDWATER
USING USEPA TOXICITY SCREENING METHOD

Groundwater/Surface Water Investigation Plan
Industri-Plex Superfund Site
Woburn, MA

Metal	Cij (ug/L)		Location of Maximum	Tij (kg-day/ug) 1/RID	Rij		Ri/Rj		RANK BY:		
	Maximum	Geometric Mean			Maximum	Geometric Mean	Maximum	Geometric Mean	MAX	GEOM. MEAN	SITE/REF RATIO
Aluminum	224000	848.9	OW-28	no data							9
Antimony	143	23.4	OW-40	3E+00	4E+02	6E+01	3E-02	3E-01	6	1	13
Arsenic	2350	16.7	OW-16	1E+00	2E+03	2E+01	2E-01	9E-02	3	4	1
Barium	505	22.8	OW-17	2E-01	1E+02	5E+00	7E-03	2E-02	7	6	3
Beryllium	8.8	0.6	OW-28	4E-03	4E-02	2E-03	3E-06	1E-05	15	15	21
Cadmium	27.4	3.2	OW-18	2E-01	5E+00	6E-01	4E-04	3E-03	14	11	14
Calcium	517000	56605.0	OW-12	no data							19
Chromium	428	11.8	OW-9	2E-01	9E+01	2E+00	6E-03	1E-02	8	8	7
Cobalt	157	7.3	OW-28	no data							15
Copper	1350	9.1	OW-10	no data							18
Iron	226000	5887.1	OW-28	no data							6
Lead	299	3.9	OW-14	7E+00	2E+03	3E+01	2E-01	1E-01	4	3	4
Magnesium	224000	11711.5	OW-16	no data							10
Manganese	28700	536.1	G123	1E-01	3E+03	5E+01	2E-01	3E-01	2	2	22
Mercury	1600	0.2	OW-40	3E+00	5E+03	5E-01	4E-01	3E-03	1	13	17
Nickel	322	13.2	OW-9	5E-02	2E+01	7E-01	1E-03	4E-03	11	10	12
Potassium	74800	4984.4	OW-16	no data							8
Selenium	78	1.4	OW-40	2E-01	2E+01	3E-01	1E-03	2E-03	12	14	16
Silver	21.2	1.6	OW-1A	3E-01	7E+00	5E-01	5E-04	3E-03	13	12	23
Sodium	757000	30528.7	OW-17	no data							11
Thallium	2.7	1.0	OW-18	1E+01	4E+01	1E+01	3E-03	8E-02	10	5	20
Vanadium	370	8.3	OW-28	1E-01	5E+01	1E+00	4E-03	6E-03	9	9	5
Zinc	9470	57.6	OW-10	5E-02	5E+02	3E+00	3E-02	2E-02	5	7	2
				Rj =	1E+04	2E+02					
Carcinogens				CPF							
Arsenic	2350	16.7	OW-16	2E-03	4E+00	3E-02	1E+00	9E-01	1	1	1
Beryllium	8.8	0.6	OW-28	4E-03	4E-02	2E-03	9E-03	7E-02	2	2	21
				Rj =	4E+00	3E-02					

(1) Shading indicates chemicals above 1% of total risk factor, mean concentrations above reference values, and a detection frequency > 5/65.

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APPENDIX G
Risk Assessment Documentation
Section 2. Toxicity Profiles

ACETONE

Acetone is a clear, flammable liquid that is used extensively as a solvent and an synthetic intermediate in the chemical and pharmaceutical industry. It is a product of the breakdown of fats and fatty acids in the body, and is a normal component in the blood and urine.

Because it is easily metabolized in the liver, acetone is fairly non-toxic to humans and laboratory animals. The NOEL (no adverse effect level) in humans exposed by inhalation is estimated at $35,000 \mu\text{g}/\text{m}^3$, and exposure at levels between 60 and $2200 \text{ mg}/\text{m}^3$ will cause strong eye and mucous membrane irritation in man (Verschueren, 1983). The estimated lethal dose by ingestion is 50 ml (about 2 ounces). Acetone is not known to cause reproductive effects in humans. It is not carcinogenic.

The USEPA recommends that the chronic oral daily intake of acetone does not exceed 0.1 mg/kg/day, based on the following study (EPA, 1986) ¹:

Acetone was given to rats at levels of 0, 100, 500 or 2500 mg/kg/day. No effects were seen at the 100 mg/kg/day dose level throughout the study. Kidney weights were increased in 500 and 2500 mg/kg groups (females only), and increased liver weights, as well increased kidney and brain weights relative to body weight were observed in the 2500 mg/kg group (both sexes). Histopathologic studies revealed tubular degeneration of the kidneys. Hyaline droplet accumulation was significant in the 500 and 2500 mg/kg males and the 2500 mg/kg females. Thus, a no observed effect level (NOEL) for acetone in this experiment was 100 mg/kg/day. EPA used a safety factor of 1000 (10 for cross species extrapolation, 10 for species variability, and 10 for extrapolation from a subchronic study to chronic exposure) to calculate a reference dose (RfD) of 0.1 mg/kg/day.

Regulatory Values

There are currently no Federal drinking water standards nor Ambient Water Quality Criteria for acetone.

¹ EPA (1986). Ninety-day gavage study in albino rats using acetone. Office of Solid Waste, Washington, D.C.

ANTIMONY

Antimony is used in paints, batteries, ceramics, semiconductors, and alloys of lead (solder) and tin (pewter). Antimony was also used medicinally as an antiparasitic compound and as an emetic (antimony potassium tartrate).

The medicinal use of antimony has given rise to a reasonably good understanding of its absorption, distribution and excretion in humans. Antimony is slowly absorbed from the gastrointestinal tract, or in some forms and doses, is emetic, precluding absorption. As with many metals, toxicity may vary greatly among different salts, due to differences in absorption. The distribution of antimony is dependent on its valence state; trivalent forms distribute to red blood cells, while pentavalent antimony is transported in plasma. Trivalent antimony is accumulated in the liver and excreted in the feces, while pentavalent antimony accumulates in both liver and spleen and tends to be excreted in the urine.

The effects of high, acute doses of antimony produce symptoms somewhat like arsenic, vomiting, diarrhea, irregular respiration, and collapse. Reduction of antimony under acidic conditions can produce stibine, a gas, which like arsine can produce hemolysis.

Chronic toxicity is known from the former use of therapeutic antimonials. The best characterized effect is heart damage, but this effect is seen with the organic antimony drugs and in industrial exposure to antimony sulfide. Cardiac effects were not seen with antimony trioxide, suggesting differential toxicity of various antimony compounds.²

Cardiac effects have also been seen in male rats given potassium antimony tartrate in drinking water at a concentration of 5,000 ppb (Schroeder, et al, 1970)³. In this study, it was also observed that exposed animals has a shorter lifespan, decreased blood glucose levels and increased cholesterol levels. This finding was similar to that reported by the same

² Stokinger, H.E. (1981) The Metals. in Patty's Industrial Hygiene and Toxicology, 3rd Edition, Volume IIA. Clayton and Clayton, editors. Wiley Interscience, New York. pages 1505-1517.

³ Schroeder, H.A., Mitchner, M., and Nasor, A.P. (1970). Zirconium, niobium, antimony, vanadium, and lead in rats: life term studies. J. Nutrition 100:59-66.

author for mice, but other authors report less toxicity in rats when antimony trioxide was used (Kanisawa and Schroeder, 1969; Sunagawa, 1981)⁴. No cancers have been reported as a result of antimony exposure.

EPA used the data of Schroeder, et al to arrive at an oral RfD for antimony. The only dose reported (5,000 ppb in drinking water was estimated to be a dose of 0.35 mg/kg-day) was assumed to be a Lowest Observed Effect Level. This dose was lowered 1000-fold (10 fold each to account for interspecies extrapolation, possible sensitive individuals in the human population, and the fact that the dose used produced an effect), to arrive at an RfD of 4E-4 mg/kg-day.

Regulatory Values

EPA proposed a health-based MCLG of 3 ppb for antimony in July 1990⁵. Interestingly, this value was based on the Schroeder, et al 1970 study cited above, but the LOAEL in this case was estimated at 0.43 mg/kg-day. A non-health based MCL of either 5 or 10 ppb was proposed in July, 1990, based on analytical detection limits for antimony.

⁴Kanisawa, M. and Schroeder, H.A. (1969) Life time studies on the effect of trace elements on spontaneous tumors in mice and rats. *Cancer Res.* 29:892-895.

⁵ Sunagawa, S (1981) Experimental Studies on antimony poisoning. *Igaki Kenkyu* 51:129-142 (Japanese). 55 FR No. 143, July 25, 1990 pages 30377-30378 and page 30411.

ARSENIC

Inorganic arsenic exists in various chemical and physical states, and the effect that this toxic metal has on biological systems is highly dependent on the chemical species present or available. In general, the trivalent form, arsenite (+3), is ten times more toxic than the pentavalent form, arsenate (+5). In addition, the metal can be reductively methylated and/or transformed from the +5 to the +3 state by biotransformation *in vivo*.

Arsenic is found naturally in certain foods (e.g. shellfish, potatoes) and some evidence suggests that it may be an essential nutrient. It is, however, a health risk to humans where increased levels are found in drinking water, from pesticide use, or improper disposal of arsenic chemicals. Smelting operations emit fairly high concentrations of arsenic and people working in these facilities or living in the adjacent areas are at high risk to the metal (ATSDR, 1989a)⁶. Symptoms of arsenic intoxication include malaise and fatigue, GI disturbances, hyperpigmentation, anemia, and peripheral neuropathy. The normal body burden for arsenic is 100 mg/70 kg. The January, 1991 HEAST reports an oral RfD of 1E-3 mg/kg/day, which is under review by the RfD/RfC Work Group.

There is evidence that chronic oral exposure to elevated levels of arsenic increase the risk of skin cancer. This is based on an epidemiological study of a Taiwanese population by Tseng et al. (1968)⁷. There was a strong correlation between the incidence of skin cancer and other signs of arsenic poisoning. The incidence was also highly dose-dependent (op. cit.). There are limitations to this study including the possibility that the population was exposed to other chemicals and life-style factors that may be different between the studied population and U.S. residents. Studies of U.S. populations exposed to somewhat lower doses of arsenic in water were negative, although the statistical power to differentiate the studies is not strong.

Studies on chemical carcinogenesis suggest that, for some compounds, no threshold for the effect exists. That is, certain carcinogens, even in extremely small doses will pose some risk

⁶ ATSDR (Agency for Toxic Substances and Disease Registry) (1989) Toxicological Profile for Arsenic. ATSDR/TP-88/02.

⁷ Tseng, W.P., et al (1968) Prevalence of skin cancer in an endemic area of chronic arsenicalism in Taiwan. J. Natl. Cancer Inst. 40:453-463.

of cancer. This assumption is incorporated into the cancer dose-response assessment for arsenic. The EPA Carcinogen Assessment Group has applied a "multi-stage" model of carcinogenesis to the epidemiologic data of Tseng et al. (1968) to derive a dose-response slope function, or "potency factor" for arsenic. Multiplying the predicted intake of arsenic by the potency slope gives an estimate of the excess risk of contracting cancer due to exposure at the site. The "potency factor" used for arsenic in evaluating the carcinogenic risk is 2 mg/kg/day^{-1} .

Regulatory Values

The EPA Office of Drinking Water has set an MCL for Arsenic of 50 ug/l. The Ambient Water Quality Criteria for protection of freshwater species is 100 ug/l.

BARIUM

The mineral barium (as ground barite, which is the natural form of barium sulfate) is used in the manufacture of glass, paints, and rubber. Barium is also used in aluminum-barium alloys and in the production of ceramics. Barium sulfate is radiopaque, and is used as an X-ray dye in medial diagnosis of gastrointestinal tract disorders.

The absorption of barium is dependent on the water solubility of its salts. Water soluble salts are absorbed from the gastrointestinal tract whereas insoluble salts are not. Insoluble salts, such as barium sulfate are practically non-toxic because of their minimal absorption, while soluble salts such as barium chloride are quite toxic. Therefore, it is important to understand the particular form of barium present to fully characterize its toxicity.

Barium that is absorbed is excreted by the kidneys; nonabsorbed barium salts are eliminated in feces. Small amounts of barium are retained in the body.

Acute toxicity in humans has been reported as a result of accidental ingestion of soluble barium salts, which caused gastroenteritis, slowed pulse rate, and muscular paralysis. In animal studies various effects on muscle function have been observed. In the industrial setting, it has been found that the carbonate and oxide salts of barium are irritant to the lungs and nasal passages as well as the skin⁸.

Chronic inhalation of barium in industrial settings causes baritosis, which includes changes notable on chest X-ray, but has no apparent effect on pulmonary function. IRIS reports that baritosis produces significant incidence of hypertension, although no citation of a report on this effect is provided.

Studies of the effects of long term administration of barium in water have been largely

⁸Stokinger, H.E. (1981). The metals., in Patty's Industrial Hygiene and Toxicology, 3rd Edition. Volume IIA. Clayton and Clayton, editors. Wiley Interscience, New york pages 1531-1537.

negative, although Perry, et al (1983)⁹ observed increased blood pressure in rats given 10,000 ppb barium in drinking water for 16 months (other negative toxicity studies did not test for blood pressure changes). However, Wones, et al (1990) and Brenniman and Levy (1983)¹⁰ did not find changes in blood pressure in human volunteers and epidemiologic data, respectively. Brenniman and Levy's study did find an increased death from cardiovascular disease in a population drinking water containing 7300 ppb barium when compared to a group with little barium in its drinking water, but the deaths were in elder members of the population and it was not ascertained if these individuals had been exposed to the water for long periods or recently moved to the area. Additionally, the high metal content of the water required the use of softeners, and how these chemical affected health was not ascertained.

In determining the RfD, EPA used the No Adverse Effect Level from the Wones study (no effect were seen in the highest dose tested; 10,000 ppb barium in 1.5 L water per day, or 0.21 mg/kg-day in a 70 kg adult), which was similar to the dose estimated for the exposed population in the Brenniman and Levy study (7300 ppb in 2 L per day in a 70 kg adult). As the highest dose produced no effect, was conducted in humans, and was consistent with findings of a large epidemiologic study, an uncertainty factor of only 3 was applied to the data, to arrive at an RfD of 0.07 mg/kg/day. The January, 1991 HEAST updates this RfD to 5E-2 mg/kg/day; however, IRIS has not yet changed.

Regulatory Levels

The MCL for barium is 1000 ppb, based on protection against circulatory effects in exposed populations.

⁹ Perry, H.M., Kopp, S.J., Erlanger, M.W., and Perry, E.F. (1983) Cardiovascular effects of chronic barium ingestion. In Trace Substances in Environmental Health, XVII. D.D. Hemphill, editor. University of Missouri Press, Columbia, MO.

¹⁰Wones, R.G., Stadler, B.L., and Frohman, L.A. (1990) Lack of effect of drinking water barium on cardiovascular risk factors. Environ. Health Perspectives 85:1-13.

Brenniman, G.R. and Levy, P.S. (1984) High barium levels in public drinking water and its association with high blood pressure. Advances in Modern Toxicology, Volume IX. E.J. Calabrese, editor. Princeton Scientific Publications, Princeton, N.J. pages 231-249.

BENZENE

Benzene is a volatile, colorless, liquid hydrocarbon that is produced from coal and petroleum distillation. It is a hexagonal (six-sided) molecule that is lighter than, and soluble in water. It is a widely used solvent, even more so in the past, before its latent toxic effects were realized. Its uses include degreasing and cleaning, a solvent in the rubber industry, a synthetic intermediate in the chemical and pharmaceutical industry, preparation and use of graphic inks, and an anti-knock fuel additive (largely replacing tetraethyllead in gasoline).

The physical properties of benzene are such that, if a sufficient amount of benzene were spilled to overcome the adsorption capacity of a soil column, the free product would tend to float on the water table. Transport of benzene dissolved in water will be retarded by interaction with immobile matrix (soil), but not to the same extent as more complex organic structures. The most prominent feature of benzene is its volatility, the chemical readily vaporizes from a pool (its vapor pressure is 95 mm Hg at 25°C, as compared to 23.8 mm Hg for water; the higher the vapor pressure value, the greater likelihood of volatilization). Benzene will also volatilize readily from water, as indicated by its Henry's law coefficient.

Yearly production of benzene today is about 4 million metric tons. Although benzene has been detected in U.S. water supplies, its concentration is very low, generally <0.1 parts per billion. The majority of the human population is exposed to benzene through inhalation, and the primary source is automobile and gasoline fumes. Gasoline contains an average of 0.8 percent benzene, and automobile exhaust averages 4 percent benzene. It is estimated that about 940 million pounds of benzene are released to the environment by this route, roughly one half of the total amount of benzene released to the environment. Concentrations of air around gas stations have been found to be 0.3 to 2.4 parts per million. The average ambient (rural) concentration is about 0.017 ppb (EPA, 1980)¹¹.

Benzene is readily absorbed via inhalation or ingestion, although its presence on particles (soils or dusts) slows the process. Dermal absorption also occurs, but at a reduced rate, relative to inhalation or ingestion. Of course, because benzene is so volatile, a competing process to dermal absorption is loss to the air.

¹¹ EPA (1980) Ambient Water Quality Criteria for Benzene. EPA 440/5-80-024.

Once absorbed, benzene will preferentially distribute to fatty tissue. The fat and bone marrow will contain the highest concentrations of benzene, with lesser amounts in the blood, liver, and kidney. Benzene is transformed in the liver to more water soluble forms, and is then excreted in the urine. Another mechanism of loss is exhalation of unchanged benzene. The chemical changes (metabolism) to benzene in the liver appear to be necessary for toxicity to this organ. However, benzene is also toxic to blood cells and metabolism in bone marrow is limited.

Acute exposures to benzene will cause typical solvent effects (staggering gait, loss of consciousness, central nervous system depression).

Bone marrow, which is responsible for the production of blood cells, is affected by chronic benzene exposure. Benzene is reported to cause pancytopenia (a decrease in number of all types of blood cells) or aplastic anemia (absence of blood cell formation) at doses previously found in some workplaces. Many studies have examined the effects of benzene on workers in industry (e.g. rubber manufacturing), and it is clear that chronic exposure to air concentrations greater than 100 ppm will cause various blood disorders. Symptoms include lassitude, dizziness, headache, heart palpitation, and shortness of breath. Cases of aplastic anemia often reverse once exposure stops, but may be lethal.

Benzene has been shown to be both mutagenic and carcinogenic in laboratory animals. The evidence that benzene causes leukemia in man comes from epidemiologic studies of Turkish shoe workers (Aksoy et al. 1974)¹² and American workers in the rubber or chemical industries (Infante, 1977; Rinsky/y et al. 1981, 1987; Ott et al, 1978; Wong, et al 1983)¹³.

¹² Aksoy, M., et al (1974) Leukemia in shoe-workers exposed chronically to benzene. *Blood* 44:837.

¹³ Infante, P.F., R.A. Rinsky, J.K. Waggoner, and R.J. Young (1977) Leukemia in benzene workers. *Lancet* 2:76-78.
Rinsky, R.A., R.J. Young and A.B. Smith (1981) Leukemia in benzene workers. *Am. J. Ind. Med.* 2:217-245.
Rinsky, R.A., et al (1987) Benzene and leukemia: An epidemiological risk assessment *N. Eng. J. Med.* 316:1044-1050.
Ott, M.G., J.C. Townsend, W.A. Fishbeck and R.A. Langner (1978) Mortality among individuals occupationally exposed to benzene. *Arch. Environ. Health* 33:3-10.
Wong, O., R.W. Morgan and M.D. Whorton (1983) Comments on the NIOSH study of leukemia in benzene workers. Technical report submitted to Gulf Canada, Ltd., by Environmental Health Associates.

The increases in leukemias were statistically significant and dose-related in at least some of the studies, although there is some disagreement as to the dose levels among effected workers (Wong et al., 1983). Based on these findings, EPA has classified benzene as an "A" carcinogen.

The toxic effect considered in risk assessment of benzene is the potential carcinogenicity of the compound. EPA takes the position that chemical carcinogens have no threshold for action; that is, that any dose will be associated with some risk of cancer induction. This may or may not be true, depending on the mechanism of carcinogenesis of benzene, which is unknown, but it is a conservative approach. Most of the models used for fitting dose-response curves to data for carcinogenesis are also linear at low doses.

The EPA risk estimate for benzene is a mean value taken from the application of several dose-risk models to data from Rinsky et al. (1981) and Ott et al. (1978) as well as the data of Wong et al. (1983), following adjustments. The models applied to the data include both relative and absolute risk models (a relative risk model assumes the chemical causes a proportional increase in the "background" cancer rate, whereas an absolute model assumes cancer induction by the chemical is independent and adds to the background cancer incidence). Doses were calculated based on cumulative exposure as well as cumulative exposure weighed for time of exposure. Although these reports indicate the route of exposure was most likely by inhalation, a route-to-route extrapolation was made to derive an oral cancer potency factor that is identical to that for inhalation. The values are $0.029 \text{ [mg/kg/day]}^{-1}$.

Regulatory Values

The EPA Office of Drinking Water has set an MCL for benzene of 5 ug/l and an MCLG of zero. The MCL is not health based.

BENZOIC ACID

Benzoic acid is used as a food preservative, and can be esterified with certain simple alcohols to produce fragrances, flavoring agents, and solvents.

Benzoic acid does not cross the skin well but may be absorbed from the gastrointestinal tract. The benzoic acid esters are liquids that can cause dermal irritation in undiluted form.

Studies of the effects of benzoic acid have produced minimal adverse effects; however, IRIS indicates that studies in laboratory animals are not appropriate for predicting the toxicity of the compound to man. No further elaboration is available.

Benzoic acid have been used as a food additive for a period long enough that the Food and Drug Administration (FDA) has had an opportunity to consider the effects (or lack of effect) of ingesting the material. Benzoic acid is among the additives rates as "GRAS" (Generally Recognized As Safe) by FDA. EPA has therefore used the estimated per capita intake of benzoic acid in food as a No Observed Adverse Effect Level, and applied no uncertainty factors in deriving an RfD. The estimated intake of benzoic acid is 312 mg/day, which would convert to approximately 4 mg/kg-day in an adult. Thus, the RfD is 4 mg/kg-day.

Regulatory Levels

There is no MCL or MCLG for benzoic acid.

BERYLLIUM

Beryllium is a hard, grayish metal that is lighter but stronger than aluminum. Because it is the lightest structural metal, it is used in space and nuclear technology. It also finds extensive use in the manufacturing of electronic components, chemicals, ceramics, and X-ray tubes. It is added to many alloys to increase strength, corrosion-resistance, and fatigue. The major source of beryllium in the environment is the combustion of fossil fuels. Emissions from these sources accounts for 99% of all U.S. emissions.

Beryllium is present in very low concentrations in the food and the air. The typical American takes in approximately 0.4 ug/day, mostly from food and water. Variations in this number can be expected depending on the types of food and beverages ingested, and the background concentrations of beryllium in the air. The background concentrations of beryllium in air and water are generally less than .0001 ug/m³ and 1 ug/l, respectively. Soil concentrations average from 2.8 to 5.0 µg/g.

The populations at highest risk to beryllium exposure are people who work in and/or live near beryllium industries. Since tobacco also contains beryllium, smokers are also at a relatively high risk to exposure. Additionally, it is believed that a very small segment of the population may be unusually sensitive to the metal.

Beryllium can exist as different compounds, and all are poorly absorbed through the gastrointestinal tract and the skin. The lung appears to be the organ most affected by exposure to the metal, and what we know concerning human toxicity has been gathered in the workplace. In both humans and laboratory animals, acute exposure to beryllium in the air will result in pneumonitis. Chronic, low-level exposure may lead to "berylliosis", in which non-malignant growths (granulomas) appear in the lung. Chronic disease differs from the acute form in that it is progressive and prolonged in spite of cessation of exposure, and its occurrence is often separated from the time of exposure by periods ranging up to several years.

The form of the inhaled beryllium compound will affect its toxicity. In general, most of the soluble salts are more toxic than the insoluble forms, even though they are cleared from the lungs more rapidly. The insoluble forms, such as beryllium oxides, may remain in the lungs

metabolism.

The most common reported effect of high-level exposure to zinc is copper deficiency, which is readily reversible. The effect occurs at exposure levels at least an order of magnitude above the RDA for zinc. Long-term oral administration of zinc sulphate in daily doses of 135 to 150 mg of zinc has been well tolerated by patients given the compound to promote wound healing. In patients with metabolic diseases such treatment might cause reduction in serum copper levels. Using a safety factor as high as 10, this means that additional intake of 15 mg of zinc does not constitute any health hazard. This corresponds to an intake of 2 liters of water containing 7.5 mg Zn/l. This concentration is above the present standard for drinking water which is 5 mg/l based on organoleptic effects.

The literature on such adverse health effects is limited. One possible reason for the limited information is that zinc has generally been accepted as a beneficial substance and adverse effects have neither been expected nor looked for. Effects on the lungs and systemic effects after inhalation of zinc compounds have only been reported from occupation settings.

Storing liquids and foodstuff in galvanized containers:

Ingesting liquid with high concentrations of zinc could result in symptoms such as nausea, vomiting, and fever.

Ingesting foodstuff with high concentrations of zinc could result in symptoms such as severe diarrhea with abdominal cramping.

The National Institute of Occupational Safety and Health (NIOSH, 1975) has recently reviewed the occupational hazards of exposure to zinc oxide and no changes were suggested regarding the existing standard for zinc oxide of 5 mg/m³. The American Conference of Government Industrial Hygienists (ACGIH, 1976) has an adopted threshold limit value (TLV) for zinc oxide of 5 mg/m³ and the Occupational Safety and Health Administration (OSHA) (29 FR 1910.1000) has a workplace standard for zinc oxide of 5 mg/ m³, 8-hour time-weighted average. The TLV value has also been adopted in other countries. For zinc chloride a limit of 1 mg/m³ has been adopted by ACGIH and OSHA.

There is not acceptable daily intake for zinc in food, zinc is an essential nutrient and there has been no reason to restrict the zinc levels in food.

Regulatory Levels

The MCL and MCLG for Zinc zero. There are no drinking water standards for zinc.

500 ppm. Rat pups born to dams exposed to 500 ppm or 250 ppm displayed reduced weight of ovaries, but the effect was transient. Developmental toxicity was seen in another inhalation study in rats.

The no effect level from the NTP study has been used to calculate an oral RfD for xylene. The highest level where no effects were seen was 250 mg/kg/day (this dose was converted to 179 mg/kg/day to account for the fact that the animals were only dosed 5 days per week). This dose was lowered by a factor of 100: a factor of 10 to account for uncertainty associated with extrapolating from animals to man, 10 to account for potentially sensitive people in the general population. The resulting RfD is 1.79 mg/kg/day, rounded to 2 mg/kg/day.

Regulatory Levels

The proposed MCL and MCLG for mixed xylenes is 10,000 ug/l.

ZINC

Zinc is a bluish-white metal which dissolves readily in strong acids. Its principal uses include electroplating and the production of alloys. Zinc is never found free in nature, but occurs as the sulfide, oxide, or carbonate (Lange, 1956). It is generally found in low concentrations (well below the current drinking water standard of 5 mg/l) in natural waters and drinking waters, however, concentrations may increase due to pollution of water systems or release of zinc from distribution systems and household plumbing, respectively.

The major source of zinc for the general population in the United States (U.S) is in the food we eat. Zinc is also found as a common trace constituent of natural waters and is a required trace element in the metabolism of most organisms. The uptake of zinc from the environment, either via ingestion or absorption, must exceed some minimum rate in order for an organism to function properly. In the "recommended dietary allowances" the National Research Council [National Academy of Sciences (NAS), 1974] based on the body weight the requirements for zinc would be about 0.5 mg/kg for the infant and 0.2 mg/kg in the adult. The average intake is generally above 10 mg in adults.

Zinc which is an essential trace element, is not carcinogenic but can be toxic at higher concentrations. An oral RfD of 0.2 mg/kg/day has been established for zinc.

It has been well established in several studies that the present intake of zinc via food for the adult U.S. population is from 10 to 20 mg/day. For the majority of the population, the intake of zinc via drinking water will be only a few percent of the intake via food, but some individuals the zinc concentration in tap water may cause an additional daily intake of 2 to 10 mg of zinc. The average exposure to zinc via ambient air will, even in the vicinity of zinc emitting industries, be in the order of only a few tenths of a milligram. Smoking will contribute even less.

Zinc may interfere with certain metals such as copper, cadmium, calcium, iron, and lead. Studies on experimental animals and on human beings given zinc for therapeutic purposes, together with observations of occupationally exposed persons, indicate that large doses of zinc can be tolerate for long periods if the copper status is adequate. Certain drugs such as contraceptives and drugs with chelating properties may have an influence on zinc

XYLENE

Xylene has an aromatic ring structure, with two methyl substitutions. There are three isomers of xylene; o-, m-, and p-xylene. Most commercially available xylene and the xylene component of gasoline is a mixture of the isomers. Xylenes are a slightly volatile liquid that is somewhat soluble in water. Xylenes are used as paint thinners, solvents, in glues, and as intermediates in the production of plasticizers, resins, and polyester fibers.

Xylenes are lighter than water and only slightly soluble. Transport of solubilized xylenes in groundwater will be retarded to a greater extent than benzene, a chemical often found as a co-contaminant with xylene. Xylenes are not as volatile as benzene, but will tend to vaporize from water to approximately the same extent as benzene, due to its lower water solubility.

Xylene has been detected in 3% and 6% of groundwater and surface water supplies sampled in a survey sponsored by the EPA. Xylene is a typical groundwater contaminant where gasoline releases have occurred.

The free absorption of xylene from the gastrointestinal tract has been inferred from the observation that toxic effects are produced on oral dosing with the compound. Absorption from the lung at high air concentrations (100 -1300 mg/M³) has been estimated to be about 60% of the administered dose in human volunteers (Astrand, et al, 1978)⁶⁴. Absorption through the skin of the hands has been demonstrated in humans, and estimated to occur at a rate of 2 ug/cm²/minute (Engstrom, et al 1977, as cited in Sandemeyer, 1982)⁶⁵. Xylene will also cross the placenta and enter fetal tissue.

Once absorbed xylene distributes to many tissues in the body, although this occurs to a lesser extent than benzene. Chemical alteration of xylene occurs in the liver and the lung, where the compound is changed to more water soluble metabolites, and excreted in the urine. In animals it has been shown that metabolism is qualitatively different in the lung

⁶⁴ Astrand, I., Engstrom, and Ovrum, P. (1978) Exposure to xylene and ethylbenzene I. Uptake, distribution, and elimination in man. *Scand J. Work Environ. Health* 4: 185-194.

⁶⁵ Sandemeyer, E.E. (1982) Aromatic hydrocarbons in - Patty's Industrial Hygiene and Toxicology - Clayton and Clayton, eds. Wiley Interscience. pages 3291-3303.

versus liver. Greater than 95% of absorbed xylene is excreted as a water-soluble metabolite, the remaining fraction being exhaled unchanged. Excretion appears to occur rapidly; animal studies indicate complete clearance of the compound in 24 hours.

Xylene is irritant to the eyes and gastrointestinal tract. appears to have low acute toxic potency. At high air concentrations xylene may be lethal and death appears to be due to lung congestion and hemorrhage. This is a similar finding to a close structural analog of xylene, ethylbenzene. Xylene appears to be more acutely toxic than other structural analogs, toluene and xylene. Central nervous system depression, a typical effect seen in solvent exposures, has been seen with xylene. Females are reported to be more susceptible to the effects of xylene than males (Sandemeyer, 1982).

Chronic exposure to xylene has been associated with lung and eye irritation, central nervous system depression (this may occur to a minor extent even at the occupational exposure limit of 100 ppm), bleeding of mucous membranes, anemia, mild liver enlargement, and kidney effects. There a suggestion of an association of teratogenic effects with xylene exposure, based on an epidemiologic study in Czechoslovakian women.

In animals, the National Toxicology Program (NTP, 1986)⁶⁶ conducted a study of rats and mice, in which mixed xylenes were given orally. The only effect seen was decreased body weight, in both sexes. This occurred at doses of 1000 mg/kg/day, 5 days a week for 13 weeks. No effects were seen at the next lowest dose, 500 mg/kg/day. No carcinogenic effect was apparent. EPA has interpreted the weight of evidence for carcinogenicity of xylene as D.

In reproductive studies, effects on the fetus have been seen at oral doses higher than the no effect level discussed above, and were usually associated with concurrent maternal toxicity. In an inhalation study (Biodynamics, 1982)⁶⁷ exposed pregnant female rats to mixed xylenes 6 hours per day for 190 days. Toxicity to the fetus was apparent in the group exposed to

⁶⁶ NTP (1986) NTP technical report of the toxicology and carcinogenesis of xylenes (mixed) (60.2% m-xylene, 13.6% p-xylene, 17.0% ethylbenzene and 9.1% o-xylene) in F344/N rats and B6C3F mice (gavage studies).

⁶⁷ Biodynamics (1982) Parental and fetal reproduction inhalation toxicity study in rats with mixed xylenes. FYI submission from American Petroleum Institute to EPA.

TRICHLOROETHENE

Trichloroethene (TCE) is a highly volatile industrial solvent that is used primarily in the degreasing of metals.

The U.S. production of the compound is estimated at 130,000 metric tons per year. Of all the TCE used in the U.S., about 90 percent is eventually released to the atmosphere. It has been detected in the ambient air and water, and a concentration of about 1 ppb would be expected in an urban environment (concentrations as high as 47 ppb have been measured in urban Tokyo). Like the chloroethanes, TCE has been detected in finished drinking water (up to 32 ppb). In natural waters, TCE has a very short residence time, and little tendency to bioaccumulate in fish (EPA, 1980)⁵⁸.

Inhalation is the primary route of exposure for humans. Ingestion of TCE is also of concern. Once TCE is ingested, almost all of it is absorbed by the intestine. It is eliminated from the body primarily by the lungs and, after conversion to a water soluble metabolite by the liver, the urine. At air concentrations of 500 ppm, humans are estimated to metabolize 60 - 90 percent of the absorbed TCE.

Acute exposure to high levels of trichloroethene in air causes dizziness and loss of consciousness; an anesthetic effect shared by many volatile organic compounds. Concentrations as high as 200 ppm are needed to observe an effect on the central nervous system in humans. Evidence from acute animal and human exposures suggests that the toxicity of TCE is low and does not present a significant health threat. There is also no evidence to suggest that chronic, low level exposures produce adverse effects in humans. Reproductive and developmental studies in which animals were exposed to relatively high concentrations of TCE in the air were negative.

Studies investigating the mutagenic potential of TCE showed a very weak response. A recent update of the experimental evidence implicating TCE as a carcinogen indicates that TCE is carcinogenic in several species of laboratory animals (EPA, 1987)⁵⁹. However,

⁵⁸ EPA (1980) Ambient Water Quality Criteria Document for Trichloroethylene.

⁵⁹ EPA (1987) Addendum to the Health Assessment Document for Trichloroethylene: Updated Carcinogenicity Assessment for Trichloroethylene. EPA/600/8-82/006FA.

studies investigating whether it is a causative factor in human carcinogenesis have uncovered no conclusive evidence to date.

Although HEAST reports the weight of evidence for TCE to be B-2, in fact, the Science Advisory Board to the EPA rates the compound as somewhere in the "continuum" between B-2 and C.

EPA (1987)⁶⁰ developed potency factors for TCE from the mouse inhalation data of Maltoni (1986)⁶¹, and Fukuda (1983)⁶²; and the stomach tube exposures in mice in studies by NTP (1982, 1986)⁶³. The potency factors for inhalation and ingestion are 0.017 and 0.011 [mg/kg[day]⁻¹], respectively. The inhalation potency factor is based on delivered dose and probably should not be used with administered dose exposure estimates.

Regulatory Levels

The MCL for TCE is 5 ppb. It is not health-based. The MCLG is zero.

⁶⁰ EPA (Environmental Protection Agency). 1987. Addendum to the Health Assessment Document for Trichloroethylene: Update Carcinogenicity Assessment for Trichloroethylene. Review draft. June 1987. EPA/600/8-82/006FA.

⁶¹ Maltoni C, Lefimine G, Cotti G. 1986. Experimental Research on Trichloroethylene Carcinogenesis. Archives of Industrial Carcinogenesis Series. Maltoni C, Mehlman MA, eds., Vol. V. Princeton, N.J.: Princeton Scientific Publishing Co., p. 393.

⁶² Fukuda K., Takemoto K., Tsuruta H. 1983. Inhalation carcinogenicity of trichloroethylene in mice and rats. Ind. Health 21: 243-254.

⁶³ NTP (National Toxicology Program). 1986a. Toxicology and Carcinogenesis Studies of Trichloroethylene in F344/N Rats and B6C3F1 Mice. NTP TR 243. Department of Health and Human Services, National Institutes of Health, Bethesda, Md.

1,1,1-TRICHLOROETHANE

1,1,1-Trichloroethane (TCA) is a volatile chlorinated hydrocarbon that is used widely as an industrial solvent and in consumer products, such as spot removers. Production increased 3-fold between 1970 and 1980, and 88 percent of TCA consumed is eventually released to the atmosphere. Background contamination levels for air and water are around 20 ppb (0.11 mg/m³) and <1ppb, respectively (EPA, 1982C). In the atmosphere, TCA is persistent and is considered to contribute to the destruction of the ozone layer.

Because it is so volatile, the risk to TCA exposure is greatest by inhalation. The amount absorbed is related to the concentration in the air and the degree of physical activity the person may perform. Like other chloroethanes, TCA is lipophilic ("fat-loving") and will prefer to concentrate into fatty tissue once inside the body. It will therefore pass the blood-brain and placental barrier, and will concentrate into mother's milk. However, TCA is rapidly excreted via the lungs once exposure is stopped. Also, available studies have shown that the likelihood of adverse health effects in humans exposed to TCA are "extremely low". It is estimated that a NOEL (no observed effect level) for short-term (acute) exposure of humans is in the range of 350-500 ppm (1890 to 2700 mg/m³). This level is many times higher than the background concentrations cited above. The main health affects are seen at levels >1000ppm, where one would experience lightheadedness and irritation of the throat.

There is no evidence to implicate TCA as a reproductive, developmental, or carcinogenic threat to the general population. For non-carcinogenic effects, the EPA recommends that a person ingest no more than 0.09 mg/kg/day, nor inhale more than 0.3 mg/kg/day. The oral RfD is based on the following calculations:

Torkelson et al. (1958)⁵⁶ exposed groups of rats, rabbits, guinea pigs and monkeys to 1,1,1-trichloroethane vapor at concentrations of 500, 1000, 2000, or 10,000 ppm. The guinea pig was found to be the most sensitive species. Increase liver weight and histopathologic changes of the liver were found in guinea pigs at the 1000 ppm, when exposure was 3 hours per day, 5 days per week for 3 months. 500 ppm for 7 hours per day, 5 days per week for

⁵⁶ 1 Torkelson, T.R., F. Oyen, D.D. McCollister and V.K. Rowe (1958) Toxicity of 1,1,1-trichloroethane as determined on laboratory animals and human subjects. Am. Ind. Hyg. Assoc. J. 19: 353-362.

6 months was without effect. This value is consistent with the findings of Adams et al. (1950)⁵⁷. Thus, 500 ppm (equivalent to 90 mg/kg/day) was defined as the No Observed Effect Level. This value was divided by 1000 to arrive at a RfD of 0.09 mg/kg/day.

Although the use of this RfD would lead to an MCL of approximately 600 ug/l, based on the usual calculations, the actual MCL and MCLG for 1,1,1-trichloroethane is 200 ug/l, based on findings of an inhalation study in mice.

Regulatory Levels

The MCL and MCLG for TCA is 200 ppb.

⁵⁷ Adams, E.M., H.C. Spencer, V.K. Rowe and D.D. Irish (1950) Vapor toxicity of 1,1,1-trichloroethane (methyl chloroform) determined by experiments on laboratory animals. Arch. Ind. Hyg. Occup. Med. 1: 225-236.

and body tremors. Some animals died at this dose. There were changes in organ weights and microscopic pathologic changes of several organs at 1250 mg/kg in rats. Organ weight changes, but not pathologic changes were seen at 2500 mg/kg in mice, but not at lower doses. The effect seen at the lowest dose was increased liver and kidney weights at 625 mg/kg in rats.

NTP also conducted a 2 year inhalation study in mice and rats (doses of 600 or 1200 ppm in rats and 120, 600, and 1200 ppm in mice, 6.5 hours per day, 5 days per week). Lesions of the nasal cavity (in rats) and overgrowth (hyperplasia) of the bronchial lining (in mice) was seen, but no deaths nor significant body weight change was observed during the course of the study. There was no evidence of cancer induction in this study.

Although there was no evidence of cancer in the CIIT or NTP studies, and most mutagenicity tests have been negative, EPA considers the data inadequate to classify toluene relative to its carcinogenicity; it is rated D in the weight of evidence system.

Although HEAST indicates that the RfD for toluene is based in the findings of the CIIT study, a more recent IRIS file indicates that the oral RfD is based on the NTP (1989) study. EPA used the findings in rats, as effects were seen at lower doses than in mice. The 312 mg/kg dose (223 mg/kg/day, based on a 7 rather than 5 day exposure) was considered a no adverse effect level. This is conservative, because the finding at the next highest dose was increased liver and kidney weight, rather than overt dysfunction of any organ system.

The no effect dose was decreased by 1000 to account for uncertainty associated with extrapolating from animals to man (10), to account for particularly sensitive individuals (10), and to extrapolate from a short duration study to chronic exposure, as well as consideration of the limited data on reproductive and developmental effects of toluene (10) to arrive at an RfD of 0.2 mg/kg/day.

There is no published information on the development of the inhalation RfD, reported by HEAST to be 2.0 mg/kg/day.

Regulatory Values

The Maximum Contaminant Level and Maximum Contaminant Level Goal for toluene is 1000 ppb.

TOLUENE

Toluene is a derivative of benzene, substituted with one methyl group. It is a clear, flammable liquid, with a sweet odor that is slightly soluble in water. It is derived mainly from petroleum refining, and only a small percentage of that produced is used directly, with the remainder being reintroduced into gasoline mixtures (benzene-xylene-toluene) to increase octane ratings. Toluene is an excellent solvent and is used extensively in the chemical and pharmaceutical industry, and in glue production (toluene is responsible for the narcosis seen after "glue sniffing").

Automobile emissions contribute the majority of toluene to the atmosphere. Toluene is the most prevalent aromatic hydrocarbon in the atmosphere, with levels ranging from 0.14 to 59 ppb. Toluene has also been detected in surface water and treated wastewater effluents at levels generally below 10 $\mu\text{g/l}$. Toluene is readily biodegradable and will not bioconcentrate to a great degree. In a study of edible aquatic organisms, 95% of the tissues sampled had levels less than 1 ppm.

Toluene is readily absorbed from the lung and gastrointestinal tract, although studies in animals suggest absorption occurs more slowly in the gastrointestinal tract. Slow absorption also occurs through skin. Studies of humans and animals indicate that inhaled toluene distributes to tissues that are high in fat content (e.g., body fat stores, brain) or well supplied with blood (e.g., liver). It seems reasonable that similar distribution would occur for other routes of exposure.

Toluene is converted in the liver to metabolites that are water soluble (hippuric acid, conjugated cresols) and can be excreted in the urine. This conversion has been demonstrated in man and animals exposed via inhalation, although it is expected to occur for other exposure routes as well. Another excretion route for toluene is exhalation of the unchanged chemical. This excretion route might be expected to operate for all exposure routes, but be more effective for exposures via inhalation.

Much of the information on toxicity of toluene to humans comes from studies of solvent abuse (such as glue sniffing) and in occupational exposure (e.g. painters, printers). Interpretation of the data can be difficult due to the fact that these individuals are exposed

to complex mixtures of chemicals. Blood abnormalities, psychomotor disorders, changes in the lens of the eye, immune system changes, kidney effects, menstrual disorders, and birth defects have been observed in some, but not all, studies of workers or abusers, and the possible confounding effect of mixed chemical exposure is mentioned in most. Liver effects, which figure prominently in the animal studies discussed below, have not been observed in occupationally exposed individuals.

Toxicity to the embryo or fetus, and teratogenic effects have been observed rarely in animal studies. These effects were seen in only one experiment where the dose was not high enough to be toxic to the mother as well. More frequently, when maternal toxicity was not present, fetal toxicity or tetragenicity was not found. Growth inhibition of rat pups born during inhalation exposure to toluene through two generations has been observed.

The USEPA recommends that the chronic oral daily intake of toluene does not exceed 0.3 mg/kg-day, or the inhalation exposure not exceed 2.0 mg/kg-day. No information is available of the inhalation RfD, but the oral RfD was calculated in the following manner:

The Chemical Industry Institute of Toxicology (CIIT, 1980)⁵⁴ conducted a 2 year inhalation study of rats exposed to toluene. The concentrations were 30, 100, or 300 ppm (113, 377, or 1130 mg/cu.m) 6 hours per day, 5 days per week. The only finding was a dose-related reduction in hematocrit values (number or volume of red blood cells) in female rats exposed to 100 and 300 ppm toluene. This may not be considered a significant toxic effect. Therefore, a No Observed Adverse Effect Level was set at the highest exposure level, 300 ppm (equivalent to 29 mg/kg/day). This value was divided by a 100-fold safety factor to arrive at an oral RfD of 0.3 mg/kg/day, rounded.

(NTP, 1989)⁵⁵ Rats and mice were given toluene orally at one of five doses (312 to 5000 mg/kg) five days per week for 13 weeks. General toxic effects were seen in both species at 2500 mg/kg, which included decreased movement or prostration, tearing and salivation,

⁵⁴ CIIT (1980) A 24-month inhalation toxicology study in Fischer-344 rats exposed to atmospheric toluene. CIIT, Research Triangle Park, NC.

⁵⁵ NTP (National Toxicology Program). 1989. Toxicology and Carcinogenesis Studies of toluene in F344/N rats and B6C3F1 mice. Technical Report Series No. 371. Research Triangle Park, NC.

the more potent tumor inducers of the PAH's present. Since then, many more studies have confirmed that BaP is a carcinogen.

Very little data exists on the non-carcinogenic effects of BaP. Death in laboratory animals that have received acute doses of BaP appears to be from bone marrow depression leading to hemorrhage and infection. Adverse reproductive/developmental effects have been reported in rodents exposed to BaP, but not in humans.

Only a subset of the general chemical category of PAH have the potential to cause cancer, and no PAH has been unequivocally demonstrated to cause cancer in humans. However, by extrapolation from individuals who smoke or have been exposed to high levels of PAH mixtures, and from animal data on certain PAH compounds, there is reason to believe that some PAH are likely to be carcinogenic in humans. The PAH observed at Industriplex rated as probable (B-2) human carcinogens are B[a]P, benzo[a]anthracene, benzo[b and k]-fluroanthene, chrysene, and indeno(1,2,3,cd) pyrene. Anthracene, fluroanthene, and pyrene are not carcinogenic.

The EPA is currently reassessing approaches to estimating the cancer potency of the PAH expected to cause tumors. A previous risk assessment strategy for assessing the carcinogenic potential of PAH mixtures was to set the potency of each compound equal to that of BaP, calculate the risk based on the amount of each individual compound, and treat the risks in an additive fashion. As it appears that B[a]P is the most potent carcinogen among the PAH, this approach is quite conservative. The former potency factor for BaP is $11.5 \text{ [mg/kg/day]}^{-1}$ based on a linearized multistage model applied to the data of Neal and Rigdon (1967)⁵⁰.

This method will be used here for purposes of completing the risk assessment. It must be recognized that this approach is subject to change based on new data from EPA.

The other PAH have been subjected to different experiments:

⁵⁰ Neal, J. and R.H. Rigdon. 1967. Gastric tumors in mice fed benzo[a]pyrene: A quantitative study. *Tex. Rep. Biol. Med.* 25: 553.

Anthracene was given to mice by stomach tube at 1000 mg/kg-day for 90 days (EPA, 1989)⁵¹. No effects were seen in the dosed animals. EPA applied a safety factor of 3000 to this data to derive an RfD of 0.3 mg/kg-day.

Fluoranthene was subjected to a similar stomach tube feeding study in mice (EPA, 1988)⁵². The dose was 125 mg/kg-day for 90 days, and changes in liver weight, as well as effects on the blood were observed. EPA applied a safety factor of 3000 to this dose, to derive an RfD of 0.04 mg/kg-day.

Pyrene was also given by stomach tube to mice (75 mg/kg-day, for 90 days EPA, 1989)⁵³. Mild effects on the kidney were observed in this study, and EPA again applied a 3000-fold safety factor. The RfD is 0.03 mg/kg-day.

Regulatory Levels

The EPA Office of Drinking Water has proposed MCLs for several of the PAH's (55FR30445, July 25, 1990). They are as follows:

Benz(a)anthracene 0.1 ug/l

Benzo(a)pyrene 0.2 ug/l

Benzo(b)fluoranthene 0.2 ug/l

Benzo(k)fluoranthene 0.2 ug/l

Chrysene 0.2 ug/l

Indeno(1,2,3,cd)pyrene 0.4 ug/l

The values are not health-based.

The MCLG for the Carcinogenic PAH's is zero.

⁵¹ USEPA. 1989. Subchronic toxicity in mice with anthracene. Final Report. Hazelton Laboratories America, Inc. Prepared for the Office of Solid Waste, Washington, DC.

⁵² USEPA. 1988. 13-week mouse oral subchronic toxicity study. Prepared by Toxicity Research Laboratories, LTD. Muskegon, MI for the Office of Solid Waste, Washington, DC.

⁵³ USEPA. 1989. Mouse oral subchronic toxicity study of pyrene. Study conducted by Toxicity Research Laboratories, Muskegon, MI for the Office of Solid Waste, Washington, DC.

exposure can result in gastrointestinal disturbances, skin rash, mouth sores, dizziness, fever, back pain, burning on urination, and dark urine. Based on available data, phenol does not cause adverse reproductive effects, nor does it appear to be mutagenic or carcinogenic.

The oral RfD for phenol is 0.6 mg/kg/day, which is based on a developmental study in rats.⁴⁹ Pregnant rats received 0, 30, 60, or 120 mg/day of phenol in distilled water by gavage on day 6 to 15 of gestation. The rats were weighed daily and observed for clinical symptoms of toxicity during the treatment period. On the 20th day of gestation, the animals were sacrificed, and the fetuses were evaluated for abnormalities. Phenol treatment of pregnant rats did not affect their weight or cause clinical symptoms of toxicity, and both treated and untreated females had the same number of live fetuses. The major effect on fetuses of treated rats was a highly significant reduction in fetal body weights in the high-dose (120 mg/kg/day) group. The highest no observed adverse effect level (NOAEL) for fetuses was 60 mg/kg/day. This value was divided by a 100-fold uncertainty factor (10 for interspecies extrapolation and 10 for sensitive human populations), to establish the RfD.

Regulatory Levels

The MCL and MCLG have not been established. The lowest effect concentration (LEC) for freshwater is 2.56E+3 ug/l.

⁴⁹ NTP (National Toxicology Program) (1983) Teratologic evaluation of phenol in CD rats and mice. Report prepared by Research Triangle Institute, Research Triangle Park, N.C. NTIS PB83-247726. Gov. Rep. Announce. Index. 83(25):6247.

POLYCYCLIC AROMATIC HYDROCARBONS (PAH)

There are a large number of compounds in the chemical class of PAH. PAH observed at the Industriplex site are: anthracene, benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, benzo[k]fluoranthene, chrysene, dibenzo[a,h]anthracene, fluoranthene, indeno (1,2,3,cd)pyrene, phenanthrene, and pyrene.

PAH's are formed as a result of incomplete combustion processes or found naturally in fossil fuels, such as coal, petroleum, and petroleum by-products. PAH are widely distributed in the environment and have been detected in animal and plant tissue, soil, water, and air. They may arise from natural sources (e.g. volcanos, wildfires), but the most significant contribution to the environment arise from mobile and stationary combustion sources, such as automobile and aircraft emissions, utilities using fossil fuels, refuse burning, home heating, and industrial processes (e.g. thermal cracking). In industry, PAH are not intentionally synthesized, but obtained by refining coal or oil for use as fuels, lubricants, preservatives, and starting materials in petrochemical manufacture. Subsequently, it can be expected that the highest levels of PAH can be expected to occur in urban areas.

Structurally, PAH can generally be described as relatively large, flat molecules with a high carbon content (two or more adjacent benzene molecules, with different configurations or substitution, depending on the compound). Physically, they have very low water solubility and a very high affinity for organic material. In turn, the highest levels of PAH detected in the environment are usually associated with particulate organic matter. Therefore, higher levels of PAH in the ambient environment are usually detected in suspended matter in air or water, or found in soil or sediment. It is believed that unbound (free) PAH in soil and water is short-lived due to degradation by photolysis. Bioconcentration factors for certain PAH are predicted, based on their insoluble nature, to be quite high. In reality, however, they have been shown to be much lower because aquatic organisms readily metabolize and excrete PAH.

Historically, it was observed that chimney sweeps, who were exposed to high levels of soot, had high incidences of skin tumors. Subsequently, the first experiments that demonstrated the induction of cancer in laboratory animals were the result of "painting" mouse skin with coal tar. Various extracts of this coal tar revealed that benzo[a]pyrene (BaP) was one of

toxicity. Inhalation of nickel (II) over long periods of time have been associated with an asthmatic condition and rhinitis.

Blood nickel concentrations generally reflect the degree of nickel exposure. Treatment by chelation therapy is recommended and usually very effective. The National Institute for Occupational Safety and Health recommend a TLV of 1 ppb for nickel carbonyl; for soluble and insoluble nickel compounds, the recommended time-weighted average is 1 and 0.1 mg/m³, respectively.

Only specific forms of nickel (nickel subsulfide, nickel carbonyl) have been determined to cause cancer, and then only by the inhalation route. For airborne nickel and its compounds, the USEPA has derived a the cancer potency factor of 1.19 mg/kd/day⁻¹. The RfD for nickel was calculated in the following manner:

A 2-year feeding study using rats was conducted by Ambrose et al. (1976)⁴⁸. Animals were given a specific nickel salt (nickel sulfate hexahydrate) in concentrations of 0, 100, 1000 or 2500 ppm (estimated as 0, 5, 50, and 125 mg Ni/kg bw) in the diet. Body weights were found to be decreased in animals given the highest dose. Variable reductions in body weight during the experiment were also observed in animals given 1000 ppm. No changes were seen at 100 ppm (equivalent to 5 mg/kg/day), making this a No Observed Effect Level. This value was divided by a 100-fold safety factor (10 each for intraspecies extrapolation and for potential sensitivity in the human population) and modified downward an additional 3 fold due to concerns over the small sample size and inadequate study of the reproductive outcomes in the animals, giving an RfD value of 0.02 mg/kg/day, rounded.

Regulatory Levels

The MCL and the MCLG for Nickel is 100 ug/l.

⁴⁸ Ambros, A.M., D.S. Larson, J.R. Borzelleca and G.R. Hennigar, Jr. (1976) Long-term toxicologic assessment of nickel in rats and dogs. J. Food Sci. Technol. 13: 181-187.

PHENOL

Phenol, occasionally referred to as "carbolic acid", is a water soluble aromatic (monohydroxybenzene) compound. It has a "medicinal" odor and a sharp, burning taste. It occurs naturally in oil, coal, wood, human and animal wastes, and decomposing organic matter and can be distilled in quantity from petroleum products. It is also a by-product of combustion and some industrial processes. The largest production is as a synthetic intermediate for large volume industrial chemicals such as phenolic resins, bis-phenol, caprolactam plastic intermediates, and chlorinated phenols. Historically, phenol was widely used in medicine and is still used today in mouth, throat, and skin medications.

Free (unconjugated) phenol is rarely found in food or water supplies. As noted earlier, phenol is found in several medications, and exposure may vary with individual consumption. The taste and odor of phenol, and particularly some of its derivatives, are noticeable at relatively low concentrations. Because phenol is very water soluble, it will not bioaccumulate to any significant degree.

Inhalation of phenol vapors is primarily restricted to the industrial environment. Although phenol is efficiently absorbed from the lungs, it is also rapidly absorbed by the skin, which appears to be the major route of exposure in the workplace. This rapid rate of absorption is illustrated by the fact that acute doses can produce symptoms within minutes of administration, regardless of the route.

Phenol is rapidly distributed to all tissues in animals that have been exposed to the chemical. Within 15 minutes, the highest concentrations are found in the liver, followed by the heart, kidneys, lungs, blood, and muscle. Fortunately, phenol is a normal by-product of protein metabolism and is thus rapidly detoxified by the liver. Some of the phenol is oxidized to carbon dioxide and water but the largest percentage of the dose is excreted in the urine as free or conjugated phenol. Finally, phenol is a by-product of the metabolism of benzene and a few other aromatic compounds.

Regardless of the route of administration, the signs and/or symptoms of acute toxicity in humans and experimental animals are similar. In humans, acute exposure appears to affect the central nervous system, resulting in sudden collapse and unconsciousness. Chronic

mg/kg/day and an uncertainty factor of 1000 (10 for interspecies and 10 for intraspecies variability, and 10 for extrapolation from subchronic to chronic effects).

4-methylphenol has been classified as a possible human carcinogen, although no potency factor has been established for any of the cresols.

Regulatory Values

Currently, no Federal drinking water standards or Ambient Water Quality Criteria are available for 4-methylphenol.

NICKEL

Nickel exists in many oxidation states, but the most prevalent form in the environment is Ni^{+2} (II). Studies of workmen in the nickel-refining industry have shown that nickel is a respiratory tract carcinogen. In ambient air, nickel is present in low concentrations (average 0.008 ug/m^3) as a constituent of particulate matter. The primary anthropogenic sources are the combustion of fossil fuels, sludge, and waste and nickel-dependent industries (stainless steel, electroplating, catalysts, batteries).

It is estimated that airborne nickel emitted from combustion sources exists primarily in the form of nickel oxide(s) or sulfate. Of this, one-third to one-half is soluble. The average air concentration of nickel in the urban U.S. is 9 ug/m^3 , about four times that of rural levels. In natural waters, nickel exists prenatally as a precipitate on particles or associated with organic matter. As the free ion, it may complex with organic or inorganic ligands, which, like other metals, are pH dependent. The mean concentration found in drinking water from eight metropolitan areas was 4.8 ug/l (EPA, 1985)⁴⁷. There is increasing evidence that nickel is a nutritionally essential trace metal. Studies with rats reveal that nickel deficiency is associated with retarded body growth and anemia. The major route of nickel exposure for the general human population is ingestion. Although dietary intake is high (300 - 500 ug/day), intestinal absorption is low (1 - 10% of intake). Generally, the amount of nickel in the feces is about 100 times that found in the urine.

Acutely, oral exposure to nickel is relatively non-toxic. However, dermal exposure to the metal is one of the most common causes of acute contact dermatitis. It is estimated that 4 - 9% of the U.S. population reacts positively to a nickel skin patch test, thus representing a significantly large segment of the population that is sensitive to the metal. Non-occupational exposure can result from contact with nickel-containing jewelry, coins, prostheses, stainless-steel cookware, etc. Acute effects via inhalation are well documented for nickel carbonyl, a compound formed when nickel comes into contact with carbon monoxide during processing. In both man and animals, the lung is the target organ, and the effects resemble a form of viral pneumonia. Little work has been done on nickel

⁴⁷ EPA (1985) Health Assessment Document for Nickel. Environmental Criteria and Assessment Office, Office of Research and Development, Research Triangle Park, N.C. 27711. EPA/600/8-83/012F.

mg/kg/day.

The inhalation potency slope is based on results of the linearized multistage model applied to the combined liver and lung tumors observed in female mice in the NTP study. A pharmacokinetic model was used to develop a "unit risk" value (a risk per unit air concentration) of $4.7 \times 10^{-7} [\mu\text{g}/\text{m}^3]^{-1}$. Because pharmacokinetic considerations were applied to the unit risk, EPA advises that a CPF not be back-calculated. As such, cancer risk from inhalation should be calculated from the unit risk value.

4-METHYLPHENOL (p-Cresol)

4-Methylphenol, an extract of coal tar, is a yellowish liquid with a distinctive, disagreeable odor. By itself, 4-methylphenol is used as a disinfectant. More commonly, it is used in combination with 2-methylphenol (o-cresol) and 3-methylphenol (m-cresol), a mixture known as cresol. Cresol also serves as a disinfectant and is used in the manufacture of chemicals, dyes, and plastic.

Animals absorb cresol through their skin, open wounds, and mucous membranes of the respiratory and gastrointestinal tracts. The rate at which cresol is absorbed through skin is influenced more by the total area of skin exposed than by the concentration of cresol. In the body cresols are converted to water soluble compounds and excreted in the urine. Cresols, especially 4-methylphenol, are normal components of human urine; between 16 and 39 mg of 4-methylphenol are normally excreted each day.

When applied topically, cresol can burn and corrode human skin, and can cause allergic dermatitis in sensitive individuals. Prolonged or extensive skin contact can be fatal. Eye exposure can cause extensive damage and ultimately blindness. When inhaled or ingested, cresol irritates the lungs and causes inflammation of the liver and kidneys. The central nervous system is the primary target of prolonged exposure by any route; symptoms of such exposure include muscular weakness, gastrointestinal disturbance, severe depression, coma, and death by respiratory failure.

An RfD of 0.05 mg/kg/day has been established for 4-methylphenol based on the following subchronic toxicity study.⁴⁶ Rats received 0, 50, 175, or 600 mg/kg/day of p-cresol for 90 days. Animals treated with 600 mg/kg/day showed significant weight loss, increased kidney weight, and a 47% mortality rate (9 of 30 males and 19 of 30 females). Within 15 to 30 minutes of high-dose treatment, central nervous system effects were observed, including lethargy, loss of muscular coordination, coma, and convulsions. These symptoms abated within an hour. The mortality rate at 450 mg/kg/day dropped to 20% (1 of 30 males and 1 of 30 females). At 50 mg/kg/day, no significant adverse effects were recorded. The RfD (0.05 mg/kg/day) was calculated using the no observed adverse effect level (NOAEL) of 50

⁴⁶ EPA (1987). o, m, p-Cresol. 90-Day oral subchronic neurotoxicity study in rats. Office of Solid Waste, Washington, D.C.

METHYLENE CHLORIDE

Methylene Chloride (MeCl) is a single carbon molecule bonded with two chlorine and two hydrogen atoms. It is a solvent used for a wide variety of purposes (e.g. degreasing, paint remover, aerosols) in the United States. Annual U.S. production is about 590 million pounds. It is estimated that 85% of this is eventually lost to the atmosphere through dispersive use. The average background concentration for MeCl is approximately 35 parts per trillion (ppt). Concentrations in urban areas may be 10 to 100 times higher than this.

In finished drinking water, concentrations in the low part per billion range have been measured. MeCl is readily degraded by bacteria in concentrations up to 400 ppm. In landfill areas, it is believed that MeCl may easily reach groundwater because it is not easily retained in the soil. There is no evidence that MeCl bioaccumulates through the food chain.

Like other solvents, inhalation of MeCl is the most rapid form of exposure. However, it is also efficiently absorbed after ingestion. Because MeCl is very fat-soluble, it will distribute easily throughout the body. It will cross the blood-brain and placental barrier, and (like other solvents) cause narcotic effects on the central nervous system. Studies in experimental animals indicate that MeCl is not toxic to the developing fetus.

Once in the body, the liver will detoxify MeCl to carbon dioxide (CO₂) and carbon monoxide (CO), which is eventually eliminated via the lungs. Blood normally contains a small percentage of CO at all times, and exposure to a few ppb of MeCl is of no consequence. However, continuous exposure (500 ppm or above) may increase this percentage (as much as 15%). This level is still below the that considered hazardous to normal, healthy individuals, but may place added stress on high risk groups, such as pregnant women or people with cardiovascular disease or on people engaged in high oxygen demand activities.

The NOEL (no observed effect level) for MeCl in humans (inhalation) appears to be around 100 ppm. Potential adverse effects, which are primarily a narcotic effect, are seen at concentrations that greatly exceed this level. The lowest concentration reported to affect eye-hand coordination was 200 ppm (695 mg/m³).

From the available evidence, MeCl is of very low toxicity to humans and animals. Even at near lethal levels, toxic effects on the liver, kidney, and heart are minimal.

Studies in the laboratory have shown that MeCl tests positive in mutagenic tests. Based on limited animal data designed to test if MeCl is carcinogenic, the USEPA Carcinogen Assessment Group has classified it as a "B2", meaning that it is a "probable human carcinogen".

In a 2-year study conducted by the National Coffee Association (as cited in EPA, 1985)⁴⁴ rats were given 0, 5, 50, 125, or 250 mg dichloromethane/kg-day in drinking water, while mice were given 0, 60, 125, 185, or 250 mg/kg/day in water. Female rats were observed to have an increased incidence of neoplastic nodules, believed to be a reversible, precancerous condition, and carcinomas of the liver, relative to controls (although the incidence was not increased in comparison to typical incidence as determined from historical controls). Male rats did not show an increased incidence of liver tumors. Male but not female mice had elevated incidences of neoplastic nodules and liver tumors when the two pathological changes were combined (the increased was not statistically significant and not dose related). A linearized multistage model was applied to the male mouse data.

In a study by the National Toxicology Program (NTP, 1986)⁴⁵, rats and mice were exposed to dichloromethane 6 hours per day, 5 days per week for 2 years. Non-malignant tumors of the mammary gland (adenomas and fibroadenomas) were observed in male and female rats. Leukemias were increased in female rats. There were increased incidences of liver tumors and lung tumors in mice.

The linearized multistage model was applied to the female mouse liver tumor response in this study, and the oral potency slope is the mean value of slope found for this data and the National Coffee Association data discussed previously. The potency factor is 0.0075 [mg/kg-day]⁻¹. The National Coffee Association Study is also the basis for the RfD fo 0.06

⁴⁴ EPA (1985) Addendum to the Health Assessment Document for Dichloromethane (methylene chloride). Updated carcinogenicity assessment. Prepared by the Carcinogen Assessment Group, Washington, DC. EPA 600/8-B2/004FF.

⁴⁵ NTP (1986) Toxicology and carcinogenesis studies of dichloromethane (methylene chloride) in F344/N rats and B6C3F1 mice (inhalation studies). NTP-TRS-306.

Because most of the lead in the cell is bound up by these inclusions, and because they can be found in the urine, it is believed that they are a major detoxification mechanism for this metal. Treatment of lead intoxication involves identification and removal of the exposure source and chelation therapy.

The carcinogenic potential of lead compounds has been demonstrated in rats and mice. The cancer response is different among studies, but is often kidney carcinoma. Based on this information, EPA has classified lead as a B-2 carcinogen. However, no CPF have been developed due to concerns about the application of the usual risk extrapolation methods to the case of lead.

EPA has not developed an RfD for lead either, again due to the complexity of findings concerning toxicity and its relation to dose. The EPA IRIS file refers the reader to documentation of the Ambient Air Quality Standard for lead. In the former guidance for conducting risk assessments (the Superfund Public Health Evaluation Manual), now obsolete, it was suggested that an acceptable chronic intake values, similar to an RfD, of $1.4E-3$ mg/kg/day be used to characterize the potential impact of ingested lead. This is a back-calculation of intake at the current primary drinking water standard (MCL) for lead of 50 ug/L.

In order to produce a characterization of lead contamination at the Site, this value will be used. However, it should be noted that this value cannot be considered a true RfD. In all likelihood, characterization of potential health effects of lead exposure will require methods different from those currently in use. An approach from EPA is still pending.

The current MCL for lead in drinking water, as mentioned, is 50 ug/L and is considered temporary. A change in this value to 20 ppb was proposed in 1985 (FR50 page 46936, November 13), and, in 1988, a still lower MCL of 5 ppb was proposed (FR53 page 31516, August 18), along with an MCLG of zero. No final rule has been passed. In May, 1991, EPA issued an "Action Level" of 15 ppb for water distribution systems that relates to treatment techniques that must be applied if this value is exceeded. The value is not health-based.

MERCURY

Mercury is unique in that the elemental form (Hg^0) of the metal exists as a liquid at room temperature. This form has a very high vapor pressure, and represents a considerable hazard at room temperature. Other inorganic compounds containing mercury in different oxidation states (Hg^{+2} , Hg^+). The major sources of mercury input into the atmosphere are from chloralkali plants, wood preservatives and fungicides, and the burning of fossil fuels and municipal waste. Once released into the general environment, the inorganic forms of the metal may be methylated by indigenous microbial flora, and are available for bioaccumulation in the food chain. Thus, the major source of mercury exposure for the general population is fish, especially species at the top of the food chain, such as tuna and swordfish.

In general, elemental mercury and the organomercury compounds affect primarily the central nervous system, whereas inorganic mercury salts target the kidney. With organomercury compounds, the symptoms of intoxication include tremor, insomnia, emotional instability, depression, and irritability. For inorganic mercury, symptoms of nephrotoxicity are apparent, such as proteinuria and edema of the lower extremities.

There is no evidence to date that either inorganic or organic forms of mercury cause cancer. For alkyl and inorganic forms of mercury, the current reference dose (U.S. EPA, HEAST, 4th quarter) in mg/kg/day, is 0.0003. This value was derived from observations on humans that blood levels of mercury greater than 200 ng/ml are associated with adverse effects on the central nervous system.

Regulatory Value

The EPA Office of Drinking Water has set an MCL and MCLG for mercury of 2 ug/l.

A cancer study of ethylbenzene has not been conducted, however ethylbenzene has not been found to be mutagenic in cell assays. EPA has interpreted the weight of evidence for carcinogenicity of ethylbenzene as D.

The no effect level from the Wolf, et al study has been used to calculate an RfD for ethylbenzene. The highest level where no effects were seen was 136 mg/kg/day (this dose was converted to 97.1 mg/kg/day to account for the fact that the animals were only dosed 5 days per week, despite the finding that excretion of the compound is rapid). This dose was lowered by a factor of 1000: a factor of 10 to account for uncertainty associated with extrapolation from animals to man, 10 to account for potentially sensitive people in the general population, and 10 to account for the short duration of the experiment relative to possible lifetime exposures that might be analyzed in a risk assessment. The resulting RfD is 0.1 mg/kg/day.

An inhalation RfD for ethylbenzene has yet to be developed.

Regulatory Levels

The MCL and MCLG for ethylbenzene is 700 ug/l.

LEAD

The widespread use of lead in paints and gasoline early in this century has made it the most widespread of all the toxic metals, and it has been detected in all media in the environment. Food and beverages contribute to the human body burden of lead, and, although quite variable, the average adult diet contains about 150 ug/day, 0.75 - 120 ug/day for children. However, it is environmental exposure, principally ingestion of lead-based paint chips in children and workplace exposure (e.g. smelting operations, battery factories) in the adult, that comprise the majority of clinical lead poisoning cases. Human populations living near lead smelting operations are at extra risk of exposure to the metal.

Symptoms of chronic lead poisoning include malaise, loss of appetite, anemia, irritability, palsy, analgesia, and reproductive and kidney dysfunction (Hammond and Beliles, 1980)⁴³.
1 Hammond, P.B., and Beliles, R.P. (1980) -Metals-. In "Toxicology: The Basic Science of Poisons", 2nd Edition, Casarett and Doull, eds., Macmillan Publishing Co., Inc., N.Y., N.Y. In order of decreasing sensitivity, the major systems affected by lead are the blood, the nervous system, and the kidneys. Low blood lead levels (30 - 50 ug/dl) will interfere with the production of heme, significantly shorten the life span of red blood cells, and possibly result in microcytic (small), hypochromic (pale) anemia. Although recent studies have shown that these low levels may also cause "subclinical lead toxicity" (neuropsychological dysfunction), more serious health impacts are seen at higher blood levels (40 - 80 ug/dl) and involve a debilitating encephalopathy in children and peripheral neuropathy in adults. Although rarely seen today because of effective lead toxicity screening programs, the signs of encephalopathy in children varies from ataxia to stupor, coma, and convulsions. In the adult, the peripheral neuropathy seen in chronically exposed workers was characterized by footdrop or wristdrop that was a result of motor nerve degeneration and dysfunction. The renal effects caused by lead also differ with age. Usually, in children with acute lead poisoning, there is a reversible renal tubular dysfunction, usually associated with central nervous system effects. In the chronically exposed adult, there is seen an irreversible chronic interstitial nephropathy. In both cases, a "pathognomonic feature" is the presence of intranuclear inclusion bodies that are comprised of an acidic lead-protein complex.

⁴³ Hammond, P.B., and Beliles, R.P. (1980) -Metals-. In "Toxicology: The Basic Science of Poisons", 2nd Edition, Casarett and Doull, eds., MacMillan Publishing Co. Inc., N.Y., N.Y.

ETHYLBENZENE

Ethylbenzene is a slightly volatile liquid that is somewhat soluble in water. Ethylbenzene is used in the production of styrene and polymers, but is also a component of fuels.

As a constituent of gasoline, it would be expected that ethylbenzene would be found frequently in water supplies. However, the compound was only detected in 3 of 466 water distributions randomly for sampling in the Groundwater Protection Survey sponsored by EPA. The detection frequency was similarly small in water supply systems selected specifically because they were expected to be contaminated with organic compounds.

As a constituent of gasoline, ethylbenzene is detected frequently in the air, and EPA estimates that exposure via this route is more important than that of water.

Few studies have been done on the gastrointestinal absorption of ethylbenzene, although it is clear that absorption is easy, given the toxic effects observed following oral dosing.

Absorption from the lung has been measured to be 44 and 64% of the administered dose in experiments with rats and human volunteers, respectively (EPA, 1985)⁴⁰. These values are comparable within the uncertainties of the experiments. The amount of uptake of ethylbenzene in inhalation exposures is related to the concentration inhaled, as well as the amount of body fat.

Skin absorption of pure ethylbenzene and ethylbenzene in water solution has been demonstrated by Dutkiewicz and Tyras (1967)⁴¹ who observed rates of absorption that varied with concentration (approximately 2 ug/cm²/min for a solution of approximately 100 ppm and 3.2 to 3.6 ug/cm²/min for solutions of approximately 150 ppm). However, due to the methods used for the experiments (absorbed dose was measured as the amount that could not be retrieved by washing the administered material off the hands), it is not clear how much ethylbenzene actually enters the system, rather than remaining on or absorbed in the skin.

⁴⁰ EPA (1985) Drinking Water Criteria for Ethylbenzene (Final Draft). EPA/600/x-84/163-1.

⁴¹ Dutkiewicz, T. and Tyras, H. (1967) Study of skin absorption of ethylbenzene in man. Brit. J. Ind. Med. 24: 330-332.

Once absorbed, ethylbenzene distribute to many tissues in the body. Chemical alteration of ethylbenzene occurs in the liver, where the compound is changed to more water soluble metabolites, and excreted in the urine. Greater than 90% of absorbed ethylbenzene is excreted in this manner. It is of note that the metabolites of ethylbenzene appear to differ in man versus experimental animals, reflecting different biochemical mechanisms of elimination. Excretion appears to occur rapidly; in a study of human volunteers, less than 5% of the dose of ethylbenzene administered by inhalation was left 22 hours following exposure.

Ethylbenzene appears to have low acute toxic potency. Lethal doses (LD50) in animals have been observed at 4-6 g ethylbenzene per kilogram body weight. At these doses, death appears to be due to lung congestion and hemorrhage. Central nervous system depression, a typical effect seen in solvent exposures, has been with ethylbenzene. Above 100 ppm for 8 hours, the effects reported in humans are mild throat and eye irritation, as well as sleeplessness fatigue and headache.

In a longer term general toxicity study of ethylbenzene, Wolf, et al (1956)⁴² gave rats oral doses of ethylbenzene between 13.6 and 680 mg/kg/day for 130 days. No effects were seen on gross or microscopic examination of tissues from animals given 13.6, or the next highest dose, 136 mg/kg/day. Increased liver and kidney weights, as well as changes observable under the microscope were noted in animals given 408 mg/kg/day.

In other studies, degeneration of testicular tissue has been observed in rabbits and monkeys given high doses of ethylbenzene by inhalation. In reproductive studies, no effect was seen on fertility in rabbits, nor was toxicity to the fetus observed (however, since ethylbenzene has been measured in blood from animal fetuses when the mother was exposed, it might be expected that fetal toxicity would occur at doses where the mother was effected as well). Studies of the potential for ethylbenzene to cause birth defects are inadequate.

⁴² Wolf, M.A., Rowe, V.K., McCollister, D.D., Hollingsworth, R.L., and Oyen, F. (1956) Toxicological studies of certain alkylated benzenes and benzene. Arch. Ind. Health 14: 387-398.

Other systemic actions are minimal. Carpenter, et al (1952)³⁷ fed guinea pigs DEHP at a level of 19 and 64 mg/kg-day for a year and found no difference in mortality, body weight, or pathology of any organ. However, both treated groups were found to have increased liver weights. Other studies indicate that DEHP is a reproductive toxin and that it effects the testes, producing reduced testicular weight, degeneration, and decreased sperm density and motility³⁸. The National Toxicology Program conducted a study in which rats were given feed containing 12,000 or 6,000 ppm DEHP, and mice were fed chow containing 3000 or 6000 ppm DEHP for life³⁹. Female rats and both sexes of mice were found to develop tumors of the liver. If neoplastic nodules (liver lesions that reverse if exposure is ceased, but may proceed to cancer if exposure continues) were added to liver tumors, treated male rats were also found to have statistically elevated tumor rates. Based on this information EPA rates DEHP as a B-2 or Probable Human Carcinogen.

EPA used the Carpenter study to develop an oral RfD. The lower dose (19 mg/kg-day) was considered a Lowest Observed Adverse Effect Level and was reduced 1000 fold (uncertainty factors of 10 each to account for interspecies extrapolation, possible sensitive individuals in the human population, and the fact that the study was not conducted for a lifetime) to arrive at an RfD of 0.02 mg/kg-day.

The EPA used a linearized multistage model of dose response that assumes no threshold

³⁷Carpenter, C.P., Weil, C.S., and Smyth, H.F. (1953) Chronic oral toxicity of di-(2-ethylhexyl)phthalate for rats and guinea pigs. Arch. Indust. Hyg. Occup. Med. 8:219-226.

³⁸ Price, C.J., Tyl, R.W., Marr, M.C., Sadler, B.M., and Kimmel, C.A. (1986) Reproduction and fertility evaluation of diethylhexylphthalate (CAS 117-81-7) in Fischer 344 rats exposed during gestation. Final Report. National Toxicology Program Report No. NTP 86-309.

Agarwal, D.K., Eustis, S., Lamb, J.C., Reel, J.R., and Kluwe, W.M. (1986) Effects of di-(2-ethylhexyl)phthalate on gonadal pathophysiology, sperm morphology, and reproductive performance of male rats. Environ. Health Pers. 65:343-350.

³⁹National Toxicology Program (1982) National Toxicology Program carcinogenesis assay of DEHP (CAS 117-81-7) in Fisher 344 rats and B6C3F1 mice (Feeding Study). NTP 80-37.

for carcinogenic effect and linear response at low doses to derive a cancer slope factor for DEHP. This value is $1.4E-2[\text{mg/kg-day}]^{-1}$. There is some controversy over this approach, as DEHP does not appear to be mutagenic, and plausible mechanisms for the carcinogenicity of the compound may have a threshold for action.

Regulatory Value

EPA proposed an MCLG of zero and MCL of 4 ppb for DEHP in July, 1990.

pathology studies at the end of the dosing. The authors noted decreased body and organ weight in the 50,000 ppm group (estimated to be a dose of 3160 mg/kg/day) and a slight decrease in body weight in female rats only, in 10,000 ppm group (estimated dose 750 mg/kg/day). Other toxic signs and pathology were not seen.

DEP has been shown to test negative in mutagenicity tests in vitro, and is currently being tested for carcinogenic effects (NTP, 1989). Available evidence suggests that the phthalate esters are not carcinogenic.

EPA used the Brown study to develop an oral RfD. The 750 mg/kg/day dose was considered a No Observed Adverse Effect Level. EPA reduced the dose 1000-fold (a factor of 10 each for species to species extrapolation, possible sensitive individuals in the human population, and the fact that the study was not of long duration). The resulting RfD was 0.8 mg/kg/day.

DI-[2-ETHYLHEXYL]PHTHALATE

Di-[2-ethylhexyl]phthalate (DEHP) is a liquid used primarily as a plasticizer; a compound added to plastics to make them more flexible. Items such as raincoats, shower curtains, and food packaging materials can be up to 40% DEHP by weight.

DEHP is readily absorbed by the gastrointestinal tract, although much of the compound may be converted to mono-ethylhexyl-phthalate (MEHP) prior to absorption. Less is known about skin and lung absorption. About 5% of administered compound was reported to have been absorbed from rats³⁵, but quantitative estimates of lung absorption are not available. DEHP and MEHP are widely distributed in the body. Much is found in the liver, where the compounds are converted by liver enzymes to more water soluble metabolites and excreted in the urine. Excretion is rapid and virtually complete. There are significant quantitative and qualitative differences in the way laboratory animals and man metabolize DEHP. This may be important in extrapolating observations of toxic effects in laboratory animals to man.

Studies of the effect of acute exposure in animals indicate DEHP is of relatively low toxicity. No information is available on the acute toxicity of DEHP in humans.

Epidemiologic studies in workers chronically exposed to DEHP (Theiss, et al, 1978)³⁶ do not indicate increased mortality or disease (1 case each of death from pancreatic cancer and uremia was found to be statistically elevated, but statistical inference on a single case is usually considered equivocal), however the size of the study and absence of exposure data limit its usefulness.

On a chronic basis, DEHP appears to affect the liver and testes of experimental animals.

³⁵ El Sisi, A.E., Carter, D.E., and Sipes, I.G. (1985) Dermal absorption and tissue distribution of phthalate esters. *The Toxicologist* 5:246.

³⁶Theiss, A.M., Korte, and Fleig, H. (1978) Untersuchungen zur morbiditat bei Mitarbeitern mitexposition gegenüber Di-2-aethylhexylphthalat. *Vortr. Anl. d. Jahrestg. d. Deutschen Gesellschaft f. arb. Med. in Frankfurt* 25:137-151 (German).

trans-1,2-DICHLOROETHENE

Commercially used dichloroethene is usually a mixture of two isomers, cis-1,2-dichloroethene and trans-1,2-dichloroethene. Both are slightly soluble in water and very soluble in ethers, alcohols, and most organic solvents. They are volatile liquids with a slight acrid, ethereal odor and are used as solvents, in perfumes, lacquers, thermoplastics, and organic synthesis.

Non-occupational exposure to these compounds would occur primarily by inhalation and ingestion. Although no human or animal studies have been carried out, it can be assumed that absorption of these compounds would be similar to tri- and tetrachloroethene, or 35-50% by inhalation and 100% by ingestion. Metabolism and elimination of the cis or trans forms of 1,2-DCE from the body is similar to that of 1,1-DCE, although these alkenes are less toxic and do not appear to be mutagenic or carcinogenic.

The primary effect of 1,2-dichloroethene exposure is central nervous system narcosis and irritation. It has been used as an anesthetic in both animals and man; animals recovered well and tolerated repeated exposures.

Barnes, et al (1985)³³ gave trans-1,2-dichloroethene in drinking to mice, for 90 days (0.1, 1.0, or 2.0 mg/ml). Increases in serum alkaline phosphatase, a finding often associated with liver damage, were seen in male mice in the 1 and 2 mg/ml levels (equivalent to 175 and 387 mg/kg/day). On this basis, the no observed effect level is 0.1 mg/ml (equivalent to 17.5 mg/kg/day), which was divided by a 1000-fold safety factor (a factor of 10 each for uncertainty in the extrapolation of dose levels from laboratory animals to humans, potential for sensitivity in humans, and extrapolation of subchronic to chronic exposure) to arrive at an RfD of 0.02 mg/kg day, rounded.

Regulatory Values

This RfD was used by EPA in proposing an MCL and MCLG of 100 ug/l.

³³ Barnes, D.W., W.M. Sanders, K.L. Shite, Jr., et al. 1985. Toxicology of trans-1,2-dichloroethylene in the mouse. Drug Chem. Toxicol. 8: 373-392.

DIETHYLPHTHALATE

Diethylphthalate (DEP) is the "ortho" ethyl ester of benzenedicarboxylic acid. DEP and other phthalic acid esters are most commonly used as "plasticizers", i.e. chemicals which, when mixed with a plastic polymer, will impart flexibility into the plastic product. They are also used in defoaming agents, as vehicles in perfumes, and in lubricating oils. On a global scale, 3 to 4 billion pounds of phthalate esters are produced annually.

Because of the extremely large production rates and the wide variety of uses for these compounds, they are considered ubiquitous contaminants and can be detected in different phases of the environment. The most common route of exposure to humans occurs when phthalic acid esters migrate into foods that come in contact with plastic packaging. People may also be exposed by dermal contact (cosmetics) and even via parenteral administration (blood bags and tubes in which the ester is extracted by an intravenous solution). Inhalation exposure may occur by breathing fumes of burning plastic products (e.g. exposure during fires) or in poorly ventilated rooms containing PVC plastics.

Phthalic acid esters are slowly absorbed through the skin, lung, and digestive tract. Once absorbed, they (or their metabolites) will distribute quite rapidly to various organs and tissues in both man and animals. Excretion occurs via the urine and feces.

These compounds have a relatively low order of toxicity. Rats can tolerate doses of up to 0.5% of their body weight without death occurring, and rabbits can withstand 3 ml/kg/day without significant toxic effects. When applied to the skin, DEP will not invoke any allergic or inflammatory responses. Long-term studies in experimental rats and dogs also showed limited significant adverse effects.

Brown, et al (1978)³⁴ fed rats chow containing from 2000 to 50,000 ppm diethylphthalate for 16 weeks. The researchers monitored weight gain and food and water intake during the experiment and conducted urinalysis, hematology, organ weight determination and organ

³⁴ Brown, D, Butterworth, K.R., Gaunt, I.F., Grasso, P., and Gangolli, S.D. (1978) Short-term oral toxicity study of diethyl phthalate in the rat. *Food Cosmet Toxicol.* 16: 415-422.

also toxic to the mother. When administered in drinking water to rats, 1,1-DCE had no effect on the reproductive outcome.

The data implicating 1,1-DCE as mutagenic or carcinogenic are controversial. A large number of studies have been conducted on 1,1-DCE carcinogenicity in animals, and only one produced an indication of a carcinogenic response. Also, an epidemiologic study showed no carcinogenic effect associated with 1,1-DCE in workers (Ott et al., 1976). However, most of the studies suffered from inadequacies of sample size, design, or duration of dosing so that it is not possible to rule out the carcinogenicity of 1,1-DCE with confidence. Thus, EPA has classified 1,1-DCE as a C, or possible human carcinogen, based on a positive inhalation study in mice (Maltoni et al, 1985)²⁹.

Maltoni, et al. (1985) observed kidney adenocarcinoma in mice exposed to 10 or 25 ppm 1,1-DCE in air for 4-5 days/week for 12 months. A statistically significant increase in kidney adenocarcinoma was noted in male mice only. EPA (1985a) applied a linearized multistage model of dose-risk to this data to arrive at a CPF of 1.2 [mg/kg/day]⁻¹. This value is based on metabolized dose, so that it probably should not be used with administered doses.

Five oral dosing cancer studies in rats or mice have been negative (Maltoni et al., 1985; Quast et al., 1983; Humiston et al., 1978³⁰; NTP, 1982³¹; Ponomarkov and Tomatis, 1980³²). However, only the NTP study was conducted for a full two years (the average lifetime of the rats used for the study) and the doses may not have been high enough for accepted EPA testing requirements.

EPA applied the linearized multistage model to the data from the NTP and Quast, et al

²⁹ Maltoni, C., et al (1985) Experimental Research on Vinylidene Chloride Carcinogenesis. Archives of Research on Industrial Carcinogenics Vol 3. Princeton, NJ. Princeton Sci. Publishers.

³⁰ Humiston, C.G., et al (1978) Results of a two-year toxicity and oncogenicity study with vinylidene chloride incorporated into the drinking water of rats. Manufacturing Chemists Association Report VCD 1.3 Tox-ori-Drw.

³¹ NTP (1982) NTP Technical Report on the Carcinogenesis Bioassay of Vinylidene Chloride in F344/W Rats and B6C3F1B Mice (Garage Study) Publication No. 82-17841.

³² Ponomarkov, V and L. Tomatis (1980) Long-term testing of vinylidene chloride and chloroprene for carcinogenicity in rats. Oncology 37: 136-141.

studies, despite the lack of statistical significance, and selected the highest of the CPFs, $6E-1$ [mg/kg/day]⁻¹, from the NTP study of kidney tumors in male rats. In the IRIS notation on this compound, EPA states "The estimate is based on a data set in which there is no significant increase in tumor incidence. The confidence that the upper limit is not greater than 0.6/mg/kg/day is high, since it is the largest value by a factor of 3 from four rat data sets in two studies. If drinking water exposure alone is considered the estimates might be reduced by a factor of 3."

Regulatory Values

The MCL and MCLG for 1,1-dichloroethene is 7.00 ug/l, based on the RfD (with an additional 10 fold safety factor, in recognition of the C cancer rating), rather than the cancer potency factor.

unequivocal reproductive or carcinogenic effects. However, based on positive findings in a single sex, EPA classifies 1,1-Dichloroethane as a C; possible human carcinogen. No potency factor has been derived.

No reports are available for exposure of humans under controlled conditions. In the workplace, exposure to 1,1-dichloroethane by inhalation will cause central nervous system effects. Prolonged skin contact will result in irritation. The current occupational limit for a 5 day/40 hr. exposure in an industrial environment is 100 ppm (400 mg/m³).

EPA has determined that the average daily human exposure dose, below which no adverse effects are to be expected, is 0.12 mg/kg/day for ingestion and 0.14 mg/kg/day for the inhalation route.

Regulatory Values

No MCL or MCLG has been developed for 1,1-dichloroethane.

1,1-DICHLOROETHENE (Vinylidene Chloride)

1,1-Dichloroethene (1,1-DCE) is a reactive, flammable, colorless liquid that can form toxic or explosive compounds in the presence of air. It is heavier than water and readily soluble in it (2.25 g/l). Production in the U.S. is approximately 178 million pounds per year, and almost all of this goes into the production of copolymers with vinyl chloride or acrylonitrile. The amount of 1,1-DCE that is lost to the atmosphere during production is estimated at 1.3 million pounds/year. Being so reactive, it breaks down quickly in the atmosphere (50% decay takes 5-12 hours). 1,1-DCE is not persistent in the aquatic environment, with most being lost to the air by volatilization. 1,1-DCE has been detected in 3% of the nations water supplies at an estimated mean concentration of 0.3 $\mu\text{g/liter}$. For the majority of the U.S. population, the daily exposure to 1,1-DCE from ingestion of drinking water has been estimated to be less than 0.6 μg (EPA. 1980)²⁷.

1,1-DCE is readily absorbed by animals following oral or inhalation exposure. Because it is reactive, it is easily transformed by the liver into products that may be more toxic than the original molecule. These metabolites will form toxic lesions in the liver and kidney, sometimes within 2 hours of exposure.

The RfD for 1,1-DCE is based on the study of Quast, et al (1983)²⁸, who administered the compound to rats in drinking water (50, 100, or 200 ppm) for 2 years. Microscopic lesions of the liver were noted in female rats of all treatment groups (only the 200 ppm treatment group was statistically different for males). Therefore, a lowest effect level was the 50 ppm dose (equivalent to 9 mg/kg/day). A safety factor of 1000 (10 each was used for use of a LOAEL, for interspecies variation, and potential variability in human sensitivity) was applied, to arrive at a RfD of 0.009 mg/kg/day.

In reproductive studies, there is conflicting evidence that 1,1-DCE will produce adverse effects to the fetus. Toxic effects to the fetus are only observed at concentrations that are

²⁷ EPA (1980) Ambient Water Quality Criteria for Dichloroethylenes. Criteria and Standards Division. EPA 440/5-80-041.

²⁸ Quast, J.F., C.G. Humiston, C.E. Wade, et al. (1983) A chronic toxicity and oncogenicity study in rats and subchronic toxicity study in dogs on ingested vinylidene chloride. Fund. Appl. Toxicol. 3: 55-62.

Regulatory Levels

The MCL and MCLG for chromium is 100 ppb.

1,1-DICHLOROETHANE

1,1-Dichloroethane is a colorless, liquid. It is used in industry (although in limited use compared to other solvents) as a chlorinated solvent intermediate, in paint removers, in metal degreasing, and as an organic synthesis intermediate. It is not produced in the United States, being imported for these specialized processes.

Based on the physical properties of 1,1-dichloroethane, it can be predicted that transport of dissolved 1,1-dichloroethane in water will be somewhat retarded, but not to the same extent as other chlorinated solvents (such as tetrachloroethene). The vapor pressure and Henry's law coefficient for the compound suggest it will readily volatilize from water or other media.

1,1-Dichloroethane has limited use and is not commercially produced in the United States. It is found in some drinking water, perhaps as a result of chlorination, and is often found in water contaminated with tetrachloroethene or trichloroethene, probably as a result of degradation of these compounds.

Limited information is available on the absorption of 1,1-dichloroethane. However, comparisons of the physical properties of the chemical with its isomer 1,2-dichloroethane, suggest that gastrointestinal absorption of 1,1-dichloroethane should be faster than 1,2-dichloroethane. The absorption of 1,1-dichloroethane should be less, and exhalation greater than 1,2-dichloroethane, based on blood-air partition coefficients of 4.7 and 19.5, respectively (EPA, 1984)²⁶.

Less information is available for this compound than for its more toxic isomer, 1,2-dichloroethane. Studies in animals have shown that 1,1-dichloroethane is considerably less toxic than other chlorinated solvents. Rats, guinea pigs, rabbits, and dogs exposed to fairly high air concentrations for six months showed no adverse changes in gross or microscopic observations. Longer-term studies at relatively high doses have shown no

²⁶ USEPA. 1984. Health Effects Assessment for 1,1-Dichloroethane. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC.

CHROMIUM

Chromium is an element that exists primarily in two oxidation states: the trivalent (Cr^{+3} or Cr III) and the hexavalent (Cr^{+6} or Cr VI) form. Cr(III) is the most stable and the most important chemically. It is less soluble (and less toxic) than Cr(VI), and is required in the mammalian diet for normal metabolism. Hexavalent chromium occurs rarely in nature because it is easily reduced to Cr(III) in the presence of organic matter.

Chromium is mainly used in steel and other alloys, but also for refractory brick, metal finishing, pigments, leather tanning, and wood and water treatment. Chromium input into the environment is mainly through stationary combustion sources, primarily fossil fuel combustion, and chemical manufacturing. Background levels in the air of urban and non-urban areas range from 0.005-0.157 $\mu\text{g}/\text{m}^3$. Chromium levels in U.S. waters varies, with concentrations ranging from 0.4-8 ppb. Levels in soil can range from 14 to 70 ppm, but because there is little bioaccumulation potential, food sources are relatively low in chromium. These low levels found in food and water are the primary sources of exposure for the general population. Exposure by inhalation is the primary route of exposure in industrial workers. The levels of chromium essential for maintaining good health are considerably lower than those associated with toxic effects.

The hexavalent form of chromium is a strong oxidant and, in contrast to the trivalent form, is easily absorbed. Once absorbed, it is converted to Cr(III), although Cr(VI) has been implicated as the reactive intermediate responsible for its toxicity. Cr(VI) is very irritating, and short term high-level exposures can result in skin ulcers, nasal septum perforation, and irritation of the gastrointestinal tract.

Little information exists that indicates the toxicity of chronic low level exposure to Cr (III). Ivanovic and Preussman (1975)²³ fed rats chromic oxide in bread for one year and observed body weight and food consumption changes, as well histologic indications of cancer after death. The highest dose level was 1800 mg/kg/day (equivalent to approximately 1400 mg of Cr(III)). No effects were seen at any dose level. Additionally, these authors reported a

²³ Ivankovic, S. and R. Preussman. 1975. Absence of toxic and carcinogenic effects after administrations of high doses of chromic oxide pigment in subacute and long-term feeding experiments in rats. Food Cosmet. Toxicol. 13: 347-351.

3-month study where other signs of toxicity were monitored. No effect were seen except for a decrease in liver and spleen weight in a group given 1400 mg/kg/day. EPA considered 1800 mg/kg/day chromic oxide (1400 mg/kg day Cr(III)) a NOAEL. This dose level was decreased 100 fold to account for interspecies extrapolation and potentially sensitive human populations. The value was decreased an additional 10-fold to account for uncertainty concerning the study used and its application to environmental exposures (where systemic absorption of Cr(III) may be greater). Thus the RfD for Cr(III) is rounded to 1 mg/kg/day.

The oral RfD for Cr(VI) is based on the studies of MacKenzie et al. (1958)²⁴. This group gave rats drinking water containing potassium chromate. No adverse signs were observed, nor were there histopathologic changes in the treated animals. The highest dose was approximately 2.4 mg CrVI/kg/day, and was considered a NOAEL. The EPA lowered this dose 100 fold to account for interspecies extrapolation and potential variation in human sensitivity, and an additional 5 fold to account for the short duration of exposure in the study. Thus, the oral RfD for Cr(VI) is 0.005 mg/kg/day.

Although evidence for the carcinogenicity of Cr(VI) by the oral route is inadequate, a variety of epidemiologic studies of chromium production workers and chrome pigment workers indicate an increased risk of lung cancer. for this reason, EPA has classified Cr(VI) as an A or known human carcinogen by the inhalation route.

The cancer mortality data of Mancuso (1975)²⁵ was used in a linearized multistage model of cancer dose-risk relation, where the offending agent was assumed to be Cr(VI), and that approximately 1/7 of the chromium exposure was Cr(VI). Using this method, the CPF for Cr(VI) is $4.1E+1$ [mg/kg/day]⁻¹.

²⁴ MacKenzie, R.D., R.U. Byerrum, C.F. Decker et al. 1958. Chronic toxicity studies. II. Hexavalent and trivalent chromium administered in drinking water to rats. Am. Med. Assoc. Arch. Ind. Health. 18: 232-234.

²⁵ Mancuso, T.F. 1975. International Conference on Heavy Metals in the Environment, Toronto, Canada, Oct. 27-31. Cited in EPA Health Assessment Document for Chromium EPA 600/8-83-014F.

(20,000 - 40,000 ppm). Excitement due to release of inhibitions is followed by central nervous system depression (i.e. decreased respiration and heart rate, loss of motor activity, decreased temperature regulation). There is a rapid decrease in the liver glycogen (sugar) stores accompanied by an increase in blood glucose levels.

Extended anesthesia or acute CHCl_3 poisoning results in liver damage. Signs of CHCl_3 poisoning include sweetish odor on the breath, cold and clammy skin, and dilated pupils. Symptoms, which develop from the first to the third day after exposure ("delayed" CHCl_3 poisoning) include progressive weakness, prolonged vomiting, delirium, coma, and death. Signs associated with liver dysfunction, such as jaundice and abnormal blood chemistry, accompany these symptoms. Autopsy results will reveal degeneration and necrosis of the liver tissue, which will be marked around the central veins.

Acute oral poisoning can result in death from ingestion of as little as one-third of an ounce (10 ml). Unconsciousness is rapid and death will occur within 12 hours due to respiratory or cardiac failure. The symptoms are very similar to those mentioned above.

Persons chronically exposed to CHCl_3 in the workplace (with average air concentrations in the ppm range) have shown symptoms of tiredness, dull-wittedness, depression, gastrointestinal distress, and frequent and "scalding" urination. It was reported that the air concentrations in the workplace where symptoms were observed may have reached 1163 ppm lasting 1 - 2 minutes, and management observed the workers acting silly and staggering about during the workday. There is also evidence that, after long term exposure, the workers became sensitive to the chemical and would become nauseated after exposure for even a few minutes. Other studies have reported similar symptoms, but, for the majority, there have been no reports of liver dysfunction. Animals exposed chronically by inhalation showed no observed adverse effects at 25 ppm (4 hrs/day, 5 days/week for 6 months). However, male rats exposed to the same level for 7 hours/day exhibited physical changes in the liver and kidney (females were unaffected). Reproductive or developmental anomalies have only been reported in studies that used dose levels that were toxic to the dam.

EPA used a study by Heywood, et al (1979)²⁰ to derive an RfD. Heywood found that 15 mg/kg-day in beagle dogs (12.9 mg/kg-day on a daily basis) 6 days per week for 7.5 years had an effect on the liver. EPA divided this Lowest Observable Adverse Effect Level (LOAEL) by 1000 to arrive at a RfD of 0.01 mg/kg/day.

CHCl₃ is a suspect carcinogen. In animals, it has been shown to induce liver or kidney tumors when given orally. Epidemiological studies of human populations in communities that have been exposed to CHCl₃ in drinking water supplies have found an increased incidence of bladder, colon and rectal cancer. Arguments have been put forth that the studies can only show a weak (but statistically significant) correlation because of the confounding effects of uncontrolled factors, such as smoking, diet, pollution, occupation, and lifestyle. The U.S. EPA summarizes that "there may be a suggestion of an increased risk of certain forms of cancer (bladder, large intestine, and especially rectum) due to exposure to chlorinated drinking water with organic material".

The inhalation potency factor for chloroform is based on a study by The National Cancer Institute (1976)²¹. The linearized multistage model was applied to the NCI data on increased liver tumors in mice and rats given by stomach tube. The data was then subjected to route-to-route extrapolation to derive a potency factor of 0.081 [mg/kg-day]⁻¹. The oral potency factor was derived using the same model procedure to the data of Jorgenson, et al (1985)²² who observed kidney tumors in mice and rats given chloroform in the drinking water. The potency factor is 0.061 [mg/kg-day]⁻¹.

Regulatory Values

There is no specific MCL for chloroform, but this compound is among the trihalomethanes for which an MCL of 10 ppb is in effect.

²⁰ Heywood, R., R.J. Sortwell, P.R.B. Noel, et al. 1979. Safety evaluation of toothpaste containing chloroform. III. Long-term study in beagle dogs. *J. Environ. Pathol. Toxicol.* 835-851.

²¹ NCI (National Cancer Institute). 1976. Report on Carcinogenesis Bioassay of Chloroform. NTIS PB264-018.

²² Jorgenson, T.A., E.F. Meierhenry, C.J. Rushbrook et al. 1985. Carcinogenicity of chloroform in drinking water to male Osborne-Mendel rats and female B6C3F1 mice. *Fund. Appl. Toxicol.* (U.S.A.) 5(4): 760-769.

indefinitely but are markedly less toxic. With the oxide, it appears that the higher the temperature the oxide is formed at, the less toxicity is associated with it. Particle size and surface area also play important roles.

Beryllium has been shown to be carcinogenic in laboratory animals (inhalation route). Studies of cancer incidence in human populations exposed in the workplace are controversial. The EPA has concluded that there is insufficient epidemiological evidence to implicate this metal as a carcinogen in humans, but has, based on the positive findings in animals, classed it as a B2, or probable human carcinogen.

Although EPA places low confidence in epidemiology studies of beryllium, the epidemiologic data of Wagoner, et al (1980)¹⁴ on beryllium workers was used to develop a dose-response curve. A relative risk model was used to determine a cancer potency factor of 8.4 [mg/kg-day]⁻¹ by the inhalation route.

Beryllium has never been shown to cause cancer by the oral route in humans. However, Schroeder and Mitchener (1975)¹⁵ observed tumors in rats given 5 mg/l beryllium in their drinking water. The tumor rate was not significantly elevated, but EPA used the data in a linearized multistage model of cancer dose-response to derive a potency factor of 4.3 [mg/kg-day]⁻¹. This value is extremely uncertain. Schroeder and Mitchener's studies were also used to establish an oral RfD of 5E-3 mg/kg/day.

Regulatory Values

The EPA Office of Drinking Water has set an MCL for Beryllium of 1.00 ug/l and an MCLG of zero. The Ambient Water Quality Criteria for protection of freshwater species (chronic) is 5.3 ug/l.

¹⁴ Wagoner, J.K., P.F. Infante and D.L. Bayliss. 1980. Beryllium: An etiologic agent in the induction of lung cancer, nonneoplastic respiratory disease, and heart disease among industrially exposed workers. *Environ. Res.* 21: 15-34.

¹⁵ Schroeder, H.A. and M. Mitchener. 1975a. Life-term studies in rats: Effects of aluminum, vanium, beryllium and tungsten. *J. Nutr.* 105: 421-427.

2-BUTANONE (Methyl Ethyl Ketone)

Methyl ethyl ketone (MEK) is a colorless liquid with a pleasant, sharp mint-like odor. It is commonly used as a solvent in cements and adhesives, cleaning fluids, flush-off paint stripper, and the manufacture of nitrocellulose and vinyl films. It is also used in the manufacture of drugs and smokeless powders.

There is less information available on MEK than other solvents. Other sources of this chemical in the environment include evaporation from paints and glues, cigarette smoke (500 ppm), and gasoline exhaust (<0.1- 1.0 ppm).

MEK is frequently used as a solvent because of its low toxicity. However, used in combination with other solvents, it may cause greater toxicity than if the other solvent was used alone. A case in point is the deliberate inhalation of glue vapors by "glue sniffers". Inhalation of glue solvent containing 31% n-hexane produced no recorded cases of neuropathy (nerve damage of the extremities). After addition of 11% MEK, cases of neuropathy were reported. These reports ceased when the manufacturer reformulated the solvent mixture. MEK will also potentiate the effect of chloroform on the liver of rats.

In a subchronic (90 day) inhalation study in rats, the NOAEL (no observed adverse effect level) of MEK was determined to be 2500 ppm. However, fetal malformations have been observed after exposure of pregnant rats to very high concentrations (1000-3000 ppm) of MEK (LaBelle and Brieger, 1955)¹⁶. The NOAEL in this study was 235 ppm, seven hours per day (evaluated as 92 mg/kg-day). MEK has not been tested for mutagenic or carcinogenic activity in laboratory animals, nor is it scheduled to be tested by the National Toxicology Program.

EPA divided the NOAEL of LaBelle and Brieger by 1000 to derive an RfD of 0.09 mg/kg/day for the inhalation route. Using route-to-route extrapolation methods, EPA (1989)¹⁷ determined this dose was equivalent to 0.05 mg/kg-day by the oral route.

¹⁶ LaBelle, C.W. and H. Brieger (1955) Vapor toxicity of a composite solvent and its principal components Arch. Ind. Health 12: 623-627.

¹⁷ EPA (1989) Updated Health Effects Assessment for Methyl Ethyl Ketone.

A model of cadmium distribution in the body has been developed by EPA (1985)¹⁸. This model has been used to predict what exposure to cadmium would cause the kidney to contain 200 ug cadmium/g tissue, the maximum level expected to be without toxic effect. The model estimates that intake of .005 mg/kg-day cadmium from water (.01 mg/kg-day, if the cadmium source is food) would produce this tissue level. By applying safety factor of 10, EPA derived an RfD of 0.0005 mg/kg-day (.001 mg/kg-day from food) from this information.

There is limited information from epidemiologic studies that cadmium causes lung cancer in humans, which are supported by observations in experimental animals. Based on this information, EPA rates cadmium a B-1, probable human carcinogen. EPA applied a two stage model of cancer response to the data of Thun, et al (1985)¹⁹ to derive a cancer potency factor of 6.1 [mg/kg-day]⁻¹. This value applies to the inhalation route only, the chemical does not appear to be carcinogenic by other exposure routes.

Regulatory Values

The EPA Office of Drinking Water set an MCL and MCLG for cadmium of 5 ug/l in January, 1991. The Ambient Water Quality Criteria for protection freshwater aquatic species is 11 ug/l.

¹⁸ USEPA. 1985. Drinking Water Criteria Document on Cadmium. Office of Drinking Water, Washington, DC. (Final draft).

¹⁹ Thun, M.J., T.M. Schnorr, A.B. Smith and W.E. Halperin. 1985. Mortality among a cohort of U.S. cadmium production workers: An update. J. Natl. Cancer Inst. 74(2): 325-333.

CHLOROFORM

Chloroform (CHCl_3) is a clear, colorless, volatile liquid with an ether-like odor and a sweet taste. It is slightly soluble in water and miscible with both polar (alcohols) and non-polar (benzene, carbon tetrachloride) compounds.

Chloroform was first used as an anesthetic in 1847, but has since been replaced with other anesthetics with more desirable properties. Today, it is widely used as a solvent and as an intermediate in the production of chlorodifluoromethane (a refrigerant) and, to a lesser degree, plastics and pharmaceuticals. Annual production of chloroform, based on 1981 data, approaches 184 million kilograms. Of this, roughly 7.2, 2.6, and 0.6 million kg is released to the air, water, and soil, respectively. Other, more significant sources of CHCl_3 include pulp and paper mills, drinking and wastewater chlorination, ethylene dichloride manufacture, degradation of trichloroethylene, and automobile exhaust.

Chloroform is ubiquitous in the environment, and natural sources (e.g. tropical oceans) also contribute significant amounts to the atmosphere. On a global scale, the average background concentration is 8 parts per trillion. Generally, urban concentrations are less than 1 part per billion, while rural areas average one hundred times lower.

In addition to inhalation, significant human exposure also occurs from ingestion of food and water. The majority of drinking water supplies in the U.S. are disinfected by chlorination, which generates CHCl_3 . The average concentration of CHCl_3 in drinking water in this country (weighted average) is $41 \mu\text{g/l}$ (ppb); levels have been measured to range from 0.1 to $311 \mu\text{g/l}$. CHCl_3 is also classified as an "indirect" food additive arising from contact of food with packaging materials and as a result of its use as a grain fumigant.

Chloroform is efficiently absorbed from both the respiratory and gastrointestinal systems. The toxic effects seen in humans or experimental animals after exposure to CHCl_3 , mainly depression of the central nervous system and hepatic damage, are similar for both inhalation and ingestion exposures. Only dermal exposures differ, resulting in renal damage with no other symptomology.

The concentration of CHCl_3 required to induce anesthesia in humans is 2 - 3 volumes %

Regulatory Values

There is no MCL or Ambient Water Quality Criteria for MEK currently.

CADMIUM

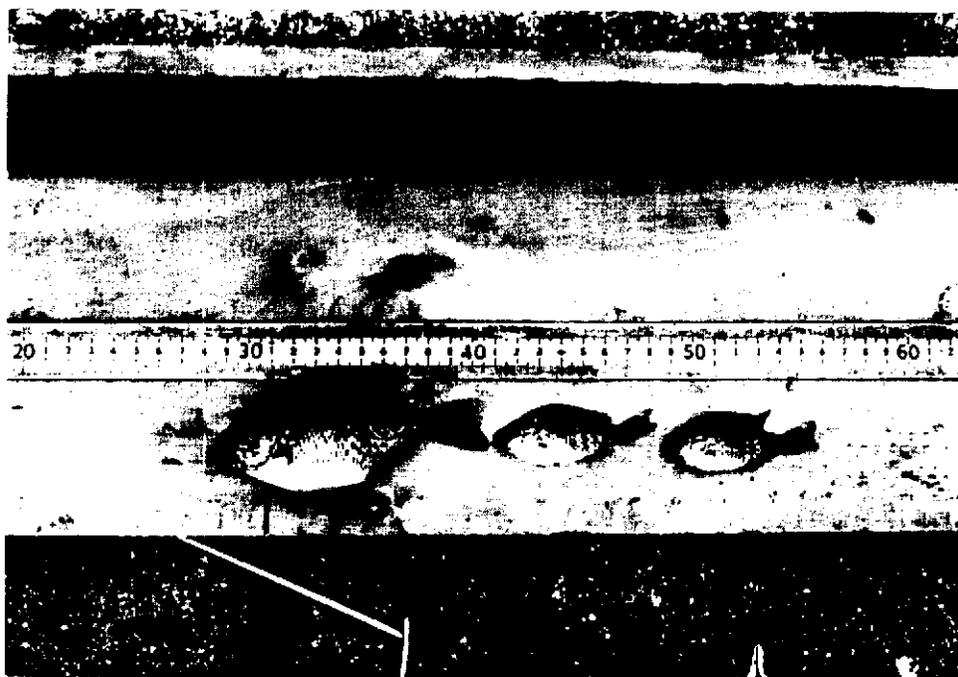
This metal is obtained as a by-product in the smelting of lead and zinc ores. Discovered in 1817, this "modern metal" has recently seen high demand due to its excellent conductivity (electronics), anti-corrosive properties (metal-plating), and bright oxide pigments (paints). Because it is not recycled, this highly toxic metal has become a ubiquitous contaminant of air, water, and soil, especially in urban areas.

The most significant route of exposure for the general population is oral, although less than 10% of ingested cadmium is absorbed. The average daily intake for people in North America and Europe is approximately 18 ug/day. Cadmium levels are high in organ meats and shellfish, which can contain from 100 to 1000 ug/kg. Plants also accumulate cadmium. Grains and cereals contain relatively high cadmium levels, as does tobacco. A person who smokes a pack of cigarettes a day can have 2 - 3 times the average body burden of cadmium. The respiratory absorption of cadmium varies from 15 to 30%. Industrial workers (e.g. smelting, welding) exposed to high air levels of cadmium develop lung damage (fibrosis, emphysema) and prolonged exposures have been associated with lung cancer in both animals and man. Ambient urban air levels are approximately 15 times higher than rural air levels.

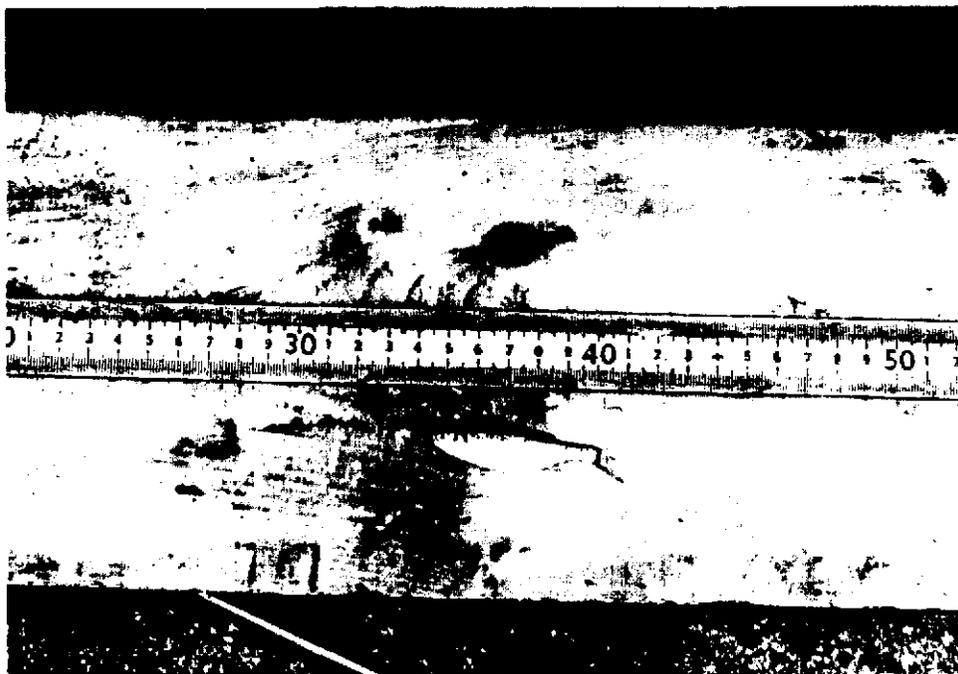
Cadmium is cumulative and has a very long half-life, estimated to be about 25 years in humans. About 50 to 75 percent of the body burden of cadmium is found in the liver and kidneys. These organs detoxify the metal by producing large amounts of a metal-binding protein called metallothionein. Exposure to zinc, copper, and mercury will also stimulate the production of this protein, but cadmium is the most effective and has a very high affinity for it. The kidney will accumulate the highest levels of cadmium and is considered the "critical organ" for both acute and chronic exposure to the metal. In humans, the amount of cadmium in the kidney is proportional to age, and very high exposures are associated with an increased frequency of kidney dysfunction (e.g. β - microglobulinuria, renal stones). Cadmium has also been implicated as an etiological factor in human cardiovascular disease, although the evidence is controversial.

APPENDIX G
Risk Assessment Documentation
Section 3. Photographs

Appendix G

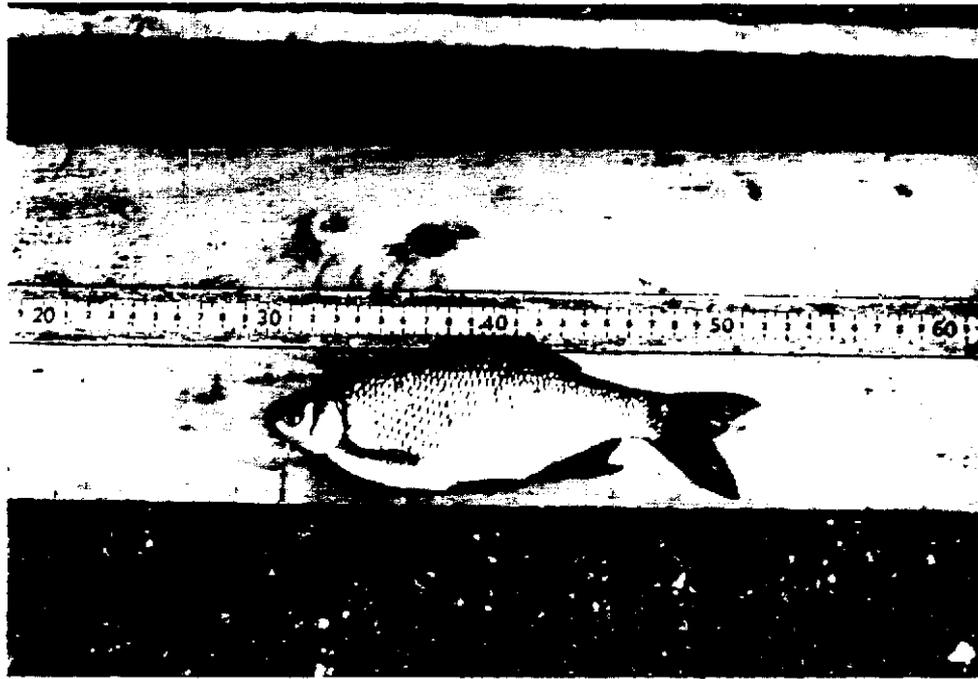


Pumpkinseed - Station 1 (Lower South Pond)



Golden Shiner - Station 1 (Lower South Pond)

Originals in color.

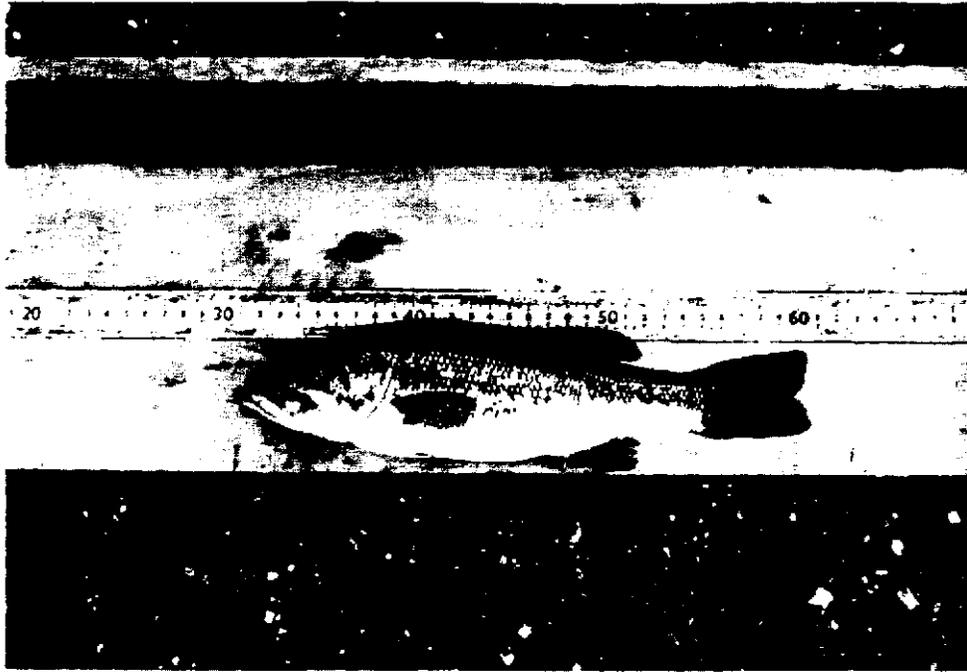


Golden Shiner - Station BS-26 (Phillip's Pond)

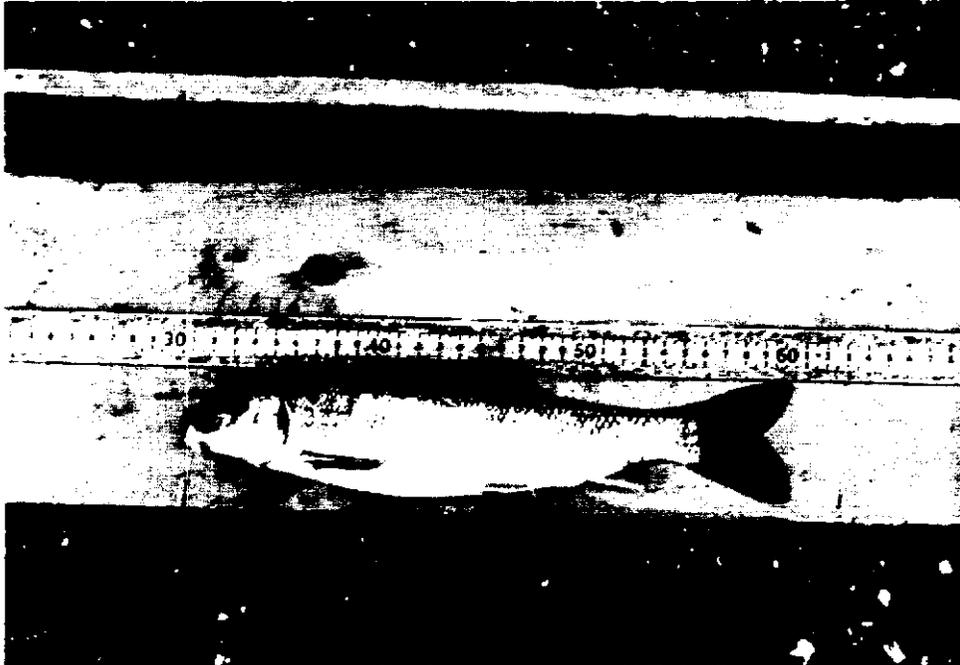


Golden Shiner - Station BS-11 (HBRA)

Originals in color.



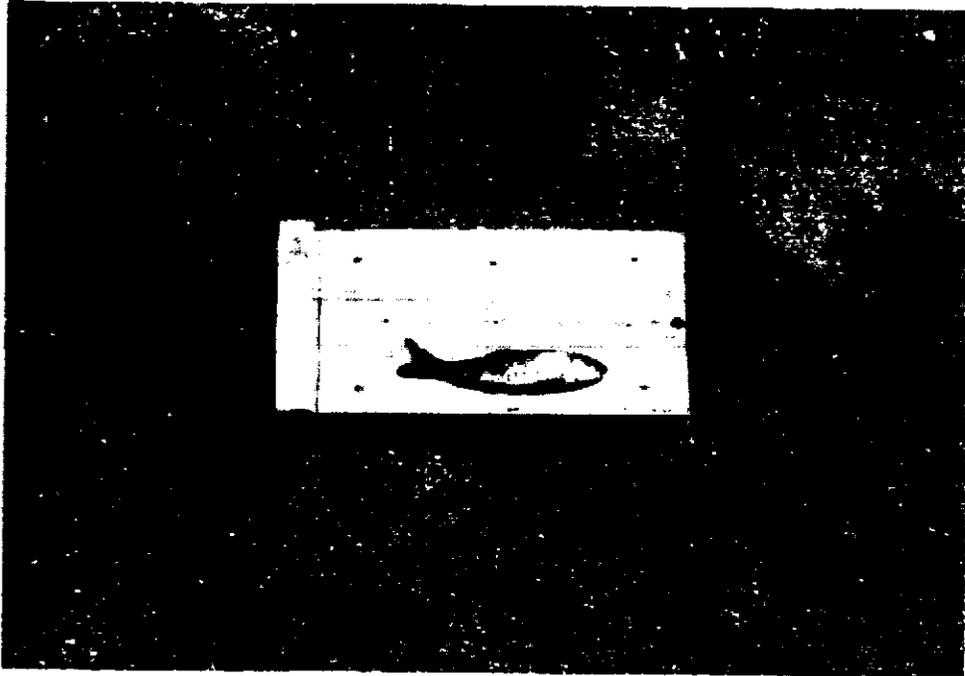
Largemouth Bass - Station BS-26 (Phillip's Pond)



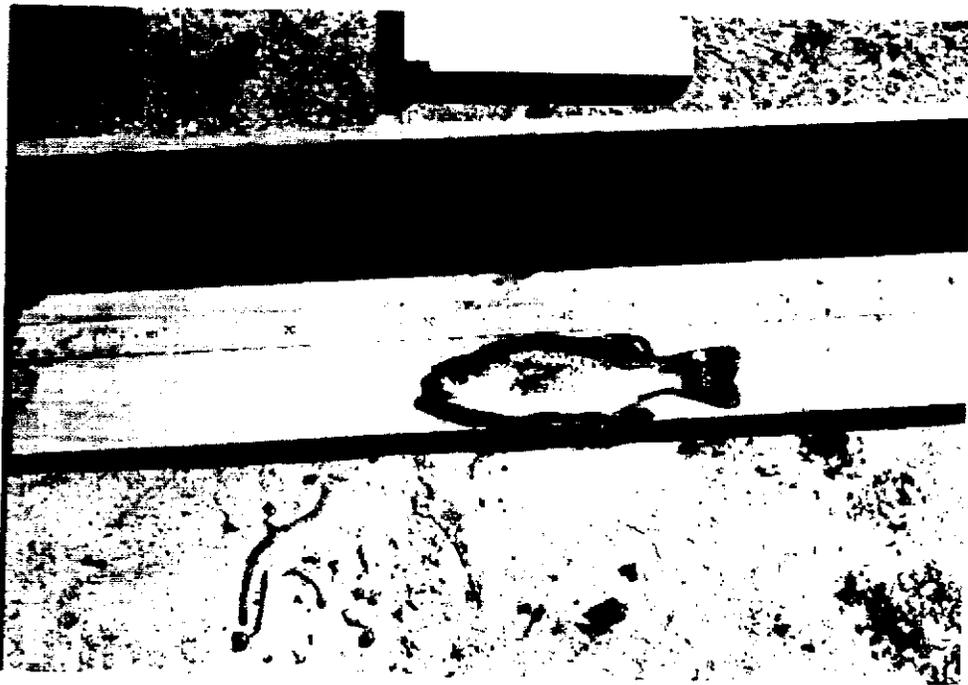
White Sucker - Station BS 26 (Phillip's Pond)

Originals in color.

Appendix G



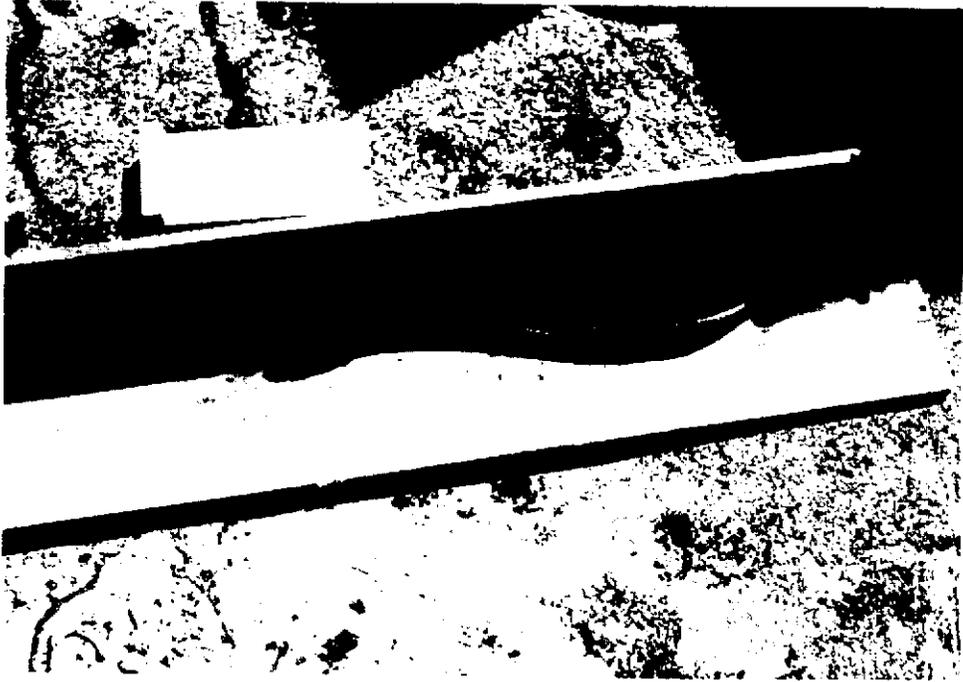
White Sucker - Station BS-14



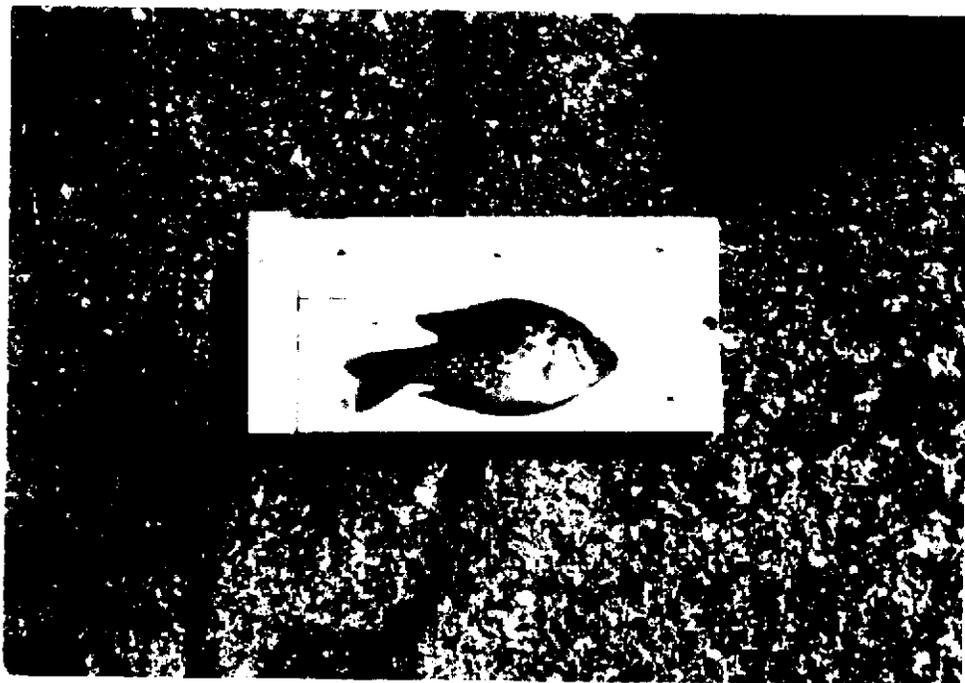
Largemouth Bass - Station BS-14

Originals in color.

Appendix C



American eel - Station BS-14



Pumpkinseed - Station BS-14

Originals in color.