

Appendix F.3

Background Concentrations

Appendix F-3
Raymark OUS Background Data

AOC	Matrix	Species	DEPCODE	Fraction	Parameter	Units	Frequency	Range Of Detects	Range Of Nondetects	Average	Maximum	Location of Maximum
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	1,2,3,4,6,7,8-HPCDD	UG/KG	4/4	0.00726 - 0.34857	-	0.11011	0.34857	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	1,2,3,4,6,7,8-HPCDF	UG/KG	4/4	0.00263 - 0.10316	-	0.043245	0.10316	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	1,2,3,4,7,8-HXCDD	UG/KG	2/4	0.00022 - 0.00622	0.0006 - 0.00984	0.002915	0.00622	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	1,2,3,4,7,8-HXCDF	UG/KG	1/4	0.00078 - 0.00078	0.00068 - 0.00973	0.0024325	0.00078	RM-SD-GM07-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	1,2,3,6,7,8-HXCDD	UG/KG	2/4	0.00033 - 0.01788	0.00075 - 0.00988	0.00585625	0.01788	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	1,2,3,6,7,8-HXCDF	UG/KG	1/4	0.00055 - 0.00055	0.00081 - 0.0068	0.0018375	0.00055	RM-SD-GM02-01
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	1,2,3,7,8,9-HXCDD	UG/KG	1/4	0.00033 - 0.00033	0.00072 - 0.01678	0.003745	0.00033	RM-SD-GM02-01
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	1,2,3,7,8,9-HXCDF	UG/KG	2/4	0.00031 - 0.00755	0.00103 - 0.00644	0.00289875	0.00755	RM-SD-GM08-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	2,3,7,8-TCDF	UG/KG	3/4	0.00097 - 0.00994	0.00053 - 0.00053	0.00418625	0.00994	RM-SD-GM08-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	OCDD	UG/KG	4/4	0.16671 - 3.64659	-	1.6016375	3.64659	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	OCDF	UG/KG	4/4	0.00672 - 0.2442	-	0.115875	0.2442	RM-SD-GM08-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	TOTAL HPCDD	UG/KG	4/4	0.01586 - 0.76351	-	0.2595375	0.76351	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	TOTAL HPCDF	UG/KG	4/4	0.01013 - 0.84704	-	0.23091	0.64704	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	TOTAL HXCDD	UG/KG	4/4	0.00112 - 0.05984	-	0.0254	0.05984	RM-SD-GM08-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	TOTAL HXCDF	UG/KG	4/4	0.00637 - 0.85412	-	0.2633475	0.65412	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	TOTAL PCDF	UG/KG	4/4	0.00616 - 0.87881	-	0.4017375	0.87881	RM-SD-GM08-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	TOTAL TCDD	UG/KG	3/4	0.00048 - 0.00546	0.00013 - 0.00013	0.00277125	0.00546	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	TOTAL TCDF	UG/KG	3/4	0.01123 - 0.72167	0.00399 - 0.00399	0.25400625	0.72167	RM-SD-GM08-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	DIOXI	TOXICITY EQUIVALENCY FACTOR	UG/KG	4/4	0.000461 - 0.01133	-	0.00451775	0.01133	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	ALUMINUM	MG/KG	43/43	926 - 22600	-	12784.32558	22600	SMS-G3
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	ARSENIC	MG/KG	42/43	0.62 - 14.2	1.5 - 1.5	5.836511628	14.2	RM-SD-GM07-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	BARIIUM	MG/KG	42/43	5.3 - 329	4.1 - 4.1	55.13837209	329	EX-91
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	BERYLLIUM	MG/KG	37/43	0.26 - 1.3	0.25 - 0.82	0.694302326	1.3	EW5-G5B
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	BERYLLIUM	MG/KG	37/43	0.26 - 1.3	0.25 - 0.82	0.694302326	1.3	THN-G2
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	CADIUM	MG/KG	6/43	0.43 - 1.4	0.39 - 1.4	0.388139535	1.4	EX-91
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	CALCIUM	MG/KG	43/43	181 - 7420	-	1637.978744	7420	UMC-92
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	CHROMIUM	MG/KG	43/43	6.2 - 107	-	21.04418605	107	RM-SD-GM07-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	COBALT	MG/KG	33/43	1.6 - 14.9	2 - 8.8	6.585116279	14.9	RM-SD-GM08-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	COPPER	MG/KG	42/43	9.2 - 338	11.9 - 11.9	41.06860465	338	RM-SD-GM07-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	IRON	MG/KG	43/43	3110 - 35300	-	16604.65116	35300	RM-SD-GM08-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	LEAD	MG/KG	40/42	3.7 - 344	19.1 - 21.7	81.83095238	344	EX-91
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	MAGNESIUM	MG/KG	43/43	368 - 10400	-	3530.1866047	10400	RM-SD-GM08-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	MANGANESE	MG/KG	43/43	35.8 - 660	-	297.0674419	660	LSSE+125
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	MERCURY	MG/KG	28/43	0.07 - 1.2	0.07 - 0.12	0.158139535	1.2	RM-SD-GM07-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	NICKEL	MG/KG	33/43	4.4 - 40.4	3 - 18.1	13.25465116	40.4	LSSA+00
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	POTASSIUM	MG/KG	27/43	517 - 5020	53.7 - 894	1134.054651	5020	RM-SD-GM08-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	SELENIUM	MG/KG	6/43	0.95 - 3.3	0.31 - 3.4	0.54	3.3	THN-G2
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	SILVER	MG/KG	2/43	0.58 - 3.3	0.31 - 1.9	0.509883721	3.3	GLC004
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	SODIUM	MG/KG	25/38	66.4 - 15000	50 - 168	953.7026316	15000	RM-SD-GM08-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	VANADIUM	MG/KG	42/43	6.5 - 81.9	3.1 - 3.1	34.38255814	81.9	SH-A+00
BKG	SOIL/SEDIMENT/WETLAND	None	a	M	ZINC	MG/KG	43/43	9.8 - 604	-	114.3651163	604	LBP012
BKG	SOIL/SEDIMENT/WETLAND	None	a	OS	ANTHRACENE	UG/KG	1/4	1300 - 1300	430 - 820	577.5	1300	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	OS	BENZO(A)ANTHRACENE	UG/KG	2/4	460 - 7000	430 - 770	2015	7000	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	OS	BENZO(A)PYRENE	UG/KG	1/4	5800 - 5800	430 - 820	1702.5	5800	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	OS	BENZO(B)FLUORANTHENE	UG/KG	3/4	300 - 12000	430 - 430	3291.25	12000	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	OS	BENZO(G,H,I)PERYLENE	UG/KG	1/4	2700 - 2700	430 - 820	827.5	2700	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	OS	BIS(2-ETHYLHEXYL)PHTHALATE	UG/KG	2/4	270 - 1600	430 - 770	617.5	1600	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	OS	CARBAZOLE	UG/KG	1/4	1100 - 1100	430 - 820	527.5	1100	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	OS	CHRYSENE	UG/KG	2/4	450 - 6700	430 - 770	1937.5	6700	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	OS	DIBENZO(A,H)ANTHRACENE	UG/KG	1/4	2000 - 2000	430 - 820	752.5	2000	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	OS	FLUORANTHENE	UG/KG	4/4	23 - 14000	-	3770.75	14000	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	OS	INDENO(1,2,3-CD)PYRENE	UG/KG	1/4	5200 - 5200	430 - 820	1552.5	5200	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	OS	PHENANTHRENE	UG/KG	2/4	300 - 6700	430 - 770	1900	6700	RM-SD-RF01-04

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BKG	SOIL/SEDIMENT/WETLAND	None	a	OS	PYRENE	UG/KG	4/4	22 - 9300	-	2485.5	9300	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	OV	CARBON DISULFIDE	UG/KG	2/4	8 - 31	13 - 18	13.625	31	RM-SD-GM08-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	OV	TOLUENE	UG/KG	1/4	7 - 7	13 - 25	9.375	7	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	4,4'-DDD	UG/KG	3/39	0.28 - 5.8	3.3 - 20	4.478974359	5.8	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	4,4'-DDE	UG/KG	14/38	0.15 - 240	3.3 - 20	15.94315789	240	THG005
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	4,4'-DDT	UG/KG	15/38	0.22 - 400	3.3 - 20	27.77026316	400	THG005
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	ALDRIN	UG/KG	3/40	0.14 - 2.8	1.7 - 10	2.2645	2.8	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	ALPHA-CHLORDANE	UG/KG	12/39	0.05 - 44	1.7 - 9.8	4.535307692	44	NS-E+200
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	AROCLOR, TOTAL	UG/KG	10/41	154 - 810	165.5 - 1005	252.4390244	810	NS-G+300
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	AROCLOR, TOTAL	UG/KG	10/41	154 - 810	165.5 - 1005	252.4390244	810	NS-B+200
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	DELTA-BHC	UG/KG	1/40	1.3 - 1.3	1.7 - 10	2.17125	1.3	THG005
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	DIELDRIN	UG/KG	8/37	2.6 - 190	3.3 - 19	12.92182182	190	NS-E+200
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	ENDOSULFAN I	UG/KG	3/39	22 - 47	1.7 - 9.8	4.284102564	47	NS-E+200
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	ENDOSULFAN II	UG/KG	7/40	0.16 - 6	3.3 - 20	4.34925	6	SH-A+00
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	ENDRIN	UG/KG	4/40	0.12 - 4.5	3.3 - 20	4.412	4.5	HBN-G4
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	ENDRIN ALDEHYDE	UG/KG	3/40	0.2 - 3.7	3.3 - 20	4.21575	3.7	HP-GR7
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	ENDRIN KETONE	UG/KG	4/39	1.8 - 9.5	3.3 - 20	5.071794872	9.5	LSSE+125
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	GAMMA-BHC (LINDANE)	UG/KG	3/40	0.03 - 2.2	1.7 - 10	2.249	2.2	RM-SD-RF01-04
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	GAMMA-CHLORDANE	UG/KG	8/37	0.15 - 13	1.7 - 9.8	2.82027027	13	EWS-G5B
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	HEPTACHLOR	UG/KG	2/39	0.28 - 1	1.7 - 10	2.090512821	1	THG005
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	HEPTACHLOR EPOXIDE	UG/KG	3/39	1.6 - 2.3	1.7 - 10	2.266666667	2.3	LSSB+365
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	METHOXYCHLOR	UG/KG	4/38	4.1 - 18	3.6 - 100	21.79736842	18	CC5
BKG	SOIL/SEDIMENT/WETLAND	None	a	PESTP	TOXAPHENE	UG/KG	2/40	1.4 - 5.7	170 - 1000	221.4275	5.7	HBN-G4
BKG	SW	None	None	M	ALUMINIUM	UG/L	4/8	85.2 - 400	25 - 92	156.36875	400	RM-SW-GM05-02
BKG	SW	None	None	M	ANTIMONY	UG/L	2/8	5.2 - 6.7	5 - 21	4.3625	6.7	RM-SW-GM03-02
BKG	SW	None	None	M	ARSENIC	UG/L	1/8	40.8 - 40.8	7 - 86	14.3125	40.8	RM-SW-GM03-01
BKG	SW	None	None	M	BARIUM	UG/L	8/8	9.1 - 42.3	10.3 - 17.8	17.09375	42.3	RM-SW-GM03-01
BKG	SW	None	None	M	CALCIUM	UG/L	8/8	199000 - 284000	-	219687.5	284000	RM-SW-GM07-04
BKG	SW	None	None	M	CHROMIUM	UG/L	1/8	22.3 - 22.3	5 - 5	4.975	22.3	RM-SW-GM07-04
BKG	SW	None	None	M	COBALT	UG/L	1/8	2.3 - 2.3	2 - 2.5	1.19375	2.3	RM-SW-GM06-02
BKG	SW	None	None	M	COPPER	UG/L	5/8	13.1 - 51.8	3 - 37.4	19.75	51.8	RM-SW-GM05-02
BKG	SW	None	None	M	IRON	UG/L	8/8	502 - 1280	-	698.25	1280	RM-SW-GM03-01
BKG	SW	None	None	M	MAGNESIUM	UG/L	8/8	480000 - 951000	-	691312.5	951000	RM-SW-GM07-04
BKG	SW	None	None	M	MANGANESE	UG/L	8/8	36.8 - 347	-	134.65	347	RM-SW-GM07-04
BKG	SW	None	None	M	MERCURY	UG/L	1/8	0.49 - 0.49	0.2 - 0.2	0.14875	0.49	RM-SW-GM07-04
BKG	SW	None	None	M	POTASSIUM	UG/L	8/8	284000 - 403000	-	344000	403000	RM-SW-GM06-02
BKG	SW	None	None	M	SODIUM	UG/L	8/8	884000 - 9330000	-	6916125	9330000	RM-SW-GM04-02
BKG	SW	None	None	M	THALLIUM	UG/L	1/8	7.8 - 7.8	7 - 104	10.20625	7.8	RM-SW-GM03-02
BKG	SW	None	None	M	VANADIUM	UG/L	3/8	1.55 - 7.9	2 - 2	2.08125	7.9	RM-SW-GM07-04
BKG	SW	None	None	M	ZINC	UG/L	5/7	19.1 - 70.7	4 - 7	34.39285714	70.7	RM-SW-GM03-02
BKG	SW	None	None	OV	ACETONE	UG/L	1/8	14 - 14	10 - 10	6.125	14	RM-SW-GM07-04
BKG	SW	None	None	OV	CARBON DISULFIDE	UG/L	1/8	3 - 3	10 - 10	4.75	3	RM-SW-GM01-01
BKG	SW	None	None	PESTP	ALPHA-BHC	UG/L	1/8	0.0029 - 0.0029	0.05 - 0.05	0.0222375	0.0029	RM-SW-GM03-02
BKG	SW	None	None	PESTP	ALPHA-CHLORDANE	UG/L	1/8	0.0013 - 0.0013	0.05 - 0.05	0.0220375	0.0013	RM-SW-GM06-02
BKG	SW	None	None	PESTP	GAMMA-BHC (LINDANE)	UG/L	1/8	0.013 - 0.013	0.05 - 0.05	0.0235	0.013	RM-SW-GM03-01
BKG	SW	None	None	PESTP	HEPTACHLOR EPOXIDE	UG/L	1/8	0.0015 - 0.0015	0.05 - 0.05	0.0220625	0.0015	RM-SW-GM03-02

ANALYTICAL RESULTS
 DRAFT REMEDIAL INVESTIGATION REPORT
 RAYMARK-FERRY CREEK - OU3
 STRATFORD, CONNECTICUT

Sample Number	RM-SD-GM02-01	RM-SD-GM07-04	RM-SD-GM08-04	RM-SD-RF01-04
Sample Location	GM02	GM07	GM08	RF01
Date Sampled	8/16/94	8/16/95	8/16/95	8/7/95
OC Type	None	None	None	None
MATRIX	SEDIMENT	SEDIMENT	SEDIMENT	SEDIMENT
Filtering	NA	NA	NA	NA
Volatile Organic Compounds (UG/KG)				
1,1,1-TRICHLOROETHANE	13 U	23 U	25 U	18 U
1,1,2,2-TETRACHLOROETHANE	13 U	23 U	25 U	18 U
1,1,2-TRICHLOROETHANE	13 U	23 U	25 U	18 U
1,1-DICHLOROETHANE	13 U	23 U	25 U	18 U
1,1-DICHLOROETHENE	13 U	23 U	25 U	18 U
1,2-DICHLOROETHANE	13 U	23 U	25 U	18 U
1,2-DICHLOROETHENE (TOTAL)	13 U	23 U	25 U	18 U
1,2-DICHLOROPROPANE	13 U	23 U	25 U	18 U
2-BUTANONE	13 U	23 U	25 U	18 U
2-HEXANONE	13 U	23 U	25 U	18 U
4-METHYL-2-PENTANONE	13 U	23 U	25 U	18 U
ACETONE	13 U	81 UJ	110 UJ	38 U
BENZENE	13 U	23 U	25 U	18 U
BROMODICHLOROMETHANE	13 U	23 U	25 U	18 U
BROMOFORM	13 U	23 U	25 U	18 U
BROMOMETHANE	13 UJ	23 UJ	25 UJ	18 U
CARBON DISULFIDE	13 U	8 J	31	18 U
CARBON TETRACHLORIDE	13 U	23 U	25 U	18 U
CHLOROENZENE	13 U	23 U	25 U	18 U
CHLOROETHANE	13 U	23 U	25 U	18 U
CHLOROFORM	13 U	23 U	25 U	18 U
CHLOROMETHANE	13 U	23 U	25 U	18 U
CIS-1,3-DICHLOROPROPENE	13 U	23 U	25 U	18 U
DIBROMOCHLOROMETHANE	13 U	23 U	25 U	18 U
ETHYLBENZENE	13 U	23 U	25 U	18 U
METHYLENE CHLORIDE	13 U	23 U	25 U	18 U
STYRENE	13 U	23 U	25 U	18 U
TETRACHLOROETHENE	13 U	23 U	25 U	18 U
TOLUENE	13 U	23 U	25 U	7 J
TRANS-1,3-DICHLOROPROPENE	13 U	23 U	25 U	18 U
TRICHLOROETHENE	13 U	23 U	25 U	18 U
VINYL CHLORIDE	13 U	23 U	25 U	18 U
XYLENES, TOTAL	13 U	23 U	25 U	18 U

ANALYTICAL RESULTS
 DRAFT REMEDIAL INVESTIGATION REPORT
 RAYMARK-FERRY CREEK - OU3
 STRATFORD, CONNECTICUT

Sample Number	RM-SD-GM02-01	RM-SD-GM07-04	RM-SD-GM08-04	RM-SD-RF01-04
Sample Location	GM02	GM07	GM08	RF01
Date Sampled	8/16/94	8/16/95	8/16/95	8/7/95
OC Type	None	None	None	None
MATRIX	SEDIMENT	SEDIMENT	SEDIMENT	SEDIMENT
Filtering	NA	NA	NA	NA
Semivolatile Organic Compounds (UG/KG)				
1,2,4-TRICHLOROBENZENE	430 U	770 U	820 U	2900 UJ
1,2-DICHLOROBENZENE	430 U	770 U	820 U	2900 UJ
1,3-DICHLOROBENZENE	430 U	770 U	820 U	2900 UJ
1,4-DICHLOROBENZENE	430 U	770 U	820 U	2900 UJ
2,2-OXYBIS(1-CHLOROPROPANE)	430 UJ	770 U	820 U	2900 UJ
2,4,5-TRICHLOROPHENOL	1000 U	1900 U	2000 U	7100 UJ
2,4,6-TRICHLOROPHENOL	430 U	770 U	820 U	2900 UJ
2,4-DICHLOROPHENOL	430 U	770 U	820 U	2900 UJ
2,4-DIMETHYLPHENOL	430 U	770 U	820 U	2900 UJ
2,4-DINITROPHENOL	1000 U	1900 U	2000 U	7100 UJ
2,4-DINITROTOLUENE	430 U	770 U	820 U	2900 UJ
2,6-DINITROTOLUENE	430 U	770 U	820 U	2900 UJ
2-CHLORONAPHTHALENE	430 U	770 U	820 U	2900 UJ
2-CHLOROPHENOL	430 U	770 U	820 U	2900 UJ
2-METHYLNAPHTHALENE	430 U	770 U	820 U	2900 UJ
2-METHYLPHENOL	430 U	770 U	820 U	2900 UJ
2-NITROANILINE	1000 U	1900 U	2000 U	7100 UJ
2-NITROPHENOL	430 U	770 U	820 U	2900 UJ
3,3'-DICHLOROBENZIDINE	430 U	770 U	820 U	2900 UJ
3-NITROANILINE	1000 U	1900 UJ	2000 UJ	7100 UJ
4,6-DINITRO-2-METHYLPHENOL	1000 U	1900 U	2000 U	7100 UJ
4-BROMOPHENYL PHENYL ETHER	430 U	770 U	820 U	2900 UJ
4-CHLORO-3-METHYLPHENOL	430 U	770 U	820 U	2900 UJ
4-CHLOROANILINE	430 U	770 U	820 U	2900 UJ
4-CHLOROPHENYL PHENYL ETHER	430 U	770 U	820 U	2900 UJ
4-METHYLPHENOL	430 U	770 U	820 U	2900 UJ
4-NITROANILINE	1000 U	1900 U	2000 U	7100 UJ
4-NITROPHENOL	1000 U	1900 U	2000 U	7100 UJ
ACENAPHTHENE	430 U	770 U	820 U	2900 UJ
ACENAPHTHYLENE	430 U	770 U	820 U	2900 UJ
ANTHRACENE	430 U	770 U	820 U	1300 J
BENZO(A)ANTHRACENE	430 U	770 U	460 J	7000 J
BENZO(A)PYRENE	430 U	770 U	820 U	5800 J
BENZO(B)FLUORANTHENE	430 U	300 J	650 J	12000 J
BENZO(G,H,I)PERYLENE	430 U	770 U	820 U	2700 J
BENZO(K)FLUORANTHENE	430 U	770 U	820 U	2900 UJ
BIS(2-CHLOROETHOXY)METHANE	430 U	770 U	820 U	2900 UJ
BIS(2-CHLOROETHYL)ETHER	430 U	770 U	820 U	2900 UJ
BIS(2-ETHYLHEXYL)PHTHALATE	430 U	770 U	270 J	1600 J
BUTYLBENZYL PHTHALATE	430 U	770 U	820 U	2900 UJ
CARBAZOLE	430 U	770 U	820 U	1100 J
CHRYSENE	430 U	770 U	450 J	6700 J
DI-N-BUTYL PHTHALATE	430 U	770 U	820 U	2900 UJ
DI-N-OCTYL PHTHALATE	430 U	770 U	820 U	2900 UJ
DIBENZO(A,H)ANTHRACENE	430 U	770 U	820 U	2000 J

ANALYTICAL RESULTS
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 RAYMARK-FERRY CREEK - OU3
 STRATFORD, CONNECTICUT

Sample Number	RM-SD-GM02-01	RM-SD-GM07-04	RM-SD-GM08-04	RM-SD-RF01-04
Sample Location	GM02	GM07	GM08	RF01
Date Sampled	8/16/94	8/16/95	8/16/95	8/7/95
OC Type	None	None	None	None
MATRIX	SEDIMENT	SEDIMENT	SEDIMENT	SEDIMENT
Filtering	NA	NA	NA	NA
DIBENZOFURAN	430 U	770 U	820 U	2900 UJ
DIETHYL PHTHALATE	430 U	770 U	820 U	2900 UJ
DIMETHYL PHTHALATE	430 U	770 U	820 U	2900 UJ
FLUORANTHENE	23 J	320 J	740 J	14000 J
FLUORENE	430 U	770 U	820 U	2900 UJ
HEXACHLOROBENZENE	430 U	770 U	820 U	2900 UJ
HEXACHLOROBUTADIENE	430 U	770 U	820 U	2900 UJ
HEXACHLOROCYCLOPENTADIENE	430 U	770 U	820 U	2900 UJ
HEXACHLOROETHANE	430 U	770 U	820 U	2900 UJ
INDENO(1,2,3-CD)PYRENE	430 U	770 U	820 U	2900 UJ
ISOPHORONE	430 U	770 U	820 U	5200 J
N-NITROSO-DI-N-PROPYLAMINE	430 U	770 U	820 U	2900 UJ
N-NITROSODIPHENYLAMINE	430 U	770 U	820 U	2900 UJ
NAPHTHALENE	430 U	770 U	820 U	2900 UJ
NITROBENZENE	430 U	770 U	820 U	2900 UJ
PENTACHLOROPHENOL	1000 U	1900 U	2000 U	7100 UJ
PHENANTHRENE	430 U	770 U	300 J	6700 J
PHENOL	430 U	770 U	820 U	2900 UJ
PYRENE	22 J	280 J	380 J	6300 J
Pesticides/PCBs (UG/KG)				
4,4'-DDD	3.3 U	0.28 J	1.5 J	5.8
4,4'-DDE	3.3 U	0.15 J	0.54 J	3.6 U
4,4'-DDT	3.3 U	3.3 U	0.22 J	4.4
ALDRIN	1.7 U	0.14 J	0.19 J	2.6
ALPHA-BHC	1.7 U	1.7 U	1.7 U	6.1 U
ALPHA-CHLORDANE	0.077 J	0.05 J	0.15 J	1.8 U
AROCLOR, TOTAL	165.5 U	165.5 U	165.5 U	180 U
AROCLOR-1016	33 U	33 U	33 U	36 U
AROCLOR-1221	67 U	67 U	67 U	72 U
AROCLOR-1232	33 U	33 U	33 U	36 U
AROCLOR-1242	33 U	33 U	33 U	36 U
AROCLOR-1248	33 U	33 U	33 U	36 U
AROCLOR-1254	33 U	33 U	33 U	36 U
AROCLOR-1260	33 U	33 U	33 U	36 U
AROCLOR-1262	33 U	33 U	33 U	36 U
AROCLOR-1268	33 U	33 U	33 U	36 U
BETA-BHC	1.7 U	1.7 U	1.7 U	1.8 U
DELTA-BHC	1.7 U	1.7 U	1.7 U	1.8 U
DIELDRIN	3.3 U	3.3 U	3.3 U	3.6 U
ENDOSULFAN I	1.7 U	1.7 U	1.7 U	1.8 U
ENDOSULFAN II	0.16 J	0.31 J	3.3 U	3.6 U
ENDOSULFAN SULFATE	3.3 U	3.3 U	3.3 U	3.6 U
ENDRIN	0.12 J	3.3 U	0.26 J	2.7 J
ENDRIN ALDEHYDE	0.53 J	3.3 U	0.2 J	4.3 U
ENDRIN KETONE	3.3 U	3.3 U	3.3 U	3.6 U
GAMMA-BHC (LINDANE)	1.7 U	0.08 J	0.03 J	2.2

ANALYTICAL RESULTS
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 RAYMARK-FERRY CREEK - OJ3
 STRATFORD, CONNECTICUT

Sample Number	RM-SD-GM02-01	RM-SD-GM07-04	RM-SD-GM08-04	RM-SD-RF01-04
Sample Location	GM02	GM07	GM08	RF01
Date Sampled	8/16/94	8/16/95	8/16/95	8/7/95
QC Type	None	None	None	None
MATRIX	SEDIMENT	SEDIMENT	SEDIMENT	SEDIMENT
Filtering	NA	NA	NA	NA
GAMMA-CHLORDANE	1.7 U	1.7 U	0.15 J	8.3
HEPTACHLOR	1.7 U	1.7 U	1.7 U	0.28 J
HEPTACHLOR EPOXIDE	1.7 U	1.7 U	1.7 U	1.9
METHOXYCHLOR	17 U	17 U	17 U	3.6 U
TOXAPHENE	170 U	170 U	170 U	180 U
Dioxin (UG/KG)				
1,2,3,4,6,7,8-HPCDD	0.00726 J	0.00849 J	0.07612 J	0.34857
1,2,3,4,6,7,8-HPCDF	0.00409 J	0.00263 J	0.0631 J	0.10318
1,2,3,4,7,8,9-HPCDF	0.00034 U	0.00218 UJ	0.01215 UJ	0.01778 U
1,2,3,4,7,8-HXCDD	0.00022 J	0.0006 UJ	0.00984 UJ	0.00622 J
1,2,3,4,7,8-HXCDF	0.00066 UJ	0.00078 J	0.00973 UJ	0.00751 UJ
1,2,3,6,7,8-HXCDD	0.00033 J	0.00075 UJ	0.00968 UJ	0.01788
1,2,3,6,7,8-HXCDF	0.00055 J	0.00081 UJ	0.00599 UJ	0.0068 U
1,2,3,7,8,9-HXCDD	0.00033 J	0.00072 UJ	0.0118 UJ	0.01678 UJ
1,2,3,7,8,9-HXCDF	0.00031 J	0.00103 UJ	0.00755 J	0.00844 U
1,2,3,7,8-PCDD	0.00042 U	0.00102 UJ	0.00309 UJ	0.00605 U
1,2,3,7,8-PCDF	0.00038 UJ	0.00188 UJ	0.00579 UJ	0.00647 U
2,3,4,6,7,8-HXCDF	0.00046 U	0.0014 UJ	0.00533 UJ	0.0108 U
2,3,4,7,8-PCDF	0.0002 U	0.00171 UJ	0.00567 UJ	0.00629 U
2,3,7,8-TCDD	0.00013 U	0.00034 UJ	0.00128 UJ	0.00123 U
2,3,7,8-TCDF	0.00053 UJ	0.00097 J	0.00994 J	0.00557 J
OCDD	0.16671 J	0.25361 J	2.33964 J	3.64659
OCDF	0.00872 J	0.00977 J	0.2442 J	0.20281 J
TOTAL HPCDD	0.01589 J	0.02729 J	0.23146 J	0.78351 J
TOTAL HPCDF	0.01013 J	0.01564 J	0.25083 J	0.64704 J
TOTAL HXCDD	0.00112 J	0.00864 J	0.05964 J	0.0322 J
TOTAL HXCDF	0.00779 J	0.00637 J	0.38511 J	0.65412 J
TOTAL PCDD	0.00042 UJ	0.00102 UJ	0.00309 UJ	0.00605 UJ
TOTAL PCDF	0.00616 J	0.00904 J	0.87881 J	0.71294 J
TOTAL TCDD	0.00013 UJ	0.00048 J	0.00508 J	0.00548 J
TOTAL TCDF	0.00399 UJ	0.01123 J	0.72187 J	0.28113 J
TOXICITY EQUIVALENCY FACTOR	0.000461 J	0.00055 J	0.00573 J	0.01133 J
Metals (MG/KG)				
ALUMINUM	2950	18400	19000	5590
ANTIMONY	1.2 U	6.3 UJ	8.8 UJ	5.1 UJ
ARSENIC	1.5 U	14.2	12.5	2.2
BARIUM	4.1 U	51.2 J	50.1 J	26.4
BERYLLIUM	0.25 U	0.8	0.58 J	0.31
CADMIUM	0.5 U	0.65 UJ	0.64 UJ	0.66 U
CALCIUM	898	2580 J	2650 J	2200 J
CHROMIUM	7.8 J	107 J	89.2 J	39
COBALT	1.6	12.5	14.8	5.7
COPPER	11 J	338	194	102 J
IRON	4940	33900	35300	14100
LEAD	8.1	91.8 J	48.4 J	141
MAGNESIUM	1210	9920	10400	3460

ANALYTICAL RESULTS
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 RAYMARK-FERRY CREEK - OU3
 STRATFORD, CONNECTICUT

Sample Number	RM-SD-GM02-01	RM-SD-GM07-04	RM-SD-GM08-04	RM-SD-RF01-04
Sample Location	GM02	GM07	GM08	RF01
Date Sampled	8/16/94	8/16/95	8/16/95	8/7/95
QC Type	None	None	None	None
MATRIX	SEDIMENT	SEDIMENT	SEDIMENT	SEDIMENT
Filtering	NA	NA	NA	NA
MANGANESE	43.5	354	321	106
MERCURY	0.12 U	1.2	1.1	0.13
NICKEL	4.4	28.8 J	33.9 J	14.7
POTASSIUM	420 U	4920	5020	1130
SELENIUM	0.99 UJ	2.2 UJ	3.4 U	0.94 U
SILVER	0.74 UJ	1.2 U	1.3 U	1 U
SODIUM	2070	14400	15000	1790
THALLIUM	1.7 U	2.4 U	2.8 U	1.9 U
VANADIUM	8.3 J	55	56.2	24.7 J
ZINC	24.1 J	183	192	158

U - Not detected; UJ - Detection limit approximate; J - Quantitation approximate;
 * - From dilution analysis; R - Rejected; NA - Not Analyzed

ANALYTICAL RESULTS
 DRAFT REMEDIAL INVESTIGATION REPORT
 RAYMARK FERRY CREEK - OU3
 STRATFORD, CONNECTICUT

Sample Number	AHP008	BES002	BESB*300	BESD*300	BS-D*00	BS-G7	BSC*400	CC5	CF-A*00	CF-B*480
Sample Locallon	AHP008	BES002	BESB*300	BESD*300	BS-D*00	BS-G7	BSC*400	CC5	CF-A*00	CF-B*480
Date Sampled										
QC Type	None	None	None	None	Field Dup. (3002)	None	Field Dup. (3003)	None	None	None
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL
Filering	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Pesticides/PCBs (UG/RG)										
4,4'-DDD	3.4 UJ	21 UJ	3.5 UJ	3.5 UJ	6.8 U	220 J	2.3	3.5 UJ	21 U	3.8 U
4,4'-DDE	110 J	11 J	14 J	19 J		NA 230	10	3.5 UJ	6.8 J	10
4,4'-DDT	49 J		12 J	18 J		R 200	7.5	3.5 UJ	R	9.1 J
ALDRIN	1.7 U	11 UJ	1.8 UJ	1.8 UJ	3.5 U	1.7 U	1.8 U	1.8 UJ	11 UJ	2 UJ
ALPHA-BHC	1.7 U	11 UJ	1.8 UJ	1.8 UJ	3.5 U	1.7 U	1.8 U	1.8 UJ	11 U	2 U
ALPHA-CHLORDANE	1.7 UJ		1.8 UJ	1.8 UJ		2.7 J		1.8 UJ	940 *	2 U
AROCLOR_TOTAL	156	1215	151.5	228	366	191	192	175.5 U	945	193.5
AROCLOR-1016	34 U	420 UJ	35 UJ	35 UJ	68 U	34 U	34 U	35 UJ	210 U	38 U
AROCLOR-1221	68 U	210 UJ	71 UJ	72 UJ	140 U	68 U	70 U	71 UJ	420 U	77 U
AROCLOR-1232	34 U	210 UJ	35 UJ	35 UJ	68 U	34 U	34 U	35 UJ	210 U	38 U
AROCLOR-1242	34 U	210 UJ	35 UJ	35 UJ	68 U	34 U	34 U	35 UJ	210 U	38 U
AROCLOR-1248	34 U	210 UJ	35 UJ	35 UJ	68 U	34 U	34 U	35 UJ	210 U	38 U
AROCLOR-1254	34 U	210 UJ	35 UJ	35 UJ	68 U	34 U	34 U	35 UJ	210 U	38 U
AROCLOR-1260	34 U	210 UJ	35 UJ	35 UJ	68 U	34 U	34 U	35 UJ	210 U	38 U
AROCLOR-1262	R	270 J	R	87 J	78	38 J	38	35 UJ	R	22 J
AROCLOR-1268	20 J	210 UJ	11 J	R	68 U	34 U	34 U	35 UJ	210 U	38 U
BETA-BHC	1.7 U	11 UJ	1.8 UJ	0.56 J	3.5 U	1.7 U	1.8 U		R	11 U
DELTA-BHC	1.7 UJ	1.7 J	1.8 UJ	1.8 UJ	3.5 U	1.7 U	1.8 U			2 U
DIELDRIN	3.4 UJ	21 UJ	3.5 UJ	3.5 UJ	3.5 U	NA 3.4 UJ	3.4 U	3.5 UJ	4.4 J	3.8 UJ
ENDOSULFAN I	1.7 U	11 UJ	1.8 UJ	1.8 UJ	3.5 U	NA	1.8 U	1.8 UJ	11 U	2 U
ENDOSULFAN II	3.4 U	21 UJ	0.75 J	0.86 J	NA	3.4 U	3.4 U	3.5 UJ	21 U	0.78 J
ENDOSULFAN SULFATE	3.4 U	12 J	3.5 UJ	3.5 UJ	6.8 U	3.4 U	3.4 U	3.5 UJ	21 U	3.8 U
ENDRIN	3.4 U	13 J	3.5 UJ	3.5 UJ	3.5 U	3.4 U	3.4 U	3.5 UJ	13 J	3.8 UJ
ENDRIN ALDEHYDE	3.4 U	11 UJ	3.5 UJ	3.5 UJ	NA	3.4 U	3.4 U	3.5 UJ	21 U	3.8 U
ENDRIN KETONE	3.4 UJ	21 UJ	3.5 UJ	3.5 UJ	7.5	3.4 U	3.4 U	3.5 UJ	21 U	0.86 J
GAMMA-BHC (LINDANE)	1.7 U	11 UJ	1.8 UJ	1.8 UJ	3.5 U	1.7 U	1.8 U	1.8 UJ	11 UJ	2 UJ
GAMMA-CHLORDANE	1.7 UJ	1100 UJ	1.8 UJ	1.8 UJ		NA	1.8 U	1.8 UJ	R	2 U
HEPTACHLOR	1.7 UJ	R	0.36 J	1.8 UJ	3.5 U	1.7 U	1.8 U	1.8 UJ	5.2 J	0.24 J
HEPTACHLOR EPOXIDE	1.7 U	11 UJ	1.8 UJ	1.8 UJ	3.5 U	1.7 U	1.8 U	1.8 UJ	R	2 U
METHOXYCHLOR	R	16 J	2.6 J	3 J	3.5 U	1.7 U	1.8 U	1.8 UJ	110 U	2.1 J
TOXAPHENE	170 U	270 UJ	180 UJ	180 UJ	350 U	170 U	180 U	180 UJ	1100 U	200 U

ANALYTICAL RESULTS
 DRAFT REMEDIAL INVESTIGATION REPORT
 RAYMARK-FERRY CREEK - 0UJ
 STRATFORD, CONNECTICUT

Sample Number	AHP008	BES002	BESB+300	BESD+300	BS-D+00	BS-G7	BSC+400	CC5	CF-A+00	CF-B+480
Sample Location	AHP008	BES002	BESB+300	BESD+300	BS-D+00	BS-G7	BSC+400	CC5	CF-A+00	CF-B+480
Date Sampled										
QC Type	None	None	None	None	Field Dup. (3002)	None	Field Dup. (3003)	None	None	None
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL
Filtering	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Metals (MG/KG)										
ALUMINUM	9900 J	11300 J	9480 J	8910 J	11100 J	9750 J	11900 J	926 J	NA	17900 J
ANTIMONY	5.6 UJ	6.8 UJ	6 UJ	5.9 UJ	6.6 UJ	6.6 UJ	6.6 U	5.6 UJ	NA	6.3 UJ
ARSENIC	15.3 J	6.1 J	4 J	4 J	5.4 J	1.4 J	5.3 J	3.8 J	NA	3.7 J
BARIUM	34.7 J	38.5 J	32.4 J	33.1 J	40.1 J	44.8 J	38.6 J	5.3 J	NA	63.7 J
BERYLLIUM	0.76 U	0.43 U	0.53 U	0.42 U	0.71 U	0.46 U	0.64 U	0.35 U	NA	1.4 U
CADMIUM	0.63 J	0.94 U	0.42 UJ	0.5 UJ	0.84 U	0.64 U	0.64 U	0.39 UJ	NA	0.44 U
CALCIUM	897 J	1130 J	894 J	536 J	2720 J	1450 J	1400 J	308 J	NA	28100 J
CHROMIUM	23.4 J	15.6 J	10.7 J	8.8 J	11.6 J	7.7 J	13.1 J	9 J	NA	20.4 J
COBALT	6.5 J	4.2 J	5.3 J	7.5 J	5.5 J	4 J	7 J	2 U	NA	9.4 J
COPPER	63.7 J	39.7 J	15.1 J	16.3 J	21 J	12.4 J	24.9 J	9.7 J	NA	34.3 J
IRON	12700 J	9150 J	11100 J	9050 J	16300 J	8520 J	16400 J	3110 J	NA	19500 J
LEAD	132 J	158 J	38.2 J	49.5 J	27.5 J	17 J	34.4 J	5.4 J	NA	67.3 J
MAGNESIUM	2780 J	1710 J	2030 J	1000 J	2390 J	2330 J	3160 J	368 J	NA	10400 J
MANGANESE	234 J	126 J	196 J	146 J	247 J	264 J	281 J	35.8 J	NA	438 J
MERCURY	0.26 U	0.14 J	0.08 J	0.12 J	0.11 J	0.1 UJ	0.12 UJ	0.08 UJ	NA	0.13 J
NICKEL	12 J	17.1 U	10.9 U	8.8 U	13.8 J	7.6 J	13.6 J	3 U	NA	15.2 J
POTASSIUM	884 U	413 UJ	265 UJ	89.3 UJ	1120 UJ	858 UJ	1280 UJ	53.7 UJ	NA	983 UJ
SELENIUM	0.36 J	0.48 J	0.33 UJ	0.34 UJ	0.69 UJ	0.4 UJ	0.4 UJ	0.33 UJ	NA	3.6 UJ
SILVER	0.31 U	0.39 U	0.33 U	0.33 U	R	0.72 UJ	0.72 U	0.31 U	NA	0.35 U
SODIUM	111 U	187 U	89.6 U	84.2 U	338 UJ	323 U	284 U	69.2 U	NA	109 U
THALLIUM	0.4 UJ	0.19 UJ	0.16 UJ	0.17 UJ	0.47 J	0.23 UJ	0.5 J	0.17 UJ	NA	0.45 U
VANADIUM	36.9 J	37.1 J	23.2 J	23.3 J	23.4 J	20.9 J	29.9 J	3.1 UJ	NA	46.2 J
ZINC	67.9 J	173 J	65 J	40.5 J	136 UJ	72.7 J	91.7 J	8.8 J	NA	114 J

U - Not detected; UJ - Detection limit approximate; J - Quantitation approximate;
 * - From dilution analysis; R - Rejected; NA - Not Analyzed

ANALYTICAL IN SIK 15
 DRAFT REMEDIAL INVESTIGATION REPORT
 RAYMARK-FERRY CREEK - 01/3
 STRATFORD, CONNECTICUT

Sample Number	CF-G8	CS-B*00	CS-D*300	EWS-G5A	EWS-G5B	EWS-G7	EX-01	FLS-A*250	FLS-G1	FLS-G2	FS-A*150
Sample Location	CF-G8	CS-B*00	CS-D*300	EWS-G5	EWS-G5	EWS-G7	EX-01	FLS-A*250	FLS-G1	FLS-G2	FS-A*150
Date Sampled											
QC Type	None	None	None			None	None	None	None	Field Dup. (3004)	None
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL
Filtering	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Pesticides/PCBs (UG/KG)											
4,4'-DDO	8.2 U	3.6 U	3.7 U	18 U	18 U	3.4 U	4.1 U	6.9 U	3.4 U	3.4 U	18 U
4,4'-DDE	12 J	2.8 J	5.8	18 U	18 U	3.4 U	5.9 J	4.9 J	3.4 U	3.4 U	18 U
4,4'-DDT	3.8 J	7 J	12 J	18 U	18 U	2.6 J	7.8 J	6.9 U	3.4 U	2	18 U
ALDRIN	2.9 J	1.8 U	1.9 U	9 U	9.2 U	1.8 U	2.1 U	3.5 U	1.8 U	1.8 U	9.3
ALPHA-BHC	4.2 UJ	1.8 U	1.9 U	9 U	9.2 U	1.8 U	2.1 U	3.5 U	1.8 U	1.8 U	9.3
ALPHA-CHLORDANE	14 J	1.8 UJ	1.3 J	21	20	1.8 U	2.1 U	1.8 J	1.8 U	2.8	9.3
AROCLOR TOTAL	373	180.8 U	269	900 U	900 U	170.5 U	208 U	348 U	171 U	171 U	905
AROCLOR-1016	82 U	36 U	37 U	180 U	180 U	34 U	41 U	69 U	34 U	34 U	180
AROCLOR-1221	170 U	73 U	74 U	360 U	360 U	69 U	84 U	140 U	70 U	70 U	370
AROCLOR-1232	82 U	36 U	37 U	180 U	180 U	34 U	41 U	69 U	34 U	34 U	180
AROCLOR-1242	82 U	36 U	37 U	180 U	180 U	34 U	41 U	69 U	34 U	34 U	180
AROCLOR-1248	82 U	36 U	37 U	180 U	180 U	34 U	41 U	69 U	34 U	34 U	180
AROCLOR-1254	82 U	36 U	37 U	180 U	180 UJ	34 U	41 U	69 U	34 U	34 U	180
AROCLOR-1260	82 U	36 U	37 U	180 U	180 UJ	34 U	41 U	69 U	34 U	34 U	180
AROCLOR-1262	R	36 U	88 J	180 U	180 UJ	34 U	41 U	69 U	34 U	34 U	180
AROCLOR-1268	82 U	36 U	23 J	180 U	180 UJ	34 U	41 U	69 U	34 U	34 U	180
BETA-BHC	4.2 UJ	1.8 U	1.9 U	9 U	9.2 U	1.8 U	2.1 U	3.5 U	1.8 U	1.8 U	9.3
DELTA-BHC	5.7 J	1.8 U	1.9 U	9 U	9.2 U	1.8 U	2.1 U	3.5 UJ	1.8 U	1.8 U	9.3
DIELDRIN	8.2 UJ	3.8 U	3.7 U	74	74	3.4 U	4.1 U	8 J	3.4 U	3.4 U	18 U
ENDOSULFAN I	1.1 J	1.8 UJ	1.9 U	22	22	1.8 U	2.1 U	3.5 UJ	1.8 U	1.8 U	9.3
ENDOSULFAN II	R	3.8 U	3.7 U	18 U	18 U	3.4 U	2.8 J	6.9 U	3.4 U	3.4 U	18 U
ENDOSULFAN SULFATE	8.2 U	3.8 U	3.7 U	18 U	18 U	3.4 U	4.1 U	8.8 U	3.4 U	3.4 U	18 U
ENDRIN	4 J	3.8 U	3.7 U	18 U	18 U	3.4 U	4.1 U	8.8 U	3.4 U	3.4 U	18 U
ENDRIN ALDEHYDE	5.9 J	3.8 U	3.7 U	18 U	18 U	3.4 U	4.1 U	8.8 U	3.4 U	3.4 U	18 U
ENDRIN KETONE	14 J	3.8 U	3.7 U	18 U	18 U	3.4 U	4.1 U	8.8 U	3.4 U	3.4 U	18 U
GAMMA-BHC (LINDANE)	4.2 UJ	1.8 U	1.9 U	9 U	9.2 U	1.8 U	2.1 U	3.5 U	1.8 U	1.8 U	9.3
GAMMA-CHLORDANE	R	1.8 UJ	3.5 J	R	13 J	1.8 U	2.1 U	2.8 J	1.8 U	5.8	9.3
HEPTACHLOR	4.2 UJ	1.8 U	1.9 U	9 U	9.2 U	1.8 U	2.1 U	3.5 U	1.8 U	1.8 U	9.3
HEPTACHLOR EPOXIDE	4.2 UJ	1.8 U	1.9 U	9 U	9.2 U	1.8 U	2.1 U	3.5 U	1.8 U	1.8 U	9.3
METHOXYCHLOR	13 J	18 U	18 U	90 U	92 U	18 U	21 U	35 U	18 U	18 U	93
TOXAPHENE	426 U	180 U	180 U	900 U	920 U	180 U	210 U	350 U	180 U	180 U	930

ANALYTICAL RESULTS
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 RAYMARK-FERRY CREEK - OU3
 STRATFORD, CONNECTICUT

Sample Number	CF-G8	CS-B*00	CS-D*300	EWS-G5A	EWS-G5B	EWS-G7	EX-91	FLS-A*250	FLS-G1	FLS-G2	FS-A*150
Sample Location	CF-G8	CS-B*00	CS-D*300	EWS-G5	EWS-G5	EWS-G7	EX-91	FLS-A*250	FLS-G1	FLS-G2	FS-A*150
Date Sampled											
QC Type	None	None	None			None	None	None	None	Field Dup. (3004)	None
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL
Filtering	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Metals (MG/KG)											
ALUMINUM	NA	15300	16600	16100 J	16200 J	3930 J	15800		NA	NA	NA
ANTIMONY	NA	R	6.7 J	8.1 UJ	5.8 UJ	5.5 UJ	5.7 UJ		NA	NA	NA
ARSENIC	NA	7.5	16.7	5.7 J	4.7 J	1.5 J	5.8		NA	NA	NA
BARIUM	NA	55	54.8	46.4	45	22.3	328 J		NA	NA	NA
BERYLLIUM	NA	0.65	0.77	1.2	1.3	0.35 U	0.7		NA	NA	NA
CADMIUM	NA	0.65 U	0.66 U	0.42 UJ	0.55 J	0.43 J	1.4 J		NA	NA	NA
CALCIUM	NA	2400 J	821 J	1400 J	1410 J	1210 J	5170 J		NA	NA	NA
CHROMIUM	NA	18	24.4	16.8	17.1	6.7	19.1		NA	NA	NA
COBALT	NA	6.9	6.3	8.7	10.2	4.7	7.6 UJ		NA	NA	NA
COPPER	NA	23.6 J	58.6 J	23.5 J	21.5 J	9.4 J	123 J		NA	NA	NA
IRON	NA	15600	18000	20200 J	20000	8300 J	18500		NA	NA	NA
LEAD	NA	64.3	224	51.8 J		R	13.5 J	344 J	NA	NA	NA
MAGNESIUM	NA	3390	3090	3610 J	3620 J	1790 J	3350		NA	NA	NA
MANGANESE	NA	322	233	307 J	329 J	223 J	813 J		NA	NA	NA
MERCURY	NA	0.25	0.14	0.11 J	0.12 J	0.06 UJ	0.14 J		NA	NA	NA
NICKEL	NA	12.9 J	17.2 J	14.3	12.6	7.3	14.9 J		NA	NA	NA
POTASSIUM	NA	1420	746	1270	1270	634 U	1680		NA	NA	NA
SELENIUM	NA	0.66 U	1.2	0.33 UJ	0.31 UJ	0.31 UJ	1 U		NA	NA	NA
SILVER	NA	1.5 UJ	2.1 UJ	0.34 U	0.31 U	0.31 U	1.9 UJ		NA	NA	NA
SODIUM	NA	162 UJ	153 UJ	221		R	96	109 UJ	NA	NA	NA
THALLIUM	NA	1.5 U	1.8 U	0.18 UJ	0.16 UJ	0.15 UJ	1.7 U		NA	NA	NA
VANADIUM	NA	30.1	55.4	36.2	35.4	14.4	39.1		NA	NA	NA
ZINC	NA	85.6 J	111 J	66.7 J	62.6 J	245 J	553 J		NA	NA	NA

U - Not detected; UJ - Detection limit approximate; J - Quantitation approximate;
 * - From dilution analysis; R - Rejected; NA - Not Analyzed

ANALYTICAL RESULTS
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 RAYMARK-FERRY CREEK - OJ3
 STRATFORD, CONNECTICUT

Sample Number	FS-AH50	GC-94	GLC004	HBN-94	HBN-G4	HP-C*500	HP-GR7	HP-GR9	JA-C*400	JA-C*800	LBB012	
Sample Location	FS-AH50	GC-94	GLC004	HBN-94	HBN-G4	HP-C*500	HP-GR7	HP-GR9	JA-C*400	JA-C*800	LBB012	
Date Sampled												
OC Type	None	None	None	None	None	None	None	None	None	None	None	
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	
Filtering	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	
Pesticides/PCBs (UG/KG)												
4,4'-DDD	U	NA	3.5 UJ	NA	NA	3.3 U	3.6 U	1.7 UJ	1.9 U	3.7 UJ	NA	3.7 UJ
4,4'-DDE	U	NA	5	NA	NA	3.9 U	9.3	9.4 J	1.6 J	2.2 J	NA	2.6 J
4,4'-DDT	U	NA	11	NA	NA	3.3 U	12	14 J	2.9 J	4.5 J	NA	3.6 J
ALDRIN	U	NA	1.8 U	NA	NA	1.7 U	1.9 U	0.8 UJ	0.8 U	1.9 UJ	NA	1.8 UJ
ALPHA-BHC	U	NA	1.8 U	NA	NA	1.7 U	1.9 U	0.8 UJ	0.8 U	1.9 UJ	NA	1.8 UJ
ALPHA-CHLORDANE	U	NA	4.2	NA	NA	3.3 U	1.9 U	0.8 UJ	0.8 U	1.9 UJ	NA	1.9 UJ
AROCLOR, TOTAL	U	NA	175.5 U	NA	NA	234 U	181 U	855 U	955 U	466.3	NA	167.5
AROCLOR-1016	U	NA	35 U	NA	NA	170 U	36 U	170 UJ	190 U	37 UJ	NA	37 UJ
AROCLOR-1221	U	NA	71 U	NA	NA	33 U	74 U	350 UJ	390 U	74 UJ	NA	76 UJ
AROCLOR-1232	U	NA	35 U	NA	NA	67 U	36 U	170 UJ	190 U	37 UJ	NA	37 UJ
AROCLOR-1242	U	NA	35 U	NA	NA	33 U	36 U	170 UJ	190 U	37 UJ	NA	37 UJ
AROCLOR-1248	U	NA	35 U	NA	NA	33 U	36 U	170 UJ	190 U	37 UJ	NA	37 UJ
AROCLOR-1254	U	NA	35 U	NA	NA	33 U	36 U	170 UJ	190 UJ	37 UJ	NA	37 UJ
AROCLOR-1260	U	NA	35 U	NA	NA	33 U	36 U	170 UJ	190 UJ	37 UJ	NA	37 UJ
AROCLOR-1262	U	NA	35 UJ	NA	NA	33 U	36 U	170 UJ	190 UJ	37 UJ	NA	37 UJ
AROCLOR-1268	U	NA	35 U	NA	NA	33 U	36 U	170 UJ	190 UJ	37 UJ	NA	37 UJ
BETA-BHC	U	NA	1.8 U	NA	NA	1.7 U	1.9 U	0.8 UJ	0.8 U	1.9 UJ	NA	1.8 UJ
DELTA-BHC	U	NA	1.8 U	NA	NA	1.7 U	1.9 U	0.8 UJ	0.8 U	1.9 UJ	NA	1.8 UJ
DIELDRIN	U	NA	3.5 UJ	NA	NA	3.3 U	3.6 U	1.7 UJ	1.9 U	3.7 UJ	NA	3.7 UJ
ENDOSULFAN I	U	NA	1.8 U	NA	NA	1.7 U	1.9 U	0.8 UJ	0.8 U	1.9 UJ	NA	1.8 UJ
ENDOSULFAN II	U	NA	3.5 U	NA	NA	3.3 U	3.6 U	1.7 UJ	1.9 U	3.7 UJ	NA	3.7 UJ
ENDOSULFAN SULFATE	U	NA	3.5 U	NA	NA	3.3 U	3.6 U	1.7 UJ	1.9 U	3.7 UJ	NA	3.7 UJ
ENDRIN	U	NA	3.5 U	NA	NA	3.3 U	3.6 U	1.7 UJ	1.9 U	3.7 UJ	NA	3.7 UJ
ENDRIN ALDEHYDE	U	NA	3.5 U	NA	NA	3.3 U	3.6 U	1.7 UJ	1.9 U	3.7 UJ	NA	3.7 UJ
ENDRIN KETONE	U	NA	3.5 U	NA	NA	3.3 U	3.6 U	1.7 UJ	1.9 U	3.7 UJ	NA	3.7 UJ
GAMMA-BHC (LINDANE)	U	NA	1.8 U	NA	NA	1.7 U	1.9 U	0.8 UJ	0.8 U	1.9 UJ	NA	1.8 UJ
GAMMA-CHLORDANE	U	NA	2.1 J	NA	NA	0.2 J	1.9 U	0.8 UJ	0.8 U	1.9 UJ	NA	1.9 UJ
HEPTACHLOR	U	NA	1.8 U	NA	NA	1.7 U	1.9 U	0.8 UJ	0.8 U	1.9 UJ	NA	1.8 UJ
HEPTACHLOR EPOXIDE	U	NA	1.6 J	NA	NA	1.7 U	1.9 U	0.8 UJ	0.8 U	1.9 UJ	NA	1.6 J
METHOXYCHLOR	U	NA	1.8 U	NA	NA	4.1 J	1.9 U	0.8 UJ	0.8 U	1.9 UJ	NA	2.2 J
TOXAPHENE	U	NA	180 U	NA	NA	5.7 J	180 U	880 UJ	880 U	190 UJ	NA	190 UJ

ANALYTICAL RESULTS
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 RAYMARK-FERRY CREEK - OU3
 STRATFORD, CONNECTICUT

Sample Number	FS-AH50	GC-94	GLC004	HBN-94	HBN-G4	HP-C+500	HP-GR7	HP-GR9	JA-C+400	JA-C+900	LBB012
Sample Location	FS-AH50	GC-94	GLC004	HBN-94	HBN-G4	HP-C+500	HP-GR7	HP-GR9	JA-C+400	JA-C+900	LBB012
Date Sampled											
QC Type	None	None	None	None	None	None	None	None	None	None	None
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL
Filtering	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Metals (MG/KG)											
ALUMINUM	NA 16400 J	NA 16700	14000	NA 10400 J	11400 J	15400 J	NA 11300				NA
ANTIMONY	NA 6.1 UJ	NA 9.3 U	8.3 UJ	NA 6.1 UJ	5.5 UJ	6.2 UJ	NA 9.1 UJ				NA
ARSENIC	NA 4.5	NA 9	8.8	NA 5.4	8.8	11	NA 3.6				NA
BARIUM	NA 172 J	NA 48.4	55 J	NA 54 J	53.8 J	126 J	NA 93.7 J				NA
BERYLLIUM	NA 1.1	NA 1.2	0.64	NA 0.58 J	0.65 J	0.83	NA 0.34 J				NA
CADMIUM	NA 0.48 UJ	NA 0.72 U	0.64 U	NA 0.42 UJ	0.47 UJ	1.4 U	NA 1.1 J				NA
CALCIUM	NA 161 J	NA 1110	1310 J	NA 1210 J	1470 J	1560 J	NA 1160 J				NA
CHROMIUM	NA 35.2	NA 17.5	15	NA 14.9	15.7	23.9	NA 14.5				NA
COBALT	NA 7.5	NA 8.5	6.9 UJ	NA 8.8	6.7	7.7	NA 2.8 UJ				NA
COPPER	NA 54	NA 49.2	23.8 J	NA 17.1	24.7	44	NA 45.3 J				NA
IRON	NA 19400 J	NA 17800	17400	NA 16100 J	16600 J	17800 J	NA 11100				NA
LEAD	NA 193	NA 97	40.3 J	NA 28.1	64.3	300	NA 286 J				NA
MAGNESIUM	NA 3390 J	NA 3250	3610	NA 3620 J	2590 J	2990 J	NA 1730				NA
MANGANESE	NA 262 J	NA 247	263 J	NA 338 J	316 J	341 J	NA 126 J				NA
MERCURY	NA 0.08 J	NA 0.12 U	0.12 J	NA 0.08 UJ	0.21 J	0.14 J	NA 0.18 J				NA
NICKEL	NA 17.9 U	NA 17	11.5 J	NA 15.2 U	13.4 U	18.1 U	NA 10.1 J				NA
POTASSIUM	NA 894 UJ	NA 1330	1500	NA 1710	984 J	731 UJ	NA 517				NA
SELENIUM	NA 0.33 UJ	NA 0.72 UJ	0.85 U	NA 0.34 UJ	0.31 UJ	0.35 UJ	NA 1.3 J				NA
SILVER	NA 0.34 U	NA 3.3 J	1.5 UJ	NA 0.34 U	0.31 U	0.34 U	NA 1.9 UJ				NA
SODIUM	NA 116	NA 112 U	65.4 UJ	NA 116	248	106	NA 50 UJ				NA
THALLIUM	NA 0.16 U	NA 1.2 U	1.5 U	NA 0.17 U	0.15 UJ	0.16 U	NA 1.6 U				NA
VANADIUM	NA 46.7	NA 42.5	32.8	NA 31.8	33.4	48.4	NA 29.7				NA
ZINC	NA 229 J	NA 80.8	76 J	NA 45.4 J	109 J	235 J	NA 254 J				NA

ANALYTICAL RESULTS
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RAYMARK-FERRY CREEK - OJ3
STRAITFORD, CONNECTICUT

Sample Number	LBP005	LBP012	LBP019	LBP029	LBP039	LBPAA+400																			
Sample Location	LBP005	LBP012	LBP019	LBP029	LBP039	LBPAA+400																			
Date Sampled																									
OC Type	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	
Filtering	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	
Pesticides/PCBs (UG/KG)																									
4,4'-DDD	3.7 UJ	NA	4 UJ	2.4 J	3.5 UJ	R		R		2.05		3.5 U	3.4 UJ	NA										NA	
4,4'-DDE	7.7 J	NA	R	3.8 UJ	3.9 J	81 J		72 J		2		3.5 U	3.4 UJ	NA											NA
4,4'-DDT	5.4 J	NA	4 UJ	3.2 J	6.6 J	120 J		62 J		2		3.5 U	3.4 UJ	NA											NA
ALDRIN	1.9 U	NA	2 UJ	1.8 UJ	1.8 UJ	2 UJ		1.9 U		2.1 U		1.8 U	1.7 U	NA											NA
ALPHA-BHC	1.9 U	NA	2 UJ	1.8 UJ	1.8 UJ	2 UJ		1.9 U		2.1 U		1.8 U	1.7 U	NA											NA
ALPHA-CHLORDANE	R	NA	4.8 UJ	9.6 J	1.8 UJ	21 J		R		4		1.8 U	1.7 U	NA											NA
AROCLOR, TOTAL	172.5	NA	200.5 U	162.5	176 U	305		183		205		175.5 U	170 U	NA											NA
AROCLOR-1016	37 U	NA	40 UJ	36 UJ	35 UJ	38 UJ		37 U		41 U		35 U	34 U	NA											NA
AROCLOR-1221	75 U	NA	81 UJ	73 UJ	72 UJ	78 UJ		75 U		83 U		71 U	68 U	NA											NA
AROCLOR-1232	37 U	NA	40 UJ	36 UJ	35 UJ	38 UJ		37 U		41 U		35 U	34 U	NA											NA
AROCLOR-1242	37 U	NA	40 UJ	36 UJ	35 UJ	38 UJ		37 U		41 U		35 U	34 U	NA											NA
AROCLOR-1248	37 U	NA	40 UJ	36 UJ	35 UJ	38 UJ		37 U		41 U		35 U	34 U	NA											NA
AROCLOR-1254	37 U	NA	40 UJ	36 UJ	35 UJ	38 UJ		37 U		41 U		35 U	34 U	NA											NA
AROCLOR-1260	37 U	NA	40 UJ	36 UJ	35 UJ	38 UJ		37 U		41 U		35 U	34 U	NA											NA
AROCLOR-1262	R	NA	40 UJ	R	35 UJ	110 J		16 J		20		35 UJ	34 U	NA											NA
AROCLOR-1268	24 J	NA	40 UJ	36 UJ	35 UJ	42 J		37 U		41 U		35 U	34 U	NA											NA
BETA-BHC	1.8 U	NA	2 UJ	1.8 UJ	1.8 UJ	2 UJ		1.9 U		2.1 U		1.8 U	1.7 U	NA											NA
DELTA-BHC	R	NA	2 UJ	1.8 UJ	1.8 UJ	2 UJ		1.8 UJ		1.05		1.8 U	1.7 U	NA											NA
DIELDRIN	R	NA	R	3.6 UJ	3.5 UJ	3.8 UJ		R		R		3.5 U	3.4 U	NA											NA
ENDOSULFAN I	1.8 U	NA	2 UJ	1.8 UJ	1.8 UJ	2.3 J		1.8 U		NA		1.8 U	1.7 U	NA											NA
ENDOSULFAN II	3.7 U	NA	4 UJ	3.8 UJ	3.5 UJ	3.8 UJ		3.7 U		4.1 U		3.5 U	3.4 U	NA											NA
ENDOSULFAN SULFATE	3.7 U	NA	4 UJ	3.8 UJ	3.5 UJ	3.8 UJ		3.7 U		4.1 U		3.5 U	3.4 U	NA											NA
ENDRIN	3.7 U	NA	4 UJ	R	3.5 UJ	3.8 UJ		3.7 U		4.1 U		3.5 U	3.4 U	NA											NA
ENDRIN ALDEHYDE	3.7 U	NA	4 UJ	3.6 UJ	3.5 UJ	3.8 UJ		3.7 U		4.1 U		3.5 U	3.4 U	NA											NA
ENDRIN KETONE	3.7 UJ	NA	R	R	3.5 UJ	3.8 UJ		3.7 UJ		3.8		3.5 U	3.4 U	NA											NA
GAMMA-BHC (LINDANE)	1.9 U	NA	2 UJ	1.8 UJ	1.8 UJ	2 UJ		1.9 U		2.1 U		1.8 U	1.7 U	NA											NA
GAMMA-CHLORDANE	R	NA	R	R	1.8 UJ	R		1.8 UJ		2.1 U		1.8 U	1.7 U	NA											NA
HEPTACHLOR	R	NA	2 UJ	1.8 UJ	1.8 UJ	2 UJ		R		R		1.8 U	1.7 U	NA											NA
HEPTACHLOR EPOXIDE	1.9 U	NA	2 UJ	1.8 UJ	1.8 UJ	2 UJ		1.9 U		2.1 U		1.8 U	1.7 UJ	NA											NA
METHOXYCHLOR	5 J	NA	3.6 J	18 UJ	18 UJ	20 UJ		5.8 J		NA		18 U	17 U	NA											NA
TOXAPHENE	190 U	NA	200 UJ	180 UJ	180 UJ	200 UJ		190 U		210 U		180 U	170 U	NA											NA

U - Not detected; UJ - Detection limit approximate; J - Quantitation approximate;
- From dilution analysis; R - Rejected; NA - Not Analyzed

ANALYTICAL RESULTS
 DRAFT REMEDIAL INVESTIGATION REPORT
 RAYMARK-FERRY CREEK - OJ3
 STRATFORD, CONNECTICUT

Sample Number	LBP005	LBP012	LBP019	LBP029	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400
Sample Location	LBP005	LBP012	LBP019	LBP029	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400	LBP039	LBPAA+400
Date Sampled																								
QC Type	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None	None
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL
Filtering	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Metals (MG/KG)																								
ALUMINUM	15000 J	12200 J	9520 J	3740 J	15100 J	13800 J	18500 J	21500 J	7130															
ANTIMONY	6.3 UJ	6.3 UJ	6.7 UJ	5.7 UJ	6 UJ	6.1 UJ	6.3 UJ	6.7 U	6.5 UJ															
ARSENIC	8.9	7.5	7.7	1.1	8.5	10.3	4.3																	
BARIIUM	45.7	42.1	42.9	22	42.4	48.5	49.1																	
BERYLLIUM	1.1	0.82 U	0.87 U	0.4 UJ	0.97 U	1.1	1.3																	
CADMIUM	0.44 U	0.78 UJ	0.78 UJ	0.4 UJ	0.7 UJ	1.4	0.44 U	0.53	0.63 U															
CALCIUM	637 J	3830 J	1910 J	2010 J	781 J	938 J	731 J	751	1300 J															
CHROMIUM	15.7	14.8	15.3	10.2	16.2	20.2	18	20.1	10.9															
COBALT	6.9	7.2	7.7	2.6 J	8.9	7.6	7.2	9.4	5.7 UJ															
COPPER	26.9 J	42.8 J	31.5 J	18.3 J	22.9 J	30.1 J	22.7 J	17.8	11.9 UJ															
IRON	17300 J	16000 J	17300 J	8220 J	18700 J	17100 J	17700 J	21600	13100															
LEAD	82.6	85.5 J	67.7 J	43.2 J	74 J	185 J	55.2	60	3.7 J															
MAGNESIUM	2860 J	3910 J	2710 J	1840 J	2720 J	2580 J	2990 J	3350	3310															
MANGANESE	284 J	224 J	406 J	98.2 J	251 J	299 J	411 J	308	201 J															
MERCURY	0.12 J	0.08 U	0.09 U	0.08 J	0.08 J	0.14 J	0.12 J	0.17	0.11 U															
NICKEL	11.9 J	11.7	16.3	10.7	13.7	12.8	14.2 J	15.3	7.9 J															
POTASSIUM	834 UJ	388 UJ	594 UJ	529 J	610 J	691 UJ	740 UJ	884 U	2880															
SELENIUM	0.24 UJ	0.34 UJ	0.39 UJ	0.32 UJ	0.33 UJ	0.34 UJ	0.84 J	0.86	0.84 U															
SILVER	0.35 U	0.35 U	0.37 U	0.32 U	0.33 U	0.34 U	0.35 U	0.37 U	1.8 UJ															
SODIUM	105	128 U	126 U	118 U	95.9 U	71.8 U	104	99.3 U	85.8 UJ															
THALLIUM	0.43 UJ	0.17 U	0.19 UJ	0.16 U	0.17 UJ	0.43 U	0.42 UJ	0.48 U	1.5 U															
VANADIUM	38.8	28.8	29	15.3	38.1	41.5	39.7	52.3	19.7															
ZINC	66.7 J	604 J	76.7 J	42.6 J	55.4 J	450 J	52.3 J	58.9	28.4 J															

ANALYTICAL RESULTS
 DRAFT REMEDIAL INVESTIGATION REPORT
 RAYMARK FERRY CREEK - 0U3
 STURFORD, CONNECTICUT

Sample Number	LP-A+50	LP-G1	LSSA+00	LSSB+365	LSSC+125
Sample Location	LP-A+50	LP-G1	LSSA+00	LSSB+365	LSSC+125
Date Sampled					
OC Type	None	None	None	None	None
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL
Filtering	NA	NA	NA	NA	NA
Pesticides/PCBs (UG/KG)					
4,4'-DDD	16 U	17 U	35 UJ	3.7 UJ	R
4,4'-DDE	16 U	74	35 UJ	R	110 J
4,4'-DDT	18 U	280	35 UJ	R	160 J
ALDRIN	8.5 U	8.9 U	1.8 UJ	1.9 UJ	3.5 UJ
ALPHA-BHC	8.5 U	8.9 U	1.8 UJ	1.9 UJ	3.5 UJ
ALPHA-CHLORDANE	8.5 U	8.9 U	1.8 UJ	3.5 J	R
AROCLOR, TOTAL	810 U	855 U	158.5	166.5	311.5
AROCLOR-1016	160 U	170 U	35 UJ	37 UJ	69 UJ
AROCLOR-1221	340 U	350 U	72 UJ	74 UJ	140 UJ
AROCLOR-1232	160 U	170 U	35 UJ	37 UJ	69 UJ
AROCLOR-1242	160 U	170 U	35 UJ	37 UJ	69 UJ
AROCLOR-1248	160 U	170 U	35 UJ	37 UJ	69 UJ
AROCLOR-1254	160 UJ	170 U	35 UJ	37 UJ	69 UJ
AROCLOR-1260	160 UJ	170 U	35 UJ	37 UJ	69 UJ
AROCLOR-1262	160 UJ	170 U	R	R	R
AROCLOR-1268	160 U	170 U	35 UJ	37 UJ	69 UJ
BETA-BHC	8.5 U	8.9 U	1.8 UJ	1.9 UJ	3.5 UJ
DELTA-BHC	8.5 U	8.9 U	1.8 UJ	1.9 UJ	3.5 UJ
DIELDRIN	16 U	17 U	35 UJ	6.8 J	R
ENDOSULFAN I	8.5 U	8.9 U	1.8 UJ	1.8 UJ	3.5 UJ
ENDOSULFAN II	18 U	17 U	35 UJ	1.8 J	8.9 UJ
ENDOSULFAN SULFATE	18 U	17 U	35 UJ	3.7 UJ	8.9 UJ
ENDRIN	18 U	17 U	35 UJ	3.7 UJ	8.9 UJ
ENDRIN ALDEHYDE	18 U	17 U	35 UJ	3.7 UJ	8.9 UJ
ENDRIN KETONE	18 U	17 U	35 UJ	1.8 J	8.5 J
GAMMA-BHC (LINDANE)	8.5 U	8.9 U	1.8 UJ	1.9 UJ	3.5 UJ
GAMMA-CHLORDANE	8.5 U	8.9 U	1.8 UJ	1.9 UJ	3.5 UJ
HEPTACHLOR	8.5 U	8.9 U	1.8 UJ	1.9 UJ	3.5 UJ
HEPTACHLOR EPOXIDE	8.5 U	8.9 U	1.8 UJ	2.3 J	3.5 UJ
METHOXYCHLOR	8.5 U	8.9 U	1.8 UJ	1.9 UJ	R
TOXAPHENE	850 U	890 U	180 UJ	190 UJ	350 UJ

U - Not detected; UJ - Detection limit approximate; J - Quantitation approximate;
 * - From dilution analysis; R - Rejected; NA - Not Analyzed

ANALYTICAL RESULTS
 DRAFT REMEDIAL INVESTIGATION REPORT
 RAYMARK-FERRY CREEK - OJ3
 STRATFORD, CONNECTICUT

Sample Number	LP-A+50	LP-G1	LSSA+00	LSSB+365	LSSC+125
Sample Location	LP-A+50	LP-G1	LSSA+00	LSSB+365	LSSC+125
Date Sampled					
QC Type	None	None	None	None	None
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL
Filtering	NA	NA	NA	NA	NA
Metals (MG/KG)					
ALUMINIUM	13500 J	NA	16500	13400	17100
ANTIMONY	6 UJ	NA		R	R
ARSENIC	3.6 J	NA	8.3	5.4	10.1
BARIUM	64.3 J	NA	44.2 J	49.6	69.9 J
BERYLLIUM	0.7 J	NA	0.65	0.49	0.72
CADMIUM	0.41 UJ	NA	0.64 UJ	0.67 UJ	0.98 J
CALCIUM	545 J	NA	960 J	1780 J	1280 J
CHROMIUM	14.1	NA	31.1	21.6	28.6
COBALT	6.2	NA	11.7	6.4	12.4
COPPER	22.9 J	NA	32.6 J	25.9 J	60.8 J
IRON	14500 J	NA	24100	16200	21700
LEAD	84.2 J	NA	22.2 J	72.9	187
MAGNESIUM	2190 J	NA	5690	3630	5210
MANGANESE	262 J	NA	483 J	305 J	660 J
MERCURY	0.12 J	NA	0.11 U	0.11 U	0.22
NICKEL	11.4	NA	40.4 J	17 J	31.6 J
POTASSIUM	316 UJ	NA	1490	1070	1590
SELENIUM	0.34 UJ	NA	0.85 UJ	0.89 UJ	0.95 J
SILVER	0.33 U	NA	1.5 U	1.6 U	1.5 U
SODIUM	97.8	NA	R	150 UJ	R
THALLIUM	0.17 UJ	NA	1.5 UJ	1.6 U	1.5 UJ
VANADIUM	33.4	NA	39.1 J	31.8	54.2 J
ZINC	66.3 J	NA	109 J	79.5 J	203 J

ANALYTICAL RESULTS
 DRAFT REMEDIAL INVESTIGATION REPORT
 RAYMARK FERRY CREEK - 0113
 STRATFORD, CONNECTICUT

Sample Number	NEP-C+200	NEP-GR8	NEP-GRG	NS-B+200	NS-E+200	NS-F+00	NS-G+300	SB-925-D	SB-950-F	SB-970-L
Sample Location	NEP-C+200	NEP-GR8	NEP-GRG	NS-B+200	NS-E+200	NS-F+00	NS-G+300	SB-925-D	SB-950-F	SB-970-L
Date Sampled										
QC Type	None	None	None	None	None	None	None	None	None	None
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL
Filtering	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Pesticides/PCBs (UG/KG)										
4,4'-DDD	3.4 U	NA	18 U	18 U	20 UJ	3.8 U	18 U	3.8 U	3.3 U	3.6 U
4,4'-DDE	2.5 J	NA	18 U	18 U	20 UJ	8	18 U	3.4 J	3.3 U	4.5 J
4,4'-DDT	3.4 U	NA	18 U	18 U	20 UJ	9.7 J	18 U	0.3	2.6 J	R
ALDRIN	1.8 U	NA	9.1 U	9.2 U	10 UJ	1.9 U	9 U	2 U	1.7 U	1.8 U
ALPHA-BHC	1.8 U	NA	9.1 U	9.2 U	10 UJ	1.9 U	9 U	2 U	1.7 U	1.8 U
ALPHA-CHLORDANE	3.4 U	NA	9.1 U	9.2 U	4.4 J	1.9 U	9 U	2 U	1.7 U	10.4
AROCLOR TOTAL	244 U	NA	900 U	810	1005 U	163	810	190.3 U	166 U	241.5
AROCLOR-1016	180 U	NA	180 U	180 U	200 UJ	36 U	180 U	36 U	33 U	36 U
AROCLOR-1221	34 U	NA	360 U	360 U	410 UJ	74 U	360 U	77 U	68 U	73 U
AROCLOR-1232	70 U	NA	180 U	180 U	200 UJ	36 U	180 U	36 U	33 U	36 U
AROCLOR-1242	34 U	NA	180 U	180 U	200 UJ	36 U	180 U	36 U	33 U	36 U
AROCLOR-1248	34 U	NA	180 U	180 U	200 UJ	36 U	180 U	36 U	33 U	36 U
AROCLOR-1254	34 U	NA	180 U	180 UJ	200 UJ	36 U	180 U	36 U	33 U	36 U
AROCLOR-1260	34 U	NA	180 U	180 UJ	200 UJ	36 U	180 U	36 U	33 U	36 U
AROCLOR-1262	34 U	NA	180 U	R	200 UJ	36 U	R	36 U	33 UJ	36 U
AROCLOR-1268	34 U	NA	180 U	180 UJ	200 UJ	36 U	180 U	36 U	33 UJ	79 J
BETA-BHC	1.8 U	NA	9.1 U	9.2 U	10 UJ	1.9 U	9 U	2 U	1.7 U	1.8 U
DELTA-BHC	1.8 U	NA	9.1 U	9.2 U	10 UJ	1.9 U	9 U	2 U	1.7 U	1.8 U
DIELDRIN	2.8 J	NA	18 U	18 U	190 J	4.8 J	18 U	3.8 U	3.3 U	R
ENDOSULFAN I	1.8 U	NA	9.1 U	9.2 U	47 J	1.9 U	9 U	2 U	1.7 U	1.8 U
ENDOSULFAN II	3.4 U	NA	18 U	18 U	20 UJ	3.6 U	18 U	3.6 U	3.3 U	3.6 U
ENDOSULFAN SULFATE	3.4 U	NA	18 U	18 U	20 UJ	3.6 U	18 U	3.6 U	3.3 U	3.6 U
ENDRIN	3.4 U	NA	18 U	18 U	20 UJ	3.6 U	18 U	3.6 U	3.3 U	3.6 U
ENDRIN ALDEHYDE	3.4 U	NA	18 U	18 U	20 UJ	3.6 U	18 U	3.6 U	3.3 U	3.6 U
ENDRIN KETONE	9.3 J	NA	18 U	18 U	20 UJ	3.6 U	18 U	3.6 U	3.3 U	4.2 J
GAMMA-BHC (LINDANE)	1.8 U	NA	9.1 U	9.2 U	10 UJ	1.9 U	9 U	2 U	1.7 U	1.8 U
GAMMA-CHLORDANE	1 J	NA	9.1 U	9.2 U	R	1.9 U	9 U	2 U	1.7 U	R
HEPTACHLOR	1.8 U	NA	9.1 U	9.2 U	10 UJ	1.9 U	9 U	2 U	1.7 U	1.8 U
HEPTACHLOR EPOXIDE	1.8 U	NA	9.1 U	9.2 U	10 UJ	1.9 U	9 U	2 U	1.7 U	1.8 U
METHOXYCHLOR	4.6	NA	91 U	92 U	100 UJ	19 U	90 U	20 U	17 U	R
TOXAPHENE	1.4 J	NA	910 U	920 U	1000 UJ	190 U	900 U	200 U	170 U	180 U

ANALYTICAL RESULTS
DRAFT REMEDIAL INVESTIGATION REPORT
RAYMARK-FERRY CREEK - OJ3
STRATFORD, CONNECTICUT

Sample Number	NEP-C*200	NEP-GR6	NEP-GRG	NS-B*200	NS-E*200	NS-F*00	NS-G*300	SB-925-D	SB-950-F	SB-970-L
Sample Location	NEP-C*200	NEP-GR6	NEP-GRG	NS-B*200	NS-E*200	NS-F*00	NS-G*300	SB-925-D	SB-950-F	SB-970-L
Date Sampled										
QC Type	None	None	None	None	None	None	None	None	None	None
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL
Filtering	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Metals (MG/KG)										
ALUMINUM	13200 J	10200 J	NA	15000 J	17100 J	14800 J	18100 J	9810 J	2320 J	4190 J
ANTIMONY	5.8 UJ	5.7 UJ	NA	6.2 UJ	6.6 UJ	6 UJ	5.9 UJ	6.3 UJ	5.5 UJ	5.8 UJ
ARSENIC	3.5	2.9	NA	5.8 J	6 J	5.5 J	4.3 J	4.1 J	0.99 J	1.8 J
BARIUM	30.6 J	32.6 J	NA	60.4	47.8	49.1	67	30.5	8	24.4
BERYLLIUM	0.98	0.61 J	NA	1.1	1.1	1.1	0.85	0.52 J	0.35 U	0.37 U
CADMIUM	0.41 UJ	0.4 UJ	NA	0.73 J	0.46 UJ	0.42 UJ	0.55 J	0.44 UJ	0.39 UJ	0.41 UJ
CALCIUM	775 J	1020 J	NA	800 J	1150 J	600 J	1030 J	821 J	2600 J	1170 J
CHROMIUM	14.2	9.5	NA	19.4	18.5	16.1	15.4	12.4	4.9	7.5
COBALT	8	6.6	NA	8.5	9.6	8	6.7	6.9	2 U	3.1 J
COPPER	18.3	14.2	NA	35.8 J	20 J	33.4 J	24.3 J	14.8 J	15.3 J	13.3 J
IRON	18900 J	14100 J	NA	18500 J	18700 J	17500 J	18300 J	12600 J	3600 J	6140 J
LEAD	43	22.7	NA	129 J	53.8 J	79.1 J	69.3 J	13.6 J	13.2 UJ	27.4 U
MAGNESIUM	3200 J	2980 J	NA	3080 J	3240 J	3150 J	3620 J	3290 J	1170 J	1830 J
MANGANESE	304 J	182 J	NA	336 J	291 J	307 J	409 J	329 J	65.8 J	94.5 J
MERCURY	0.16 J	0.08 UJ	NA	0.17 J	0.13 J	0.13 J	0.13 J	0.09 U	0.07 U	0.08 U
NICKEL	16.9 U	9.8 U	NA	15.5	13.1	14.1	14.2	9.8	4.2 J	7.3
POTASSIUM	1300 J	1770	NA	760 J	845 U	633 U	1570	1000 J	273 UJ	345 UJ
SELENIUM	0.32 UJ	0.32 UJ	NA	0.35 UJ	0.37 UJ	0.34 UJ	0.33 UJ	0.34 UJ	0.32 UJ	0.3 UJ
SILVER	0.33 U	0.32 U	NA	0.34 U	0.37 U	0.33 U	0.33 U	0.33 UJ	0.31 UJ	0.33 UJ
SODIUM	72.4	88.3 U	NA	123	154	109	120	105 J	43.8 J	50.4 J
THALLIUM	0.16 U	0.16 U	NA	0.17 UJ	0.19 UJ	0.17 UJ	0.18 UJ	0.17 U	0.18 U	0.15 U
VANADIUM	36.7	24.2	NA	47.6	33.5	44.9	34.5	23.1	4.8 J	9.9 J
ZINC	50.9 J	34.8 J	NA	60.5 J	58.5 J	69.2 J	79.2 J	39.4 J	27.4 J	46.1 J

U - Not detected; UJ - Detection limit approximate; J - Quantitation approximate;
* - From dilution analysis; R - Rejected; NA - Not Analyzed

ANALYTICAL RESULTS
 DRAFT REMEDIAL INVESTIGATION REPORT
 RAYMARK-FERRY CREEK - OJ3
 STRATFORD, CONNECTICUT

Sample Number	SBI*300														
Sample Location	SBI*300														
Date Sampled															
OC Type	None														
MATRIX	SOIL														
Filtering	NA														
Pesticides/PCBs (UG/KG)															
4,4'-DDD	R	R	3.5 U	NA	3.4 U	3.5 U	17 UJ	NA	0.44 J	NA	NA	NA	NA	NA	NA
4,4'-DDE	3.5 UJ	19 UJ	3.5 U	NA	3.4 U	3.9 J	17 UJ	NA	2.4 J	NA	NA	NA	NA	NA	NA
4,4'-DDT	6.7 J	19 UJ	3.5 U	NA	3.4 U	10 J	17 UJ	NA	4.7 J	NA	NA	NA	NA	NA	NA
ALDRIN	1.8 UJ	10 UJ	1.8 U	NA	1.8 U	1.8 U	8.9 UJ	NA	2 UJ	NA	NA	NA	NA	NA	NA
ALPHA-BHC	1.8 UJ	10 UJ	1.8 U	NA	1.8 U	1.8 U	8.9 UJ	NA	2 UJ	NA	NA	NA	NA	NA	NA
ALPHA-CHLORDANE	2.9 J	10 UJ	1.8 U	NA	1.8 U	3.5 U	8.9 UJ	NA	2 UJ	NA	NA	NA	NA	NA	NA
AROCLOR, TOTAL	241.5	955 U	175 U	NA	171 U	195.5	655 U	NA	251	NA	NA	NA	NA	NA	NA
AROCLOR-1016	35 UJ	190 UJ	35 U	NA	34 U	35 U	170 UJ	NA	39 UJ	NA	NA	NA	NA	NA	NA
AROCLOR-1221	72 UJ	390 UJ	70 U	NA	70 U	72 U	350 UJ	NA	79 UJ	NA	NA	NA	NA	NA	NA
AROCLOR-1232	35 UJ	190 UJ	35 U	NA	34 U	35 U	170 UJ	NA	39 UJ	NA	NA	NA	NA	NA	NA
AROCLOR-1242	35 UJ	190 UJ	35 U	NA	34 U	35 U	170 UJ	NA	39 UJ	NA	NA	NA	NA	NA	NA
AROCLOR-1248	35 UJ	190 UJ	35 U	NA	34 U	35 U	170 UJ	NA	39 UJ	NA	NA	NA	NA	NA	NA
AROCLOR-1254	36 UJ	190 UJ	35 U	NA	34 U	35 U	170 UJ	NA	39 UJ	NA	NA	NA	NA	NA	NA
AROCLOR-1260	35 UJ	190 UJ	35 U	NA	34 U	35 U	170 UJ	NA	39 UJ	NA	NA	NA	NA	NA	NA
AROCLOR-1262	83 J	190 UJ	35 UJ	NA	34 UJ	37 J	170 UJ	NA	75 J	NA	NA	NA	NA	NA	NA
AROCLOR-1268	35 UJ	190 UJ	35 UJ	NA	34 UJ	35 UJ	170 UJ	NA	39 UJ	NA	NA	NA	NA	NA	NA
BETA-BHC	1.8 UJ	10 UJ	1.8 U	NA	1.8 U	1.8 U	8.9 UJ	NA	2 UJ	NA	NA	NA	NA	NA	NA
DELTA-BHC	1.8 UJ	10 UJ	1.8 U	NA	1.8 U	1.8 U	8.9 UJ	NA	2 UJ	NA	NA	NA	NA	NA	NA
DIELDRIN	R	19 UJ	3.5 U	NA	3.4 U	R	17 UJ	NA	3.8 UJ	NA	NA	NA	NA	NA	NA
ENDOSULFAN I	1.8 UJ	10 UJ	1.8 U	NA	1.8 U	1.8 U	8.9 UJ	NA	2 UJ	NA	NA	NA	NA	NA	NA
ENDOSULFAN II	3.5 UJ	19 UJ	3.5 U	NA	3.4 U	3.5 U	17 UJ	NA	2.4 J	NA	NA	NA	NA	NA	NA
ENDOSULFAN SULFATE	3.5 UJ	19 UJ	3.5 U	NA	3.4 U	3.5 U	17 UJ	NA	3.9 UJ	NA	NA	NA	NA	NA	NA
ENDRIN	3.5 UJ	19 UJ	3.5 U	NA	3.4 U	3.5 U	17 UJ	NA	3.9 UJ	NA	NA	NA	NA	NA	NA
ENDRIN ALDEHYDE	3.5 UJ	19 UJ	3.5 U	NA	3.4 U	3.5 U	17 UJ	NA	3.9 UJ	NA	NA	NA	NA	NA	NA
ENDRIN KETONE	R	R	2.1 J	NA	R	3.9 J	R	NA	1.3 J	NA	NA	NA	NA	NA	NA
GAMMA-BHC (LINDANE)	1.8 UJ	10 UJ	1.8 U	NA	1.8 U	1.8 U	8.9 UJ	NA	2 UJ	NA	NA	NA	NA	NA	NA
GAMMA-CHLORDANE	R	R	1.8 U	NA	1.8 U	3.3 U	8.9 UJ	NA	2 UJ	NA	NA	NA	NA	NA	NA
HEPTACHLOR	1.8 UJ	10 UJ	1.8 U	NA	1.8 U	1.8 U	8.9 UJ	NA	0.23 J	NA	NA	NA	NA	NA	NA
HEPTACHLOR EPOXIDE	1.8 UJ	10 UJ	1.8 U	NA	1.8 U	1.8 U	8.9 UJ	NA	2 UJ	NA	NA	NA	NA	NA	NA
METHOXYCHLOR	18 UJ	100 UJ	18 U	NA	18 U	R	89 UJ	NA	4.1 J	NA	NA	NA	NA	NA	NA
TOXAPHENE	180 UJ	1000 UJ	180 U	NA	180 U	180 U	850 UJ	NA	200 UJ	NA	NA	NA	NA	NA	NA

ANALYTICAL RESULTS
DRAFT REMEDIAL INVESTIGATION REPORT
RAYMARK-FERRY CREEK - OJ3
STRATFORD, CONNECTICUT

Sample Number	SBI+300											
Sample Location	SBI+300											
Date Sampled												
OC Type	None											
MATRIX	SOIL											
Filtering	NA											
Metals (MG/KG)												
ALUMINUM	NA	6640 J	2360 J	2500 J	NA	2550 J	2660 J	4380 J	NA	15000 J		
ANTIMONY	NA	6.4 UJ	5.6 UJ	5.6 UJ	NA	5.7 UJ	6 UJ	5.6 UJ	NA	6.3 UJ		
ARSENIC	NA	5.7 J	0.53 J	0.62 J	NA	0.87 J	0.78 J	1.6 J	NA	12.3 J		
BARIUM	NA	23.2 J	8.8 J	16.4 J	NA	14 J	26.6 J	22.6 J	NA	54.2 J		
BERYLLIUM	NA	0.51 J	0.35 U	0.35 U	NA	0.36 U	0.37 U	0.35 U	NA	1.1 J		
CADMIUM	NA	0.44 UJ	0.39 UJ	0.39 UJ	NA	0.4 UJ	0.46 J	0.39 UJ	NA	1.2 U		
CALCIUM	NA	1600 J	33100 J	3760 J	NA	797 J	2430 J	1040 J	NA	1500 J		
CHROMIUM	NA	13.5 J	5.9 J	6.2 J	NA	5 J	6.8 J	14.9 J	NA	21.8 J		
COBALT	NA	4.6 J	2 U	2.4 J	NA	2.1 U	2.2 U	3.1 J	NA	6.2 J		
COPPER	NA	68.4 J	16.5 J	16.7 J	NA	11.2 J	13.1 J	38.3 J	NA	36.7 J		
IRON	NA	14700 J	5560 J	4420 J	NA	3930 J	4560 J	7730 J	NA	26000 J		
LEAD	NA	50.4 J	10.5 UJ	19.1 U	NA	47.7 J	65 J	21.7 U	NA	76.4 J		
MAGNESIUM	NA	2340 J	1420 J	1390 J	NA	1240 J	1230 J	1710 J	NA	5410 J		
MANGANESE	NA	152 J	73.1 J	61.6 J	NA	68.3 J	71.2 J	95.6 J	NA	253 J		
MERCURY	NA	0.18 J	0.09 U	0.07 U	NA	0.09 U	0.09 U	0.07 J	NA	0.14 J		
NICKEL	NA	17 J	8.3 J	5.4 J	NA	5 J	6.4 J	7.6 J	NA	14.5 UJ		
POTASSIUM	NA	518 UJ	268 UJ	387 UJ	NA	263 UJ	324 UJ	396 UJ	NA	1780 UJ		
SELENIUM	NA	0.37 UJ	0.3 UJ	0.32 UJ	NA	0.33 UJ	0.31 UJ	0.33 UJ	NA	0.37 UJ		
SILVER	NA	0.35 UJ	0.31 UJ	0.31 UJ	NA	0.32 UJ	0.33 UJ	0.31 UJ	NA	0.35 U		
SODIUM	NA	86.9 J	290 J	66.4 J	NA	31.4 J	36.1 J	76.9 J	NA	R		
THALLIUM	NA	0.18 UJ	0.15 UJ	0.16 UJ	NA	0.17 U	0.16 U	0.16 U	NA	0.16 UJ		
VANADIUM	NA	16.3 J	6.8 J	6.5 J	NA	6.2 J	6.7 J	10.4 J	NA	49.4 J		
ZINC	NA	117 J	28.6 J	37 J	NA	50.7 J	68.6 J	61.4 J	NA	710 J		

U - Not detected; UJ - Detection limit approximate; J - Quantitation approximate;
* - From dilution analysis; R - Rejected; NA - Not Analyzed

ANALYTICAL RESULTS
 DRAFT REMEDIAL INVESTIGATION REPORT
 RAYMARK-FERRY CREEK - OU3
 STRATFORD, CONNECTICUT

Sample Number	SBP005	SBPF+00	SH-97	SH-A+00	SH-D+695	SH-E+400	SH400	SMS-G3	SPB 005	THG005	THN-62	THN-G2
Sample Location	SBP005	SBPF+00	SH-97	SH-A+00	SH-D+695	SH-E+400	SH400	SMS-G3	SPB 005	THG005	THN-62	THN-G2
Date Sampled												
OC Type	None	None	None	None	None	None	None	None	None	None	None	None
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	None	None	None
Filtering	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Pesticides/PCBs (UG/KG)												
4,4'-DDD	NA	3.4 UJ	3.6 UJ	4.4 UJ	4 UJ	3.4 UJ	NA	3.6 U	R	18 UJ	19 UJ	NA
4,4'-DDE	NA	3.4 UJ	3.6 UJ	R	4 UJ	3.4 UJ	NA	3.6 UJ	2.9 J	240 J	19 UJ	NA
4,4'-DDT	NA	1.4 J	3.6 UJ	R	4 UJ	3.2 J	NA	3.6 U	2.7 UJ	400 J	19 UJ	NA
ALDRIN	NA	1.8 UJ	1.8 UJ	2.3 UJ	2.1 UJ	1.8 UJ	NA	1.8 U	1.8 UJ	9.4 UJ	9.8 UJ	NA
ALPHA-BHC	NA	1.8 UJ	1.8 UJ	2.3 UJ	2.1 UJ	1.8 UJ	NA	1.8 U	1.8 UJ	9.4 UJ	9.8 UJ	NA
ALPHA-CHLORDANE	NA	1.8 UJ	1.8 UJ	1.3 J	2.1 UJ	1.8 UJ	NA	1.8 U	0.33 J	13 J	9.8 UJ	NA
AROCLOR TOTAL	NA	303.5	182.5	198.5	201 U	154	170 U	180.5 U	187.5	605 U	655 U	NA
AROCLOR-1016	NA	34 UJ	36 UJ	44 UJ	40 UJ	34 UJ	34 U	36 U	37 UJ	180 UJ	190 UJ	NA
AROCLOR-1221	NA	69 UJ	73 UJ	89 UJ	82 UJ	70 UJ	68 U	73 U	76 UJ	370 UJ	390 UJ	NA
AROCLOR-1232	NA	34 UJ	36 UJ	44 UJ	40 UJ	34 UJ	34 U	36 U	37 UJ	180 UJ	190 UJ	NA
AROCLOR-1242	NA	34 UJ	36 UJ	44 UJ	40 UJ	34 UJ	34 U	36 U	37 UJ	180 UJ	190 UJ	NA
AROCLOR-1248	NA	34 UJ	36 UJ	44 UJ	40 UJ	34 UJ	34 U	36 U	37 UJ	180 UJ	190 UJ	NA
AROCLOR-1254	NA	34 UJ	36 UJ	44 UJ	40 UJ	34 UJ	34 U	36 U	37 UJ	180 UJ	190 UJ	NA
AROCLOR-1260	NA	34 UJ	36 UJ	44 UJ	40 UJ	34 UJ	34 U	36 U	37 UJ	180 UJ	190 UJ	NA
AROCLOR-1262	NA	150 J	R	R	40 UJ	R	34 U	36 U	R	180 UJ	190 UJ	NA
AROCLOR-1268	NA	34 UJ	36 UJ	44 UJ	40 UJ	34 UJ	34 U	36 U	37 UJ	180 UJ	190 UJ	NA
BETA-BHC	NA	1.8 UJ	1.8 UJ	2.3 UJ	2.1 UJ	1.8 UJ	NA	1.8 U	1.8 UJ	9.4 UJ	9.8 UJ	NA
DELTA-BHC	NA	1.8 UJ	1.8 UJ	2.3 UJ	2.1 UJ	1.8 UJ	NA	1.8 U	0.34 J	1.3 J	9.8 UJ	NA
DIELDRIN	NA	3.4 UJ	5.9 J	4.4 UJ	4 UJ	3.4 UJ	NA	3.6 U	R	R	19 UJ	NA
ENDOSULFAN I	NA	1.8 UJ	1.8 UJ	2.3 UJ	2.1 UJ	1.8 UJ	NA	1.8 U	1.9 UJ	9.4 UJ	9.8 UJ	NA
ENDOSULFAN II	NA	3.9 J	3.6 UJ	6 J	4 UJ	3.4 UJ	NA	3.6 U	1.4 J	18 UJ	19 UJ	NA
ENDOSULFAN SULFATE	NA	3.4 UJ	3.6 UJ	4.4 UJ	4 UJ	3.4 UJ	NA	3.6 U	3.7 UJ	18 UJ	19 UJ	NA
ENDRIN	NA	R	3.6 UJ	4.4 UJ	4 UJ	3.4 UJ	NA	3.6 U	3.7 UJ	18 UJ	19 UJ	NA
ENDRIN ALDEHYDE	NA	R	3.6 UJ	4.4 UJ	4 UJ	3.4 UJ	NA	3.6 U	3.7 UJ	18 UJ	19 UJ	NA
ENDRIN KETONE	NA	4.8 J	18 UJ	5.2 J	4 UJ	3.4 UJ	NA	3.6 U	0.81 J	18 UJ	19 UJ	NA
GAMMA-BHC (LINDANE)	NA	1.8 UJ	1.8 UJ	2.3 UJ	2.1 UJ	1.8 UJ	NA	1.8 U	1.9 UJ	9.4 UJ	9.8 UJ	NA
GAMMA-CHLORDANE	NA	1.2 J	1.8 UJ	2.3 UJ	2.1 UJ	1.8 UJ	NA	1.8 U	1.8 UJ	R	9.8 UJ	NA
HEPTACHLOR	NA	1.8 UJ	1.8 UJ	2.3 UJ	2.1 UJ	1.8 UJ	NA	1.8 U	1.8 UJ	1 J	9.8 UJ	NA
HEPTACHLOR EPOXIDE	NA	1.8 UJ	1.8 UJ	2.3 UJ	2.1 UJ	1.8 UJ	NA	1.8 U	1.9 UJ	9.4 UJ	9.8 UJ	NA
METHOXYCHLOR	NA	4.3 J	3.6 UJ	13 J	21 UJ	18 UJ	NA	18 U	2.2 J	84 UJ	88 UJ	NA
TOXAPHENE	NA	180 UJ	180 UJ	230 UJ	210 UJ	180 UJ	NA	180 U	180 UJ	940 UJ	980 UJ	NA

ANALYTICAL RESULTS
 DRAFT REMEDIAL INVESTIGATION REPORT
 RAYMARK-FERRY CREEK - OJ3
 STRATFORD, CONNECTICUT

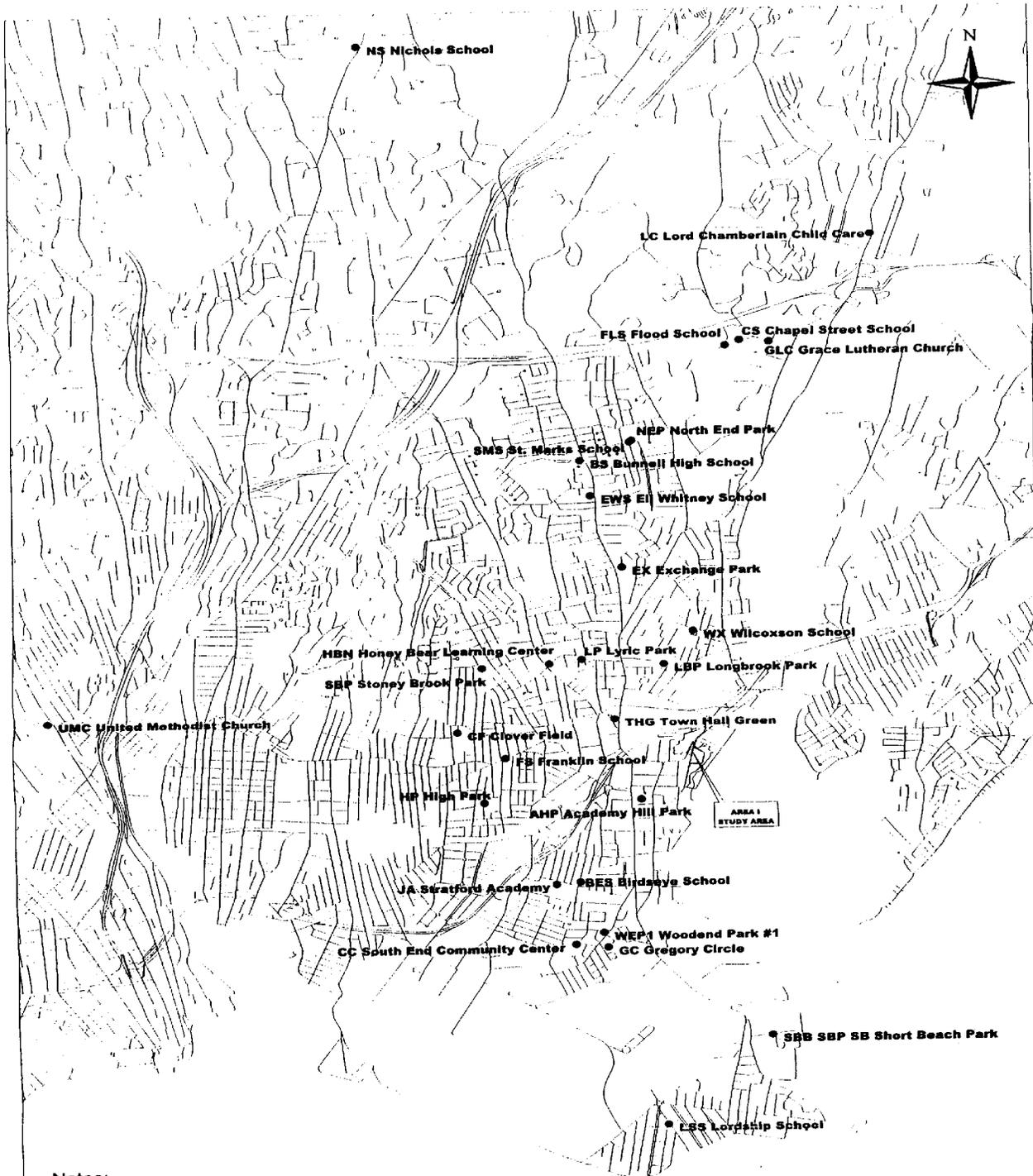
Sample Number	SBP005	SBPF+00	SH-97	SH-A+00	SH-D+695	SH-E+400	SH400	SMS-G3	SPB 005	THG005	THN-62	THN-G2		
Sample Location	SBP005	SBPF+00	SH-97	SH-A+00	SH-D+695	SH-E+400	SH400	SMS-G3	SPB 005	THG005	THN-62	THN-G2		
Date Sampled														
OC Type	None	None	None	None	None	None	None	None	None	None	None	None		
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	SOIL	None	SOIL	SOIL	SOIL	SOIL		
Filtration	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA		
Metals (MG/KG)														
ALUMINUM	7960 J	10800 J	18900	21200	16400	10300		NA	22600		NA	15800 J	NA	21200
ANTIMONY	6.4 UJ	5.6 UJ	9.8 UJ	12.6 UJ	5.6 UJ	10.1 UJ		NA		R	NA	5.9 UJ	NA	
ARSENIC	3.9	10.5	3.5	11.6	3.4	4.3		NA	8.4		NA	11.2	NA	6.9
BARIUM	29.4 J	53.1 J	35.7 J	41.8 J	57.1 J	40 J		NA	62.1 J		NA	58 J	NA	64.1 J
BERYLLIUM	0.61 J	0.62 J	0.76	1.1	0.81	0.43		NA	1.1		NA	0.7 J	NA	1.3
CADMIUM	0.45 UJ	0.73 UJ	0.65 U	0.82 U	0.74 U	0.63 U		NA	0.66 U		NA	0.47 UJ	NA	0.89 J
CALCIUM	850 J	2090 J	653 J	1390 J	2320 J	1310 J		NA	646 J		NA	2760 J	NA	3640 J
CHROMIUM	8.8	14.8	19.3	24.8	19.5	18.4		NA	17.3		NA	25.2	NA	18.9
COBALT	6.3	10.5	6.5 UJ	8.8 UJ	7.5 UJ	8.2 UJ		NA	9.5		NA	8	NA	8
COPPER	15.2	34.7	20.5 J	38.6 J	18.1 J	15.7 J		NA	18.5 J		NA	35.2	NA	37.9 J
IRON	15800 J	16900 J	17800	21500	18500	14400		NA	22800		NA	17800 J	NA	22800
LEAD	22.8	53	60.8 J	118 J	32 J	42.3 J		NA	21.7		NA	90.9	NA	124
MAGNESIUM	2270 J	3600 J	3730	4520	4190	4380		NA	3980		NA	3520 J	NA	4960
MANGANESE	185 J	272 J	213 J	567 J	812 J	234 J		NA	597		NA	328 J	NA	522
MERCURY	0.09 UJ	0.06 UJ	0.16 J	0.28	0.14 J	0.11 J		NA	0.11 U		NA	0.16 J	NA	0.12 U
NICKEL	11.8 U	15.6 U	13 J	19.8 J	14.3 J	18.6 J		NA	14.4 J		NA	17.1 U	NA	21.8 J
POTASSIUM	605 UJ	1260 J	1180	1170	1370	1710		NA	1720		NA	767 UJ	NA	1480
SELENIUM	0.33 UJ	0.31 UJ	1.3 J	2.2 J	0.96 U	0.84 U		NA	1.4		NA	0.33 UJ	NA	3.3 J
SILVER	0.38 U	0.31 U	1.3 UJ	1.8 UJ	1.7 UJ	1.5 UJ		NA	1.5 UJ		NA	0.58 J	NA	1.9 U
SODIUM	82.4	105	75.3 UJ	168 UJ	97.3 UJ	62.7 UJ		NA		R	NA	118	NA	
THALLIUM	0.17 UJ	0.16 J	1.5 U	1.6 U	1.7 U	1.5 U		NA	1.5 U		NA	0.16 U	NA	1.6 UJ
VANADIUM	21.3	29.6	49.6	81.6	45.1	30.6		NA	37.2 J		NA	34.5	NA	53.5 J
ZINC	35.2 J	66 J	45 J	87.6 J	76.1 J	49.6 J		NA	63.5 J		NA	77.8 J	NA	134 J

ANALYTICAL RESULTS
 DRAFT REMEDIAL INVESTIGATION REPORT
 RAYMARK-FERRY CREEK - OJ3
 STRATFORD, CONNECTICUT

Sample Number	UMC-92	WBG008	WEP-2-63	WEP-2-G3	WX-G3
Sample Location	UMC-92	WBG008	WEP-2-63	WEP-2-G3	WX-G3
Date Sampled					
QC Type	None	None	None	None	None
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL
Filtering	NA	NA	NA	NA	NA
Pesticides/PCBs (UG/KG)					
4,4'-DDD	NA	NA	NA	18 U	NA
4,4'-DDE	NA	NA	NA	18 U	NA
4,4'-DDT	NA	NA	NA	18 U	NA
ALDRIN	NA	NA	NA	9.3 U	NA
ALPHA-BHC	NA	NA	NA	9.3 U	NA
ALPHA-CHLORDANE	NA	NA	NA	9.3 U	NA
AROCLOR, TOTAL	NA	NA	NA	905 U	NA
AROCLOR-1016	NA	NA	NA	180 U	NA
AROCLOR-1221	NA	NA	NA	370 U	NA
AROCLOR-1232	NA	NA	NA	180 U	NA
AROCLOR-1242	NA	NA	NA	180 U	NA
AROCLOR-1248	NA	NA	NA	180 U	NA
AROCLOR-1254	NA	NA	NA	180 U	NA
AROCLOR-1260	NA	NA	NA	180 U	NA
AROCLOR-1262	NA	NA	NA	180 U	NA
AROCLOR-1268	NA	NA	NA	180 U	NA
BETA-BHC	NA	NA	NA	9.3 U	NA
DELTA-BHC	NA	NA	NA	9.3 U	NA
DIELDRIN	NA	NA	NA	18 U	NA
ENDOSULFAN I	NA	NA	NA	9.3 U	NA
ENDOSULFAN II	NA	NA	NA	18 U	NA
ENDOSULFAN SULFATE	NA	NA	NA	18 U	NA
ENDRIN	NA	NA	NA	18 U	NA
ENDRIN ALDEHYDE	NA	NA	NA	18 U	NA
ENDRIN KETONE	NA	NA	NA	18 U	NA
GAMMA-BHC (LINDANE)	NA	NA	NA	9.3 U	NA
GAMMA-CHLORDANE	NA	NA	NA	9.3 U	NA
HEPTACHLOR	NA	NA	NA	9.3 U	NA
HEPTACHLOR EPOXIDE	NA	NA	NA	9.3 U	NA
METHOXYCHLOR	NA	NA	NA	93 U	NA
TOXAPHENE	NA	NA	NA	930 U	NA

ANALYTICAL RESULTS
 DRAFT REMEDIAL INVESTIGATION REPORT
 RAYMARK-FERRY CREEK - OU3
 STRATFORD, CONNECTICUT

Sample Number	UMC-92	WBG008	WEP-2-63	WEP-2-G3	WX-G3
Sample Location	UMC-92	WBG008	WEP-2-63	WEP-2-G3	WX-G3
Date Sampled					
QC Type	None	None	None	None	None
MATRIX	SOIL	SOIL	SOIL	SOIL	SOIL
Filtering	NA	NA	NA	NA	NA
Metals (MG/KG)					
ALUMINUM	7120	12400 J	11600 J		NA 7840
ANTIMONY	4.8 UJ	5.7 UJ	6 UJ		NA 5 UJ
ARSENIC	4.2	8.5	4 J		NA 2 J
BARIUM	32.8 J	34.4	33.7		NA 33.2
BERYLLIUM	0.31 J	1	0.55 J		NA 0.35 J
CADMIUM	0.63 U	0.4 U	0.42 UJ		NA 0.62 UJ
CALCIUM	7420 J	775 J	1260 J		NA 1230 J
CHROMIUM	11.3	16.2	12.7		NA 12.3
COBALT	4.2 UJ	7	7.1		NA 4.7
COPPER	17 J	27.3 J	18.4 J		NA 8.2 J
IRON	11300	18100 J	12800 J		NA 11200
LEAD	65.4 J	192	44.8 J		NA 11.5
MAGNESIUM	4710	2760 J	2540 J		NA 2380
MANGANESE	212 J	253 J	216 J		NA 247
MERCURY	0.17 J	0.11 J	0.19 J		NA 0.1 U
NICKEL	9.7 J	12.8 J	11.2		NA 8.8 J
POTASSIUM	941	615 UJ	548 U		NA 1150
SELENIUM	0.84 U	2.1 UJ	0.33 UJ		NA 0.83 UJ
SILVER	1.5 UJ	0.33 U	0.33 U		NA 1.7 U
SODIUM	74.4 UJ	109	100		NA 77.7 J
THALLIUM	1.5 U	0.38 UJ	0.16 UJ		NA 1.4 UJ
VANADIUM	20.6	46.3	23		NA 21.3
ZINC	78.1 J	58.9 J	57.1 J		NA 40.8 J



Notes:
 All Locations Considered Approximate
 Plan Not to be Used For Design
 Coordinates Obtained from EPA Region I GIS
 Coordinates for THN Second Hill Lane School
 and SH Tree House Nursery Not Available



SOIL BACKGROUND LOCATIONS
 DRAFT REMEDIAL INVESTIGATION REPORT - AREA I
 RAYMARK - FERRY CREEK - OU3
 STRATFORD, CONNECTICUT



TETRA TECH NUS, INC.

Drawn By: D.A. Chisholm
 Scale: As Shown

Date: February 27, 1999
 File: ...Raymark\OU3FIGS.APR

55 JONSPIN ROAD WILMINGTON, MA 01887
 (978)658-7899

Appendix F.4

**State of Connecticut
Water Quality Standards**

TABLE F.4.1
STATE OF CONNECTICUT WATER QUALITY STANDARDS
AREA B, DELBUONO WETLANDS
SURFACE WATER
FERRY CREEK, STRATFORD, CT
PAGE 1 OF 2

Parameter	Frequency	Range Of Detects	Range Of Nondetects	Average	Location of Maximum	State WQS Freshwater Chronic ⁽¹⁾	State WQS Saltwater Chronic ⁽¹⁾	State WQS Water Only ⁽¹⁾	State WQS Water and Organisms ⁽¹⁾
Volatiles (ug/L)									
1,1,1-TRICHLOROETHANE	4/26	12 - 160	10 - 10	13	RM-SW-SD20-04			3100	
1,1-DICHLOROETHANE	3/26	4 - 28	10 - 10	6	RM-SW-SD20-04				
1,1-DICHLOROETHENE	4/26	4 - 50	10 - 10	7	RM-SW-SD20-04				
1,2-DICHLOROETHENE (TOTAL)	4/26	5 - 59	10 - 10	8	RM-SW-SD20-04				
ACETONE	1/26	37 - 37	10 - 10	6	RM-SW-SD19-03				
CHLOROBEZENE	2/26	1 - 3	10 - 10	5	RM-SW-SD20-04			680	21000
CHLOROFORM	1/26	4 - 4	10 - 10	5	RM-SW-SD20-04			5.7	470
CHLOROMETHANE	1/26	16 - 16	10 - 10	5	RM-SW-SD19-03			5.7	470
TRICHLOROETHENE	3/26	4 - 49	10 - 20	7	RM-SW-SD20-04				81
VINYL CHLORIDE	2/26	5 - 7	10 - 10	5	RM-SW-SD20-04				525
Semivolatiles (ug/L)									
BIS(2-ETHYLHEXYL)PHTHALATE	2/26	11.5 - 29	10 - 10	6	RM-SW-SD09-02				
DIETHYL PHTHALATE	1/26	0.6 - 0.6	10 - 10	5	RM-SW-SD20-03			23000	120000
FLUORANTHENE	1/26	0.9 - 0.9	10 - 10	5	RM-SW-SD20-03			300	370
PHENANTHRENE	1/26	0.5 - 0.5	10 - 10	5	RM-SW-SD20-03				
PYRENE	1/26	0.6 - 0.6	10 - 10	5	RM-SW-SD20-03			960	11000
Pesticides/PCBs (ug/L)									
4,4'-DDD	4/26	0.002 - 0.004	0.1 - 0.1	0.04	RM-SW-SD09-04				
ALDRIN	1/26	0.0007 - 0.0007	0.05 - 0.05	0.02	RM-SW-SD20-04	1.5	0.65		
DIELDRIN	2/26	0.0007 - 0.0255	0.05 - 0.1	0.04	RM-SW-SD19-04				
ENDOSULFAN II	1/26	0.004 - 0.004	0.1 - 0.1	0.05	RM-SW-SD20-03	0.056	0.0087	0.93	2
ENDOSULFAN SULFATE	1/26	0.012 - 0.012	0.1 - 0.1	0.05	RM-SW-SD20-03			0.93	2
ENDRIN ALDEHYDE	1/26	0.006 - 0.006	0.1 - 0.1	0.05	RM-SW-SD19-03			0.76	0.81
ENDRIN KETONE	1/26	0.002 - 0.002	0.1 - 0.1	0.05	RM-SW-SD09-03				
GAMMA-BHC (LINDANE)	1/26	0.013 - 0.013	0.05 - 0.05	0.02	RM-SW-SD19-04	0.08	0.08	0.019	0.063
GAMMA-CHLORDANE	1/26	0.004 - 0.004	0.05 - 0.05	0.02	RM-SW-SD31-04	0.0043			
HEPTACHLOR	1/26	0.002 - 0.002	0.05 - 0.05	0.02	RM-SW-SD08-01	0.0038	0.0036		
HEPTACHLOR EPOXIDE	2/26	0.0008 - 0.002	0.05 - 0.05	0.02	RM-SW-SD37-04	0.0038	0.0036		
METHOXYCHLOR	2/26	0.007 - 0.03	0.1 - 0.5	0.22	RM-SW-SD08-01				
Inorganics (ug/L)									
ALUMINIUM	6/26	31.6 - 792	34.6 - 219	94	RM-SW-SD08-02				
ANTIMONY	3/26	15 - 27.4	5 - 40.9	10	RM-SW-SD28-04				4300
ARSENIC	9/26	3.9 - 93.4	1.8 - 66	15	RM-SW-SD37-04	190			
BARIIUM	23/26	5.5 - 52.1	8.9 - 10.2	15	RM-SW-SD20-04				
CALCIUM	25/26	9670 - 319000	241000 - 241000	189299	RM-SW-SD09-01				
CHROMIUM	15/26	7.7 - 16.4	3.2 - 5	8	RM-SW-SD31-04		50	170	3400
COBALT	1/26	9.5 - 9.5	2 - 3.4	2	RM-SW-SD09-03				
COPPER	4/26	18.3 - 65.4	3 - 52.4	15	RM-SW-SD20-03				
IRON	20/26	133 - 1570	38.4 - 190	321	RM-SW-SD08-02				
LEAD	8/26	2.3 - 16.7	1.1 - 42	8	RM-SW-SD20-03			50	
MAGNESIUM	26/26	17300 - 922000	-	599669	RM-SW-SD32-04				

TABLE F.4.1
 STATE OF CONNECTICUT WATER QUALITY STANDARDS
 AREA B, DELBUONO WETLANDS
 SURFACE WATER
 FERRY CREEK, STRATFORD, CT
 PAGE 2 OF 2

Parameter	Frequency	Range Of Detects	Range Of Nondetects	Average	Location of Maximum	State WQS Freshwater Chronic ⁽¹⁾	State WQS Saltwater Chronic ⁽¹⁾	State WQS Water Only ⁽¹⁾	State WQS Water and Organisms ⁽¹⁾
MANGANESE	26/26	4.9 - 976	-	141	RM-SW-SD20-04				
MERCURY	10/26	0.27 - 1.95	0.1 - 0.2	0.39	RM-SW-SD19-04				
POTASSIUM	26/26	7330 - 337000	-	202082	RM-SW-SD08-02				
SELENIUM	2/26	2.6 - 3.3	2.5 - 50	8	RM-SW-SD28-04	5	71	100	6800
SODIUM	26/26	144000 - 8720000	-	5048192	RM-SW-SD31-04				
THALLIUM	2/26	7.7 - 9.1	2.7 - 104	17	RM-SW-SD19-04				
VANADIUM	7/26	2.4 - 4.6	2 - 10.9	2	RM-SW-SD31-04				
ZINC	5/18	27.7 - 179	4.5 - 31.4	28	RM-SW-SD20-03				

(1) State of Connecticut Department of Environmental Protection, Water Quality Standards, Appendix D: Numerical Water Quality Criteria for Chemical Constituents, March 17, 1997.

TABLE F.4.2
STATE OF CONNECTICUT WATER QUALITY STANDARDS
AREA C, HOUSATONIC BOAT CLUB WETLANDS AREA
SURFACE WATER
FERRY CREEK, STRATFORD, CT
PAGE 1 OF 1

Parameter	Frequency	Range Of Detects	Range Of Nondetects	Average	Location of Maximum	State WQS Freshwater Chronic ⁽¹⁾	State WQS Saltwater Chronic ⁽¹⁾	State WQS Water Only ⁽¹⁾	State WQS Water and Organisms ⁽¹⁾
Volatiles (ug/L)									
CHLOROMETHANE	1/19	1 - 1	10 - 10	5	RM-SW-HB14-02			5.7	470
Semivolatiles (ug/L)									
CHRYSENE	1/19	0.6 - 0.6	10 - 10	5	RM-SW-HB01-01				
DI-N-BUTYL PHTHALATE	1/19	0.8 - 0.8	10 - 10	5	RM-SW-HB06-01			2700	12000
FLUORANTHENE	1/19	0.5 - 0.5	10 - 10	5	RM-SW-HB01-01			300	370
PYRENE	1/19	0.8 - 0.8	10 - 10	5	RM-SW-HB01-01			960	11000
Pesticides/PCBs (ug/L)									
4,4'-DDD	1/19	0.005 - 0.005	0.1 - 0.1	0.05	RM-SW-HB20-03				
ALPHA-BHC	3/19	0.0023 - 0.0053	0.05 - 0.05	0.02	RM-SW-HB14-02				0.013
AROCLOR-1268	4/19	0.14 - 0.19	0.5 - 1	0.27	RM-SW-HB3A-02		0.03		
BETA-BHC	1/19	0.008 - 0.008	0.05 - 0.05	0.02	RM-SW-HB01-01			0.014	0.046
DIELDRIN	3/19	0.0023 - 0.0047	0.1 - 0.1	0.04	RM-SW-HB01-02			0.00014	0.00014
ENDOSULFAN SULFATE	3/19	0.0054 - 0.011	0.1 - 0.1	0.04	RM-SW-HB20-03			0.93	2
ENDRIN	1/19	0.009 - 0.009	0.1 - 0.1	0.05	RM-SW-HB20-03			0.76	0.81
ENDRIN ALDEHYDE	1/19	0.005 - 0.005	0.05 - 0.1	0.04	RM-SW-HB21-03			0.76	0.81
GAMMA-BHC (LINDANE)	1/19	0.001 - 0.001	0.05 - 0.05	0.02	RM-SW-HB06-04	0.08	0.08	0.019	0.063
METHOXYCHLOR	1/19	0.0026 - 0.0026	0.1 - 0.5	0.21	RM-SW-HB07-01				
Inorganics (ug/L)									
ALUMINIUM	12/19	110 - 4010	25 - 130	785	RM-SW-HB12-04				
ANTIMONY	4/19	4.2 - 29.4	5 - 26.3	7	RM-SW-HB23-04				4300
ARSENIC	5/19	3.6 - 51.1	1.8 - 33.1	11	RM-SW-HB01-01	190			
BARIUM	16/19	11.1 - 67.7	7.7 - 8.3	21	RM-SW-HB01-02				
CADMIUM	3/19	1.5 - 2.5	1.4 - 2.4	1	RM-SW-HB01-02		9.3	16	170
CALCIUM	19/19	109000 - 263000	-	186816	RM-SW-HB06 & 12-04				
CHROMIUM	9/19	6.4 - 59.2	5 - 7	12	RM-SW-HB12-04			170	3400
COBALT	1/19	2 - 2	2 - 5.7	1	RM-SW-HB02-01				
COPPER	13/19	2.4 - 286	3 - 54.1	59	RM-SW-HB01-02				
IRON	19/19	149 - 6710	-	1290	RM-SW-HB12-04				
LEAD	11/19	3 - 147	3 - 7.8	27	RM-SW-HB02-01				
MAGNESIUM	19/19	295000 - 873000	-	574974	RM-SW-HB12-04				
MANGANESE	19/19	18 - 750	-	180	RM-SW-HB12-04				
MERCURY	3/19	0.57 - 3.5	0.1 - 0.2	0.41	RM-SW-HB12-04				
NICKEL	1/19	41 - 41	3.6 - 15.4	7	RM-SW-HB01-02	88		610	4600
POTASSIUM	19/19	129000 - 344000	-	258974	RM-SW-HB11-02				
SODIUM	19/19	738000 - 7510000	-	5423684	RM-SW-HB10-02				
THALLIUM	1/19	8.9 - 8.9	4.2 - 9.8	4	RM-SW-HB02-02				
VANADIUM	8/18	2.1 - 16.8	2 - 11.3	4	RM-SW-HB12-04				
ZINC	8/17	23.2 - 128	4.4 - 62	34	RM-SW-HB01-02				

(1) State of Connecticut Department of Environmental Protection, Water Quality Standards, Appendix D: Numerical Water Quality Criteria for Chemical Constituents, March 17, 1997.

TABLE F.4.3
 STATE OF CONNECTICUT WATER QUALITY STANDARDS
 AREA F, SELBY POND
 SURFACE WATER
 FERRY CREEK, STRATFORD, CT
 PAGE 1 OF 1

Parameter	Frequency	Range Of Detects	Range Of Nondetects	Average	Location of Maximum	State WQS Freshwater Chronic ⁽¹⁾	State WQS Saltwater Chronic ⁽¹⁾	State WQS Water Only ⁽¹⁾	State WQS Water and Organisms ⁽¹⁾
Pesticides/PCBs (ug/L)									
ENDOSULFAN I	1/4	0.013 - 0.013	0.05 - 0.05	0.02	RM-SW-SP04-03	0.056		0.93	2
ENDOSULFAN SULFATE	1/4	0.028 - 0.028	0.1 - 0.1	0.04	RM-SW-SP04-03			0.93	2
ENDRIN ALDEHYDE	1/4	0.028 - 0.028	0.1 - 0.1	0.04	RM-SW-SP04-03			0.76	0.81
HEPTACHLOR EPOXIDE	1/4	0.003 - 0.003	0.05 - 0.05	0.02	RM-SW-SP01-01	0.0038	0.0036		
Inorganics (ug/L)									
ALUMINUM	2/11	89.7 - 561	40 - 342	95	RM-SW-SP19-04				
ANTIMONY	1/11	4.4 - 4.4	5 - 26.3	4	RM-SW-SP14-04				
ARSENIC	1/11	6 - 6	1.8 - 11.4	4	RM-SW-SP14-04	190	36	14	4300
BARIUM	5/11	8.4 - 10.6	14.4 - 21.5	9	RM-SW-SP14-04				
BERYLLIUM	1/10	0.75 - 0.75	1 - 1	1	RM-SW-SP14-04				
CADMIUM	1/11	0.75 - 0.75	1 - 2.4	1	RM-SW-SP14-04		9.3	16	170
CALCIUM	11/11	164000 - 280000	-	207636	RM-SW-SP01-01				
CHROMIUM	1/11	2.4 - 2.4	1.4 - 10.4	3	RM-SW-SP14-04	11	50	170	3400
COBALT	1/7	1 - 1	1 - 7.4	1	RM-SW-SP14-04				
COPPER	1/11	17.3 - 17.3	3 - 46.6	12	RM-SW-SP14-04				
IRON	5/11	159 - 1450	312 - 1080	430	RM-SW-SP14-04				
LEAD	1/11	3.3 - 3.3	2 - 27.6	3	RM-SW-SP14-04		8.5	50	
MAGNESIUM	11/11	519000 - 888000	-	654455	RM-SW-SP01-01				
MANGANESE	10/11	26.8 - 149	5 - 5	58	RM-SW-SP03-01				
MERCURY	1/11	0.15 - 0.15	0.1 - 0.2	0.1	RM-SW-SP14-04				
NICKEL	1/7	27.3 - 27.3	10 - 21.9	9	RM-SW-SP14-04	88		610	4600
POTASSIUM	11/11	231500 - 404000	-	339341	RM-SW-SP16-04				
SELENIUM	1/11	3 - 3	2.5 - 4	2	RM-SW-SP14-04	5	71	100	6800
SILVER	4/11	2.8 - 9	2 - 20.5	4	RM-SW-SP08-04			105	65000
SODIUM	11/11	4250000 - 9040000	-	5333636	RM-SW-SP01-01				
THALLIUM	1/11	6.8 - 6.8	4.9 - 9	4	RM-SW-SP14-04				
VANADIUM	7/11	1.3 - 4	2 - 8.6	2	RM-SW-SP18-04				
ZINC	1/11	48.5 - 48.5	4 - 129	29	RM-SW-SP14-04		86		

(1) State of Connecticut Department of Environmental Protection, Water Quality Standards, Appendix D: Numerical Water Quality Criteria for Chemical Constituents, March 17, 1997.

Appendix F.5

Sample Subsets

TABLE F-5.1
 SAMPLE LIST AREA B
 CURRENT/FUTURE COMMERCIAL WORKERS

AOC	RECEPTOR	MATRIX	BORING
B	bscm	SOIL	DBL-018
B	bscm	SOIL	DBL017
B	bscm	SOIL	SA640 A+160
B	bscm	SOIL	SA654 A+164
B	bscm	SOIL	SA654 B+164
B	bscm	SOIL	SA654 C+164
B	bscm	SOIL	SA654A N153,E164
B	bscm	SOIL	SA654A N172,E164
B	bscm	SOIL	SA658 A+050
B	bscm	SOIL	SA658 A+075
B	bscm	SOIL	SA658 B+075
B	bscm	SOIL	SA658 C+050
B	bscm	SOIL	SA658 C+075
B	bscm	SOIL	SA658 C+155
B	bscm	SOIL	SA666 A+136
B	bscm	SOIL	SA666 B+136
B	bscm	SOIL	SA666 C+136
B	bscm	SOIL	SA666A N80,E123
B	bscm	SOIL	SA674 A+056
B	bscm	SOIL	SA674 A+112
B	bscm	SOIL	SA674 B+066
B	bscm	SOIL	SA674 B+112
B	bscm	SOIL	SA674 C+112
B	bscm	SOIL	SA674A N12,E12
B	bscm	SOIL	SA674A N13,E85
B	bscm	SOIL	SA674A N37,E103

TABLE F-5.2
SAMPLE LIST AREA B
FUTURE COMMERCIAL WORKERS - AREA 1

AOC	RECEPTOR	MATRIX	BORING
B	bscn1	SOIL	B2-SB07
B	bscn1	SOIL	DBL-018
B	bscn1	SOIL	DBL017
B	bscn1	SOIL	SA640 A+160
B	bscn1	SOIL	SA654 A+164
B	bscn1	SOIL	SA654 B+164
B	bscn1	SOIL	SA654 C+164
B	bscn1	SOIL	SA654A N153,E164
B	bscn1	SOIL	SA654A N172,E164
B	bscn1	SOIL	SA658 A+050
B	bscn1	SOIL	SA658 A+075
B	bscn1	SOIL	SA658 B+075
B	bscn1	SOIL	SA658 C+050
B	bscn1	SOIL	SA658 C+075
B	bscn1	SOIL	SA658 C+155
B	bscn1	SOIL	SA666 A+136
B	bscn1	SOIL	SA666 B+136
B	bscn1	SOIL	SA666 C+136
B	bscn1	SOIL	SA666A N80,E123
B	bscn1	SOIL	SA674 A+056
B	bscn1	SOIL	SA674 A+112
B	bscn1	SOIL	SA674 B+066
B	bscn1	SOIL	SA674 B+112
B	bscn1	SOIL	SA674 C+112
B	bscn1	SOIL	SA674A N12,E12
B	bscn1	SOIL	SA674A N13,E85
B	bscn1	SOIL	SA674A N27,E68
B	bscn1	SOIL	SA674A N37,E103

TABLE F-5.3
 SAMPLE LIST AREA B
 FUTURE COMMERCIAL WORKERS - AREA 2

AOC	RECEPTOR	MATRIX	BORING
B	bscn2	SOIL	B2-SB01
B	bscn2	SOIL	B2-SB02
B	bscn2	SOIL	B2-SB04
B	bscn2	SOIL	DBL-001
B	bscn2	SOIL	DBL-003
B	bscn2	SOIL	DBL-004
B	bscn2	SOIL	DBL-005
B	bscn2	SOIL	DBL-006
B	bscn2	SOIL	DBL-008
B	bscn2	SOIL	DBL-009
B	bscn2	SOIL	DBL-010
B	bscn2	SOIL	DBL-011
B	bscn2	SOIL	DBL-012
B	bscn2	SOIL	DBL-013
B	bscn2	SOIL	DBL-014
B	bscn2	SOIL	DBL007
B	bscn2	WETLAND	B2-SB03
B	bscn2	WETLAND	B2-SB08
B	bscn2	WETLAND	DB01
B	bscn2	WETLAND	DB02
B	bscn2	WETLAND	DB03
B	bscn2	WETLAND	DB04
B	bscn2	WETLAND	DB05
B	bscn2	WETLAND	DBL-002
B	bscn2	WETLAND	DBL-016

TABLE F-5.4
 SAMPLE LIST AREA B
 WETLANDS/MARSH RECEPTORS

AOC	RECEPTOR	MATRIX	BORING
B	bsir	SEDIMENT	B2-SD04
B	bsir	SEDIMENT	B2-SD05
B	bsir	SEDIMENT	HR24
B	bsir	SEDIMENT	HR25
B	bsir	SEDIMENT	HR26
B	bsir	SEDIMENT	HR27
B	bsir	SEDIMENT	HR29
B	bsir	SEDIMENT	SD05W
B	bsir	SEDIMENT	SD07
B	bsir	SEDIMENT	SD09
B	bsir	SEDIMENT	SD25
B	bsir	SEDIMENT	SD26
B	bsir	SEDIMENT	SD27
B	bsir	SEDIMENT	SD28
B	bsir	SEDIMENT	SD29
B	bsir	SEDIMENT	SD30
B	bsir	WETLAND	SM C+240
B	bsir	WETLAND	SM-004
B	bsir	WETLAND	SM-005
B	bsir	WETLAND	SM-006
B	bsir	WETLAND	SM-007
B	bwir	SW	SD07
B	bwir	SW	SD09
B	bwir	SW	SD25
B	bwir	SW	SD28
B	bwir	SW	SD29
B	bwir	SW	SD30

TABLE F-5.5
SAMPLE LIST AREA B
ADOLESCENT TRESPASSERS

AOC	RECEPTOR	MATRIX	BORING
B	bstr	SEDIMENT	B2-SD03
B	bstr	SEDIMENT	B2-SD11
B	bstr	SEDIMENT	SD08
B	bstr	SEDIMENT	SD19-03
B	bstr	SEDIMENT	SD19-04
B	bstr	SEDIMENT	SD20-03
B	bstr	SEDIMENT	SD26
B	bstr	SOIL	B2-SB01
B	bstr	SOIL	B2-SB02
B	bstr	SOIL	B2-SB04
B	bstr	SOIL	DBL-001
B	bstr	SOIL	DBL-003
B	bstr	SOIL	DBL-004
B	bstr	SOIL	DBL-005
B	bstr	SOIL	DBL-006
B	bstr	SOIL	DBL-008
B	bstr	SOIL	DBL-009
B	bstr	SOIL	DBL-010
B	bstr	SOIL	DBL-011
B	bstr	SOIL	DBL-012
B	bstr	SOIL	DBL-013
B	bstr	SOIL	DBL-014
B	bstr	SOIL	DBL-015
B	bstr	SOIL	DBL007
B	bstr	WETLAND	B2-SB03
B	bstr	WETLAND	B2-SB05
B	bstr	WETLAND	B2-SB06
B	bstr	WETLAND	B2-SB08
B	bstr	WETLAND	B2-SB09
B	bstr	WETLAND	DB01
B	bstr	WETLAND	DB02
B	bstr	WETLAND	DB03
B	bstr	WETLAND	DB04
B	bstr	WETLAND	DB05
B	bstr	WETLAND	DB06
B	bstr	WETLAND	DB07
B	bstr	WETLAND	DB08
B	bstr	WETLAND	DB09
B	bstr	WETLAND	DB10
B	bstr	WETLAND	DBL-002
B	bstr	WETLAND	DBL-016
B	bstr	WETLAND	DBL-019
B	bstr	WETLAND	DBL-020
B	bstr	WETLAND	DBL-021
B	bstr	WETLAND	DBL-022
B	bstr	WETLAND	DBL-023
B	bstr	WETLAND	DBL-024
B	bstr	WETLAND	DBL-025
B	bstr	WETLAND	DBL-026
B	bstr	WETLAND	DBL027

TABLE F-5.6
 SAMPLE LIST AREA B
 LOCAL FISHERMEN INGESTING OYSTERS

AOC	RECEPTOR	MATRIX	BORING
B	bbolf	BIOTA	FC01-8244
B	bbolf	BIOTA	FC01-8308
B	bbolf	BIOTA	FC05-8301
B	bbolf	BIOTA	FC05-8305
B	bbolf	BIOTA	FC06-8044
B	bbolf	BIOTA	FC06-8295
B	bbolf	BIOTA	FC06-8296
B	bbolf	BIOTA	FC07-8273
B	bbolf	BIOTA	FC07-8276
B	bbolf	BIOTA	FC08-8278
B	bbolf	BIOTA	FC08-8285/86
B	bbolf	BIOTA	FC08-8287
B	bbolf	BIOTA	FC09-8042
B	bbolf	BIOTA	FC09-8054
B	bbolf	BIOTA	FC09-8269
B	bbolf	BIOTA	FC10-8252
B	bbolf	BIOTA	FC10-8256
B	bbolf	BIOTA	FC10-8258
B	bbolf	BIOTA	FC11-8051
B	bbolf	BIOTA	FC11-8260
B	bbolf	BIOTA	FC11-8262
B	bbolf	BIOTA	FC12-8040
B	bbolf	BIOTA	FC12-8052
B	bbolf	BIOTA	FC12-8261

TABLE F-5.7
 SAMPLE LIST AREA B
 LOCAL FISHERMEN INGESTING MUSSELS

AOC	RECEPTOR	MATRIX	BORING
B	bbmlf	BIOTA	FC01-8246/49/50
B	bbmlf	BIOTA	FC01-8248
B	bbmlf	BIOTA	FC06-8294
B	bbmlf	BIOTA	FC06-8297
B	bbmlf	BIOTA	FC06-8299
B	bbmlf	BIOTA	FC07-8272
B	bbmlf	BIOTA	FC07-8275
B	bbmlf	BIOTA	FC08-8284
B	bbmlf	BIOTA	FC08-8288
B	bbmlf	BIOTA	FC09-8049
B	bbmlf	BIOTA	FC09-8263
B	bbmlf	BIOTA	FC09-8279
B	bbmlf	BIOTA	FC09-8280
B	bbmlf	BIOTA	FC09-8280D
B	bbmlf	BIOTA	FC10-8039/253/282
B	bbmlf	BIOTA	FC10-8257
B	bbmlf	BIOTA	FC10-8259
C	bbmlf	BIOTA	C-1
C	bbmlf	BIOTA	C-2
C	bbmlf	BIOTA	C-3
C	bbmlf	BIOTA	HB-9
D	bbmlf	BIOTA	D-1
D	bbmlf	BIOTA	D-2
D	bbmlf	BIOTA	D-3
D	bbmlf	BIOTA	D-4
D	bbmlf	BIOTA	D-6

TABLE F-5.8
SAMPLE LIST AREA C
WETLAND/MARSH RECEPTOR

AOC	RECEPTOR	MATRIX	BORING
C	csir	SEDIMENT	C-1
C	csir	SEDIMENT	C-2
C	csir	SEDIMENT	C-3
C	csir	SEDIMENT	C-SD01
C	csir	SEDIMENT	C-SD02
C	csir	SEDIMENT	C-SD03
C	csir	SEDIMENT	C-SD04
C	csir	SEDIMENT	CSD1
C	csir	SEDIMENT	HB-1
C	csir	SEDIMENT	HB-2
C	csir	SEDIMENT	HB-3
C	csir	SEDIMENT	HB-4
C	csir	SEDIMENT	HB-5
C	csir	SEDIMENT	HB-6
C	csir	SEDIMENT	HB-7
C	csir	SEDIMENT	HB-8
C	csir	SEDIMENT	HB-9
C	csir	SEDIMENT	HB05
C	csir	SEDIMENT	HB06
C	csir	SEDIMENT	HB07
C	csir	SEDIMENT	HB10
C	csir	SEDIMENT	HB12
C	csir	SEDIMENT	HB13
C	csir	SEDIMENT	HB14
C	csir	SEDIMENT	HB15
C	csir	SEDIMENT	HB20
C	csir	SEDIMENT	HB21
C	csir	SEDIMENT	HB22
C	csir	SEDIMENT	HB24
C	csir	SEDIMENT	HB3A
C	csir	SOIL	1564ELM A+100
C	csir	SOIL	1564ELM B+100
C	csir	SOIL	1564ELM B+135
C	csir	SOIL	1564ELM B+145
C	csir	SOIL	1564ELM G1
C	csir	SOIL	ES1564 001
C	csir	SOIL	ES1564 003
C	csir	SOIL	ES1564 A+094
C	csir	SOIL	ES1564 A+132
C	csir	SOIL	ES1564 A+145 (W)
C	csir	SOIL	ES1564 B+124
C	csir	SOIL	ES1564 BC+145 (W)
C	csir	SOIL	ES1564 C+123
C	csir	SOIL	ES1564 CD+145 (W)
C	csir	SOIL	ES1564 CD+170 (W)
C	csir	SOIL	ES1564 D+080 (W)
C	csir	SOIL	HWCO A+500
C	csir	SOIL	HWCO AA+500
C	csir	SOIL	HWCO AB+200
C	csir	SOIL	HWCO AB+300
C	csir	SOIL	HWCO AB+600

**TABLE F-5.8
SAMPLE LIST AREA C
WETLAND/MARSH RECEPTOR**

AOC	RECEPTOR	MATRIX	BORING
C	csir	SOIL	HWCO B+250
C	csir	SOIL	HWCO B+375
C	csir	SOIL	HWCO BC+050
C	csir	SOIL	HWCO BC+225
C	csir	SOIL	HWCO BC+625
C	csir	SOIL	HWCO C+025
C	csir	SOIL	HWCO C+225
C	csir	SOIL	HWCO C+600
C	csir	SOIL	HWCO CD+075
C	csir	SOIL	HWCO CD+225
C	csir	SOIL	HWCO CD+570
C	csir	SOIL	HWCO D+050
C	csir	SOIL	HWCO D+135
C	csir	SOIL	HWCO D+225
C	csir	SOIL	HWCO DE+610
C	csir	WETLAND	BC-SB2
C	csir	WETLAND	BC-SB8
C	csir	WETLAND	C-SS02
C	csir	WETLAND	HB01
C	csir	WETLAND	HB02
C	csir	WETLAND	HB03
C	csir	WETLAND	HB04
C	csir	WETLAND	HB08
C	csir	WETLAND	HB09
C	csir	WETLAND	HB11
C	csir	WETLAND	HB16
C	csir	WETLAND	HB17
C	csir	WETLAND	HB18
C	csir	WETLAND	HB19
C	csir	WETLAND	HB23
C	csir	WETLAND	HB3A
C	csir	WETLAND	HB4A
C	csir	WETLAND	HB8A
C	csir	WETLAND	HBC 001
C	csir	WETLAND	HBC 002
C	csir	WETLAND	HBC 003
C	csir	WETLAND	HBC C+00
C	csir	WETLAND	HBC D+00
C	csir	WETLAND	HBC E+00
C	csir	WETLAND	HBCBB+1150
C	csir	WETLAND	HWCO A+400
C	csir	WETLAND	HWCO A+600
C	csir	WETLAND	HWCO AA+460
C	csir	WETLAND	HWCO AA+550
C	csir	WETLAND	HWCO B+650
C	cwir	SW	HB01
C	cwir	SW	HB02
C	cwir	SW	HB05
C	cwir	SW	HB06
C	cwir	SW	HB07
C	cwir	SW	HB10

TABLE F-5.8
SAMPLE LIST AREA C
WETLAND/MARSH RECEPTOR

AOC	RECEPTOR	MATRIX	BORING
C	cwir	SW	HB11
C	cwir	SW	HB12
C	cwir	SW	HB14
C	cwir	SW	HB15
C	cwir	SW	HB20
C	cwir	SW	HB21
C	cwir	SW	HB22
C	cwir	SW	HB23
C	cwir	SW	HB3A

TABLE F-5.9
 SAMPLE LIST AREA F
 FREQUENT RECREATIONAL USERS

AOC	RECEPTOR	MATRIX	BORING
F	fsfr	WETLAND	SP04
F	fsfr	WETLAND	SP05
F	fsfr	WETLAND	SP06
F	fsfr	WETLAND	SP07
F	fsfr	WETLAND	SP18
F	fsfr	WETLAND	SP19
F	fwfr	SW	SP01
F	fwfr	SW	SP02
F	fwfr	SW	SP03
F	fwfr	SW	SP04
F	fwfr	SW	SP08
F	fwfr	SW	SP14
F	fwfr	SW	SP15
F	fwfr	SW	SP16
F	fwfr	SW	SP17
F	fwfr	SW	SP18
F	fwfr	SW	SP19

TABLE F-5.10
SAMPLE LIST AREA F
LOCAL FISHERMEN INGESTING EELS

AOC	RECEPTOR	MATRIX	BORING
F	fbelf	BIOTA	SP-AE-F-1
F	fbelf	BIOTA	SP-AE-F-10
F	fbelf	BIOTA	SP-AE-F-2
F	fbelf	BIOTA	SP-AE-F-3
F	fbelf	BIOTA	SP-AE-F-4
F	fbelf	BIOTA	SP-AE-F-5
F	fbelf	BIOTA	SP-AE-F-6
F	fbelf	BIOTA	SP-AE-F-7
F	fbelf	BIOTA	SP-AE-F-8
F	fbelf	BIOTA	SP-AE-F-9

TABLE F-5.11
SAMPLE LIST AREA F
LOCAL FISHERMEN INGESTING WHITE PERCH

AOC	RECEPTOR	MATRIX	BORING
F	fbplf	BIOTA	SP-PF-F-1
F	fbplf	BIOTA	SP-PF-F-2
F	fbplf	BIOTA	SP-PF-F-3
F	fbplf	BIOTA	SP-PF-F-4
F	fbplf	BIOTA	SP-PF-F-5
F	fbplf	BIOTA	SP-PF-F-6
F	fbplf	BIOTA	SP-PF-F-7
F	fbplf	BIOTA	SP-PF-F-8

Appendix F.6

**Sample Calculations for UCL
Statistics**

APPENDIX F.6

STATISTICS AND EXPOSURE POINT CONCENTRATIONS

1 INTRODUCTION

This appendix presents the methodology for statistical analysis of environmental data collected at the site. Tables 1 through 4 are referenced statistical tables.

The statistical methods presented were used to develop summary statistics (e.g., range, mean, standard deviation, 95% Upper Confidence Limits) which describe environmental contaminant concentrations at the facility.

The statistical methods presented were based on:

1. EPA Publication 9285.7-081. "Supplemental Guidance to RAGS: Calculating the Concentration Term." May 1992.
2. Gilbert, Richard O., Statistical Methods for Environmental Pollution Monitoring. Van Nostrand Reinhold Company. New York, New York. 1987.
3. Cochran, William G. and Snedecor, George W. Statistical Methods. The Iowa State University Press. 1980.

2 LIMIT OF DETECTION

In the chemical analysis of environmental samples, some analytes may be present at concentrations which are below the sample quantitation limit (SQL) of the analytical procedure. The results are generally reported as not detected (rather than zero), and the appropriate limit of detection is given. The nondetects were replaced with the SQL divided by two prior to statistical analysis. Clearly, if all the observations are nondetect results, no statistical analysis is warranted. In addition, field duplicate results were processed prior to use in statistical analysis. The maximum value was used for solid matrix duplicates. The average value was used for aqueous duplicates.

3 STATISTICAL METHODS

3.1.1 The Shapiro and Wilk "W-test"

The data must be analyzed to determine whether they were drawn from an underlying normal, lognormal or undetermined distribution. A number of statistical evaluations may be used to determine which, if either, of the distributions are exhibited by a given data set. As recommended by the EPA, the Shapiro and Wilk "W-test" (for sample sets ≤ 50) and the Shapiro-Francia "W-test" (for sample sets > 50) will be used to determine whether the data are normally or lognormally distributed (EPA, 1992).

The null hypothesis (H_0) that is tested is that the population has a normal (or lognormal when the data is log-transformed) distribution.

The alternate hypothesis (H_A) is that the population does not have a normal (or lognormal when the data is log-transformed) distribution.

The equation for the W statistic is:

$$W = \left[\frac{b}{S_R \sqrt{n-1}} \right]^2$$

where

$$b = \sum_{i=1}^k a_i (x_{[n+1]} - x) = \sum_{i=1}^k b_i$$

and the coefficients $a_1, a_2, a_3, \dots, a_k$ are found in Table 1.

A "W" statistic (W_{calc}) is computed for a data set (or a log transformed data set) and compared to a test statistic (W_{test}). The test statistic is determined at the 5% significance level from Table 2. If $W_{\text{calc}} > W_{\text{test}}$, then the null hypothesis is not rejected (i.e. the data are assumed to be normally distributed [or lognormally distributed if log transformed data are tested]). If $W_{\text{calc}} < W_{\text{test}}$, then the null hypothesis is rejected and the alternative hypothesis is accepted (i.e., the data are not assumed to be normally

distributed [or not log-normally distributed if log transformed data are tested]).

3.1.2 Representative Concentration for a Normal Distribution (Upper One-sided 95% Confidence Limit for the Arithmetic Mean)

The 100(1- α) Upper Confidence Limit ($UCL_{100(1-\alpha)}$) of the population mean (\bar{x}) is often used as a descriptive statistic for environmental data. When $\alpha = 0.05$, the 95 percent upper confidence limit (one-tailed test) may be calculated as follows:

$$UCL_{0.95} = \bar{x} + t_{0.95, n-1} \frac{S_x}{\sqrt{n}}$$

$$\bar{X} = \frac{1}{n} \sum_{i=1}^n x_i = \text{arithmetic mean}$$

$$S_x = \sqrt{\frac{\sum_{i=1}^n (x_i - \bar{x})^2}{n-1}} = \text{the sample standard deviation}$$

where: $t_{0.95, n-1}$ = Value from *t* - distribution (Table 4)

It should be noted that the 95 percent confidence interval for a second sample of size n drawn from the same population will most likely not be the same as that for the first sample. In theory if an interval estimate is calculated for the means of a very large set of samples of size n , the true population mean will be within 95 percent of this limit.

3.1.3 Representative Concentration for a Lognormal Distribution (Upper One-sided 95% Confidence Limit for the Geometric Mean)

The following formula may be used to calculate the upper 95% confidence interval ($UCL_{95\%}$) for the geometric mean (x_g):

$$UCL_{0.95} = \exp \left[\bar{y} + 0.5 (S_y)^2 + \frac{S_y (H_{0.95,n})}{\sqrt{n}} \right]$$

where: \bar{y} = arithmetic mean of the log-transformed data, $y = \ln(x)$

S_y = standard deviation of y

$H_{0.95,n}$ = factor for sample size n (Table 3)

4 HANDLING OF MULTIPLE ROUND SAMPLES

Multiple round samples are samples collected from the same location on different dates. The individual multiple round sample results were used in the determination of the contaminants of potential concern. The average of multiple round sample results was used to calculate the exposure point concentrations.

Table 2
PERCENTAGE POINTS OF THE W TEST FOR N=3 to 50

n	0.01	0.05
3	0.753	0.767
4	0.687	0.748
5	0.686	0.762
6	0.713	0.788
7	0.730	0.803
8	0.749	0.818
9	0.764	0.829
10	0.781	0.842
11	0.792	0.850
12	0.805	0.859
13	0.814	0.866
14	0.825	0.874
15	0.835	0.881
16	0.844	0.887
17	0.851	0.892
18	0.858	0.897
19	0.863	0.901
20	0.868	0.905
21	0.873	0.908
22	0.878	0.911
23	0.881	0.914
24	0.884	0.916
25	0.888	0.918
26	0.891	0.920
27	0.894	0.923
28	0.896	0.924
29	0.898	0.926
30	0.900	0.927

n	0.01	0.05
31	0.902	0.929
32	0.904	0.930
33	0.906	0.931
34	0.908	0.933
35	0.910	0.934
36	0.912	0.935
37	0.914	0.936
38	0.916	0.938
39	0.917	0.939
40	0.919	0.940
41	0.920	0.941
42	0.922	0.942
43	0.923	0.943
44	0.924	0.944
45	0.926	0.945
46	0.927	0.945
47	0.928	0.946
48	0.929	0.947
49	0.929	0.947
50	0.930	0.947

TA 3
**VALUES OF $H_{0.95}$ FOR COMPUTING A ONE-SIDED
 UPPER 95% CONFIDENCE LIMIT ON A LOGNORMAL MEAN**

Sy/n	3	5	7	10	12	15	21	31	51	101	201	301	401	601
0.10	2.75	2.035	1.886	1.802	1.775	1.749	1.722	1.701	1.684	1.670	1.662	1.659	1.658	1.656
0.20	3.295	2.198	1.992	1.881	1.843	1.809	1.771	1.742	1.718	1.697	1.685	1.680	1.677	1.674
0.30	4.109	2.402	2.125	1.977	1.927	1.882	1.833	1.793	1.761	1.733	1.716	1.709	1.705	1.700
0.40	5.22	2.651	2.282	2.089	2.026	1.968	1.905	1.856	1.813	1.770	1.755	1.746	1.740	1.734
0.50	6.495	2.947	2.465	2.220	2.141	2.068	1.989	1.928	1.876	1.830	1.802	1.790	1.784	1.776
0.60	7.807	3.287	2.673	2.368	2.271	2.181	2.085	2.010	1.946	1.891	1.857	1.843	1.835	1.825
0.70	9.12	3.662	2.904	2.532	2.414	2.306	2.191	2.102	2.025	1.960	1.919	1.902	1.892	1.881
0.80	10.43	4.062	3.155	2.710	2.570	2.443	2.307	2.202	2.112	2.035	1.988	1.968	1.957	1.944
0.90	11.74	4.478	3.420	2.902	2.738	2.589	2.432	2.310	2.206	2.117	2.062	2.040	2.027	2.012
1.00	13.05	4.905	3.698	3.103	2.915	2.744	2.564	2.423	2.306	2.205	2.143	2.117	2.102	2.085
1.25	16.33	6.001	4.426	3.639	3.389	3.163	2.923	2.737	2.580	2.447	2.364	2.330	2.310	2.288
1.50	19.6	7.12	5.184	4.207	3.896	3.612	3.311	3.077	2.881	2.713	2.609	2.566	2.542	2.514
1.75	22.87	8.25	5.960	4.795	4.422	4.081	3.719	3.437	3.200	2.997	2.872	2.820	2.791	2.757
2.00	26.14	9.387	6.747	5.396	4.962	4.564	4.141	3.912	3.533	3.295	3.148	3.088	3.053	3.013
2.50	32.69	11.673	8.339	6.621	6.067	5.557	5.013	4.588	4.228	3.920	3.729	3.650	3.605	3.553
3.00	39.23	13.97	9.945	7.864	7.191	6.570	5.907	5.388	4.947	4.569	4.334	4.238	4.183	4.119
3.50	45.77	16.27	11.560	9.118	8.326	7.596	6.815	6.201	5.681	5.233	4.956	4.842	4.776	4.700
4.00	52.31	18.58	13.180	10.380	9.469	8.630	7.731	7.024	6.424	5.908	5.588	5.456	5.380	5.293
4.50	58.85	20.88	14.800	11.640	10.620	9.669	8.652	7.854	7.174	6.590	6.227	6.077	5.991	5.892
5.00	65.39	23.19	16.430	12.910	11.770	10.710	9.579	8.688	7.929	7.277	6.871	6.704	6.608	6.497
6.00	78.47	27.81	19.680	15.450	14.080	12.810	11.440	10.360	9.449	8.661	8.170	7.968	7.852	7.718
7.00	91.55	32.43	22.940	18.000	16.390	14.900	13.310	12.050	10.980	10.050	9.479	9.242	9.106	8.949
8.00	104.6	37.06	26.200	20.550	18.710	17.010	15.180	13.740	12.510	11.450	10.790	10.520	10.370	10.190
9.00	117.7	41.68	29.460	23.100	21.030	19.110	17.050	15.430	14.050	12.850	12.110	11.810	11.630	11.430
10.00	130.8	46.31	32.730	25.660	23.350	21.220	18.930	17.130	15.590	14.260	13.430	13.100	12.900	12.670

TABLE 4
PERCENTILES OF STUDENT'S t-DISTRIBUTION WITH n DEGREES OF FREEDOM

n\F	0.60	0.75	0.90	0.95	0.975	0.99	0.995	0.9995
1	0.325	1.000	3.078	6.314	12.706	31.821	63.656	636.578
2	0.289	0.816	1.886	2.920	4.303	6.965	9.925	31.600
3	0.277	0.765	1.638	2.353	3.182	4.541	5.841	12.924
4	0.271	0.741	1.533	2.132	2.776	3.747	4.604	8.610
5	0.267	0.727	1.476	2.015	2.571	3.365	4.032	6.869
6	0.265	0.718	1.440	1.943	2.447	3.143	3.707	5.959
7	0.263	0.711	1.415	1.895	2.365	2.998	3.499	5.408
8	0.262	0.706	1.397	1.860	2.306	2.896	3.355	5.041
9	0.261	0.703	1.383	1.833	2.262	2.821	3.250	4.781
10	0.260	0.700	1.372	1.812	2.228	2.764	3.169	4.587
11	0.260	0.697	1.363	1.796	2.201	2.718	3.106	4.437
12	0.259	0.695	1.356	1.782	2.179	2.681	3.055	4.318
13	0.259	0.694	1.350	1.771	2.160	2.650	3.012	4.221
14	0.258	0.692	1.345	1.761	2.145	2.624	2.977	4.140
15	0.258	0.691	1.341	1.753	2.131	2.602	2.947	4.073
16	0.258	0.690	1.337	1.746	2.120	2.583	2.921	4.015
17	0.257	0.689	1.333	1.740	2.110	2.567	2.898	3.965
18	0.257	0.688	1.330	1.734	2.101	2.552	2.878	3.922
19	0.257	0.688	1.328	1.729	2.093	2.539	2.861	3.883
20	0.257	0.687	1.325	1.725	2.086	2.528	2.845	3.850
21	0.257	0.686	1.323	1.721	2.080	2.518	2.831	3.819
22	0.256	0.686	1.321	1.717	2.074	2.508	2.819	3.792
23	0.256	0.685	1.319	1.714	2.069	2.500	2.807	3.768
24	0.256	0.685	1.318	1.711	2.064	2.492	2.797	3.745
25	0.256	0.684	1.316	1.708	2.060	2.485	2.787	3.725
26	0.256	0.684	1.315	1.706	2.056	2.479	2.779	3.707
27	0.256	0.684	1.314	1.703	2.052	2.473	2.771	3.689
28	0.256	0.683	1.313	1.701	2.048	2.467	2.763	3.674
29	0.256	0.683	1.311	1.699	2.045	2.462	2.756	3.660
30	0.256	0.683	1.310	1.697	2.042	2.457	2.750	3.646
40	0.255	0.681	1.303	1.684	2.021	2.423	2.704	3.551
60	0.254	0.679	1.296	1.671	2.000	2.390	2.660	3.460
120	0.254	0.677	1.289	1.658	1.980	2.358	2.617	3.373
1,000,000	0.253	0.674	1.282	1.645	1.960	2.326	2.576	3.290

F = 1 - α

CLIENT Raymark 003		JOB NUMBER	
SUBJECT Check - UCL Calculations		Aroclor-1268, Trespasser, Surface Soil	
BASED ON		DRAWING NUMBER Area B	
BY Gary Glennon	CHECKED BY	APPROVED BY	DATE 3/27/00

Interpolation of $H_{0.95}$ Table

$$n = 20 \quad s = 1.554$$

$$n_1 = 15 \quad s_1 = 1.50 \quad H_1 = 3.612 \quad H_2 = 3.311$$

$$n_2 = 21 \quad s_2 = 1.75 \quad H_3 = 4.081 \quad H_4 = 3.719$$

$$\begin{aligned} H_{\text{interpol}_1} &= (((H_2 - H_1) / (n_2 - n_1)) \times (n - n_1)) + H_1 \\ &= (((3.311 - 3.612) / (21 - 15)) \times (20 - 15)) + 3.612 \\ &= 3.361 \end{aligned}$$

$$\begin{aligned} H_{\text{interpol}_2} &= (((H_4 - H_3) / (n_2 - n_1)) \times (n - n_1)) + H_3 \\ &= (((3.719 - 4.081) / (21 - 15)) \times (20 - 15)) + 4.081 \\ &= 4.003 \end{aligned}$$

$$\begin{aligned} H &= (((H_{\text{interpol}_2} - H_{\text{interpol}_1}) / (s_2 - s_1)) \times (s - s_1)) + H_{\text{interpol}_1} \\ &= (((4.003 - 3.361) / (1.75 - 1.50)) \times (1.554 - 1.50)) + 3.361 \\ &= 3.50 \end{aligned}$$

Interpolation of t

$$df = 19 \quad t = 1.729$$

CLIENT		JOB NUMBER	
SUBJECT			
BASED ON		DRAWING NUMBER	
BY	CHECKED BY	APPROVED BY	DATE

Calculation of UCL-NORMAL

$$\bar{x} = 357.95$$

$$t = 1.729$$

$$s = 552.53$$

$$n = 20$$

$$\begin{aligned} UCL-N &= \bar{x} + t(s/\sqrt{n}) \\ &= 357.95 + 1.729(552.53/\sqrt{20}) \\ &= \boxed{571.57 \text{ ug/kg}} \quad \checkmark \end{aligned}$$

Calculation of UCL-LOGNORMAL

$$\bar{x} = 4.736$$

$$H = 3.50$$

$$s = 1.554$$

$$n = 20$$

$$\begin{aligned} UCL-LOGNORMAL &= e^{(\bar{x} + 0.5s^2 + sH/\sqrt{n-1})} \\ &= e^{(4.736 + 0.5(1.554)^2 + (1.554)(3.50)/\sqrt{20-1})} \\ &= \boxed{1327.8 \text{ ug/kg}} \quad \checkmark \approx 1.3 \text{ mg/kg} \end{aligned}$$

Location	Fraction	Parameter	CasNo	Units	Detects	Count	Average	W	WL	WStat	Distribution	UCL N	UCL T	MaxOfDetects	MaxQual	EPC	EPCStat	EPC CTE	EPC CTEStat	MaxLocation	
bstf	OS	2,4-Dimethylphenol	105679	UG/KG	2	17	420	0.751	0.943	0.892	Lognormal	550	580	170	J	170	Max	170	Max	DB01	
bstf	OS	2-Methylnaphthalene	91576	UG/KG	4	17	460	0.854	0.926	0.892	Lognormal	620	870	850	J	850	Max	480	Mean-N	DR10	
bstf	OS	4-Methylphenol	106445	UG/KG	3	17	470	0.887	0.907	0.892	Lognormal	630	1000	850	J	850	Max	470	Mean-N	DR10	
bstf	OS	Acenaphthene	83329	UG/KG	7	17	370	0.767	0.978	0.892	Lognormal	510	650	380	J	380	Max	370	Mean-N	DB05	
bstf	OS	Acenaphthylene	208968	UG/KG	15	18	870	0.745	0.967	0.897	Lognormal	1300	2700	4100	J	2700	95% UCL-T	2700	95% UCL-T	DB05	
bstf	OS	Anthracene	120127	UG/KG	14	18	730	0.67	0.949	0.897	Lognormal	1100	1800	3700	J	1800	95% UCL-T	1800	95% UCL-T	DB05	
bstf	OS	Benzo(a)anthracene	56553	UG/KG	17	18	1800	0.678	0.962	0.897	Lognormal	2800	6100	10000	J	6100	95% UCL-T	6100	95% UCL-T	DB05	
bstf	OS	Benzo(a)pyrene	50328	UG/KG	16	18	1300	0.763	0.973	0.897	Lognormal	1900	4000	4800	J	4000	95% UCL-T	4000	95% UCL-T	DB05	
bstf	OS	Benzo(b)fluoranthene	205992	UG/KG	17	18	2500	0.79	0.93	0.897	Lognormal	3700	10000	10000	J	10000	Max	2500	Mean-N	DB05	
bstf	OS	Benzo(g,h)perylene	191242	UG/KG	9	17	810	0.89	0.899	0.892	Lognormal	800	1500	1500	J	1500	Max	810	Mean-N	B2-SD03	
bstf	OS	Benzo(k)fluoranthene	207089	UG/KG	15	18	1500	0.819	0.969	0.897	Lognormal	2100	3600	5700	J	3600	95% UCL-T	3600	95% UCL-T	DB05	
bstf	OS	bis(2-Ethylhexyl)phthalate	117817	UG/KG	15	18	1800	0.360	0.553	0.897	Lognormal	3300	6100	18000	J	6100	95% UCL-T	6100	95% UCL-T	SD26	
bstf	OS	Butybenzylphthalate	85687	UG/KG	2	17	470	0.842	0.956	0.892	Lognormal	620	750	180	J	180	Max	180	Max	SD08	
bstf	OS	Carbazole	86748	UG/KG	13	18	350	0.649	0.823	0.897	Lognormal	510	650	1300	J	650	95% UCL-T	650	95% UCL-T	DB05	
bstf	OS	Chrysene	218019	UG/KG	17	18	2100	0.74	0.952	0.897	Lognormal	3100	6400	10000	J	6400	95% UCL-T	6400	95% UCL-T	DB05	
bstf	OS	Dibenz(a,h)anthracene	53703	UG/KG	14	18	400	0.878	0.939	0.897	Lognormal	530	730	1100	J	730	95% UCL-T	730	95% UCL-T	DB05	
bstf	OS	Dibenzofuran	132649	UG/KG	6	17	420	0.849	0.97	0.892	Lognormal	580	860	640	J	640	Max	420	Mean-N	DB05	
bstf	OS	Diethylphthalate	84662	UG/KG	1	17	500	0.816	0.907	0.892	Lognormal	640	890	850	J	850	95% UCL-T	850	95% UCL-T	DB10	
bstf	OS	Di-n-Butylphthalate	84742	UG/KG	4	17	460	0.833	0.946	0.892	Lognormal	600	650	1300	J	650	95% UCL-T	650	95% UCL-T	SD26	
bstf	OS	Di-n-octylphthalate	117840	UG/KG	4	17	440	0.783	0.937	0.892	Lognormal	590	640	230	J	230	Max	230	Max	SD08	
bstf	OS	Fluoranthene	206440	UG/KG	16	18	4300	0.711	0.969	0.897	Lognormal	6800	20000	23000	J	20000	95% UCL-T	20000	95% UCL-T	DB05	
bstf	OS	Fluorene	86737	UG/KG	10	18	400	0.566	0.868	0.897	Undefined	600	580	1800	J	580	95% UCL-T	580	95% UCL-T	DB05	
bstf	OS	Indeno(1,2,3-cd)pyrene	193395	UG/KG	17	18	850	0.885	0.926	0.897	Lognormal	870	1800	1700	J	1700	Max	650	Mean-N	DB05	
bstf	OS	Naphthalene	51203	UG/KG	4	17	430	0.83	0.914	0.892	Lognormal	580	850	230	J	230	Max	230	Max	DB05	
bstf	OS	Perfluorobiphenyl	87865	UG/KG	1	17	1200	0.848	0.939	0.892	Lognormal	1600	2000	150	J	150	Max	150	Max	DB05	
bstf	OS	Phenanthrene	85018	UG/KG	17	18	2100	0.524	0.934	0.897	Lognormal	3500	8200	15000	J	8200	95% UCL-T	8200	95% UCL-T	DB05	
bstf	OS	Pyrene	129000	UG/KG	18	18	2900	0.741	0.959	0.897	Lognormal	4300	12000	14000	J	12000	95% UCL-T	12000	95% UCL-T	DB05	
bstf	OS	Total PAH	10TPAH	UG/KG	18	18	21000	0.742	0.927	0.897	Lognormal	32000	150000	108700	J	110000	Max	21000	Mean-N	DB05	
bstf	OV	2-Butanone	78933	UG/KG	3	16	95	0.302	0.604	0.897	Undefined	240	110	1300	J	110	95% UCL-T	110	95% UCL-T	DB04	
bstf	OV	Acetone	87641	UG/KG	5	16	120	0.322	0.794	0.897	Undefined	280	210	1500	J	210	95% UCL-T	210	95% UCL-T	DB04	
bstf	OV	Carbon Disulfide	75150	UG/KG	3	15	12	0.808	0.937	0.881	Lognormal	15	15	31	J	31	15	95% UCL-T	15	95% UCL-T	DB10
bstf	OV	Styrene	100425	UG/KG	1	16	10	0.896	0.926	0.887	Lognormal	11	11	10	J	10	Max	10	Mean-N	DB04	
bstf	OV	Toluene	108883	UG/KG	1	16	11	0.789	0.906	0.887	Lognormal	13	13	28	J	28	13	95% UCL-T	13	95% UCL-T	DB04
bstf	PESTP	4,4'-DDD	72548	UG/KG	8	20	8.2	0.722	0.944	0.905	Lognormal	12	20	38	J	20	95% UCL-T	20	95% UCL-T	DB04	
bstf	PESTP	4,4'-DDE	72559	UG/KG	15	20	5.1	0.903	0.836	0.905	Undefined	8.6	23	12	J	12	Max	5.1	Mean-N	B2-SD03	
bstf	PESTP	4,4'-DDT	50283	UG/KG	10	19	6	0.809	0.955	0.901	Lognormal	8.6	17	24	J	17	95% UCL-T	17	95% UCL-T	B2-SD03	
bstf	PESTP	Aldrin	309002	UG/KG	12	20	1.5	0.855	0.853	0.905	Undefined	1.9	4.5	3	J	3	Max	1.5	Mean-N	DB05	
bstf	PESTP	alpha-BHC	319646	UG/KG	11	20	0.87	0.747	0.983	0.905	Lognormal	1.3	1.7	1.3	J	1.3	Max	0.87	Mean-N	B2-SD03	
bstf	PESTP	alpha-Chlordane	5103719	UG/KG	15	20	3.7	0.701	0.969	0.905	Lognormal	5.6	17	17	J	17	95% UCL-T	17	95% UCL-T	DB03	
bstf	PESTP	Aroclor, Total	AROCLORTOT	UG/KG	18	20	900	0.828	0.898	0.905	Undefined	1500	4300	4900	J	4300	95% UCL-T	4300	95% UCL-T	DB05	
bstf	PESTP	Aroclor, Total (Conservative)	AROCLORTOTC	UG/KG	18	20	1100	0.649	0.926	0.905	Lognormal	1800	4000	5625	J	4000	95% UCL-T	4000	95% UCL-T	DB05	
bstf	PESTP	Aroclor-1254	11097691	UG/KG	2	20	270	0.25	0.579	0.905	Undefined	700	190	4900	J	190	95% UCL-T	190	95% UCL-T	DB05	
bstf	PESTP	Aroclor-1262	37324235	UG/KG	7	20	320	0.489	0.785	0.905	Undefined	570	920	1900	J	920	95% UCL-T	920	95% UCL-T	DB01	
bstf	PESTP	Aroclor-1268	11100144	UG/KG	14	20	360	0.642	0.907	0.905	Lognormal	570	1300	1800	J	1300	95% UCL-T	1300	95% UCL-T	DB01	
bstf	PESTP	beta-BHC	319657	UG/KG	2	20	1.6	0.688	0.837	0.905	Undefined	2	2.1	0.85	J	0.85	Max	0.85	Max	SD19-04	
bstf	PESTP	delta-BHC	319668	UG/KG	1	20	1.4	0.621	0.75	0.905	Undefined	1.8	1.8	0.74	J	0.74	Max	0.74	Max	DB05	
bstf	PESTP	Dieldrin	60571	UG/KG	10	19	3.7	0.831	0.938	0.901	Lognormal	4.9	6.1	8.7	J	6.1	95% UCL-T	6.1	95% UCL-T	DB07	
bstf	PESTP	Endosulfan II	33213859	UG/KG	3	19	3.4	0.674	0.781	0.901	Undefined	4.5	4.7	8.9	J	4.7	95% UCL-T	4.7	95% UCL-T	SD08	
bstf	PESTP	Endosulfan Sulfate	1031078	UG/KG	4	20	3.3	0.799	0.921	0.905	Lognormal	4.2	4.5	4.4	J	4.4	Max	3.3	Mean-N	B2-SD11	
bstf	PESTP	Endrin	72208	UG/KG	4	20	2.8	0.722	0.922	0.905	Lognormal	3.7	4.4	1.4	J	1.4	Max	1.4	Max	DB08	
bstf	PESTP	Endrin Aldehyde	7421934	UG/KG	8	19	5.9	0.571	0.971	0.901	Lognormal	9.2	12	11	J	11	Max	5.9	Mean-N	DB04	
bstf	PESTP	Endrin Ketone	53494705	UG/KG	3	20	3.2	0.758	0.856	0.905	Undefined	4.2	5	6.9	J	5	95% UCL-T	5	95% UCL-T	B2-SD08	
bstf	PESTP	gamma-BHC	58899	UG/KG	3	20	1.7	0.766	0.892	0.905	Undefined	2.2	2.4	3.2	J	2.4	95% UCL-T	2.4	95% UCL-T	SD08	
bstf	PESTP	gamma-Chlordane	5103742	UG/KG	14	18	4.1	0.687	0.931	0.897	Lognormal	6.3	9.3	17	J	9.3	95% UCL-T	9.3	95% UCL-T	DB03	
bstf	PESTP	Heptachlor	76448	UG/KG	2	20	1.5	0.720	0.896	0.905	Undefined	2	2.2	0.33	J	0.33	Max	0.33	Max	SD08	
bstf	PESTP	Heptachlor Epoxide	1024573	UG/KG	11	20	1.2	0.79	0.961	0.905	Lognormal	1.6	3.1	1.4	J	1.4	Max	1.2	Mean-N	SD26	
bstf	PESTP	Methoxychlor	72435	UG/KG	1	19	1.5	0.695	0.82	0.901	Undefined	21	24	1.5	J	1.5	Max	1.5	Max	B2-SD11	
bstf	TOC	Total Organic Carbon	7440440	MG/KG	2	2	80000	-1	-1	-1	< 11 Samples	-1	-1	138000	J	140000	Max	80000	Mean-N	B2-SD11	
bwf	M	Aluminum	7428905	UG/L	2	15	57.3	0.924	0.951	0.881	Lognormal	62.5	62.9	75.5	J	75.5	82.9	95% UCL-T	82.9	95% UCL-T	SD07
bwf	M	Antimony	7440360	UG/L	2	15	10.7	0.758	0.851	0.881	Undefined	13.4	14.6	27.4	J	14.6	95% UCL-T	14.6	95% UCL-T		

TABLE 4
PERCENTILES OF STUDENT'S t-DISTRIBUTION WITH n DEGREES OF FREEDOM

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n\F	0.60	0.75	0.90	0.95	0.975	0.99	0.995	0.9995
1	0.325	1.000	3.078	6.314	12.706	31.821	63.656	636.578
2	0.289	0.816	1.886	2.920	4.303	6.965	9.925	31.600
3	0.277	0.765	1.638	2.353	3.182	4.541	5.841	12.924
4	0.271	0.741	1.533	2.132	2.776	3.747	4.604	8.610
5	0.267	0.727	1.476	2.015	2.571	3.365	4.032	6.869
6	0.265	0.718	1.440	1.943	2.447	3.143	3.707	5.959
7	0.263	0.711	1.415	1.895	2.365	2.998	3.499	5.408
8	0.262	0.706	1.397	1.860	2.306	2.896	3.355	5.041
9	0.261	0.703	1.383	1.833	2.262	2.821	3.250	4.781
10	0.260	0.700	1.372	1.812	2.228	2.764	3.169	4.587
11	0.260	0.697	1.363	1.796	2.201	2.718	3.106	4.437
12	0.259	0.695	1.356	1.782	2.179	2.681	3.055	4.318
13	0.259	0.694	1.350	1.771	2.160	2.650	3.012	4.221
14	0.258	0.692	1.345	1.761	2.145	2.624	2.977	4.140
15	0.258	0.691	1.341	1.753	2.131	2.602	2.947	4.073
16	0.258	0.690	1.337	1.746	2.120	2.583	2.921	4.015
17	0.257	0.689	1.333	1.740	2.110	2.567	2.898	3.965
18	0.257	0.688	1.330	1.734	2.101	2.552	2.878	3.922
19	0.257	0.688	1.328	1.729	2.093	2.539	2.861	3.883
20	0.257	0.687	1.325	1.725	2.086	2.528	2.845	3.850
21	0.257	0.686	1.323	1.721	2.080	2.518	2.831	3.819
22	0.256	0.686	1.321	1.717	2.074	2.508	2.819	3.792
23	0.256	0.685	1.319	1.714	2.069	2.500	2.807	3.768
24	0.256	0.685	1.318	1.711	2.064	2.492	2.797	3.745
25	0.256	0.684	1.316	1.708	2.060	2.485	2.787	3.725
26	0.256	0.684	1.315	1.706	2.056	2.479	2.779	3.707
27	0.256	0.684	1.314	1.703	2.052	2.473	2.771	3.689
28	0.256	0.683	1.313	1.701	2.048	2.467	2.763	3.674
29	0.256	0.683	1.311	1.699	2.045	2.462	2.756	3.660
30	0.256	0.683	1.310	1.697	2.042	2.457	2.750	3.646
40	0.255	0.681	1.303	1.684	2.021	2.423	2.704	3.551
60	0.254	0.679	1.296	1.671	2.000	2.390	2.660	3.460
120	0.254	0.677	1.289	1.658	1.980	2.358	2.617	3.373
1,000,000	0.253	0.674	1.282	1.645	1.960	2.326	2.576	3.290

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$F = 1 - \alpha$

TABLE 3
VALUES OF $H_{0.95}$ FOR COMPUTING A ONE-SIDED
UPPER 95% CONFIDENCE LIMIT ON A LOGNORMAL MEAN

Sy/n	3	5	7	10	12	15	21	31	51	101	201	301	401	601
0.10	2.75	2.035	1.886	1.802	1.775	1.749	1.722	1.701	1.684	1.670	1.662	1.659	1.658	1.656
0.20	3.295	2.198	1.992	1.881	1.843	1.809	1.771	1.742	1.718	1.697	1.685	1.680	1.677	1.674
0.30	4.109	2.402	2.125	1.977	1.927	1.882	1.833	1.793	1.761	1.733	1.716	1.709	1.705	1.700
0.40	5.22	2.651	2.282	2.089	2.026	1.968	1.905	1.856	1.813	1.770	1.755	1.746	1.740	1.734
0.50	6.495	2.947	2.465	2.220	2.141	2.068	1.989	1.928	1.876	1.830	1.802	1.790	1.784	1.776
0.60	7.807	3.287	2.673	2.368	2.271	2.181	2.085	2.010	1.946	1.891	1.857	1.843	1.835	1.825
0.70	9.12	3.662	2.904	2.532	2.414	2.306	2.191	2.102	2.025	1.960	1.919	1.902	1.892	1.881
0.80	10.43	4.062	3.155	2.710	2.570	2.443	2.307	2.202	2.112	2.035	1.988	1.968	1.957	1.944
0.90	11.74	4.478	3.420	2.902	2.738	2.589	2.432	2.310	2.206	2.117	2.062	2.040	2.027	2.012
1.00	13.05	4.905	3.698	3.103	2.915	2.744	2.564	2.423	2.306	2.205	2.143	2.117	2.102	2.085
1.25	16.33	6.001	4.426	3.639	3.389	3.163	2.923	2.737	2.580	2.447	2.364	2.330	2.310	2.288
1.50	19.6	7.12	5.184	4.207	3.896	H_1 3.612	H_2 3.311	3.077	2.881	2.713	2.609	2.566	2.542	2.514
1.75	22.87	8.25	5.960	4.795	4.422	H_3 4.081	H_4 3.719	3.437	3.200	2.997	2.872	2.820	2.791	2.757
2.00	26.14	9.387	6.747	5.396	4.962	4.564	4.141	3.912	3.533	3.295	3.148	3.088	3.053	3.013
2.50	32.69	11.673	8.339	6.621	6.067	5.557	5.013	4.588	4.228	3.920	3.729	3.650	3.605	3.553
3.00	39.23	13.97	9.945	7.864	7.191	6.570	5.907	5.388	4.947	4.569	4.334	4.238	4.183	4.119
3.50	45.77	16.27	11.560	9.118	8.326	7.596	6.815	6.201	5.681	5.233	4.956	4.842	4.776	4.700
4.00	52.31	18.58	13.180	10.380	9.469	8.630	7.731	7.024	6.424	5.908	5.588	5.456	5.380	5.293
4.50	58.85	20.88	14.800	11.640	10.620	9.669	8.652	7.854	7.174	6.590	6.227	6.077	5.991	5.892
5.00	65.39	23.19	16.430	12.910	11.770	10.710	9.579	8.688	7.929	7.277	6.871	6.704	6.608	6.497
6.00	78.47	27.81	19.680	15.450	14.080	12.810	11.440	10.360	9.449	8.661	8.170	7.968	7.852	7.718
7.00	91.55	32.43	22.940	18.000	16.390	14.900	13.310	12.050	10.980	10.050	9.479	9.242	9.106	8.949
8.00	104.6	37.06	26.200	20.550	18.710	17.010	15.180	13.740	12.510	11.450	10.790	10.520	10.370	10.190
9.00	117.7	41.68	29.460	23.100	21.030	19.110	17.050	15.430	14.050	12.850	12.110	11.810	11.630	11.430
10.00	130.8	46.31	32.730	25.660	23.350	21.220	18.930	17.130	15.590	14.260	13.430	13.100	12.900	12.670

S_1 →
 S_2 →

receptor	NSAMPLE	PARAMETER	LAB	RESULT	QUAL	UNITS	USE
bstr	DBL007	Aroclor-1268		67	UJ	UG/KG	33.5
r	DBL027	Aroclor-1268		76	J	UG/KG	76
bstr	OU3-B2-SB08-0002	Aroclor-1268		100		UG/KG	100
bstr	OU3-B2-SD03-0002	Aroclor-1268		160		UG/KG	160
bstr	OU3-B2-SD11-0002	Aroclor-1268		41	U	UG/KG	20.5
bstr	RM-SD-DB01-03	Aroclor-1268		1800		UG/KG	1800
bstr	RM-SD-DB02-03	Aroclor-1268		240		UG/KG	240
bstr	RM-SD-DB03-03	Aroclor-1268		1600		UG/KG	1600
bstr	RM-SD-DB04-03	Aroclor-1268		48	U	UG/KG	24
bstr	RM-SD-DB05-03	Aroclor-1268		160	U	UG/KG	80
bstr	RM-SD-DB06-03	Aroclor-1268		33	J	UG/KG	33
bstr	RM-SD-DB07-03	Aroclor-1268		780		UG/KG	780
bstr	RM-SD-DB08-03	Aroclor-1268		58		UG/KG	58
bstr	RM-SD-DB09-03	Aroclor-1268		37		UG/KG	37
bstr	RM-SD-DB10-03	Aroclor-1268		780		UG/KG	780
bstr	RM-SD-SD08	Aroclor-1268		74		UG/KG	74
bstr	RM-SD-SD19-03	Aroclor-1268		33	U	UG/KG	16.5
bstr	RM-SD-SD19-04	Aroclor-1268		130		UG/KG	130
bstr	RM-SD-SD20-03	Aroclor-1268		33	U	UG/KG	16.5
bstr	RM-SD-SD26-04	Aroclor-1268		1100		UG/KG	1100

Receptor	PARAMETER	n	AvgUse	StDevUse	AvgLogUse	StDevLogUse	UNITS
bstr	Aroclor-1268	20	357.95	552.52877753109	4.7362454997622	1.5544523261134	UG/KG

II TEF Calculation Check

4/10/00

Gary Glenon

RECEPTOR	NSAMPLE	FRACTION	PARAMETER	LAB RESULT	QUAL	UNITS	TEF	DETECT	DATE	CRF	1/2 ND	1/2 ND x TEF			
bscn2	RM-SD-DB03-03	DIOXI	2,3,4,7,8-PeCDF	0.22183	J	UG/KG	0.5	0.22183	0.110915						
bscn2	RM-SD-DB03-03	DIOXI	1,2,3,4,6,7,8-HpCDD	1.12828	J	UG/KG	0.01	1.12828	0.0112828						
bscn2	RM-SD-DB03-03	DIOXI	1,2,3,4,6,7,8-HpCDF	0.59526	J	UG/KG	0.01	0.59526	0.0059526						
bscn2	RM-SD-DB03-03	DIOXI	1,2,3,4,7,8,9-HpCDF	0.02173	UJ	UG/KG	0.01			0.010865	0.00010865				
bscn2	RM-SD-DB03-03	DIOXI	1,2,3,4,7,8-HxCDD	0.03797	UJ	UG/KG	0.1			0.018985	0.0018985				
bscn2	RM-SD-DB03-03	DIOXI	1,2,3,4,7,8-HxCDF	0.24961	J	UG/KG	0.1	0.24961	0.024961						
bscn2	RM-SD-DB03-03	DIOXI	1,2,3,6,7,8-HxCDD	0.0364	UJ	UG/KG	0.1			0.0182	0.00182				
bscn2	RM-SD-DB03-03	DIOXI	1,2,3,6,7,8-HxCDF	0.06338	UJ	UG/KG	0.1			0.03169	0.003169				
bscn2	RM-SD-DB03-03	DIOXI	1,2,3,7,8,9-HxCDD	0.04919	UJ	UG/KG	0.1			0.024595	0.0024595				
bscn2	RM-SD-DB03-03	DIOXI	1,2,3,7,8,9-HxCDF	0.14404	J	UG/KG	0.1	0.14404	0.014404						
bscn2	RM-SD-DB03-03	DIOXI	1,2,3,7,8-PeCDD	0.00813	UJ	UG/KG	1			0.004065	0.004065				
bscn2	RM-SD-DB03-03	DIOXI	2,3,4,6,7,8-HxCDF	0.00443	UJ	UG/KG	0.1			0.002215	0.0002215				
bscn2	RM-SD-DB03-03	DIOXI	Total TCDF	0.9481	J	UG/KG		0.9481							
bscn2	RM-SD-DB03-03	DIOXI	2,3,7,8-TCDD	0.00272	UJ	UG/KG	1			0.00136	0.00136				
bscn2	RM-SD-DB03-03	DIOXI	2,3,7,8-TCDF	0.22156	J	UG/KG	0.1	0.22156	0.022156						
bscn2	RM-SD-DB03-03	DIOXI	OCDD	4.96505	J	UG/KG	0.0001	4.96505	0.000496505						
bscn2	RM-SD-DB03-03	DIOXI	OCDF	0.15677	J	UG/KG	0.0001	0.15677	0.00015677						
bscn2	RM-SD-DB03-03	DIOXI	Total HpCDD	3.88902	J	UG/KG		3.88902							
bscn2	RM-SD-DB03-03	DIOXI	Total HpCDF	1.73706	J	UG/KG		1.73706							
bscn2	RM-SD-DB03-03	DIOXI	Total HxCDD	0.00589	J	UG/KG		0.00589							
bscn2	RM-SD-DB03-03	DIOXI	Total HxCDF	1.98002	J	UG/KG		1.98002							
bscn2	RM-SD-DB03-03	DIOXI	Total PeCDD	0.00062	J	UG/KG		0.00062							
bscn2	RM-SD-DB03-03	DIOXI	Total PeCDF	1.46102	J	UG/KG		1.46102							
bscn2	RM-SD-DB03-03	DIOXI	Total TCDD	0.00272	UJ	UG/KG				0.00136					
bscn2	RM-SD-DB03-03	DIOXI	1,2,3,7,8-PeCDF	0.1109	UJ	UG/KG	0.05			0.05545	0.0027725				
Toxicity Equivalency =											0.19018	+	0.017875	=	0.208058

Appendix F.7

Toxicity Profiles

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REFERENCES

F.7.1 Antimony

F.7.1.1 Pharmacokinetics

Ingested antimony is absorbed slowly and incompletely from the gastrointestinal (GI) tract (Iffland 1988). Within a few days of acute exposure, highest tissue concentrations are found in the liver, kidney, and thyroid. Organs of storage include skin, bone, and teeth. Highest concentrations in deceased smelter workers (inhalation exposure) occurred in the lungs and skeleton. Excretion is largely via the urine or feces, although some is incorporated into the hair.

F.7.1.2 Noncancer Toxicity

Acute intoxication from ingestion of large doses of antimony induces GI disturbances, dehydration, and cardiac effects in humans (Iffland 1988). Chronic effects from occupational exposure include irritation of the respiratory tract, pneumoconiosis, pustular eruptions of the skin called "antimony spots," allergic contact dermatitis, and cardiac effects, including abnormalities of the electrocardiograph (ECG) and myocardial changes. Cardiac effects were also observed in rats and rabbits exposed by inhalation for six weeks and in animals (dogs, and possibly other species) treated by intravenous injection (Elinder and Friberg 1986).

Chronic oral exposure studies in laboratory animals include two briefly reported lifetime drinking water studies in rats and mice (Kanisawa and Schroeder 1969; Schroeder et al. 1970). The only dose tested, 5 ppm potassium antimony tartrate, resulted in reduced longevity in both species and in reduced mean heart weight in the rats. The EPA (2000) verifies a RfD of 0.0004 mg/kg/day for chronic oral exposure to antimony from the LOAEL of 5 ppm potassium antimony tartrate (0.35 mg antimony/kg body weight-day) in the lifetime study in rats (Schroeder et al. 1970). An uncertainty factor of 1000 was applied; factors of 10 each for inter- and intraspecies variation and to estimate an NOAEL from an LOAEL. The heart is considered a likely target organ for chronic oral exposure of humans.

F.7.1.3 Carcinogenicity

Data were not located regarding the carcinogenicity of antimony to humans. Antimony fed to rats did not produce an excess of tumors (Goyer 1991), but a high frequency of lung tumors was observed in rats exposed by inhalation to antimony trioxide for one year (Elinder and Friberg 1986). Antimony is classified in EPA cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans) (EPA 1997).

F.7.2 Arsenic

F.7.2.1 Pharmacokinetics

Several studies confirm that soluble inorganic arsenic compounds and organic arsenic compounds are almost completely (>90 percent) absorbed from the GI tract in both animals and humans (Ishinishi et al. 1986). The absorption efficiency of insoluble inorganic arsenic compounds depends on particle size and stomach pH. Initial distribution of absorbed arsenic is to the liver, kidneys, and lungs, followed by redistribution to hair, nails, teeth, bone, and skin, which are considered tissues of accumulation. Arsenic has a long half-life in the blood of rats, compared with other animals and humans, because of firm binding to the hemoglobin in erythrocytes.

Metabolism of inorganic arsenic includes reversible oxidation-reduction so that both arsenite (valence of 3) and arsenate (valence of 5) are present in the urine of animals treated with arsenic of either valence (Ishinishi et al. 1986). Arsenite is subsequently oxidized and methylated by a saturable mechanism to form mono- or dimethylarsenate; the latter is the predominant metabolite in the urine of animals or humans. Organic arsenic compounds (arsenilic acid, cacodylic acid) are not readily converted to inorganic arsenic. Excretion of organic or inorganic arsenic is largely via the urine, but considerable species variation exists. Continuously exposed humans appear to excrete 60 to 70 percent of their daily intake of arsenate or arsenite via the urine.

F.7.2.2 Noncancer Toxicity

A lethal dose of arsenic trioxide in humans is 70 to 180 mg. (approximately 50 to 140 mg arsenic; Ishinishi et al. 1986). Acute oral exposure of humans to high doses of arsenic produces liver swelling, skin lesions, disturbed heart function, and neurological effects. The only noncancer effects in humans clearly attributable to chronic oral exposure to arsenic are dermal hyperpigmentation and keratosis, as revealed by studies of several hundred Chinese exposed to naturally occurring arsenic in well water (Tseng 1977; Tseng et al. 1968; EPA 2000). Similar effects were observed in persons exposed to high levels of arsenic in water in Utah and the northern part of Mexico (Cebrian et al. 1983; Southwick et al. 1983). Occupational (predominantly inhalation) exposure is also associated with neurological deficits, anemia, and cardiovascular effects (Ishinishi et al. 1986), but concomitant exposure to other chemicals cannot be ruled out. The EPA (2000) derived an RfD of 0.3 ug/kg/day for chronic oral exposure, based on an NOAEL of 0.8 ug/kg/day for skin lesions from Chinese data. The principal target organ for arsenic appears to be the skin. The nervous system and cardiovascular systems appear to be less significant target organs. Inorganic arsenic may be an essential nutrient, exerting beneficial effects on growth, health, and feed conversion efficiency (Underwood 1977).

F.7.2.3 Carcinogenicity

Inorganic arsenic is clearly a carcinogen in humans. Inhalation exposure is associated with increased risk of lung cancer in persons employed as smelter workers, in arsenical pesticide applicators, and in a population residing near a pesticide manufacturing plant (EPA 2000). Oral exposure to high levels in well water is associated with increased risk of skin cancer (Tseng 1977; EPA 2000). Extensive animal testing with various forms of arsenic given by many routes of exposure to several species, however, has not demonstrated the carcinogenicity of arsenic (International Agency for Research on Cancer [IARC] 1980). The EPA (2000) classifies inorganic arsenic in cancer weight-of-evidence Group A (human carcinogen), and recommends an oral unit risk of 0.00005 ug/L in drinking water, based on the incidence of skin cancer in the Tseng (1977) study. The EPA presents a chronic oral slope factor of 1.5 per mg/kg/day based on the same information. The EPA (2000) notes that the uncertainties associated with the oral unit risk are considerably less than those for most

carcinogens, so that the unit risk might be reduced in order of magnitude. An inhalation unit risk of 0.0043 per $\mu\text{g}/\text{m}^3$ was derived for inorganic arsenic from the incidence of lung cancer in occupationally exposed men (EPA 2000). An inhalation cancer slope factor, equivalent to 15.1 per $\text{mg}/\text{kg}/\text{day}$, was derived from the same data assuming an inhalation rate of $20 \text{ m}^3/\text{day}$ and a body weight of 70 kg for humans.

F.7.3 Barium

F.7.3.1 Noncancer Toxicity

Barium is a naturally occurring alkaline earth metal that comprises approximately 0.04 percent of the earth's crust (Reeves 1986). Acute oral toxicity was manifested by GI upset, altered cardiac performance, and transient hypertension, convulsions, and muscular paralysis. Repeated oral exposures were associated with hypertension. Occupational exposure to insoluble barium sulfate induced benign pneumoconiosis (ACGIH 1991). The EPA (2000) presents a verified chronic oral RfD of $0.07 \text{ mg}/\text{kg}/\text{day}$, based on an NOAEL of $0.21 \text{ mg}/\text{kg}/\text{day}$ in a ten-week study in humans exposed to barium in drinking water and an uncertainty factor of 3. The EPA (1997) presented the same value as a provisional RfD for subchronic oral exposure. A provisional chronic inhalation RfC of $0.0005 \text{ mg}/\text{m}^3$ and a provisional subchronic inhalation RfC of $0.005 \text{ mg}/\text{m}^3$ were based on an NOEL for fetotoxicity in a four-month intermittent-exposure inhalation study in rats (EPA 1997). Uncertainty factors of 1000 and 100 were used for the chronic and subchronic RfC values, respectively. The chronic and subchronic inhalation RfC values are equivalent to 0.0001 and $0.001 \text{ mg}/\text{kg}/\text{day}$, assuming a human inhalation rate of $20 \text{ m}^3/\text{day}$ and body weight of 70 kg. Barium is principally a muscle toxin. Its targets are the GI system, skeletal muscle, the cardiovascular system, and the fetus.

F.7.3.2 Carcinogenicity

The EPA (2000) classifies barium as a cancer weight-of-evidence Group D substance (not classifiable as to carcinogenicity in humans). Cancer risk is not estimated for Group D substances.

F.7.4 Cadmium

F.7.4.1 Pharmacokinetics

Estimates of cadmium uptake by the respiratory tract range from 10 to 50 percent; uptake is greatest for fumes and small particles and least for large dust particles (Friberg et al., 1986; Goyer, 1991). GI absorption of ingested cadmium is ordinarily 5 to 8 percent, but may reach 20 percent in cases of serious dietary iron deficiency. Highest tissue levels are normally found in the kidneys followed by the liver, although levels in the liver may exceed those in the kidneys of persons suffering from cadmium-induced renal dysfunction. The half-life of cadmium in the kidneys and liver may be as long as 10-30 years. Fecal and urinary excretion of cadmium are approximately equivalent to normal humans exposed to small amounts. Urinary excretion increases markedly in humans with cadmium-induced renal disease.

F.7.4.2 Noncancer Toxicity

Acute inhalation exposure to fumes or particles of cadmium induces respiratory symptoms, general weakness, and, in severe cases, respiratory insufficiency, shock, and death (Friberg et al., 1986). Acute oral exposure induces GI disturbances. Chronic inhalation exposure induces pulmonary emphysema, and chronic exposure by either route consistently produces renal tubular disease in humans and laboratory animals. Proteinuria is a reliable early indicator of cadmium-induced kidney disease. The combination of pulmonary emphysema and renal tubular disease, if severe, may result in early mortality. Painful osteomalacia and osteoporosis may arise from altered metabolism of bone minerals secondary to renal damage. The combination of renal and skeletal damage is called itai-itai disease in Japan. Cadmium exposure has been associated with liver damage, but the liver appears to be less sensitive than the kidney. The kidney is the primary target organ of cadmium toxicity. The EPA (2000) derived chronic oral RfD values of 0.5 ug/kg/day for cadmium ingested in water and 1 ug/kg/day for cadmium ingested in food, based on a toxicokinetic model that predicted NOAELs from renal cortical concentration of cadmium. The different RfD values reflect assumed differences in GI absorption of cadmium from water (5 percent) and food (2.5 percent).

F.7.4.3 Carcinogenicity

Carcinogenicity data in humans consist of several occupational studies that associate cadmium exposure with lung cancer, but concomitant exposure to other carcinogenic chemicals and smoking were not adequately controlled. Other occupational studies reported significantly increased risk of prostatic cancer, but this effect was not observed in the largest occupational study of workers exposed to high levels (Thun et al., 1985). The animal data consist of an inhalation study in rats that showed a significant increase in lung tumors, and several parenteral injection studies that produced injection site tumors. No evidence of carcinogenicity, however, was observed in seven oral studies in rats and mice. The EPA (2000) classifies cadmium a cancer weight-of-evidence Group B1 substance for inhalation exposure on the basis of limited evidence of carcinogenicity in humans and sufficient evidence in animals. The data were insufficient to classify cadmium as carcinogenic to humans exposed by the oral route. An inhalation unit risk of 0.0018 ug/m^3 , equivalent to 6.3 per mg/kg/day, was derived from the occupational exposure study by Thun et al. (1985) assuming an inhalation rate of $20 \text{ m}^3/\text{day}$ and a body weight of 70 kg for humans.

F.7.5 Chromium

F.7.5.1 Noncancer Toxicity

In nature, chromium (III) predominates over chromium (VI) (Langård and Norseth, 1986). Little chromium (VI) exists in biological materials, except shortly after exposure, because reduction to chromium (III) occurs rapidly. Chromium (III) is considered a nutritionally essential trace element and is considerably less toxic than chromium (VI). No effects were observed in rats consuming 5% chromium (III)/kg/day in the diet for over two years (EPA, 1997). The NOEL of 5% Cr₂O₃ was the basis for a verified chronic oral RfD of 1.5 mg/kg/day (EPA, 1997). The same NOEL and an uncertainty factor of 1000 were the basis for a provisional subchronic oral RfD of 1 mg/kg/day (EPA, 1997).

Acute oral exposure of humans to high doses of chromium (VI) induced neurological effects, GI hemorrhage and fluid loss, and kidney and liver effects. Parenteral dosing of animals with chromium (VI) is selectively toxic to the kidney tubules. An NOAEL of 2.4 mg chromium (VI)

/kg/day in a one-year drinking water study in rats and an uncertainty factor of 500 was the basis of a verified RfD of 0.003 mg/kg/day for chronic oral exposure (EPA, 2000). The same NOAEL and an uncertainty factor of 100 were the basis of a provisional subchronic oral RfD of 0.02 mg/kg/day (EPA, 1997).

Occupational (inhalation and dermal) exposure to chromium (III) compounds induced dermatitis (ACGIH, 1991). Similar exposure to chromium (VI) induced ulcerative and allergic contact dermatitis, irritation of the upper respiratory tract including ulceration of the mucosa and perforation of the nasal septum, and possibly kidney effects. An inhalation RfC values was not located for chromium (III), however, EPA (2000) presents an inhalation RfD of 0.03 ug/kg/day for chromium (VI).

A target organ was not identified for chromium (III). The kidney appears to be the principal target organ for repeated oral dosing with chromium (VI). Additional target organs for dermal and inhalation exposure include the skin and respiratory tract.

F.7.5.2 Carcinogenicity

Data were not located regarding the carcinogenicity of chromium (III). The EPA (2000) classifies chromium (VI) in cancer weight-of-evidence Group A (human carcinogen), based on the consistent observation of increased risk of lung cancer in occupational studies of workers in chromate production or the chrome pigment industry. Parenteral dosing of animals with chromium (VI) compounds consistently induced injection-site tumors. There is no evidence that oral exposure to chromium (VI) induces cancer. An inhalation unit risk of 0.012 per ug/m³, equivalent to 41 per mg/kg/day, assuming humans inhale 20 m³/day and weigh 70 kg, was based on increased risk of lung cancer deaths in chromate production workers (EPA, 2000).

F.7.6 Copper

F.7.6.1 Noncancer Toxicity

Copper is a nutritionally essential element that functions as a cofactor in several enzyme systems (Aaseth and Norseth 1986). Acute exposure to large oral doses of copper salts was

associated with GI disturbances, hemolysis, and liver and kidney lesions. Chronic oral toxicity in humans has not been reported. Chronic oral exposure of animals was associated with an iron-deficiency type of anemia, hemolysis, and lesions in the liver and kidneys. Occupational exposure may induce metal fume fever, and, in cases of chronic exposure to high levels, hemolysis and anemia (ACGIH 1991). Neither oral nor inhalation RfD or RfC values were located for copper. The target organs for copper are the erythrocyte, liver, and kidney, and, for inhalation exposure, the lung. An oral RfD of 0.04 mg/kg/day was presented for copper (EPA, 1997). A RfC value was not located for copper.

F.7.6.2 Carcinogenicity

Copper is classified in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans) (EPA 1997). Quantitative risk estimates are not derived for Group D chemicals.

F.7.7 Lead

F.7.7.1 Pharmacokinetics

Studies in humans indicate that an average of 10 percent of ingested lead is absorbed, but estimates as high as 40 percent were obtained in some individuals (Tsuchiya, 1986). Nutritional factors have a profound effect on GI absorption efficiency. Children absorb ingested lead more efficiently than adults; absorption efficiencies up to 53 percent were recorded for children three months to eight years of age. Similar results were obtained for laboratory animals; absorption efficiencies of 5 to 10 percent were obtained for adults and > 50 percent were obtained for young animals. The deposition rate of inhaled lead averages approximately 30 to 50 percent, depending on particle size, with as much as 60 percent deposition of very small particles (0.03 mm) near highways. All lead deposited in the lungs is eventually absorbed.

Approximately 95 percent of the lead in the blood is located in the erythrocytes (EPA, 2000). Lead in the plasma exchanges with several body compartments, including the internal organs, bone, and several excretory pathways. In humans, lead concentrations in bone increase with

age (Tsuchiya, 1986). About 90 percent of the body burden of lead is located in the skeleton. Neonatal blood concentrations are about 85 percent of maternal concentrations (EPA, 2000). Excretion of absorbed lead is principally through the urine, although GI secretion, biliary excretion, and loss through hair, nails, and sweat are also significant.

F.7.7.2 Noncancer Toxicity

The noncancer toxicity of lead to humans has been well characterized through decades of medical observation and scientific research (EPA, 2000). The principal effects of acute oral exposure are colic with diffuse paroxysmal abdominal pain (probably due to vagal irritation), anemia, and, in severe cases, acute encephalopathy, particularly in children (Tsuchiya, 1986). The primary effects of long-term exposure are neurological and hematological. Limited occupational data indicate that long-term exposure to lead may induce kidney damage. The principal target organs of lead toxicity are the erythrocyte and the nervous system. Some of the effects on the blood, particularly changes in levels of certain blood enzymes, and subtle neurobehavioral changes in children, appear to occur at levels so low as to be considered nonthreshold effects.

The USEPA (1986b and 1990a) determined that it is inappropriate to derive an RfD for oral exposure to lead for several reasons. First, the use of an RfD assumes that a threshold for toxicity exists, below which adverse effects are not expected to occur; however, the most sensitive effects of lead exposure, impaired neurobehavioral development in children and altered blood enzyme levels associated with anemia, may occur at blood lead concentrations so low as to be considered practically nonthreshold in nature. Second, RfD values are specific for the route of exposure for which they are derived. Lead, however, is ubiquitous, so that exposure occurs from virtually all media and by all pathways simultaneously, making it practically impossible to quantify the contribution to blood lead from any one route of exposure. Finally, the dose-response relationships common to many toxicants, and upon which derivation of an RfD is based, do not hold true for lead. This is because the fate of lead within the body depends, in part, on the amount and rate of previous exposures, the age of the recipient, and the rate of exposure. There is, however, a reasonably good correlation between blood lead concentration and effect. Therefore, blood lead concentration is the appropriate parameter on which to base the regulation of lead.

USEPA (1997) presented no inhalation RfC for lead, but referred to the National Ambient Air Quality Standard (NAAQS) for lead, which could be used in lieu of an inhalation RfC. The NAAQSs are based solely on human health considerations and are designed to protect the most sensitive subgroup of the human population. The NAAQS for lead is 1.5 mg/m³, averaged quarterly.

F.7.7.3 Carcinogenicity

USEPA (2000) classifies lead in cancer weight-of-evidence Group B2 (probable human carcinogen), based on inadequate evidence of cancer in humans and sufficient animal evidence. The human data consist of several epidemiologic occupational studies that yielded confusing results. All of the studies lacked quantitative exposure data and failed to control for smoking and concomitant exposure to other possibly carcinogenic metals. Rat and mouse bioassays showed statistically significant increases in renal tumors following dietary and subcutaneous exposure to several soluble lead salts. Various lead compounds were observed to induce chromosomal alterations in vivo and in vitro, sister chromatic exchange in exposed workers, and cell transformation in Syrian hamster embryo cells; to enhance simian adenovirus induction; and to alter molecular processes that regulate gene expression. USEPA (1997) declined to estimate risk for oral exposure to lead because many factors (e.g., age, general health, nutritional status, existing body burden and duration of exposure) influence the bioavailability of ingested lead, introducing a great deal of uncertainty into any estimate of risk.

The USEPA IEUBK lead model is an iterated set of equations that estimate blood lead concentration in children aged 0 to 7 years (USEPA, 1994a). The biokinetic part of the model describes the movement of lead between the plasma and several body compartments and estimates the resultant blood lead concentration. The rate of the movement of lead between the plasma and each compartment is a function of the transition or residence time (i.e., the mean time for lead to leave the plasma and enter a given compartment, or the mean residence time for lead in that compartment). Compartments modeled include the erythrocytes, liver, kidneys, all the other soft tissue of the body, cortical bone, and trabecular bone. Excretory pathways and their rates are also modeled. These include the mean time for excretion from the plasma to the urine, from the liver to the bile, and from the other soft tissues to the hair, skin, sweat, etc. The model permits the user to adjust the transition and residence times.

USEPA guidance (USEPA, 1994b) recommends using 400 mg/kg as a screening level for lead in soil for residential scenarios at CERCLA sites and at RCRA Corrective Action sites. Residential areas with soil lead below 400 mg/kg generally require no further action. However, in some special situations, further study is warranted below the screening level (e.g., wetlands, agricultural areas).

F.7.8 Manganese

F.7.8.1 Noncancer Toxicity

Manganese is nutritionally required in humans for normal growth and health (EPA, 2000). Humans exposed to approximately 0.8 mg manganese/kg/day in drinking water exhibited lethargy, mental disturbances (1/16 committed suicide), and other neurologic effects. The elderly appeared to be more sensitive than children. Oral treatment of laboratory rodents induced biochemical changes in the brain, but rodents did not exhibit the neurological signs exhibited by humans. Occupational exposure to high concentrations in air induced a generally typical spectrum of neurological effects and an increased incidence of pneumonia (ACGIH, 1986).

EPA presented the oral RfD for manganese of 0.02 mg/kg/day (EPA, 2000) based on drinking water and an oral RfD of 0.14 mg/kg/day based on food. The EPA (2000) presented a verified chronic inhalation RfC based on a LOAEL for impairment of neurobehaviorial function in occupationally exposed humans. The inhalation RfC is equivalent to 0.0143 ug/kg/day, assuming humans inhale 20 m³ of air/day and weigh 70 kg. The CNS and respiratory tract are target organs of inhalation exposure to manganese.

F.7.8.2 Carcinogenicity

The EPA (2000) classifies manganese in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans). Quantitative cancer risk estimates are not derived from Group D chemicals.

F.7.9 Mercury

Mercury occurs in three forms: elemental, organic, and inorganic. Although the toxicity of all forms is mediated by the mercury cation, the extent of absorption and pattern of distribution within the body, which determines the effects observed, depends on the form to which the organism is exposed (Goyer, 1991). Bacterial activity in the environment converts inorganic mercury to methyl mercury (Berlin, 1986). It is likely that either inorganic mercury or methyl mercury may be taken up by plants and enter the food chain, and this discussion will focus on inorganic and methyl mercury. Exposure to elemental mercury, which is more likely to occur in an occupational setting, is not discussed herein.

F.7.9.1 Pharmacokinetics

The GI absorption of inorganic mercury salts is about 2 to 10 percent in humans, and slightly higher in experimental animals (Berlin, 1986; Goyer, 1991). Inorganic mercury in the blood is roughly equally divided between the plasma and erythrocytes. Distribution is preferentially to the kidney, with somewhat lower concentrations found in the liver, and even lower levels found in the skin, spleen, testes, and brain (Berlin, 1986). Inorganic mercury is excreted principally through the feces and urine, with minor pathways including the secretions of exocrine glands and exhalation of elemental mercury vapor.

Methyl mercury is nearly completely (90 to 95 percent) absorbed from the GI tract (Berlin, 1986). The concentration of methyl mercury in the erythrocytes is about 10 times that in the plasma. Methyl mercury leaves the blood slowly, showing particular affinity for the brain, particularly in primates. In rats, 1 percent of the body burden of methyl mercury is found in the brain, but in humans, 10 percent of the body burden is found in the brain. Somewhat lower levels are found in the liver and kidney. During pregnancy, methyl mercury accumulates in the fetal brain, often at levels higher than in the maternal brain. Most tissues except the brain transform methyl mercury to inorganic mercury. Excretion of methyl mercury is principally via the bile, with a half-life of 70 days in humans not suffering from toxicity. Following exposure to methyl mercury, some of the mercury in the bile exists as methyl mercury and some as the inorganic form. The inorganic form is largely passed in the feces, but the methyl mercury is

subject to enterohepatic recirculation. Another important excretory pathway for methyl mercury is lactation.

F.7.9.2 Noncancer Toxicity

Target organs for inorganic or methyl mercury include the kidney, nervous system, fetus, and neonate. Acute oral exposure to high doses of inorganic mercury causes severe damage to the GI mucosa because of the corrosive nature of mercury salts, which may lead to bloody diarrhea, shock, circulatory collapse, and death (Berlin, 1986; Goyer, 1991). Acute sublethal poisoning induces severe kidney damage. Chronic exposure induces an autoimmune glomerular disease and renal tubular injury. The U.S. EPA (1997) presented a verified RfD of 0.3 ug/mg-day for chronic oral exposure to inorganic mercury, based on kidney effects in rats.

Acute or chronic exposure to methyl mercury leads to neurologic dysfunction (Berlin, 1986; Goyer, 1991). The region of the nervous system affected is species-dependent. Methyl mercury poisoning in rats induces peripheral nerve damage and kidney effects. In humans, the sensory cortex appears to be the most sensitive. The brain of the fetus and the neonate may be unusually sensitive to methyl mercury; retarded neurologic development was observed in prenatally exposed children whose mothers showed no clinical signs of poisoning. The U.S. EPA (1997) derived an RfD of 0.1 ug/kg/day for chronic oral exposure to methyl mercury based on developmental neurological abnormalities in human infants. An inhalation RfC of 0.0003 mg/m³ (uncertainty factor of 30) has been established for inorganic mercury based on neurotoxic effects in humans. This translates into a chronic RfD of 0.000086 mg/kg/day (U.S. EPA, 2000).

F.7.9.3 Carcinogenicity

The U.S. EPA (2000) classifies inorganic mercury in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans), based on no data regarding cancer in humans, and inadequate animal and supporting data. In an intraperitoneal injection study with metallic mercury in rats, sarcomas developed only in those tissues in direct contact with the test material (ATSDR, 1992d). A two-year dietary study in rats with mercuric acetate (inorganic mercury) yielded no evidence of carcinogenicity (ATSDR, 1992d). In mice, however, dietary

exposure to high doses of mercury chloride for up to 78 weeks induced renal adenomas and adenocarcinomas (ATSDR, 1992d). The U.S. EPA has not yet evaluated the carcinogenicity of organic mercury. No carcinogenic effect, however, was observed in a two-year feeding study with phenylmercuric acetate in rats (ATSDR, 1992d).

F.7.10 Nickel

F.7.10.1 Noncancer Toxicity

In a subchronic gavage study with nickel chloride in water, clinical signs of toxicity in rats included lethargy, ataxia, irregular breathing, reduced body temperature, salivation, and discolored extremities (EPA 2000). Inhalation exposure was associated with asthma and pulmonary fibrosis in welders using nickel alloys (ACGIH 1986). Lung effects were observed in laboratory animals exposed by inhalation. The EPA (2000) presented a verified RfD of 0.02 for chronic oral exposure to nickel, based on an NOAEL for decreased organ and body weights in a two-year dietary study with nickel sulfate in rats and an uncertainty factor of 300. The EPA (1997) presented the same value as a provisional subchronic oral RfD. The CNS appears to be the target organ for the oral toxicity of nickel. The lung is clearly the target organ for inhalation exposure.

F.7.10.2 Carcinogenicity

Occupational exposure to nickel was associated with increased risk of nasal, laryngeal and lung cancer (ATSDR 1995a). Inhalation exposure of rats to nickel subsulfide increased the incidence of lung tumors. The EPA (2000) presents a cancer weight-of-evidence Group A classification (human carcinogen) for nickel, and presents an inhalation unit risk of 0.00024 per mg/m^3 for nickel refinery dust. The unit risk is equivalent to 0.84 per $\text{ug}/\text{kg}/\text{day}$, assuming humans inhale 20 m^3 of air/day and weigh 70 kg. The quantitative estimate was derived from the human occupational studies.

F.7.11 Thallium

F.7.11.1 Noncancer Toxicity

Thallium is highly toxic; acute ingestion by humans or laboratory animals induced gastroenteritis, neurological dysfunction, and renal and liver damage (Kazantzis, 1986). Chronic ingestion of more moderate doses characteristically caused alopecia. Thallium was used medicinally to induce alopecia in cases of ringworm of the scalp, sometimes with disastrous results. In industrial (inhalation, oral, dermal) exposure, neurologic signs preceded alopecia, suggesting that the nervous system is more sensitive than the hair follicle. The EPA (2000) presented verified chronic oral RfD values for several thallium compounds (thallium acetate, thallium acetate, thallium carbonate, thallium chloride, thallium nitrate, thallium sulfate, and thallic oxide) based on increased incidence of alopecia and increased serum levels of liver enzymes indicative of hepatocellular damage in rats treated with thallium sulfate for 90 days. EPA (2000) presented a chronic oral RfD for thallium of 0.07 ug/kg/day.

F.7.11.2 Carcinogenicity

Thallium was classified as a cancer weight-of-evidence Group D substance (not classifiable as to carcinogenicity to humans) (EPA, 2000).

F.7.12 Vanadium

F.7.12.1 Noncancer Toxicity

The oral toxicity of vanadium compounds to humans is very low (Lagerkvist et al. 1986), probably because little vanadium is absorbed from the GI tract. Effects in humans exposed by inhalation include upper and lower respiratory tract irritation. A provisional subchronic and chronic oral RfD of 0.007 mg/kg/day was derived from an NOEL in rats in a lifetime drinking water study with vanadyl sulfate and an uncertainty factor of 100 (USEPA, 1997). A target organ could not be identified for oral exposure. The respiratory tract is the target organ for inhalation exposure.

F.7.12.2 Carcinogenicity

No information was located regarding the carcinogenicity of vanadium.

F.7.13 Zinc

F.7.13.1 Pharmacokinetics

Zinc is a nutritionally required trace element. Estimates of the efficiency of GI absorption of zinc in animals range from <10 to 90 percent (Elinder 1986). Estimates in normal humans range from approximately 20 to 77 percent (Elinder 1986; Goyer 1991). The net absorption of zinc appears to be homeostatically controlled, but it is unclear whether GI absorption, intestinal secretion, or both are regulated. Distribution of absorbed zinc is primarily to the liver (Goyer 1991), with subsequent redistribution to bone, muscle, and kidney (Elinder 1986). Highest tissue concentrations are found in the prostate. Excretion appears to be principally through the feces, in part from biliary secretion, but the relative importance of fecal and urinary excretion is species-dependent. The half-life of zinc absorbed from the GI tracts of humans in normal zinc homeostasis is approximately 162 to 500 days.

F.7.13.2 Noncancer Toxicity

Humans exposed to high concentrations of aerosols of zinc compounds may experience severe pulmonary damage and death (Elinder 1986). The usual occupational exposure is to freshly formed fumes of zinc, which can induce a reversible syndrome known as metal fume fever. Orally, zinc exhibits a low order of acute toxicity. Animals dosed with 100 times dietary requirement showed no evidence of toxicity (Goyer 1991). In humans, acute poisoning from foods or beverages prepared in galvanized containers is characterized by GI upset (Elinder 1986). Chronic oral toxicity in animals is associated with poor growth, GI inflammation, arthritis, lameness, and a microcytic, hypochromic anemia (Elinder 1986), possibly secondary to copper deficiency (Underwood 1977). The EPA (1997) presented a verified RfD of 0.3 mg/kg/day for chronic oral exposure to zinc, based on anemia in humans.

F.7.13.3 Carcinogenicity

The EPA (2000) classifies zinc in cancer weight-of-evidence Group D (not classifiable as to carcinogenicity to humans) based on inadequate evidence for carcinogenicity in humans and animals. The human data consist largely of occupational exposure studies not designed to detect a carcinogenic response, and of reports that prostatic zinc concentrations were lower in cancerous than in noncancerous tissue. The animal data consist of several dietary, drinking water, and zinc injection studies, none of which provided convincing data for a carcinogenic response.

F.7.14 Polyaromatic Hydrocarbons

PAHs are a large class of ubiquitous natural and anthropogenic chemicals, all with similar chemical structures (ATSDR 1990a).

F.7.14.1 Pharmacokinetics

Although quantitative absorption data for the PAHs were not located, benzo(a)pyrene was readily absorbed across the GI (Rees et al. 1971) and respiratory epithelia (Kotin et al. 1969; Vainich et al. 1976). The high lipophilicity of other compounds in this class suggests that other PAHs also would be readily absorbed across GI and respiratory epithelia.

Benzo(a)pyrene was distributed widely in the tissues of treated rats and mice, but primarily to tissues high in fat, such as adipose tissue and mammary gland (Kotin et al. 1969; Schlede et al. 1970a). Patterns of tissue distribution of other PAHs would be expected to be similar because of the high lipophilicity of the members of this class.

Studies of the metabolism of benzo(a)pyrene provide information relevant to other PAHs because of the structural similarities of all members of the class. Metabolism involves microsomal mixed function oxidase hydroxylation of one or more of the phenyl rings with the formation of phenols and dihydrodiols, probably via formation of arene oxide intermediates (EPA 1979a). The dihydrodiols may be further oxidized to diol epoxides, which, for certain members of the class, are known to be the ultimate carcinogens (LaVoie et al. 1982).

Conjugation with glutathione or glucuronic acid, and reduction to tetrahydrotetraols are important detoxification pathways. Metabolism of naphthalene resulted in the formation of 1,2-naphthoquinone, which induced cataract formation and retinal damage in rats and rabbits.

Excretion of benzo(a)pyrene or dibenzo(a,h)anthracene residues was reported to be rapid, although quantitative data were not located (EPA 1979b). Excretion occurred mainly via the feces, probably largely due to biliary secretion (Schlede et al. 1970a, 1970b). The EPA (1980) concluded that accumulation in the body tissues of PAHs from chronic low level exposure would be unlikely.

F.7.14.2 Noncancer Toxicity

Oral noncancer toxicity data are available for acenaphthene, anthracene, fluoranthene, fluorene, and naphthalene. Newborn infants, children, and adults exposed to naphthalene by ingestion, inhalation, or possibly by skin contact developed hemolytic anemia with associated jaundice and occasionally renal disease (EPA 1979c). In a 13-week gavage study in rats, treatment with 50 mg naphthalene/kg, 5 days/week for 13 weeks (35.7 mg/kg/day) induced no effects; higher doses presumably reduced the growth rate (National Toxicology Program (NTP) 1980). Application of an uncertainty factor of 1000 yielded a provisional RfD for chronic oral exposure of 0.04 mg/kg/day (EPA 1997). The very mild effect (decreased growth rate) apparently observed at higher doses suggests that the RfD is very conservatively protective.

F.7.14.3 Carcinogenicity

The PAHs are ubiquitous, being released to the environment from anthropogenic as well as from natural sources (ATSDR 1992a). Benzo(a)pyrene is the most extensively studied member of the class, inducing tumors in multiple tissues of virtually all laboratory species tested by all routes of exposure. Although epidemiology studies suggested that complex mixtures that contain PAHs (coal tar, soots, coke oven emissions, cigarette smoke) are carcinogenic to humans (EPA 1984), the carcinogenicity cannot be attributed to PAHs alone because of the presence of other potentially carcinogenic substances in these mixtures (ATSDR 1992a). In addition, recent investigations showed that the PAH fraction of roofing tar, cigarette smoke, and coke oven emissions accounted for only 0.1 to 8 percent of the total

mutagenic activity of the unfractionated complex mixture in Salmonella (Lewtas 1988). Aromatic amines, nitrogen heterocyclic compounds, highly oxygenated quinones, diones, and nitrooxygenated compounds, none of which would be expected to arise from in vivo metabolism of PAHs, probably accounted for the majority of the mutagenicity of coke oven emissions and cigarette smoke. Furthermore, coal tar, which contains a mixture of many PAHs, has a long history of use in the clinical treatment of a variety of skin disorders in humans (ATSDR 1990a).

Because of the lack of human cancer data, assignment of individual PAHs to EPA cancer weight-of-evidence groups was based largely on the results of animal studies with large doses of purified compound (EPA 1984). Frequently, unnatural routes of exposure, including implants of the test chemical in beeswax and trioctanoin in the lungs of female Osborne-Mendel rats, intratracheal instillation, and subcutaneous or intraperitoneal injection, were used. Benzo(a)anthracene, benzo(a)pyrene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene were classified in Group B2 (probable human carcinogens).

The EPA (2000) verifies a slope factor for oral exposure to benzo(a)pyrene of 7.3 per mg/kg/day, based on several dietary studies in mice and rats. Neither verified nor provisional quantitative risk estimates were available for the other PAHs in Group B2. The EPA (1980) promulgated an ambient water quality criterion for "total carcinogenic PAHs," based on an oral slope factor derived from a study with benzo(a)pyrene, as being sufficiently protective for the class. Largely because of this precedent, the quantitative risk estimates for benzo(a)pyrene were adopted for the other carcinogenic PAHs when quantitative estimates were needed.

Recent reevaluations of the carcinogenicity and mutagenicity of the Group B2 PAHs suggest that there are large differences between individual PAHs in cancer potency (Krewski et al., 1989). Based on the available cancer and mutagenicity data, and assuming that there is a constant relative potency between different carcinogens across different bioassay systems and that the PAHs under consideration have similar dose-response curves, Thorslund and Charnley (1988) derived relative potency values for several PAHs. A more recent Relative Potency Factor (RPF) scheme for the Group B2 PAHs was based only on the induction of lung epidermoid carcinomas in female Osborne-Mendel rats in the lung-implantation experiments (Clement International 1990).

F.7.15 Benzo[A]Anthracene

F.7.15.1 Noncancer Toxicity

The oral and inhalation RfD and RfC are not available at this time (EPA 2000).

F.7.15.2 Carcinogenicity

Benzo[a]anthracene has a weight of evidence classification of B2, a probable human carcinogen. The classification was based on sufficient data from animal bioassays. Benzo[a]anthracene produced tumors in mice exposed by gavage; intraperitoneal, subcutaneous or intramuscular injection; and topical application. Benzo[a]anthracene produced mutations in bacteria and in mammalian cells, and transformed mammalian cells in culture.

Although there are no human data that specifically link exposure to benzo[a]anthracene to human cancers, benzo[a]anthracene is a component of mixtures that have been associated with human cancer. These include coal tar, soot, coke oven emissions and cigarette smoke (U.S. EPA, 1984, 1990; IARC, 1984; Lee et al., 1976; Brockhaus and Tomingas, 1976).

Benzo[a]anthracene administration caused an increase in the incidence of tumors by gavage (Klein, 1963); dermal application (IARC, 1973); and both subcutaneous injection (Steiner and Faulk, 1951; Steiner and Edgecomb, 1952) and intraperitoneal injection (Wislocki et al., 1986) assays. A group of male mice was exposed to gavage solutions containing 3% benzo[a]anthracene for 5 weeks. There was an increased incidence of pulmonary adenomas and hepatomas.

Supporting data for carcinogenicity include genetic mutations in five different strains of Salmonella typhimurium. Benzo[a]anthracene produced positive results in an assay for mutations in Drosophila melongaster (Fahmy and Fahmy, 1973).

The currently used Oral Slope Factor (CSF) for Benzo[a]anthracene is 7.3E-01 per (mg/kg)/day which is extrapolated from the CSF for Benzo[a]pyrene (BaP), i.e., 0.1×7.3 (BaP) = 7.3E-01 per (mg/kg)/day (USEPA Region III Risk-Based Concentration Table, 10/1/99).

The inhalation CSF is not available.

F.7.16 Benzo(B)Fluorantene

F.7.16.1 Noncancer Toxicity

Little information is available on benzo(b)fluoranthene. However based on the similarities of chemical structures, most properties should be similar to benzo(a)pyrene.

F.7.16.2 Carcinogenicity

A Toxicity Equivalency Factor (TEF) has been developed (EPA, 1993) for benzo(b)fluoranthene which allows the estimation of an oral CSF of 0.73 mg/g/day. The EPA (2000) has classified benzo(b)fluoranthene in cancer weight-of-evidence Group B2 (Probable Human Carcinogen, sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans) based on lung tumors in mice.

F.7.17 Benzo[A]Pyrene (Bap)

F.7.17.1 Pharmacokinetics

Benzo(a)pyrene was readily absorbed across the GI (Rees et al. 1971) and respiratory epithelia (Kotin et al. 1969; Vainich et al. 1976). Benzo(a)pyrene was distributed widely in the tissues of treated rats and mice, but primarily to tissues high in fat, such as adipose tissue and mammary gland (Kotin et al. 1969; Schleder et al. 1970a).

Studies of the metabolism of benzo(a)pyrene provide information relevant to other PAHs because of the structural similarities of all members of the class. Metabolism involves microsomal mixed function oxidase hydroxylation of one or more of the phenyl rings with the

formation of phenols and dihydrodiols, probably via formation of arene oxide intermediates (EPA 1979a). The dihydrodiols may be further oxidized to diol epoxides, which, for certain members of the class, are known to be the ultimate carcinogens (LaVoie et al. 1982). Conjugation with glutathione or glucuronic acid, and reduction to tetrahydrotetraols are important detoxification pathways.

Excretion of benzo(a)pyrene residue was reported to be rapid, although quantitative data were not located (EPA 1979b). Excretion occurred mainly via the feces, probably largely due to biliary secretion (Schlede et al. 1970a, 1970b). The EPA (1980) concluded that accumulation in the body tissues of PAHs from chronic low level exposure would be unlikely.

F.7.17.2 Noncancer Toxicity

The oral RfD and inhalation RfC are not available at this time.

F.7.17.3 Carcinogenicity

The PAHs are ubiquitous, being released to the environment from anthropogenic as well as from natural sources (ATSDR 1992a). Benzo (a)pyrene is the most extensively studied member of the class, inducing tumors in multiple tissues of virtually all laboratory species tested by all routes of exposure. Although epidemiology studies suggested that complex mixtures that contain PAHs (coal tar, soots, coke oven emissions, cigarette smoke) are carcinogenic to humans (EPA 1984), the carcinogenicity cannot be attributed to PAHs alone because of the presence of other potentially carcinogenic substances in these mixtures (ATSDR 1990a). In addition, recent investigations showed that the PAH fraction of roofing tar, cigarette smoke, and coke oven emissions accounted for only 0.1 to 8 percent of the total mutagenic activity of the unfractionated complex mixture in Salmonella (Lewtas 1988). Aromatic amines, nitrogen heterocyclic compounds, highly oxygenated quinones, diones, and nitrooxygenated compounds, none of which would be expected to arise from in vivo metabolism of PAHs, probably accounted for the majority of the mutagenicity of coke oven emissions and cigarette smoke. Coal tar, which contains a mixture of many PAHs, has a long history of use in the clinical treatment of a variety of skin disorders in humans (ATSDR 1990a).

Because of the lack of human cancer data, assignment of individual PAHs to EPA cancer weight-of-evidence groups was based largely on the results of animal studies with large doses of purified compound (EPA 1984). Frequently, unnatural routes of exposure, including implants of the test chemical in beeswax and trioctanoin in the lungs of female Osborne-Mendel rats, intratracheal instillation, and subcutaneous or intraperitoneal injection, were used. Benzo (a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenzo(a,h)anthracene, and indeno(1,2,3-cd)pyrene were classified in Group B2 (probable human carcinogens).

The EPA (2000) verifies a slope factor for oral exposure to benzo(a)pyrene of 7.3 per mg/kg/day, based on several dietary studies in mice and rats. Neither verified nor provisional quantitative risk estimates were available for the other PAHs in Group B2. The EPA (1980) promulgated an ambient water quality criterion for "total carcinogenic PAHs," based on an oral slope factor derived from a study with benzo(a)pyrene, as being sufficiently protective for the class. Largely because of this precedent, the quantitative risk estimates for benzo(a)pyrene were adopted for the other carcinogenic PAHs when quantitative estimates were needed.

Human data specifically linking benzo[a]pyrene (BAP) to a carcinogenic effect are lacking. There are, however, multiple animal studies in many species demonstrating BAP to be carcinogenic following administration by numerous routes. In addition, BAP has produced positive results in numerous genotoxicity assays.

The data for animal carcinogenicity was sufficient. The animal data consist of dietary, gavage, inhalation, intratracheal instillation, dermal and subcutaneous studies in numerous strains of at least four species of rodents and several primates. Repeated BAP administration has been associated with increased incidences of total tumors and of tumors at the site of exposure. The tumor types in mice from oral diet studies include forestomach, squamous cell papillomas and carcinomas (Neal and Rigdon 1967).

Benzo [a]pyrene has been shown to cause genotoxic effects in a broad range of prokaryotic and mammalian cell assay systems (EPA 1990).

The oral slope factor presented in the Region III Risk-Based Concentration Table is 7.3E+0 per mg/kg/day. The cancer slope factor for inhalation is not available.

F.7.18 Dibenzo[A,H]Anthracene

F.7.18.1 Noncancer Toxicity

The oral RfD and inhalation RfC are not available.

F.7.18.2 Carcinogenicity

Classification -- B2; probable human carcinogen

The EPA (1997) has classified dibenzo(a,h)anthracene in cancer weight-of-evidence group B2 (Probable Human Carcinogen, sufficient evidence of carcinogenicity in animals). Based on carcinomas in mice following oral or dermal exposure and injection site tumors in several species following subcutaneous or intramuscular administration. Dibenzo[a,h]anthracene has induced DNA damage and gene mutations in bacteria as well as gene mutations and transformation in several types of mammalian cell cultures.

Although there are no human data that specifically link exposure to dibenzo[a,h]anthracene with human cancers, dibenzo[a]anthracene is a component of mixtures that have been associated with human cancer. These include coal tar, soot, coke oven emissions and cigarette smoke (EPA, 1984, 1990; IARC, 1984).

Dibenzo[a,h]anthracene has been shown to be carcinogenic when administered to mice by the oral route (Snell and Stewart, 1962, 1963) in a water-olive oil emulsion. Mice developed pulmonary adenomas, pulmonary carcinomas, and mammary carcinomas.

Dibenzo[a,h]anthracene has produced positive results in bacterial DNA damage and mutagenicity assays and in mammalian cell DNA damage, mutagenicity and cell transformation assays.

The currently used Oral Slope Factor (CSF) for Dibenzo[a,h]anthracene is 7.3E+00 per (mg/kg)/day which is extrapolated from the CSF for Benzo[a]pyrene i.e., 1.0×7.3 (BaP) = 7.3 per (mg/kg)/day (USEPA Region III Risk-Based Concentration Table, 10/1/99).

The inhalation Cancer Slope Factor for dibenzo(a,h)anthracene is not available.

F.7.19 Indeno(1,2,3-Cd)Pyrene

F.7.19.1 Noncancer Toxicity

Little information was found on the toxicity of indeno(1,2,3-cd)pyrene. Because of its structural similarity its properties should resemble benzo(a)pyrene.

F.7.19.2 Carcinogenicity

A Toxicity Equivalency Factor (TEF) has been developed for indeno(1,2,3-cd)pyrene (EPA 1993). This allows the estimation of an oral CSF of 0.73 mg/kg/day. The EPA (2000) has classified indeno(1,2,3-cd)pyrene in cancer weight-of-evidence Group B2 (Probable Human Carcinogen, sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans) based on tumors in mice following lung implants.

F.7.20 Bis(2-Ethylhexyl)Phthalate (Di[2-Ethylhexyl]Phthalate)

F.7.20.1 Noncancer Toxicity

The acute oral toxicity of bis(2-ethylhexyl)phthalate is very low; oral LD_{50/30} (lethal dose to 50 percent of population within 30 days without medical testament) values in rats and mice were 33,800 and 26,300 mg/kg, respectively (ACGIH, 1991). Repeated high-dose oral exposures were associated with decreased growth, altered organ weights, testicular degeneration, and developmental effects. The EPA (2000) presents a verified chronic oral RfD of 0.02 mg/kg/day based on an LOAEL for increased relative liver weight in guinea pigs and an uncertainty factor of 1000. The EPA (1997) adopted the chronic oral RfD as the provisional subchronic

oral RfD. The principal target organs for the toxicity of bis(2-ethylhexyl)phthalate are the liver and testis.

F.7.20.2 Carcinogenicity

The EPA (2000) classifies bis(2-ethylhexyl)phthalate in cancer weight-of evidence Group B2 (probable human carcinogen), based on inadequate human cancer data (one limited occupational study) and sufficient cancer data in laboratory animals. An oral slope factor of 0.014 per mg/kg/day was based on the increased incidence of liver tumors in a dietary study in male mice. An inhalation slope factor of 0.014 per mg/kg/day was presented by EPA (2000).

F.7.21 Aldrin/Dieldrin (Clement 1985)

F.7.21.1 Pharmacokinetics

Both aldrin and dieldrin are carcinogens, causing increases in a variety of tumors in rats at low but not at high doses and producing a higher incidence of liver tumors in mice. The reason for this reversed dose-response relationship is unclear. Neither appears to be mutagenic when tested in a number of systems. Aldrin and dieldrin are both toxic to the reproductive system and teratogenic. Reproductive effects include decreased fertility, increased fetal death, and effects on gestation; while teratogenic effects include cleft palate, webbed foot, and skeletal anomalies. Chronic effects attributed to aldrin and dieldrin include liver toxicity and central nervous system abnormalities. Both chemicals are acutely toxic; the oral LD₅₀ is around 50 mg/kg, and the dermal LD₅₀ is about 100 mg/kg.

F.7.21.1 Non-Carcinogenic Toxicity

Chronic feeding with aldrin induced evidence of degeneration of the liver in rats. (EPA 1997). The EPA (1997) presented a verified chronic oral RfD of 0.03 ug/kg/day based on a LOAEL for liver effects in rats and an uncertainty factor of 1000. The principal target organ of aldrin is the liver.

F.7.21.2 Carcinogenicity

The EPA (1997) classifies aldrin in cancer weight-of-evidence Group B2 (probable human carcinogen), based on inadequate human data and sufficient animal data. The human data consist of epidemiologic studies that had results that were statistically insignificant. Animal studies associated treatment with liver tumors in male and female mice. The EPA (1997) presented a verified oral slope factor of 17 per mg/kg/day, based on the increased incidence of liver tumors in mice treated in the diet. An inhalation risk estimate was not derived.

F.7.22 Chlordane

Technical chlordane is a mixture of at least 50 related compounds (ATSDR 1992b). The principal components of the mixture are cis- and trans-chlordane, heptachlor, cis- and trans-nonachlor, and alpha-, beta- and gamma-chlordane. Each component has its own environmental fate and transport kinetics, so it is unlikely that the chlordane identified at the site would have the same chemical composition as technical chlordane. It is unclear which chlordane component(s) were found at the site.

F.7.22.1 Pharmacokinetics

Kinetic studies in rats, in which the area under the curve was compared following intravenous and oral dosing, indicate that approximately 80 percent of an oral dose of trans-chlordane is absorbed from the GI tract (Ohno et al. 1986). In animals, absorbed chlordane is distributed most rapidly to the liver and kidneys, probably because of the extensive vascularity of these organs (Ohno et al. 1986), followed by redistribution to adipose tissue (Barnett and Dorough 1974). In humans, levels of chlordane residues in adipose tissue increase with increasing duration of exposure (ATSDR 1992b). Metabolism involves principally oxidation, dechlorination, and conjugation, yielding lipophilic products that accumulate in adipose tissue, as well as more polar products that are excreted. Chlordane residues are excreted principally through the bile, although considerable species differences occur. Lactation is an important mechanism of excretion of chlordane residues retained in body fat.

F.7.22.2 Non-Carcinogenic Toxicity

An acute oral lethal dose of chlordane in humans is estimated to be 25 to 50 mg/kg (ATSDR 1992b). Symptoms of acute oral or inhalation intoxication in humans consistently include GI disturbances such as vomiting, cramps, and diarrhea, and neurological effects including headache, irritability, dizziness, incoordination, convulsions, and coma. Data were not located regarding symptoms or effects in humans chronically exposed by the oral route, and no noncancer effects were observed in several studies of occupationally exposed humans. Mild liver lesions were observed in chronic oral studies in rats and mice. Prenatal or early postnatal exposure of mice to chlordane damages the developing immune system and nervous system. Target organs of chlordane include the liver, nervous system, and the fetus and neonate.

The EPA (1997) derived an RfD of 0.06 mg/kg/day for chronic oral exposure to chlordane, based on an NOEL of 0.055 mg/kg/day for liver effects in a 30-month dietary study in rats (Velsicol Chemical Company 1983). An uncertainty factor of 1,000 was applied; factors of 10 each for inter- and intraspecies variation, and to reflect deficiencies in the database.

F.7.22.3 Carcinogenicity

The EPA (2000) classifies chlordane in cancer weight-of-evidence Group B2, based on inadequate evidence in humans and sufficient evidence in animals. The human data consist of several epidemiologic studies of chlordane manufacturing workers and pesticide applicators. The only indication of a carcinogenic effect was a borderline significantly increased incidence of bladder cancer in one study of pesticide applicators, but chlordane exposure was not quantified and the workers were concomitantly exposed to other carcinogenic pesticides. The animal data consist of several studies in which oral exposure induced a dose-related increase in the incidence of liver tumors. The evidence for carcinogenicity in rats is equivocal. The EPA (1997) derived an oral slope factor of 1.3 per mg/kg/day and an inhalation unit risk of 0.00037 per ug/m³ based on liver tumor incidence in two dietary studies in mice. The unit risk is equivalent to an inhalation reference dose of 1.29 per mg/kg/day (EPA 1997), assuming humans inhale 20m³ of air/day and weigh 70 kg.

F.7.23 Dieldrin

F.7.23.1 Noncancer Toxicity

The EPA (2000) derived a RfD of 5×10^{-5} mg/kg/day for chronic oral exposure based on a NOAEL of 0.005 mg/kg/day for liver lesions in a two-year rat feeding study (Walker et al., 1969) with an uncertainty factor of 100. The LOAEL was identified as 0.05 mg/kg/day.

At the end of two years the rats had increased liver weights and histopathological examinations revealed liver parenchymal cell changes. These hepatic lesions were considered to be characteristic of exposure to an organochlorine insecticide.

The chronic inhalation RfC is not available at this time.

F.7.23.2 Carcinogenicity

EPA (2000) classifies dieldrin in cancer weight-of-evidence B2. Dieldrin is carcinogenic in seven strains of mice when administered orally. Dieldrin is structurally related to compounds (aldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid) which produce tumors in rodents.

Human carcinogenicity data is considered inadequate. Two studies of workers exposed to aldrin and to dieldrin reported no increased incidence of cancer. Both studies were limited in their ability to detect an excess of cancer deaths.

Animal carcinogenicity data was sufficient. Dieldrin has been shown to be carcinogenic in various strains of mice of both sexes. At different dose levels the effects range from benign liver tumors, to hepatocarcinomas with transplantation confirmation, to pulmonary metastases.

Supporting data for carcinogenicity include genotoxicity tests. Dieldrin causes chromosomal aberrations in mouse cells (Markaryan, 1966; Majumdar et al., 1976) and in human lymphoblastoid cells (Trepanier et al., 1977), mutation in Chinese hamster cells (Ahmed et al.,

1977), and unscheduled DNA synthesis in rat (Probst et al., 1981) and human cells (Rocchi et al., 1980).

EPA (2000) reports an Oral Slope Factor of 16 per (mg/kg)/day based on a diet study in mice that produced liver carcinomas.

This inhalation cancer slope factor of 16 per mg/kg/day was calculated from the oral slope factor.

F.7.24 Heptachlor Epoxide (Clement, 1985)

F.7.24.1 Health Effects

Heptachlor epoxide is a liver carcinogen when administered orally to mice. Results from mutagenicity bioassays suggest that this compound also may have genotoxic activity. Reproductive and teratogenic effects in rats include decreased litter size, shortened life span of suckling rats, and development of cataracts in offspring.

Tests with laboratory animals, primarily rodents, demonstrate acute and chronic toxic effects due to heptachlor exposure. Although heptachlor epoxide is absorbed most readily through the gastrointestinal tract, inhalation and skin contact are also potential routes of exposure. Acute exposure by various routes can cause development of hepatic vein thrombi and can affect the central nervous system and cause death. Chronic exposure induces liver changes, affects hepatic microsomal enzyme activity, and causes increased mortality in offspring. The oral LD₅₀ for heptachlor epoxide in the rat is 47 mg/kg.

Although there are reports of acute and chronic toxicity in humans, with symptoms including tremors, convulsions, kidney damage, respiratory collapse, and death, details of such episodes are not well documented. Heptachlor epoxide has been found in a high percentage of human adipose tissue samples, and also in human milk samples and biomagnification of heptachlor epoxide occurs. This compound also has been found in the tissues of stillborn infants, suggesting an ability to cross the placenta and bioaccumulate in the fetus.

The oral RfD for heptachlor epoxide is 1.30E-05 mg/kg-day based on increased liver to weight ratios in male and female dogs. Heptachlor epoxide is classified as a B2 carcinogen oral CSF for heptachlor epoxide is 9.1 per mg/kg-day based on an increased incidence of liver carcinomas. The inhalation CSF for heptachlor epoxide is 9.1 per mg/kg-day.

F.7.25 DDT (4,4'-Dichlorodiphenyl-Trichloroethane)

F.7.25.1 Pharmacokinetics

Dichlorodiphenyltrichloroethane (DDT) is readily absorbed when dissolved in oils, fats, or lipid solvents, but is poorly absorbed as dry powder or aqueous suspension. Once absorbed, DDT concentrates in adipose tissue. Storage in fat is protective because it decreases the amount of chemicals at the site of toxic action, the brain. At a constant rate of intake, concentrations in adipose tissue reach a steady state and remain relatively constant. When exposure ceases, DDT is slowly eliminated. The rate of elimination is estimated to be 1 percent of stored DDT excreted per day (Gartrell 1985).

After absorption in mammals, DDT degrades by dehydrochlorination to unsaturated DDE and by substitution of hydrogen for one chlorine atom yielding DDD. DDD is further metabolized through a series of intermediates yielding DDA. DDA is relatively water soluble and excreted primarily in the urine. Ingestion studies of DDT administered to volunteers demonstrated that within 24 hours, urinary DDA excretion increased detectably. Excretion of DDT as DDA appeared to be totally dependent on preferential reductive dechlorination of DDT to DDD (rather than DDE) and then to DDA (Clayton 1981).

F.7.25.2 Noncancer Toxicity

The CNS is an important target organ in humans acutely exposed to DDT. Symptoms include altered sensory perception, headache, nausea, disequilibrium, confusion, tremors, and convulsions (Hayes 1982; ATSDR 1993). Tremors and hyperirritability were observed in chronically exposed animals (NCI 1978; Rossi et al. 1977). The liver appears to be the other important target organ, at least in animals. Liver effects include enzyme induction, increased liver weight, increased serum levels of liver enzymes, hepatocellular hypertrophy, and necrosis (ATSDR 1993). The EPA (2000) derived an RfD of 0.5 mg/kg/day for chronic oral exposure

from an NOEL of 0.05 mg/kg/day for liver effects in a 15- to 27-week feeding study in rats (Laug et al. 1950). An uncertainty factor of 100 was applied with factors of 10 each for inter- and intraspecies variation.

Dermal exposure has been associated with no illness and usually no irritation. Subcutaneous injection of colloidal suspensions of DDT in saline up to 30 ppm caused no irritation. Studies of DDT-impregnated clothing have found it to cause no irritation (Hayes 1982). The earliest symptom of acute DDT poisoning is paresthesia of the mouth and lower part of the face. This is followed by paresthesia of same areas and of the tongue and then dizziness, and tremors of extremities, confusion, malaise, headache, fatigue, and delayed vomiting. Vomiting is probably of central origin and not due to local irritation. Convulsions occur only in severe poisoning. Onset may be as soon as 30 minutes after ingestion of a large dose or as late as six hours after smaller but still-toxic doses. Recovery from mild poisoning usually is essentially complete in 24 hours, but recovery from severe poisoning requires several days (Hayes 1982).

There is no documented evidence that dietary absorption of DDT, alone or in combination with insecticides of the aldrin-toxaphene group, has caused cancer in the general population. No evidence has been presented that DDT has caused cancer among the millions of individuals (almost entirely men) who have been handling or spraying DDT (as dust, solution, and suspension) in all parts of the world and under all possible climatic conditions.

DDT is a mixture of *o,p*-DDT and related compounds. One of the more important of the DDT isomers is *o,p*-DDT. These agents have prominent estrogenic effects that have been well-characterized in a number of assay systems (Johnson, et al. 1988). The estrogenicity of DDT has led to the supposition that it may adversely affect reproductive outcome by causing birth defects, increasing pregnancy complications, or affecting fertility (RTC 1990).

F.7.25.3 Carcinogenicity

The EPA (1997) has classified DDT in cancer weight-of-evidence Group B 2 (probable human carcinogen) based on the observation of tumors (generally of the liver) in seven studies in various mouse strains and in three studies in rats. The EPA (1997) derived an oral slope factor of 3.4×10^{-1} per mg/kg/day from liver tumors in oral (diet) studies in the mouse and the

rat. An inhalation unit risk of 9.7×10^{-5} per $\mu\text{g}/\text{m}^3$, equivalent to 0.34 per $\text{mg}/\text{kg}/\text{day}$ (assuming a 70 kg adult inhales 20 m^3 of air/day), was derived from the same oral (diet) studies.

F.7.26 1,1-Dichloroethene

F.7.26.1 Noncancer Toxicity

Chronic oral exposure of laboratory animals to 1,1-dichloroethene induced liver effects (EPA 2000). In animals, inhalation exposure induced degenerative changes in the liver and kidneys (ATSDR 1992c). No health effects were observed in a limited study of 138 exposed workers (ACGIH 1986). The EPA (2000) presents a verified RfD for chronic oral exposure of 0.009 $\text{mg}/\text{kg}/\text{day}$, based on an NOAEL for liver effects in a chronic drinking water study in rats and an uncertainty factor of 1000. The EPA (1997) presented the same value as a provisional subchronic oral RfD. The liver and kidneys are the target organs for exposure to 1,1-dichloroethene.

F.7.26.2 Carcinogenicity

EPA classified 1,1-dichloroethene as a cancer weight-of-evidence Group C compound (possible human carcinogen), based on an inadequate occupational exposure cancer study, limited data in several animal studies, its mutagenicity and ability to alkylate deoxyribonucleic acid (DNA), and its structural similarity to vinyl chloride, a known human carcinogen (EPA 2000). The eighteen available animal studies (11 by inhalation exposure, 5 by oral exposure, and 1 each by dermal application and subcutaneous injection) were limited in sensitivity by various deficiencies in design. Credible evidence that 1,1-dichloroethene was a complete carcinogen was provided only by one 12-month inhalation study in mice, in which the incidence of kidney adenocarcinomas was significantly greater in the high-dose males than in the control males. A slope factor of 0.6 per $\text{mg}/\text{kg}/\text{day}$ for oral exposure was based on the increase in incidence of adrenal pheochromocytomas in male rats treated by gavage for two years, even though the increase was not statistically significant (EPA, 2000). A unit risk for inhalation exposure of $5.0\text{E}-05$ per $\mu\text{g}/\text{m}^3$ was based on the incidence of kidney adenocarcinomas in male mice in the inhalation study mentioned above (EPA, 2000). The unit risk is equivalent to 0.175 per $\text{mg}/\text{kg}/\text{day}$, assuming humans inhale 20 m^3 of air/day and weight 70 kg.

F.7.27 1,2-Dichloroethene, Total (1,2-Dichloroethylene, Total)

EPA presents an oral reference dose of 0.009 mg/kg/day for total 1,2-dichloroethene.

F.7.27.1 Cis-1,2-Dichloroethene (Cis-1,2-Dichloroethylene)

F.7.27.1.1 Noncancer Toxicity

Repeated oral exposure of rats to cis-1,2-dichloroethene was associated with signs of anemia (decreased hematocrit and hemoglobin) (EPA, 2000). Inhalation exposure to isomeric mixtures of 1,2-dichloroethene induced narcosis, and mixed isomers of 1,2-dichloroethene were used as an anesthetic gas (ACGIH, 1991). The EPA (2000) presented a provisional chronic oral RfD of 0.01 mg/kg/day based on an NOAEL for signs of anemia in rats and an uncertainty factor of 3000. A provisional subchronic oral RfD of 0.1 mg/kg/day was derived from the same NOAEL and an uncertainty factor of 300. Target organs appear to be the erythrocyte for oral exposure and the CNS for inhalation exposure.

F.7.27.1.2 Carcinogenicity

The EPA (2000) classifies cis-1,2-dichloroethene as a cancer weight-of-evidence Group D compound (not classifiable as to carcinogenicity to humans), based on an absence of human or animal cancer data. Quantitative estimates of cancer risk are not derived for Group D chemicals.

F.7.27.2 Trans-1,2-Dichloroethene (Trans-1,2,-Dichloroethylene)

F.7.27.2.1 Noncancer Toxicity

The oral LD_{50/30} for trans-1,2-dichloroethene in rats was 1275 mg/kg; death was preceded by CNS and respiratory depression (ACGIH, 1991). Histopathologic examination revealed lesions in the lungs and heart. Prolonged oral administration induced clinicopathologic evidence of mild liver damage (EPA, 2000). An NOAEL for this effect in a 90-day drinking water study in

mice and an uncertainty factor of 1000 was the basis for a verified chronic oral RfD of 0.02 mg/kg/day. A provisional subchronic oral RfD of 0.2 mg/kg/day was derived from the same NOAEL and an uncertainty factor of 100 (EPA, 2000). The target organs for inhalation exposure to trans-1,2-dichloroethene are the CNS, heart, and lungs; the liver appears to be the principal target of oral exposure.

F.7.27.2.2 Carcinogenicity

Data regarding the carcinogenicity of trans-1,2-dichloroethene were not located.

F.7.28 1,1,1-Trichloroethane

F.7.28.1 **Non-Carcinogenic Toxicity**

The toxicity of oral exposure to 1,1,1-trichloroethane is low (ACGIH 1986). Chronic ingestion by laboratory animals reduced growth rate, but produced little pathology in internal organs (ATSDR 1990b). Acute inhalation exposure of humans or animals to high levels induced death due to narcosis or cardiac sensitization (ACGIH 1986). Occupational exposure was not associated with systemic effects. A provisional chronic inhalation RfC of 1 mg/m³ was derived from an NOAEL for slight growth retardation in guinea pigs and an uncertainty factor of 1,000. The provisional subchronic inhalation RfC, based on the same NOAEL and an uncertainty factor of 100, was 10 mg/m³. The chronic and subchronic inhalation RfC values are equivalent to 0.3 and 3 mg/kg/day, respectively, assuming humans inhale 20 m³ of air/day and weigh 70 kg. Target organs for inhalation exposure to 1,1,1-trichloroethane are the CNS and heart.

F.7.28.2 **Carcinogenicity**

The EPA (2000) classifies 1,1,1-trichloroethane as a cancer weight-of-evidence Group D compound (not classifiable as to carcinogenicity to humans). There are no reported human cancer data, and animal studies (78-week gavage studies in rats and mice, and a 12-month inhalation study in rats) were inadequate to determine the carcinogenicity of 1,1,1-trichloroethane in animals. Quantitative cancer risk estimates are not derived for Group D compounds.

F.7.29 Trichloroethene

F.7.29.1 Noncancer Toxicity

Little is known about the toxicity of prolonged oral exposure to trichloroethene. Acute inhalation exposure to high levels induced anesthesia, tachypnea, and ventricular arrhythmias (ACGIH, 1986). Occupational exposure was associated with headache, dizziness, lassitude, and other CNS effects. Prolonged inhalation exposure of animals affected the liver and kidneys. An oral RfD of 0.006 mg/kg/day was presented by EPA (2000). An inhalation RfC value was not located for trichloroethene in IRIS or HEAST. The principal target organs for trichloroethene are the CNS and heart, and, to a lesser extent, the liver and kidney.

F.7.29.2 Carcinogenicity

Carcinogenicity studies in laboratory animals showed increased incidence of hepatocellular carcinomas (gavage exposure) and malignant lymphomas (inhalation exposure) in mice and increased incidence of renal adenocarcinomas in male rats (gavage) (ATSDR, 1995b). Cancer studies in humans were inadequate. Interpretation of the data regarding the carcinogenicity of trichloroethene is controversial, and the EPA (2000) has not adopted a final position on a cancer weight-of-evidence classification. Currently, EPA believes the weight-of-evidence to be on the C-B2 continuum (possible-probable human carcinogen), and presents the slope factor of 0.011 per mg/kg/day for oral exposure and 0.006 per mg/kg/day for inhalation exposure (EPA, 2000).

F.7.30 Vinyl Chloride

F.7.30.1 Noncancer Toxicity

Data were not located regarding oral exposure of humans to vinyl chloride (ATSDR 1995c). In rats, lifetime dietary ingestion of vinyl chloride slightly but significantly increased mortality and induced mild histopathologic effects in the liver. Several early occupational studies associated vinyl chloride exposure with a syndrome known as vinyl chloride disease, which includes acroosteolysis (dissolution of the ends of the distal phalanges of the hands), circulatory

disturbances in the extremities, Raynaud syndrome (sudden, recurrent bilateral cyanosis of the digits), scleroderma, hematologic effects, effects on the lungs, and impaired liver function and liver damage. Mild neurologic effects were also associated with occupational exposure. Long-term inhalation studies in rats and mice identified elevated relative liver weight as a sensitive indicator of liver effects. Neither inhalation RfC values nor oral RfD values for vinyl chloride were located. The principal target organs for vinyl chloride appear to be the CNS and the liver.

F.7.30.2 Carcinogenicity

The EPA (2000) lists vinyl chloride as an EPA cancer weight-of-evidence Group A compound (human carcinogen) and presents a verified oral slope factor of 1.9 per mg/kg/day, based on the increased incidence of liver and lung tumors in a lifetime dietary study in rats. An inhalation unit risk of $8.4E-05$ per $\mu\text{g}/\text{m}^3$, equivalent to 0.3 per mg/kg/day, assuming humans inhale 20 m^3 of air/day and weigh 70 kg, is based on liver tumors in rats intermittently exposed by inhalation for 12 months.

F.7.31 Polychlorinated Biphenyls

F.7.31.1 Noncancer Toxicity

Epidemiologic studies of women in the United States associated oral PCB exposure with low birth weight or retarded musculoskeletal or neurobehavioral development of their infants (ATSDR 1992e). Oral studies in animals established the liver as the target organ in all species, and the thyroid as an additional target organ in the rat. Effects observed in monkeys included gastritis, anemia, chloracne-like dermatitis, and immunosuppression. Oral treatment of animals induced developmental effects, including retarded neurobehavioral and learning development in monkeys. Oral RfD values of 0.02 $\mu\text{g}/\text{kg}/\text{day}$ for Aroclor-1254 and 0.07 $\mu\text{g}/\text{kg}/\text{day}$ for Aroclor-1016 were located.

Occupational exposure to PCBs was associated with upper respiratory tract and ocular irritation, loss of appetite, liver enlargement, increased serum concentrations of liver enzymes, skin irritation, rashes and chloracne, and, in heavily exposed female workers, decreased birth

weight of their infants (ATSDR 1992e). Concurrent exposure to other chemicals confounded the interpretation of the occupational exposure studies. Laboratory animals exposed by inhalation to Aroclor-1254 vapors exhibited moderate liver degeneration, decreased body weight gain and slight renal tubular degeneration. Neither subchronic nor chronic inhalation RfC values were available.

Target organs for PCBs include the skin, liver, fetus, and neonate.

F.7.31.2 Carcinogenicity

The EPA (2000) classifies the PCBs as EPA cancer weight-of-evidence Group B2 substances (probable human carcinogens), based on inadequate data in humans and sufficient data in animals. The human data consist of several epidemiologic occupational and accidental oral exposure studies with serious limitations, including poorly quantified concentrations of PCBs and durations of exposure, and probable exposures to other potential carcinogens.

The animal data consist of several oral studies in rats and mice with various aroclors, kanexchors, or clophens (commercial PCB mixtures manufactured in the United States, Japan and Germany, respectively) that reported increased incidence of liver tumors in both species (EPA 2000).

The EPA (2000) presents a verified oral slope factor and an inhalation slope factor of 2.0 per mg/kg/day for PCBs based on liver tumors in rats treated with Aroclor-1260.

F.7.32 Dioxins

Specific congeners and homologues of these classes of interest at this site include 1,2,3,4,6,7,8-heptachlorodibenzofuran and -heptachlorodibenzo-p-dioxin; 1,2,3,4,7,8,9-heptachlorodibenzofuran and -heptachlorodibenzo-p-dioxin; 1,2,3,4,7,8- hexachlorodibenzofuran and -hexachlorodibenzo-p-dioxin; 1,2,3,6,7,8- and 2,3,4,6,7,8- hexachlorodibenzofuran; 1,2,3,7,8,9-hexachlorodibenzofuran and -hexachlorodibenzo-p- dioxin; 1,2,3,6,7,8-hexachlorodibenzo-p-dioxin; unspecified hexachlorodibenzofurans and dibenzo-p-dioxins; 1,2,3,7,8- and 2,3,4,7,8-pentachlorodibenzofuran; unspecified

pentachlorodibenzofurans; 2,3,7,8-tetrachlorodibenzofuran; and unspecified tetrachlorodibenzofurans.

F.7.32.1 Noncancer Toxicity

Of the members of these classes, the toxicity of 2,3,7,8-TCDD has been studied most extensively. The only effect in humans clearly attributable to 2,3,7,8-TCDD was chloracne (ATSDR 1999). The data, however, also associated exposure to 2,3,7,8-TCDD with hepatotoxicity and neurotoxicity in humans. In animals, toxicity of 2,3,7,8-TCDD is most commonly manifested as a wasting syndrome with thymic atrophy, terminating in death, with a large number of organ systems showing nonspecific effects. Chronic treatment of animals with 2,3,7,8-TCDD or a mixture of two isomers of hexachlorodibenzo-p-dioxin resulted in liver damage. Immunologic effects may be among the more sensitive endpoints of exposure to the PCDDs in animals. In animals 2,3,7,8-TCDD is a developmental and reproductive toxicant. No verified or provisional noncancer toxicity values were located for any of the chemicals of interest in these classes (EPA 1999, 2000).

F.7.32.3 Carcinogenicity

Data regarding the carcinogenicity of 2,3,7,8-TCDD to humans, obtained from epidemiologic studies of workers exposed to pesticides or to other chlorinated chemicals known to be contaminated with 2,3,7,8-TCDD, are conflicting (ATSDR 1999). The interpretation of these studies is not clear because exposure to 2,3,7,8-TCDD was not quantified, multiple routes of exposure (dermal, inhalation, oral) were involved, and the workers were exposed to other potentially carcinogenic compounds. In animals, however, 2,3,7,8-TCDD is clearly carcinogenic, inducing thyroid, lung, and liver tumors in orally treated rats and mice (EPA 1985). Similarly, oral treatment with a mixture of two hexachlorodibenzo-p-dioxin isomers induced liver tumors in rats and mice. On the basis of the animal data, 2,3,7,8-TCDD and the hexachlorodibenzo-p-dioxins were assigned to EPA cancer weight-of-evidence Group B2 (probable human carcinogen). Although the other PCDDs and PCDFs were not formally classified as to carcinogenicity to humans, for regulatory purposes they are treated as probable human carcinogens.

The EPA (1997) presents provisional oral and inhalation slope factors for 2,3,7,8-TCDD of 150,000 per mg/kg/day, based on the incidence of liver and lung tumors in an oral study in rats.

Much less is known about the toxicity of other CDD and CDF congeners. Based on available toxicity data, EPA has developed a method for expressing toxicities of these compounds in terms of equivalent amounts of 2,3,7,8-TCDD. "Toxicity equivalency factors", or TEFs, are used to convert the concentration of a given CDD/CDF into an equivalent concentration of 2,3,7,8-TCDD.

F.7.33 Asbestos

F.7.33.1 Noncancer Toxicity

Data not available at this time.

F.7.33.2 Carcinogenicity

This section provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to the following sections for information on long-term toxic effects other than carcinogenicity.

Weight-Of-Evidence Classification

Classification -- A; human carcinogen

Basis -- Observation of increased mortality and incidence of lung cancer, mesotheliomas and gastrointestinal cancer in occupationally exposed workers are consistent across investigators and study populations. Animal studies by inhalation in two strains of rats showed similar findings for lung cancer and mesotheliomas. Animal evidence for carcinogenicity via ingestion is limited (male rats fed intermediate-range chrysotile fibers; i.e., >10 um length, developed benign polyps), and epidemiologic data in this regard are inadequate.

Human Carcinogenicity Data

Sufficient. Numerous epidemiologic studies have reported an increased incidence of deaths due to cancer, primarily lung cancer and mesotheliomas associated with exposure to inhaled asbestos. Among 170 asbestos insulation workers in North Ireland followed for up to 26 years, an increased incidence of death was seen due to all cancers (SMR=390), cancers of the lower respiratory tract and pleura (SMR=1760) (Elmes and Simpson, 1971) and mesothelioma (7 cases). Exposure was not quantified.

Selikoff (1976) reported 59 cases of lung cancer and 31 cases of mesothelioma among 1249 asbestos insulation workers followed prospectively for 11 years. Exposure was not quantified. A retrospective cohort mortality study (Selikoff et al., 1979) of 17,800 U.S. and Canadian asbestos insulation workers for a 10-year period using best available information (autopsy, surgical, clinical) reported an increased incidence of cancer at all sites (319.7 expected vs. 995 observed, SMR=311) and cancer of the lung (105.6 expected vs. 486 observed, SMR=460). A modest increase in deaths from gastrointestinal cancer was reported along with 175 deaths from mesothelioma (none expected). Years of exposure ranged from less than 10 to greater than or equal to 45. Levels of exposure were not quantified. In other epidemiologic studies, the increase for lung and pleural cancers has ranged from a low of 1.9 times the expected rate, in asbestos factory workers in England (Peto et al., 1977), to a high of 28 times the expected rate, in female asbestos textile workers in England (Newhouse et al., 1972). Other occupational studies have demonstrated asbestos exposure-related increases in lung cancer

and mesothelioma in several industries including textile manufacturing, friction products manufacture, asbestos cement products, and in the mining and milling of asbestos. The studies used for the inhalation quantitative estimate of risk are listed in the table in Section II.C.2.

A case-control study (Newhouse and Thompson, 1965) of 83 patients with mesothelioma reported 52.6% had occupational exposure to asbestos or lived with asbestos workers compared with 11.8% of the controls. Of the remaining subjects, 30.6% of the mesothelioma cases lived within one-half mile of an asbestos factory compared with 7.6% of the controls.

The occurrence of pleural mesothelioma has been associated with the presence of asbestos fibers in water, fields and streets in a region of Turkey with very high environmental levels of naturally-occurring asbestos (Baris et al., 1979).

Kanarek et al. (1980) conducted an ecologic study of cancer deaths in 722 census tracts in the San Francisco Bay area, using cancer incidence data from the period of 1969-1971. Chrysotile asbestos concentrations in drinking water ranged from nondetectable to $3.6E+7$ fibers/L. Statistically significant dose-related trends were reported for lung and peritoneal cancer in white males and for gall bladder, pancreatic and peritoneal cancer in white females. Weaker correlations were reported between asbestos levels and female esophageal, pleural and kidney cancer, and stomach cancer in both sexes. In an extension of this study, Conforti et al. (1981) included cancer incidence data from the period of 1969-1974. Statistically significant positive associations were found between asbestos concentration and cancer of the digestive organs in white females, cancers of the digestive tract in white males and esophageal, pancreatic and stomach cancer in both sexes. These associations appeared to be independent of socioeconomic status and occupational exposure to asbestos.

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Marsh (1983) reviewed eight independent ecologic studies of asbestos in drinking water carried out in five geographic areas. It was concluded that even though one or more studies found an association between asbestos in water and cancer mortality (or incidence) due to neoplasms of various organs, no individual study or aggregation of studies exists that would establish risk levels from ingested asbestos. Factors confounding the results of these studies

include the possible underestimates of occupational exposure to asbestos and the possible misclassification of peritoneal mesothelioma as GI cancer.

Polissar et al. (1984) carried out a case-control study which included better control for confounding variables at the individual level. The authors concluded that there was no convincing evidence for increased cancer risk from asbestos ingestion. At the present time, an important limitation of both the case-control and the ecologic studies is the short follow-up time relative to the long latent period for the appearance of tumors from asbestos exposure.

Animal Carcinogenicity Data

Sufficient. There have been about 20 animal bioassays of asbestos. Gross et al. (1967) exposed 61 white male rats (strain not reported) to 86 mg chrysotile asbestos dust/cu.m for 30 hours/week for 16 months. Of the 41 animals that survived the exposure period, 10 had lung cancer. No lung cancer was observed in 25 controls.

Reeves (1976) exposed 60-77 rats/group for 4 hours/day, 4 days/week for 2 years to doses of 48.7-50.2 mg/cu.m crocidolite, 48.2-48.6 mg/cu.m amosite and 47.4-47.9 mg/cu.m chrysotile. A 5-14% incidence of lung cancer was observed among concentration groups and was concentration-dependent.

Wagner et al. (1974) exposed CD Wistar rats (19-52/group) to 9.7-14.7 mg/cu.m of several types of asbestos for 1 day to 24 months for 7 hours/day, 5 days/week. A duration-dependent increased incidence of lung carcinomas and mesotheliomas was seen for all types of asbestos after 3 months of exposure compared with controls.

F344 rats (88-250/group) were exposed to intermediate range chrysotile asbestos ($1291E+8$ f/g) in drinking water by gavage to dams during lactation and then in diet throughout their lifetime (NTP, 1985). A statistically significant increase in incidence of benign epithelial neoplasms (adenomatous polyps in the large intestine) was observed in male rats compared with pooled controls of all NTP oral lifetime studies (3/524). In the same study, rats exposed to short range chrysotile asbestos ($6081E+9$ f/g) showed no significant increase in tumor incidence.

Ward et al. (1980) administered 10 mg UICC amosite asbestos 3 times/week for 10 weeks by gavage to 50 male F344 rats. The animals were observed for an additional 78-79 weeks post-treatment. A total of 17 colon carcinomas were observed. This result was statistically significant compared with historical controls; no concurrent controls were maintained.

Syrian golden hamsters (126-253/group) were exposed to short and intermediate range chrysotile asbestos at a concentration of 1% in the diet for the lifetime of the animals (NTP, 1983). An increased incidence of neoplasia of the adrenal cortex was observed in both males and females exposed to intermediate range fibers and in males exposed to short range fibers. This increase was statistically significant by comparison to pooled controls but not by comparison to concurrent controls. NTP suggested that the biologic importance of adrenal tumors in the absence of target organ (GI tract) neoplasia was questionable.

Quantitative Estimate Of Carcinogenic Risk From Oral Exposure

Not available.

Quantitative Estimate Of Carcinogenic Risk From Inhalation Exposure

SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk -- 2.3E-1 per (f/mL)

Extrapolation Method -- Additive risk of lung cancer and mesothelioma, using relative risk model for lung cancer and absolute risk model for mesothelioma.

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	4E-4 f/mL
E-5 (1 in 100,000)	4E-5 f/mL

E-6 (1 in 1,000,000) 4E-6 f/mL

Additional Comments (Carcinogenicity, Inhalation Exposure)

Risks have been calculated for males and females according to smoking habits for a variety of exposure scenarios (U.S. EPA, 1986). The unit risk value is calculated for the additive combined risk of lung cancer and mesothelioma, and is calculated as a composite value for males and females. The epidemiological data show that cigarette smoking and asbestos exposure interact synergistically for production of lung cancer and do not interact with regard to mesothelioma. The unit risk value is based on risks calculated using U.S. general population cancer rates and mortality patterns without consideration of smoking habits. The risks associated with occupational exposure were adjusted to continuous exposure by applying a factor of 140 cu.m/50 cu.m based on the assumption of 20 cu.m/day for total ventilation and 10 cu.m/8-hour workday in the occupational setting.

The unit risk is based on fiber counts made by phase contrast microscopy (PCM) and should not be applied directly to measurements made by other analytical techniques. The unit risk uses PCM fibers because the measurements made in the occupational environment use this method. Many environmental monitoring measurements are reported in terms of fiber counts or mass as determined by transmission electron microscopy (TEM). PCM detects only fibers longer than 5 μm and $>0.4 \mu\text{m}$ in diameter, while TEM can detect much smaller fibers. TEM mass units are derived from TEM fiber counts. The correlation between PCM fiber counts and TEM mass measurements is very poor. Six data sets which include both measurements show a conversion between TEM mass and PCM fiber count that range from 5-150 (ug/cu.m)/(f/mL). The geometric mean of these results, 30 (ug/cu.m)/(f/mL), was adopted as a conversion factor (U.S. EPA, 1986), but it should be realized that this value is highly uncertain. Likewise, the correlation between PCM fiber counts and TEM fiber counts is very uncertain and no generally applicable conversion factor exists for these two measurements.

In some cases TEM results are reported as numbers of fibers $<5 \mu\text{m}$ long and of fibers longer than 5 μm . Comparison of PCM fiber counts and TEM counts of fibers $>5 \mu\text{m}$ show that the fraction of fibers detected by TEM that are also $>0.4 \mu\text{m}$ in diameter (and detectable by PCM) varies from 22-53% (U.S. EPA, 1986).

It should be understood that while TEM can be specific for asbestos, PCM is a nonspecific technique and will measure any fibrous material. Measurements by PCM which are made in conditions where other types of fibers may be present may not be reliable.

In addition to the studies cited above, there were three studies of asbestos workers in mining and milling which showed an increase in lung cancer (McDonald et al., 1980, Nicholson et al., 1979; Rubino et al., 1979). The slope factor calculated from these studies was lower than the other studies, possibly because of a substantially different fiber size distribution, and they were not included in the calculation. The slope factor was calculated by life table methods for lung cancer using a relative risk model, and for mesothelioma using an absolute risk model. The final slope factor for lung cancer was calculated as the weighted geometric mean of estimates from the 11 studies cited in section II.C.2. The final slope factor for mesothelioma is based on the calculated values from the studies of Selikoff et al. (1979), Peto et al. (1982), Seidman et al. (1979), Peto (1980) and Finkelstein (1983) adjusted for the mesothelioma incidence from several additional studies cited previously.

There is some evidence which suggests that the different types of asbestos fibers vary in carcinogenic potency relative to one another and site specificity. It appears, for example, that the risk of mesothelioma is greater with exposure to crocidolite than with amosite or chrysotile exposure alone. This evidence is limited by the lack of information on fiber exposure by mineral type. Other data indicates that differences in fiber size distribution and other process differences may contribute at least as much to the observed variation in risk as does the fiber type itself.

The unit risk should not be used if the air concentration exceeds $4E-2$ fibers/ml, since above this concentration the slope factor may differ from that stated.

Discussion Of Confidence (Carcinogenicity, Inhalation Exposure)

A large number of studies of occupationally-exposed workers have conclusively demonstrated the relationship between asbestos exposure and lung cancer or mesothelioma. These results have been corroborated by animal studies using adequate numbers of animals. The

quantitative estimate is limited by uncertainty in the exposure estimates, which results from a lack of data on early exposure in the occupational studies and the uncertainty of conversions between various analytical measurements for asbestos.

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