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LEXINGTON-BLUEGRASS ARMY DEPOT GROUNDWATER INVESTIGATION REPORT PHASE I - FINAL

Volume IV

Lexington-Bluegrass Army Depot
Lexington, Kentucky

Submitted to:

Commander
Department of the Army
United States Army Environmental Center
Aberdeen Proving Ground, Maryland

Submitted by:

Metcalf & Eddy, Inc.
2800 Corporate Exchange Drive
Suite 250
Columbus, Ohio 43231

Prepared Under:

Contract No. DAAA15-90-D-0016
Task Order Number 4

September 1995

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SUMMARY OF SWMU-SPECIFIC SAMPLING RESULTS

This appendix provides the sampling results which serve as the basis for the baseline risk assessment evaluation of each of the two groundwater areas at the LBAD. The tables also provide the frequency of detection, average concentration detected, the range of concentrations detected, the 95 percent UCL, and the exposure concentration associated with each of the chemicals detected in the two areas. In addition, results from the two groundwater background samples are also included.

U.S.EPA guidelines for calculating the 95 percent UCL for chemicals were employed (U.S. EPA, 1992a; 1991b). It should be noted that for small sample sizes, the U.S. EPA recommended procedure for calculating the 95 percent UCL results in extremely high levels. This particular artifact of the statistical methodology was noted during identification of representative site specific concentrations for comparisons of site and background chemical concentrations, selection of chemicals of concern, and determination of exposure point concentrations. For the most part, the maximum or 95 percent UCL, whichever was lower, was employed. In addition, the 95 percent UCL was only calculated for cases where three or more sample results were available for a particular environmental media from each site. If less than three samples were obtained, the maximum chemical concentration was employed for selection of chemicals of concern, and determination of exposure point concentrations. In calculating the 95 percent UCL of the arithmetic mean, an assumption that the data were lognormally distributed was made. Transformation of lognormal data is necessary to compute the UCL of the data. The data were transformed by using the natural logarithm function (i.e., ln(x)).

To calculate the 95 percent UCL of the arithmetic mean for lognormally distributed data, the following steps were followed:

1. transform the data using the natural logarithm (ln(x));
2. calculate the standard deviation of the transformed data;
3. calculate the mean of the transformed data;
4. determine the H-statistic (Gilbert, 1987); and
5. calculate the UCL using this equation:

$$UCL = e^{(\bar{x} + (0-5)(s)^2 + \frac{sH}{\sqrt{n-1}})}$$

where:

e	=	constant (base of natural log)
$\bar{x}$	=	mean of transformed data
s	=	standard deviation of the transformed data
H	=	H - statistic
n	=	number of samples

Summary of Sampling Results from the Northern Portion of LBAD

ANALYTE	A00CMW1009 12/5/91 ug/L	A00CMW1009 9/2/92 ug/L	S001MW1134 12/5/91 ug/L	S001MW2300 8/19/92 ug/L	S001MW23D0 8/19/92 ug/L	S001MW43I0 8/19/92 ug/L	S001MW44I0 8/19/92 ug/L	FREQUENCY OF DETECTION	AVERAGE ug/L
Acetone	<8.0	<8.0	<8.0	<8.0	<8.0	100	<8.0	1/7	17.7
Benzene	1	<1.0	<1.0	<1.0	2.8	1.24	<1.0	3/7	1.01
Chloromethane	4.4	<1.2	<1.2	<1.2	<1.2	<1.2	<1.2	1/7	1.14
1,3-Dimethylbenzene	<1.0	1.3	<1.0	<1.4	<4.3	<1.0	<1.0	1/7	1.29
Toluene	<1.0	<1.0	<1.0	2.6	3.7	3.1	<1.0	3/7	1.63
Aluminum	24300	15900	22900	17000	10800	1060	1980	7/7	13420
Arsenic	NS	3.32	8.78	5.93	<2.35	<2.35	<2.35	3/6	3.59
Barium	84.3	48.3	183	173	570	29.8	27.4	7/7	159
Beryllium	1.65	<1.12	1.73	<1.12	3.1	<1.12	<1.12	3/7	1.25
Boron	NS	389	NS	NS	NS	535	450	3/3	458
Calcium	80400	33600	174000	510000	1700000	139000	55900	7/7	384700
Chromium	34.4	42.5	47.1	<16.8	<16.8	<16.8	<16.8	3/7	22.5
Copper	25.7	<18.8	36.2	28.9	33.7	<18.8	<18.8	4/7	21.8
Iron	37800	16400	53400	29600	17100	1560	1180	7/7	22434
Lead	25.1	19.3	18.6	62.7	30	<4.47	<4.47	5/7	22.9
Magnesium	22700	15500	54600	46200	920000	19300	29700	7/7	158286
Manganese	764	288	1700	935	1330	70.3	34.5	7/7	732
Mercury	<0.1	<0.1	<0.1	<0.1	0.105	<0.1	<0.1	1/7	0.058
Nickel	38.3	<32.1	49.7	41.4	<32.1	<32.1	<32.1	3/7	27.7
Potassium	11600	8700	28200	11900	154000	9260	31100	7/7	36394
Sodium	55000	94000	1100000	110000	9800000	120000	62000	7/7	1.62E+06
Thallium	<125.0	<125.0	189	<125.0	<125.0	<125.0	<125.0	1/7	80.6
Vanadium	42.2	<27.6	36.9	<27.6	<27.6	<27.6	<27.6	2/7	21.2
Zinc	103	<66.7	111	56.1	44.2	30.9	40.7	6/7	64.7

NS – Not Sampled

Summary of Sampling Results from the Northern Portion of LBAD

ANALYTE	RANGE		95% UCL	EXPOSURE CONCENTRATION
	MINIMUM	MAXIMUM		
	ug/L	ug/L	ug/L	ug/L
Acetone	ND	100	114	100
Benzene	ND	2.80	2.19	2.19
Chloromethane	ND	4.40	2.69	2.69
1,3-Dimethylbenzene	ND	1.30	3.84	1.30
Toluene	ND	3.70	7.61	3.70
Aluminum	1060	24300	184642	24300
Arsenic	ND	8.78	19.3	8.78
Barium	27.4	570	1052	570
Beryllium	ND	3.10	3.09	3.09
Boron	389	535	651	535
Calcium	33600	1.70E+06	5.58E+06	1.70E+06
Chromium	ND	47.1	74.9	47.1
Copper	ND	36.2	47.4	36.2
Iron	1180	53400	1.05E+06	53400
Lead	ND	62.7	352	62.7
Magnesium	15500	920000	1.94E+06	920000
Manganese	34.5	1700	30560	1700
Mercury	ND	0.105	0.074	0.074
Nickel	ND	49.7	48.7	48.7
Potassium	8700	154000	168893	154000
Sodium	55000	9.80E+06	2.41E+08	9.80E+06
Thallium	ND	189	119	119
Vanadium	ND	42.2	35.9	35.9
Zinc	ND	111	105	105

ND – Not Determined

Summary of Sampling Results in the Southern Portion of LBAD

ANALYTE	A00BMW1123 12/6/91 ug/L	A00BMW1123 9/3/92 ug/L	B004MW4700 9/9/93 ug/L	B004MW47D0 9/9/93 ug/L	B004FD47D0* 9/9/93 ug/L	B018MW4800 9/21/93 ug/L	B018MW48D0 9/21/93 ug/L	S003MW1052 12/6/91 ug/L	S003FD1052* 12/6/91 ug/L
Acetone	<8.0	<8.0	<8.0	<8.0	<8.0	100	240	<8.0	<8.0
Benzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-ethylhexyl)phthalate	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	13	<7.7
Carbon tetrachloride	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
1,1-Dichloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
1,2-Dichloroethenes	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
2,4-Dimethylphenol	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4
1,3-Dimethylbenzene	<1.0	2.2	16	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
alpha-Endosulfan	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
Ethylbenzene	<1.0	<1.0	7.7	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Methyl isobutyl ketone	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4
Phenol	<2.2	17	<2.2	<2.2	<2.2	<2.2	<2.2	5.5	<2.2
Tetrachloroethene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Toluene	<1.0	<1.0	33	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Trichloroethene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Vinyl chloride	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0
Xylenes	<2.0	<2.0	24	<2.0	<2.0	<2.0	<2.0	<2.0	<2.0
alpha-BHC	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	0.003	<0.003
delta-BHC	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
DDT	0.008	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
Lindane	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
Aluminum	6450	65100	NS	NS	NS	NS	NS	34400	52400
Antimony	73.2	<60.0	NS	NS	NS	NS	<60.0	<60.0	<60.0
Arsenic	NS	10.9	NS	NS	NS	NS	NS	NS	NS
Barium	62.4	307	NS	NS	NS	NS	NS	327	421
Beryllium	<1.12	3.02	NS	NS	NS	NS	<1.12	2.86	4
Boron	NS	305	NS	NS	NS	NS	NS	NS	NS
Cadmium	<6.78	<6.78	NS	NS	NS	NS	<6.78	343	417
Calcium	162000	210000	NS	NS	NS	NS	NS	240000	280000
Chromium	71	139	NS	NS	NS	NS	NS	173	244
Cobalt	<25.0	35.9	NS	NS	NS	NS	<25.0	28	43.5
Copper	<18.8	55.5	NS	NS	NS	NS	<18.8	156	213
Iron	17600	84000	NS	NS	NS	NS	NS	152000	229000
Lead	11.6	95.4	NS	NS	NS	NS	NS	27.1	32.1
Magnesium	21000	40600	NS	NS	NS	NS	NS	55300	63400
Manganese	4740	5120	NS	NS	NS	NS	NS	6670	8750
Mercury	<0.1	<0.1	NS	NS	NS	NS	<0.1	<0.1	<0.1
Molybdenum	NS	<52.7	NS	NS	NS	NS	NS	NS	NS
Nickel	<32.1	10.9	NS	NS	NS	NS	<32.1	111	136
Potassium	4310	26600	NS	NS	NS	NS	NS	12000	14300
Sodium	10100	17400	NS	NS	NS	NS	NS	40100	40200
Tellurium	NS	<118.0	NS	NS	NS	NS	NS	NS	NS
Thallium	<125.0	<125.0	NS	NS	NS	NS	<125.0	<125.0	266
Tin	NS	<59.9	NS	NS	NS	NS	NS	NS	NS
Vanadium	<27.6	85.4	NS	NS	NS	NS	<27.6	53.8	78.3
Zinc	48.4	244	NS	NS	NS	NS	NS	386	533

NS -- Not Sampled

*Duplicate

Summary of Sampling Results in the Southern Portion of LBAD

ANALYTE	S003MW1052 9/2/92 ug/L	S003FD1052* 9/2/92 ug/L	S003MW4000 8/20/92 ug/L	S003FD4000* 8/20/92 ug/L	S003MW40D0 8/21/92 ug/L	S003FD40D0* 8/21/92 ug/L	S003MW0600 12/11/91 ug/L	S003MW6D00 8/21/92 ug/L	S003MW1051 11/24/91 ug/L
Acetone	<8.0	<8.0	<8.0	<8.0	<8.0	<8.0	52	100	52
Benzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	33	<1.0
Bis(2-ethylhexyl)phthalate	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7
Carbon tetrachloride	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
1,1-Dichloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
1,2-Dichloroethenes	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
2,4-Dimethylphenol	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4	13	<4.4
1,3-Dimethylbenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	1.8	14	<1.0
alpha-Endosulfan	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
Ethylbenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	1.1	6.8	<1.0
Methyl isobutyl ketone	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4
Phenol	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2
Tetrachloroethene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Toluene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	3.7	69	<1.0
Trichloroethene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Vinyl chloride	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0
Xylenes	<2.0	<2.0	<2.0	<2.0	<2.0	<2.0	3.1	110	<2.0
alpha-BHC	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
delta-BHC	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
DDT	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	0.011	<0.003	<0.003
Lindane	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	0.005	<0.003	<0.003
Aluminum	3840	9150	666	746	2520	1620	2470	87600	3500
Antimony	<60.0	<60.0	<60.0	<60.0	<60.0	<60.0	<60.0	<60.0	<60.0
Arsenic	2.9	3.89	<2.35	<2.35	<2.35	<2.35	<2.35	<2.35	<2.35
Barium	83.7	143	266	205	106	105	99.9	911	161
Beryllium	<1.12	<1.12	<1.12	<1.12	<1.12	<1.12	<1.12	36.5	<1.12
Boron	257	274	557	393	1110	1180	NS	1930	NS
Cadmium	147	291	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78	7.22
Calcium	153000	160000	115000	93200	131000	115000	165000	6300000	118000
Chromium	41.8	119	<16.8	<16.8	<16.8	<16.8	28.7	273	18.1
Cobalt	<25.0	<25.0	<25.0	<25.0	<25.0	<25.0	<25.0	26.1	<25.0
Copper	<18.8	33.9	<18.8	<18.8	<18.8	<18.8	<18.8	77.6	<18.8
Iron	18600	47100	1680	1260	4080	2580	3200	111000	8940
Lead	6.71	14.6	<4.47	<4.47	<4.47	<4.47	<4.47	470	5.55
Magnesium	30800	32700	37500	28900	43000	46600	54200	750000	31200
Manganese	1610	4000	157	142	292	201	306	17000	342
Mercury	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	0.174	<0.1
Molybdenum	<52.7	<52.7	<52.7	<52.7	<52.7	<52.7	NS	<52.7	NS
Nickel	39.7	79.6	<32.1	<32.1	<32.1	<32.1	<32.1	152	<32.1
Potassium	3610	5350	5680	5410	10900	12000	6160	122000	4940
Sodium	31200	31600	48900	37800	190000	220000	48900	4900000	23200
Tellurium	<118.0	<118.0	<118.0	<118.0	<118.0	<118.0	NS	<118	NS
Thallium	<125.0	<125.0	<125.0	<125.0	<125.0	<125.0	<125.0	<125.0	<125.0
Tin	<59.9	<59.9	<59.9	<59.9	<59.9	<59.9	NS	94.9	NS
Vanadium	<27.6	<27.6	<27.6	<27.6	<27.6	<27.6	<27.6	523	<27.6
Zinc	110	161	34.9	27	58.3	72.3	29.5	1850	36.2

NS – Not Sampled *Duplicate

Summary of Sampling Results in the Southern Portion of LBAD

ANALYTE	S003MW1051 9/1/92 ug/L	S003MW1053 11/24/91 ug/L	S003MW1600 12/10/91 ug/L	S003MW1600 9/2/92 ug/L	S003MW3900 8/21/92 ug/L	S003MW4100 8/19/92 ug/L	S004MW0500 12/9/91 ug/L	S004FD0500* 12/9/91 ug/L	S004MW1900 5/26/92 ug/L
Acetone	<8.0	<8.0	<8.0	<8.0	100	<8.0	<8.0	<8.0	<8.0
Benzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Bis(2-ethylhexyl)phthalate	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7
Carbon tetrachloride	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
1,1-Dichloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
1,2-Dichloroethenes	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
2,4-Dimethylphenol	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4
1,3-Dimethylbenzene	<1.0	<1.0	<1.0	<1.0	1.6	<1.0	<1.0	<1.0	<1.0
alpha-Endosulfan	<0.003	<0.003	0.5	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
Ethylbenzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Methyl isobutyl ketone	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4
Phenol	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2
Tetrachloroethene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Toluene	<1.0	<1.0	1.7	<1.0	2.7	<1.0	<1.0	<1.0	1.1
Trichloroethene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	1.1
Vinyl chloride	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0
Xylenes	<2.0	<2.0	<2.0	<2.0	2.3	<2.0	<2.0	<2.0	<2.0
alpha-BHC	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
delta-BHC	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
DDT	<0.003	"U"	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
Lindane	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
Aluminum	<112.0	143	2390	262	94800	605	<112.0	326	1220
Antimony	<60.0	<60.0	<60.0	<60.0	<60.0	<60.0	<60.0	<60.0	<60.0
Arsenic	<2.35	<2.35	<2.35	<2.35	25	<2.35	<2.35	<2.35	<2.35
Barium	145	168	262	74	849	38.5	<2.82	74.7	53.9
Beryllium	<1.12	<1.12	<1.12	<1.12	36.5	<1.12	<1.12	<1.12	<1.12
Boron	388	NS	NS	271	2420	408	NS	NS	<230
Cadmium	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78
Calcium	95300	93900	141000	107000	5000000	88200	<105.0	102000	86400
Chromium	<16.8	<16.8	<16.8	<16.8	210	<16.8	<16.8	<16.8	<16.8
Cobalt	<25.0	<25.0	<25.0	<25.0	69	<25.0	<25.0	<25.0	<25.0
Copper	<18.8	<18.8	<18.8	<18.8	66.2	<18.8	<18.8	23.1	<18.8
Iron	624	1020	19300	1570	135000	793	<77.5	2180	3060
Lead	<4.47	<4.47	9.32	<4.47	180	<4.47	<4.47	<4.47	<4.47
Magnesium	29600	36200	35000	32400	830000	9930	<135.0	13600	13900
Manganese	150	42.3	377	141	14000	1080	<9.67	193	252
Mercury	<0.1	<0.1	<0.1	<0.1	0.299	<0.1	<0.1	0.116	<0.1
Molybdenum	<52.7	NS	NS	<52.7	<52.7	<52.7	NS	NS	<52.7
Nickel	<32.1	<32.1	<32.1	<32.1	169	<32.1	<32.1	<32.1	<32.1
Potassium	3520	4280	3090	4010	125000	2530	<1240.0	1610	<1240.0
Sodium	21800	32900	19600	40800	5600000	15800	<279.0	13000	12800
Tellurium	<118.0	NS	NS	<118.0	<118.0	<118.0	NS	NS	<118.0
Thallium	<125.0	<125.0	196	<125.0	<125.0	<125.0	<125.0	<125.0	<125.0
Tin	<59.9	NS	NS	<59.9	102	<59.9	NS	NS	<59.9
Vanadium	<27.6	<27.6	<27.6	<27.6	523	<27.6	<27.6	<27.6	<27.6
Zinc	<18.0	<18.0	24.2	<18.0	1470	43.1	<18.0	32.2	29.2

NS – Not Sampled

*Duplicate

Summary of Sampling Results in the Southern Portion of LBAD

ANALYTE	S004FD1900* 5/26/92 ug/L	S004MW19DD 9/10/92 ug/L	S004MW4200 8/19/92 ug/L	S004FD4200* 8/19/92 ug/L	S004MW0200 12/10/91 ug/L	S004MW0300 12/10/91 ug/L	S004MW0400 12/4/91 ug/L	S004MW0400 9/2/92 ug/L	S004MW4700 8/21/92 ug/L
Acetone	<8.0	<8.0	<8.0	<8.0	<8.0	<8.0	<8.0	<8.0	100
Benzene	<1.0	0.78	<1.0	<1.0	1.9	<1.0	<1.0	<1.0	4.8
Bis(2-ethylhexyl)phthalate	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7
Carbon tetrachloride	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
1,1-Dichloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
1,2-Dichloroethenes	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
2,4-Dimethylphenol	<4.4	20	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4
1,3-Dimethylbenzene	<1.0	<1.0	<1.0	<1.11	3.7	<1.0	<1.0	<1.0	14
alpha-Endosulfan	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
Ethylbenzene	<1.0	<1.0	<1.0	<1.0	2.1	<1.0	<1.0	3.9	7.2
Methyl isobutyl ketone	<1.4	5.3	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4
Phenol	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2
Tetrachloroethene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Toluene	1.3	<1.0	<1.0	<1.0	11	<1.0	<1.0	<1.0	37
Trichloroethene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Vinyl chloride	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0
Xylenes	<2.0	42	<2.0	<2.0	3.4	<2.0	<2.0	<2.0	13
alpha-BHC	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
delta-BHC	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
DDT	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
Lindane	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
Aluminum	1560	350000	285	322	992	1360	22900	2710	44200
Antimony	<60.0	217	<60.0	<60.0	<60.0	<60.0	63	<60.0	<60.0
Arsenic	<2.35	<2.35	<2.35	<2.35	3.89	<2.35	10.8	<2.35	<2.35
Barium	59.7	1870	72.6	76.2	78.7	69.3	300	189	194
Beryllium	<1.12	18.2	<1.12	<1.12	<1.12	<1.12	1.66	<1.12	1.1
Boron	<230	<230.0	<230.0	<230.0	NS	NS	NS	<230.0	367
Cadmium	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78
Calcium	89300	17000000	78200	81100	130000	60800	172000	89700	26600
Chromium	<16.8	448	<16.8	<16.8	<16.8	34.8	32.8	<16.8	41.6
Cobalt	<25.0	186	<25.0	<25.0	<25.0	<25.0	32.6	<25.0	<25.0
Copper	<18.8	257	<18.8	<18.8	24.2	30.3	41.1	<18.8	57.2
Iron	3600	342000	2380	2590	3590	3070	51200	5560	39100
Lead	7.58	<4.47	<4.47	<4.47	21.3	9.12	101	9.65	106
Magnesium	14300	980000	11600	12000	36600	9870	18400	13000	8440
Manganese	270	65000	124	129	162	74.2	1000	361	324
Mercury	<0.1	1.54	<0.1	<0.1	<0.1	0.127	<0.1	<0.1	0.101
Molybdenum	<52.7	58.8	<52.7	<52.7	NS	NS	NS	<52.7	<52.7
Nickel	<32.1	427	<32.1	<32.1	<32.1	55.6	<32.1	<32.1	59.3
Potassium	1660	83500	1830	1780	4860	1690	5310	2700	7630
Sodium	13300	5300000	20300	21100	47000	89000	27800	70000	180000
Tellurium	<118.0	211	<118.0	<118.0	NS	NS	NS	<118.0	<118.0
Thallium	<125.0	437	<125.0	<125.0	<125.0	<125.0	<125.0	<125.0	<125.0
Tin	<59.9	62.9	<59.9	<59.9	NS	NS	NS	<59.9	<59.9
Vanadium	<27.6	237	<27.6	<27.6	<27.6	<27.6	39.8	<27.6	34.1
Zinc	21.6	1140	<18.0	<18.0	25.6	32.3	119	<35.1	154

NS – Not Sampled

*Duplicate

Summary of Sampling Results in the Southern Portion of LBAD

ANALYTE	S2567MW080 12/16/91 ug/L	S2567MW8D0 8/22/92 ug/L	S2567MW090 12/17/91 ug/L	S2567MW122 12/6/91 ug/L	S2567MW124 12/11/91 ug/L	S2567MW180 12/17/91 ug/L	S2567MW180 8/22/92 ug/L	S2567MW18D 8/19/92 ug/L	S2567FD18D* 8/19/92 ug/L
Acetone	<8.0	<8.0	<8.0	<8.0	<8.0	100	<8.0	<8.0	<8.0
Benzene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	1.9	1.5
Bis(2-ethylhexyl)phthalate	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7
Carbon tetrachloride	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
1,1-Dichloroethane	9	22	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
1,2-Dichloroethenes	<5.0	34	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
2,4-Dimethylphenol	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4	14.8	20.5
1,3-Dimethylbenzene	<1.0	<1.0	1.1	<1.0	<1.0	<1.0	<1.0	<16	<13
alpha-Endosulfan	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
Ethylbenzene	<1.0	<1.0	2.2	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Methyl isobutyl ketone	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4
Phenol	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2
Tetrachloroethene	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Toluene	1.7	<1.0	4.7	<1.0	<1.0	<1.0	<1.0	4.4	3.5
Trichloroethene	<1.0	6.6	<1.0	<1.0	1.3	<1.0	<1.0	<1.0	<1.0
Vinyl chloride	<12.0	<12.0	<12.0	<12.0	<12.0	17	<12.0	<12.0	<12.0
Xylenes	2.6	<2.0	4.4	<2.0	<2.0	<2.0	<2.0	<2.0	<2.0
alpha-BHC	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
delta-BHC	<0.003	<0.003	<0.003	<0.003	<0.003	0.015	<0.003	<0.003	<0.003
DDT	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	"U"
Lindane	0.006	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
Aluminum	1730	512	952	3000	4340	2870	279	187	258
Antimony	<60.0	<60.0	<60.0	<60.0	<60.0	<60.0	<60.0	<60.0	<60.0
Arsenic	<2.35	<2.35	<2.35	NS	<2.35	<2.35	<2.35	<2.35	<2.35
Barium	184	174	307	350	113	235	213	449	464
Beryllium	<1.12	<1.12	<1.12	<1.12	<1.12	<1.12	<1.12	<1.12	110
Boron	NS	613	NS	NS	NS	NS	471	NS	NS
Cadmium	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78
Calcium	143000	169000	82600	88000	123000	115000	99500	670000	760000
Chromium	36.4	<16.8	<16.8	<16.8	<16.8	<16.8	<16.8	<16.8	<16.8
Cobalt	<25.0	<25.0	<25.0	<25.0	<25.0	<25.0	<25.0	<25.0	<25.0
Copper	<18.8	<18.8	<18.8	<18.8	<18.8	<18.8	<18.8	<18.8	<18.8
Iron	2190	7910	1240	9170	5580	1600	146	335	414
Lead	<4.47	<4.47	<4.47	<4.47	12	15.3	6.38	<4.47	<4.47
Magnesium	18900	20000	36700	17400	12800	35400	33400	490000	550000
Manganese	425	626	137	727	1680	256	235	99.7	102
Mercury	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1
Molybdenum	NS	<52.7	NS	NS	NS	NS	<52.7	NS	NS
Nickel	<32.1	<32.1	<32.1	<32.1	<32.1	<32.1	<32.1	<32.1	<32.1
Potassium	3210	2470	5960	3050	2510	4500	11200	74200	75900
Sodium	41200	38400	36100	13600	17100	44500	43100	4700000	5300000
Tellurium	NS	<118.0	NS	NS	NS	NS	<118.0	NS	NS
Thallium	<125.0	<125.0	<125.0	<125.0	<125.0	<125.0	<125.0	<125.0	<125.0
Tin	NS	<59.9	NS	NS	NS	NS	<59.9	NS	NS
Vanadium	<27.6	<27.6	<27.6	<27.6	<27.6	<27.6	<27.6	<27.6	<27.6
Zinc	19.1	39.4	20.9	39.5	19.2	34.9	110	1900	<18.0

NS – Not Sampled

*Duplicate

Summary of Sampling Results in the Southern Portion of LBAD

ANALYTE	S2567MW320 8/19/92 ug/L	S2567MW32D 8/21/92 ug/L	S2567MW330 8/19/92 ug/L	S2567FD330* 8/19/92 ug/L	S2567MW450 8/21/92 ug/L	S2567MW460 8/22/92 ug/L	W008WS0176 11/22/91 ug/L	W01WS01150 11/5/91 ug/L	W09WS01150 11/6/91 ug/L
Acetone	<8.0	100	<8.0	<8.0	<8.0	<8.0	<8.0	<8.0	<8.0
Benzene	<1.0	6.9	<1.0	0.93	<1.0	<1.0	0.85	<1	<1
Bis(2-ethylhexyl)phthalate	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7	<7.7
Carbon tetrachloride	1.1	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
1,1-Dichloroethane	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
1,2-Dichloroethenes	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0	<5.0
2,4-Dimethylphenol	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4	<4.4
1,3-Dimethylbenzene	<1.0	5.8	<4	<6.8	<1.0	<1.0	<1.0	<1.0	<1.0
alpha-Endosulfan	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
Ethylbenzene	<1.0	7.2	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Methyl isobutyl ketone	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4	<1.4
Phenol	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2	<2.2
Tetrachloroethene	1.1	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Toluene	<1.0	36	2.7	5.9	<1.0	<1.0	<1.0	<1.0	<1.0
Trichloroethene	1.2	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0	<1.0
Vinyl chloride	150	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0	<12.0
Xylenes	<2.0	38	<4.2	<6.3	<2.0	<2.0	<2.0	<2.0	<2.0
alpha-BHC	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<5.3	<0.003	<0.003
delta-BHC	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<3R	<0.003	<0.003
DDT	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	0.017	<0.003
Lindane	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003	<0.003
Aluminum	9040	270000	4400	2730	171	272	<112	2400	119
Antimony	<60.0	<60.0	<60.0	<60.0	<60.0	<60.0	<60	<60	<60
Arsenic	3.39	<2.35	<2.35	<2.35	<2.35	<2.35	<2.35	4.52	<2.35
Barium	807	1330	229	214	177	207	37.3	196	138
Beryllium	<1.12	28.3	2.36	<1.12	<1.12	<1.12	<1.12	<1.12	<1.12
Boron	<230.0	712	NS	NS	276	259	NS	NS	NS
Cadmium	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78	<6.78
Calcium	270000	17000000	190000	165000	79600	74400	116000	103000	77000
Chromium	27	119	<16.8	<16.8	<16.8	<16.8	<16.8	<16.8	<16.8
Cobalt	<25.0	211	<25.0	<25.0	<25.0	<25.0	<25.0	<25.0	<25.0
Copper	<18.8	237	<18.8	<18.8	<18.8	<18.8	<18.8	<18.8	<18.8
Iron	18100	36400	8630	6660	132	235	<77.5	5520	107
Lead	18.2	<4.47	25.8	17.1	<4.47	<4.47	<4.47	8.3	<4.47
Magnesium	54200	640000	43300	40400	24200	20100	15300	19300	23800
Manganese	1240	58000	418	337	53.2	50.6	143	666	647
Mercury	<0.1	0.76	<0.1	<0.1	<0.1	<0.1	<0.1	0.119	<0.1
Molybdenum	<52.7	<52.7	NS	NS	<52.7	<52.7	NS	NS	NS
Nickel	34.6	499	<32.1	<32.1	<32.1	<32.1	<32.1	<32.1	<32.1
Potassium	22800	48500	10200	6620	6310	3570	3440	2080	3390
Sodium	48300	1200000	49800	46800	29300	820000	25400	22100	34900
Tellurium	<118.0	<118.0	NS	NS	<118.0	<118.0	NS	NS	NS
Thallium	<125.0	<125.0	<125.0	<125.0	<125.0	<125.0	<125.0	<125.0	<125.0
Tin	<59.9	76.1	NS	NS	<59.9	<59.9	NS	NS	NS
Vanadium	31.2	208	<27.6	<27.6	<27.6	<27.6	<27.6	<27.6	<27.6
Zinc	89.8	1350	63.9	28.8	77	134	<18	123	<18

NS – Not Sampled

*Duplicate

Summary of Sampling Results in the Southern Portion of LBAD

ANALYTE	FREQUENCY OF DETECTION	AVERAGE ug/L	RANGE		95% UCL ug/L	EXPOSURE CONCENTRATION ug/L
			MINIMUM ug/L	MAXIMUM ug/L		
Acetone	9/44	24.6	ND	240	30.9	30.9
Benzene	8/44	1.57	ND	33.0	1.27	1.27
Bis(2-ethylhexyl)phthalate	1/44	4.06	ND	13.0	4.22	4.22
Carbon tetrachloride	1/44	0.514	ND	1.10	0.529	0.529
1,1-Dichloroethane	2/44	1.18	ND	22.0	0.94	0.94
1,2-Dichloroethenes	1/44	3.22	ND	34.0	3.20	3.20
2,4-Dimethylphenol	3/44	3.27	ND	20.5	3.41	3.41
1,3-Dimethylbenzene	9/44	2.26	ND	16.0	2.55	2.55
alpha-Endosulfan	1/44	0.013	ND	0.500	0.003	0.003
Ethylbenzene	8/44	1.28	ND	7.70	1.40	1.40
Methyl isobutyl ketone	1/44	0.805	ND	5.30	0.834	0.834
Phenol	2/44	1.56	ND	17.0	1.56	1.56
Tetrachloroethene	1/44	0.514	ND	1.10	0.529	0.529
Toluene	13/44	5.18	ND	69.0	5.68	5.68
Trichloroethene	4/44	0.698	ND	6.60	0.718	0.718
Vinyl chloride	2/44	9.52	ND	150	8.70	8.70
Xylenes	9/44	6.41	ND	110	5.80	5.80
alpha-BHC	1/44	0.062	ND	0.003	0.005	0.003
delta-BHC	1/42	0.002	ND	0.015	0.002	0.002
DDT	3/42	0.095	ND	0.017	0.008	0.008
Lindane	2/44	0.0017	ND	0.006	0.002	0.002
Aluminum	37/40	26324	ND	350000	124704	124704
Antimony	3/41	36.4	ND	217	38.5	38.5
Arsenic	5/37	2.6	ND	25.0	2.88	2.88
Barium	39/40	302	ND	1870	398	398
Beryllium	10/41	6.32	ND	110	5.77	5.77
Boron	15/28	524	ND	2420	909	909
Cadmium	3/41	21	ND	417	10.20	10.20
Calcium	39/40	1.26E+06	ND	1.70E+07	1.02E+06	1.02E+06
Chromium	15/39	51.3	ND	448	68.4	68.4
Cobalt	7/41	25.1	ND	211	25.6	25.6
Copper	13/41	34	ND	257	37.9	37.9
Iron	38/40	30380	ND	342000	176723	176723
Lead	18/40	30	ND	470	41.9	41.9
Magnesium	39/40	118034	ND	980000	137654	137654
Manganese	39/40	4735	ND	65000	8461	8461
Mercury	8/41	0.119	ND	1.54	0.116	0.116
Molybdenum	1/20	26.6	ND	58.8	30.0	30.0
Nickel	10/42	51	ND	499	54.7	54.7
Potassium	39/40	16591	1610	125000	21554	21554
Sodium	38/40	614905	ND	5.60E+06	823095	823095
Tellurium	1/19	63.4	ND	211	73.8	73.8
Thallium	3/41	74.9	ND	437	79.1	79.1
Tin	4/20	38.8	ND	102	48.5	48.5
Vanadium	9/41	53.7	ND	523	53.4	53.4
Zinc	32/40	255	ND	1900	437	437

NS – Not Sampled

ND – Not Determined

Summary of Chemical Concentrations Detected in Upgradient Wells at LBAD

ANALYTE	FACLMW0700 12/4/91 ug/L	S2567MW070 12/12/91 ug/L	FREQUENCY OF DETECTION	AVERAGE ug/L	RANGE MINIMUM ug/L	RANGE MAXIMUM ug/L	Average X 2 ug/L
DDT	0.026	<0.003	1/2	0.014	ND	0.026	0.0275
Aluminum	4520	3310	2/2	3915	3310	4520	7830
Arsenic	2.35	<2.35	1/2	1.763	ND	2.35	3.525
Barium	221	211	2/2	216	211	221	432
Beryllium	1.11	<1.12	1/2	0.835	ND	1.11	1.67
Calcium	89300	82000	2/2	85650	82000	89300	171300
Copper	<18.8	100	1/2	54.7	ND	100	109.4
Iron	4250	1900	2/2	3075	1900	4250	6150
Lead	13.2	5.91	2/2	9.56	5.91	13.2	19.11
Magnesium	28100	27500	2/2	27800	27500	28100	55600
Manganese	96.7	72.6	2/2	84.65	72.6	96.7	169.3
Potassium	7080	5900	2/2	6490	5900	7080	12980
Sodium	57000	63000	2/2	60000	57000	63000	120000
Zinc	138	50.6	2/2	94.3	50.6	138	188.6

ND – Not Determined

APPENDIX L

METHODS FOR ESTIMATING INTAKES FOR CHEMICALS OF POTENTIAL CONCERN

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ATTACHMENT 1 Groundwater Air Inhalation Model

METHODS FOR ESTIMATING INTAKE LEVELS

The methods utilized for determining intake levels of the study chemicals are described in this Appendix. The following groundwater exposure pathways are addressed:

- ingestion of groundwater;
- dermal contact with groundwater;
- inhalation of volatiles while showering;

All other (non-groundwater) applicable exposure pathways were evaluated in the previous RFI. In all cases, conservative assumptions are employed in order to estimate the reasonable maximum exposures to potential receptor populations.

The parameter values used in calculating intakes are based primarily on USEPA guidance (USEPA 1989a,c, and USEPA 1991a, unless otherwise noted in the exposure equation tables). Exposure is averaged over 70 years (25,550 days) for carcinogenic effects. For non-carcinogenic effects, exposure is averaged over the product of the exposure duration (in years) times 365 days per year. Chronic exposures are assumed to occur over a period of at least seven years. Subchronic exposures are defined as exposures which occur over a period of up to seven years. Average body weights of 70 kg for an adult and 16 kg for child, are assumed for all of the exposure calculations (USEPA, 1991, and USEPA, 1989a,c). [These body weights are derived from the average body weight for adult men and women (USEPA, 1989)].

This appendix provides a detailed accounting of the equations and corresponding parameter assumptions for the existing and future exposure scenarios described in Section 3 of this report. The descriptions of the methods for quantifying intake are provided below. The receptor populations, activities which result in exposure, exposure frequencies, exposure durations, and exposure events are based on the receptor populations and corresponding exposure pathways described in Section 3.

Ingestion of Chemicals in Drinking Water

Ingestion of chemicals in drinking water assumes a residential scenario in which a home is constructed on or near the areas of concern and a drinking water well is installed and used as the domestic water

supply source. The equation and parameter values used for determining intakes under this scenario are presented in Table L-1.

Dermal Contact While Bathing

Dermal contact with the chemicals of concern is investigated in light of the fact that use of groundwater by a residential receptor could result in exposure via this route. The equation and parameter values used for estimating exposure levels from dermal contact with groundwater are presented in Table L-2. This equation results in the estimation of an absorbed dose. It is assumed that the entire body surface will come in contact with the water. Total body surface areas of 19,400 cm^2 and 7,280 cm^2 are assumed for adults and children, respectively (50th percentile data for adult males and 3-6 year old male children, respectively; U.S. EPA, 1989b). The exposure time is based on an average shower duration of 10 minutes, assuming that a resident takes at least one shower every day at the residence. Permeability constants (PC) values are based on chemical-specific values provided in U.S. EPA guidance for dermal exposure assessment (U.S. EPA, 1992c).

It should be noted that the intake values resulting from the equation in Table L-2 are absorbed doses. For the computation of hazards quotients and risk estimates, these absorbed doses must be compared to absorbed RfDs and cancer slope factors. This adjustment was made in calculating risks.

Chemical-specific permeability constants (PCs) are utilized as recommended in the U.S. EPA guidance for Dermal Exposure Assessment Table 5-7 (U.S. EPA, 1992c). Dermal PCs are identified in Table 1 of Appendix N.

Inhalation of Vapors While Bathing

Receptors may be exposed to chemicals [volatile organic compounds (VOCs)] while showering with groundwater from a residential well. Concentrations of VOCs in ambient air during time spent in the room during and after showering are examined assuming receptor exposure through use of groundwater as the source of shower water. The equation and parameter values used for determining intakes under this scenario are presented in Table L-3.

Because actual sampling data were not available to characterize chemical concentrations in air resulting from showering, the chemical levels were estimated using a model recommended by the U.S. EPA's

Environmental Criteria and Assessment Office (ECAO). The general model description is provided in Attachment 1. In this model, inhalation exposures are modeled by estimating the quantity of airborne volatile organic chemicals during the showering time and during the time period after showering where a decrease in the air concentrations would be expected. Based on the air model, ambient air concentrations for the chemicals of concern were derived. The following parameter values were assumed: a bathroom volume of 9.1 m^3, a combined showering, and after showering period of 0.37 hours. The water flow rate was assumed to be 0.6 m^3/hour. The fraction of volatilization was set at 75 percent. All of these parameter values were based on the mean or typical values recommended in the model description. The results of the showering model for the northern and southern portions of LBAD are presented in Tables L-4 and L-5.

TABLE L.1
Ingestion of Ground Water
[Equation and Parameter Values Derived from U.S.EPA, 1989a, b and U.S. EPA 1991a, Unless Otherwise Noted]

$$Intake\ (mg/kgday) = \frac{CW \times IR \times EF \times ED}{BW \times AT}$$

VARIABLE	VARIABLE DESCRIPTION	VARIABLE ASSUMPTIONS
CW	Chemical Concentration in Water (mg/liter)	Chemical Specific (mg/liter)
IR	Ingestion Rate (liters/day)	2 liters/day (adult - resident) 1 liter /day (child) 1 liter/day (adult - worker)
EF	Exposure Frequency (days/year)	350 days/year (residential) 250 days/year (occupational)
ED	Exposure Duration (years)	30 years for adults (chronic and carcinogenic effects - residential) 7 years for adults (subchronic effects - residential and workers) 7 years for child (subchronic and carcinogenic effects)
BW	Body Weight (kg)	70 kg (adult) 16 kg (child)
AT	Averaging Time (period over which exposure is averaged - days)	25,550 days (carcinogenic effects) ED x 365 days/year (chronic noncarcinogenic and subchronic effects

TABLE L.2
Dermal Contact with Ground Water
While Showering
[Equation and Parameter Values Derived from U.S.EPA, 1989a, b and U.S. EPA 1991a, Unless Otherwise Noted]

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

VARIABLE	VARIABLE DESCRIPTION	VARIABLE ASSUMPTIONS
CW	Chemical Concentration in Water (mg/liter)	Chemical Specific (mg/liter)
SA	Skin Surface Available for Contact (cm)	19,400 cm^2 (adult) 7,280 cm^2 (child)
PC	Chemical Specific Dermal Permeability Constant (cm/hr)	Chemical Specific (cm/hr)
ET	Exposure Time	0.08 hours/day
EF	Exposure Frequency (days/year)	350 days/year
ED	Exposure Duration (years)	30 years for adults (chronic and carcinogenic effects - residential) 7 years for adults (subchronic effects - residential and workers) 7 years for child (subchronic and carcinogenic effects)
CF	Volumetric Conversion Factor for Water (1 liter/1,000 cm)	1 liter/1,000 cm^3
BW	Body Weight (kg)	70 kg (adult) 16 kg (child)
AT	Averaging Time (period over which exposure is averaged - days)	25,550 days (carcinogenic effects) ED x 365 days/year (chronic noncarcinogenic and subchronic effects

TABLE L.3

Inhalation of Vapors from Groundwater While Showering

[Equation and Parameter Values Derived from U.S.EPA, 1989a, b and U.S. EPA 1991a, Unless Otherwise Noted; Ei = explained in Attachment A]

$$Intake\ (mg/kg\ day) = \frac{Ei\ x\ SF\ x\ EF\ x\ ED}{BW\ x\ AT}$$

VARIABLE	VARIABLE DESCRIPTION	VARIABLE ASSUMPTIONS
Ei*	Exposure	Chemical Specific (mg)
SF	Shower Frequency	1 shower/day
EF	Exposure Frequency (days/year)	350 days/year
ED	Exposure Duration	30 years for adults (chronic and carcinogenic effects - residential) 7 years for adults (subchronic effects - residential and workers) 7 years for child (subchronic and carcinogenic effects)
BW	Body Weight (kg)	70 kg (adult) 16 kg (child)
AT	Averaging Time (period over which exposure is averaged - days)	25,550 days (carcinogenic effects) ED x 365 days/year (chronic noncarcinogenic and subchronic effects)

* Refer to Attachment 1

TABLE L–4 **AMBIENT AIR CONCENTRATIONS OF VOLATILE ORGANIC CHEMICALS – NORTHERN PORTION OF LBAD**

Cmax(Ca2) = Cw x f x Fw x t / Va
Ei = [Ca1 X Bt1] + [Ca2 X Bt2]
B = 833 L/hr
t2 = 0.2 hr

where:
Cmax = maximum air concentration in bathroom (mg/L)
Cw = water concentration (mg/L)
f = fraction volatilization (unitless)
Fw = water flow rate (m3/hr)
t1 = shower period (hr)
Va = bathroom size (m3)
Ei = Exposure concentration (mg)
B = Breathing rate
t2 = after shower period

ANALYTE	GROUNDWATER CONC. (mg/l) Cw	VOLATILIZATION FACTOR (unitless) f	SHOWER PERIOD (hr) t1	WATER FLOW RATE (m3/hr) Fw	BATHROOM VOLUME (m3) Va	AIR CONCENTRATION (mg/l) Ca2	Ca1	EXPOSURE CONC. (mg) Ei
Acetone	0.100	0.75	0.17	0.6	9.1	8.41E–04	4.20E–04	0.168
Benzene	0.002	0.75	0.17	0.6	9.1	1.84E–05	9.19E–06	0.004
Chloromethane	0.003	0.75	0.17	0.6	9.1	2.26E–05	1.13E–05	0.005
1,3–Dimethylbenzene	0.001	0.75	0.17	0.6	9.1	1.09E–05	5.46E–06	0.002
Toluene	0.004	0.75	0.17	0.6	9.1	3.11E–05	1.56E–05	0.006

TABLE L–5 **AMBIENT AIR CONCENTRATIONS OF VOLATILE ORGANIC CHEMICALS – SOUTHERN PORTION OF LBAD**

Cmax(Ca2) = Cw x f x Fw x t / Va
Ei = [Ca1 X Bt1] + [Ca2 X Bt2]
B = 833 L/hr
t2 = 0.2 hr

where:
Cmax = maximum air concentration in bathroom (mg/L)
Cw = water concentration (mg/L)
f = fraction volatilization (unitless)
Fw = water flow rate (m3/hr)
t = shower period (hr)
Va = bathroom size (m3)
Ei = Exposure concentration (mg)
B = Breathing rate
t2 = after shower period

ANALYTE	GROUNDWATER CONC. (mg/l) Cw	VOLATILIZATION FACTOR (unitless) f	SHOWER PERIOD (hr) t1	WATER FLOW RATE (m3/hr) Fw	BATHROOM VOLUME (m3) Va	AIR CONCENTRATION (mg/l) Ca2	AIR CONCENTRATION (mg/l) Ca1	EXPOSURE CONC. (mg) Ei
Acetone	0.0309	0.75	0.17	0.6	9.1	2.60E–04	1.30E–04	0.052
Benzene	0.0013	0.75	0.17	0.6	9.1	1.06E–05	5.32E–06	0.002
Carbon tetrachloride	0.0005	0.75	0.17	0.6	9.1	4.44E–06	2.22E–06	0.001
1,1–Dichloroethane	0.0009	0.75	0.17	0.6	9.1	7.87E–06	3.93E–06	0.002
1,2–Dichloroethenes	0.0032	0.75	0.17	0.6	9.1	2.69E–05	1.34E–05	0.005
1,3–Dimethylbenzene	0.0026	0.75	0.17	0.6	9.1	2.15E–05	1.07E–05	0.004
Ethylbenzene	0.0014	0.75	0.17	0.6	9.1	1.17E–05	5.87E–06	0.002
Methyl isobutyl ketone	0.0008	0.75	0.17	0.6	9.1	7.01E–06	3.51E–06	0.001
Tetrachloroethene	0.0005	0.75	0.17	0.6	9.1	4.44E–06	2.22E–06	0.001
Toluene	0.0057	0.75	0.17	0.6	9.1	4.78E–05	2.39E–05	0.010
Trichloroethene	0.0007	0.75	0.17	0.6	9.1	6.04E–06	3.02E–06	0.001
Vinyl chloride	0.0087	0.75	0.17	0.6	9.1	7.32E–05	3.66E–05	0.015
Xylenes	0.0058	0.75	0.17	0.6	9.1	4.87E–05	2.44E–05	0.010

ATTACHMENT 1

GROUNDWATER AIR INHALATION MODEL

SCREENING METHOD FOR ESTIMATING INHALATION EXPOSURE TO VOLATILE CHEMICALS FROM DOMESTIC WATER

1. Introduction

The following discussion has been developed to provide a screening method for estimating the indoor air concentrations of volatile chemicals from indoor water uses and the resulting human inhalation exposures, with an emphasis on showers. A computerized model titles MAVRIQ (Model for Analysis of volatiles and Residential Indoor Air Quality), which is under development, may also be used to refine the exposure estimates, since it more accurately accounts for human behavioral and water use patterns.

This procedure evolved from research done by Julian Andelman at the University of Pittsburg under funding from the Exposure Assessment Group at U.S. EPA in Washington, D.C. The references given provide a more detailed description of these procedures and related work.

2. When is Inhalation Exposure of Concern?

In order to determine the significance of the inhalation pathway the ratio for the vapor inhalation exposure to the water ingestion exposure can be calculated. Using Henry's Law Constant to obtain the equilibrium concentration in air, and setting a ratio of < 0.1 as criteria, the equation can be derived as follows:

$$\frac{\text{max inhalation exposure}}{\text{Water ingestion exposure}} < 0.1 \qquad (1)$$

$$\frac{H\,C_w \times (20{,}000\ \text{L/day})}{C_w \times (2\ \text{L/day})} < 0.1 \qquad (2)$$

$$H < 10^{-5} \qquad (3)$$

Where

C_w = contaminant concentration in water (mg/L)
H = Henry's Law Constant (unitless)

The unitless Henry's Law constant can be calculated by using the following equation:

$$H = H'/RT$$

Where

H'	=	Henry's Law Constant in atm-m^3/mol
R	=	gas constant in atm-m^3/mol °K
T	=	temperature in °K

Assuming a typical water temperature in a shower scenario of 40° C, RT is 2.6 x 10^{-2} atm-m^3/mol.

Equation (3) suggests that for compounds with Henry's Law Constants of $< 10^{-5}$, the inhalation exposure would not exceed ingestion and is probably much less; therefore, the inhalation pathway may not be of concern when compared to ingestion. Caution should be used when applying this criterion. If the i ingestion exposure is significant, the inhalation exposure, although orders of magnitude less, may also be significant when considered separately.

3. Showering Exposure

The derivations and assumptions used to estimate exposure through the showering scenario are included in Appendix 1. The exposure equation below accounts for the exposure during the showering time and the exposure during the period subsequent to the shower where there is a decay of the chemical concentration.

$$E_1 = [C_{aAVG1}Bt_1]_{shower} + [C_{aAVG2}Bt_2]_{after\ shower} \quad (4)$$

Where

E_1	=	exposure [mg]
C_{aAVG1}	=	average air concentration during shower [mg/L]
C_{aAVG2}	=	average air concentration after shower [mg/L]
B	=	breathing rate [L/hr]
t_1	=	shower period [hr]
t_2	=	after shower period [hr]

C_{aAVG1} and C_{aAVG2} are estimated using equations (5) and (6) and (7) below.

$$C_{aAVG1} = C_{aMAX}/2 \quad (5)$$

$$C_{aAVG2} = C_{aMAX} \quad (6)$$

$$C_{aMAX} = \frac{C_a \times f \times F_w \times t_1}{V_a} \quad (7)$$

Where

C_{aMAX}	=	maximum air concentration in bathroom [mg/L]
C_a	=	water concentration [mg/L]
f	=	fraction volatilization [unitless]
F_w	=	water flow rate [L/hr]
V_a	=	bathroom size [L]

Default values for the variables in these equations are tabulated in Table 1.

Using equations (4) through (7) and the average or most probable values from Table 1.

TABLE 1

Variable	Value or range	Reference
Fraction Volatilization (f)	0.5 - 0.9 (typical = 0.75)	1
Water Flow Rate (F_w) [L/hr]	600 - 1,800 (mean = 600)	2
Shower Period (t_1) [hr]	0.08 - 0.3 (mean = 0.08)	2
After Shower Period (t_2)	0.2 (typical)	1
Bathroom Size (V_a) [L]	8,300 - 9,800	3
Breathing Rate (B) [L/hr]	833 (20 m^3/day)	4

1. Andelman, J., Total Exposure to Volatile Organic Compounds in Potable Water, Chapter 20, Significance and Treatment of volatile Organic Compounds in Water Supplies

2. U.S. Department of Housing and Urban Development, Residential Water Conservation Projects, March 1984, Contract H-5230

3. Giardino, N.J., Gumerman, E., Andelman, J.B., Wilkes, C.R., Small, M.J., Borrazo, J.E., Davidson, C.I. (1990), Real-Time Air Measurements of Trichloroethylene in Domestic Bathrooms Using Contaminated Water.

4. U.S. EPA Factors Handbook.

APPENDIX M
PARAMETER VALUES AND RESULTS OF THE LEAD UPTAKE/BIOKINETIC MODEL

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LEAD UPTAKE/BIOKINETIC MODEL RESULTS

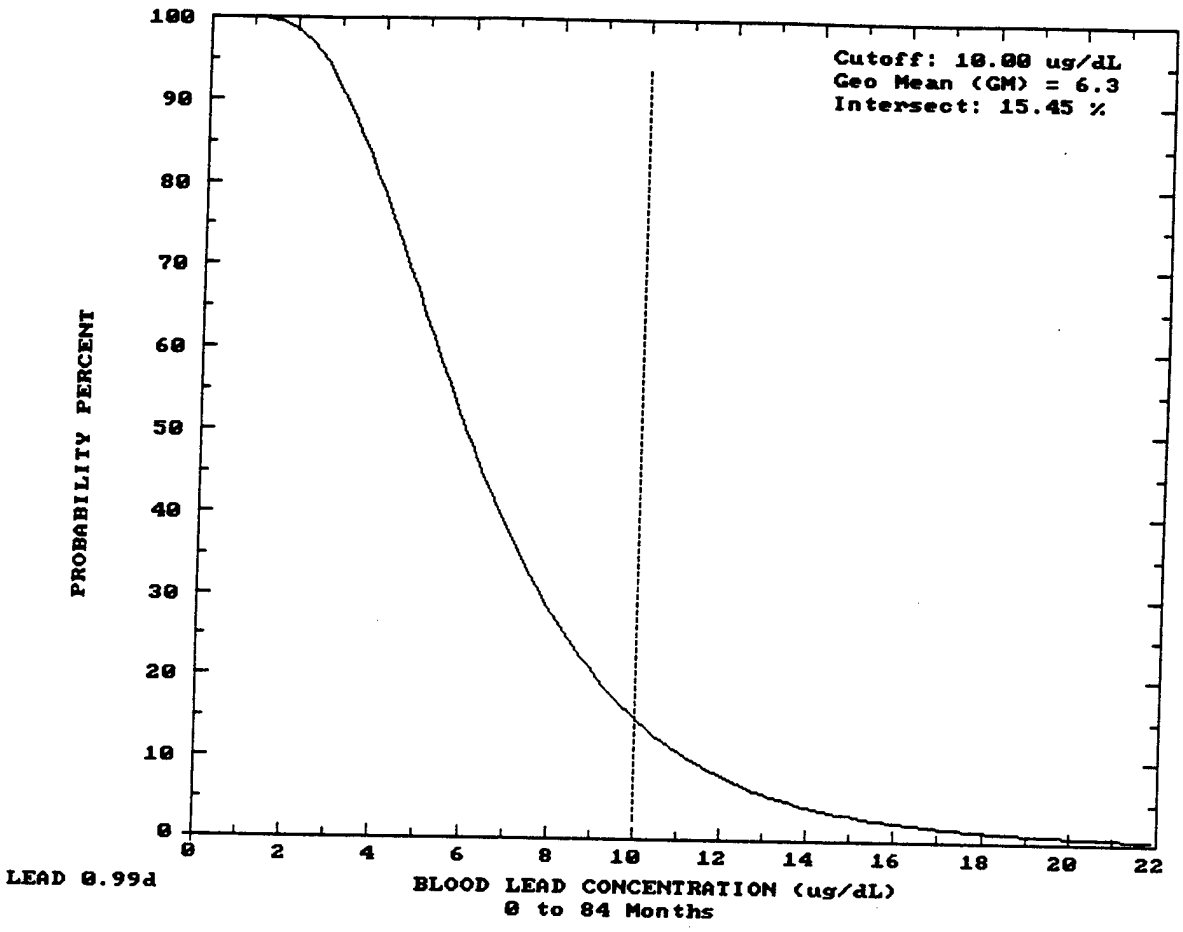
This appendix provides the output of the U.S. EPA integrated exposure uptake/biokinetic model for lead in children (U.S. EPA, 1994) utilized for groundwater concentrations that exceeded background concentrations for the LBAD site. Please note that the groundwater concentration of 41.9 μg/L from the southern portion of LBAD is less than the Kentucky water domestic supply source criterion of 50 μg/L (KDEP, 1994). The following table provides the lead concentrations for the groundwater sampled in both the southern and northern portions of the LBAD site.

LBAD Area	Groundwater Lead Concentration (μg/L)
Southern Portion	41.9
Northern Portion	62.7

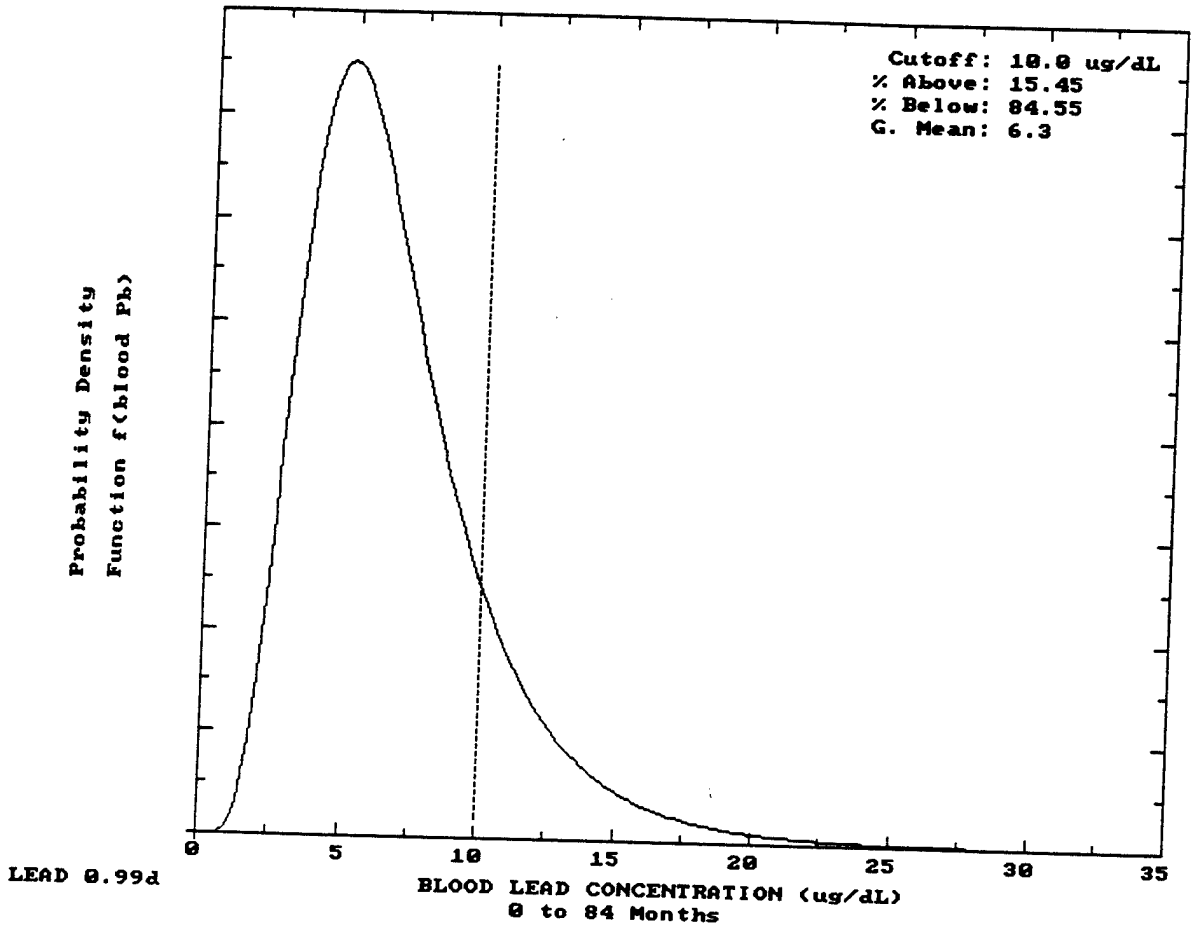
The site-specific parameter values employed in the model are presented in Table 1. The model incorporates exposures through indoor and outdoor air, drinking water ingestion (using the site-specific lead levels), ingestion of lead in soil and dust (using the default values of 200 μg/g for soil and 0.10 μg/m^3 for dust), and the default maternal blood contribution (using the default of 2.5 μg/dl lead).

Figures 1 and 3 present the probability plot relative to the geometric mean blood lead level predicted by the model and also show the geometric mean blood lead concentrations which would be associated with the conditions. The intersect on this figure represents the percentage of children who would be expected to have blood lead levels above 10 μg/dl.

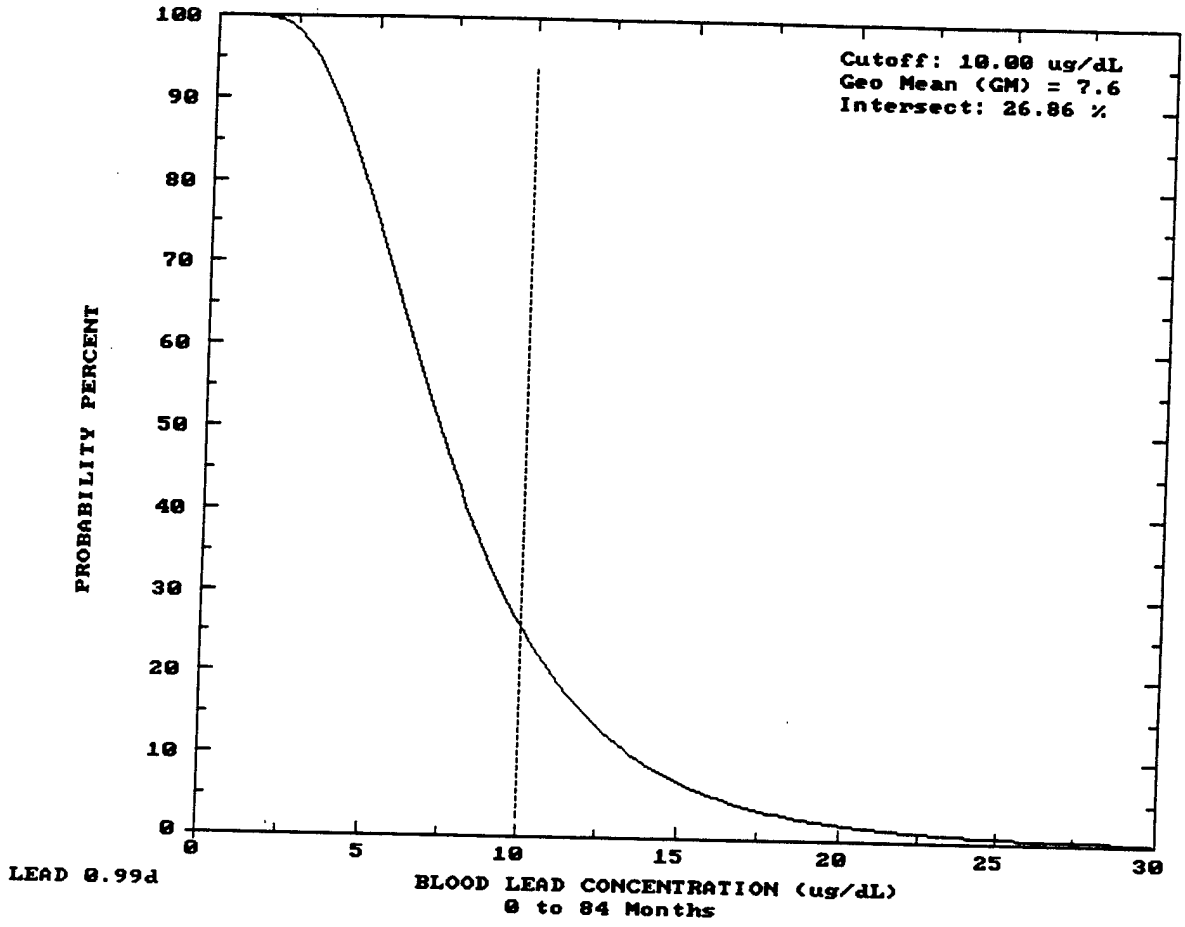
Figures 2 and 4 present the probability density distribution about the geometric mean and standard deviation of the blood lead levels. The model predicts the percent of the exposed population which would be expected to have blood lead levels above 10 μg/dl.



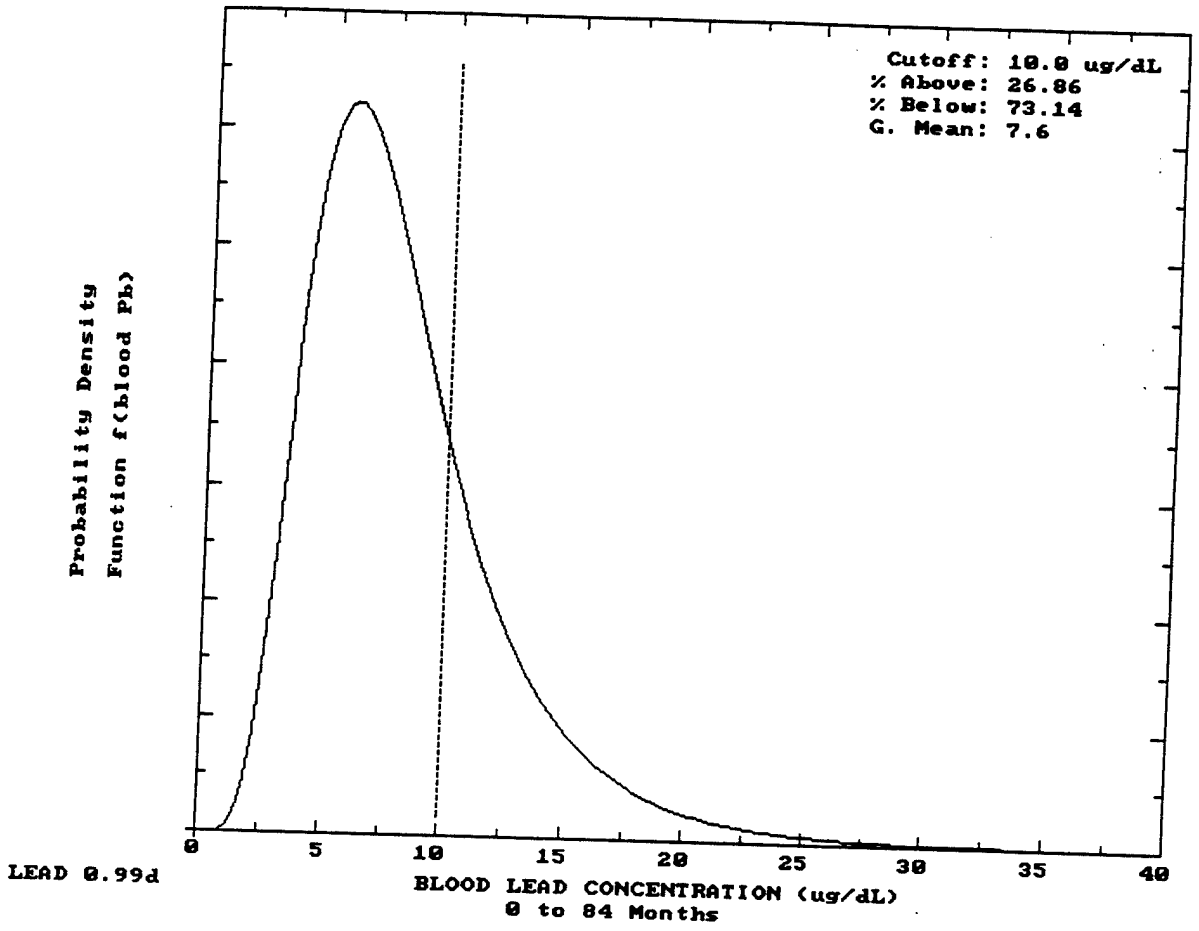
M&E	**PROBABILITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR THE SOUTHERN PORTION**	Project Number #007248-0005 Figure 1



M&E	**PROBABILITY DENSITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR THE SOUTHERN PORTION**	Project Number #007248-0005
		Figure 2



M&E	**PROBABILITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR THE NORTHERN PORTION**	Project Number #007248-0005 Figure 3



M&E	**PROBABILITY DENSITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR THE NORTHERN PORTION**	Project Number #007248-0005 Figure 4

TABLE 1
PARAMETER VALUE ASSUMPTIONS FOR THE LEAD UPTAKE BIOKINETIC MODEL FOR THE SOUTHERN PORTION

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 41.90 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	200.0	200.0
1-2	200.0	200.0
2-3	200.0	200.0
3-4	200.0	200.0
4-5	200.0	200.0
5-6	200.0	200.0
6-7	200.0	200.0

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

TABLE 1 (continued)
PARAMETER VALUE ASSUMPTIONS FOR THE LEAD UPTAKE BIOKINETIC MODEL FOR THE SOUTHERN PORTION

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	5.7	10.68	4.51
1-2:	7.3	18.27	6.88
2-3:	7.1	19.29	7.00
3-4:	6.8	19.73	7.13
4-5:	6.3	18.59	5.41
5-6:	5.8	18.98	4.92
6-7:	5.4	19.32	4.68

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.45	3.71	0.00	0.02
1-2:	2.46	8.90	0.00	0.03
2-3:	2.81	9.42	0.00	0.06
3-4:	2.75	9.78	0.00	0.07
4-5:	2.71	10.40	0.00	0.07
5-6:	2.89	11.07	0.00	0.09
6-7:	3.21	11.34	0.00	0.09

TABLE 2
PARAMETER VALUE ASSUMPTIONS FOR THE LEAD UPTAKE BIOKINETIC MODEL FOR THE NORTHERN PORTION

LEAD MODEL Version 0.99d

AIR CONCENTRATION: 0.100 ug Pb/m3 DEFAULT
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 62.70 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: constant conc.

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	200.0	200.0
1-2	200.0	200.0
2-3	200.0	200.0
3-4	200.0	200.0
4-5	200.0	200.0
5-6	200.0	200.0
6-7	200.0	200.0

Additional Dust Sources: None DEFAULT

PAINT Intake: 0.00 ug Pb/day DEFAULT

MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 2.50 ug Pb/dL

TABLE 2 (continued)
PARAMETER VALUE ASSUMPTIONS FOR THE LEAD UPTAKE BIOKINETIC MODEL FOR THE NORTHERN PORTION

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	6.6	12.29	4.43
1-2:	8.7	21.93	6.65
2-3:	8.5	23.23	6.79
3-4:	8.2	23.91	6.94
4-5:	7.7	23.15	5.28
5-6:	7.3	23.88	4.80
6-7:	6.8	24.37	4.57

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.40	5.44	0.00	0.02
1-2:	2.37	12.87	0.00	0.03
2-3:	2.72	13.66	0.00	0.06
3-4:	2.67	14.23	0.00	0.07
4-5:	2.64	15.16	0.00	0.07
5-6:	2.82	16.17	0.00	0.09
6-7	3.14	16.57	0.00	0.09

APPENDIX N
DETERMINATION OF DERMAL PERMEABILITY CONSTANTS AND ORAL ABSORPTION FACTORS FOR THE CHEMICALS OF CONCERN AT THE LEXINGTON-BLUEGRASS ARMY DEPOT

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N-2 Review of Oral Absorption Percentages for Chemicals of Concern N-5

DETERMINATION OF DERMAL PERMEABILITY CONSTANTS AND ORAL ABSORPTION FACTOR

This appendix provides the rationale and data sources utilized to derive appropriate permeability constants and absorption factors required for the evaluation of dermal exposure to groundwater. Similar information is also presented for the oral absorption factors for the chemicals of concern. The oral absorption factors are needed to adjust administered dose toxicity factors (i.e., noncarcinogen reference doses and cancer slope factors) to absorbed dose values as per U.S. EPA Risk Assessment Guidance for Superfund. Table 1 lists the chemical-specific values.

Dermal Permeability Constants

As detailed in Appendix L, quantification of dermal exposures to groundwater require consideration of the chemical-specific permeability constants (PCs), or the rate at which chemicals will move across the stratum corneum (in units of cm/hr). Inclusion of the PC value in the equation for quantifying dermal exposure to chemicals in water results in the calculation of an absorbed dose. Most PC values identified in the scientific literature are related to medicinal compounds, rather than chemicals typically found at hazardous waste sites. However, the U.S. EPA Exposure Assessment Group has compiled lists of PC values which have been derived in experimental studies or through modeling/predictive techniques (U.S. EPA, 1992a). Chemical-specific PC values reported in the U.S. EPA dermal exposure assessment guidance were employed for dermal exposures to the chemicals of concern for the LBAD site. The PC values utilized in the exposure calculations for the baseline Risk Assessment are provided in Table N-1. These PC values were taken from Table 5-7 in the guidance manual which provides predicted PC values for priority pollutants. In the case where PC values were not reported for the chemicals of concern, PC values were estimated for organics and relied upon a default value of 1.0E-03 for inorganics. For organics, the equation for estimating the PC value is:

$$\text{Log } K_p = -2.75 + 0.71 \log K_{ow} - 0.0061\ MW \quad (1)$$

where:

$\log K_{ow}$ = chemical specific

MW = molecular weight of chemical

Acetone was the only chemical of concern with a PC value estimated by this equation. The log K_{ow} of acetone is -0.24 and its molecular weight is 58.

Oral Absorption Factors

Oral absorption factors for the contaminants of concern were derived from reviews of relevant U. S. EPA Health Effects Assessment (HEA) documents, Agency for Toxic Substances and Disease Registry (ATSDR) toxicological profiles, and information provided by the U. S. EPA Environmental Criteria and Assessment Office (ECAO) as shown in Table N-2. These oral absorption factors were selected based on an approach wherein human absorption factors were selected prior to animal oral absorption factors where data were available. The highest absorption factor was utilized where only animal data were available. Where data were unavailable for a particular compound, the absorption factor of a chemical with similar chemical and physical properties was utilized.

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Table N-1 Permeability Constants and Oral Absorption Factors for Chemicals of Concern at LBAD

	CHEMICAL	PERMEABILITY CONSTANTS (CM/HR) (a)	ORAL ABSORPTION FACTOR (UNITLESS) (b)
VOLATILES	Acetone	0.00057	1.000
	Benzene	0.021	0.900
	Carbon tetrachloride	0.022	0.800
	Chloromethane	0.0042	1.000
	1,1-Dichloroethane	0.0089	1.000
	1,2-Dichloroethenes	0.01	0.900
	1,3-Dimethylbenzene	0.08	0.800
	Ethylbenzene	0.074	0.800
	Methyl isobutyl ketone	0.00326	0.800
	Tetrachloroethene	0.048	1.000
	Toluene	0.045	1.000
	Trichloroethene	0.016	1.000
	Vinyl chloride	0.0073	0.800
	Xylenes	0.08	0.920
SEMIVOLATILES	Bis(2-Ethylhexyl)phthalate	0.033	0.250
	2,4-Dimethylphenol	0.015	0.500
	Phenol	0.0055	0.900
PESTICIDES	alpha-BHC	0.016	0.900
	delta-BHC	0.016	0.900
	DDT	0.43	0.900
	Endosulfan	0.0033	0.500
	Lindane	0.014	0.990
INORGANICS	Aluminum	0.001	0.200
	Antimony	0.001	0.070
	Arsenic	0.001	0.950
	Barium	0.001	0.050
	Beryllium	0.001	0.100
	Boron	0.001	1.000
	Cadmium	0.001	0.070
	Calcium	0.001	0.200
	Chromium	0.001	0.030
	Cobalt	0.001	0.200
	Copper	0.001	0.990
	Iron	0.001	0.200
	Lead	0.001	0.500
	Magnesium	0.001	0.040
	Manganese	0.001	0.040
	Mercury	0.001	0.150
	Molybdenum	0.001	0.150
	Nickel	0.001	0.100
	Potassium	0.001	0.200
	Sodium	0.001	0.200
	Tellurium	0.001	0.200
	Thallium	0.001	0.100
	Tin	0.001	0.030
	Vanadium	0.001	0.010
	Zinc	0.001	0.300

NA – Not applicable/Not available

(a) Permeability constants and dermal absorption factors from Dermal Exposure Assessment: Principles and Applications, U.S. EP. Office of Health and Environmental Assessment, January 1992.

(b) Oral absorption factors from U.S. EPA, Environmental Criteria and Assessment Office (ECAO).

Default values, as per USEPA Region IV, were used as follows: 80% for VOCs, 50% for SVOCs, and 20% for inorganics

Table N-2 Review of Oral Absorption Percentages for Chemicals of Concern for LBAD (cont'd)

COMPOUND	ATSDR PROFILE (1988, 1989)	EPA DOCUMENTS
Acetone	Animal: 100%	HEA (1988) – readily absorbed; no quantitative data
Benzene	Animal: >90%	NA
Carbon tetrachloride	Animal: 80–85%	HEA (1987) – Animal: 65–86%
Chloromethane	NA	NA
1,2–Dichloroethenes	Animal: >90%	HEA(1984) – NA
1,3–Dimethylbenzene	NA	NA
1,1–Dichloroethane	Animal: 81–100%	NA
2,4–Dimethylphenol	NA	NA
Ethylbenzene	NA	HEEP (1986) – Animal: >90%
Methyl isobutyl ketone	NA	NA
Tetrachloroethene	Animal: Rapid and complete	HEA (1988) – Animal: 100%
Toluene	NA	HEA/DWCD (1984/1987) – Animal: 100%
Trichloroethene	Animal: 91–98%	HEA (1988) – Animal: 92–100%
Vinyl chloride	Human: No data Animal: Rapid and probably complete absorption	HEA(1984) – Rapid absorption, No quantitative data
Xylenes	Human: Some absorption, no data Animal: 87–92%	HEA(1980) – Animal: Nearly complete
Bis(2–Ethylhexyl)phthalate	Animal: 4.5–25%	NA
Phenol	Animal: 31–95% Human: 90%	HEEP(1987), HEA(1989) – Animal: 30%
alpha–BHC	Animal: 97.4%	NA
delta–BHC	Animal: 91.9%	NA
DDT	Animal: 70–90%	NA
Alpha–Endosulfan	NA	NA
Lindane	Animal: 91–99%	DWCD (1985) – Animal: 70% DWCD (1986) – Animal: 3–4% Human:3–4%
Aluminum	Animal/Human: Low	NA
Antimony	Animal: 2–7% Human: 1.0%	NA
Arsenic	Animal: 30–90% Human: 46–95%	AWQCD (1980) – Animal/Human: 70–>95%
Barium	Animal: 7.0% Human: <5.0%	HEA (1984) – Some absorption
Beryllium	Animal: <1.0%	AWQCD/DWCD (1980/1980) – Animal: 0.006–10%
Boron	NA	NA
Cadmium	Animal: 1.1–7% Human: 1.1–7%	HEA (1987) – Animal: 2–12% AWQCD (1980) – Human: 5.0%
Calcium	NA	NA
Chromium III	Animal: 2–3% Human: 0.4%	NA
Cobalt	NA	NA

Table N-2 Review of Oral Absorption Percentages for Chemicals of Concern for LBAD (cont'd)

COMPOUND	ATSDR PROFILE (1988, 1989)	EPA DOCUMENTS
Copper	Human: 15-99%	NA
Iron	NA	NA
Lead	Animal: 1-15% Human: 8-50%	HEA (1988)- Animal: 66% AWQCD (1980)- Animal/Human: 8-50%
Magnesium	NA	NA
Manganese	Animal: 2.5-5.5% Human: 3-5	DWCD (1986)- Animal: 3-4% Human: 3-4%
Mercury	Animal: 1-2% Human: 15%	HEA (1984)- Human: 15%
Molybdenum	NA	NA
Nickel	NA	DWCD/HEA (1980/1984)- Animal/Human: 1-10%
Potassium	NA	NA
Sodium	NA	NA
Tellurium	NA	NA
Thallium	Animal: 100%	NA
Tin	Animal: 3%	NA
Vanadium	Animal: 0.1-2.6% Human: Poorly absorbed	HEA (1987)- Animal: 1.6-2.6% Human: 0.1-1%
Zinc	Human: 20-30%	NA

NA - Not Available
ATSDR - Agency for Toxic Substances and Disease Registry
AWQCD - Ambient Water Quality Criteria Document
DWCD - Drinking Water Criteria Document
HEA - Health Effect Assessment Document
HEEP - Health and Environmental Effects Profile
HEED - Health and Environmental Effects Document
DWHA - Drinking Water Health Advisory

APPENDIX O
AREA-SPECIFIC EXPOSURE, NONCANCER HAZARD, AND CANCER RISK CALCULATIONS FOR CHEMICALS OF CONCERN AT THE LEXINGTON-BLUEGRASS ARMY DEPOT

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SWMU-SPECIFIC CALCULATIONS OF EXPOSURE, HAZARD, AND RISK

This appendix presents the detailed calculations of exposure, noncancer hazard, and cancer risk for each of the two LBAD groundwater areas quantitatively evaluated in the baseline risk assessment as previously described in Section 6.0. The calculations are presented on an exposure pathway-specific basis for the chemicals of concern detected in each of the two areas. The tables provide the parameter values utilized for each of the chemicals and exposure pathways to estimate the exposure levels for each of the pertinent receptor populations. The exposure estimates are then evaluated with respect to the appropriate noncarcinogenic reference dose and cancer slope factors to derive the chemical-specific noncancer hazards and cancer risks. The total chemical- and pathway-specific hazard and risk levels are then presented in the last column of each table. These results were, in turn, utilized to determine the total risks and hazards associated with each of the LBAD groundwater areas in Sections 6.5 and 6.7 of the baseline risk assessment.

INGESTION OF GROUNDWATER – FUTURE RESIDENTIAL ADULT SHORT TERM RISK FOR LBAD NORTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)−1	RISK
Acetone	0.1	2	350	7	70	25550	2.74E−04	NA	ND
Benzene	0.002	2	350	7	70	25550	5.48E−06	2.90E−02	1.59E−07
Chloromethane	0.003	2	350	7	70	25550	8.22E−06	1.30E−02	1.07E−07
1,3−Dimethylbenzene	0.001	2	350	7	70	25550	2.74E−06	NA	ND
Toluene	0.004	2	350	7	70	25550	1.10E−05	NA	ND
Aluminum	24.3	2	350	7	70	25550	6.66E−02	NA	ND
Arsenic	0.009	2	350	7	70	25550	2.47E−05	1.50E+00	3.70E−05
Barium	0.57	2	350	7	70	25550	1.56E−03	NA	ND
Beryllium	0.003	2	350	7	70	25550	8.22E−06	4.30E+00	3.53E−05
Boron	0.54	2	350	7	70	25550	1.48E−03	NA	ND
Calcium	1700	2	350	7	70	25550	4.66E+00	NA	ND
Chromium	0.047	2	350	7	70	25550	1.29E−04	NA	ND
Iron	53	2	350	7	70	25550	1.45E−01	NA	ND
Lead	0.06	2	350	7	70	25550	1.64E−04	NA	ND
Magnesium	920	2	350	7	70	25550	2.52E+00	NA	ND
Manganese	1.7	2	350	7	70	25550	4.66E−03	NA	ND
Mercury	0.00007	2	350	7	70	25550	1.92E−07	NA	ND
Nickel	0.05	2	350	7	70	25550	1.37E−04	NA	ND
Potassium	154	2	350	7	70	25550	4.22E−01	NA	ND
Sodium	9800	2	350	7	70	25550	2.68E+01	NA	ND
Thallium	0.12	2	350	7	70	25550	3.29E−04	NA	ND
Vanadium	0.04	2	350	7	70	25550	1.10E−04	NA	ND
							TOTAL RISK		7.26E−05

N/ – Not Available/Not Detected

INGESTION OF GROUNDWATER – FUTURE RESIDENTIAL ADULT SHORT TERM HAZARD FOR LBAD NORTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	SUBCHRONIC RfD (MG/KG/DAY)	HAZARD
Acetone	0.1	2	350	7	70	2555	2.74E−03	1.00E+00	2.74E−03
Benzene	0.002	2	350	7	70	2555	5.48E−05	NA	ND
Chloromethane	0.003	2	350	7	70	2555	8.22E−05	NA	ND
1,3−Dimethylbenzene	0.001	2	350	7	70	2555	2.74E−05	NA	ND
Toluene	0.004	2	350	7	70	2555	1.10E−04	2.00E+00	5.48E−05
Aluminum	24.3	2	350	7	70	2555	6.66E−01	NA	ND
Arsenic	0.009	2	350	7	70	2555	2.47E−04	3.00E−04	8.22E−01
Barium	0.57	2	350	7	70	2555	1.56E−02	7.00E−02	2.23E−01
Beryllium	0.003	2	350	7	70	2555	8.22E−05	5.00E−03	1.64E−02
Boron	0.54	2	350	7	70	2555	1.48E−02	9.00E−02	1.64E−01
Calcium	1700	2	350	7	70	2555	4.66E+01	NA	ND
Chromium	0.047	2	350	7	70	2555	1.29E−03	1.00E+00	1.29E−03
Iron	53	2	350	7	70	2555	1.45E+00	NA	ND
Lead	0.06	2	350	7	70	2555	1.64E−03	1.40E−03	1.17E+00
Magnesium	920	2	350	7	70	2555	2.52E+01	NA	ND
Manganese	1.7	2	350	7	70	2555	4.66E−02	5.00E−03	9.32E+00
Mercury	0.00007	2	350	7	70	2555	1.92E−06	3.00E−04	6.39E−03
Nickel	0.05	2	350	7	70	2555	1.37E−03	2.00E−02	6.85E−02
Potassium	154	2	350	7	70	2555	4.22E+00	NA	ND
Sodium	9800	2	350	7	70	2555	2.68E+02	NA	ND
Thallium	0.12	2	350	7	70	2555	3.29E−03	8.00E−04	4.11E+00
Vanadium	0.04	2	350	7	70	2555	1.10E−03	7.00E−03	1.57E−01
								TOTAL HAZARD	1.61E+01

NA/ND – Not Available/Not Detected

DERMAL CONTACT WITH GROUNDWATER – FUTURE RESIDENTIAL ADULT SHORT TERM RISK FOR LBAD NORTH

ANALYTE	CW (MG/L)	SA (CM2)	PC (CM/HR)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	CF (L/CM3)	BW (KG)	AT (DAYS)	ABSORBED INTAKE (MG/KG/DAY)	ADJUSTED CSF (MG/KG/DAY)−1	RISK
Acetone	0.1	19400	5.7E−04	0.17	350	7	1.0E−03	70	25550	2.58E−07	NA	ND
Benzene	0.002	19400	1.1E−01	0.17	350	7	1.0E−03	70	25550	9.94E−07	3.22E−02	3.20E−08
Chloromethane	0.003	19400	4.2E−03	0.17	350	7	1.0E−03	70	25550	5.69E−08	1.62E−02	9.25E−10
1,3−Dimethylbenzene	0.001	19400	8.0E−02	0.17	350	7	1.0E−03	70	25550	3.61E−07	NA	ND
Toluene	0.004	19400	1.0E+00	0.17	350	7	1.0E−03	70	25550	1.81E−05	NA	ND
Aluminum	24.3	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	1.10E−04	NA	ND
Arsenic	0.009	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	4.07E−08	1.58E+00	6.42E−08
Barium	0.57	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	2.58E−06	NA	ND
Beryllium	0.003	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	1.36E−08	4.30E+01	5.83E−07
Boron	0.54	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	2.44E−06	NA	ND
Calcium	1700	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	7.68E−03	NA	ND
Chromium	0.047	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	2.12E−07	NA	ND
Iron	53	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	2.39E−04	NA	ND
Lead	0.06	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	2.71E−07	NA	ND
Magnesium	920	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	4.16E−03	NA	ND
Manganese	1.7	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	7.68E−06	NA	ND
Mercury	0.00007	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	3.16E−10	NA	ND
Nickel	0.05	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	2.26E−07	NA	ND
Potassium	154	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	6.96E−04	NA	ND
Sodium	9800	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	4.43E−02	NA	ND
Thallium	0.12	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	5.42E−07	NA	ND
Vanadium	0.04	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	1.81E−07	NA	ND
											TOTAL RISK	6.80E−07

N. Not Available/Not Determined

DERMAL CONTACT WITH GROUNDWATER – FUTURE RESIDENTIAL ADULT SHORT TERM HAZARD FOR LBAD NORTH

ANALYTE	CW (MG/L)	SA (CM2)	PC (CM/HR)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	CF (L/CM3)	BW (KG)	AT (DAYS)	ABSORBED INTAKE (MG/KG/DAY)	ADJUSTED RfD (MG/KG/DAY)	HAZARD
Acetone	0.1	19400	5.70E-04	0.17	350	7	1.0E-03	70	2555	2.58E-06	1.00E+00	2.58E-06
Benzene	0.002	19400	1.10E-01	0.17	350	7	1.0E-03	70	2555	9.94E-06	NA	ND
Chloromethane	0.003	19400	4.20E-03	0.17	350	7	1.0E-03	70	2555	5.69E-07	NA	ND
1,3-Dimethylbenzene	0.001	19400	8.00E-02	0.17	350	7	1.0E-03	70	2555	3.61E-06	NA	ND
Toluene	0.004	19400	1.00E+00	0.17	350	7	1.0E-03	70	2555	1.81E-04	2.00E+00	9.04E-05
Aluminum	24.3	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	1.10E-03	NA	ND
Arsenic	0.009	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	4.07E-07	2.85E-04	1.43E-03
Barium	0.57	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	2.58E-05	3.50E-03	7.36E-03
Beryllium	0.003	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	1.36E-07	5.00E-04	2.71E-04
Boron	0.54	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	2.44E-05	9.00E-02	2.71E-04
Calcium	1700	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	7.68E-02	NA	ND
Chromium	0.047	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	2.12E-06	3.00E-02	7.08E-05
Iron	53	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	2.39E-03	NA	ND
Lead	0.06	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	2.71E-06	7.00E-04	3.87E-03
Magnesium	920	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	4.16E-02	NA	ND
Manganese	1.7	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	7.68E-05	2.00E-04	3.84E-01
Mercury	0.00007	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	3.16E-09	4.50E-05	7.03E-05
Nickel	0.05	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	2.26E-06	2.00E-03	1.13E-03
Potassium	154	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	6.96E-03	NA	ND
Sodium	9800	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	4.43E-01	NA	ND
Thallium	0.12	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	5.42E-06	8.00E-05	6.78E-02
Vanadium	0.04	19400	1.00E-03	0.17	350	7	1.0E-03	70	2555	1.81E-06	7.00E-05	2.58E-02
											TOTAL HAZARD	4.92E-01

NA/ND – Not Available/Not Determined

INHALATION OF VAPORS WHILE SHOWERING – FUTURE RESIDENTIAL ADULT SHORT TERM RISK FOR LBAD NORTH

ANALYTE	Ei (MG)	SF (SHOWERS/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)–1	RISK
Acetone	0.17	1.0	350	7	70	25550	2.30E–04	NA	ND
Benzene	0.00	1.0	350	7	70	25550	5.03E–06	2.90E–02	1.46E–07
Chloromethane	0.00	1.0	350	7	70	25550	6.20E–06	6.30E–03	3.91E–08
1,3–Dimethylbenzene	0.00	1.0	350	7	70	25550	2.99E–06	NA	ND
Toluene	0.01	1.0	350	7	70	25550	8.52E–06	NA	ND
								TOTAL RISK	1.85E–07

INHALATION OF VAPORS WHILE SHOWERING – FUTURE RESIDENTIAL ADULT SHORT TERM HAZARD FOR LBAD NORTH

ANALYTE	Ei (MG)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	SUBCHRONIC RfD (MG/KG/DAY)	HAZARD
Acetone	0.17	1.0	350	7	70	2555	2.30E–03	NA	ND
Benzene	0.00	1.0	350	7	70	2555	5.03E–05	NA	ND
Chloromethane	0.00	1.0	350	7	70	2555	6.20E–05	NA	ND
1,3–Dimethylbenzene	0.00	1.0	350	7	70	2555	2.99E–05	NA	ND
Toluene	0.01	1.0	350	7	70	2555	8.52E–05	3.00E–01	2.84E–04
								TOTAL HAZARD	2.84E–04

NA/ND – Not Applicable/Not Determined

INGESTION OF GROUNDWATER – FUTURE RESIDENTIAL ADULT LONG TERM RISK FOR LBAD NORTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)–1	RISK
Acetone	0.1	2	350	30	70	25550	1.17E–03	NA	ND
Benzene	0.002	2	350	30	70	25550	2.35E–05	2.90E–02	6.81E–07
Chloromethane	0.003	2	350	30	70	25550	3.52E–05	1.30E–02	4.58E–07
1,3–Dimethylbenzene	0.001	2	350	30	70	25550	1.17E–05	NA	ND
Toluene	0.004	2	350	30	70	25550	4.70E–05	NA	ND
Aluminum	24.3	2	350	30	70	25550	2.85E–01	NA	ND
Arsenic	0.009	2	350	30	70	25550	1.06E–04	1.50E+00	1.59E–04
Barium	0.57	2	350	30	70	25550	6.69E–03	NA	ND
Beryllium	0.003	2	350	30	70	25550	3.52E–05	4.30E+00	1.51E–04
Boron	0.54	2	350	30	70	25550	6.34E–03	NA	ND
Calcium	1700	2	350	30	70	25550	2.00E+01	NA	ND
Chromium	0.047	2	350	30	70	25550	5.52E–04	NA	ND
Iron	53	2	350	30	70	25550	6.22E–01	NA	ND
Lead	0.06	2	350	30	70	25550	7.05E–04	NA	ND
Magnesium	920	2	350	30	70	25550	1.08E+01	NA	ND
Manganese	1.7	2	350	30	70	25550	2.00E–02	NA	ND
Mercury	0.00007	2	350	30	70	25550	8.22E–07	NA	ND
Nickel	0.05	2	350	30	70	25550	5.87E–04	NA	ND
Potassium	154	2	350	30	70	25550	1.81E+00	NA	ND
Sodium	9800	2	350	30	70	25550	1.15E+02	NA	ND
Thallium	0.12	2	350	30	70	25550	1.41E–03	NA	ND
Vanadium	0.04	2	350	30	70	25550	4.70E–04	NA	ND
								TOTAL RISK	**3.11E–04**

NA/ND – Not Available/Not Detected

INGESTION OF GROUNDWATER – FUTURE RESIDENTIAL ADULT LONG TERM HAZARD FOR LBAD NORTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	RfD (MG/KG/DAY)	HAZARD
Acetone	0.1	2	350	30	70	10950	2.74E−03	1.00E−01	2.74E−02
Benzene	0.002	2	350	30	70	10950	5.48E−05	3.00E−04	1.83E−01
Chloromethane	0.003	2	350	30	70	10950	8.22E−05	NA	ND
1,3−Dimethylbenzene	0.001	2	350	30	70	10950	2.74E−05	2.00E+00	1.37E−05
Toluene	0.004	2	350	30	70	10950	1.10E−04	2.00E−01	5.48E−04
Aluminum	24.3	2	350	30	70	10950	6.66E−01	1.00E+00	6.66E−01
Arsenic	0.009	2	350	30	70	10950	2.47E−04	3.00E−04	8.22E−01
Barium	0.57	2	350	30	70	10950	1.56E−02	7.00E−02	2.23E−01
Beryllium	0.003	2	350	30	70	10950	8.22E−05	5.00E−03	1.64E−02
Boron	0.54	2	350	30	70	10950	1.48E−02	9.00E−02	1.64E−01
Calcium	1700	2	350	30	70	10950	4.66E+01	NA	ND
Chromium	0.047	2	350	30	70	10950	1.29E−03	1.00E+00	1.29E−03
Iron	53	2	350	30	70	10950	1.45E+00	NA	ND
Lead	0.06	2	350	30	70	10950	1.64E−03	1.40E−03	1.17E+00
Magnesium	920	2	350	30	70	10950	2.52E+01	NA	ND
Manganese	1.7	2	350	30	70	10950	4.66E−02	5.00E−03	9.32E+00
Mercury	0.00007	2	350	30	70	10950	1.92E−06	3.00E−04	6.39E−03
Nickel	0.05	2	350	30	70	10950	1.37E−03	2.00E−02	6.85E−02
Potassium	154	2	350	30	70	10950	4.22E+00	5.00E+01	8.44E−02
Sodium	9800	2	350	30	70	10950	2.68E+02	3.40E+01	7.90E+00
Thallium	0.12	2	350	30	70	10950	3.29E−03	8.00E−05	4.11E+01
Vanadium	0.04	2	350	30	70	10950	1.10E−03	7.00E−03	1.57E−01
								TOTAL HAZARD	**6.19E+01**

N. = Not Available/Not Detected

DERMAL CONTACT WITH GROUNDWATER – FUTURE RESIDENTIAL ADULT LONG TERM RISK FOR LBAD NORTH

ANALYTE	CW (MG/L)	SA (CM2)	PC (CM/HR)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	CF (L/CM3)	BW (KG)	AT (DAYS)	ABSORBED INTAKE (MG/KG/DAY)	ADJUSTED CSF (MG/KG/DAY)-1	RISK
Acetone	0.1	19400	5.7E-04	0.17	350	30	1.0E-03	70	25550	1.10E-06	NA	ND
Benzene	0.002	19400	1.1E-01	0.17	350	30	1.0E-03	70	25550	4.26E-06	3.22E-02	1.37E-07
Chloromethane	0.003	19400	4.2E-03	0.17	350	30	1.0E-03	70	25550	2.44E-07	1.62E-02	3.96E-09
1,3-Dimethylbenzene	0.001	19400	8.0E-02	0.17	350	30	1.0E-03	70	25550	1.55E-06	NA	ND
Toluene	0.004	19400	1.0E+00	0.17	350	30	1.0E-03	70	25550	7.74E-05	NA	ND
Aluminum	24.3	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	4.70E-04	NA	ND
Arsenic	0.009	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.74E-07	1.58E+00	2.75E-07
Barium	0.57	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.10E-05	NA	ND
Beryllium	0.003	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	5.81E-08	4.30E+01	2.50E-06
Boron	0.54	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.05E-05	NA	ND
Calcium	1700	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	3.29E-02	NA	ND
Chromium	0.047	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	9.10E-07	NA	ND
Iron	53	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.03E-03	NA	ND
Lead	0.06	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.16E-06	NA	ND
Magnesium	920	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.78E-02	NA	ND
Manganese	1.7	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	3.29E-05	NA	ND
Mercury	0.00007	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.36E-09	NA	ND
Nickel	0.05	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	9.68E-07	NA	ND
Potassium	154	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	2.98E-03	NA	ND
Sodium	9800	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.90E-01	NA	ND
Thallium	0.12	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	2.32E-06	NA	ND
Vanadium	0.04	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	7.74E-07	NA	ND
											TOTAL RISK	2.91E-06

NA/ND – Not Available/Not Determined

DERMAL CONTACT WITH GROUNDWATER – FUTURE RESIDENTIAL ADULT LONG TERM HAZARD FOR LBAD NORTH

ANALYTE	CW (MG/L)	SA (CM2)	PC (CM/HR)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	CF (L/CM3)	BW (KG)	AT (DAYS)	ABSORBED INTAKE (MG/KG/DAY)	ADJUSTED RfD (MG/KG/DAY)	HAZARD
Acetone	0.1	19400	5.70E−04	0.17	350	30	1.0E−03	70	10950	2.58E−06	1.00E−01	2.58E−05
Benzene	0.002	19400	1.10E−01	0.17	350	30	1.0E−03	70	10950	9.94E−06	2.70E−04	3.68E−02
Chloromethane	0.003	19400	4.20E−03	0.17	350	30	1.0E−03	70	10950	5.69E−07	NA	ND
1,3−Dimethylbenzene	0.001	19400	8.00E−02	0.17	350	30	1.0E−03	70	10950	3.61E−06	1.60E+00	2.26E−06
Toluene	0.004	19400	1.00E+00	0.17	350	30	1.0E−03	70	10950	1.81E−04	2.00E−01	9.04E−04
Aluminum	24.3	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	1.10E−03	2.00E−01	5.49E−03
Arsenic	0.009	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	4.07E−07	2.85E−04	1.43E−03
Barium	0.57	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	2.58E−05	3.50E−03	7.36E−03
Beryllium	0.003	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	1.36E−07	5.00E−04	2.71E−04
Boron	0.54	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	2.44E−05	9.00E−02	2.71E−04
Calcium	1700	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	7.68E−02	NA	ND
Chromium	0.047	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	2.12E−06	3.00E−02	7.08E−05
Iron	53	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	2.39E−03	NA	ND
Lead	0.06	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	2.71E−06	7.00E−04	3.87E−03
Magnesium	920	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	4.16E−02	NA	ND
Manganese	1.7	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	7.68E−05	2.00E−04	3.84E−01
Mercury	0.00007	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	3.16E−09	4.50E−05	7.03E−05
Nickel	0.05	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	2.26E−06	2.00E−03	1.13E−03
Potassium	154	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	6.96E−03	1.00E+01	6.96E−04
Sodium	9800	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	4.43E−01	6.80E+00	6.51E−02
Thallium	0.12	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	5.42E−06	8.00E−06	6.78E−01
Vanadium	0.04	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	1.81E−06	7.00E−05	2.58E−02
											TOTAL HAZARD	1.21E+00

N. = Not Available/Not Determined

INHALATION OF VAPORS WHILE SHOWERING – FUTURE RESIDENTIAL ADULT LONG TERM RISK FOR LBAD NORTH

ANALYTE	Ei (MG)	SF (SHOWERS/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)–1	RISK
Acetone	0.17	1.0	350	30	70	25550	9.87E–04	NA	ND
Benzene	0.004	1.0	350	30	70	25550	2.16E–05	2.90E–02	6.25E–07
Chloromethane	0.005	1.0	350	30	70	25550	2.66E–05	6.30E–03	1.67E–07
1,3–Dimethylbenzene	0.002	1.0	350	30	70	25550	1.28E–05	NA	ND
Toluene	0.006	1.0	350	30	70	25550	3.65E–05	NA	ND
								TOTAL RISK	7.93E–07

INHALATION OF VAPORS WHILE SHOWERING – FUTURE RESIDENTIAL ADULT LONG TERM HAZARD FOR LBAD NORTH

ANALYTE	Ei (MG)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	RfD (MG/KG/DAY)	HAZARD
Acetone	0.168	1.0	350	30	70	10950	2.30E–03	NA	ND
Benzene	0.004	1.0	350	30	70	10950	5.03E–05	1.70E–03	2.96E–02
Chloromethane	0.005	1.0	350	30	70	10950	6.20E–05	NA	ND
1,3–Dimethylbenzene	0.002	1.0	350	30	70	10950	2.99E–05	NA	ND
Toluene	0.006	1.0	350	30	70	10950	8.52E–05	1.00E–01	8.52E–04
								TOTAL HAZARD	3.05E–02

NA/ND – Not Applicable/Not Determined

INGESTION OF GROUNDWATER – FUTURE RESIDENTIAL CHILD RISK FOR LBAD NORTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)−1	RISK
Acetone	0.1	1	350	7	16	25550	5.99E−04 NA		ND
Benzene	0.002	1	350	7	16	25550	1.20E−05	2.90E−02	3.48E−07
Chloromethane	0.003	1	350	7	16	25550	1.80E−05	1.30E−02	2.34E−07
1,3−Dimethylbenzene	0.001	1	350	7	16	25550	5.99E−06 NA		ND
Toluene	0.004	1	350	7	16	25550	2.40E−05 NA		ND
Aluminum	24.3	1	350	7	16	25550	1.46E−01 NA		ND
Arsenic	0.009	1	350	7	16	25550	5.39E−05	1.50E+00	8.09E−05
Barium	0.57	1	350	7	16	25550	3.42E−03 NA		ND
Beryllium	0.003	1	350	7	16	25550	1.80E−05	4.30E+00	7.73E−05
Boron	0.54	1	350	7	16	25550	3.24E−03 NA		ND
Calcium	1700	1	350	7	16	25550	1.02E+01 NA		ND
Chromium	0.047	1	350	7	16	25550	2.82E−04 NA		ND
Iron	53	1	350	7	16	25550	3.18E−01 NA		ND
Lead	0.06	1	350	7	16	25550	3.60E−04 NA		ND
Magnesium	920	1	350	7	16	25550	5.51E+00 NA		ND
Manganese	1.7	1	350	7	16	25550	1.02E−02 NA		ND
Mercury	0.00007	1	350	7	16	25550	4.20E−07 NA		ND
Nickel	0.05	1	350	7	16	25550	3.00E−04 NA		ND
Potassium	154	1	350	7	16	25550	9.23E−01 NA		ND
Sodium	9800	1	350	7	16	25550	5.87E+01 NA		ND
Thallium	0.12	1	350	7	16	25550	7.19E−04 NA		ND
Vanadium	0.04	1	350	7	16	25550	2.40E−04 NA		ND
								TOTAL RISK	1.59E−04

N – Not Available/Not Detected

INGESTION OF GROUNDWATER – FUTURE RESIDENTIAL CHILD SHORT TERM HAZARD FOR LBAD NORTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	SUBCHRONIC RfD (MG/KG/DAY)	HAZARD
Acetone	0.1	1	350	7	16	2555	5.99E−03	1.00E+00	5.99E−03
Benzene	0.002	1	350	7	16	2555	1.20E−04 NA		ND
Chloromethane	0.003	1	350	7	16	2555	1.80E−04 NA		ND
1,3−Dimethylbenzene	0.001	1	350	7	16	2555	5.99E−05 NA		ND
Toluene	0.004	1	350	7	16	2555	2.40E−04	2.00E+00	1.20E−04
Aluminum	24.3	1	350	7	16	2555	1.46E+00 NA		ND
Arsenic	0.009	1	350	7	16	2555	5.39E−04	3.00E−04	1.80E+00
Barium	0.57	1	350	7	16	2555	3.42E−02	7.00E−02	4.88E−01
Beryllium	0.003	1	350	7	16	2555	1.80E−04	5.00E−03	3.60E−02
Boron	0.54	1	350	7	16	2555	3.24E−02	9.00E−02	3.60E−01
Calcium	1700	1	350	7	16	2555	1.02E+02 NA		ND
Chromium	0.047	1	350	7	16	2555	2.82E−03	1.00E+00	2.82E−03
Iron	53	1	350	7	16	2555	3.18E+00 NA		ND
Lead	0.06	1	350	7	16	2555	3.60E−03	1.40E−03	2.57E+00
Magnesium	920	1	350	7	16	2555	5.51E+01 NA		ND
Manganese	1.7	1	350	7	16	2555	1.02E−01	5.00E−03	2.04E+01
Mercury	0.00007	1	350	7	16	2555	4.20E−06	3.00E−04	1.40E−02
Nickel	0.05	1	350	7	16	2555	3.00E−03	2.00E−02	1.50E−01
Potassium	154	1	350	7	16	2555	9.23E+00 NA		ND
Sodium	9800	1	350	7	16	2555	5.87E+02 NA		ND
Thallium	0.12	1	350	7	16	2555	7.19E−03	8.00E−04	8.99E+00
Vanadium	0.04	1	350	7	16	2555	2.40E−03	7.00E−03	3.42E−01
								TOTAL HAZARD	**3.51E+01**

NA/ND – Not Available/Not Detected

DERMAL CONTACT WITH GROUNDWATER – FUTURE RESIDENTIAL CHILD RISK FOR LBAD NORTH

ANALYTE	CW (MG/L)	SA (CM2)	PC (CM/HR)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	CF (L/CM3)	BW (KG)	AT (DAYS)	ABSORBED INTAKE (MG/KG/DAY)	ADJUSTED CSF (MG/KG/DAY)−1	RISK
Acetone	0.1	7280	5.7E−04	0.17	350	7	1.0E−03	16	25550	4.23E−07	NA	ND
Benzene	0.002	7280	1.1E−01	0.17	350	7	1.0E−03	16	25550	1.63E−06	3.22E−02	5.26E−08
Chloromethane	0.003	7280	4.2E−03	0.17	350	7	1.0E−03	16	25550	9.35E−08	1.62E−02	1.52E−09
1,3−Dimethylbenzene	0.001	7280	8.0E−02	0.17	350	7	1.0E−03	16	25550	5.93E−07	NA	ND
Toluene	0.004	7280	1.0E+00	0.17	350	7	1.0E−03	16	25550	2.97E−05	NA	ND
Aluminum	24.3	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	1.80E−04	NA	ND
Arsenic	0.009	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	6.68E−08	1.58E+00	1.05E−07
Barium	0.57	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	4.23E−06	NA	ND
Beryllium	0.003	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	2.23E−08	4.30E+01	9.57E−07
Boron	0.54	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	4.01E−06	NA	ND
Calcium	1700	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	1.26E−02	NA	ND
Chromium	0.047	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	3.49E−07	NA	ND
Iron	53	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	3.93E−04	NA	ND
Lead	0.06	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	4.45E−07	NA	ND
Magnesium	920	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	6.82E−03	NA	ND
Manganese	1.7	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	1.26E−05	NA	ND
Mercury	0.00007	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	5.19E−10	NA	ND
Nickel	0.05	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	3.71E−07	NA	ND
Potassium	154	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	1.14E−03	NA	ND
Sodium	9800	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	7.27E−02	NA	ND
Thallium	0.12	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	8.90E−07	NA	ND
Vanadium	0.04	7280	1.0E−03	0.17	350	7	1.0E−03	16	25550	2.97E−07	NA	ND
											TOTAL RISK	1.12E−06

N/A – Not Available/Not Determined

DERMAL CONTACT WITH GROUNDWATER – FUTURE RESIDENTIAL CHILD SHORT TERM HAZARD FOR LBAD NORTH

ANALYTE	CW (MG/L)	SA (CM2)	PC (CM/HR)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	CF (L/CM3)	BW (KG)	AT (DAYS)	ABSORBED INTAKE (MG/KG/DAY)	ADJUSTED RfD (MG/KG/DAY)	HAZARD
Acetone	0.1	7280	5.7E-04	0.17	350	7	1.0E-03	16	2555	4.23E-06	1.00E+00	4.23E-06
Benzene	0.002	7280	1.1E-01	0.17	350	7	1.0E-03	16	2555	1.63E-05	NA	ND
Chloromethane	0.003	7280	4.2E-03	0.17	350	7	1.0E-03	16	2555	9.35E-07	NA	ND
1,3-Dimethylbenzene	0.001	7280	8.0E-02	0.17	350	7	1.0E-03	16	2555	5.93E-06	NA	ND
Toluene	0.004	7280	1.0E+00	0.17	350	7	1.0E-03	16	2555	2.97E-04	2.00E+00	1.48E-04
Aluminum	24.3	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	1.80E-03	NA	ND
Arsenic	0.009	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	6.68E-07	2.85E-04	2.34E-03
Barium	0.57	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	4.23E-05	3.50E-03	1.21E-02
Beryllium	0.003	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	2.23E-07	5.00E-04	4.45E-04
Boron	0.54	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	4.01E-05	9.00E-02	4.45E-04
Calcium	1700	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	1.26E-01	NA	ND
Chromium	0.047	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	3.49E-06	3.00E-02	1.16E-04
Iron	53	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	3.93E-03	NA	ND
Lead	0.06	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	4.45E-06	7.00E-04	6.36E-03
Magnesium	920	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	6.82E-02	NA	ND
Manganese	1.7	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	1.26E-04	2.00E-04	6.30E-01
Mercury	0.00007	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	5.19E-09	4.50E-05	1.15E-04
Nickel	0.05	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	3.71E-06	2.00E-03	1.85E-03
Potassium	154	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	1.14E-02	NA	ND
Sodium	9800	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	7.27E-01	NA	ND
Thallium	0.12	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	8.90E-06	8.00E-05	1.11E-01
Vanadium	0.04	7280	1.0E-03	0.17	350	7	1.0E-03	16	2555	2.97E-06	7.00E-05	4.24E-02
											TOTAL HAZARD	8.08E-01

NA/ND – Not Available/Not Determined

INHALATION OF VAPORS WHILE SHOWERING – FUTURE RESIDENTIAL CHILD RISK FOR LBAD NORTH

ANALYTE	Ei (MG)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)–1	RISK
Acetone	0.17	1.0	350	7	16	25550	1.01E–03	NA	ND
Benzene	0.00	1.0	350	7	16	25550	2.20E–05	2.90E–02	6.38E–07
Chloromethane	0.00	1.0	350	7	16	25550	2.71E–05	6.30E–03	1.71E–07
1,3–Dimethylbenzene	0.00	1.0	350	7	16	25550	1.31E–05	NA	ND
Toluene	0.01	1.0	350	7	16	25550	3.73E–05	NA	ND
								TOTAL RISK	**8.09E–07**

INHALATION OF VAPORS WHILE SHOWERING – FUTURE RESIDENTIAL CHILD SHORT TERM HAZARD FOR LBAD NORTH

ANALYTE	Ei (MG)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	SUBCHRONIC RfD (MG/KG/DAY)	HAZARD
Acetone	0.17	1.0	350	7	16	2555	1.01E–02	NA	ND
Benzene	0.00	1.0	350	7	16	2555	2.20E–04	NA	ND
Chloromethane	0.00	1.0	350	7	16	2555	2.71E–04	NA	ND
1,3–Dimethylbenzene	0.00	1.0	350	7	16	2555	1.31E–04	NA	ND
Toluene	0.01	1.0	350	7	16	2555	3.73E–04	3.00E–01	1.24E–03
								TOTAL HAZARD	**1.24E–03**

NA/ND – Not Applicable/Not Determined

INGESTION OF GROUNDWATER -- FUTURE OCCUPATIONAL ADULT SHORT TERM RISK FOR LBAD NORTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)-1	RISK
Acetone	0.1	1	250	7	70	25550	9.78E-05	NA	ND
Benzene	0.002	1	250	7	70	25550	1.96E-06	2.90E-02	5.68E-08
Chloromethane	0.003	1	250	7	70	25550	2.94E-06	1.30E-02	3.82E-08
1,3-Dimethylbenzene	0.001	1	250	7	70	25550	9.78E-07	NA	ND
Toluene	0.004	1	250	7	70	25550	3.91E-06	NA	ND
Aluminum	24.3	1	250	7	70	25550	2.38E-02	NA	ND
Arsenic	0.009	1	250	7	70	25550	8.81E-06	1.50E+00	1.32E-05
Barium	0.57	1	250	7	70	25550	5.58E-04	NA	ND
Beryllium	0.003	1	250	7	70	25550	2.94E-06	4.30E+00	1.26E-05
Boron	0.54	1	250	7	70	25550	5.28E-04	NA	ND
Calcium	1700	1	250	7	70	25550	1.66E+00	NA	ND
Chromium	0.047	1	250	7	70	25550	4.60E-05	NA	ND
Iron	53	1	250	7	70	25550	5.19E-02	NA	ND
Lead	0.06	1	250	7	70	25550	5.87E-05	NA	ND
Magnesium	920	1	250	7	70	25550	9.00E-01	NA	ND
Manganese	1.7	1	250	7	70	25550	1.66E-03	NA	ND
Mercury	0.00007	1	250	7	70	25550	6.85E-08	NA	ND
Nickel	0.05	1	250	7	70	25550	4.89E-05	NA	ND
Potassium	154	1	250	7	70	25550	1.51E-01	NA	ND
Sodium	9800	1	250	7	70	25550	9.59E+00	NA	ND
Thallium	0.12	1	250	7	70	25550	1.17E-04	NA	ND
Vanadium	0.04	1	250	7	70	25550	3.91E-05	NA	ND
								TOTAL RISK	2.59E-05

NA/ND -- Not Available/Not Detected

INGESTION OF GROUNDWATER – FUTURE OCCUPATIONAL ADULT SHORT TERM HAZARD FOR LBAD NORTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	SUBCHRONIC RfD (MG/KG/DAY)	HAZARD
Acetone	0.1	1	250	7	70	2555	9.78E−04	1.00E+00	9.78E−04
Benzene	0.002	1	250	7	70	2555	1.96E−05	NA	ND
Chloromethane	0.003	1	250	7	70	2555	2.94E−05	NA	ND
1,3−Dimethylbenzene	0.001	1	250	7	70	2555	9.78E−06	NA	ND
Toluene	0.004	1	250	7	70	2555	3.91E−05	2.00E+00	1.96E−05
Aluminum	24.3	1	250	7	70	2555	2.38E−01	NA	ND
Arsenic	0.009	1	250	7	70	2555	8.81E−05	3.00E−04	2.94E−01
Barium	0.57	1	250	7	70	2555	5.58E−03	7.00E−02	7.97E−02
Beryllium	0.003	1	250	7	70	2555	2.94E−05	5.00E−03	5.87E−03
Boron	0.54	1	250	7	70	2555	5.28E−03	9.00E−02	5.87E−02
Calcium	1700	1	250	7	70	2555	1.66E+01	NA	ND
Chromium	0.047	1	250	7	70	2555	4.60E−04	1.00E+00	4.60E−04
Iron	53	1	250	7	70	2555	5.19E−01	NA	ND
Lead	0.06	1	250	7	70	2555	5.87E−04	1.40E−03	4.19E−01
Magnesium	920	1	250	7	70	2555	9.00E+00	NA	ND
Manganese	1.7	1	250	7	70	2555	1.66E−02	5.00E−03	3.33E+00
Mercury	0.00007	1	250	7	70	2555	6.85E−07	3.00E−04	2.28E−03
Nickel	0.05	1	250	7	70	2555	4.89E−04	2.00E−02	2.45E−02
Potassium	154	1	250	7	70	2555	1.51E+00	NA	ND
Sodium	9800	1	250	7	70	2555	9.59E+01	NA	ND
Thallium	0.12	1	250	7	70	2555	1.17E−03	8.00E−04	1.47E+00
Vanadium	0.04	1	250	7	70	2555	3.91E−04	7.00E−03	5.59E−02
								TOTAL HAZARD	5.74E+00

Not Available/Not Detected

INGESTION OF GROUNDWATER – FUTURE OCCUPATIONAL ADULT LONG TERM RISK FOR LBAD NORTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)–1	RISK
Acetone	1.00E–01	1	250	25	70	25550	3.49E–04	NA	ND
Benzene	2.00E–03	1	250	25	70	25550	6.99E–06	2.90E–02	2.03E–07
Chloromethane	3.00E–03	1	250	25	70	25550	1.05E–05	1.30E–02	1.36E–07
1,3–Dimethylbenzene	1.00E–03	1	250	25	70	25550	3.49E–06	NA	ND
Toluene	4.00E–03	1	250	25	70	25550	1.40E–05	NA	ND
Aluminum	2.43E+01	1	250	25	70	25550	8.49E–02	NA	ND
Arsenic	9.00E–03	1	250	25	70	25550	3.15E–05	1.50E+00	4.72E–05
Barium	5.70E–01	1	250	25	70	25550	1.99E–03	NA	ND
Beryllium	3.00E–03	1	250	25	70	25550	1.05E–05	4.30E+00	4.51E–05
Boron	5.40E–01	1	250	25	70	25550	1.89E–03	NA	ND
Calcium	1.70E+03	1	250	25	70	25550	5.94E+00	NA	ND
Chromium	4.70E–02	1	250	25	70	25550	1.64E–04	NA	ND
Iron	5.30E+01	1	250	25	70	25550	1.85E–01	NA	ND
Lead	6.00E–02	1	250	25	70	25550	2.10E–04	NA	ND
Magnesium	9.20E+02	1	250	25	70	25550	3.21E+00	NA	ND
Manganese	1.70E+00	1	250	25	70	25550	5.94E–03	NA	ND
Mercury	7.00E–05	1	250	25	70	25550	2.45E–07	NA	ND
Nickel	5.00E–02	1	250	25	70	25550	1.75E–04	NA	ND
Potassium	1.54E+02	1	250	25	70	25550	5.38E–01	NA	ND
Sodium	9.80E+03	1	250	25	70	25550	3.42E+01	NA	ND
Thallium	1.20E–01	1	250	25	70	25550	4.19E–04	NA	ND
Vanadium	4.00E–02	1	250	25	70	25550	1.40E–04	NA	ND
								TOTAL RISK	9.26E–05

NA/ND – Not Available/Not Detected

INGESTION OF GROUNDWATER – FUTURE OCCUPATIONAL ADULT LONG TERM HAZARD FOR LBAD NORTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	RfD (MG/KG/DAY)	HAZARD
Acetone	1.00E−01	1	250	25	70	9125	9.78E−04	1.00E−01	9.78E−03
Benzene	2.00E−03	1	250	25	70	9125	1.96E−05	3.00E−04	ND
Chloromethane	3.00E−03	1	250	25	70	9125	2.94E−05	NA	ND
1,3−Dimethylbenzene	1.00E−03	1	250	25	70	9125	9.78E−06	2.00E+00	4.89E−06
Toluene	4.00E−03	1	250	25	70	9125	3.91E−05	2.00E−01	1.96E−04
Aluminum	2.43E+01	1	250	25	70	9125	2.38E−01	1.00E+00	ND
Arsenic	9.00E−03	1	250	25	70	9125	8.81E−05	3.00E−04	2.94E−01
Barium	5.70E−01	1	250	25	70	9125	5.58E−03	7.00E−02	7.97E−02
Beryllium	3.00E−03	1	250	25	70	9125	2.94E−05	5.00E−03	5.87E−03
Boron	5.40E−01	1	250	25	70	9125	5.28E−03	9.00E−02	5.87E−02
Calcium	1.70E+03	1	250	25	70	9125	1.66E+01	NA	ND
Chromium	4.70E−02	1	250	25	70	9125	4.60E−04	1.00E+00	4.60E−04
Iron	5.30E+01	1	250	25	70	9125	5.19E−01	NA	ND
Lead	6.00E−02	1	250	25	70	9125	5.87E−04	1.40E−03	4.19E−01
Magnesium	9.20E+02	1	250	25	70	9125	9.00E+00	NA	ND
Manganese	1.70E+00	1	250	25	70	9125	1.66E−02	5.00E−03	3.33E+00
Mercury	7.00E−05	1	250	25	70	9125	6.85E−07	3.00E−04	2.28E−03
Nickel	5.00E−02	1	250	25	70	9125	4.89E−04	2.00E−02	2.45E−02
Potassium	1.54E+02	1	250	25	70	9125	1.51E+00	5.00E+01	ND
Sodium	9.80E+03	1	250	25	70	9125	9.59E+01	3.40E+01	ND
Thallium	1.20E−01	1	250	25	70	9125	1.17E−03	8.00E−05	1.47E+01
Vanadium	4.00E−02	1	250	25	70	9125	3.91E−04	7.00E−03	5.59E−02
								TOTAL HAZARD	1.90E+01

N/ Not Available/Not Detected

INGESTION OF GROUNDWATER – FUTURE RESIDENTIAL ADULT SHORT TERM RISK FOR LBAD SOUTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)−1	RISK
Acetone	0.03	2	350	7	70	25550	8.22E−05 NA		ND
Benzene	0.001	2	350	7	70	25550	2.74E−06	2.90E−02	7.95E−08
Carbon tetrachloride	0.0005	2	350	7	70	25550	1.37E−06	1.30E−01	1.78E−07
1,1−Dichloroethane	0.0009	2	350	7	70	25550	2.47E−06 NA		ND
1,2−Dichloroethenes	0.003	2	350	7	70	25550	8.22E−06 NA		ND
1,3−Dimethylbenzene	0.003	2	350	7	70	25550	8.22E−06 NA		ND
Ethylbenzene	0.001	2	350	7	70	25550	2.74E−06 NA		ND
Methyl isobutyl ketone	0.0008	2	350	7	70	25550	2.19E−06 NA		ND
Tetrachloroethene	0.0005	2	350	7	70	25550	1.37E−06	5.20E−02	7.12E−08
Toluene	0.006	2	350	7	70	25550	1.64E−05 NA		ND
Trichloroethene	0.0007	2	350	7	70	25550	1.92E−06	1.10E−02	2.11E−08
Vinyl chloride	0.009	2	350	7	70	25550	2.47E−05	1.90E+00	4.68E−05
Xylenes	0.006	2	350	7	70	25550	1.64E−05 NA		ND
alpha−BHC	0.000003	2	350	7	70	25550	8.22E−09	6.30E+00	5.18E−08
delta−BHC	0.000002	2	350	7	70	25550	5.48E−09 NA		ND
Bis(2−ethylhexyl)phthalate	0.004	2	350	7	70	25550	1.10E−05	1.40E−02	1.53E−07
DDT	0.000008	2	350	7	70	25550	2.19E−08	3.40E−01	7.45E−09
2,4−Dimethylphenol	0.003	2	350	7	70	25550	8.22E−06 NA		ND
alpha−Endosulfan	0.000003	2	350	7	70	25550	8.22E−09 NA		ND
Lindane	0.000002	2	350	7	70	25550	5.48E−09	1.30E+00	7.12E−09
Phenol	0.0016	2	350	7	70	25550	4.38E−06 NA		ND
Aluminum	125	2	350	7	70	25550	3.42E−01 NA		ND
Antimony	0.04	2	350	7	70	25550	1.10E−04 NA		ND
Arsenic	0.003	2	350	7	70	25550	8.22E−06	1.50E+00	1.23E−05
Barium	0.4	2	350	7	70	25550	1.10E−03 NA		ND
Beryllium	0.006	2	350	7	70	25550	1.64E−05	4.30E+00	7.07E−05
Boron	0.91	2	350	7	70	25550	2.49E−03 NA		ND
Cadmium	0.01	2	350	7	70	25550	2.74E−05 NA		ND
Calcium	1025	2	350	7	70	25550	2.81E+00 NA		ND
Chromium	0.07	2	350	7	70	25550	1.92E−04 NA		ND
Cobalt	0.03	2	350	7	70	25550	8.22E−05 NA		ND
Copper	0.04	2	350	7	70	25550	1.10E−04 NA		ND
Iron	177	2	350	7	70	25550	4.85E−01 NA		ND
Lead	0.04	2	350	7	70	25550	1.10E−04 NA		ND
Magnesium	138	2	350	7	70	25550	3.78E−01 NA		ND
Manganese	8.5	2	350	7	70	25550	2.33E−02 NA		ND
Mercury	0.0001	2	350	7	70	25550	2.74E−07 NA		ND
Molybdenum	0.03	2	350	7	70	25550	8.22E−05 NA		ND
Nickel	0.05	2	350	7	70	25550	1.37E−04 NA		ND
Potassium	22	2	350	7	70	25550	6.03E−02 NA		ND
Sodium	823	2	350	7	70	25550	2.25E+00 NA		ND
Tellurium	0.07	2	350	7	70	25550	1.92E−04 NA		ND
Thallium	0.08	2	350	7	70	25550	2.19E−04 NA		ND
Tin	0.05	2	350	7	70	25550	1.37E−04 NA		ND
Vanadium	0.05	2	350	7	70	25550	1.37E−04 NA		ND
Zinc	0.44	2	350	7	70	25550	1.21E−03 NA		ND
								TOTAL RISK	1.30E−04

NA/ND – Not Available/Not Detected

INGESTION OF GROUNDWATER – FUTURE RESIDENTIAL ADULT SHORT TERM HAZARD FOR LBAD SOUTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	SUBCHRONIC RfD (MG/KG/DAY)	HAZARD
Acetone	0.03	2	350	7	70	2555	8.22E−04	1.00E+00	8.22E−04
Benzene	0.001	2	350	7	70	2555	2.74E−05 NA		ND
Carbon tetrachloride	0.0005	2	350	7	70	2555	1.37E−05	2.00E−03	6.85E−03
1,1−Dichloroethane	0.0009	2	350	7	70	2555	2.47E−05	1.00E+00	2.47E−05
1,2−Dichloroethenes	0.003	2	350	7	70	2555	8.22E−05	9.00E−03	9.13E−03
1,3−Dimethylbenzene	0.003	2	350	7	70	2555	8.22E−05 NA		ND
Ethylbenzene	0.001	2	350	7	70	2555	2.74E−05	1.00E−01	2.74E−04
Methyl isobutyl ketone	0.0008	2	350	7	70	2555	2.19E−05	8.00E−01	2.74E−05
Tetrachloroethene	0.0005	2	350	7	70	2555	1.37E−05	1.00E−01	1.37E−04
Toluene	0.006	2	350	7	70	2555	1.64E−04	2.00E+00	8.22E−05
Trichloroethene	0.0007	2	350	7	70	2555	1.92E−05 NA		ND
Vinyl chloride	0.009	2	350	7	70	2555	2.47E−04 NA		ND
Xylenes	0.006	2	350	7	70	2555	1.64E−04 NA		ND
alpha−BHC	0.000003	2	350	7	70	2555	8.22E−08 NA		ND
delta−BHC	0.000002	2	350	7	70	2555	5.48E−08 NA		ND
Bis(2−ethylhexyl)phthalate	0.004	2	350	7	70	2555	1.10E−04 NA		ND
DDT	0.000008	2	350	7	70	2555	2.19E−07	5.00E−04	4.38E−04
2,4−Dimethylphenol	0.003	2	350	7	70	2555	8.22E−05	2.00E−01	4.11E−04
alpha−Endosulfan	0.000003	2	350	7	70	2555	8.22E−08	6.00E−03	1.37E−05
Lindane	0.000002	2	350	7	70	2555	5.48E−08	3.00E−03	1.83E−05
Phenol	0.0016	2	350	7	70	2555	4.38E−05	6.00E−01	7.31E−05
Aluminum	125	2	350	7	70	2555	3.42E+00 NA		ND
Antimony	0.04	2	350	7	70	2555	1.10E−03	4.00E−04	2.74E+00
Arsenic	0.003	2	350	7	70	2555	8.22E−05	3.00E−04	2.74E−01
Barium	0.4	2	350	7	70	2555	1.10E−02	7.00E−02	1.57E−01
Beryllium	0.006	2	350	7	70	2555	1.64E−04	5.00E−03	3.29E−02
Boron	0.91	2	350	7	70	2555	2.49E−02	9.00E−02	2.77E−01
Cadmium	0.01	2	350	7	70	2555	2.74E−04 NA		ND
Calcium	1025	2	350	7	70	2555	2.81E+01 NA		ND
Chromium	0.07	2	350	7	70	2555	1.92E−03	1.00E+00	1.92E−03
Cobalt	0.03	2	350	7	70	2555	8.22E−04 NA		ND
Copper	0.04	2	350	7	70	2555	1.10E−03 NA		ND
Iron	177	2	350	7	70	2555	4.85E+00 NA		ND
Lead	0.04	2	350	7	70	2555	1.10E−03	1.40E−03	7.83E−01
Magnesium	138	2	350	7	70	2555	3.78E+00 NA		ND
Manganese	8.5	2	350	7	70	2555	2.33E−01	5.00E−03	4.66E+01
Mercury	0.0001	2	350	7	70	2555	2.74E−06	3.00E−04	9.13E−03
Molybdenum	0.03	2	350	7	70	2555	8.22E−04	5.00E−03	1.64E−01
Nickel	0.05	2	350	7	70	2555	1.37E−03	2.00E−02	6.85E−02
Potassium	22	2	350	7	70	2555	6.03E−01 NA		ND
Sodium	823	2	350	7	70	2555	2.25E+01 NA		ND
Tellurium	0.07	2	350	7	70	2555	1.92E−03 NA		ND
Thallium	0.08	2	350	7	70	2555	2.19E−03	8.00E−04	2.74E+00
Tin	0.05	2	350	7	70	2555	1.37E−03	6.00E−01	2.28E−03
Vanadium	0.05	2	350	7	70	2555	1.37E−03	7.00E−03	1.96E−01
Zinc	0.44	2	350	7	70	2555	1.21E−02	3.00E−01	4.02E−02
								TOTAL HAZARD	5.41E+01

NA Not Available/Not Detected

DERMAL CONTACT WITH GROUNDWATER – FUTURE RESIDENTIAL ADULT SHORT TERM RISK FOR LBAD SOUTH

ANALYTE	CW (MG/L)	SA (CM2)	PC (CM/HR)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	CF (L/CM3)	BW (KG)	AT (DAYS)	ABSORBED INTAKE (MG/KG/DAY)	ADJUSTED CSF (MG/KG/DAY)−1	RISK
Acetone	0.03	19400	5.7E−04	0.17	350	7	1.0E−03	70	25550	7.73E−08	NA	ND
Benzene	0.001	19400	1.1E−01	0.17	350	7	1.0E−03	70	25550	4.97E−07	3.22E−02	1.60E−08
Carbon tetrachloride	0.0005	19400	2.2E−02	0.17	350	7	1.0E−03	70	25550	4.97E−08	1.51E−01	7.51E−09
1,1−Dichloroethane	0.0009	19400	8.9E−03	0.17	350	7	1.0E−03	70	25550	3.62E−08	NA	ND
1,2−Dichloroethenes	0.003	19400	1.0E−02	0.17	350	7	1.0E−03	70	25550	1.36E−07	NA	ND
1,3−Dimethylbenzene	0.003	19400	8.0E−02	0.17	350	7	1.0E−03	70	25550	1.08E−06	NA	ND
Ethylbenzene	0.001	19400	1.0E+00	0.17	350	7	1.0E−03	70	25550	4.52E−06	NA	ND
Methyl isobutyl ketone	0.0008	19400	3.3E−03	0.17	350	7	1.0E−03	70	25550	1.18E−08	NA	ND
Tetrachloroethene	0.0005	19400	3.7E−01	0.17	350	7	1.0E−03	70	25550	8.36E−07	5.20E−02	4.35E−08
Toluene	0.006	19400	1.0E+00	0.17	350	7	1.0E−03	70	25550	2.71E−05	NA	ND
Trichloroethene	0.0007	19400	2.3E−01	0.17	350	7	1.0E−03	70	25550	7.27E−07	1.10E−02	8.00E−09
Vinyl chloride	0.009	19400	7.3E−03	0.17	350	7	1.0E−03	70	25550	2.97E−07	2.37E+00	7.05E−07
Xylenes	0.006	19400	8.0E−02	0.17	350	7	1.0E−03	70	25550	2.17E−06	NA	ND
alpha−BHC	0.000003	19400	1.6E−02	0.17	350	7	1.0E−03	70	25550	2.17E−10	7.00E+00	1.52E−09
delta−BHC	0.000002	19400	1.6E−02	0.17	350	7	1.0E−03	70	25550	1.45E−10	NA	ND
Bis(2−ethylhexyl)phthalate	0.004	19400	3.3E−02	0.17	350	7	1.0E−03	70	25550	5.96E−07	5.60E−02	3.34E−08
DDT	0.000008	19400	4.3E−01	0.17	350	7	1.0E−03	70	25550	1.55E−08	3.78E−01	5.87E−09
2,4−Dimethylphenol	0.003	19400	1.1E−01	0.17	350	7	1.0E−03	70	25550	1.49E−06	NA	ND
alpha−Endosulfan	0.000003	19400	3.3E−03	0.17	350	7	1.0E−03	70	25550	4.47E−11	NA	ND
Lindane	0.000002	19400	1.4E−02	0.17	350	7	1.0E−03	70	25550	1.26E−10	1.31E+00	1.66E−10
Phenol	0.0016	19400	8.2E−03	0.17	350	7	1.0E−03	70	25550	5.93E−08	NA	ND
Aluminum	125	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	5.65E−04	NA	ND
Antimony	0.04	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	1.81E−07	NA	ND
Arsenic	0.003	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	1.36E−08	1.58E+00	2.14E−08
Barium	0.4	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	1.81E−06	NA	ND
Beryllium	0.006	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	2.71E−08	4.30E+01	1.17E−06
Boron	0.91	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	4.11E−06	NA	ND
Cadmium	0.01	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	4.52E−08	NA	ND
Calcium	1025	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	4.63E−03	NA	ND
Chromium	0.07	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	3.16E−07	NA	ND
Cobalt	0.03	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	1.36E−07	NA	ND
Copper	0.04	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	1.81E−07	NA	ND
Iron	177	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	8.00E−04	NA	ND
Lead	0.04	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	1.81E−07	NA	ND
Magnesium	138	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	6.23E−04	NA	ND
Manganese	8.5	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	3.84E−05	NA	ND
Mercury	0.0001	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	4.52E−10	NA	ND
Molybdenum	0.03	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	1.36E−07	NA	ND
Nickel	0.05	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	2.26E−07	NA	ND
Potassium	22	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	9.94E−05	NA	ND
Sodium	823	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	3.72E−03	NA	ND
Tellurium	0.07	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	3.16E−07	NA	ND
Thallium	0.08	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	3.61E−07	NA	ND
Tin	0.05	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	2.26E−07	NA	ND
Vanadium	0.05	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	2.26E−07	NA	ND
Zinc	0.44	19400	1.0E−03	0.17	350	7	1.0E−03	70	25550	1.99E−06	NA	ND
											TOTAL RISK	2.01E−06

NA/ND – Not Available/Not Determined

DERMAL CONTACT WITH GROUNDWATER – FUTURE RESIDENTIAL ADULT SHORT TERM HAZARD FOR LBAD SOUTH

ANALYTE	CW (MG/L)	SA (CM2)	PC (CM/HR)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	CF (L/CM3)	BW (KG)	AT (DAYS)	ABSORBED INTAKE (MG/KG/DAY)	ADJUSTED RfD (MG/KG/DAY)	HAZARD
Acetone	0.03	19400	5.70E−04	0.17	350	7	1.0E−03	70	2555	7.73E−07	1.00E+00	7.73E−07
Benzene	0.001	19400	1.10E−01	0.17	350	7	1.0E−03	70	2555	4.97E−06 NA		ND
Carbon tetrachloride	0.0005	19400	2.20E−02	0.17	350	7	1.0E−03	70	2555	4.97E−07	1.72E−03	2.89E−04
1,1−Dichloroethane	0.0009	19400	8.90E−03	0.17	350	7	1.0E−03	70	2555	3.62E−07	1.00E+00	3.62E−07
1,2−Dichloroethenes	0.003	19400	1.00E−02	0.17	350	7	1.0E−03	70	2555	1.36E−06	8.10E−03	1.67E−04
1,3−Dimethylbenzene	0.003	19400	8.00E−02	0.17	350	7	1.0E−03	70	2555	1.08E−05 NA		ND
Ethylbenzene	0.001	19400	1.00E+00	0.17	350	7	1.0E−03	70	2555	4.52E−05	9.00E−02	5.02E−04
Methyl isobutyl ketone	0.0008	19400	3.26E−03	0.17	350	7	1.0E−03	70	2555	1.18E−07	6.40E−01	1.84E−07
Tetrachloroethene	0.0005	19400	3.70E−01	0.17	350	7	1.0E−03	70	2555	8.36E−06	1.00E−01	8.36E−05
Toluene	0.006	19400	1.00E+00	0.17	350	7	1.0E−03	70	2555	2.71E−04	2.00E+00	1.36E−04
Trichloroethene	0.0007	19400	2.30E−01	0.17	350	7	1.0E−03	70	2555	7.27E−06 NA		ND
Vinyl chloride	0.009	19400	7.30E−03	0.17	350	7	1.0E−03	70	2555	2.97E−06 NA		ND
Xylenes	0.006	19400	8.00E−02	0.17	350	7	1.0E−03	70	2555	2.17E−05 NA		ND
alpha−BHC	0.000003	19400	1.60E−02	0.17	350	7	1.0E−03	70	2555	2.17E−09 NA		ND
delta−BHC	0.000002	19400	1.60E−02	0.17	350	7	1.0E−03	70	2555	1.45E−09 NA		ND
Bis(2−ethylhexyl)phthalate	0.004	19400	3.30E−02	0.17	350	7	1.0E−03	70	2555	5.96E−06 NA		ND
DDT	0.000008	19400	4.30E−01	0.17	350	7	1.0E−03	70	2555	1.55E−07	4.50E−04	3.45E−04
2,4−Dimethylphenol	0.003	19400	1.10E−01	0.17	350	7	1.0E−03	70	2555	1.49E−05	1.30E−01	1.15E−04
alpha−Endosulfan	0.000003	19400	3.30E−03	0.17	350	7	1.0E−03	70	2555	4.47E−10	4.80E−03	9.32E−08
Lindane	0.000002	19400	1.40E−02	0.17	350	7	1.0E−03	70	2555	1.26E−09	2.97E−03	4.26E−07
Phenol	0.0016	19400	8.20E−03	0.17	350	7	1.0E−03	70	2555	5.93E−07	5.40E−01	1.10E−06
Aluminum	125	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	5.65E−03 NA		ND
Antimony	0.04	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	1.81E−06	2.40E−04	7.53E−03
Arsenic	0.003	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	1.36E−07	2.85E−04	4.76E−04
Barium	0.4	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	1.81E−05	3.50E−03	5.16E−03
Beryllium	0.006	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	2.71E−07	5.00E−04	5.42E−04
Boron	0.91	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	4.11E−05	9.00E−02	4.57E−04
Cadmium	0.01	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	4.52E−07 NA		ND
Calcium	1025	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	4.63E−02 NA		ND
Chromium	0.07	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	3.16E−06	3.00E−02	1.05E−04
Cobalt	0.03	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	1.36E−06 NA		ND
Copper	0.04	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	1.81E−06 NA		ND
Iron	177	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	8.00E−03 NA		ND
Lead	0.04	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	1.81E−06	7.00E−04	2.58E−03
Magnesium	138	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	6.23E−03 NA		ND
Manganese	8.5	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	3.84E−04	2.00E−04	1.92E+00
Mercury	0.0001	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	4.52E−09	4.50E−05	1.00E−04
Molybdenum	0.03	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	1.36E−06	1.00E−03	1.36E−03
Nickel	0.05	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	2.26E−06	2.00E−03	1.13E−03
Potassium	22	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	9.94E−04 NA		ND
Sodium	823	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	3.72E−02 NA		ND
Tellurium	0.07	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	3.16E−06 NA		ND
Thallium	0.08	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	3.61E−06	8.00E−05	4.52E−02
Tin	0.05	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	2.26E−06	1.80E−02	1.25E−04
Vanadium	0.05	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	2.26E−06	7.00E−05	3.23E−02
Zinc	0.44	19400	1.00E−03	0.17	350	7	1.0E−03	70	2555	1.99E−05	9.00E−02	2.21E−04
											TOTAL HAZARD	2.02E+00

NA = Not Available/Not Determined

INHALATION OF VAPORS WHILE SHOWERING – FUTURE RESIDENTIAL ADULT SHORT TERM RISK FOR LBAD SOUTH

ANALYTE	Ei (MG)	SF (SHOWERS/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)–1	RISK
Acetone	0.052	1.0	350	7	70	25550	7.11E–05	NA	ND
Benzene	0.002	1.0	350	7	70	25550	2.91E–06	2.90E–02	8.45E–08
Carbon tetrachloride	0.001	1.0	350	7	70	25550	1.22E–06	5.30E–02	6.45E–08
1,1–Dichloroethane	0.002	1.0	350	7	70	25550	2.15E–06	NA	ND
1,2–Dichloroethenes	0.005	1.0	350	7	70	25550	7.36E–06	NA	ND
1,3–Dimethylbenzene	0.004	1.0	350	7	70	25550	5.88E–06	NA	ND
Ethylbenzene	0.002	1.0	350	7	70	25550	3.21E–06	NA	ND
Methyl isobutyl ketone	0.001	1.0	350	7	70	25550	1.92E–06	NA	ND
Tetrachloroethene	0.001	1.0	350	7	70	25550	1.22E–06	2.00E–03	2.43E–09
Toluene	0.010	1.0	350	7	70	25550	1.31E–05	NA	ND
Trichloroethene	0.001	1.0	350	7	70	25550	1.65E–06	6.00E–03	9.92E–09
Vinyl chloride	0.015	1.0	350	7	70	25550	2.00E–05	3.00E–01	6.01E–06
Xylenes	0.010	1.0	350	7	70	25550	1.33E–05	NA	ND
								TOTAL RISK	**6.17E–06**

INHALATION OF VAPORS WHILE SHOWERING – FUTURE RESIDENTIAL ADULT SHORT TERM HAZARD FOR LBAD SOUTH

ANALYTE	Ei (MG)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	SUBCHRONIC RfD (MG/KG/DAY)	HAZARD
Acetone	0.052	1.0	350	7	70	2555	7.11E–04	NA	ND
Benzene	0.002	1.0	350	7	70	2555	2.91E–05	NA	ND
Carbon tetrachloride	0.001	1.0	350	7	70	2555	1.22E–05	1.70E–02	7.16E–04
1,1–Dichloroethane	0.002	1.0	350	7	70	2555	2.15E–05	1.00E+00	2.15E–05
1,2–Dichloroethenes	0.005	1.0	350	7	70	2555	7.36E–05	NA	ND
1,3–Dimethylbenzene	0.004	1.0	350	7	70	2555	5.88E–05	NA	ND
Ethylbenzene	0.002	1.0	350	7	70	2555	3.21E–05	3.00E–01	1.07E–04
Methyl isobutyl ketone	0.001	1.0	350	7	70	2555	1.92E–05	2.00E–01	9.60E–05
Tetrachloroethene	0.001	1.0	350	7	70	2555	1.22E–05	NA	ND
Toluene	0.010	1.0	350	7	70	2555	1.31E–04	3.00E–01	4.36E–04
Trichloroethene	0.001	1.0	350	7	70	2555	1.65E–05	NA	ND
Vinyl chloride	0.015	1.0	350	7	70	2555	2.00E–04	NA	ND
Xylenes	0.010	1.0	350	7	70	2555	1.33E–04	NA	ND
								TOTAL HAZARD	**1.38E–03**

NA/ND – Not Applicable/Not Determined

INGESTION OF GROUNDWATER – FUTURE RESIDENTIAL ADULT LONG TERM RISK FOR LBAD SOUTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)−1	RISK
Acetone	0.03	2	350	30	70	25550	3.52E−04 NA		ND
Benzene	0.001	2	350	30	70	25550	1.17E−05	2.90E−02	3.41E−07
Carbon tetrachloride	0.0005	2	350	30	70	25550	5.87E−06	1.30E−01	7.63E−07
1,1−Dichloroethane	0.0009	2	350	30	70	25550	1.06E−05 NA		ND
1,2−Dichloroethenes	0.003	2	350	30	70	25550	3.52E−05 NA		ND
1,3−Dimethylbenzene	0.003	2	350	30	70	25550	3.52E−05 NA		ND
Ethylbenzene	0.001	2	350	30	70	25550	1.17E−05 NA		ND
Methyl isobutyl ketone	0.0008	2	350	30	70	25550	9.39E−06 NA		ND
Tetrachloroethene	0.0005	2	350	30	70	25550	5.87E−06	5.20E−02	3.05E−07
Toluene	0.006	2	350	30	70	25550	7.05E−05 NA		ND
Trichloroethene	0.0007	2	350	30	70	25550	8.22E−06	1.10E−02	9.04E−08
Vinyl chloride	0.009	2	350	30	70	25550	1.06E−04	1.90E+00	2.01E−04
Xylenes	0.006	2	350	30	70	25550	7.05E−05 NA		ND
alpha−BHC	0.000003	2	350	30	70	25550	3.52E−08	6.30E+00	2.22E−07
delta−BHC	0.000002	2	350	30	70	25550	2.35E−08 NA		ND
Bis(2−ethylhexyl)phthalate	0.004	2	350	30	70	25550	4.70E−05	1.40E−02	6.58E−07
DDT	0.000008	2	350	30	70	25550	9.39E−08	3.40E−01	3.19E−08
2,4−Dimethylphenol	0.003	2	350	30	70	25550	3.52E−05 NA		ND
alpha−Endosulfan	0.000003	2	350	30	70	25550	3.52E−08 NA		ND
Lindane	0.000002	2	350	30	70	25550	2.35E−08	1.30E+00	3.05E−08
Phenol	0.0016	2	350	30	70	25550	1.88E−05 NA		ND
Aluminum	125	2	350	30	70	25550	1.47E+00 NA		ND
Antimony	0.04	2	350	30	70	25550	4.70E−04 NA		ND
Arsenic	0.003	2	350	30	70	25550	3.52E−05	1.50E+00	5.28E−05
Barium	0.4	2	350	30	70	25550	4.70E−03 NA		ND
Beryllium	0.006	2	350	30	70	25550	7.05E−05	4.30E+00	3.03E−04
Boron	0.91	2	350	30	70	25550	1.07E−02 NA		ND
Cadmium	0.01	2	350	30	70	25550	1.17E−04 NA		ND
Calcium	1025	2	350	30	70	25550	1.20E+01 NA		ND
Chromium	0.07	2	350	30	70	25550	8.22E−04 NA		ND
Cobalt	0.03	2	350	30	70	25550	3.52E−04 NA		ND
Copper	0.04	2	350	30	70	25550	4.70E−04 NA		ND
Iron	177	2	350	30	70	25550	2.08E+00 NA		ND
Lead	0.04	2	350	30	70	25550	4.70E−04 NA		ND
Magnesium	138	2	350	30	70	25550	1.62E+00 NA		ND
Manganese	8.5	2	350	30	70	25550	9.98E−02 NA		ND
Mercury	0.0001	2	350	30	70	25550	1.17E−06 NA		ND
Molybdenum	0.03	2	350	30	70	25550	3.52E−04 NA		ND
Nickel	0.05	2	350	30	70	25550	5.87E−04 NA		ND
Potassium	22	2	350	30	70	25550	2.58E−01 NA		ND
Sodium	823	2	350	30	70	25550	9.66E+00 NA		ND
Tellurium	0.07	2	350	30	70	25550	8.22E−04 NA		ND
Thallium	0.08	2	350	30	70	25550	9.39E−04 NA		ND
Tin	0.05	2	350	30	70	25550	5.87E−04 NA		ND
Vanadium	0.05	2	350	30	70	25550	5.87E−04 NA		ND
Zinc	0.44	2	350	30	70	25550	5.17E−03 NA		ND
								TOTAL RISK	5.59E−04

N = Not Available/Not Detected

INGESTION OF GROUNDWATER – FUTURE RESIDENTIAL ADULT LONG TERM HAZARD FOR LBAD SOUTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	RfD (MG/KG/DAY)	HAZARD
Acetone	0.03	2	350	30	70	10950	8.22E-04	1.00E-01	8.22E-03
Benzene	0.001	2	350	30	70	10950	2.74E-05	3.00E-04	9.13E-02
Carbon tetrachloride	0.0005	2	350	30	70	10950	1.37E-05	7.00E-04	1.96E-02
1,1-Dichloroethane	0.0009	2	350	30	70	10950	2.47E-05	1.00E-01	2.47E-04
1,2-Dichloroethenes	0.003	2	350	30	70	10950	8.22E-05	9.00E-03	9.13E-03
1,3-Dimethylbenzene	0.003	2	350	30	70	10950	8.22E-05	2.00E+00	4.11E-05
Ethylbenzene	0.001	2	350	30	70	10950	2.74E-05	1.00E-01	2.74E-04
Methyl isobutyl ketone	0.0008	2	350	30	70	10950	2.19E-05	8.00E-02	2.74E-04
Tetrachloroethene	0.0005	2	350	30	70	10950	1.37E-05	1.00E-02	1.37E-03
Toluene	0.006	2	350	30	70	10950	1.64E-04	2.00E-01	8.22E-04
Trichloroethene	0.0007	2	350	30	70	10950	1.92E-05	NA	ND
Vinyl chloride	0.009	2	350	30	70	10950	2.47E-04	NA	ND
Xylenes	0.006	2	350	30	70	10950	1.64E-04	2.00E+00	8.22E-05
alpha-BHC	0.000003	2	350	30	70	10950	8.22E-08	NA	ND
delta-BHC	0.000002	2	350	30	70	10950	5.48E-08	NA	ND
Bis(2-ethylhexyl)phthalate	0.004	2	350	30	70	10950	1.10E-04	2.00E-02	5.48E-03
DDT	0.000008	2	350	30	70	10950	2.19E-07	5.00E-04	4.38E-04
2,4-Dimethylphenol	0.003	2	350	30	70	10950	8.22E-05	2.00E-02	4.11E-03
alpha-Endosulfan	0.000003	2	350	30	70	10950	8.22E-08	6.00E-03	1.37E-05
Lindane	0.000002	2	350	30	70	10950	5.48E-08	3.00E-04	1.83E-04
Phenol	0.0016	2	350	30	70	10950	4.38E-05	6.00E-01	7.31E-05
Aluminum	125	2	350	30	70	10950	3.42E+00	1.00E+00	3.42E+00
Antimony	0.04	2	350	30	70	10950	1.10E-03	4.00E-04	2.74E+00
Arsenic	0.003	2	350	30	70	10950	8.22E-05	3.00E-04	2.74E-01
Barium	0.4	2	350	30	70	10950	1.10E-02	7.00E-02	1.57E-01
Beryllium	0.006	2	350	30	70	10950	1.64E-04	5.00E-03	3.29E-02
Boron	0.91	2	350	30	70	10950	2.49E-02	9.00E-02	2.77E-01
Cadmium	0.01	2	350	30	70	10950	2.74E-04	5.00E-04	5.48E-01
Calcium	1025	2	350	30	70	10950	2.81E+01	NA	ND
Chromium	0.07	2	350	30	70	10950	1.92E-03	1.00E+00	1.92E-03
Cobalt	0.03	2	350	30	70	10950	8.22E-04	NA	ND
Copper	0.04	2	350	30	70	10950	1.10E-03	4.00E-02	2.74E-02
Iron	177	2	350	30	70	10950	4.85E+00	NA	ND
Lead	0.04	2	350	30	70	10950	1.10E-03	1.40E-03	7.83E-01
Magnesium	138	2	350	30	70	10950	3.78E+00	NA	ND
Manganese	8.5	2	350	30	70	10950	2.33E-01	5.00E-03	4.66E+01
Mercury	0.0001	2	350	30	70	10950	2.74E-06	3.00E-04	9.13E-03
Molybdenum	0.03	2	350	30	70	10950	8.22E-04	5.00E-03	1.64E-01
Nickel	0.05	2	350	30	70	10950	1.37E-03	2.00E-02	6.85E-02
Potassium	22	2	350	30	70	10950	6.03E-01	5.00E+01	1.21E-02
Sodium	823	2	350	30	70	10950	2.25E+01	3.40E+01	6.63E-01
Tellurium	0.07	2	350	30	70	10950	1.92E-03	NA	ND
Thallium	0.08	2	350	30	70	10950	2.19E-03	8.00E-05	2.74E+01
Tin	0.05	2	350	30	70	10950	1.37E-03	6.00E-01	2.28E-03
Vanadium	0.05	2	350	30	70	10950	1.37E-03	7.00E-03	1.96E-01
Zinc	0.44	2	350	30	70	10950	1.21E-02	3.00E-01	4.02E-02
								TOTAL HAZARD	8.35E+01

NA/ND – Not Available/Not Detected

DERMAL CONTACT WITH GROUNDWATER – FUTURE RESIDENTIAL ADULT LONG TERM RISK FOR LBAD SOUTH

ANALYTE	CW (MG/L)	SA (CM2)	PC (CM/HR)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	CF (L/CM3)	BW (KG)	AT (DAYS)	ABSORBED INTAKE (MG/KG/DAY)	ADJUSTED CSF (MG/KG/DAY)-1	RISK
Acetone	0.03	19400	5.7E-04	0.17	350	30	1.0E-03	70	25550	3.31E-07 NA		ND
Benzene	0.001	19400	1.1E-01	0.17	350	30	1.0E-03	70	25550	2.13E-06	3.22E-02	6.86E-08
Carbon tetrachloride	0.0005	19400	2.2E-02	0.17	350	30	1.0E-03	70	25550	2.13E-07	1.51E-01	3.22E-08
1,1-Dichloroethane	0.0009	19400	8.9E-03	0.17	350	30	1.0E-03	70	25550	1.55E-07 NA		ND
1,2-Dichloroethenes	0.003	19400	1.0E-02	0.17	350	30	1.0E-03	70	25550	5.81E-07 NA		ND
1,3-Dimethylbenzene	0.003	19400	8.0E-02	0.17	350	30	1.0E-03	70	25550	4.65E-06 NA		ND
Ethylbenzene	0.001	19400	1.0E+00	0.17	350	30	1.0E-03	70	25550	1.94E-05 NA		ND
Methyl isobutyl ketone	0.0008	19400	3.3E-03	0.17	350	30	1.0E-03	70	25550	5.05E-08 NA		ND
Tetrachloroethene	0.0005	19400	3.7E-01	0.17	350	30	1.0E-03	70	25550	3.58E-06	5.20E-02	1.86E-07
Toluene	0.006	19400	1.0E+00	0.17	350	30	1.0E-03	70	25550	1.16E-04 NA		ND
Trichloroethene	0.0007	19400	2.3E-01	0.17	350	30	1.0E-03	70	25550	3.12E-06	1.10E-02	3.43E-08
Vinyl chloride	0.009	19400	7.3E-03	0.17	350	30	1.0E-03	70	25550	1.27E-06	2.37E+00	3.02E-06
Xylenes	0.006	19400	8.0E-02	0.17	350	30	1.0E-03	70	25550	9.29E-06 NA		ND
alpha-BHC	0.000003	19400	1.6E-02	0.17	350	30	1.0E-03	70	25550	9.29E-10	7.00E+00	6.51E-09
delta-BHC	0.000002	19400	1.6E-02	0.17	350	30	1.0E-03	70	25550	6.20E-10 NA		ND
Bis(2-ethylhexyl)phthalate	0.004	19400	3.3E-02	0.17	350	30	1.0E-03	70	25550	2.56E-06	5.60E-02	1.43E-07
DDT	0.000008	19400	4.3E-01	0.17	350	30	1.0E-03	70	25550	6.66E-08	3.78E-01	2.52E-08
2,4-Dimethylphenol	0.003	19400	1.1E-01	0.17	350	30	1.0E-03	70	25550	6.39E-06 NA		ND
alpha-Endosulfan	0.000003	19400	3.3E-03	0.17	350	30	1.0E-03	70	25550	1.92E-10 NA		ND
Lindane	0.000002	19400	1.4E-02	0.17	350	30	1.0E-03	70	25550	5.42E-10	1.31E+00	7.12E-10
Phenol	0.0016	19400	8.2E-03	0.17	350	30	1.0E-03	70	25550	2.54E-07 NA		ND
Aluminum	125	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	2.42E-03 NA		ND
Antimony	0.04	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	7.74E-07 NA		ND
Arsenic	0.003	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	5.81E-08	1.58E+00	9.17E-08
Barium	0.4	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	7.74E-06 NA		ND
Beryllium	0.006	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.16E-07	4.30E+01	5.00E-06
Boron	0.91	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.76E-05 NA		ND
Cadmium	0.01	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.94E-07 NA		ND
Calcium	1025	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.98E-02 NA		ND
Chromium	0.07	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.36E-06 NA		ND
Cobalt	0.03	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	5.81E-07 NA		ND
Copper	0.04	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	7.74E-07 NA		ND
Iron	177	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	3.43E-03 NA		ND
Lead	0.04	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	7.74E-07 NA		ND
Magnesium	138	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	2.67E-03 NA		ND
Manganese	8.5	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.65E-04 NA		ND
Mercury	0.0001	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.94E-09 NA		ND
Molybdenum	0.03	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	5.81E-07 NA		ND
Nickel	0.05	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	9.68E-07 NA		ND
Potassium	22	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	4.26E-04 NA		ND
Sodium	823	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.59E-02 NA		ND
Tellurium	0.07	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.36E-06 NA		ND
Thallium	0.08	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	1.55E-06 NA		ND
Tin	0.05	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	9.68E-07 NA		ND
Vanadium	0.05	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	9.68E-07 NA		ND
Zinc	0.44	19400	1.0E-03	0.17	350	30	1.0E-03	70	25550	8.52E-06 NA		ND
											TOTAL RISK	8.61E-06

NA – Not Available/Not Determined

DERMAL CONTACT WITH GROUNDWATER – FUTURE ADULT RESIDENTIAL LONG TERM HAZARD FOR LBAD SOUTH

ANALYTE	CW (MG/L)	SA (CM2)	PC (CM/HR)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	CF (L/CM3)	BW (KG)	AT (DAYS)	ABSORBED INTAKE (MG/KG/DAY)	ADJUSTED RfD (MG/KG/DAY)	HAZARD
Acetone	0.03	19400	5.70E−04	0.17	350	30	1.0E−03	70	10950	7.73E−07	1.00E−01	7.73E−06
Benzene	0.001	19400	1.10E−01	0.17	350	30	1.0E−03	70	10950	4.97E−06	2.70E−04	1.84E−02
Carbon tetrachloride	0.0005	19400	2.20E−02	0.17	350	30	1.0E−03	70	10950	4.97E−07	6.02E−04	8.26E−04
1,1−Dichloroethane	0.0009	19400	8.90E−03	0.17	350	30	1.0E−03	70	10950	3.62E−07	1.00E−01	3.62E−06
1,2−Dichloroethenes	0.003	19400	1.00E−02	0.17	350	30	1.0E−03	70	10950	1.36E−06	8.10E−03	1.67E−04
1,3−Dimethylbenzene	0.003	19400	8.00E−02	0.17	350	30	1.0E−03	70	10950	1.08E−05	1.60E+00	6.78E−06
Ethylbenzene	0.001	19400	1.00E+00	0.17	350	30	1.0E−03	70	10950	4.52E−05	9.00E−02	5.02E−04
Methyl isobutyl ketone	0.0008	19400	3.26E−03	0.17	350	30	1.0E−03	70	10950	1.18E−07	6.40E−02	1.84E−06
Tetrachloroethene	0.0005	19400	3.70E−01	0.17	350	30	1.0E−03	70	10950	8.36E−06	1.00E−02	8.36E−04
Toluene	0.006	19400	1.00E+00	0.17	350	30	1.0E−03	70	10950	2.71E−04	2.00E−01	1.36E−03
Trichloroethene	0.0007	19400	2.30E−01	0.17	350	30	1.0E−03	70	10950	7.27E−06	NA	ND
Vinyl chloride	0.009	19400	7.30E−03	0.17	350	30	1.0E−03	70	10950	2.97E−06	NA	ND
Xylenes	0.006	19400	8.00E−02	0.17	350	30	1.0E−03	70	10950	2.17E−05	1.84E+00	1.18E−05
alpha−BHC	0.000003	19400	1.60E−02	0.17	350	30	1.0E−03	70	10950	2.17E−09	NA	ND
delta−BHC	0.000002	19400	1.60E−02	0.17	350	30	1.0E−03	70	10950	1.45E−09	NA	ND
Bis(2−ethylhexyl)phthalate	0.004	19400	3.30E−02	0.17	350	30	1.0E−03	70	10950	5.96E−06	5.00E−03	1.19E−03
DDT	0.000008	19400	4.30E−01	0.17	350	30	1.0E−03	70	10950	1.55E−07	4.50E−04	3.45E−04
2,4−Dimethylphenol	0.003	19400	1.10E−01	0.17	350	30	1.0E−03	70	10950	1.49E−05	1.30E−02	1.15E−03
alpha−Endosulfan	0.000003	19400	3.30E−03	0.17	350	30	1.0E−03	70	10950	4.47E−10	4.80E−03	9.32E−08
Lindane	0.000002	19400	1.40E−02	0.17	350	30	1.0E−03	70	10950	1.26E−09	2.97E−04	4.26E−06
Phenol	0.0016	19400	8.20E−03	0.17	350	30	1.0E−03	70	10950	5.93E−07	5.40E−01	1.10E−06
Aluminum	125	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	5.65E−03	2.00E−01	2.82E−02
Antimony	0.04	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	1.81E−06	2.40E−04	7.53E−03
Arsenic	0.003	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	1.36E−07	2.85E−04	4.76E−04
Barium	0.4	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	1.81E−05	3.50E−03	5.16E−03
Beryllium	0.006	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	2.71E−07	5.00E−04	5.42E−04
Boron	0.91	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	4.11E−05	9.00E−02	4.57E−04
Cadmium	0.01	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	4.52E−07	3.50E−05	1.29E−02
Calcium	1025	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	4.63E−02	NA	ND
Chromium	0.07	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	3.16E−06	3.00E−02	1.05E−04
Cobalt	0.03	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	1.36E−06	NA	ND
Copper	0.04	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	1.81E−06	3.96E−02	4.56E−05
Iron	177	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	8.00E−03	NA	ND
Lead	0.04	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	1.81E−06	7.00E−04	2.58E−03
Magnesium	138	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	6.23E−03	NA	ND
Manganese	8.5	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	3.84E−04	2.00E−04	1.92E+00
Mercury	0.0001	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	4.52E−09	4.50E−05	1.00E−04
Molybdenum	0.03	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	1.36E−06	1.00E−03	1.36E−03
Nickel	0.05	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	2.26E−06	2.00E−03	1.13E−03
Potassium	22	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	9.94E−04	1.00E+01	9.94E−05
Sodium	823	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	3.72E−02	6.80E+00	5.47E−03
Tellurium	0.07	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	3.16E−06	NA	ND
Thallium	0.08	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	3.61E−06	8.00E−06	4.52E−01
Tin	0.05	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	2.26E−06	1.80E−02	1.25E−04
Vanadium	0.05	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	2.26E−06	7.00E−05	3.23E−02
Zinc	0.44	19400	1.00E−03	0.17	350	30	1.0E−03	70	10950	1.99E−05	9.00E−02	2.21E−04
											TOTAL HAZARD	2.50E+00

NA/ND – Not Available/Not Determined

INHALATION OF VAPORS WHILE SHOWERING – FUTURE RESIDENTIAL ADULT LONG TERM RISK FOR LBAD SOUTH

ANALYTE	Ei (MG)	SF (SHOWERS/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)–1	RISK
Acetone	0.052	1.0	350	30	70	25550	3.05E–04 NA		ND
Benzene	0.002	1.0	350	30	70	25550	1.25E–05	2.90E–02	3.62E–07
Carbon tetrachloride	0.001	1.0	350	30	70	25550	5.22E–06	5.30E–02	2.76E–07
1,1–Dichloroethane	0.002	1.0	350	30	70	25550	9.23E–06 NA		ND
1,2–Dichloroethenes	0.005	1.0	350	30	70	25550	3.16E–05 NA		ND
1,3–Dimethylbenzene	0.004	1.0	350	30	70	25550	2.52E–05 NA		ND
Ethylbenzene	0.002	1.0	350	30	70	25550	1.38E–05 NA		ND
Methyl isobutyl ketone	0.001	1.0	350	30	70	25550	8.23E–06 NA		ND
Tetrachloroethene	0.001	1.0	350	30	70	25550	5.22E–06	2.00E–03	1.04E–08
Toluene	0.010	1.0	350	30	70	25550	5.61E–05 NA		ND
Trichloroethene	0.001	1.0	350	30	70	25550	7.08E–06	6.00E–03	4.25E–08
Vinyl chloride	0.015	1.0	350	30	70	25550	8.59E–05	3.00E–01	2.58E–05
Xylenes	0.010	1.0	350	30	70	25550	5.72E–05 NA		ND
								TOTAL RISK	2.65E–05

INHALATION OF VAPORS WHILE SHOWERING – FUTURE RESIDENTIAL ADULT LONG TERM HAZARD FOR LBAD SOUTH

ANALYTE	Ei (MG)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	RfD (MG/KG/DAY)	HAZARD
Acetone	0.052	1.0	350	30	70	10950	7.11E–04 NA		ND
Benzene	0.002	1.0	350	30	70	10950	2.91E–05	1.70E–03	1.71E–02
Carbon tetrachloride	0.001	1.0	350	30	70	10950	1.22E–05 NA		ND
1,1–Dichloroethane	0.002	1.0	350	30	70	10950	2.15E–05	1.00E–01	2.15E–04
1,2–Dichloroethenes	0.005	1.0	350	30	70	10950	7.36E–05 NA		ND
1,3–Dimethylbenzene	0.004	1.0	350	30	70	10950	5.88E–05 NA		ND
Ethylbenzene	0.002	1.0	350	30	70	10950	3.21E–05	3.00E–01	1.07E–04
Methyl isobutyl ketone	0.001	1.0	350	30	70	10950	1.92E–05	2.00E–02	9.60E–04
Tetrachloroethene	0.001	1.0	350	30	70	10950	1.22E–05 NA		ND
Toluene	0.010	1.0	350	30	70	10950	1.31E–04	1.00E–01	1.31E–03
Trichloroethene	0.001	1.0	350	30	70	10950	1.65E–05	1.14E–01	1.45E–04
Vinyl chloride	0.015	1.0	350	30	70	10950	2.00E–04 NA		ND
Xylenes	0.010	1.0	350	30	70	10950	1.33E–04	8.60E–02	1.55E–03
								TOTAL HAZARD	2.14E–02

NA/ND – Not Applicable/Not Determined

INGESTION OF GROUNDWATER – FUTURE RESIDENTIAL CHILD RISK FOR LBAD SOUTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)–1	RISK
Acetone	0.03	1	350	7	16	25550	1.80E–04	NA	ND
Benzene	0.001	1	350	7	16	25550	5.99E–06	2.90E–02	1.74E–07
Carbon tetrachloride	0.0005	1	350	7	16	25550	3.00E–06	1.30E–01	3.90E–07
1,1–Dichloroethane	0.0009	1	350	7	16	25550	5.39E–06	NA	ND
1,2–Dichloroethenes	0.003	1	350	7	16	25550	1.80E–05	NA	ND
1,3–Dimethylbenzene	0.003	1	350	7	16	25550	1.80E–05	NA	ND
Ethylbenzene	0.001	1	350	7	16	25550	5.99E–06	NA	ND
Methyl isobutyl ketone	0.0008	1	350	7	16	25550	4.79E–06	NA	ND
Tetrachloroethene	0.0005	1	350	7	16	25550	3.00E–06	5.20E–02	1.56E–07
Toluene	0.006	1	350	7	16	25550	3.60E–05	NA	ND
Trichloroethene	0.0007	1	350	7	16	25550	4.20E–06	1.10E–02	4.61E–08
Vinyl chloride	0.009	1	350	7	16	25550	5.39E–05	1.90E+00	1.02E–04
Xylenes	0.006	1	350	7	16	25550	3.60E–05	NA	ND
alpha–BHC	0.000003	1	350	7	16	25550	1.80E–08	6.30E+00	1.13E–07
delta–BHC	0.000002	1	350	7	16	25550	1.20E–08	NA	ND
Bis(2–ethylhexyl)phthalate	0.004	1	350	7	16	25550	2.40E–05	1.40E–02	3.36E–07
DDT	0.000008	1	350	7	16	25550	4.79E–08	3.40E–01	1.63E–08
2,4–Dimethylphenol	0.003	1	350	7	16	25550	1.80E–05	NA	ND
alpha–Endosulfan	0.000003	1	350	7	16	25550	1.80E–08	NA	ND
Lindane	0.000002	1	350	7	16	25550	1.20E–08	1.30E+00	1.56E–08
Phenol	0.0016	1	350	7	16	25550	9.59E–06	NA	ND
Aluminum	125	1	350	7	16	25550	7.49E–01	NA	ND
Antimony	0.04	1	350	7	16	25550	2.40E–04	NA	ND
Arsenic	0.003	1	350	7	16	25550	1.80E–05	1.50E+00	2.70E–05
Barium	0.4	1	350	7	16	25550	2.40E–03	NA	ND
Beryllium	0.006	1	350	7	16	25550	3.60E–05	4.30E+00	1.55E–04
Boron	0.91	1	350	7	16	25550	5.45E–03	NA	ND
Cadmium	0.01	1	350	7	16	25550	5.99E–05	NA	ND
Calcium	1025	1	350	7	16	25550	6.14E+00	NA	ND
Chromium	0.07	1	350	7	16	25550	4.20E–04	NA	ND
Cobalt	0.03	1	350	7	16	25550	1.80E–04	NA	ND
Copper	0.04	1	350	7	16	25550	2.40E–04	NA	ND
Iron	177	1	350	7	16	25550	1.06E+00	NA	ND
Lead	0.04	1	350	7	16	25550	2.40E–04	NA	ND
Magnesium	138	1	350	7	16	25550	8.27E–01	NA	ND
Manganese	8.5	1	350	7	16	25550	5.09E–02	NA	ND
Mercury	0.0001	1	350	7	16	25550	5.99E–07	NA	ND
Molybdenum	0.03	1	350	7	16	25550	1.80E–04	NA	ND
Nickel	0.05	1	350	7	16	25550	3.00E–04	NA	ND
Potassium	22	1	350	7	16	25550	1.32E–01	NA	ND
Sodium	823	1	350	7	16	25550	4.93E+00	NA	ND
Tellurium	0.07	1	350	7	16	25550	4.20E–04	NA	ND
Thallium	0.08	1	350	7	16	25550	4.79E–04	NA	ND
Tin	0.05	1	350	7	16	25550	3.00E–04	NA	ND
Vanadium	0.05	1	350	7	16	25550	3.00E–04	NA	ND
Zinc	0.44	1	350	7	16	25550	2.64E–03	NA	ND
								TOTAL RISK	2.85E–04

NA/ND – Not Available/Not Detected

INGESTION OF GROUNDWATER – FUTURE RESIDENTIAL CHILD SHORT TERM HAZARD FOR LBAD SOUTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	SUBCHRONIC RfD (MG/KG/DAY)	HAZARD
Acetone	0.03	1	350	7	16	2555	1.80E−03	1.00E+00	1.80E−03
Benzene	0.001	1	350	7	16	2555	5.99E−05 NA		ND
Carbon tetrachloride	0.0005	1	350	7	16	2555	3.00E−05	2.00E−03	1.50E−02
1,1−Dichloroethane	0.0009	1	350	7	16	2555	5.39E−05	1.00E+00	5.39E−05
1,2−Dichloroethenes	0.003	1	350	7	16	2555	1.80E−04	9.00E−03	2.00E−02
1,3−Dimethylbenzene	0.003	1	350	7	16	2555	1.80E−04 NA		ND
Ethylbenzene	0.001	1	350	7	16	2555	5.99E−05	1.00E−01	5.99E−04
Methyl isobutyl ketone	0.0008	1	350	7	16	2555	4.79E−05	8.00E−01	5.99E−05
Tetrachloroethene	0.0005	1	350	7	16	2555	3.00E−05	1.00E−01	3.00E−04
Toluene	0.006	1	350	7	16	2555	3.60E−04	2.00E+00	1.80E−04
Trichloroethene	0.0007	1	350	7	16	2555	4.20E−05 NA		ND
Vinyl chloride	0.009	1	350	7	16	2555	5.39E−04 NA		ND
Xylenes	0.006	1	350	7	16	2555	3.60E−04 NA		ND
alpha−BHC	0.000003	1	350	7	16	2555	1.80E−07 NA		ND
delta−BHC	0.000002	1	350	7	16	2555	1.20E−07 NA		ND
Bis(2−ethylhexyl)phthalate	0.004	1	350	7	16	2555	2.40E−04 NA		ND
DDT	0.000008	1	350	7	16	2555	4.79E−07	5.00E−04	9.59E−04
2,4−Dimethylphenol	0.003	1	350	7	16	2555	1.80E−04	2.00E−01	8.99E−04
alpha−Endosulfan	0.000003	1	350	7	16	2555	1.80E−07	6.00E−03	3.00E−05
Lindane	0.000002	1	350	7	16	2555	1.20E−07	3.00E−03	4.00E−05
Phenol	0.0016	1	350	7	16	2555	9.59E−05	6.00E−01	1.60E−04
Aluminum	125	1	350	7	16	2555	7.49E+00 NA		ND
Antimony	0.04	1	350	7	16	2555	2.40E−03	4.00E−04	5.99E+00
Arsenic	0.003	1	350	7	16	2555	1.80E−04	3.00E−04	5.99E−01
Barium	0.4	1	350	7	16	2555	2.40E−02	7.00E−02	3.42E−01
Beryllium	0.006	1	350	7	16	2555	3.60E−04	5.00E−03	7.19E−02
Boron	0.91	1	350	7	16	2555	5.45E−02	9.00E−02	6.06E−01
Cadmium	0.01	1	350	7	16	2555	5.99E−04 NA		ND
Calcium	1025	1	350	7	16	2555	6.14E+01 NA		ND
Chromium	0.07	1	350	7	16	2555	4.20E−03	1.00E+00	4.20E−03
Cobalt	0.03	1	350	7	16	2555	1.80E−03 NA		ND
Copper	0.04	1	350	7	16	2555	2.40E−03 NA		ND
Iron	177	1	350	7	16	2555	1.06E+01 NA		ND
Lead	0.04	1	350	7	16	2555	2.40E−03	1.40E−03	1.71E+00
Magnesium	138	1	350	7	16	2555	8.27E+00 NA		ND
Manganese	8.5	1	350	7	16	2555	5.09E−01	5.00E−03	1.02E+02
Mercury	0.0001	1	350	7	16	2555	5.99E−06	3.00E−04	2.00E−02
Molybdenum	0.03	1	350	7	16	2555	1.80E−03	5.00E−03	3.60E−01
Nickel	0.05	1	350	7	16	2555	3.00E−03	2.00E−02	1.50E−01
Potassium	22	1	350	7	16	2555	1.32E+00 NA		ND
Sodium	823	1	350	7	16	2555	4.93E+01 NA		ND
Tellurium	0.07	1	350	7	16	2555	4.20E−03 NA		ND
Thallium	0.08	1	350	7	16	2555	4.79E−03	8.00E−04	5.99E+00
Tin	0.05	1	350	7	16	2555	3.00E−03	6.00E−01	4.99E−03
Vanadium	0.05	1	350	7	16	2555	3.00E−03	7.00E−03	4.28E−01
Zinc	0.44	1	350	7	16	2555	2.64E−02	3.00E−01	8.79E−02
								TOTAL HAZARD	1.18E+02

N/ Not Available/Not Detected

DERMAL CONTACT WITH GROUNDWATER – FUTURE RESIDENTIAL CHILD RISK FOR LBAD SOUTH

ANALYTE	CW (MG/L)	SA (CM2)	PC (CM/HR)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	CF (L/CM3)	BW (KG)	AT (DAYS)	ABSORBED INTAKE (MG/KG/DAY)	ADJUSTED CSF (MG/KG/DAY)-1	RISK
Acetone	0.03	7280	5.7E-04	0.17	350	7	1.0E-03	16	25550	1.27E-07	NA	ND
Benzene	0.001	7280	1.1E-01	0.17	350	7	1.0E-03	16	25550	8.16E-07	3.22E-02	2.63E-08
Carbon tetrachloride	0.0005	7280	2.2E-02	0.17	350	7	1.0E-03	16	25550	8.16E-08	1.51E-01	1.23E-08
1,1-Dichloroethane	0.0009	7280	8.9E-03	0.17	350	7	1.0E-03	16	25550	5.94E-08	NA	ND
1,2-Dichloroethenes	0.003	7280	1.0E-02	0.17	350	7	1.0E-03	16	25550	2.23E-07	NA	ND
1,3-Dimethylbenzene	0.003	7280	8.0E-02	0.17	350	7	1.0E-03	16	25550	1.78E-06	NA	ND
Ethylbenzene	0.001	7280	1.0E+00	0.17	350	7	1.0E-03	16	25550	7.42E-06	NA	ND
Methyl isobutyl ketone	0.0008	7280	3.3E-03	0.17	350	7	1.0E-03	16	25550	1.93E-08	NA	ND
Tetrachloroethene	0.0005	7280	3.7E-01	0.17	350	7	1.0E-03	16	25550	1.37E-06	5.20E-02	7.14E-08
Toluene	0.006	7280	1.0E+00	0.17	350	7	1.0E-03	16	25550	4.45E-05	NA	ND
Trichloroethene	0.0007	7280	2.3E-01	0.17	350	7	1.0E-03	16	25550	1.19E-06	1.10E-02	1.31E-08
Vinyl chloride	0.009	7280	7.3E-03	0.17	350	7	1.0E-03	16	25550	4.87E-07	2.37E+00	1.16E-06
Xylenes	0.006	7280	8.0E-02	0.17	350	7	1.0E-03	16	25550	3.56E-06	NA	ND
alpha-BHC	0.000003	7280	1.6E-02	0.17	350	7	1.0E-03	16	25550	3.56E-10	7.00E+00	2.49E-09
delta-BHC	0.000002	7280	1.6E-02	0.17	350	7	1.0E-03	16	25550	2.37E-10	NA	ND
Bis(2-ethylhexyl)phthalate	0.004	7280	3.3E-02	0.17	350	7	1.0E-03	16	25550	9.79E-07	5.60E-02	5.48E-08
DDT	0.000008	7280	4.3E-01	0.17	350	7	1.0E-03	16	25550	2.55E-08	3.78E-01	9.64E-09
2,4-Dimethylphenol	0.003	7280	1.1E-01	0.17	350	7	1.0E-03	16	25550	2.45E-06	NA	ND
alpha-Endosulfan	0.000003	7280	3.3E-03	0.17	350	7	1.0E-03	16	25550	7.34E-11	NA	ND
Lindane	0.000002	7280	1.4E-02	0.17	350	7	1.0E-03	16	25550	2.08E-10	1.31E+00	2.73E-10
Phenol	0.0016	7280	8.2E-03	0.17	350	7	1.0E-03	16	25550	9.73E-08	NA	ND
Aluminum	125	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	9.27E-04	NA	ND
Antimony	0.04	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	2.97E-07	NA	ND
Arsenic	0.003	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	2.23E-08	1.58E+00	3.51E-08
Barium	0.4	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	2.97E-06	NA	ND
Beryllium	0.006	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	4.45E-08	4.30E+01	1.91E-06
Boron	0.91	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	6.75E-06	NA	ND
Cadmium	0.01	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	7.42E-08	NA	ND
Calcium	1025	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	7.60E-03	NA	ND
Chromium	0.07	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	5.19E-07	NA	ND
Cobalt	0.03	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	2.23E-07	NA	ND
Copper	0.04	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	2.97E-07	NA	ND
Iron	177	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	1.31E-03	NA	ND
Lead	0.04	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	2.97E-07	NA	ND
Magnesium	138	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	1.02E-03	NA	ND
Manganese	8.5	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	6.30E-05	NA	ND
Mercury	0.0001	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	7.42E-10	NA	ND
Molybdenum	0.03	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	2.23E-07	NA	ND
Nickel	0.05	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	3.71E-07	NA	ND
Potassium	22	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	1.63E-04	NA	ND
Sodium	823	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	6.10E-03	NA	ND
Tellurium	0.07	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	5.19E-07	NA	ND
Thallium	0.08	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	5.93E-07	NA	ND
Tin	0.05	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	3.71E-07	NA	ND
Vanadium	0.05	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	3.71E-07	NA	ND
Zinc	0.44	7280	1.0E-03	0.17	350	7	1.0E-03	16	25550	3.26E-06	NA	ND
											TOTAL RISK	3.30E-06

NA/ND – Not Available/Not Determined

DERMAL CONTACT WITH GROUNDWATER – FUTURE RESIDENTIAL CHILD SHORT TERM HAZARD FOR LBAD SOUTH

ANALYTE	CW (MG/L)	SA (CM2)	PC (CM/HR)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	CF (L/CM3)	BW (KG)	AT (DAYS)	ABSORBED INTAKE (MG/KG/DAY)	ADJUSTED RfD (MG/KG/DAY)	HAZARD
Acetone	0.03	7280	5.7E−04	0.17	350	7	1.0E−03	16	2555	1.27E−06	1.00E+00	1.27E−06
Benzene	0.001	7280	1.1E−01	0.17	350	7	1.0E−03	16	2555	8.16E−06 NA		ND
Carbon tetrachloride	0.0005	7280	2.2E−02	0.17	350	7	1.0E−03	16	2555	8.16E−07	1.72E−03	4.74E−04
1,1−Dichloroethane	0.0009	7280	8.9E−03	0.17	350	7	1.0E−03	16	2555	5.94E−07	1.00E+00	5.94E−07
1,2−Dichloroethenes	0.003	7280	1.0E−02	0.17	350	7	1.0E−03	16	2555	2.23E−06	8.10E−03	2.75E−04
1,3−Dimethylberzene	0.003	7280	8.0E−02	0.17	350	7	1.0E−03	16	2555	1.78E−05 NA		ND
Ethylberzene	0.001	7280	1.0E+00	0.17	350	7	1.0E−03	16	2555	7.42E−05	9.00E−02	8.24E−04
Methyl isobutyl ketone	0.0008	7280	3.3E−03	0.17	350	7	1.0E−03	16	2555	1.93E−07	6.40E−01	3.02E−07
Tetrachloroethene	0.0005	7280	3.7E−01	0.17	350	7	1.0E−03	16	2555	1.37E−05	1.00E−01	1.37E−04
Toluene	0.006	7280	1.0E+00	0.17	350	7	1.0E−03	16	2555	4.45E−04	2.00E+00	2.23E−04
Trichloroethene	0.0007	7280	2.3E−01	0.17	350	7	1.0E−03	16	2555	1.19E−05 NA		ND
Vinyl chloride	0.009	7280	7.3E−03	0.17	350	7	1.0E−03	16	2555	4.87E−06 NA		ND
Xylenes	0.006	7280	8.0E−02	0.17	350	7	1.0E−03	16	2555	3.56E−05 NA		ND
alpha−BHC	0.000003	7280	1.6E−02	0.17	350	7	1.0E−03	16	2555	3.56E−09 NA		ND
delta−BHC	0.000002	7280	1.6E−02	0.17	350	7	1.0E−03	16	2555	2.37E−09 NA		ND
Bis(2−ethylhexyl)phthalate	0.004	7280	3.3E−02	0.17	350	7	1.0E−03	16	2555	9.79E−06 NA		ND
DDT	0.000008	7280	4.3E−01	0.17	350	7	1.0E−03	16	2555	2.55E−07	4.50E−04	5.67E−04
2,4−Dimethylphenol	0.003	7280	1.1E−01	0.17	350	7	1.0E−03	16	2555	2.45E−05	1.30E−01	1.88E−04
alpha−Endosulfan	0.000003	7280	3.3E−03	0.17	350	7	1.0E−03	16	2555	7.34E−10	4.80E−03	1.53E−07
Lindane	0.000002	7280	1.4E−02	0.17	350	7	1.0E−03	16	2555	2.08E−09	2.97E−03	6.99E−07
Phenol	0.0016	7280	8.2E−03	0.17	350	7	1.0E−03	16	2555	9.73E−07	5.40E−01	1.80E−06
Aluminum	125	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	9.27E−03 NA		ND
Antimony	0.04	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	2.97E−06	2.40E−04	1.24E−02
Arsenic	0.003	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	2.23E−07	2.85E−04	7.81E−04
Barium	0.4	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	2.97E−05	3.50E−03	8.48E−03
Beryllium	0.006	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	4.45E−07	5.00E−04	8.90E−04
Boron	0.91	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	6.75E−05	9.00E−02	7.50E−04
Cadmium	0.01	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	7.42E−07 NA		ND
Calcium	1025	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	7.60E−02 NA		ND
Chromium	0.07	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	5.19E−06	3.00E−02	1.73E−04
Cobalt	0.03	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	2.23E−06 NA		ND
Copper	0.04	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	2.97E−06 NA		ND
Iron	177	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	1.31E−02 NA		ND
Lead	0.04	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	2.97E−06	7.00E−04	4.24E−03
Magnesium	138	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	1.02E−02 NA		ND
Manganese	8.5	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	6.30E−04	2.00E−04	3.15E+00
Mercury	0.0001	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	7.42E−09	4.50E−05	1.65E−04
Molybdenum	0.03	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	2.23E−06	1.00E−03	2.23E−03
Nickel	0.05	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	3.71E−06	2.00E−03	1.85E−03
Potassium	22	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	1.63E−03 NA		ND
Sodium	823	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	6.10E−02 NA		ND
Tellurium	0.07	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	5.19E−06 NA		ND
Thallium	0.08	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	5.93E−06	8.00E−05	7.42E−02
Tin	0.05	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	3.71E−06	1.80E−02	2.06E−04
Vanadium	0.05	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	3.71E−06	7.00E−05	5.30E−02
Zinc	0.44	7280	1.0E−03	0.17	350	7	1.0E−03	16	2555	3.26E−05	9.00E−02	3.63E−04
											TOTAL HAZARD	3.31E+00

NA = Not Available/Not Determined

INHALATION OF VAPORS WHILE SHOWERING – FUTURE RESIDENTIAL CHILD RISK FOR LBAD SOUTH

ANALYTE	Ei (MG)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)–1	RISK
Acetone	0.052	1.0	350	7	16	25550	3.11E–04	NA	ND
Benzene	0.002	1.0	350	7	16	25550	1.27E–05	2.90E–02	3.70E–07
Carbon tetrachloride	0.001	1.0	350	7	16	25550	5.32E–06	5.30E–02	2.82E–07
1,1–Dichloroethane	0.002	1.0	350	7	16	25550	9.42E–06	NA	ND
1,2–Dichloroethenes	0.005	1.0	350	7	16	25550	3.22E–05	NA	ND
1,3–Dimethylbenzene	0.004	1.0	350	7	16	25550	2.57E–05	NA	ND
Ethylbenzene	0.002	1.0	350	7	16	25550	1.41E–05	NA	ND
Methyl isobutyl ketone	0.001	1.0	350	7	16	25550	8.40E–06	NA	ND
Tetrachloroethene	0.001	1.0	350	7	16	25550	5.32E–06	2.00E–03	1.06E–08
Toluene	0.010	1.0	350	7	16	25550	5.72E–05	NA	ND
Trichloroethene	0.001	1.0	350	7	16	25550	7.23E–06	6.00E–03	4.34E–08
Vinyl chloride	0.015	1.0	350	7	16	25550	8.77E–05	3.00E–01	2.63E–05
Xylenes	0.010	1.0	350	7	16	25550	5.84E–05	NA	ND
								TOTAL RISK	2.70E–05

INHALATION OF VAPORS WHILE SHOWERING – FUTURE RESIDENTIAL CHILD SHORT TERM HAZARD FOR LBAD SOUTH

ANALYTE	Ei (MG)	ET (HR/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	SUBCHRONIC RfD (MG/KG/DAY)	HAZARD
Acetone	0.052	1.0	350	7	16	2555	3.11E–03	NA	ND
Benzene	0.002	1.0	350	7	16	2555	1.27E–04	NA	ND
Carbon tetrachloride	0.001	1.0	350	7	16	2555	5.32E–05	1.70E–02	3.13E–03
1,1–Dichloroethane	0.002	1.0	350	7	16	2555	9.42E–05	1.00E+00	9.42E–05
1,2–Dichloroethenes	0.005	1.0	350	7	16	2555	3.22E–04	NA	ND
1,3–Dimethylbenzene	0.004	1.0	350	7	16	2555	2.57E–04	NA	ND
Ethylbenzene	0.002	1.0	350	7	16	2555	1.41E–04	3.00E–01	4.69E–04
Methyl isobutyl ketone	0.001	1.0	350	7	16	2555	8.40E–05	2.00E–01	4.20E–04
Tetrachloroethene	0.001	1.0	350	7	16	2555	5.32E–05	NA	ND
Toluene	0.010	1.0	350	7	16	2555	5.72E–04	3.00E–01	1.91E–03
Trichloroethene	0.001	1.0	350	7	16	2555	7.23E–05	NA	ND
Vinyl chloride	0.015	1.0	350	7	16	2555	8.77E–04	NA	ND
Xylenes	0.010	1.0	350	7	16	2555	5.84E–04	NA	ND
								TOTAL HAZARD	6.02E–03

NA/ND – Not Applicable/Not Determined

INGESTION OF GROUNDWATER – FUTURE OCCUPATIONAL ADULT SHORT TERM RISK FOR LBAD SOUTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)–1	RISK
Acetone	3.00E–02	1	250	7	70	25550	2.94E–05 NA		ND
Benzene	1.00E–03	1	250	7	70	25550	9.78E–07	2.90E–02	2.84E–08
Carbon tetrachloride	5.00E–04	1	250	7	70	25550	4.89E–07	1.30E–01	6.36E–08
1,1–Dichloroethane	9.00E–04	1	250	7	70	25550	8.81E–07 NA		ND
1,2–Dichloroethenes	3.00E–03	1	250	7	70	25550	2.94E–06 NA		ND
1,3–Dimethylbenzene	3.00E–03	1	250	7	70	25550	2.94E–06 NA		ND
Ethylbenzene	1.00E–03	1	250	7	70	25550	9.78E–07 NA		ND
Methyl isobutyl ketone	8.00E–04	1	250	7	70	25550	7.83E–07 NA		ND
Tetrachloroethene	5.00E–04	1	250	7	70	25550	4.89E–07	5.20E–02	2.54E–08
Toluene	6.00E–03	1	250	7	70	25550	5.87E–06 NA		ND
Trichloroethene	7.00E–04	1	250	7	70	25550	6.85E–07	1.10E–02	7.53E–09
Vinyl chloride	9.00E–03	1	250	7	70	25550	8.81E–06	1.90E+00	1.67E–05
Xylenes	6.00E–03	1	250	7	70	25550	5.87E–06 NA		ND
alpha–BHC	3.00E–06	1	250	7	70	25550	2.94E–09	6.30E+00	1.85E–08
delta–BHC	2.00E–06	1	250	7	70	25550	1.96E–09 NA		ND
Bis(2–ethylhexyl)phthalate	4.00E–03	1	250	7	70	25550	3.91E–06	1.40E–02	5.48E–08
DDT	8.00E–06	1	250	7	70	25550	7.83E–09	3.40E–01	2.66E–09
2,4–Dimethylphenol	3.00E–03	1	250	7	70	25550	2.94E–06 NA		ND
alpha–Endosulfan	3.00E–06	1	250	7	70	25550	2.94E–09 NA		ND
Lindane	2.00E–06	1	250	7	70	25550	1.96E–09	1.30E+00	2.54E–09
Phenol	1.60E–03	1	250	7	70	25550	1.57E–06 NA		ND
Aluminum	1.25E+02	1	250	7	70	25550	1.22E–01 NA		ND
Antimony	4.00E–02	1	250	7	70	25550	3.91E–05 NA		ND
Arsenic	3.00E–03	1	250	7	70	25550	2.94E–06	1.50E+00	4.40E–06
Barium	4.00E–01	1	250	7	70	25550	3.91E–04 NA		ND
Beryllium	6.00E–03	1	250	7	70	25550	5.87E–06	4.30E+00	2.52E–05
Boron	9.10E–01	1	250	7	70	25550	8.90E–04 NA		ND
Cadmium	1.00E–02	1	250	7	70	25550	9.78E–06 NA		ND
Calcium	1.03E+03	1	250	7	70	25550	1.00E+00 NA		ND
Chromium	7.00E–02	1	250	7	70	25550	6.85E–05 NA		ND
Cobalt	3.00E–02	1	250	7	70	25550	2.94E–05 NA		ND
Copper	4.00E–02	1	250	7	70	25550	3.91E–05 NA		ND
Iron	1.77E+02	1	250	7	70	25550	1.73E–01 NA		ND
Lead	4.00E–02	1	250	7	70	25550	3.91E–05 NA		ND
Magnesium	1.38E+02	1	250	7	70	25550	1.35E–01 NA		ND
Manganese	8.50E+00	1	250	7	70	25550	8.32E–03 NA		ND
Mercury	1.00E–04	1	250	7	70	25550	9.78E–08 NA		ND
Molybdenum	3.00E–02	1	250	7	70	25550	2.94E–05 NA		ND
Nickel	5.00E–02	1	250	7	70	25550	4.89E–05 NA		ND
Potassium	2.20E+01	1	250	7	70	25550	2.15E–02 NA		ND
Sodium	8.23E+02	1	250	7	70	25550	8.05E–01 NA		ND
Tellurium	7.00E–02	1	250	7	70	25550	6.85E–05 NA		ND
Thallium	8.00E–02	1	250	7	70	25550	7.83E–05 NA		ND
Tin	5.00E–02	1	250	7	70	25550	4.89E–05 NA		ND
Vanadium	5.00E–02	1	250	7	70	25550	4.89E–05 NA		ND
Zinc	4.40E–01	1	250	7	70	25550	4.31E–04 NA		ND
								TOTAL RISK	4.66E–05

NA – Not Available/Not Detected

INGESTION OF GROUNDWATER – FUTURE OCCUPATIONAL ADULT SHORT TERM HAZARD FOR LBAD SOUTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	SUBCHRONIC RfD (MG/KG/DAY)	HAZARD
Acetone	3.00E−02	1	250	7	70	2555	2.94E−04	1.00E+00	2.94E−04
Benzene	1.00E−03	1	250	7	70	2555	9.78E−06	NA	ND
Carbon tetrachloride	5.00E−04	1	250	7	70	2555	4.89E−06	2.00E−03	2.45E−03
1,1−Dichloroethane	9.00E−04	1	250	7	70	2555	8.81E−06	1.00E+00	ND
1,2−Dichloroethenes	3.00E−03	1	250	7	70	2555	2.94E−05	9.00E−03	3.26E−03
1,3−Dimethylbenzene	3.00E−03	1	250	7	70	2555	2.94E−05	NA	ND
Ethylbenzene	1.00E−03	1	250	7	70	2555	9.78E−06	1.00E−01	9.78E−05
Methyl isobutyl ketone	8.00E−04	1	250	7	70	2555	7.83E−06	8.00E−01	ND
Tetrachloroethene	5.00E−04	1	250	7	70	2555	4.89E−06	1.00E−01	4.89E−05
Toluene	6.00E−03	1	250	7	70	2555	5.87E−05	2.00E+00	2.94E−05
Trichloroethene	7.00E−04	1	250	7	70	2555	6.85E−06	NA	ND
Vinyl chloride	9.00E−03	1	250	7	70	2555	8.81E−05	NA	ND
Xylenes	6.00E−03	1	250	7	70	2555	5.87E−05	NA	ND
alpha−BHC	3.00E−06	1	250	7	70	2555	2.94E−08	NA	ND
delta−BHC	2.00E−06	1	250	7	70	2555	1.96E−08	NA	ND
Bis(2−ethylhexyl)phthalate	4.00E−03	1	250	7	70	2555	3.91E−05	NA	ND
DDT	8.00E−06	1	250	7	70	2555	7.83E−08	5.00E−04	1.57E−04
2,4−Dimethylphenol	3.00E−03	1	250	7	70	2555	2.94E−05	2.00E−01	1.47E−04
alpha−Endosulfan	3.00E−06	1	250	7	70	2555	2.94E−08	6.00E−03	4.89E−06
Lindane	2.00E−06	1	250	7	70	2555	1.96E−08	3.00E−03	6.52E−06
Phenol	1.60E−03	1	250	7	70	2555	1.57E−05	6.00E−01	2.61E−05
Aluminum	1.25E+02	1	250	7	70	2555	1.22E+00	NA	ND
Antimony	4.00E−02	1	250	7	70	2555	3.91E−04	4.00E−04	9.78E−01
Arsenic	3.00E−03	1	250	7	70	2555	2.94E−05	3.00E−04	9.78E−02
Barium	4.00E−01	1	250	7	70	2555	3.91E−03	7.00E−02	5.59E−02
Beryllium	6.00E−03	1	250	7	70	2555	5.87E−05	5.00E−03	1.17E−02
Boron	9.10E−01	1	250	7	70	2555	8.90E−03	9.00E−02	9.89E−02
Cadmium	1.00E−02	1	250	7	70	2555	9.78E−05	NA	ND
Calcium	1.03E+03	1	250	7	70	2555	1.00E+01	NA	ND
Chromium	7.00E−02	1	250	7	70	2555	6.85E−04	1.00E+00	6.85E−04
Cobalt	3.00E−02	1	250	7	70	2555	2.94E−04	NA	ND
Copper	4.00E−02	1	250	7	70	2555	3.91E−04	NA	ND
Iron	1.77E+02	1	250	7	70	2555	1.73E+00	NA	ND
Lead	4.00E−02	1	250	7	70	2555	3.91E−04	1.40E−03	2.80E−01
Magnesium	1.38E+02	1	250	7	70	2555	1.35E+00	NA	ND
Manganese	8.50E+00	1	250	7	70	2555	8.32E−02	5.00E−03	1.66E+01
Mercury	1.00E−04	1	250	7	70	2555	9.78E−07	3.00E−04	3.26E−03
Molybdenum	3.00E−02	1	250	7	70	2555	2.94E−04	5.00E−03	5.87E−02
Nickel	5.00E−02	1	250	7	70	2555	4.89E−04	2.00E−02	2.45E−02
Potassium	2.20E+01	1	250	7	70	2555	2.15E−01	NA	ND
Sodium	8.23E+02	1	250	7	70	2555	8.05E+00	NA	ND
Tellurium	7.00E−02	1	250	7	70	2555	6.85E−04	NA	ND
Thallium	8.00E−02	1	250	7	70	2555	7.83E−04	8.00E−04	9.78E−01
Tin	5.00E−02	1	250	7	70	2555	4.89E−04	6.00E−01	8.15E−04
Vanadium	5.00E−02	1	250	7	70	2555	4.89E−04	7.00E−03	6.99E−02
Zinc	4.40E−01	1	250	7	70	2555	4.31E−03	3.00E−01	1.44E−02
								TOTAL HAZARD	1.93E+01

NA/ND – Not Available/Not Detected

INGESTION OF GROUNDWATER – FUTURE OCCUPATIONAL ADULT LONG TERM RISK FOR LBAD SOUTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	CSF (MG/KG/DAY)–1	RISK
Acetone	3.00E–02	1	250	25	70	25550	1.05E–04 NA		ND
Benzene	1.00E–03	1	250	25	70	25550	3.49E–06	2.90E–02	1.01E–07
Carbon tetrachloride	5.00E–04	1	250	25	70	25550	1.75E–06	1.30E–01	2.27E–07
1,1–Dichloroethane	9.00E–04	1	250	25	70	25550	3.15E–06 NA		ND
1,2–Dichloroethenes	3.00E–03	1	250	25	70	25550	1.05E–05 NA		ND
1,3–Dimethylbenzene	3.00E–03	1	250	25	70	25550	1.05E–05 NA		ND
Ethylbenzene	1.00E–03	1	250	25	70	25550	3.49E–06 NA		ND
Methyl isobutyl ketone	8.00E–04	1	250	25	70	25550	2.80E–06 NA		ND
Tetrachloroethene	5.00E–04	1	250	25	70	25550	1.75E–06	5.20E–02	9.09E–08
Toluene	6.00E–03	1	250	25	70	25550	2.10E–05 NA		ND
Trichloroethene	7.00E–04	1	250	25	70	25550	2.45E–06	1.10E–02	2.69E–08
Vinyl chloride	9.00E–03	1	250	25	70	25550	3.15E–05	1.90E+00	5.98E–05
Xylenes	6.00E–03	1	250	25	70	25550	2.10E–05 NA		ND
alpha–BHC	3.00E–06	1	250	25	70	25550	1.05E–08	6.30E+00	6.60E–08
delta–BHC	2.00E–06	1	250	25	70	25550	6.99E–09 NA		ND
Bis(2–ethylhexyl)phthalate	4.00E–03	1	250	25	70	25550	1.40E–05	1.40E–02	1.96E–07
DDT	8.00E–06	1	250	25	70	25550	2.80E–08	3.40E–01	9.51E–09
2,4–Dimethylphenol	3.00E–03	1	250	25	70	25550	1.05E–05 NA		ND
alpha–Endosulfan	3.00E–06	1	250	25	70	25550	1.05E–08 NA		ND
Lindane	2.00E–06	1	250	25	70	25550	6.99E–09	1.30E+00	9.09E–09
Phenol	1.60E–03	1	250	25	70	25550	5.59E–06 NA		ND
Aluminum	1.25E+02	1	250	25	70	25550	4.37E–01 NA		ND
Antimony	4.00E–02	1	250	25	70	25550	1.40E–04 NA		ND
Arsenic	3.00E–03	1	250	25	70	25550	1.05E–05	1.50E+00	1.57E–05
Barium	4.00E–01	1	250	25	70	25550	1.40E–03 NA		ND
Beryllium	6.00E–03	1	250	25	70	25550	2.10E–05	4.30E+00	9.02E–05
Boron	9.10E–01	1	250	25	70	25550	3.18E–03 NA		ND
Cadmium	1.00E–02	1	250	25	70	25550	3.49E–05 NA		ND
Calcium	1.03E+03	1	250	25	70	25550	3.58E+00 NA		ND
Chromium	7.00E–02	1	250	25	70	25550	2.45E–04 NA		ND
Cobalt	3.00E–02	1	250	25	70	25550	1.05E–04 NA		ND
Copper	4.00E–02	1	250	25	70	25550	1.40E–04 NA		ND
Iron	1.77E+02	1	250	25	70	25550	6.19E–01 NA		ND
Lead	4.00E–02	1	250	25	70	25550	1.40E–04 NA		ND
Magnesium	1.38E+02	1	250	25	70	25550	4.82E–01 NA		ND
Manganese	8.50E+00	1	250	25	70	25550	2.97E–02 NA		ND
Mercury	1.00E–04	1	250	25	70	25550	3.49E–07 NA		ND
Molybdenum	3.00E–02	1	250	25	70	25550	1.05E–04 NA		ND
Nickel	5.00E–02	1	250	25	70	25550	1.75E–04 NA		ND
Potassium	2.20E+01	1	250	25	70	25550	7.69E–02 NA		ND
Sodium	8.23E+02	1	250	25	70	25550	2.88E+00 NA		ND
Tellurium	7.00E–02	1	250	25	70	25550	2.45E–04 NA		ND
Thallium	8.00E–02	1	250	25	70	25550	2.80E–04 NA		ND
Tin	5.00E–02	1	250	25	70	25550	1.75E–04 NA		ND
Vanadium	5.00E–02	1	250	25	70	25550	1.75E–04 NA		ND
Zinc	4.40E–01	1	250	25	70	25550	1.54E–03 NA		ND
								TOTAL RISK	1.66E–04

NA = Not Available/Not Detected

INGESTION OF GROUNDWATER – FUTURE OCCUPATIONAL ADULT LONG TERM HAZARD FOR LBAD SOUTH

ANALYTE	CW (MG/L)	IR (L/DAY)	EF (DAY/YR)	ED (YR)	BW (KG)	AT (DAYS)	INTAKE (MG/KG/DAY)	RfD (MG/KG/DAY)	HAZARD
Acetone	3.00E−02	1	250	25	70	9125	2.94E−04	1.00E−01	2.94E−03
Benzene	1.00E−03	1	250	25	70	9125	9.78E−06	3.00E−04	3.26E−02
Carbon tetrachloride	5.00E−04	1	250	25	70	9125	4.89E−06	7.00E−04	6.99E−03
1,1−Dichloroethane	9.00E−04	1	250	25	70	9125	8.81E−06	1.00E−01	8.81E−05
1,2−Dichloroethenes	3.00E−03	1	250	25	70	9125	2.94E−05	9.00E−03	3.26E−03
1,3−Dimethylbenzene	3.00E−03	1	250	25	70	9125	2.94E−05	2.00E+00	1.47E−05
Ethylbenzene	1.00E−03	1	250	25	70	9125	9.78E−06	1.00E−01	9.78E−05
Methyl isobutyl ketone	8.00E−04	1	250	25	70	9125	7.83E−06	8.00E−02	9.78E−05
Tetrachloroethene	5.00E−04	1	250	25	70	9125	4.89E−06	1.00E−02	4.89E−04
Toluene	6.00E−03	1	250	25	70	9125	5.87E−05	2.00E−01	2.94E−04
Trichloroethene	7.00E−04	1	250	25	70	9125	6.85E−06	NA	ND
Vinyl chloride	9.00E−03	1	250	25	70	9125	8.81E−05	NA	ND
Xylenes	6.00E−03	1	250	25	70	9125	5.87E−05	2.00E+00	2.94E−05
alpha−BHC	3.00E−06	1	250	25	70	9125	2.94E−08	NA	ND
delta−BHC	2.00E−06	1	250	25	70	9125	1.96E−08	NA	ND
Bis(2−ethylhexyl)phthalate	4.00E−03	1	250	25	70	9125	3.91E−05	2.00E−02	1.96E−03
DDT	8.00E−06	1	250	25	70	9125	7.83E−08	5.00E−04	1.57E−04
2,4−Dimethylphenol	3.00E−03	1	250	25	70	9125	2.94E−05	2.00E−02	1.47E−03
alpha−Endosulfan	3.00E−06	1	250	25	70	9125	2.94E−08	6.00E−03	4.89E−06
Lindane	2.00E−06	1	250	25	70	9125	1.96E−08	3.00E−04	6.52E−05
Phenol	1.60E−03	1	250	25	70	9125	1.57E−05	6.00E−01	2.61E−05
Aluminum	1.25E+02	1	250	25	70	9125	1.22E+00	1.00E+00	1.22E+00
Antimony	4.00E−02	1	250	25	70	9125	3.91E−04	4.00E−04	9.78E−01
Arsenic	3.00E−03	1	250	25	70	9125	2.94E−05	3.00E−04	9.78E−02
Barium	4.00E−01	1	250	25	70	9125	3.91E−03	7.00E−02	5.59E−02
Beryllium	6.00E−03	1	250	25	70	9125	5.87E−05	5.00E−03	1.17E−02
Boron	9.10E−01	1	250	25	70	9125	8.90E−03	9.00E−02	9.89E−02
Cadmium	1.00E−02	1	250	25	70	9125	9.78E−05	5.00E−04	1.96E−01
Calcium	1.03E+03	1	250	25	70	9125	1.00E+01	NA	ND
Chromium	7.00E−02	1	250	25	70	9125	6.85E−04	1.00E+00	6.85E−04
Cobalt	3.00E−02	1	250	25	70	9125	2.94E−04	NA	ND
Copper	4.00E−02	1	250	25	70	9125	3.91E−04	4.00E−02	9.78E−03
Iron	1.77E+02	1	250	25	70	9125	1.73E+00	NA	ND
Lead	4.00E−02	1	250	25	70	9125	3.91E−04	1.40E−03	2.80E−01
Magnesium	1.38E+02	1	250	25	70	9125	1.35E+00	NA	ND
Manganese	8.50E+00	1	250	25	70	9125	8.32E−02	5.00E−03	1.66E+01
Mercury	1.00E−04	1	250	25	70	9125	9.78E−07	3.00E−04	3.26E−03
Molybdenum	3.00E−02	1	250	25	70	9125	2.94E−04	5.00E−03	5.87E−02
Nickel	5.00E−02	1	250	25	70	9125	4.89E−04	2.00E−02	2.45E−02
Potassium	2.20E+01	1	250	25	70	9125	2.15E−01	5.00E+01	4.31E−03
Sodium	8.23E+02	1	250	25	70	9125	8.05E+00	3.40E+01	2.37E−01
Tellurium	7.00E−02	1	250	25	70	9125	6.85E−04	NA	ND
Thallium	8.00E−02	1	250	25	70	9125	7.83E−04	8.00E−05	9.78E+00
Tin	5.00E−02	1	250	25	70	9125	4.89E−04	6.00E−01	8.15E−04
Vanadium	5.00E−02	1	250	25	70	9125	4.89E−04	7.00E−03	6.99E−02
Zinc	4.40E−01	1	250	25	70	9125	4.31E−03	3.00E−01	1.44E−02
								TOTAL HAZARD	**2.98E+01**

NA/ND – Not Available/Not Detected

APPENDIX P
EVALUATION OF ESSENTIAL NUTRIENTS

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Evaluation of Essential Nutrients P-1

List of Tables

Table

1 Comparison of Essential Nutrient Maximum Concentrations Detected in Groundwater to Their Recommended Dietary Allowance P-2

EVALUATION OF ESSENTIAL NUTRIENTS

Some naturally occurring metals are essential nutrients required by humans and other organisms to maintain vital physiological processes. Nine inorganic chemicals detected in groundwater at the LBAD Facility are considered to be essential nutrients. These chemicals are calcium, chromium, copper, iron, magnesium, manganese, potassium, sodium, and zinc. The National Academy of Science has determined chemical-specific levels, termed recommended dietary allowance (RDA), that provide a level of sufficient nutritional value for most individuals.

A comparison was made between the daily intake (DI) of groundwater of each of the essential nutrients to the respective RDA. The ratio of the DI to the RDA provides an indication of whether or not the chemical concentration in groundwater when ingested, will result in exceedance of the RDA (quotient greater than one). The RDAs for essential nutrients are assumed to come from an individual's dietary intake of food stuffs. Any intake of these chemicals from groundwater would be in addition to food sources.

The daily intake from groundwater was determined using the exposure parameters for the chronic hazard calculations but assuming an ingestion rate of 2 l/day, which is consistent with the more conservative ingestion rate of a residential adult.

Results of the comparison are shown in Table 1. The DI/RDA ratios of calcium, iron, magnesium, manganese, and sodium indicate that these constitutents exist in groundwater at levels exceeding the RDA. No conclusions can be drawn from this comparison regarding potential adverse consequences of ingesting concentrations greater than the RDA.

Although four of the nine constituents did not exceed their acceptable RDA values, all ten constituents were taken through the risk assessment.

Table 1 Comparison of Essential Nutrient Concentrations Detected in Groundwater to their Recommended Dietary Allowance

ESSENTIAL NUTRIENT	SITE-WIDE MAXIMUM EXPOSURE POINT CONCENTRATION(a) (MG/L)	DAILY INTAKE (DI) FROM GROUNDWATER MG/DAY	RECOMMENDED DIETARY ALLOWANCE (RDA) (c) MG/DAY	DI/RDA (UNITLESS)
Calcium	1.70E+03	3400	1200	2.83
Chromium	0.0684	0.1368	0.2	0.68
Copper	0.0379	0.0758	3	0.03
Iron	177	354	15	23.60
Magnesium	920	1840	350	5.26
Manganese	8.46	16.92	5	3.38
Potassium	154	308	2000	0.15
Sodium	9800	19600	500	39.20
Zinc	0.437	0.874	15	0.06

(a) The exposure point concentration is the 95% UCL or the maximum concentration detected, whichever is lower
(b) The daily intake is derrived using an ingestion rate of 2 liters of groundwater per day
(c) RDA values are from the National Academy of Science.

APPENDIX Q

IRIS PRINTOUT FILES

1 - IRIS
IRSN - 125
DATE - 930802
UPDT - 08/02/93, 2 fields
STAT - Oral RfD Assessment (RDO) on-line 08/01/93
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 12/01/90
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 03/01/88 RDO Confidence levels revised
IRH - 07/01/89 REFS Bibliography on-line
IRH - 01/01/90 CAR Carcinogen assessment now under review
IRH - 07/01/90 CAR Carcinogen assessment on-line
IRH - 07/01/90 RCRA EPA contact changed
IRH - 07/01/90 CREF Carcinogen references added
IRH - 12/01/90 RDO Text edited
IRH - 12/01/90 RDO EPA contacts changed
IRH - 12/01/90 CAREV Text edited
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 08/01/93 RDO Oral RfD noted as pending change
IRH - 08/01/93 RDO Work group review date added
RLEN - 10027
NAME - Acetone
RN - 67-64-1
SY - ACETON
SY - Acetone
SY - DIMETHYLFORMALDEHYDE
SY - DIMETHYLKETAL
SY - DIMETHYL KETONE
SY - KETONE, DIMETHYL
SY - KETONE PROPANE
SY - beta-KETOPROPANE
SY - METHYL KETONE
SY - PROPANONE
SY - 2-PROPANONE
SY - PYROACETIC ACID
SY - PYROACETIC ETHER
SY - RCRA WASTE NUMBER U002
SY - UN 1090

RDO -
o ORAL RFD SUMMARY :

NOTE: The Oral RfD for acetone may change in the near future pending the outcome of a further review now being conducted by the RfD/RfC Work Group.

Critical Effect	Experimental Doses*	UF	MF	RfD

Increased liver and kidney weights and nephrotoxicity	NOEL: 100 mg/kg/day LOAEL: 500 mg/kg/day	1000	1	1E-1 mg/kg/day

Rat Oral Subchronic Study

U.S. EPA, 1986

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*Conversion Factors: Actual dose tested

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o ORAL RFD STUDIES :

U.S. EPA. 1986. Ninety-day gavage study in albino rats using acetone. Office of Solid Waste, Washington, DC.

Acetone was administered by gavage for 90 days to groups of albino rats (30/sex/group) at 0, 100, 500, or 2500 mg/kg/day. Body weights, food consumption, clinical chemistry, hematology, and histopathologic parameters, as well as organ weights and organ-to-body weight ratios, were measured and analyzed. Animals were sacrificed after 30 or 90 days of exposure. No effects were seen at the 100 mg/kg/day dose level throughout the study. RBC parameters were significantly increased in the 2500-mg/kg/day group at 30 days (males only) and at 90 days in males and females. Statistical analysis of the absolute and relative organ weight data revealed significantly increased kidney weights for females in the 500- and 2500-mg/kg/day groups and increased kidney-to-body and brain weight ratios for males and females in the 2500-mg/kg/day groups. Liver weight and liver/body weight ratios were also increased in the 2500-mg/kg/day males and females. Histopathologic studies revealed a marked increase in severity in tubular degeneration of the kidneys and hyaline droplet accumulation with increasing doses. This accumulation was significant in the 500- and 2500-mg/kg/day males and the 2500 mg/kg/day females.

Based on the above findings, the NOEL for this study is 100 mg/kg/day and the LOAEL is 500 mg/kg/day based on increased liver and kidney weights and nephrotoxicity.

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o ORAL RFD UNCERTAINTY :

UF -- An uncertainty factor of 1000 is used; 100 for inter- and intraspecies extrapolation and 10 to extrapolate from subchronic to chronic exposure.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

Limited human studies have shown that workers exposed to acetone vapors (600 to 2150 ppm) experienced transient eye and nose irritation. Animals exposed to acetone vapors at 45,134 mg/cu.m experienced slight, but not significant, decreases in organ and body weights.

---

o ORAL RFD CONFIDENCE :

Study -- Medium
Data Base -- Low
RfD -- Low

Confidence in the principal study is rated medium, since a moderate number of animals/dose/sex and an extensive number of parameters were measured. The data base is rated low because a very limited number of studies are available and no pertinent supporting studies were located. The overall confidence rating for the RfD is low.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- None

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o REVIEW DATES : 12/18/85, 05/30/86, 07/21/93
o VERIFICATION DATE : 05/30/86
o EPA CONTACTS :

Harlal Choudhury / OHEA -- (513)569-7553

W. Bruce Peirano / OHEA -- (513)569-7540

CAREV-
o CLASSIFICATION : D; not classifiable as to human carcinogenicity
o BASIS FOR CLASSIFICATION : Based on lack of data concerning carcinogenicity in humans or animals.
o HUMAN CARCINOGENICITY DATA :

None.

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o ANIMAL CARCINOGENICITY DATA :

None.

---

o SUPPORTING DATA :

Acetone did not show mutagenic activity when tested in Salmonella typhimurium strains TA98 and TA100 or in Schizosaccharomyces pombe strain P1 either in the presence or absence of liver homogenates (McCann et al., 1975; Abbondandolo et al., 1980; Maron et al., 1981; Hallstrom et al., 1981) or in cell transformation systems (Freeman et al., 1973; Rhim et al., 1974; Quarles et al., 1979a,b). Furthermore, acetone gave negative results in assays for chromosomal aberrations and sister chromatid exchange (Norppa et al., 1981; Norppa, 1981; Tates and Kriek, 1981), DNA binding (Kubinski et al., 1981), point mutation in mouse lymphoma cells (Amacher et al., 1980), and transfection of E. coli CR63 cells (Vasavada and Padayatty, 1981). In one study, however, acetone was reported to produce chromosomal aberrations but not sister chromatid exchanges (Kawachi et al., 1980).

CARDR-
o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1988

The 1988 updated Health Effects Document for Acetone has received Agency review and is approved for publication.
DOCUMENT

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o REVIEW DATES : 12/06/89
o VERIFICATION DATE : 12/06/89
o EPA CONTACTS :

Charles Ris / OHEA -- (202)260-5895

CERC -

Value (status) -- 5000 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final adjusted RQ for acetone is 5000 pounds, based on the application of the secondary criterion of biodegradation to the primary criteria RQ of 1000 pounds, determined by ignitability. Available data indicate a flash point of -4F and a boiling point of 133F, which corresponds to an RQ of 1000 pounds. The final RQ takes biodegradation into account, since acetone biodegrades when released into the environment. The biological oxygen demand for 5 days (BOD5) is 46-55%.

Reference -- 50 FR 13456 (04/04/85); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - U.S. EPA. 1986. Ninety-day gavage study in albino rats using acetone. Office of Solid Waste, Washington, DC.

IREF - None

CREF - Abbondandolo, A., S. Bonatti, C. Corsi, et al. 1980. The use of organic solvents in mutagenicity testing. Mutat. Res. 79: 141-150.

CREF - Amacher, D.E., S.C. Paillet, G.N. Turner, V.A. Ray and D.S. Salsburg. 1980. Point mutations at the thymidine kinase locus in L5178Y mouse lymphoma cells. 2. Test validation and interpretation. Mutat. Res. 72: 447-474.

CREF - Freeman, A.E., E.K. Weisburger, J.H. Weisburger, R.G. Wolford, J.M. Maryak and R.J. Huebner. 1973. Transformation of cell cultures as an indication of the carcinogenic potential of chemicals. J. Natl. Cancer Inst. 51(3): 799-808.

CREF - Hallstrom, I., A. Sundvall, U. Rannug, R. Grafstrom and C. Ramel. 1981. The metabolism of drugs and carcinogens in isolated subcellular fractions of Drosophila melangaster. I. Activation of vinyl chloride, 2-amnioanthrecene and benzo(a)pyrene as measured by mutagenic effects in Salmonella typhimurium. Chem. Biol. Inter. 34: 129-143.

CREF - Kawachi, T., T. Yahagi, T. Kada et al. 1980. Cooperative programme on short-term assays for carcinogenicity in Japan. In: Molecular and Cellular Aspects of Carcinogen Screening Tests. R. Montesano, ed. WHO, IARC, Lyon, France. p. 323-330.

CREF - Kubinski, H., G.E. Gutzke and Z.O. Kubinski. 1981. DNA-cell-binding (DCB) assay for suspected carcinogens and mutagens. Mutat. Res. 89: 95-136.

CREF - Maron, D., J. Katzenellenbogen and B.N. Ames. 1981. Compatability of organic solvents with the Salmonella/microsome test. Mutat. Res.

88: 343-350.
CREF - McCann, J., E. Choi, E. Yamasaki and B.N. Ames. 1975. Detection of carcinogens as mutagens in the Salmonella/microsome test: Assay of 300 chemicals. Proc. Natl. Acad. Sci. 72(12): 5135-5139.
CREF - Norppa, H. 1981. The in vitro induction of sister chromatid exchanges and chromosome aberrations in human lymphocytes by styrene derivatives. Carcinogenesis. 2(3): 237-242.
CREF - Norppa, H., K. Hemminki, M. Sorsa and H. Vainio. 1981. Effect of monosubstituted epoxides on chromosome aberrations and SCE in cultured human lymphocytes. Mutat. Res. 91: 243-250.
CREF - Quarles, J.M., M.W. Sega, C.K. Schenley and W. Lijinsky. 1979a. Transformation of hamster fetal cells by nitrosated pesticides in a transplacental assay. Cancer Res. 39: 4525-4533.
CREF - Quarles, J.M., M.W. Sega, C.K Schenley and R.W. Tennant. 1979b. Rapid screening for chemical carcinogens: Transforming activity of selected nitroso compounds detected in a transplacental host-mediated culture system. Natl. Cancer Inst. Monogr. 51: 257-263.
CREF - Rhim, J.S., D.K. Park, E.K. Weisburger and J.H. Weisburger. 1974. Evaluation of an in vitro assay system for carcinogens based on prior infection of rodent cells with nontransforming RNA tumor virus. J. Natl. Cancer Inst. 52(4): 1167-1173.
CREF - Tates, A.D. and E. Kriek. 1981. Induction of chromosomal aberrations and sister-chromatid exchanges in Chinese hamster cells in vitro by some proximate and ultimate carcinogenic arylamide derivatives. Mutat. Res. 88: 397-410.
CREF - Vasavada, H.A. and J.D. Padayatty. 1981. Rapid transfection assay for screening mutagens and carcinogens. Mutat. Res. 91: 9-14.
CREF - U.S. EPA. 1988. Updated Health Effects Assessment for Acetone. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.
HAREF- None

[IRIS] SS 2 /cf?
USER:

1 - IRIS
IRSN - 270
DATE - 940207
UPDT - 02/07/94, 6 fields
STAT - Oral RfD Assessment (RDO) pending
STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) on-line 02/01/94
STAT - Drinking Water Health Advisories (DWHA) on-line 08/01/90
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 12/01/88 CAREV Anderson and Richardson citation year corrected
IRH - 12/01/88 CAREV Kissling and Speck citation year corrected
IRH - 07/01/89 RDI Inhalation RfD now under review
IRH - 02/01/90 CAR Clarified citations
IRH - 02/01/90 CAREV Corrected Maltoni, 1979 to Maltoni and Scarnato, 1979
IRH - 02/01/90 CAREV Corrected Maltoni, 1983 to Maltoni et al., 1983
IRH - 02/01/90 CAREV Corrected Synder et al., 1980 to 1981
IRH - 02/01/90 REFS Bibliography on-line
IRH - 03/01/90 CREF Clarify Maltoni et al., 1983 and NTP, 1986 references
IRH - 08/01/90 HADR Primary contact changed
IRH - 08/01/90 RCRA EPA contact changed
IRH - 01/01/91 CAR Text edited
IRH - 01/01/91 CARI Inhalation slope factor removed (global change)
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 04/01/92 CARO Text revised
IRH - 02/01/94 CARDR Secondary contact's phone number changed
RLEN - 25778
NAME - Benzene
RN - 71-43-2
SY - Benzene
SY - benzol
SY - coal naphtha
SY - cyclohexatriene
SY - phene
SY - phenyl hydride
SY - polystream
SY - pyrobenzol

RDO -
o ORAL RFD SUMMARY :

A risk assessment for this substance/agent will be reviewed by an EPA work group.

RDI -
o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

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o REVIEW DATES : 05/18/89, 07/20/89
CAREV-
o CLASSIFICATION : A; human carcinogen
o BASIS FOR CLASSIFICATION : Several studies of increased incidence of nonlymphocytic leukemia from occupational exposure, increased incidence of neoplasia in rats and mice exposed by inhalation and gavage, and some supporting data form the basis for this classification.
o HUMAN CARCINOGENICITY DATA :

Aksoy et al. (1974) reported effects of benzene exposure among 28,500 Turkish workers employed in the shoe industry. Mean duration of employment was 9.7 years (1-15 year range) and mean age was 34.2 years. Peak exposure was reported to be 210-650 ppm. Twenty-six cases of leukemia and a total of 34 leukemias or preleukemias were observed, corresponding to an incidence of 13/100,000 (by comparison to 6/100,000 for the general population). A follow-up paper (Aksoy, 1980) reported eight additional cases of leukemia as well as evidence suggestive of increases in other malignancies.

In a retrospective cohort mortality study Infante et al. (1977a,b) examined leukemogenic effects of benzene exposure in 748 white males exposed while employed in the manufacturing of rubber products. Exposure occurred from 1940-1949, and vital statistics were obtained through 1975. A statistically significant increase (p less than or equal to 0.002) of leukemias was found by comparison to the general U.S. population. There was no evidence of solvent exposure other than benzene. Air concentrations were generally found to be below the recommended limits in effect during the study period.

In a subsequent retrospective cohort mortality study Rinsky et al. (1981) observed seven deaths from leukemia among 748 workers exposed to benzene and followed for at least 24 years (17,020 person-years). This increased incidence was statistically significant; standard mortality ratio (SMR) was 560. For the five leukemia deaths that occurred among workers with more than 5 years exposure, the SMR was 2100. Exposures (which ranged from 10-100 ppm 8-hour TWA) were described as less than the recommended standards for the time period of 1941-1969.

In an updated version of the Rinsky et al. (1981) study, the authors followed the same cohort to 12/31/81 (Rinsky et al., 1987). An in his earlier study, cumulative exposure was derived from historic air-sampling data or

interpolated estimates based on exisitng data. Standardized mortality rates ranged from 109 at cumulative benzene exposures under 40 ppm-years and increased montonically to 6637 (6 cases) at 400 ppm-years or more. The authors found significantly elevated risks of leukemia at cumulative exposures less than the equivalent current standard for occupational exposure which is 10 ppm over a 40-year working lifetime.

Ott et al. (1978) observed three deaths from leukemia among 594 workers followed for at least 23 years in a retrospective cohort mortality study, but the increase was not statistically significant. Exposures ranged from <2 to >25 ppm 8-hour TWA.

Wong et al. (1983) reported on the mortality of male chemical workers who had been exposed to benzene for at least 6 months during the years 1946-1975. The study population of 4062 persons was drawn from seven chemical plants, and jobs were categorized as to peak exposure. Those with at least 3 days/week exposure (3036 subjects) were further categorizeed on the basis of an 8-hour TWA. The control subjects held jobs at the same plants for at least 6 months but were never subject to benzene exposure. Dose-dependent increases were seen in leukemia and lymphatic and hematopoietic cancer. The incidence of leukemia was responsible for the majority of the increase. It was noted that the significance of the increase is due largely to a less than expected incidence of neoplasia in the unexposed subjects.

Numerous other epidemiologic and case studies have reported an increased incidence or a causal relationship between leukemia and exposure to benzene (IARC, 1982).

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o ANIMAL CARCINOGENICITY DATA :

Both gavage and inhalation exposure of rodents to benzene have resulted in development of neoplasia. Maltoni and Scarnato (1979) and Maltoni et al. (1983) administered benzene by gavage at dose levels of 0, 50, 250, and 500 mg/kg bw to 30-40 Sprague-Dawley rats/sex for life. Dose-related increased incidences of mammary tumors were seen in females and of Zymbal gland carcinomas, oral cavity carcinomas and leukemias/lymphomas in both sexes.

In an NTP (1986) study, benzene was administered by gavage doses of 0, 50, 100, or 200 mg/kg bw to 50 F344/N rats/sex or 0, 25, 50, or 100 mg/kg bw to 50 B6C3F1 mice/sex. Treatment was 5 times/week for 103 weeks. Significantly increased incidences ($p<0.05$) of various neoplasic growths were seen in both sexes of both species. Both male and female rats and mice had increased incidence of carcinomas of the Zymbal gland. Male and female rats had oral cavity tumors, and males showed increased incidences of skin tumors. Mice of both sexes had increased incidence of lymphomas and lung tumors. Males were observed to have harderian and preputial gland tumors and females had tumors of mammary gland and ovary. In general, the increased incidence was dose-related.

Slightly increased incidences of hematopoietic neoplasms were reported for

male C57Bl mice exposed by inhalation to 300 ppm benzene 6 hours/day, 5 days/ week for 488 days. There was no increase in tumor incidence in male AKR or CD-1 mice similarly exposed to 100 ppm or 100 or 300 ppm benzene, respectively. Likewise male Sprague-Dawley rats exposed by inhalation to 300 ppm benzene were not observed to have increased incidence of neoplasia (Snyder et al., 1981).

Maltoni et al. (1983) treated male and female Sprague-Dawley rats in the following manner. Starting at 13 weeks of age rats were exposed to 200 ppm benzene 4 hours/day, 5 days/week for 7 weeks; 200 ppm 7 hours/day, 5 days/week for 12 weeks; 300 ppm 7 hours/day, 5 days/week for 85 weeks. An 8-hour/day TWA for 5 days/week was calculated to be 241 ppm. A statistically significant increase was noted in hepatomas and carcinomas of the Zymbal gland.

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o SUPPORTING DATA :

Numerous investigators have found significant increases in chromosomal aberrations of bone marrow cells and peripheral lymphocytes from workers with exposure to benzene (IARC, 1982). Benzene also induced chromosomal aberrations in bone marrow cells from rabbits (Kissling and Speck, 1973), mice (Meyne and Legator, 1980) and rats (Anderson and Richardson, 1979). Several investigators have reported positive results for benzene in mouse micronucleus assays (Meyne and Legator, 1980). Benzene was not mutagenic in several bacterial and yeast systems, in the sex-linked recessive lethal mutation assay with Drosophila melanogaster or in mouse lymphoma cell forward mutation assay.

CARO -
o CLASSIFICATION : A; human carcinogen
o BASIS FOR CLASSIFICATION : Several studies of increased incidence of nonlymphocytic leukemia from occupational exposure, increased incidence of neoplasia in rats and mice exposed by inhalation and gavage, and some supporting data form the basis for this classification.
o ORAL SLOPE FACTOR : 2.9E-2 per (mg/kg)/day
o DRINKING WATER UNIT RISK : 8.3E-7 per (ug/L)
o DOSE EXTRAPOLATION METHOD : One-hit (pooled data)
o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	1E+2 ug/L
E-5 (1 in 100,000)	1E+1 ug/L
E-6 (1 in 1,000,000)	1E+0 ug/L

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o ORAL DOSE-RESPONSE DATA :

Tumor Type -- leukemia
Test Animals -- human
Route -- inhalation, occupational exposure
Reference -- Rinsky et al., 1981; Ott et al., 1978; Wong et al., 1983

The slope factor was derived from human data for inhalation exposure (see dose-response data for inhalation quantitative estimate). The human respiratory rate was assumed to be 20 cu.m/day and the human drinking water intake was assumed to be 2 L/day. The fraction of the administered dose absorbed systemically via inhalation and via drinking water were assumed to be equal.

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o ADDITIONAL COMMENTS :

The unit risk estimate is the geometric mean of four ML point estimates using pooled data from the Rinsky et al. (1981) and Ott et al. (1978) studies, which was then adjusted for the results of the Wong et al. (1983) study as described in the additional comments section for inhalation data.

The unit risk should not be used if the water concentration exceeds 1E+4 ug/L, since above this concentration the unit risk may not be appropriate.

---

o DISCUSSION OF CONFIDENCE :

The pooled cohorts were sufficiently large and were followed for an adequate time period. The increases in leukemias were statistically significant and dose-related in one of the studies. Wong et al. (1983) disagrees that exposures reported in Rinsky et al. (1981) were within the recommended standards. For the five leukemia deaths in persons with 5 or more years exposure, the author notes that mean exposure levels (range 15-70 ppm) exceeded the recommended standard (25 ppm) in 75% of the work locations sampled. A total of 21 unit risk estimates were prepared using 6 models and various combinations of the epidemiologic data. These range over slightly more than one order of magnitude. A geometric mean of these estimates is 2.7E-2. Regression models give an estimate similar to the geometric mean.

The risk estimate above based on reconsideration of the Rinsky et al. (1981) and Ott et al. (1978) studies is very similar to that of 2.4E-2/ppm (cited in U.S. EPA, 1980) based on Infante et al. (1977a,b), Ott et al. (1978) and Aksoy et al. (1974). It was felt by the authors of U.S. EPA (1985) that the exposure assessment provided by Aksoy was too imprecise to warrant inclusion in the current risk estimate.

Risk estimates based on animal gavage studies are about 5 times higher than those derived from human data. Pharmacokinetic data which could impact the risk assessment are currently being evaluated.

CARI -
o CLASSIFICATION : A; human carcinogen
o BASIS FOR CLASSIFICATION : Several studies of increased incidence of nonlymphocytic leukemia from occupational exposure, increased incidence of neoplasia in rats and mice exposed by inhalation and gavage, and some supporting data form the basis for this classification.
o INHALATION UNIT RISK : 8.3E-6 per (ug/cu.m)
o DOSE EXTRAPOLATION METHOD : One-hit (pooled data)
o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	1E+1 ug/cu.m
E-5 (1 in 100,000)	1E+0 ug/cu.m
E-6 (1 in 1,000,000)	1E-1 ug/cu.m

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o INHALATION DOSE-RESPONSE DATA :

Tumor Type -- leukemia
Test Animals -- humans
Route -- inhalation, occupational exposure
Reference -- Rinsky et al., 1981; Ott et al., 1978; Wong et al., 1983

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o ADDITIONAL COMMENTS :

The unit risk estimate is the geometric mean of four ML point estimates using pooled data from the Rinsky et al. (1981) and Ott et al. (1978) studies, which was then adjusted for the results of the Wong et al. (1983) study. The Rinsky data used were from an updated tape which reports one more case of leukemia than was published in 1981. Equal weight was given to cumulative dose and weighted cumulative dose exposure categories as well as to relative and absolute risk model forms. The results of the Wong et al. (1983) study were incorporated by assuming that the ratio of the Rinsky-Ott-Wong studies to the Rinsky-Ott studies for the relative risk cumulative dose model was the same as for other model-exposure category combinations and multiplying this ratio by the Rinsky-Ott geometric mean. The age-specific U.S. death rates for 1978 (the most current year available) were used for background leukemia and total death rates. It should be noted that a recently published paper (Rinsky et al., 1987) reported yet another case of leukemia from the study population.

The unit risk should not be used if the air concentration exceeds 100

ug/cu.m, since above this concentration the unit risk may not be appropriate.

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o DISCUSSION OF CONFIDENCE :

The pooled cohorts were sufficiently large and were followed for an adequate time period. The increases in leukemias were statistically significant and dose-related in one of the studies. Wong et al. (1983) disagrees that exposures reported in Rinsky et al. (1981) were within the recommended standards. For the five leukemia deaths in persons with 5 or more years exposure, the author notes that mean exposure levels (range 15-70 ppm) exceeded the recommended standard (25 ppm) in 75% of the work locations sampled. The risk estimate above based on reconsideration of the Rinsky et al. (1981) and Ott et al. (1978) studies is very similar to that of 2.4E-2/ppm (cited in U.S. EPA, 1980) based on Infante et al. (1977a,b), Ott et al. (1978) and Aksoy et al. (1974). It was felt by the authors of U.S. EPA (1985) that the exposure assessment provided by Aksoy was too imprecise to warrant inclusion in the current risk estimate. A total of 21 unit risk estimates were prepared using 6 models and various combinations of the epidemiologic data. These range over slightly more than one order of magnitude. A geometric mean of these estimates is 2.7E-2/ppm. Regression models give an estimate similar to the geometric mean.

CARDR-

o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1980, 1985, 1987

The 1985 Interim Evaluation was reviewed by the Carcinogen Assessment Group.

The 1987 memorandum is an internal document.
DOCUMENT

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o REVIEW DATES : 03/05/87, 10/09/87
o VERIFICATION DATE : 10/09/87
o EPA CONTACTS :

D.L. Bayliss / OHEA -- (202)260-5726

Robert E. McGaughy / OHEA -- (202)260-5889

HAONE-

Appropriate data for calculating a One-day HA are not available. It is recommended that the Ten-day HA of 0.235 mg/L used as the One-day HA.

HATEN-

Ten-day HA -- 2.35E-1 mg/L

NOAEL -- 2.35 mg/kg/day
UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)
Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Deichman et al., 1963

Rats were exposed to benzene for 6 hours/day, 4 days/week by inhalation and their hematology was monitored weekly. By the second week of treatment, hematological impairment was observed at the 2659 mg/cu.m exposure concentration and there was some indication, especially in females, that white blood cells were depressed at the 103 mg/cu.m exposure concentration. No effect was seen when animals were exposed to 96 mg/cu.m for up to 4 months. Based on the conditions of exposure and an assumed absorption factor of 50%, a NOAEL of 2.35 mg/kg/day can be calculated.

HALTC-

A Longer-term HA has not been calculated for benzene because of its potent carcinogenicity.

HALTA-

A Longer-term HA has not been calculated for benzene because of its potent carcinogenicity.

HALIF-

Drinking Water Equivalent Level (DWEL) -- None

Lifetime HA -- None

Benzene is classified in Group A: Human carcinogen. Neither a DWEL nor a Lifetime HA have been calculated for benzene. Refer to Section II of this file for information on the carcinogenicity of this substance.

OLEP -

Odor perception threshold (air) -- 4.9 mg/cu.m.

Odor perception threshold (water) -- 2.0 mg/L.

ALAB -

Analysis of benzene is by a purge-and-trap gas chromatographic procedure used for the determination of volatile aromatic and unsaturated organic compounds in water.

TREAT-

Treatment technologies which will remove benzene from water include granular activated carbon adsorption and air stripping.

HADR -

o HEALTH ADVISORY SOURCE :

Deichman, W.B., W.E. MacDonald and E. Bernal. 1963. The hemopoietic toxicity of benzene vapors. Toxicol. Appl. Pharmacol. 5: 201-224.
DOCUMENT

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o HEALTH ADVISORY REVIEW :

U.S. EPA. 1985. Drinking Water Criteria Document for Benzene. Office of Drinking Water, Washington, DC. (Final draft)

EPA review of HAs in 1985.

Public review of HAs following notification of availability in October, 1985.

Scientific Advisory Panel review of HAs in January, 1986.

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o EPA DRINKING WATER CONTACT :

Jennifer Orme Zavaleta / OST -- (202)260-7586

Edward V. Ohanian / OST -- (202)260-7571

CAA -

Considers technological or economic feasibility? -- YES

Discussion -- Benzene has been listed as a hazardous air pollutant under Section 112 of the Clean Air Act. EPA promulgated NESHAP for benzene from equipment leaks on June 6, 1984 (49 FR 23498) and proposed regulations for coke oven by-product plants.

Reference -- 40 CFR Part 61, Subpart J

EPA Contact -- Emissions Standards Division, OAQPS
(917)541-5571 / FTS 629-5571

WQCHU-

Water and Fish Consumption -- 6.6E-1 ug/L

Fish Consumption Only -- 4.0E+1 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criteria represents a E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC -- 5.3E+3 ug/L
Chronic LEC -- None

Marine:

Acute LEC -- 5.1E+3 ug/L
Chronic LEC -- 7.0E+2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 0 mg/L (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of zero mg/L for benzene is proposed based on carcinogenic effects. In humans, exposure to benzene is associated with myelocytic anemia, thrombocytopenia and leukemia (acute myelogenous and monocytic leukemia). In animals, an increase in tumors and leukemia have been reported. EPA has classified benzene in Group A: sufficient evidence from epidemiological studies.

Reference -- 50 FR 46880 (11/13/85)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value (status) -- 0.005 mg/L (Final, 1987)

Considers technological or economic feasibility? -- YES

Discussion -- EPA has set an MCL based on detection limits.

Monitoring requirements -- All systems to be monitored for four consecutive quarters; repeat monitoring dependent upon detection, vulnerability status and system size.

Analytical methodology -- Gas chromatography (EPA 502.1, 502.2, 503.1); gas chromatographic/mass spectrometry (EPA 524.1, 524.2).

Best available technology -- Packed tower aeration; granular activated carbon.

Reference -- 52 FR 25690 (07/08/87); 56 FR 30266 (07/01/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

FISTD-

No data available

FIREV-

Action -- Voluntary cancellations (1985)

Considers technological or economic feasibility? -- NO

Summary of regulatory action -- All products voluntarily canceled based on concern for oncogenicity, mutagenicity and blood disorders.

Reference -- FR or NTIS No. not available.

EPA Contact -- Special Review Branch / OPP
(703)557-7400 / FTS 557-7400

CERC -

Value (status) -- 10 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for benzene is 10 pounds, based on its potential carcinogenicity. The available data indicate a hazard ranking of medium based on a potency factor of 0.27/mg/kg/day and a weight-of-evidence group A, which corresponds to an RQ of 10 pounds.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - None
IREF - None
CREF - Aksoy, M., S. Erdem and G. Dincol. 1974. Leukemia in shoeworkers exposed chronically to benzene. Blood. 44(6): 837-841.
CREF - Aksoy, M. 1980. Different types of malignancies due to occupational exposure to benzene: A review of recent observations in Turkey. Environ. Res. 23: 181.
CREF - Anderson, D. and C.R. Richardson. 1979. Chromosome gaps are associated with chemical mutagenesis (abstract No. Ec-9). Environ. Mutat. 1: 179.
CREF - IARC (International Agency for Research on Cancer). 1982. Benzene. In: Some industrial chemicals and dyestuffs. IARC Monographs on the evaluation of carcinogenic risk of chemicals to humans. IARC, WHO, Lyon, France. 29: 93-148.
CREF - Infante, P.F., R.A. Rinsky, J.K. Wagoner and R.J. Young. 1977a. Benzene and Leukemia. The Lancet. 2(8043): 867-869.
CREF - Infante, P.F., R.A. Rinsky, J.K. Wagoner and R.J. Young. 1977b. Leukemia in benzene workers. Lancet. 19: 76-78.
CREF - Kissling, M. and B. Speck. 1973. Chromosome aberrations in experimental benzene intoxication. HELV. Med. Acta. 36: 59-66.
CREF - Maltoni, C. and C. Scarnato. 1979. First experimental demonstration of the carcinogenic effects of benzene. Long-term bioassays on Sprague-Dawley Rats by oral administration. Med. Lav. 70: 352-357.
CREF - Maltoni, C., B. Conti and G. Cotti. 1983. Benzene: A multipotential carcinogen. Results of long-term bioassays performed at the Bologna

Institute of Oncology. Am. J. Ind. Med. 4: 589-630.
CREF - Meyne, J. and M.S. Legator. 1980. Sex-related differences in cytogenetic effects of benzene in the bone marrow of Swiss mice. Environ. Mutat. 2: 43-50.
CREF - NTP (National Toxicology Program). 1986. Toxicology and carcinogenesis studies of benzene (CAS No. 71-43-2) in F344/N rats and B6C3F mice (gavage studies). NTP Technical Report Series No. 289. NIH Publication No. 86-2545.
CREF - Ott, M.G., J.C. Townsend, W.A. Fishbeck and R.A. Langner. 1978. Mortality among individuals occupationally exposed to benzene. Arch. Environ. Health. 33: 3-10.
CREF - Rinsky. R.A., R.J. Young and A.B. Smith. 1981. Leukemia in benzene workers. Am. J. Ind. Med. 2: 217-245.
CREF - Rinsky, R.A., A.B. Smith, R. Hornung, et al. 1987. Benzene and Leukemia. New England J. Med. 316(17): 1044-1050.
CREF - Snyder, C.A., M.N. Erlichman, S. Laskin, B.D. Goldstein, and R.E. Albert. 1981. The pharmacokinetics of repetitive benzene exposure at 300 and 100 ppm in AKR mice and Sprague-Dawley rats. Toxicol. Appl. Pharmacol. 57: 164-171.
CREF - U.S. EPA. 1980. Ambient Water Quality Criteria Document for Benzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office (Cincinnati, OH) and Carcinogen Assessment Group (Washington, DC), and the Environmental Research Labs (Corvalis, OR; Duluth, MN; Gulf Breeze, FL) for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-018.
CREF - U.S. EPA. 1985. Interim Quantitative Cancer Unit Risk Estimates Due to Inhalation of Benzene. Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC for the Office of Air Quality Planning and Standards, Washington, DC.
CREF - U.S. EPA. 1987. Memorandum from J. Orme, HEB, CSD/ODW to C. Vogt, Criteria and Standards Division, ODW, June 1987.
CREF - Wong, O., R.W. Morgan and M.D. Whorton. 1983. Comments on the NIOSH study of leukemia in benzene workers. Technical report submitted to Gulf Canada, Ltd., by Environmental Health Associates.
HAREF- Deichman, W.B., W.E. MacDonald and E. Bernal. 1963. The hemopoietic toxicity of benzene vapors. Toxicol. Appl. Pharmacol. 5: 201-224.
HAREF- U.S. EPA. 1985. Drinking Water Criteria Document for Benzene. Office of Drinking Water, Washington, DC. (Final draft)

[IRIS] SS 5 /cf?
USER:

prt dl ncar, car cotni^H^H^Hntinuous

1 - IRIS
IRSN - 19
DATE - 921007
UPDT - 10/07/92, 6 fields
STAT - Oral RfD Assessment (RDO) on-line 06/01/91
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 06/01/91
STAT - Drinking Water Health Advisories (DWHA) on-line 08/01/90
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 04/01/92
IRH - 03/01/88 RDO Dose conversion clarified
IRH - 03/01/88 RDO Principal study corrected
IRH - 03/01/88 RDO Text added
IRH - 03/01/88 RDO Text revised
IRH - 03/01/88 RDO Verification and meeting dates changed
IRH - 03/01/88 CAREV Text corrected
IRH - 03/01/88 CARO Confidence statement revised
IRH - 03/01/88 CARI Confidence statement revised
IRH - 03/01/88 HADV Health Advisory added
IRH - 06/30/88 RDO Primary contact changed
IRH - 12/01/89 RDO Corrected citation year for Condie et al.
IRH - 12/01/89 REFS Bibliography on-line
IRH - 06/01/90 CAA Area code for EPA contact corrected
IRH - 06/01/90 RCRA EPA contact changed
IRH - 08/01/90 HATEN Uncertainty factor text corrected
IRH - 08/01/90 HADR Primary contact changed
IRH - 01/01/91 CAR Text edited
IRH - 01/01/91 CARI Inhalation slope factor removed (global change)
IRH - 03/01/91 RDO Primary contact changed
IRH - 06/01/91 RDO Text edited
IRH - 06/01/91 CAR Text edited
IRH - 06/01/91 MCLG EPA contact changed
IRH - 06/01/91 MCL EPA contact changed
IRH - 08/01/91 CREF Blair et al., 1979 and Milham, 1976 references added
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 04/01/92 CAA CAA regulatory action withdrawn
IRH - 10/01/92 CREF Missing reference added
RLEN - 28123
NAME - Carbon tetrachloride
RN - 56-23-5
SY - Acritet
SY - Benzinoform
SY - Carbona
SY - Carbon chloride
SY - Carbon tet
SY - Carbon tetrachloride
SY - Carbo tetrachloride
SY - Czterochlorek wegla

SY - ENT 4,705
SY - Fasciolin
SY - Flukoids
SY - Freon 10
SY - Halon 104
SY - Mecatorina
SY - Methane tetrachloride
SY - Methane, tetrachloro-
SY - Necatorina
SY - Necatorine
SY - Perchloromethane
SY - R 10
SY - Tetrachloorkoolstof
SY - Tetrachloormetaan
SY - Tetrachlorkohlenstoff, tetra
SY - Tetrachlormethan
SY - Tetrachlorocarbon
SY - Tetrachloromethane
SY - Tetrachlorure de carbone
SY - Tetrachorkohlenstoff uvasol
SY - Tetraclorometano
SY - Tetracloruro di carbonio
SY - Tetrafinol
SY - Tetraform
SY - Tetrasol
SY - Univerm
SY - Ventox
SY - Vermoestricid
SY - WLN: GXGGG.

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Liver lesions Subchronic Rat Gavage Study Bruckner et al., 1986	NOAEL: 1 mg/kg/day (converted to 0.71 mg/kg/day) LOAEL: 10 mg/kg/day (converted to 7.1 mg/kg/day)	1000	1	7E-4 mg/kg/day

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*Conversion Factors: 1 mg/kg/day (NOAEL) x 5/7 = 0.71 mg/kg/day (5 day/week dosing regimen)

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o ORAL RFD STUDIES :

Bruckner, J.V., W.F. MacKenzie, S. Muralidhara, R. Luthra, G.M. Kyle and D. Acosta. 1986. Oral toxicity of carbon tetrachloride: Acute, subacute and

subchronic studies in rats. Fund. Appl. Toxicol. 6(1): 16-34.

Male Sprague-Dawley rats were given 1, 10, or 33 mg carbon tetrachloride/kg/day by corn oil gavage, 5 days/week for 12 weeks. Liver lesions, as evidenced by mild centrilobular vacuolization and statistically significant increases in serum sorbitol dehydrogenase activity, were observed at the 10 and 33 mg/kg/day dosesm in a dose-related manner. Therefore, the LOAEL was established at 10 mg/kg/day (converted to 7.1 mg/kg/day) and the NOAEL was 1 mg/kg/day (converted to 0.71 mg/kg/day).

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o ORAL RFD UNCERTAINTY :

UF -- UF allows for interspecies and intrahuman variability and extrapolation from subchronic to chronic duration of exposure.

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o ORAL RFD MODIFYING FACTOR :

MF = None

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o ORAL RFD COMMENTS :

A 1983 draft of the Bruckner et al. (1986) study was used as the basis for the RfD by the RfD Work Group at a 05/20/85 verification meeting. When this study was subsequently published (Bruckner et al., 1986), no change to the verified value was required.

Subchronic studies in mice gavaged with carbon tetrachloride in corn oil (Condie et al., 1986; Hayes et al., 1985) support the critical effect and the magnitude of the NOAEL and LOAEL found in the rat studies. Additional studies (Alumot et al., 1976; NCI, 1976) in rats lend moderate support to the choice of a NOAEL in the chosen rat study.

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o ORAL RFD CONFIDENCE :

Study -- High
Data Base -- Medium
RfD -- Medium

The principal study was well conducted and good dose-response was observed in the liver, which is the target organ for carbon tetrachloride toxicity; thus, high confidence was assigned. Four additional subchronic studies support the RfD, but reproductive and teratology endpoints are not well investigated; thus, the data base rates a medium confidence. Medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

U.S. EPA. 1985. Drinking Water Criteria Document for Carbon Tetrachloride. Office of Drinking Water, Washington, DC.

Public review of RfD following ODW proposal of RMCL in June 1984.

Science Advisory Board review of RfD on January 14, 1986.

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o REVIEW DATES : 05/20/85
o VERIFICATION DATE : 05/20/85
o EPA CONTACTS :

Krishan Khanna / OST -- (202)260-7588

Michael L. Dourson / OST -- (513)569-7544

CAREV-
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Carcinogenicity in rats, mice, and hamsters
o HUMAN CARCINOGENICITY DATA :

Inadequate. There have been three case reports of liver tumors developing after carbon tetrachloride exposure. Several studies of workers (Milham, 1976; Blair et al., 1979) who may have used carbon tetrachloride have suggested that these workers may have an excess risk of cancer.

---

o ANIMAL CARCINOGENICITY DATA :

Sufficient. Carbon tetrachloride has produced hepatocellular carcinomas in rats, mice, and hamsters, the species evaluated to date.

Hepatocellular carcinomas developed in Osborne-Mendel, Japanese, and Wistar rats, but not Sprague-Dawley or Black rats, following s.c. injection of carbon tetrachloride. Hyperplastic nodules were noted in Buffalo rats treated s.c. (Reuber and Glover, 1967a,b, 1970). Sensitivity varied among strains, and trends in incidence appeared inversely related to severity of cirrhosis.

Fifty Osborne-Mendel rats/sex were administered carbon tetrachloride by corn oil gavage at 47 and 94 mg/kg/injection for males and 80 and 159 mg/kg for females 5 times/week for 78 weeks. At 110 weeks, only 7/50 high-dose males and 14/50 high-dose females survived; 14/50 low-dose males and 20/50 low-dose females survived. The incidence of hepatocellular carcinomas was increased in animals exposed to carbon tetrachloride as compared with pooled colony controls. The apparent decrease in the incidence of hepatocellular carcinomas in high-dose female rats compared with the low-dose females (1/14 vs. 4/20, respectively) was attributed by the authors to increased lethality before tumors could be expressed (NCI, 1976a,b, 1977).

In this same study, using the same dosing schedule, male and female B6C3F1

mice received 1250 or 2500 mg/kg carbon tetrachloride. The incidences of hepatocellular carcinomas in males were 5/77, 49/49, and 47/48 in the control, low- and high-dose groups, respectively, and 1/80, 40/40, and 43/45 in the control, low- and high-dose groups, respectively.

Carbon tetrachloride administered by gavage has also been shown to produce neoplastic changes in livers of five additional strains of mice (C3H, A, Y, C, and L) (Andervont, 1958; Edwards, 1941; Eschenbrenner and Miller 1943; Edwards and Dalton, 1942; Edwards et al., 1942). In the last study, 56 male and 19 female L mice, which have a low incidence of spontaneous hepatomas, were treated with 0.1 mL of 40% carbon tetrachloride 2 or 3 times/week over 4 months, for a total of 46 treatments. Animals were killed 3 to 3.5 months after the last treatment. The combined hepatoma incidence of treated male mice was 47% (7/15 vs. 2/71 in the untreated male controls); treated females showed and incidence of 38% (3/8 vs. 0/81 in the untreated female controls).

As part of a larger study of liver carcinogens, Della Porta et al. (1961) treated Syrian golden hamsters (10/sex/dose) with carbon tetrachloride by gavage, weekly for 30 weeks. For the first 7 weeks, 0.25 mL of 0.05% carbon tetrachloride in corn oil was administered; this dose was halved for the remainder of the exposure period. All animals were observed for an additional 25 weeks. All of the 10 hamsters that were killed or dying between weeks 43 and 55 had liver cell carcinomas, compared with 0 in controls.

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o SUPPORTING DATA :

Carbon tetrachloride was not mutagenic to either S. typhimurium or E. coli (McCann et al., 1975; Simmon et al., 1977; Uehleke et al., 1976). At low concentrations, carbon tetrachloride did not produce chromatid or chromosomal aberrations in an epithelial cell line derived from rat liver (Dean and Hodson-Walker, 1979). In vivo unscheduled DNA synthesis assays have likewise been negative in male Fischer 344 rats (Mirsalis and Butterworth, 1980; Mirsalis et al., 1982). Carbon tetrachloride produced mitotic recombination and gene conversion in S. cerevisiae, but only at concentrations which reduced viability to 10% (Callen et al., 1980). Carbon tetrachloride may be metabolized to reactive intermediates capable of binding to cellular nucleophilic macromolecules. Negative responses in bacterial mutagenicity assays may have been due to inadequate metabolic activation in the test systems.

CARO -
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Carcinogenicity in rats, mice, and hamsters
o ORAL SLOPE FACTOR : 1.3E-1 per (mg/kg)/day
o DRINKING WATER UNIT RISK : 3.7E-6 per (ug/L)
o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra

risk

o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	3E+1 ug/L
E-5 (1 in 100,000)	3E+0 ug/L
E-6 (1 in 1,000,000)	3E-1 ug/L

---

o ORAL DOSE-RESPONSE DATA :

Tumor Type -- Hepatocellular carcinomas/hepatomas
Test Animals -- various, see table
Route -- gavage
Reference -- several, see table

Administered Dose (mg/day)	Human Equivalent Dose (mg/kg)/day	Tumor Incidence	Unit Risk per (ug/L)	Reference
Hamster/Syrian, male and female				
0	0	0/80	3.4E-5	Della
0.95	1.02	10/19		Porta et al., 1961
Mouse/L, male and female				
0	0	2/152	9.4E-6	Edwards
15	2.3	34/73		et al., 1942
Mouse/B6C3F1, male and female				
0	0	6/157	1.8E-6	NCI,
21	55.4	89/89		1976a,b,
42	110.8	90/93		1977
Rat/Osborne-Mendel:				
M, F 0	0	0/37	3.1E-7	NCI,
M 11	4.5	2/45		1976a,b,
F 18	7.4	4/46		1977
M 21	8.7	2/47		
F 36	14.9	1/30		

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o ADDITIONAL COMMENTS :

A geometric mean was calculated from the unit risks derived from the four data sets above. Della Porta et al. (1961) did not report controls in this study, but did give incidence rate for vehicle controls in an earlier study. Animal doses are TWA.

The unit risk should not be used if the water concentration exceeds 3E+3 ug/L, since above this concentration the unit risk may not be appropriate.

---

o DISCUSSION OF CONFIDENCE :

The studies used were all deficient in some respect, precluding the choice of any one study as most appropriate. For all studies, data from males and females were combined because of the small sample sizes. In the first and second studies (Della Porta et al., 1961; Edwards et al., 1942) one dose was tested. Della Porta et al. (1961) did not report concurrent control incidence. In the NCI (1976a,b) studies, tumor incidence in the mice was virtually 100%, and goodness-of-fit criteria were not satisfied for the multistage model. Tumor incidence in rats in these studies was higher at low doses, presumably because early mortality at higher doses precluded tumor formation. The studies lacked pharmacokinetic data. However, a common biological mechanism, cell death and regeneration, leading to development of the same tumor type, was suggested by observations in all the studies. Since the risk estimates from these data (across 3-4 species and strains) only vary by 2 orders of magnitude, a geometric mean was derived as the risk estimate to accommodate the several study deficiencies.

CARI -

o CLASSIFICATION : B2; probable human carcinogen

o BASIS FOR CLASSIFICATION : Carcinogenicity in rats, mice, and hamsters

o INHALATION UNIT RISK : 1.5E-5 per (ug/cu.m)

o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk

o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	7E+0 ug/cu.m
E-5 (1 in 100,000)	7E-1 ug/cu.m
E-6 (1 in 1,000,000)	7E-2 ug/cu.m

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o INHALATION DOSE-RESPONSE DATA :

The inhalation risk estimates were calculated from the oral exposure data in CARO.

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o ADDITIONAL COMMENTS :

Inhalation risk was calculated assuming an air intake of 20 cu.m/day and 40% absorption rate by humans (U.S. EPA, 1984). This absorption coefficient was based on 30% inhalation in monkeys, and 30% and 57-65% inhalation in humans. A range of estimates of unit risk for inhalation exposures for the

four studies cited above was determined, with 1.5E-5 per (ug/cu.m) calculated as the geometric mean for the unit risk.

The unit risk should not be used if the air concentration exceeds 7E+2 ug/cu.m, since above this concentration the unit risk may not be appropriate.

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o DISCUSSION OF CONFIDENCE :

See CARO.

CARDR-

o CARCINOGENICITY SOURCE :

U.S. EPA. 1984. Health Assessment Document for Carbon Tetrachloride. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA 600/8/82-001F.

The 1984 Health Assessment Document for Carbon Tetrachloride received Agency and external review.

DOCUMENT

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o REVIEW DATES : 11/12/86, 12/04/86
o VERIFICATION DATE : 12/04/86
o EPA CONTACTS :

Jean C. Parker / OHEA -- (202)260-5898

Arthur Chiu / OHEA -- (202)260-5898

HAONE-

One-day HA -- 4E+0 mg/L

NOAEL -- 40 mg/kg/day
UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)
Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Bruckner et al., 1986

Rats were administered single oral doses of carbon tetrachloride. Doses of 80 mg/kg and higher caused changes in liver enzymes (BUN, GPT, SDH, OCT) and histopathologic liver and kidney changes. A dose of 40 mg/kg produced

no effects and is identified as the NOAEL.

HATEN-

Ten-day HA -- 1.6E-1 mg/L

LOAEL -- 16 mg/kg/day
UF -- 1000 (allows for interspecies and intrahuman variability with the use of a LOAEL from an animal study)
Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Bruckner et al., 1986

Rats were administered nine doses of carbon tetrachloride by gavage over an 11-day period. The lowest dose tested (20 mg/kg/day) produced significant changes in serum enzyme levels and hepatic midzonal vacuolation. Higher doses caused more extensive liver damage. A LOAEL of 16 mg/kg/day is established after adjustment for the treatment schedule.

HALTC-

LONGER-TERM HEALTH ADVISORY FOR A CHILD

Longer-term (Child) HA -- 7.1E-2 mg/L

NOAEL -- 0.71 mg/kg/day
UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)
Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Bruckner et al., 1986

Rats were administered carbon tetrachloride by gavage, 5 times weekly for 12 weeks, at doses of 1, 10, or 33 mg/kg/day. Doses of 10 and 33 mg/kg/day were hepatotoxic (changes in serum enzyme levels, centrilobular vacuolation, and necrosis). The NOAEL of 1 mg/kg/day, based on a 7 days/week dosing regimen, is equivalent to 0.71 mg/kg/day.

HALTA-

Longer-term (Adult) HA -- 2.5E-1 mg/L

NOAEL -- 0.71 mg/kg/day
UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)

Assumptions -- 2 L/day water consumption for a 70-kg adult

Principal Study -- Bruckner et al., 1986

This study is the same as that for the longer-term (child) HA.

HALIF-

Drinking Water Equivalent Level (DWEL) -- 2.5E-2 mg/L

Assumptions -- 2 L/day water consumption for a 70-kg adult

RfD Verification Date -- 07/08/85 (see Section I.A. in this file)

Lifetime HA -- None.

Principal Study (DWEL) -- Bruckner et al., 1986

This study was used in the derivation of the oral chronic RfD; see the RfD Section for a description. Carbon tetrachloride is considered to be a probable human carcinogen. Refer to the carcinogenicity assessment section for information on the carcinogenicity of this substance.

OLEP -

Odor perception threshold -- 0.52 mg/L.

ALAB -

Analysis of carbon tetrachloride is by a purge-and-trap gas chromatographic procedure used for the determination of volatile organohalides in drinking water. Confirmatory analysis is by mass spectrometry.

TREAT-

Treatment techniques which will remove carbon tetrachloride from drinking water include granular activated carbon adsorption, boiling, and air stripping. Conventional treatment processes (coagulation, sedimentation, filtration), even when augmented by the addition of powdered activated carbon, provide little removal of carbon tetrachloride.

HADR -
o HEALTH ADVISORY SOURCE :

Bruckner, J.V., W.F. MacKenzie, S. Muralidhara, R. Luthra, G.M. Kyle and D.

Acosta. 1986. Oral toxicity of carbon tetrachloride: Acute, subacute and subchronic studies in rats. Fund. Appl. Toxicol. 6(1): 16-34.
DOCUMENT

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o HEALTH ADVISORY REVIEW :

U.S. EPA. 1985. Final Draft of the Drinking Water Criteria Document on Carbon Tetrachloride. Office of Drinking Water, Washington, DC.

EPA review of HAs in 1985.

Public review of HAs following notification of availability in October, 1985.

Scientific Advisory Panel review of HAs in January, 1986.

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o EPA DRINKING WATER CONTACT :

Jennifer Orme / OST -- (202)260-7586

Edward V. Ohanian / OST -- (202)260-7571

WQCHU-

Water and Fish Consumption: 4.0E-1 ug/L

Fish Consumption Only: 6.94E+0 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For maximum protection from the potential carcinogenic properties of this chemical, the ambient concentration should be zero. However, zero may not be attainable at this time so the recommended criteria represents a E-6 estimated incremental increase in cancer risk over a lifetime.

Reference -- 45 FR 791318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC -- 3.52E+4 ug/L

Chronic -- None

Marine:

Acute LEC -- 5.0E+4 ug/L
Chronic -- None

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 791318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 0 mg/L (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0 mg/L for carbon tetrachloride is proposed based on carcinogenic effects. Carbon tetrachloride has been shown to be carcinogenic in rats, mice, and hamsters through oral exposure. Hepatocellular carcinomas in several studies have been observed. EPA has classified carbon tetrachloride in Group B2: sufficient evidence in animals and inadequate evidence in humans.

Reference -- 50 FR 46880 (11/13/85)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value (status) -- 0.005 mg/L (Final, 1987)

Considers technological or economic feasibility? -- YES

Discussion -- EPA has set an MCL based on detection limits.

Monitoring requirements -- All systems to be monitored for four consecutive quarters; repeat monitoring dependent upon detection and vulnerability status and system size.

Analytical methodology -- Gas chromatography (EPA 502.1, 502.2, 503.1); gas chromatographic/mass spectrometry (EPA 524.1, 524.2).

Best available technology -- Packed tower aeration; granular activated carbon.

Reference -- 52 FR 25690 (07/08/87); 56 FR 30266 (07/01/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

FISTD-

No data available

FIREV-

Action -- Voluntary cancellation (1986)

Considers technological or economic feasibility? -- No

Summary of regulatory action -- Voluntary cancellations were made in 1985 also. For specific details on the Special Review process for this active ingredient please call the EPA Contact.

Reference -- 50 FR 42997 (10/23/85); 54 FR 41004 (11/12/86); 52 FR 38200 (10/14/87) Proposed exemption revocation; 54 FR 6126 (02/08/89) Tolerance exemption on grains

EPA Contact -- Special Review Branch / OPP -- (703)557-7400 / FTS 557-7400

CERC -

Value (status) --10 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ for carbon tetrachloride is based on potential carcinogenicity. Available data indicate a hazard ranking of medium based upon a potency factor of 59.9 mg/kg/day and assignment to weight-of-evidence group B2. This corresponds to an RQ of 10 pounds.

Reference -- 52 FR 8140 (03/16//87); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

___IV.E.1. TSCA, SECTION 6

Status -- Advance Notice of Proposed Rulemaking (ANPR) (1985)

Discussion -- EPA is developing a comprehensive and integrated strategy for a regulatory investigation of six solvents, including carbon tetrachloride.

Reference: 50 FR 42005 (10/17/85); 40 CFR 754

EPA Contact -- Chemical Control Division / OTS (202)260-3749 / FTS 260-3749

OREF - Alumot E., E. Nachtomi, E. Mandel and P. Holstein. 1976. Tolerance and acceptable daily intake of chlorinated fumigants in the rat diet. Food Cosmet. Toxicol. 14: 105-110.
OREF - Bruckner, J.V., S. Muralidhara, R. Luthra, G.M. Kyle, W.F. MacKenzie and D. Acosta. 1983. Oral toxicity of carbon tetrachloride: Acute, subacute and subchronic studies in rats. University of Georgia, Athens, GA. (Draft)
OREF - Bruckner, J.V., W.F. MacKenzie, S. Muralidhara, R. Luthra, G.M. Kyle and D. Acosta. 1986. Oral toxicity of carbon tetrachloride: Acute, subacute and subchronic studies in rats. Fund. Appl. Toxicol. 6(1): 16-34.
OREF - Condie, L.W., R.D. Laurie, T. Mills, M. Robinson and J.P. Bercz. 1986. Effect of gavage vehicle on hepatotoxicity of carbon tetrachloride in CD-1 mice: Corn oil versus Tween-60 aqueous emulsion. Fund. Appl. Toxicol. 7(2): 199-206.
OREF - NCI (National Cancer Institute). 1976. Report on the Carcinogenesis Bioassay of Chloroform. Carcinogenesis Program, Division of Cancer Cause and Prevention. March 1.
OREF - U.S. EPA. 1985. Drinking Water Criteria Document for Carbon Tetrachloride. Office of Drinking Water, Washington, DC. PB86-118155.
IREF - None
CREF - Andervont, H.B. 1958. Induction of hepatomas in strain C3H mice with 4-o- tolylazo-o-toluidine and carbon tetrachloride. J. Natl. Cancer Inst. 20(2): 431-438.
CREF - Blair, A., P. Decoufle and D. Grauman. 1979. Causes of death among laundry and dry cleaning workers. Am. J. Public Health. 69(5): 508-511.
CREF - Callen, D.F., C.R. Wolf and R.M. Philpot. 1980. Cytochrome P-450 mediated genetic activity and cytoxicity of seven halogenated aliphatic hydrocarbons in Saccharomyces cerevisiae. Mutat. Res. 77: 55-63.
CREF - Dean, B.J. and G. Hodson-Walker. 1979. An in vitro chromosome assay using cultured rat-liver cells. Mutat. Res. 64: 329-337.
CREF - Della Porta, G., B. Terracini and P. Shubik. 1961. Induction with carbon tetrachloride of liver cell carcinomas in hamsters. J. Natl. Cancer Inst. 26(4): 855-863.
CREF - Edwards, J.E. and H.A. Dalton. 1942. Induction of cirrhosis of the liver and of hepatomas in mice with carbon tetrachloride. J. Natl. Cancer Inst. 3: 19-41.
CREF - Edwards, J.E., W.E. Heston and H.A. Dalton. 1942. Induction of the carbon tetrachloride hepatoma in strain L. mice. J. Natl. Cancer Inst. 3: 297-301.
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CREF - Milham, S. 1976. Neoplasia in the wood and pulp industry. Ann. New

York Acad. Sci. 271: 294-300.
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CREF - Mirsalis, J.C., C.K. Tyson and B.E. Butterworth. 1982. Detection of genotoxic carcinogens in the in vivo-in vitro hepatocyte DNA repair assay. Environ. Mutagen. 4: 553-562.
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CREF - Reuber, M.D. and E.L. Glover. 1967a. Hyperplastic and early neoplastic lesions of the liver in buffalo strain rats of various ages given subcutaneous carbon tetrachloride. J. Natl. Cancer Inst. 38(6): 891-899.
CREF - Reuber, M.D. and E.L. Glover. 1967b. Cholangiofibrosis in the liver of Buffalo strain rats injected with carbon tetrachloride. Br. J. Exp. Pathol. 48(3): 319-322.
CREF - Reuber, M.D. and E.L. Glover. 1970. Cirrhosis and carcinoma of the liver in male rats given subcutaneous carbon tetrachloride. J. Natl. Cancer Inst. 44(2): 419-427.
CREF - Simmon, V.F., K. Kauhanen and R.G. Tardiff. 1977. Mutagenic activity of chemicals identified in drinking water. In: Progress in Genetic Toxicology, D. Scott, B.A Bridges and F.H. Sobels, Ed. Elsevier/North-Holland Biomedical Press, New York. p. 249-258.
CREF - Uehleke, H., H. Greim, M. Kramer and T. Werner. 1976. Covalent binding of haloalkanes to liver constituents, but absence of mutagenicity on bacteria in a metabolizing test system mutation. Research. 38: 114.
CREF - U.S. EPA. 1984. Health Assessment Document for Carbon Tetrachloride. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA 600/8/82-001F.
HAREF- Bruckner, J.V., W.F. MacKenzie, S. Muralidhara, R. Luthra, G.M. Kyle and D. Acosta. 1986. Oral toxicity of carbon tetrachloride: Acute, subacute and subchronic studies in rats. Fund. Appl. Toxicol. 6(1): 16-34.
HAREF- U.S. EPA. 1985. Drinking Water Criteria Document for Carbon Tetrachloride. Office of Drinking Water, Washington, DC. (Final draft)

[IRIS] SS 6 /cf?
USER:

prt dl ncar, car continuous

1 - IRIS
IRSN - 522
DATE - 920501
UPDT - 05/01/92, 52 fields
STAT - Oral RfD Assessment (RDO) pending
STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) pending 05/01/92
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) no data
IRH - 08/01/91 RDO Oral RfD now under review
IRH - 08/01/91 RDI Inhalation RfC now under review
IRH - 05/01/92 CAR Carcinogenicity assessment now under review
RLEN - 1063
NAME - Chloromethane
RN - 74-87-3
SY - ARTIC
SY - CASWELL NO. 557
SY - CHLOOR-METHAAN [DUTCH]
SY - CHLOR-METHAN [GERMAN]
SY - CHLOROMETHANE
SY - CHLORURE DE METHYLE [FRENCH]
SY - CHLORURE DE METHYLE [FRENCH]
SY - CLOROMETANO [ITALIAN]
SY - CLORURO DE METILO [SPANISH]
SY - CLORURO DI METILE [ITALIAN]
SY - EPA PESTICIDE CHEMICAL CODE 053202
SY - HSDB 883
SY - METHANE, CHLORO-
SY - METHYL CHLORIDE
SY - METHYLCHLORID [GERMAN]
SY - METYLU CHLOREK [POLISH]
SY - MONOCHLOROMETHANE
SY - R 40
SY - RCRA WASTE NUMBER U045
SY - UN 1063

RDO -
o ORAL RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

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prt dl ncar, car continuous

1 - IRIS
IRSN - 265
DATE - 940801
STAT - Oral RfD Assessment (RDO) on-line 01/01/92
STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) message 08/01/94
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 03/01/88 RDO Text clarified paragraph 1
IRH - 12/01/89 RDI Inhalation RfD now under review
IRH - 04/01/90 RDO ABC, 1986 corrected to U.S. EPA, 1986
IRH - 04/01/90 REFS Bibliography on-line
IRH - 06/01/90 RDO Oral RfD summary noted as pending change
IRH - 06/01/90 RCRA EPA contact changed
IRH - 09/01/91 RDO Oral RfD no longer pending change; no changes made
IRH - 12/01/91 RDO Text revised; additional study added
IRH - 12/01/91 RDO Text significantly revised; additional studies added
IRH - 12/01/91 OREF Oral RfD references revised to reflect new text
IRH - 01/01/92 RDO Citation year corrected for Schnegg and Kirchgessner
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 01/01/92 OREF Citation year corrected for Schnegg and Kirchgessner
IRH - 03/01/94 CAR Carcinogenicity assessment now under review
IRH - 08/01/94 CAR Message added
IRH - 08/01/94 CAR Work group review date added
RLEN - 27287
NAME - Nickel, soluble salts
RN - 7440-02-0
SY - C.I. 77775
SY - NICHEL
SY - Nickel
SY - Nickel, soluble salts

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Decreased body and organ weights	NOAEL: 100 ppm diet (5 mg/kg/day)	300	1	2E-2 mg/kg/day
Rat Chronic Oral Study	LOAEL: 1000 ppm diet (50 mg/kg/day)			

Ambrose et al., 1976

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*Conversion Factors: 1 ppm = 0.05 mg/kg/day (assumed rat consumption)

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o ORAL RFD STUDIES :

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

Ambrose et al. (1976) reported the results of a 2-year feeding study using rats given 0, 100, 1000 or 2500 ppm nickel (estimated as 0, 5, 50 and 125 mg Ni/kg bw) in the diet. Body weights in the high-dose male and female rats were significantly decreased compared with controls. Body weight was also reduced at 1000 ppm. This reduction was significant for females at week 6 and from weeks 26 through 104, whereas males showed body weight reduction only at 52 weeks. Groups of female rats on the 1000 or 2500 ppm nickel diets (50 and 125 mg Ni/kg bw) had significantly higher heart-to-body weight ratios and lower liver-to-body weight ratios than controls. No significant effects were reported at 100 ppm (5 mg Ni/kg bw). The dose of 1000 ppm (50 mg Ni/kg bw) represents a LOAEL for this study, while the dose of 100 ppm (5 mg Ni/kg bw) is a NOAEL. In this study, 2-year survival was poor, particularly in control rats of both sexes (death: 44/50), raising some concern about the interpretation of the results of this study. A subchronic study conducted by American Biogenics Corp. (ABC, 1986) also found 5 mg/kg/day to be a NOAEL, which supports the Ambrose et al. (1976) chronic NOAEL of 5 mg/kg/day.

Dietary exposure of dogs to 2500 ppm Ni (about 63 mg/kg/day) resulted in depressed body weight gain; no effects were seen at either 100 ppm (about 2.5 mg/kg/day) or 1000 ppm Ni (about 25 mg/kg/day) in the diet (Ambrose et al., 1976). This study demonstrates that rats are the more sensitive of the two species.

ABC (1986) conducted the 90-day study with nickel chloride in water (0, 5, 35 and 100 mg/kg/day) administered by gavage to both male and female CD rats (30 animals/sex/group). The data generated in this study included clinical pathology, ophthalmological evaluations, serum biochemistry, body and organ weight changes and histopathological evaluations of selected organs (heart, kidney, liver).

The body weight and food consumption values were consistently lower than those of controls for the 35 and 100 mg/kg/day dosed males. Female rats in both high-dose groups had lower body weights than controls, but food consumption was unaffected by the test article. Clinical signs of toxicity, such as lethargy, ataxia, irregular breathing, cool body temperature, salivation and discolored extremities, were seen primarily in the 100 mg/kg/day group; these signs were less severe in the 35 mg/kg/day group. The 5 mg/kg/day group did not show any significant clinical signs of toxicity. There was 100% mortality in the high-dose group; 6/30 males and 8/30 females died in the mid-dose group (35 mg/kg/day). Histopathologic evaluation indicated that deaths of 3/6 males and 5/8 females in the mid-dose group were due to gavage errors. At sacrifice, kidney, liver and spleen weights for 35 mg/kg/day treated males and right kidney weights for 35 mg/kg/day treated females were significantly lower than controls. Based on the results obtained in this study, the 5 mg/kg/day

nickel dose was a NOAEL, whereas 35 mg/kg/day was a LOAEL for decreased body and organ weights.

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o ORAL RFD UNCERTAINTY :

UF -- An uncertainty factor of 10 is used for interspecies extrapolation and 10 to protect sensitive populations. An additional uncertainty factor of 3 is used to account for inadequacies in the reproductive studies (RTI, 1987; Ambrose et al., 1976; Smith et al., 1990) (see Additional Comments section). During the gestation and postnatal development of F1b litters in the RTI (1987) study, temperatures were about 10 degrees F higher than normal at certain times, which makes evaluation of this part of the reproductive study impossible. In the Ambrose et al. (1976) study, statistical design limitations included small sample size and use of pups rather than litters as the unit for comparison. There were also problems with the statistical analysis of the Smith et al. (1990) study.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

In addition to the effects on organ weights described in the critical study, two other sensitive endpoints exist: neonatal mortality and dermatotoxicity. While no reproductive effects have been associated with nickel exposure to humans, several studies in laboratory animals have demonstrated fetotoxicity. These studies are described below.

Following the reproductive studies is a discussion of nickel-induced dermatotoxicity in hypersensitive humans. While nickel has long been recognized as a contact irritant, many studies have also demonstrated dermal effects in sensitive humans resulting from ingested nickel. The weight-of-evidence from these studies indicates that ingested nickel may invoke an eruption or worsening of eczema; however, a dose-response relationship is difficult to establish. A few representative studies and review articles are cited below.

While the systemic toxicity data (as manifested in organ weight changes) was used as the critical study for the RfD determination, the reproductive/fetotoxicity and the dermatotoxicity were both considered as possible endpoints upon which to base the quantitative risk assessment of nickel. The data for effects on the latter two endpoints do not demonstrate consistent dose-response relationships, and in both cases the available studies are sufficiently flawed so as to prevent their selection as the basis for the oral RfD. It is noted, however, that the RfD based on the Ambrose et al. (1976) study is considered to be protective of all endpoints with the possible exception of hypersensitive individuals as described below.

In addition to the 2-year feeding study used as the basis for the RfD, Ambrose

et al. (1976) also reported reproductive toxicity of nickel. The study had some statistical design limitations including small sample size and use of pups rather than litters as the unit for comparison. Furthermore, the results were equivocal and did not clearly define a NOAEL or LOAEL. Because nickel was administered in a laboratory chow diet rather than drinking water, quantifying analogous nickel exposure via drinking water was problematic.

In a 2-generation study (RTI, 1987) nickel chloride was administered in drinking water to male and female CD rats (30/sex/dose) at dose levels of 0, 50, 250 and 500 ppm (0, 7.3, 30.8 and 51.6 mg/kg/day, estimated) for 90 days before breeding (10 rats/sex/group comprised a satellite subchronic nonbreeder group). At the 500 ppm dose level there was a significant decrease in the Po maternal body weight, along with absolute and relative liver weights. Thus, 250 ppm (30.8 mg/kg/day) was a NOAEL for Po breeders. Histopathology was performed for liver, kidney, lungs, heart, pituitary, adrenals and reproductive organs to make this assessment. This NOAEL is higher than the NOAEL derived from the chronic Ambrose et al. (1976) and subchronic gavage (ABC, 1986) assays.

In the RTI (1987) F1a generation (postnatal days 1-4) at the 500 ppm dose level the number of live pups/litter was significantly decreased, pup mortality was significantly increased, and average pup body weight was significantly decreased in comparison with controls. Similar effects were seen with F1b litters of Po dams exposed to 500 ppm nickel. In the 50 and 250 ppm dose groups increased pup mortality and decreased live litter size was observed in the F1b litters. However, these effects seen with F1b litters are questionable because the room temperature tended to be 10 degrees F higher than normal at certain times (gestation-postnatal days) along with much lower levels of humidity. As evidenced in the literature, temperatures that are 10 degrees F above normal during fetal development cause adverse effects (Edwards, 1986). Therefore, the above results seen at 50 and 250 ppm cannot be considered to be genuine adverse effects.

F1b males and females of the RTI (1987) study were randomly mated on postnatal day 70 and their offspring (F2a and F2b) were evaluated through postnatal day 21. This phase included teratological evaluations of F2b fetuses. Evaluation of the data indicated that the 500 ppm dose caused significant body weight depression of both mothers and pups, and increased neonatal mortality during the postnatal development period. The intermediate dose, 250 ppm nickel, produced transient depression of maternal weight gain and water intake during gestation of the F2b litters. The 50 ppm nickel exposure caused a significant increase in short ribs (11%). However, since this effect was not seen in both the higher dose groups, the reported incidence of short ribs in the 50 ppm group is not considered to be biologically significant.

Schroeder and Mitchener (1971) conducted a 3-generation study in which 5 mating pairs of rats were provided drinking water containing 5 mg Ni/L (estimated as 0.43 mg/kg bw). Results of this study indicated significant increases in neonatal mortality and in the number of runts born to exposed rats compared with controls. The major weakness of this study, however, is

that the end result is based on a total of five matings. The matings were not randomized and the males were not rotated. The Schroeder and Mitchener (1971) study was conducted in an environmentally controlled facility where rats had access to food and water containing minimal levels of essential trace metals. Because of the interactions of nickel with other trace metals, the restricted exposure to trace metals (chromium was estimated as inadequate) may have contributed to the toxicity of nickel.

Smith et al. (1990) also studied the reproductive and fetotoxic effects of nickel. Four groups of 34 female Long-Evans rats were given drinking water containing nickel chloride in the following concentrations of nickel: 0, 10, 50 or 250 ppm (0, 1.3, 6.8 or 31.6 mg/kg/day) for 11 weeks prior to mating and during two successive gestation periods (G1, G2) and lactation periods (L1, L2). Maternal body weight gain was reduced during G1 in mid- and high-dose females. The reproductive performance of the exposed rats was not affected. Pup birth weight was unaltered by treatment, and weight gain was reduced only in male pups exposed to 50 ppm nickel during L1. The most significant toxicological finding was the increased incidence of perinatal mortality. The proportion of dead pups per litter was elevated at the high dose in L1 and at 10 and 250 ppm in L2. While the perinatal mortality reported in this study is consistent with other reproductive studies on nickel, it is hard to define a NOAEL and LOAEL because of the absence of a clear dose-response trend at the lower doses.

Many studies have been published regarding nickel sensitivity in humans. Of the general population, approximately 8-10% of women and 1-2% of men demonstrate a sensitivity to nickel as determined by a patch test (North American Contact Dermatitis Group, 1973; Prystowsky et al., 1979). Initial sensitization to nickel is believed to result from dermal contact, but recurring flares of eczema, particularly of the hands, may be triggered by ingestion.

The human studies described below are difficult to interpret for several reasons: very small numbers of subjects (mostly women already determined to be sensitive to nickel by a patch test) were used in the studies; many investigators reported a placebo effect; many studies were not conducted in a double-blind manner, thereby introducing investigator bias; and it was often not specified whether subjects had been fasted overnight or whether there were other dietary restrictions. It is important to note that the way in which nickel is consumed may greatly affect its bioavailability. Sunderman et al. (1989) demonstrated that 27+/-17% of the nickel in drinking water was absorbed by healthy humans whereas only 0.7+/-0.4% of the same dose of nickel ingested in food was absorbed (a 40-fold difference). One final point to bear in mind in interpreting these studies is that the subjects were generally given a bolus dose of nickel. The absorption and biokinetics following such an exposure may be quite different from an exposure which is given incrementally throughout the day.

Following an overnight fast, groups of 5 nickel-sensitive women were given 100 mL of water along with one oral dose of nickel sulfate containing 0.6, 1.25 or

2.5 mg nickel (Cronin et al., 1980). The clinical response was observed for the next 24 hours. Worsening of hand eczema was reported in 2/5 female subjects that received 0.6 mg, 3/5 at 1.25 mg and 5/5 at 2.5 mg. Erythema was observed in 1/5 (0.6 mg), 4/5 (1.25 mg) and 4/5 (2.5 mg) women. While there appears to be a good dose-response relationship, this study did not report controls. The response observed at the lowest dose may well be within background levels.

Numerous other studies have been conducted to attempt to establish the relationships between nickel exposure and dermal irritation. Kaaber et al. (1978, 1979) reported worsening of eczema following an oral challenge with 2.5 mg nickel. In the 1978 study, 17/28 subjects experienced aggravation of dermatitis following nickel ingestion. Nine of the 17 that experienced adverse effects from the nickel found that their condition improved when they adopted a low nickel diet. In the 1979 study 9/14 subjects responded negatively to nickel treatment.

Studies conducted by Gawrodger et al. (1986), Burrows et al. (1981) and Jordan and King (1979) offer different results. Jordan and King's double blind, placebo controlled investigation suggested that 0.5 mg supplement to a normal diet was safe with the possible exception of extremely sensitive individuals. Gawrodger et al. (1986) reported that 5/10 women responded to both the 0.4 and 2.5 mg doses of nickel, but 10/26 also reacted to a placebo. They determined the LOAEL of their experiment to be 5.6 mg of nickel, a dose at which 100% of the women responded. Burrows et al. (1981) administered 0.5 mg nickel twice a day on two consecutive days to 22 patients, each of whom served as her own control. There was no significant difference between the number of individuals responding to a placebo as compared to nickel. However, the placebo response was high (12/22). The authors concluded that there is probably no connection between nickel in an ordinary diet and exacerbation of dermatitis but that a higher level may aggravate dermatitis in some individuals.

Nielsen (1989) describes a study in which 12 nickel-sensitive women were challenged for a 4-day period with a diet providing 490 ug Ni/day. No changes were observed before the start of the nickel challenge to day 0 (start of challenge). On day 4, the eczema of 6 patients was considered to be worse according to both the patients' impressions and a dermatologist's evaluation. The delayed reaction in this study may be attributed to the fact that the dose of nickel was ingested in the diet throughout the day as opposed to studies which employed a bolus dose. This difference may greatly affect the pharmacokinetics of ingested nickel.

While the previous studies on humans with a hypersensitivity to nickel were considered in developing the RfD, none of them were adequate to serve as the basis for the quantitative risk assessment. The RfD is believed to be set at a level which would not cause individuals to become sensitized to nickel; however, those who have already developed a hypersensitivity (e.g., from a dermal exposure) may not be fully protected.

One final point to bear in mind in establishing an RfD for nickel is that nickel has been shown to be an essential trace element for several animal species. Rats deprived of nickel exhibit retarded growth and low hemoglobin levels (Schnegg and Kirchgessner, 1977). A requirement for nickel has not been conclusively demonstrated in humans, but nickel is considered to be a normal constituent of the diet. Typical daily intake of nickel ranges from 100-300 ug/day.

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o ORAL RFD CONFIDENCE :

Study -- Low
Data Base -- Medium
RfD -- Medium

The chronic study (Ambrose et al., 1976) was properly designed and provided adequate toxicological endpoints; however, high mortality occurred in the controls (44/50). Therefore, a low confidence is recommended for the study. The data base provided adequate supporting subchronic studies, one by gavage and the other in drinking water (Po animals of the RTI subchronic study, 1986). A medium confidence level in the data base is recommended since there are inadequacies in the remaining reproduction data.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- U.S. EPA, 1986, 1991

The information contained in the Quantification of Toxicologic Effects for Nickel was reviewed by the Science Advisory Board in August 1990.

Other EPA Documentation -- None

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o REVIEW DATES : 04/16/87, 05/20/87, 07/16/87, 05/17/90, 08/14/91
o VERIFICATION DATE : 07/16/87
o EPA CONTACTS :

Sue Velazquez / OHEA -- (513)569-7571

Jennifer Orme / OST -- (202)260-7586

RDI -
o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

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o REVIEW DATES : 11/16/89

WQCHU-

Water and Fish Consumption: 1.34E+1 ug/L

Fish Consumption Only: 1.0E+2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of 1.34E+1 ug/L is based on consumption of contaminated aquatic organisms and water. A WQC of 1.0E+2 ug/L has also been established based on consumption of contaminated aquatic organisms alone.

Reference -- 51 FR 43665 (12/03/86)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 1.4E+3 ug/L
Chronic -- 1.6E+2 ug/L

Marine:

Acute -- 7.5E+1 ug/L
Chronic -- 8.3E+0 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. The freshwater criteria are hardness dependent. Values given here are calculated at a hardness of 100 mg/L CaCO3. A complete discussion can be found in the referenced notice.

Reference -- 51 FR 43665 (12/03/86)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value -- 0.1 mg/L (nickel) (Proposed, 1990)

Considers technological or economic feasibility? -- NO

Discussion -- EPA is proposing to regulate nickel based on its potential adverse effects (reduced body and liver weights) reported in a two-year dietary study in rats. The MCLG is based upon a DWEL of 0.58 mg/L and an assumed drinking water contribution of 20 percent.

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Health and Ecological Criteria Division / OST / (202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 0.1 mg/L (nickel) (Proposed, 1990)

Considers technological or economic feasibility? -- YES

Discussion -- EPA is proposing an MCL equal to the proposed MCLG of 0.1 mg/L.

Monitoring requirements -- Ground water systems every 3 years; surface water systems annually; will allow monitoring at up to 10-year intervals after the system completes 3 rounds of sampling at <50% of the MCL.

Analytical methodology -- Atomic absorption (EPA 249.2; SM 304); inductively-coupled plasma (EPA 200.7; SM 305); ICP mass spectrometry (EPA 200.8): PQL= 0.050 mg/L.

Best available technology -- Ion exchange; reverse osmosis; lime softening.

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

Status -- Listed (nickel) (Final, 1991)

Discussion -- "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement -- All systems to be monitored unless a vulnerability assessment determines the system is not vulnerable.

Analytical methodology -- Atomic absorption (EPA 249.2; SM 304); inductively coupled plasma (EPA 200.7; SM 305).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

CERC -

Value -- 100 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The proposed RQ for soluble nickel salts is 100 pounds, based on potential carcinogenicity. The available data indicate a hazard ranking of low and a weight-of-evidence classification of C, which corresponds to an RQ of 100 pounds.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800) 424-9346 / (202) 260-3000 / FTS 260-3000

RCRA -

Status -- Listed (total nickel)

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Ambrose, A.M., P.S. Larson, J.R. Borzelleca and G.R. Hennigar, Jr. 1976. Long-term toxicologic assessment of nickel in rats and dogs. J. Food Sci. Technol. 13: 181-187.

OREF - ABC (American Biogenics Corp.). 1986. Ninety-day gavage study in albino rats using nickel. Draft Final Report submitted to Research Triangle Institute, P.O. Box 12194, Research Triangle Park, NC 27709.

OREF - Burrows, D., S. Creswell and J.D. Merrett. 1981. Nickel, hands and hip prostheses. Br. J. Dermatol. 105: 437-444.

OREF - Cronin, E., A. Di Michiel and S.S. Brown. 1980. Oral nickel challenge in nickel-sensitive women with hand eczema. In: Nickel Toxicology, S.S. Brown and F.W. Sunderman Jr., Ed. Academic Press, New York. p. 149-152.

OREF - Edwards, M.J. 1986. Hyperthermia as a teratogen: A review of experimental studies and their clinical significance. Terat. Carcin. Mutagen. 6: 563-582.

OREF - Gawkrodger, D.J., S.W. Cook, G.S. Fell and J.A.A. Hunter. 1986. Nickel dermatitis: The reaction to oral nickel challenge. Br. J. Dermatol. 115: 33-38.

OREF - Jordan, W.P. and S.E. King. 1979. Nickel feeding in nickel-sensitive patients with hand eczema. J. Am. Acad. Dermatol. 1(6): 506-508.

OREF - Kaaber, K., N.K. Veien and J.C. Tjell. 1978. Low nickel diet in the treatment of patients with chronic nickel dermatitis. Br. J. Derm. 98: 197-201.

OREF - Kaaber, K., T. Menne, J.C. Tjell and N. Veien. 1979. Antabuse treatment of nickel dermatitis. Chelation - a new principle in the treatment of nickel dermatitis. Contact Derm. 5: 221-228.

OREF - Nielsen, G.D. 1989. Oral challenge of nickel-allergic patients with hand eczema. In: Nickel and Human Health: Current Perspectives. Advances in Environmental Science and Technology, E. Nieboer and A. Aitio, Ed. John Wiley and Sons, Inc., New York, NY. Chapter 16: 1.

OREF - North American Contact Dermatitis Group. 1973. Epidemiology of contact dermatitis in North America: 1972. Arch. Dermatol. 108: 537-540.

OREF - Prystowsky, S.D., A.M. Allen, R.W. Smith, J.H. Nonomura, R.B. Odom and W.A. Akers. 1979. Allergic contact hypersensitivity to nickel, neomycin, ethylenediamine and benzocaine. Relationships between age, sex, history, exposure and reactivity to standard patch tests and use tests in a general population. Arch. Dermatol. 115(8): 959-962.

OREF - RTI (Research Triangle Institute). 1987. Two generation

prt dl cn^H^Hc^Hncar, car continuous

1 - IRIS
IRSN - 429
DATE - 920122
STAT - Oral RfD Assessment (RDO) pending
STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) on-line 10/01/90
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 08/01/89 RDO Oral RfD now under review
IRH - 05/01/90 RDI Inhalation RfC now under review
IRH - 10/01/90 CAR Carcinogen assessment on-line
IRH - 10/01/90 REFS Bibliography on-line
IRH - 01/01/92 EXSR Regulatory Action section on-line
RLEN - 14960
NAME - 1,1-Dichloroethane
RN - 75-34-3
SY - AETHYLIDENCHLORID [GERMAN]
SY - CHLORINATED HYDROCHLORIC ETHER
SY - CHLORURE D'ETHYLIDENE [FRENCH]
SY - CLORURO DI ETILIDENE [ITALIAN]
SY - 1,1-DICHLOORETHAAN [DUTCH]
SY - 1,1-DICHLORAETHAN [GERMAN]
SY - 1,1-DICHLORETHANE
SY - DICHLORO-1,1 ETHANE [FRENCH]
SY - 1,1-DICHLOROETHANE
SY - 1,1-DICLOROETANO [ITALIAN]
SY - 1,1-DICLOROETANO [SPANISH]
SY - ETHANE, 1,1-DICHLORO-
SY - ETHYLIDENE CHLORIDE
SY - ETHYLIDENE DICHLORIDE
SY - HSDB 64
SY - NCI-C04535
SY - RCRA WASTE NUMBER U076
SY - UN 2362

RDO -
o ORAL RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

RDI -

o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

CAREV-

o CLASSIFICATION : C; possible human carcinogen

o BASIS FOR CLASSIFICATION : Based on no human data and limited evidence of carcinogenicity in two animal species (rats and mice) as shown by an increased incidence of mammary gland adenocarcinomas and hemangiosarcomas in female rats and an increased incidence of hepatocellular carcinomas and benign uterine polyps in mice.

o HUMAN CARCINOGENICITY DATA :

None.

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o ANIMAL CARCINOGENICITY DATA :

Limited. An NCI bioassay (1978a) provides limited evidence of the carcinogenicity of 1,1-dichloroethane in Osborne-Mendel rats and B6C3F1 mice. This is based on significant dose-related increases in the incidence of hemangiosarcomas at various sites and mammary carcinomas in female rats and statistically significant increases in the incidence of liver carcinoma in male mice and benign uterine polyps in female mice. The study is limited by high mortality in many groups; the low survival rates precluded the appearance of possible late-developing tumors and decreased the statistical power of this bioassay.

Technical grade 1,1-dichloroethane in corn oil was administered by gavage 5 days/week for 78 weeks to groups of 50 Osborne-Mendel rats/sex/dose. All surviving animals were necropsied following a 33-week observation period. Due to toxicity, dosing was not continuous (3 weeks on then 1 week off), making the TWAs for 5 days/week 382 and 764 mg/kg/day for low- and high-dose males and 475 and 950 mg/kg/day for low- and high-dose females, respectively. Both a vehicle and an untreated (not intubated) control group (20 rats/sex/group) were included in the study. A high incidence of pneumonia (approximately 80%) in all 4 groups of each sex was considered to be the cause for the low survival at termination of the study. Survival at 111 weeks was 30, 5, 4,and 8% in the untreated control, the vehicle control, the low-dose, and the high-dose male rat groups, respectively. Survival at termination for the female rat groups was 40, 20, 16, and 18% for the untreated control, vehicle control, low- and high-dose groups, respectively. In female rats there was a statistically significant positive dose-related trend in incidence of hemangiosarcomas (0/19 for matched vehicle controls, 0/50 for the low-dose group, and 4/50 for the high-dose group). The incidence of mammary gland adenocarcinomas (1/20 for the untreated group, 0/19 for the vehicle control group, 1/50 for low-dose, and 5/50 for high-dose groups) showed a

statistically significant dose-related positive trend in those female rats surviving at least 52 weeks; tumor incidence was 0/16, 1/28 and 5/31 for vehicle control, low- and high-dose groups, respectively. (Tumor incidence at termination for the untreated control females surviving at least 52 weeks was not reported.) This bioassay was conducted before the life table tests were implemented, so results adjusted for mortality are not available. No mammary gland adenomas or hemangiosarcomas were observed in the dosed-male rats.

In the same NCI (1978a) study, groups of 50 B6C3F1 mice/sex/group were administered technical grade 1,1-dichloroethane in corn oil by gavage 5 days/week for 70 weeks. As in the rat study, the dosage pattern was 3 weeks on and 1 week off; the surviving animals were necropsied 13 weeks after the termination of dosing. The TWAs for 5 days/week for the low- and high-dose groups were 1442 and 2885 mg/kg/day for male and 1665 and 3331 mg/kg/day for female mice. Control groups, identical to those in the rat study and consisting of 20 mice/sex/group were also used. Survival at termination was 80, 80, 80, and 50% for the untreated control group, the vehicle control group, the low-, and high-dose females, respectively. In male mice survival was 35, 55, 62, and 32% in the untreated control group, the vehicle control group, the low-, and high-dose groups, respectively. An increased incidence of hepatocellular carcinoma in male mice was not statistically significant by either pair-wise or trend test (2/17 in the untreated control group, 1/19 in the vehicle control group, 8/49 in the low-dose and 8/47 in the high-dose groups). The incidence of hepatocellular carcinoma in male mice surviving at least 52 weeks was 1/19, 6/72, 8/48, and 8/32 in the matched vehicle control group, a pooled vehicle control group consisting of mice from this and identical controls from other concurrent experiments, and the low-, and high-dose groups, respectively; this positive trend was statistically significant. In female mice, liver carcinomas were reported in only the vehicle control (1/19) and the low-dose groups (1/47): no liver tumors were seen in the untreated controls or in the high-dose group. A statistically significant increase in benign uterine endometrial stromal polyps (4/46) was observed in high-dose females; these were not observed in any other group. A preliminary report of the NCI (1978a) study was published by Weisburger (1977).

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o SUPPORTING DATA :

To determine if 1,1-dichloroethane in drinking water could act as a tumor promoter or a complete carcinogen, Klaunig et al. (1986) exposed groups of 35 male B6C3F1 mice to 1,1-dichloroethane in drinking water at 0, 835, or 2500 mg/L for up to 52 weeks following a 4-week treatment with either drinking water containing 10 mg/L diethyl nitrosamine (DENA-initiated groups) or with deionized water (noninitiated groups). The investigators estimated that the approximate weekly dose of 1,1-dichloroethane was 3.8 mg/g/week (corresponding to 543 mg/kg/day) for the groups exposed to 2500 mg/L. Upon sacrifice at the end of either 24 weeks (10 mice/group) or 52 weeks (25 mice/group) of promotion, all tissues were examined for gross pathologic lesions and histologic sections of the liver, kidneys and lungs were examined. Neither the initiated nor the noninitiated 1,1-dichloroethane-treated groups showed a significant increase in the incidence of liver or lung tumors compared with

initiated or noninitiated controls, respectively. The authors concluded that 1,1-dichloroethane was not carcinogenic to mice and did not act as a tumor promotor following initiation with DENA. These conclusions may not be entirely justified, since the duration of the study may have been inadequate for the development of tumors in noninitiated 1,1-dichloroethane-treated animals. In addition, the incidence of liver tumors in DENA-initiated controls was 70% at 24 weeks and 100% at 52 weeks, and the number of tumors/mouse in DENA-initiated controls at these times was 3.00 and 29.30, respectively. Hence, an increase in tumors or decrease in latency in 1,1-dichloroethane-treated DENA-initiated animals would have to be marked in order to be detectable.

Milman et al. (1988) and Story et al. (1986) investigated the chlorinated ethanes and ethylenes to detect their potential tumor initiating or promoting effects in a liver foci assay in Osborne-Mendel rats. In this assay, 1,1-dichloroethane did not give positive results for initiation (with phenobarbital as promotor), or as a complete carcinogen when administered in the absence of initiation or promotion. Positive results for were seen for promotion with DENA as initiator. The assumption that the liver foci seen in this type of assay are precancerous has not been validated.

When tested by plate incorporation in a desiccator (because of volatility) in the presence and absence of metabolic activation systems, 1,1-dichloroethane was reported to be mutagenic for Salmonella typhimurium TA1535, TA98, and TA100, but not to TA1537 (Riccio et al., 1983; Mitoma et al., 1984). Negative results were reported for 1,1-dichloroethane in a cell transformation assay with BALB/c-3T3 cells, tested in the absence of an exogenous metabolic activation system in a sealed glass incubation chamber (Tu et al., 1985; Arthur D. Little, Inc., 1983). When tested in a similar manner in a DNA repair assay with hepatocyte primary cultures from rats or mice, 1,1-dichloroethane produced positive results (Williams, 1977). The results obtained in these three assays were also summarized in a joint publication (Milman et al., 1988).

Positive results were obtained in a viral transformation assay in which 1,1-dichloroethane was incubated with cultured Syrian Hamster embryo cells in a sealed glass chamber prior to addition of adenovirus SA7 (Hatch et al., 1983).

Lattanzi et al. (1988) determined that 1,1-dichloroethane, like 1,2-dichloroethane, binds covalently to DNA, forming DNA adducts. The Covalent Binding Index (CBI) of both 1,1-dichloroethane and 1,2-dichloroethane classifies them as weak initiators.

Chronic bioassays performed by NCI (1978b) on the isomer 1,2-dichloroethane resulted in many of the same tumor types as seen in the bioassays of 1,1-dichloroethane. Significant increases in the incidences of forestomach squamous cell carcinomas and hemangiosarcomas were observed in male rats and an increased incidence of mammary adenocarcinomas was observed in both female rats and mice. In addition, alveolar and bronchiolar adenomas

were reported in male and female mice; endometrial stromal polyps and sarcomas in female mice; and hepatocellular carcinomas in male mice.

Based on these findings, as well as the appearance of lung papillomas in mice after topical treatment, 1,2-dichloroethane was classified as a group B2 chemical, a probable human carcinogen (U.S. EPA, 1990). Because of similarities in structure and target organs, the carcinogenic evidence for 1,2-dichloroethane is considered to be supportive of the classification of 1,1-dichloroethane in group C, a possible human carcinogen.

CARDR-
o CARCINOGENICITY SOURCE :

U.S. EPA. 1985. Health and Environmental Effects Profile for Dichloroethanes. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

U.S. EPA. 1986. Guidelines for Carcinogen Risk Assessment. Federal Register. 51(185): 33992-34003.

U.S. EPA. 1990. Integrated Risk Information System (IRIS). Online. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.

The 1984 Health Effects Assessment for 1,1-Dichloroethane has received Office of Health and Environmental Assessment review.
DOCUMENT

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o REVIEW DATES : 12/07/89
o VERIFICATION DATE : 12/07/89
o EPA CONTACTS :

Sue Velazquez / ORD -- (513)569-7571 / FTS 684-7571

Krishan Khanna / ODW -- (202)260-7588 / FTS 260-7588

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - None
IREF - None
CREF - Arthur D. Little, Inc. 1983. Cell transformation assays of 11 chlorinated hydrocarbon analogs. Microfiche No. OTS0509392, Document No. 40-8324457.
CREF - Hatch, G.G., P.D. Mamay, M.L. Ayer, B.C. Casto and S. Nesnow. 1983. Chemical enhancement of viral transformation in Syrian hamster embryo cells by gaseous and volatile chlorinated methanes and ethanes. Cancer Res. 43(5): 1945-1950.
CREF - Klaunig, J.E., R.J. Ruch and M.A. Pereira. 1986. Carcinogenicity of chlorinated methane and ethane compounds administered in drinking water to mice. Environ. Health Perspect. 69: 89-95.
CREF - Lattanzi, G., A. Colacci, S. Grilli, et al. 1988. Binding of hexachloroethane to biological macromolecules from rat and mouse organs. J. Toxicol. Environ. Health. 24: 403-411.
CREF - Milman, H.A., D.L. Story, E.S. Riccio, et al. 1988. Rat liver foci and in vitro assays to detect initiating and promoting effects of chlorinated ethanes and ethylenes. Ann. NY Acad. Sci. 534: 521-530.
CREF - Mitoma, C., C.A. Tyson and E.S. Riccio. 1984. Investigations of the species sensitivities and mechanism of carcinogenicity of halogenated hydrocarbons. Microfiche No. OTS0509408, Document No. 40-8424225.
CREF - NCI (National Cancer Institute). 1978a. Bioassay of 1,1-dichloroethane for possible carcinogenicity. CAS No. 78-34-3. NCI/NTP Technical Report No. 066. DHEW Publ. No. (NIH) 78-1316, Washington, DC.
CREF - NCI (National Cancer Institute). 1978b. Bioassay of 1,2-Dichloroethane for Possible Carcinogenicity. NCI Carcinogenesis Technical Report Series No. 55. DHEW Publ. No. (NIH) 78-1361, Washington, DC.
CREF - Riccio, E., A. Griffin, K. Mortelmans and H.A. Milman. 1983. A comparative mutagenicity study of volatile halogenated hydrocarbons using different metabolic activation systems. Environ. Mutagen. 5: 472. (Abstract)
CREF - Story, D.L., E.F. Meierhenry, C.A. Tyson and H.A. Milman. 1986. Differences in rat liver enzyme-altered foci produced by

chlorinated aliphatics and phenobarbital. Toxicol. Ind. Health. 2(4): 351-362.
CREF - Tu, A.S., T.A. Murray, K.M. Hatch, A. Sivak and H.A. Milman. 1985. In vitro transformation of BALB/c-3T3 cells by chlorinated ethanes and ethylenes. Cancer Lett. 28(1): 85-92.
CREF - U.S. EPA. 1985. Health and Environmental Effects Profile for Dichloroethanes. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.
CREF - U.S. EPA. 1986. Guidelines for Carcinogen Risk Assessment. Federal Register. 51(185): 33992-34003.
CREF - U.S. EPA. 1990. Integrated Risk Information System (IRIS). Online. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH.
CREF - Weisburger, E.K. 1977. Carcinogenicity studies on halogenated hydrocarbons. Environ. Health Perspect. 21: 7-16.
CREF - Williams, E.M. 1977. Detection of chemical carcinogens by unscheduled DNA synthesis in rat liver primary cell cultures. Cancer Res. 37(6): 1845-1851.
HAREF- None

[IRIS] SS 16 /cf?
USER:
127-18-4
Search in progress

SS (16) PSTG (1)

[IRIS] SS 17 /cf?
USER:

108-38-3
Search in progress

SS (4) PSTG (1)

[IRIS] SS 5 /cf?
USER:
prt dl ncar ^H, car continuous

1 - IRIS
IRSN - 264
DATE - 920122
STAT - Oral RfD Assessment (RDO) on-line 09/30/87
STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) on-line 03/01/91
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 09/26/88 CAR Carcinogen summary on-line
IRH - 07/01/89 RDI Inhalation RfD now under review
IRH - 07/01/89 REFS Bibliography on-line
IRH - 03/01/91 CARDR Primary contact changed
IRH - 03/01/91 RCRA EPA contact changed
IRH - 01/01/92 RDO Secondary contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
RLEN - 17557
NAME - Xylenes
RN - 1330-20-7
SY - 108-38-3
SY - 1330-20-7
SY - 2106-42-3
SY - 95-47-6
SY - dimethylbenzene
SY - 1,2-dimethylbenzene
SY - 1,3-dimethylbenzene
SY - 1,4-dimethylbenzene
SY - mixed xylenes
SY - m-xylene
SY - meta-xylene
SY - o-xylene
SY - ortho-xylene
SY - p-xylene
SY - para-xylene
SY - Xylenes

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD

Hyperactivity, decreased body weight and increased mortality (males) Chronic Rat Gavage Study	NOAEL: 250 mg/kg/day (converted to 179 mg/kg/day) FEL: 500 mg/kg/day (converted to 357 mg/kg/day)	100	1	2E+0 mg/kg/day

NTP, 1986

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*Conversion Factors: Dose adjusted for gavage schedule (5\days/week).

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o ORAL RFD STUDIES :

NTP (National Toxicology Program). 1986. NTP Technical Report on the Toxicology and Carcinogenesis of Xylenes (mixed) (60.2% m-xylene, 13,6% p-xylene, 17.0 ethylbenzene and 9.1% 0-xylene) (CAS No. 1330-20-7) in F344/N rats and B6C3F1 mice (gavage studies). U.S. DHHS, PHS, NIH, NTP, Research Triangle Park, NC. NTP TR 327, NIH Publ. No. 86-2583.

Groups of 50 male and 50 female Fischer 344 rats and 50 male and 50 female B6C3F1 mice were given gavage doses of 0, 250, or 500 mg/kg/day (rats) and 0, 500, or 1000 mg/kg/day (mice) for 5 days/week for 103 weeks. The animals were observed for clinical signs of toxicity, body weight gain, and mortality. All animals that died or were killed at sacrifice were given gross necropsy and comprehensive histologic examinations. There was a dose-related increased mortality in male rats, and the increase was significantly greater in the high-dose group compared with controls. Although increased mortality was observed at 250 mg/kg/day, the increase was not significant. Although many of the early deaths were caused by gavage error, NTP (1986) did not rule out the possibility that the rats were resisting gavage dosing because of the behavioral effects of xylene. Mice given the high dose exhibited hyper-activity, a manifestation of CNS toxicity. There were no compound-related histopathologic lesions in any of the treated rats or mice. Therefore, the high dose is a FEL and the low dose a NOAEL.

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o ORAL RFD UNCERTAINTY :

UF = 100. An uncertainty factor of 100 was chosen: 10 for species-to-species extrapolation and 10 to protect sensitive individuals.

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o ORAL RFD MODIFYING FACTOR :

MF = 1.

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o ORAL RFD COMMENTS :

U.S. EPA (1984) reported an RfD of 0.01 mg/kg/day, based on a rat dietary NOAEL of 200 ppm or 10 mg/kg/day as defined by Bowers et al. (1982) in a 6-month study. This NOAEL was divided by an uncertainty factor of 1000. U.S.

EPA (1985, 1986) noted that this study used aged rats, loss of xylene from volatilization was not controlled, only one exposure level was used, and histopathologic examination was incomplete. An RfD of 4.31 mg/day (about 0.06 mg/kg/day) based on an inhalation study (Jenkins et al., 1970) using rats, guinea pigs, monkeys, and dogs exposed to o-xylene at 3358 mg/cu.m, 8 hours/day, 5 days/ week for 6 weeks or at 337 mg/cu.m continuously for 90 days was derived by U.S. EPA (1985). Deaths in rats and monkeys, and tremors in dogs occurred at the highest dose, whereas no effects were observed in the 337 mg/cu.m continuous exposure group. The RfD based on the NTP (1986) study is preferable because it is based on a chronic exposure in two species by a relevant route of administration, and comprehensive histology was performed. Xylene is fetotoxic and teratogenic in mice at high oral doses (Nawrot and Staples, 1981; Marks et al., 1982), but the RfD as calculated should be protective of these effects.

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o ORAL RFD CONFIDENCE :

Study: Medium
Data Base: Medium
RfD: Medium

The NTP (1986) study was given a medium confidence level because it was a well-designed study in which adequately sized groups of two species were tested over a substantial portion of their lifespan, comprehensive histology was performed, and a NOAEL was defined; but clinical chemistries, blood enzymes, and urinalysis were not performed. The data base was given a medium confidence level because, although supporting data exist for mice and teratogenicity and fetotoxicity data are available with positive results at high oral doses, a LOAEL for chronic oral exposure has not been defined. Medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

U.S. EPA. 1986. Health and Environmental Effects Profile for Xylenes (o-, m-, p-). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH and the Environmental Criteria and Assessment Office, Research Triangle Park, NC for the Office of Solid Waste and Emergency Response and the Office of Air Quality Planning and Standards, Office of Air and Radiation, Washington, DC.

Limited peer review and extensive agency-wide review, 1986.

U.S. EPA. 1985. Drinking Water Criteria Document For Xylenes. Prepared by Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

Extensive peer review agency-wide review.

U.S. EPA. 1984. Health Effects Assessment for Xylene. Prepared by the

Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC.

ECAO internal review and limited agency review.

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o REVIEW DATES : 12/05/85, 03/19/87
o VERIFICATION DATE : 03/19/87
o EPA CONTACTS :

Harlal Choudhury / ORD -- (513)569-7536 / FTS 684-7536

Kenneth Poirier / ORD -- (513)569-7553 / FTS 684-7553

RDI -
o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

CAREV-
o CLASSIFICATION : D; not classifiable as to human carcinogenicity.
o BASIS FOR CLASSIFICATION : Orally administered technical xylene mixtures did not result in significant increases in incidences in tumor responses in rats or mice of both sexes.
o HUMAN CARCINOGENICITY DATA :

None.

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o ANIMAL CARCINOGENICITY DATA :

Inadequate. In an NTP (1986) study, 50 male and 50 female F344/N rats were treated by gavage with mixed xylenes in corn oil (60% m-xylene, 14% p-xylene, 9% o-xylene and 17% ethylbenzene) at dosages of 0, 250 or 500 mg/kg/day, 5 days/week for 103 weeks. Similarly, 50 male and 50 female B6C3F1 mice were treated with the same xylene mixture at dosages of 0, 500 or 1000 mg/kg/day. Animals were killed and examined histologically when moribund or after 104-105 weeks. An apparent dose-related increased mortality was observed in male rats, but this difference was statistically significant for the high dose group, only. No other differences in survival between dosage groups of either sex were observed. Interstitial cell tumors of the testes could not be attributed to administration of the test compound observed in male rats (43/50 control, 38/50 low-dose and 41/49 high-dose). NTP (1986) reported that there were no significant changes in the incidence of neoplastic

or nonneoplastic lesions in either the rats or mice that could be considered related to the mixed xylene treatment, and concluded that under the conditions of these 2-year gavage studies, there was "no evidence of carcinogenicity" of xylene (mixed) for rats or mice of either sex at any dosage tested.

Maltoni et al. (1985), in a limited study, reported higher incidences (compared with controls) of malignant tumors in male and female Sprague-Dawley rats treated by gavage with xylene in olive oil at 500 mg/kg/day, 4 or 5 days/week for 104 weeks. This study did not report survival rates or specific tumor types; therefore, the results cannot be interpreted.

Berenblum (1941) reported that "undiluted" xylene applied at weekly intervals produced one tumor-bearing animal out of 40 after 25 weeks in skin-painting experiments in mice. No control groups were described. Pound (1970) reported negative results in initiation-promotion experiments with xylene as the initiator and croton oil as the promotor.

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o SUPPORTING DATA :

The frequency of sister chromatid exchanges and chromosomal aberrations were nearly identical between a group of 17 paint industry workers exposed to xylene and their respective referents (Haglund et al., 1980). In vitro, xylene caused no increase in the number of sister chromatid exchanges in human lymphocytes (Gerner-Smidt and Friedrich, 1978). Studies indicate that xylene isomers, technical grade xylene or mixed xylene are not mutagenic in tests with Salmonella typhimurium (Florin et al., 1980; NTP, 1986; Bos et al., 1981) nor in mutant reversion assays with Escherichia coli (McCarroll et al., 1981). Technical grade xylene, but not o- and m-xylene, was weakly mutagenic in Drosophila recessive lethal tests. Chromosomal aberrations were not increased in bone marrow cells of rats exposed to xylenes by inhalation (Donner et al., 1980).

CARDR-
o CARCINOGENICITY SOURCE :

U.S. EPA. 1987. Drinking Water Criteria Document for Xylene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. ECAO-CIN-416. Final.

The Drinking Water Criteria Document for Xylene has received Agency and external review.

DOCUMENT

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o REVIEW DATES : 12/02/87
o VERIFICATION DATE : 12/02/87
o EPA CONTACTS :

Bruce Mintz / ODW -- (202)260-9569 / FTS 260-9569

W. Bruce Peirano / ORD -- (513)569-7540 / FTS 684-7540

MCLG -

Value (status) -- 10.0 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- The EPA has promulgated a MCLG of 10.0 mg/L based upon potential adverse effects reported in a chronic oral study in rats. Cancer information on xylenes was reviewed and found to be inadequate for determining potential human carcinogenicity.

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Health and Ecological Criteria Division / OST / (202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 10.0 mg/L (Final, 1991)

Considers technological or economic feasibility? -- YES

Discussion -- The EPA has promulgated a MCL equal to the MCLG of 10.0 mg/L.

Monitoring requirements -- All systems initially monitored for four consecutive quarters; repeat monitoring dependent upon detection, vulnerability status and system size.

Analytical methodology -- Purge and trap capillary gas chromatography (EPA 502.2); gas chromatographic/mass spectrometry (EPA 524.2); purge and trap gas chromatography (EPA 503.1); gas chromatography/mass spectrometry (EPA 524.1); PQL= 0.005 mg/L.

Best available technology -- Granular activated carbon; packed tower aeration.

Reference -- 56 FR 3526 (01/30/91); 56 FR 30266 (07/01/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

Value -- 0.02 mg/L (Proposed, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- SMCLs are non-enforceable and establish limits for contaminants which may affect the aesthetic qualities (e.g. taste and odor) of drinking water. It is recommended that systems monitor for these contaminants every three years. More frequent monitoring for contaminants such as pH, color, odor or others may be appropriate under certain circumstances. The SMCL for xylenes is based on odor qualities. Promulgation has been deferred following public comment (56 FR 3526).

Reference -- 54 FR 22062 (05/22/89); 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

FISTD-

Status -- List "C" Pesticide (1989)

Reference -- 54 FR 30846 (07/24/89)

EPA Contact -- Registration Branch / OPP
(703)557-7760 / FTS 557-7760

FIREV-

No data available

CERC -

Value (status) -- 1000 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ is based on ignitability and aquatic toxicity as established for xylene under Section 311(b)(4) of the Clean Water Act (40 CFR 117.3). The available data indicate the aquatic 96-hour Median Threshold Limit for xylene is between 10 and 100 ppm, corresponding to an RQ of 1000 pounds. The ignitibility RQ of 1000 pounds is based on a flash point of 81 to 90F.

Reference -- 50 FR 13456 (04/04/85); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Bowers, D.E. Jr., M.S. Cannon and D.H. Jones. 1982. Ultrastructural changes in liver of young and aging rats exposed to methylated benzenes. Am. J. Vet. Res. 43(4): 679-683.

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OREF - U.S. EPA. 1985. Drinking Water Criteria Document for Xylenes. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

OREF - U.S. EPA. 1986. Health and Environmental Effects Profile for Xylene (o-,m-,p-). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH and the Environmental Criteria and Assessment Office, Research Triangle Park, NC for the Office of Solid Waste and Emergency Respo

IREF - None

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HAREF- None

[IRIS] SS 5 /cf?
USER:

[10] TRIFACTS - [TRIFACTS]
[11] TOXIC CHEMICAL RELEASE INVENTORY FILES - [TRI]

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The health risk assessment information contained in IRIS has been reviewed and agreed upon by work groups composed of EPA (United States Environmental Protection Agency) scientists. Background Documents explaining the methods used to derive the values in IRIS are available from EPA. For copies of these documents and more information on IRIS file content contact IRIS User Support at 513/569-7254.

[IRIS] SS 1 /cf?
USER:
100-41-4
  Search in progress

SS (1) PSTG (1)

[IRIS] SS 2 /cf?
USER:
prt dl ncar, car continuous

1 - IRIS
IRSN - 49
DATE - 920501
UPDT - 05/01/92, 52 fields
STAT - Oral RfD Assessment (RDO) on-line 06/01/91
STAT - Inhalation RfC Assessment (RDI) on-line 03/01/91
STAT - Carcinogenicity Assessment (CAR) on-line 08/01/91
STAT - Drinking Water Health Advisories (DWHA) on-line 03/01/88
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 03/01/88 RDO Dose conversion clarified
IRH - 03/01/88 RDO Documentation revised
IRH - 03/01/88 HADV Health Advisory added
IRH - 09/07/88 CAR Carcinogen summary on-line
IRH - 08/01/89 REFS Bibliography on-line
IRH - 08/01/90 RCRA EPA contact changed
IRH - 10/01/90 RDI Inhalation RfC now under review
IRH - 03/01/91 RDI Inhalation RfC summary on-line
IRH - 03/01/91 IREF Inhalation RfC references added
IRH - 06/01/91 RDO Primary contact changed
IRH - 08/01/91 CARDR Secondary contact changed
IRH - 01/01/92 RDO Secondary contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
RLEN - 47996

NAME - Ethylbenzene
RN - 100-41-4
SY - AETHYLBENZOL
SY - BENZENE, ETHYL
SY - EB
SY - ETHYLBENZEEN
SY - Ethylbenzene
SY - ETHYLBENZOL
SY - ETILBENZENE
SY - ETYLOBENZEN
SY - NCI-C56393
SY - PHENYLETHANE
SY - UN 1175

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Liver and kidney toxicity	NOEL: 136 mg/kg/day (converted to 97.1 mg/kg/day)	1000	1	1E-1 mg/kg/day
Rat Subchronic to Chronic Oral Bio-assay	LOAEL: 408 mg/kg/day (converted to 291 mg/kg/day)			
Wolf et al., 1956				

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*Conversion Factors: 5 days/7 days; thus, 136 mg/kg/day x 5 days/7 days = 97.1 mg/kg/day

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o ORAL RFD STUDIES :

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

The chosen study is a rat 182-day oral bioassay in which ethylbenzene was given 5 days/week at doses of 13.6, 136, 408, or 680 mg/kg/day in olive oil gavage. There were 10 albino female rats/dose group and 20 controls.

The criteria considered in judging the toxic effects on the test animals were growth, mortality, appearance and behavior, hematologic findings, terminal concentration of urea nitrogen in the blood, final average organ and body weights, histopathologic findings, and bone marrow counts. The LOAEL of 408 mg/kg/day is associated with histopathologic changes in liver and kidney.

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o ORAL RFD UNCERTAINTY :

UF = 1000. The uncertainty factor of 1000 reflects 10 for both intraspecies

and interspecies variability to the toxicity of this chemical in lieu of specific data, and 10 for extrapolation of a subchronic effect level to its chronic equivalent.

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o ORAL RFD MODIFYING FACTOR :

MF = 1.

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o ORAL RFD COMMENTS :

None.

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o ORAL RFD CONFIDENCE :

Study: Low
Data Base: Low
RfD: Low

Confidence in the chosen study is low because rats of only one sex were tested and the experiment was not of chronic duration. Confidence in the supporting data base is low because other oral toxicity data were not found. Low confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

U.S. EPA. 1980. Ambient Water Quality Criteria for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-048. NTIS PB 81-117590.

U.S. EPA. 1985. Drinking Water Criteria Document for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (Public review draft)

U.S. EPA. 1985. Health Effects Assessment for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC. ECAO-CIN-H008.

The 1980 Ambient Water Quality Criteria Document for Ethylbenzene received extensive Agency and public review.

The 1985 Drinking Water Criteria Document for Ethylbenzene and the 1985 Health Effects Assessment for Ethylbenzene received extensive Agency review with the help of selected outside scientists.

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o REVIEW DATES : 05/20/85
o VERIFICATION DATE : 05/20/85
o EPA CONTACTS :

Jeffrey C. Swartout / ORD -- (513)569-7811 / FTS 684-7811

Carolyn Smallwood / ORD -- (513)569-7425 / FTS 684-7425

RDI -

o INHALATION RFD SUMMARY :

Critical Effect	Exposures*	UF	MF	RfC
Developmental toxicity Rat and Rabbit Developmental Inhalation Studies Andrew et al., 1981; Hardin et al., 1981	NOAEL: 434 mg/cu.m (100 ppm) NOAEL(ADJ): 434 mg/cu.m NOAEL(HEC): 434 mg/cu.m LOAEL: 4340 mg/cu.m (1000 ppm) LOAEL(ADJ): 4340 mg/cu.m LOAEL(HEC): 4340 mg/cu.m	300	1	1E+0 mg/cu.m

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*Conversion Factors: MW = 106.18. Assuming 25C and 760 mmHg, NOAEL(mg/cu.m) =
100 ppm x MW/24.45 = 434 mg/cu.m. For developmental effects, this concentration is not adjusted; therefore, NOAEL(ADJ) = NOAEL. The NOAEL(HEC) was calculated for a gas:extrarespiratory effect, assuming periodicity was attained. Since b:a lambda values are unknown for the experimental animal species (a) and humans (h), a default value of 1.0 was used for this ratio. NOAEL(HEC) = NOAEL(ADJ) x (b:a lambda(a)/lambda(h)) = 434 mg/cu.m.

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o INHALATION RFD STUDIES :

Andrew, F.D., R.L. Buschbom, W.C. Cannon, R.A. Miller, L.F. Montgomery, D.W. Phelps, et al. 1981. Teratologic assessment of ethylbenzene and 2-ethoxyethanol. Battelle Pacific Northwest Laboratory, Richland, WA. PB 83-208074., 108.

Hardin, B.D., G.P. Bond, M.R. Sikov, F.D. Andrew, R.P. Beliles and R.W. Niemeier. 1981. Testing of selected workplace chemicals for teratogenic potential. Scand. J. Work Environ. Health. 7(suppl 4): 66-75.

Inhalation experiments were conducted with Wistar rats (n=78-107/concentration) and New Zealand white rabbits (n=29-30/concentration) exposed 6 to 7 hours/day, 7 days/week during days 1-19 and 1-24 of gestation, respectively, to nominal concentrations of 0, 100, or 1000 ppm (434 or 4342 mg/cu.m) (Andrew et al., 1981). A separate group of rats was exposed pregestationally for 3 weeks prior to mating and exposure was continued into the gestational period. Actual concentrations were within 10% of target

concentrations. All pregnant animals were sacrificed 1 day prior to term (21 days for rats; 30 days for rabbits). Maternal organs (liver, lungs, kidney, heart, spleen, adrenals, ovaries, and brain) were examined histopathologically. Uteri were examined and fetuses were weighed, sexed, and measured for crown-to-rump length, and examined for external, internal and skeletal abnormalities. For statistical analyses, the litter was chosen as the experimental unit.

Ethylbenzene did not elicit embryotoxicity, fetotoxicity, or teratogenicity in rabbits at either exposure level. There were no significant incidences of major malformations, minor anomalies, or common variants in fetal rabbits from exposed groups. Maternal toxicity in the rabbits was not evident. There was no evidence of histologic damage in any of the dams' organs. The principal observation noted by the investigators was a reduced number of live rabbit kits per litter ($p<0.05$) at both exposure levels when evaluated by ANOVA and Duncan's Multiple Range Test. The number of live kits per litter in the air-exposed controls was reported as 8 (3+/-s.d.), compared with 7 (3+/-s.d.) for each exposure group. However, if one recalculates the data presented in Table 9 of Andrew et al. (1981), the number of live kits per litter for the low concentration (100 ppm) was 8 rather than 7 as presented in the paper. Since the number of live kits per litter at the high concentration was 7, this may suggest an effect at 1000 ppm, but not at 100 ppm. However, the number of implantations per litter and the number of dead or resorbed per litter were not different from controls. Prenatal mortality ranged from 5 to 8% and preimplantation loss ranged from 18 to 27%. Neither indicated a concentration-related intrauterine mortality. The results of the rabbit study are indicative of a NOAEL of 100 ppm based on a lack of developmental effects in rabbits. The NOAEL(HEC) is 434 mg/cu.m.

In rats exposed only during gestation, there were no histopathological effects in any of the maternal organs examined. There was no effect on fertility or on any of the other measures of reproductive status. The principal observation in fetuses was an increased incidence ($p<0.05$) of supernumerary and rudimentary ribs in the high exposure group and an elevated incidence of extra ribs in both the high and 100 ppm groups. Both absolute and relative liver, kidney, and spleen weights were significantly increased in pregnant rats from the 1000 ppm group.

Groups of female rats were also exposed for 3 weeks prior to mating and exposure was continued during gestation. Like the 1000-ppm group exposed only during gestation, there was also an increased incidence of extra ribs ($p<0.05$) in the pre-gestationally exposed high exposure group. However, an increased incidence was not seen at 100 ppm in those exposed pre-gestationally, in contrast to the comparable group exposed only during gestation. There was no increase in rudimentary ribs in either of exposed groups. When extra and rudimentary ribs were grouped together, there was no significant increase in supernumerary ribs in either of the exposed groups. The apparent discrepancy in the incidence of supernumerary ribs between the pregestationally-exposed group and those exposed only during gestation may be based, in part, on the fewer numbers of litters examined in the pregestationally-exposed group.

There were no effects on fertility or on any of the of the other measures of reproductive status. No fetal toxicity was noted at either exposure level. Body weights, placental weights, and sex ratios were within normal limits. Absolute and relative liver and spleen weights were significantly increased in pregant rats from the 1000 ppm group; only relative kidney weight was increased significantly. There were no histopathological effects in any of the organs examined.

Skeletal variants were seen at both 434 and 4342 mg/cu.m in the rats with the effects at 432 mg/cu.m being reduced compared with those occurring at 4342 mg/cu.m. By themselves, the effects are marginally adverse, even at 4342 mg/cu.m. However, a weight-of-evidence approach, noting a cluster of other mild effects at 4342 mg/cu.m, is used to determine that 1000 ppm is a LOAEL. The skeletal variations are considered along with evidence of slightly reduced litter size in rabbits at 4342 mg/cu.m and an increase in "% skeletal retarded fetuses" at 600 mg/cu.m (Ungvary and Tatrai, 1985). Additional support for this position is derived from the observations of somewhat elevated maternal liver, kidney, and spleen weights (Andrew et al., 1981).

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o INHALATION RFD UNCERTAINTY :

UF = 300. The uncertainty factor of 300 reflects a factor of 10 to protect unusually sensitive individuals, 3 to adjust for interspecies conversion and 10 to adjust for the absence of multigenerational reproductive and chronic studies.

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o INHALATION RFD MODIFYING :

MF = 1.
FACTOR

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o INHALATION RFD COMMENTS :

Ungvary and Tatrai (1985) exposed CFY rats (n=17-20) to levels of 600, 1200, or 2400 mg/cu.m for 24 hours/day during days 7 to 15 of gestation. CFLP mice (n=20) were exposed to 500 mg/cu.m for 24 hours/day from gestational days 6 to 15 or for 3 days intermittently for 4 hours/day for days 6-15. It is not clear from the description if the results pertain to the continuous exposure or the intermittent exposure. New Zealand rabbits (n=3-9) were exposed for 24 hours/day to concentrations of 500 or 1000 mg/cu.m from gestational days 7 to 20. Untreated animals and those exposed to air only served as controls.

It was stated that maternal toxicity (unspecified species) was moderate and concentration-dependent; however, no data were presented to support this statement. Maternal weight gain was reported to have decreased for rabbits exposed to 1000 mg/cu.m. It was reported that rabbits exposed to 1000 mg/cu.m exhibited mild maternal toxicity manifested by reduced weight gain. However, the percent weight gain was not reported. There were no data for developmental endpoints in the 1000-ppm group because there were no live fetuses. One dam had died and three others aborted in this exposure group. Four dams had total

resorptions. However, four other compounds in addition to ethyl benzene were tested at 1000 mg/cu.m and all caused spontaneous abortions at this level. Thus, the results are not clearly indicative of a treatment-related effect. This observation, coupled with the lack of any indication of abortions in rabbits in the Hardin et al. (1981) study, suggests that this effect in rabbits is not treatment-related.

Ungvary and Tatrai (1985) did observe a significant reduction in the mean female fetal weight in rabbit dams exposed 24 hours/day to 500 mg/cu.m. Andrew et al. (1981) did not observe such an effect in rabbits exposed up to 4348 mg/cu.m. These conflicting results in rabbits might be attributable to differences in study design.

Postimplantation loss (% dead or resorbed fetuses), and exposure-related skeletal retardation were significantly elevated ($p<0.05$) in rats at all exposure levels with one exception. Exposure to 600 mg/cu.m for 6 hours/day (it was not stated if this was a single exposure or the exposure duration on each day of gestation) did not result in any statistically significant fetal effects although there was increased incidence of dead/resorbed fetuses, lower weight of fetuses, and skeletal retarded fetuses. In the 24-hour/day exposure groups, malformations characterized as "anomalies of the uropoietic apparatus" and an increased incidence of extra ribs were significantly increased only at the highest exposure level. No data were presented on the anomalies of the uropoeitic apparatus. There was a significant ($p<0.05$) increase in skeletal retardation and fetal resorption in all continuous exposure groups although the concentration-response was shallow. The percent skeletal retarded fetuses, for example, at exposure concentrations of 600, 1200, and 2400 mg/cu.m was 26, 30, and 35%, respectively; the incidence in controls was 13%. These results in rats suggest a LOAEL(HEC) of 2400 mg/cu.m for extra ribs in the absence of demonstrable maternal toxicity.

In mice, an increased incidence of "anomalies of the uropoietic apparatus" was the only observation, but no data were presented. There was no discussion concerning maternal toxicity.

A 90-day subchronic inhalation study was conducted in F344/N rats (n=10/sex/group) and B6C3F1 mice (n=10/sex/group) that were exposed to 0, 100, 250, 500, 750, and 1000 ppm (0, 434, 1086, 2171, 3257, and 4343 mg/cu.m) 6 hours/day, 5 days/week (NTP, 1988; 1989; 1990). The duration-adjusted values were 0, 77.5, 194, 388, 582, and 776 mg/cu.m, respectively. The test atmosphere concentrations monitored by gas chromatography were within a 10% range of the target concentrations. At study termination, necropsies were conducted on the lung, liver, kideny, heart, testes, and thymus with organ weight measurements. Clinical chemistry data were obtained for rats. Histopathological examinations were conducted on all animals in the high concentration groups and in controls; animals in the lower concentration groups were evaluated when lesions were observed until no observed effects were seen. Sperm morphology and vaginal cytology tests were performed. There were no mortalities, exposure-related clinical signs of toxicity, or significant adverse effects on body weight in any of the exposed rats or mice.

In rats, hematology parameters were unaffected. Of the liver enzymes evaluated, only serum alkaline phosphatase (SAP) activity was significantly reduced in a concentration-related manner (at 500 ppm and above) for both sexes with a greater sensitivity in females. The significance of this decrease is not clear since in liver damage, SAP levels usually increase. The investigators suggested the decrease may be due to reduced water and food intake. No liver histopathology was noted for any exposure group. Significant concentration-related increases in absolute liver weights occurred in males at 250 ppm and higher (12.5, 17.3, 22.0, and 23.6% at 250, 500, 750, and 1000 ppm, respectively); in females the lowest concentration at which an increase in absolute liver weight was seen was in the 500-ppm group (11.8%). The increase in the 750- and 1000-ppm groups was 11.5 and 15.8%, respectively. Relative liver weights were significantly increased in all male exposure groups except the 100-ppm group while all female exposure groups except the two lowest groups showed significant increases. Absolute kidney weight in males significantly increased only in the 500- and 750-ppm groups; relative weight was increased in the three highest exposure groups. In females, both absolute and relative kidney weights increased significantly in the three highest exposure groups. Regeneration of renal tubules in the kidneys of male rats only was seen in all groups including controls. The severity of the lesions was greatest in the rats at in the high-exposure group.

The most significant gross observation in rats was the presence of enlarged bronchial and/or mediastinal lymph nodes, but these observations were not dose-related. The incidence for minimal lung inflammation in male rats was 0/10, 3/10, 9/10, 9/10, 8/10, and 10/10 for the 0-, 100-, 250-, 500-, 750-, and 1000-ppm exposure groups, respectively. Microscopically, this enlargement was attributable to an increase in normal constituents of the lymph nodes characterized by accumulations of macrophages, lymphocytes, neutrophils, and plasma cells. It was the opinion of the NTP Pathology Working Group (PWG) that hyperplasia of the lymph nodes and lower respiratory tract was typical of an infectious agent with an associated active immune response rather than ethylbenzene exposure (NTP, 1989). This diagnosis was supported by the following observations: an uneven distribution of lesions among and within groups; foci of airway inflammation were randomly distributed throughout the lungs; considerable variability in severity within groups; and there was no consistent concentration-response relationship. No lesions were seen in the nasal cavity. The PWG described these lesions as not typical of the type of lesions which occurs with known pulmonary irritants. These lesions were not found in control animals, which were housed in separate rooms. No infectious agent was identified upon serologic examination. In the draft NTP technical report (NTP, 1990), the inflammatory lung lesions were described as probably unrelated to exposure. Antibodies to common rodent respiratory tract viruses were not detected. However, only sera from control rats were sampled. Lesions morphologically indistinguishable from those in this study have been seen in control and treatment groups of rats from other inhalation and dosed feed studies (NTP, 1990). The PWG recommended that this effect be reevaluated in another study.

In mice, no significant exposure-related gross or histopathological observations were noted at terminal necropsy of any organs, including the lung. The only exposure-related effects were significantly elevated absolute and relative liver weight in both sexes of mice at of 750 and 1000 ppm and significantly elevated relative kidney weight of the females exposed to 1000 ppm. There were no significant histopathological changes or function test alterations in either liver or kidney of either sex.

The NTP peer review of the subchronic study took place on November 20, 1990 at Research Triangle Park. The NTP Board of Scientific Counselors' panel of experts agreed with the conclusions of the NTP report that there were no indications of toxicity due to ethyl benzene. A 2-year lifetime study in both rats and mice has been initiated and exposures have been conducted through 7 months. No serial sacrifices are planned and results are not expected prior to 1992.

Clark (1983) exposed Wistar rats (n=18/sex/group) (12-13 weeks old) to 0 and 100 ppm (0 and 434 mg/cu.m) reagent grade ethylbenzene 6 hours/day, 5 days/week for 12 weeks. The duration-adjusted values were 0 and 77.5 mg/cu.m. Clinical observations, body weight, food intake, hematology, urinalysis, organ weights, and histopathology of all major organs (including the lung and nasal cavity) were used as parameters to assess toxicity. No statistically significant effects were observed at 100 ppm. There were no differences from controls in the liver enzymes, including SAP. While slight bile duct hyperplasia was seen in 15/18 exposed males and 14/18 exposed females, hyperplasia was also common in controls (10/18 females and 8/18 males), and these observations were not statistically significant. The results of this study suggest a NOAEL of 100 ppm. The NOAEL(HEC) is 77.5 mg/cu.m. The results are in general agreement with the findings of the NTP study in F344 rats.

Wolf et al. (1956) exposed rats (n=10-25/sex/group) to 400, 600 or 1250 ppm (1737, 2606, or 5428 mg/cu.m) ethylbenzene 7 hours/day, 5 days/week for about 6 months. The duration-adjusted values were 0, 362, 542, and 1131 mg/cu.m, respectively, using the 7-hour duration. Exposure ranged from 186 to 214 days. Male rats only were also exposed to 2200 ppm (9554 mg/cu.m) for 7 hours/day, 5 days/week for about 5 months. The duration-adjusted value was 1990 mg/cu.m. Histopathology was performed on a variety of organs including the lung. Data on liver and kidney weights and histopathology were not presented; these parameters were discussed only in descriptive terms. Repeated exposure of rats, guinea pigs, and rhesus monkeys was examined.

Growth was depressed moderately in male rats at 2200 ppm. Liver and kidney weights in rats were increased slightly in all exposed groups compared with matched controls, and rats exposed to 1250 and 2200 ppm developed histopathological changes manifested as cloudy swelling of the liver and renal tubules and testicular degeneration. The date indicate a NOAEL for liver histopatholgy at 600 ppm (542 mg/cu.m). However, no incidence data was reported. Since it is not clear that these effects are adverse when taken in context with the results of the NTP study, a NOAEL or LOAEL is not identified.

Guinea pigs (5-10/sex/group) and rabbits (1-2/sex/group) were exposed to 0, 400, or 600 ppm (duration-adjusted concentrations of 0, 362, or 542 mg/cu.m, respectively) ethylbenzene 7 hours/day, 5 days/week for about 6 months. Only females were exposed to 1250 ppm (duration-adjusted value of 1131 mg/cu.m). Growth was depressed in female guinea pigs exposed to 1250 ppm. Liver weight was described as slightly increased only in the 600-ppm exposure group. The study does not clearly indicate 600 ppm as a LOAEL so the NOAEL for guinea pigs is designated at 600 ppm. The NOAEL(HEC) is 542 mg/cu.m. Other than an observation of slight degeneration of the testicular germinal epithelium in the male rabbit at 600 ppm, there were no adverse effects reported for rabbits of either sex.

One male Rhesus monkey was exposed to 600 ppm (duration-adjusted value of 542 mg/cu.m) and two females were exposed to 400 ppm (duration-adjusted value of 362 mg/cu.m). A slight degeneration of the testicular germinal epithelium and increased liver weight was observed in the male monkey. No effects were reported for the female rhesus monkeys.

The small number of rabbits and monkeys preclude identification of NOAEL and LOAEL values for these species.

Cragg et al. (1989) exposed B6C3F1 mice (n=5/sex/group) and F344 rats (n=5/sex/group) to actual concentrations of 0, 99, 382, and 782 ppm (0, 430, 1659, and 3396 mg/cu.m) 6 hours/day, 5 days/week for 4 weeks. The duration-adjusted values were 0, 77, 296, 606 mg/cu.m, respectively. In the same study, New Zealand White rabbits (n=5/sex/group) were exposed to actual concentrations of 0, 382, 782, or 1610 ppm (0, 1659, 3396, or 6992 mg/cu.m). The duration-adjusted values were 0, 296, 606 and 1249 mg/cu.m, respectively. No changes were evident in mortality, clinical chemistry parameters, urinalysis, nor were there treatment-related gross or histopathological findings. Urinalysis was not performed on rabbits and clinical chemistry parameters were not performed on mice. Liver enzymes measured included AP. Hematology was performed on all species. Histopathology was only conducted on the high concentration animals except all rabbits' testes were examined. There was no liver histopathology in any of the species.

In the 382-ppm exposure group, rats exhibited sporadic incidences of salivation and lacrimation. (These observations were not noted in the NTP subchronic study). Absolute liver weights were significantly increased in male rats; relative weight was increased at 782 ppm. In females, absolute liver weight was significantly increased at 782 ppm and relative weight at both concentrations. Male rats of the 782 ppm group had a significant ($p<0.05$) increase in platelets while females only had a significant ($p<0.05$) increase in total leukocytes.

In mice, females showed a statistically significant increase in absolute, but not relative liver weight, at 782 ppm. There were no significant liver weight changes in male mice. Both males and females exhibited an increased liver weight relative to brain weight at 782 ppm only. Rabbits showed no

changes in liver weight ratios at any exposure level.

Since there were no adverse histopathological findings for the liver, a NOAEL of 782 ppm is identified for rats and mice. The NOAEL(HEC) is 606 mg/cu.m. The NOAEL for rabbits is 1610 ppm; the NOAEL(HEC) is 1249 mg/cu.m.

Elovaara et al. (1985) found concentration-related increases in drug-metabolizing enzymes of liver and kidney, with corresponding ultrastructural alterations in a subchronic inhalation study with rats. Male Wistar rats (n=5/group) were exposed to 0, 50, 300, or 600 ppm (0, 217, 1302, or 2604 mg/cu.m) ethylbenzene 6 hours/day, 5 days/week for 2, 5, 9, or 16 weeks. The duration-adjusted values were 0, 38.7, 233, and 465 mg/cu.m, respectively. The liver was the only organ examined histologically (light and electron microscopy). There were no changes in liver weight at any concentration. After 16 weeks exposure, NADPH-cytochrome reductase and UDPG-transferase were significantly elevated at 300 and 600 ppm. Aminopyrine N-demethylase and 7-ethoxycoumarin-0-deethylase (7-ECDE) were elevated at all exposure levels. The elevation in UDPG-transferase was exposure-related and may signify glucuronidation of ethylbenzene metabolites during detoxication. Electron microscopy also showed changes in hepatocyte ultrastructure [e.g., smooth endoplasmic reticulum (SER) proliferation, slight degranulation of rough endoplasmic reticulum] at all exposure levels beginning 2 to 9 weeks after exposure. Necrosis was not observed nor were there any increases in serum alanine aminotransferase. SAP was not measured. The proliferation of SER is consistent with enzyme induction. At 16 weeks, changes in ultrastructure were mainly confined to the high-exposure group. There was no effect of exposure on hepatic glutathione (GSH) content. Significant increases in relative kidney weight only were reported following 2 and 9, but not at 16 weeks of exposure to 600 ppm. Kidney 7-ECDE, and UDPG transferase activities showed statistically significant and exposure-related increases at all exposure levels.

In the absence of histologic evidence of damage, changes in absolute or relative liver weight, and no effect on serum ALT, the microsmal enzyme induction and ultrastructural changes are considered to be adaptation phenomena. The results of this study suggest a NOAEL of 600 ppm. The NOAEL(HEC) is 465 mg/cu.m for liver and kidney. The absence of liver weight changes is not consistent with the findings of the NTP (1988) subchronic study.

Angerer and Wulf (1985) evaluated 35 workers who chronically (2-24 years, average 8.2 years) sprayed varnishes containing alkyd-phenol and polyester resins dissolved in solvent mixtures consisting principally of xylene isomers and ethylbenzene. Some of the varnishes contained lead-based pigments. The air samples from personal monitors indicated average levels of 4.0 ppm for ethylbenzene. Although workers had significantly elevated lymphocytes in addition to significantly decreased erythrocyte counts and hemoglobin levels compared with controls, these effects cannot be attributed to ethylbenzene since other compounds (e.g., xylene, methylchloroform, n-butanol, toluene, C9 hydrocarbons) were detected in some of the six workplaces evaluated.

Bardodej and Cirek (1988) carried out biomonitoring of 200 ethylbenzene production workers occupationally exposed for a mean duration of 12.2 years to unspecified concentrations of ethylbenzene and benzene over a 20-year period. The workers were evaluated twice a year and ethylbenzene metabolites measured. No statistically significant differences in hematological effects (e.g., RBC, WBC, leukocyte and platelet counts) or liver function tests (e.g., aminotransferase and/or SAP and LDH activities and bilirubin tests) were observed between exposed and nonexposed workers.

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o INHALATION RFD CONFIDENCE : Study: Low Data Base: Low RfC: Low
developmental study by Hardin et al. (1981) was well-conducted and indicated no clearly adverse effects in any species. The study is given a low confidence rating because higher exposure levels may have provided more information on the potential for maternal toxicity and developmental effects. The data base is given a low rating since although other studies have examined a variety of other endpoints (e.g., liver and lung), by histopathology in rats and mice, there are no chronic studies and no multi-generation developmental studies. These latter studies would be useful to determine more conclusively the potential of ethylbenzene to affect development. NTP does not consider observations of lung lesions in rats exposed in the NTP subchronic study to be treatment-related. However, no infectious agent has been detected. Therefore, there remains a possibility that ethylbenzene may play a role in producing lung lesions. It is anticipated that this issue will be clarified upon completion of the chronic study in progress. In view of the previous considerations, the RfC is given a low confidence rating.

o INHALATION RFD SOURCE :

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- U.S. EPA, 1984; 1985; 1987.
DOCUMENT

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o REVIEW DATES : 09/19/90, 12/20/90
o VERIFICATION DATE : 12/20/90

o EPA CONTACTS :

Mark Greenberg / ORD -- (919)541-4156 / FTS 629-4156

Annie M. Jarabek / ORD -- (919)541-4847 / FTS 629-4847

CAREV-

o CLASSIFICATION : D; not classifiable as to human carcinogenicity.

o BASIS FOR CLASSIFICATION : nonclassifiable due to lack of animal bioassays and human studies.

o HUMAN CARCINOGENICITY DATA :

None.

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o ANIMAL CARCINOGENICITY DATA :

None. NTP has plans to initiate bioassay. Metabolism and excretion studies at 3.5, 35 and 350 mg/kg are to be conducted as well.

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o SUPPORTING DATA :

The metabolic pathways for humans and rodents are different (Engstrom et al., 1984). Major metabolites in humans, mandelic acid and phenylglyoxylic acid, are minor metabolites in rats and rabbits (Kiese and Lenk, 1974). The major animal metabolites were not detected in the urine of exposed workers (Engstrom et al., 1984).

Ethylbenzene at 0.4 mg/plate was not mutagenic for Salmonella strains TA98, TA1535, TA1537 and TA1538 with or without Aroclor 1254 induced rat liver homogenates (S9) (Nestmann et al., 1980). Ethylbenzene was shown to increase the mean number of sister chromatid exchanges in human whole blood lymphocyte culture at the highest dose examined without any metabolic activation system (Norppa and Vainio, 1983).

Dean et al. (1985) used a battery of short-term tests including bacterial mutation assays, mitotic gene conversion in Saccharomyces cerevisiae JD1 in the presence and absence of S9 and chromosomal damage in a cultured rat liver cell line. Ethylbenzene was not mutagenic in the range of concentrations tested (0.2, 2, 20, 50 and 200 ug/plate) for S. typhimurium TA98, TA100, TA1535, TA1537 and TA1538 or for Escherichia coli WP2 and WP2uvrA. Ethylbenzene also showed no response in the S. cerevisiae JD1 gene conversion assay. In contrast, ethylbenzene hydroperoxide showed positive responses with E. coli WP2 at 200 ug/plate in the presence of S9 and an equally significant response with the gene conversion system of yeast.

CARDR-

o CARCINOGENICITY SOURCE :

U.S. EPA. 1980. Ambient Water Quality Criteria Document for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-048. NTIS PB 81-117590.

U.S. EPA. 1984. Health Effects Assessment for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC. EPA/540/1-86/008.

U.S. EPA. 1987. Drinking Water Criteria Document for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

The Ambient Water Quality Criteria Document and the Health Assessment Document have received Agency and external review. The Drinking Water Criteria Document has been extensively reviewed.

DOCUMENT

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o REVIEW DATES : 10/07/87
o VERIFICATION DATE : 10/07/87
o EPA CONTACTS :

Arthur S. Chiu / ORD -- (202)260-6764 / FTS 260-6764

Lynn Papa / ORD -- (513)569-7523 / FTS 684-7523

HAONE-

One-day HA -- 3.2E+1 mg/L

NOAEL -- 31.8 mg/kg/day
UF -- 10 (allows for intrahuman variability with the use of a NOAEL from a human study)
Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Bardodej and Bardodejova, 1970

No adverse health effects were observed in human volunteers exposed to ethylbenzene by inhalation at a concentration of 100 ppm (435 mg/cu.m) for 8 hours. Based on the conditions of exposure and an assumed absorption factor of 64%, this is equivalent to a NOAEL of 31.8 mg/kg/day.

HATEN-

Appropriate data for calculating a Ten-day HA are not available. Therefore, the Ten-day HA has been calculated from the One-day HA by dividing the One-day HA of 32 mg/L by 10. The Ten-day HA is therefore 3.2 mg/L.

HALTC-

Appropriate data for calculating a Longer-term HA are not available. It is recommended that the modified DWEL (adjusted for a 10-kg child) of 0.97 mg/L (rounded to 1 mg/L) be used as the Longer-term HA.

HALTA-

Appropriate data for calculating a Longer-term HA are not available. It is recommended that the DWEL of 3.4 mg/L be used as the Longer-term HA for the 70-kg adult.

HALIF-

Drinking Water Equivalent Level (DWEL) -- 3.4E+0 mg/L

Assumptions -- 2 L/day water consumption for a 70-kg adult

RfD Verification Date = 05/20/85 (see RDO)

Lifetime HA -- 6.8E-1 mg/L

Assumptions -- 20% exposure by drinking water

Principal Study -- Wolf et al., 1956 (This study was used in the derivation of the chronic oral RfD; see RDO)

OLEP -

Taste perception threshold (water) -- 0.029 mg/L.

Odor perception threshold (water) -- 0.029 mg/L.

Odor perception threshold (air) -- 0.062 mg/L.

ALAB -

Analysis of ethylbenzene is by a purge-and-trap gas chromatographic procedure used for the detection of volatile organic compounds in water. Confirmatory analysis is by mass spectrometry.

TREAT-

Ethylbenzene is most effectively removed from water by air stripping. Adsorption on activated carbon is at least partially effective in the removal of ethylbenzene from solution. Conventional treatment processes may also be effective.

HADR -
o HEALTH ADVISORY SOURCE :

U.S. EPA. 1985. Final Draft of the Drinking Water Criteria Document on Ethylbenzene. Office of Drinking Water, Washington, DC.
DOCUMENT

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o HEALTH ADVISORY REVIEW :

EPA review of HAs in 1985.

Public review of HAs following notification of availability in October, 1985.

Scientific Advisory Panel review of HAs in January, 1986.

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o EPA DRINKING WATER CONTACT :

Charles O. Abernathy / ODW -- (202)260-5374 / FTS 260-5374

Edward V. Ohanian / ODW -- (202)260-7571 / FTS 260-7571

WQCHU-

Water and Fish Consumption: 1.4E+3 ug/L

Fish Consumption Only: 3.28E+3 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of 1.4E+3 ug/L is based on consumption of contaminated aquatic organisms and water. A WQC of 3.28E+3 ug/L has also been established

based on consumption of contaminated aquatic organisms alone.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC -- 3.2 E+ 4 ug/L
Chronic LEC -- none

Marine:

Acute LEC -- 4.3 E+ 2 ug/L
Chronic LEC -- none

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LEC's are given when the minimum data required to derive water quality criteria are not available.

Reference -- 43 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 0.7 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0.7 mg/L for ethylbenzene is promulgated based upon reported histopathological changes (lesion not specified) in a 6-month oral study in rats. The MCLG is based upon a DWEL of 3.4 mg/L and an assumed drinking water contribution of 20 percent.

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 0.7 mg/L (Final, 1991)

Considers technological or economic feasibility? -- YES

Discussion -- The EPA has promulgated a MCL that is equal to the MCLG of 0.7 mg/L.

Monitoring requirements -- All systems initially monitored for four consecutive quarters; repeat monitoring dependent upon detection, vulnerability status and system size.

Analytical methodology -- Gas chromatographic/mass spectrometry (EPA 524.2): purge and trap capillary gas chromatography (EPA 502.2); gas chromatography/ mass spectrometry (EPA 524.1); purge and trap gas chromatography (EPA 503.1); PQL= 0.005 mg/L.

Best available technology -- Granular activated carbon; packed tower aeration.

Reference -- 56 FR 3526 (01/30/91); 56 FR 30266 (07/01/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

Value -- 0.03 mg/L (Proposed, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- SMCLs are non-enforceable and establish limits for contaminants which may affect the aesthetic qualities (e.g. taste and odor) of drinking water. It is recommended that systems monitor for these contaminants every three years. More frequent monitoring for contaminants such as pH, color, odor or others may be appropriate under certain circumstances. The SMCL for ethylbenzene is based on odor qualities. Promulgation deferred following public comment (56 FR 3526 see below).

Reference -- 54 FR 22062 (05/22/89); 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

CERC -

Value (status) -- 1000 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ is based on aquatic toxicity, as established under Section 311(b)(4) of the Clean Water Act (40 CFR 117.3), and ignitability. Available data indicate that the aquatic 96-Hour Median Threshold Limit for ethylbenzene is between 10 and 100 ppm. The closed-cup flash point is less than 100F and the boiling point is >100F.

Reference -- 50 FR 13456 (04/04/85); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - U.S. EPA. 1980. Ambient Water Quality Criteria Document for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-048. NTIS PB 81- 117590.

OREF - U.S. EPA. 1985. Drinking Water Criteria Document for Ethylbenzene.

Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (Final draft)

OREF - U.S. EPA. 1984. Health Effects Assessment for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC.

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

IREF - Andrew, F.D., R.L. Buschbom, W.C. Cannon, R.A. Miller, L.F. Montgomery, D.W. Phelps, et al. 1981. Teratologic assessment of ethylbenzene and 2- ethoxyethanol. Battelle Pacific Northwest Laboratory, Richland, WA. PB 83- 208074., 108.

IREF - Angerer, J. and H. Wulf. 1985. Occupational chronic exposure to organic solvents. XI. Alkylbenzene exposure of varnish workers: Effects on hematopoietic system. Int. Arch. Occup. Environ. Health. 56(4): 307-21.

IREF - Bardodej, Z. and A. Cirek. 1988. Long-term study on workers occupationally exposed to ethylbenzene. J. Hyg. Epidemiol. Microbiol. Immunol. 32(1): 1-5.

IREF - Cragg, S.T., E.A. Clarke, I.W. Daly, R.R. Miller, J.B. Terrill and R.E. Quellette. 1989. Subchronic inhalation toxicity of ethylbenzene in mice, rats, and rabbits. Fund. Appl. Toxicol. 13(3): 399-408.

IREF - Clark, D.G. 1983. Ethylbenzene hydroperoxide (EBHP) and ethylbenzene (EB): 12 week inhalation study in rats. (Group research report with attachments and cover sheet.) EPA OTS Public Files. Shell Oil Co. Document No. 86870001629. Fiche Number 0516206 (2).

IREF - Elovaara, E., K. Engstrom, J. Nickels, A. Aito and H. Vainio. 1985. Biochemical and morphological effects of long-term inhalation exposure of rats to ethylbenzene. Xenobiotica. 15(4): 299-308.

IREF - Hardin, B.D., G.P. Bond, M.R. Sikov, F.D. Andrew, R.P. Beliles and R.W. Niemeier. 1981. Testing of selected workplace chemicals for teratogenic potential. Scand. J. Work Environ. Health. 7(suppl 4): 66-75.

IREF - NTP (National Toxicology Program). 1988. Subchronic and chronic toxicity study of ethylbenzene. 90-Day subchronic study report on inhalation exposure of F344/N rats and B6C3F1 mice. Principal investigator: Catherine Aranyi. IIT Research Institute, Chicago, IL.

IREF - NTP (National Toxicology Program). 1989. Chairperson's report. Pathology Working Group (PWG) review of subchronic toxicity testing on ethylbenzene administered by inhalation in F344 rats and B6C3F1 mice.

IREF - NTP (National Toxicology Program). 1990. Draft NTP Technical Report on the Toxicity Studies of ethylbenzene in F344 rats and B6C3F1 mice (Inhalation Studies). NTP TOX 10, U.S. DHHS.

IREF - Ungvary, G. and E. Tatrai. 1985. On the embryotoxic effects of

benzene and its alkyl derivatives in mice, rats, and rabbits. Arch. Toxicol. Suppl 8: 425-430.

IREF - U.S. EPA. 1984. Health Effects Assessment for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC. EPA/540/1-86-008.

IREF - U.S. EPA. 1985. Drinking Water Criteria Document for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. NTIS PB 86-117835. (Final Draft).

IREF - U.S. EPA. 1987. Health Advisory for Ethylbenzene. Office of Drinking Water, Washington, DC.

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

CREF - Dean, B.J., T.M. Brooks, G. Hodson-Walker and D.H. Hutson. 1985. Genetic toxicology testing of 41 industrial chemicals. Mutat. Res. 153: 57-77.

CREF - Engstrom, K., V. Riihimaki and A. Laine. 1984. Urinary disposition of ethylbenzene and m-xylene in man following separate and combined exposure. Int. Arch. Occup. Environ. Health. 54: 355-363.

CREF - Kiese, M. and W. Lenk. 1974. Hydroxyacetophenones: Urinary metabolites of ethylbenzene and acetophenone in the rabbit. Xenobiotica. 4(6): 337-343.

CREF - Nestmann, E.R., E.G-H. Lee, T.I. Matula, G.R. Douglas and J.C. Mueller. 1980. Mutagenicity of constituents identified in pulp and paper mill effluent using the Salmonella/mammalian-microsome assay. Mutat. Res. 79: 203-212.

CREF - Norppa, H. and H. Vainio. 1983. Induction of sister-chromatid exchanges by styrene analogues in cultured human lymphocytes. Mutat. Res. 116: 379-387.

CREF - U.S. EPA. 1980. Ambient Water Quality Criteria Document for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-048. NTIS PB 81- 117590.

CREF - U.S. EPA. 1984. Health Effects Assessment for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC.

CREF - U.S. EPA. 1987. Drinking Water Criteria Document for Ethylbenzene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (Final report)

HAREF- Bardodej, Z. and E. Bardodejova. 1970. Biotransformation of ethylbenzene, styrene and alpha-methylstyrene in man. Am. Ind. Hyg. Assoc. J. 31(2): 206-209.

HAREF- Wolf, M.A., V.K. Rowe, D.D. McCollister, R.L. Hollingsworth and F.

Oyen. 1956. Toxicological studies of certain alkylated benzenes and benzene. Arch. Ind. Health. 14: 387-398.
HAREF- U.S. EPA. 1985. Drinking Water Criteria Document on Ethylbenzene. Office of Drinking Water, Washington, DC. (Final draft)

[IRIS] SS 2 /cf?
USER:

108-10-1
Search in progress

SS (9) PSTG (1)

[IRIS] SS 10 /cf?
USER:
prt dl ncar, car continuous

1 - IRIS
IRSN - 170
DATE - 930802
UPDT - 08/02/93, 2 fields
STAT - Oral RfD Assessment (RDO) withdrawn 08/01/93
STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) no data
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 03/01/88 RDO Text revised
IRH - 07/01/89 REFS Bibliography on-line
IRH - 04/01/90 RDI Inhalation RfD now under review
IRH - 03/01/91 RDO Oral RfD withdrawn pending additional review
IRH - 03/01/91 REFS Bibliography withdrawn
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 08/01/93 RDO Work group review date added
IRH - 08/01/93 RDO EPA contact changed
RLEN - 2596
NAME - Methyl isobutyl ketone (MIBK)
RN - 108-10-1
SY - HEXON
SY - HEXONE
SY - ISOBUTYL-METHYLKETON
SY - ISOBUTYL METHYL KETONE
SY - ISOPROPYLACETONE
SY - KETONE, ISOBUTYL METHYL
SY - METHYL-ISOBUTYL-CETONE
SY - METHYLISOBUTYLKETON
SY - Methyl Isobutyl Ketone
SY - 4-METHYL-PENTAN-2-ON
SY - 2-METHYL-4-PENTANONE
SY - 4-METHYL-2-PENTANONE
SY - METILISOBUTILCHETONE
SY - 4-METILPENTAN-2-ONE
SY - METYLOIZOBUTYLOKETON
SY - MIBK
SY - MIK
SY - 2-PENTANONE, 4-METHYL-
SY - RCRA WASTE NUMBER U161
SY - SHELL MIBK

SY - UN 1245

RDO -
o ORAL RFD SUMMARY :

The Oral RfD for this substance has been withdrawn pending further review by the RfD/RfC Work Group.

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o REVIEW DATES : 05/30/86, 02/20/91, 07/22/93

RDI -
o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

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o REVIEW DATES : 11/16/88, 03/22/90, 05/16/90, 02/20/90

FISTD-

Status -- List "C" Pesticide (1989)

Reference -- 54 FR 30846 (07/24/90)

EPA Contact -- Registration Branch / OPP
(703)557-7760 / FTS 557-7760

FIREV-

No data available

CERC -

Value (status) -- 5000 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final adjusted RQ for this substance is 5000 pounds, based on application of the secondary criterion of biodegradation to the primary criterion RQ of 1000 pounds determined by ignitability. Available data indicate a flash point of 64F and a boiling point of 224F. The final RQ takes into account the biodegradation of methyl isobutyl ketone [BOD5 - 4.4%, BOD5 - 56% (sewage seed), BOD20 - 57%, BOD20 - 65%]. Therefore, the 1000-pound RQ based on ignitability has been adjusted upward one level to 5000 pounds.

Reference -- 50 FR 13456 (04/04/85); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

[IRIS] SS 10 /cf?
USER:

prt dl ncar, car continuous

1 - IRIS
IRSN - 103
DATE - 920406
UPDT - 04/06/92, 3 fields
STAT - Oral RfD Assessment (RDO) on-line 03/01/88
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) pending
STAT - Drinking Water Health Advisories (DWHA) on-line 03/01/88
STAT - U.S. EPA Regulatory Actions (EXSR) withdrawn 04/01/92
IRH - 12/23/87 RDO RfD withdrawn pending further review
IRH - 03/01/88 RDO Revised Oral RfD sumary added - RfD changed
IRH - 03/01/88 HADV Health Advisory added
IRH - 07/01/89 REFS Bibliography on-line
IRH - 05/01/90 CAR Carcinogen assessment now under review
IRH - 06/01/90 CAA Area code for EPA contact corrected
IRH - 06/01/90 RCRA EPA contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 04/01/92 EXSR Regulatory action section withdrawn
RLEN - 12623
NAME - Tetrachloroethylene
RN - 127-18-4
SY - Ankilostin
SY - Antisal 1
SY - Antisol 1
SY - Carbon bichloride
SY - Carbon dichloride
SY - Czterochloroetylen
SY - Dee-Solv
SY - Didakene
SY - Didokene
SY - Dowclene EC
SY - Dow-Per
SY - ENT 1,860
SY - Ethene, tetrachloro-
SY - Ethylene tetrachloride
SY - Ethylene, tetrachloro-
SY - Fedal-Un
SY - NCI-C04580
SY - Nema
SY - PCE
SY - PER
SY - Perawin
SY - PERC
SY - Perchloorethyleen, per
SY - Perchlor
SY - Perchloraethylen, per
SY - Perchlorethylene

SY - Perchlorethylene, per
SY - Perchloroethylene
SY - Perclene
SY - Percloroetilene
SY - Percosolv
SY - Percosolve
SY - PERK
SY - Perklone
SY - Persec
SY - Tetlen
SY - Tetracap
SY - Tetrachlooretheen
SY - Tetrachloraethen
SY - Tetrachlorethylene
SY - Tetrachloroethene
SY - Tetrachloroethylene
SY - 1,1,2,2-Tetrachloroethylene.
SY - Tetracloroetene
SY - Tetraguer
SY - Tetraleno
SY - Tetralex
SY - Tetravec
SY - Tetroguer
SY - Tetropil
SY - WLN: GYGUYGG

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Hepatotoxicity in mice, weight gain in rats	NOAEL: 20 mg/kg/day (converted to 14 mg/kg/day)	1000	1	1E-2 mg/kg/day
6-Week Mouse Gavage Study Buben and O'Flaherty, 1985	LOAEL: 100 mg/kg/day (converted to 71 mg/kg/day)			

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*Conversion Factors: Doses have been adjusted for treatment schedule (5 days/week)

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o ORAL RFD STUDIES :

Buben, J.A. and E.J. O'Flaherty. 1985. Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: a dose-effect study. Toxicol. Appl. Pharmacol. 78: 105-122.

Buben and O'Flaherty (1985) exposed Swiss-Cox mice to tetrachloroethylene in corn oil by gavage at doses of 0, 20, 100, 200, 500, 1500, and 2000 mg/kg, 5 days/ week for 6 weeks. Liver toxicity was evaluated by several parameters including liver weight/body weight ratio, hepatic triglyceride concentration, DNA content, histopathological evaluation, and serum enzyme levels. Increased liver triglycerides were first observed in mice treated with 100 mg/kg. Liver weight/body weight ratios were significantly higher than controls for animals treated with 100 mg/kg. At higher doses, hepatotoxic effects included decreased DNA content, increased SGPT, decreased levels of G6P and hepatocellular necrosis, degeneration and polyploidy.

A NOEL of 14 mg/kg/day was established in a second study, as well (Hayes et al., 1986). Groups of 20 Sprague-Dawley rats of both sexes were administered doses of 14, 400, or 1400 mg/kg/day in drinking water. Males in the high-dose group and females in the two highest groups exhibited depressed body weights. Equivocal evidence of hepatotoxicity (increased liver and kidney weight/body weight ratios) were also observed at the higher doses.

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o ORAL RFD UNCERTAINTY :

UF = 1000. The uncertainty factor of 1000 results from multiplying factors of 10 to account for intraspecies variability, interspecies variability and extrapolation of a subchronic effect level to its chronic equivalent.

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o ORAL RFD MODIFYING FACTOR :

MF = 1.

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o ORAL RFD COMMENTS :

Other data support the findings of the principal studies. Exposure of mice and rats to tetrachloroethylene by gavage for 11 days caused hepatotoxicity (centrilobular swelling) at doses as low as 100 mg/kg/day in mice (Schumann et al., 1980). Mice were more sensitive to the effects of tetrachloroethylene exposure than rats. Increased liver weight was observed in mice at 250 mg/kg, while rats did not exhibit these effects until doses of 1000 mg/kg/day were reached. Relative sensitivity to man cannot be readily established but the RfD of 1E-2 mg/kg/day is protective of the most mild effects observed in humans [diminished odor perception/modified Romberg test scores in volunteers exposed to 100 ppm for 7 hours; roughly equivalent to 20 mg/kg/day (Stewart et al., 1961)].

The principal studies are of short duration. Inhalation studies have been performed which indicate that the uncertainty factor of 10 is sufficient for extrapolation of the subchronic effect to its chronic equivalent. Liver enlargement and vacuolation of hepatocytes were found to be reversible lesions for mice exposed to low concentrations of tetrachloroethylene (Kjellstrand et al., 1984). In addition, elevated liver weight/body weight ratios observed in animals exposed to tetrachloroethylene for 30 days were similar to those in animals exposed for 120 days. Several chronic inhalation studies have also

been performed (Carpenter, 1937; NTP, 1985; Rowe et al., 1952). None are inconsistent with a NOAEL of 14 mg/kg/day for tetrachloroethylene observed by Buben and O'Flaherty (1985) and Hayes et al. (1986).

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o ORAL RFD CONFIDENCE :

Study: Low
Data Base: Medium
RfD: Medium

No one study combines the features desired for deriving an RfD: oral exposure, large number of animals, multiple dose groups, testing in both sexes and chronic exposure. Confidence in the principal studies is low mainly because of the lack of complete histopathological examination at the NOAEL in the mouse study. The data base is relatively complete but lacks studies of reproductive and teratology endpoints subsequent to oral exposure; thus, it receives a medium confidence rating. Medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

U.S. EPA. 1985. Health Assessment Document for Tetrachloroethylene (Perchloroethylene). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC for the Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA 600/8-82/005F.

U.S. EPA. 1987. Quantification of Toxicological Effects for Tetrachloroethylene. Prepared from the Health Assessment Document for Tetrachloroethylene (Perchloroethylene). Office of Drinking Water, Washington, DC.

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o REVIEW DATES : 05/20/85, 08/05/86, 09/17/87
o VERIFICATION DATE : 09/17/87
o EPA CONTACTS :

Krishan Khanna / OST -- (202)260-7588 / FTS 260-7588

Michael L. Dourson / ORD -- (513)569-7544 / FTS 684-7544

HAONE-

The available studies were not considered sufficient for calculation of a One-day HA. It is recommended that the value for the Ten-day HA, 2 mg/L, be use for the One-day HA.

HATEN-

Ten-day HA -- 2E+0 mg/L

NOAEL -- 20 mg/kg/day
UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)
Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Buben and O'Flaherty, 1985

Male Swiss-Cox mice were administered tetrachloroethylene by gavage at doses of 0, 20, 100, 200, 500, 1000, 1500, and 2000 mg/kg, 5 days/week for 6 weeks. Liver toxicity was evaluated by several parameters including liver weight-to-body weight ratio, hepatic triglyceride concentrations, DNA content, histopathological evaluation and serum enzyme levels. Increased liver triglycerides were first observed in mice treated with 100 mg/kg. Liver weight/body weight ratios were significantly higher than controls for the 100 mg/kg group, and slightly higher than controls in the 20 mg/kg group. A NOAEL of 20 mg/kg/day was identified based on the absence of hepatotoxic effects. After 5 days of exposure, a NOAEL of 20 mg/kg/day was identified.

HALTC-

Longer-term (Child) HA -- 1.4E+0 mg/L

NOAEL -- 14 mg/kg/day (adjusted for dosing schedule of 5 days/week)
UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)
Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Buben and O'Flaherty, 1985 (study described in HATEN)

HALTA-

Longer-term (Adult) HA -- 5.0E+0 mg/L

NOAEL -- 14 mg/kg/day (adjusted for dosing schedule of 5 days/week)
Assumptions -- 2 L/day water consumption for a 70-kg adult
UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)

Principal Study -- Buben and O'Flaherty, 1985 (study described in HATEN)

HALIF-

Drinking Water Equivalent Level (DWEL) -- 5E-1 mg/L

Assumptions -- 2 L/day water consumption for a 70-kg adult

RfD Verification Date -- 05/20/85 (see RDO)

NOTE: The classification of this substance is currently under review. A final decision on whether this substance should be classified B2 or C has not yet been made. If this substance is classified as C, the lifetime HA calculated below is recommended. If the classification is B2, no lifetime HA is recommended.

Lifetime HA -- 1E-2 mg/L

Assumptions -- 20% exposure by drinking water

Principal Study -- Buben and O'Flaherty, 1985 (This study was used in the derivation of the chronic oral RfD; see RDO)

NOTE: A safety factor of 10 was used in the derivation of this HA, in addition to the UF of 1000 for the RfD, to account for the possible carcinogenicity of this substance.

OLEP -

Ordor perception threshold -- 300 ug/L.

ALAB -

Analysis of tetrachloroethylene is by a purge-and-trap gas chromatographic procedure used for the determination of volatile organohalides in drinking water.

TREAT-

Treatment techniques which will remove tetrachloroethylene from water include granular activated carbon adsorption, air stripping, and boiling.

HADR -
o HEALTH ADVISORY SOURCE :

U.S. EPA. 1985. Health Effects Criteria Document for Tetrachloroethylene. Office of Drinking Water, Washington, DC.
DOCUMENT

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o HEALTH ADVISORY REVIEW :

EPA review of HAs in 1986.

Public review of HAs is currently in progress (1987).

Science Advisory Board review to be determined.

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o EPA DRINKING WATER CONTACT :

Krishan Khanna / ODW -- (202)260-7588 / FTS 260-7588

Edward V. Ohanian / ODW -- (202)260-7571 / FTS 260-7571

OREF - Buben, J.A. and E.J. O'Flaherty. 1985. Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: A dose- effect study. Toxicol. Appl. Pharmacol. 78: 105-122.

OREF - Carpenter, C.P. 1937. The chronic toxicity of tetrachloroethylene. J. Ind. Hyg. Toxicol. 19(7): 323-336.

OREF - Hayes, J.R., L.W. Condie, Jr. and J.F. Borzelleca. 1986. The subchronic toxicity of tetrachloroethylene (perchloroethylene) administered in the drinking water of rats. Fund. Appl. Toxicol. 7: 119-125.

OREF - Kjellstrand, P., B. Holmquist, M. Kanje, et al. 1984. Perchloroethylene: Effects on body and organ weights and plasma butyrylcholinesterase activity in mice. Acta Pharmacol. Toxicol. 54(5): 414-424.

OREF - NTP (National Toxicology Program). 1985. NTP Technical Report on the Toxicology and Carcinogenesis Studies of Tetrachloroethylene (perchloroethylene). U.S. Dept. Health and Human Services, NIH Publ. No. 85- 2567.

OREF - Rowe, V.K., D.D. McCollister, H.C. Spencer, E.M. Adams and D.D. Irish. 1952. Vapor toxicity of tetrachloroethylene for laboratory animals and human subjects. Arch. Ind. Hyg. Occup. Med. 5: 566-579.

OREF - Schumann, A.M., J.F. Quast and P.G. Watanabe. 1980. The pharmacokinetics and macromolecular interaction of perchloroethylene in mice and rats as related to oncogenicity. Toxicol. Appl. Pharmacol. 55: 207-219.

OREF - Stewart, R.D., H.H. Gay, D.S. Erley, C.L. Hake and A.W. Schaffer. 1961. Human exposure to tetrachloroethylene vapor. Arch. Environ. Health. 2: 40-46.

OREF - U.S. EPA. 1985. Health Assessment Document for Tetrachloroethylene (perchloroethylene). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC for the Office of Air Quality Planning and Standards, Research Triangle Park, NC. EPA 600/8-82-005F. Office of Drinking Water, Washington, DC.

OREF - U.S. EPA. 1987. Quantification of Toxicological Effects for Tetrachloroethylene. Prepared from the Health Assessment Document for Tetrachloroethylene (perchloroethylene). Office of Drinking Water, Washington, DC.
IREF - None
CREF - None
HAREF- Buben, J.A. and E.J. O'Flaherty. 1985. Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: A dose- effect study. Toxicol. Appl. Pharmacol. 78: 105-122.
HAREF- U.S. EPA. 1985. Health Effects Criteria Document for Tetrachloroethylene. Office of Drinking Water, Washington, DC.

[IRIS] SS 17 /cf?
USER:
75-01-4
Search in progress

SS (17) PSTG (1)

[IRIS] SS 18 /cf?
USER:

108-88-3
Search in progress

SS (10) PSTG (1)

[IRIS] SS 11 /cf?
USER:
prt dl ncar, car continuous

1 - IRIS
IRSN - 115
DATE - 940406
UPDT - 04/06/94, 1 field
STAT - Oral RfD Assessment (RDO) on-line 04/01/94
STAT - Inhalation RfC Assessment (RDI) on-line 08/01/92
STAT - Carcinogenicity Assessment (CAR) on-line 02/01/94
STAT - Drinking Water Health Advisories (DWHA) on-line 09/01/90
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 04/01/92
IRH - 03/01/88 RDO Text revised
IRH - 09/07/88 CAR Carcinogen summary on-line
IRH - 02/01/89 CARDR Secondary contact's phone number corrected
IRH - 07/01/89 RDI Inhalation RfD now under review
IRH - 03/01/90 REFS Bibliography on-line
IRH - 04/01/90 CREF Combs et al., 1973 citation corrected
IRH - 06/01/90 CAA Area code for EPA contact corrected
IRH - 06/01/90 RCRA EPA contact changed
IRH - 07/01/90 RDO Withdrawn; new RfD verified (in preparation)
IRH - 07/01/90 OREF Oral RfD references withdrawn
IRH - 08/01/90 RDO Oral RfD summary replaced; RfD changed
IRH - 08/01/90 CAR Text edited
IRH - 08/01/90 OREF Oral RfD references revised
IRH - 09/01/90 HADV Health Advisory on-line
IRH - 09/01/90 HAREF Health Advisory references added
IRH - 08/01/91 CREF Litton Bionetics, Inc., 1981 reference title clarified
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 04/01/92 CAA CAA regulatory action withdrawn
IRH - 08/01/92 RDI Inhalation RfC on-line
IRH - 08/01/92 IREF Inhalation references on-line
IRH - 02/01/94 CARDR Secondary contact's phone number changed
IRH - 04/01/94 RDO Primary contact changed
RLEN - 73299
NAME - Toluene
RN - 108-88-3
SY - ANTISAL 1a
SY - BENZENE, METHYL
SY - METHACIDE
SY - METHYL-BENZENE
SY - METHYLBENZOL

SY - NCI-C07272
SY - PHENYL-METHANE
SY - RCRA WASTE NUMBER U220
SY - TOLUEEN
SY - TOLUEN
SY - Toluene
SY - TOLUOL
SY - TOLUOLO
SY - TOLU-SOL
SY - UN 1294

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Changes in liver and kidney weights 13-Week Rat Gavage Study NTP, 1989	NOAEL: 312 mg/kg converted to 223 mg/kg/day LOAEL: 625 mg/kg converted to 446 mg/kg/day	1000	1	2E-1 mg/kg/day

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*Conversion Factors: Dose adjusted for gavage schedule of 5 days/week.

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o ORAL RFD STUDIES :

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

The oral toxicity of toluene was investigated in this subchronic gavage study in F344 rats. Groups of 10 rats/sex/group were administered toluene in corn oil at dosage levels of 0, 312, 625, 1250, 2500, or 5000 mg/kg for 5 days/week for 13 weeks. All animals receiving 5000 mg/kg died within the first week. One female and 8 males in the 2500 mg/kg group died, but 2 of these were due to gavage errors. No deaths occurred at lower doses. Several toxic effects were noted at doses greater than or equal to 2500 mg/kg, including prostration, hypoactivity, ataxia, piloerection, lacrimation, excessive salivation, and body tremors. No signs of biologic significance were seen in groups receiving less than or equal to 1250 mg/kg. The only significant change in body weight was a decrease ($p<0.05$) for males in the 2500 mg/kg group. There were no toxicologically significant changes in hematology or urinalysis for any group of animals. Biochemical changes, including a significant increase ($p<0.05$) in SGOT in 2500 males and a dose-related increase in cholinesterase in females receiving 2500 and 5000 mg/kg, were not considered to be biologically significant. There were several pathologic findings and organ weight changes in the liver, kidney, brain, and urinary bladder. In males, absolute and relative weights of both the liver and kidney

were significantly increased ($p<0.05$) at doses greater than or equal to 625 mg/kg. In females, absolute and relative weights of the liver, kidney, and heart were all significantly increased at doses greater than or equal to 1250 mg/kg ($p<0.01$ for all comparisons except $p<0.05$ for absolute kidney and heart weights at 1250 mg/kg). Histopathologic lesions in the liver consisted of hepatocellular hypertrophy, occurring at greater than or equal to 2500 mg/kg. Nephrosis was observed in rats that died, and damage to the tubular epithelia of the kidney occurred in terminally sacrificed rats. Histopathologic changes were also noted in the brain and urinary bladder. In the brain, mineralized foci and necrosis of neuronal cells were observed in males and females at 2500 mg/kg and males at 1250 mg/kg. In the bladder, hemorrhage of the muscularis was seen in males and females at 5000 mg/kg and males at 2500 mg/kg. The NOAEL for this study is 312 mg/kg/day based on liver and kidney weight changes in male rats at 625 mg/kg. The toxicologic significance of these organ weight changes is strengthened by the occurrence of histopathologic changes in both the liver and kidney at higher doses. Because the exposure was for 5 days/week, this dose is converted to 312 x 5/7 = 223 mg/kg/day. The LOAEL is 625 mg/kg, which is 446 mg/kg/day when converted.

NTP (1989) also conducted a 13-week gavage study in B6C3F1 mice, following the same regimen described above. All mice receiving 5000 mg/kg died and 8/20 receiving 2500 mg/kg also died. Signs of toxicity seen in animals receiving greater than or equal to 2500 mg/kg included subconvulsive jerking, prostration, impaired grasping reflex, bradypnea, hypothermia, ataxia, and hypoactivity. By week 13, the mean body weight of 2500 mg/kg males was significantly ($p<0.05$) lower than controls. No other significant changes were reported for any group, including macroscopic observation, organ weight means, or clinical pathology parameters. The NOAEL for mice in this study was 1250 mg/kg.

The subchronic study by Wolf et al. (1956) is supportive of the NTP studies. Groups of 10 female Wistar rats were administered gavage doses of 0, 118, 354, or 590 mg/kg toluene dissolved in olive oil. A total of 138 doses were administered over 193 days, resulting in average doses of approximately 0, 84, 253, or 422 mg/kg/day. Hematologic, behavioral, gross and histopathologic examinations were conducted with no toxic effects being reported at any dose. Therefore, the highest dose of 422 mg/kg/day is considered to be the NOAEL for this study. However, this study is not used as the basis for the RfD because the LOAEL of 446 mg/kg/day identified by NTP (1989) is too close to the NOAEL identified by Wolf et al. (1956). Also, the NTP study indicated that male rats are more sensitive to toluene and the Wolf study utilized only female rats.

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o ORAL RFD UNCERTAINTY :

UF -- An uncertainty factor of 1000 was applied to account for inter- and intraspecies extrapolations, for subchronic-to-chronic extrapolation and for limited reproductive and developmental toxicity data.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

Kostas and Hotchin (1981) exposed NYLAR mice pre- and post-natally to toluene provided in the drinking water at concentrations of 0, 16, 80, or 400 ppm. Effects were noted in all dosed groups on rotorod performance, measured at 45 to 55 days of age, but there was an inverse dose-response relationship. No effects of toluene exposure were seen on maternal fluid consumption, offspring mortality rate, development of eye or ear openings, or surface-righting response. This study is not suitable for use in risk assessment because only 6 to 9 pregnancies/dose group were obtained, and because the dose-response relationship was inverse.

In an abstract providing limited information, Nawrot and Staples (1979) reported an increase in embryonic lethality in mice exposed to toluene from days 6 to 15 of gestation. Pregnant CD-1 dams were administered 0.3, 0.5, or 1.0 mL/kg bw, 3 times/day (equivalent to approximately 780, 1300, or 2600 mg/kg/day). Maternal toxicity was not observed at any dose level, but toluene was shown to be teratogenic at the high dose and embryolethal at the low dose. These levels are higher than the NOAEL demonstrated by the NTP (1989) study.

Several subchronic and chronic inhalation studies have been performed on toluene but are not considered to be suitable for deriving an oral RfD. These studies are summarized nicely in the introduction to the 2-year inhalation bioassay by NTP, 1989. The studies identify the following potential target organs: kidney (male rat); hematologic effects (mice); central nervous system (rats, mice, primates); developmental toxicity (rats, rabbits). It is beyond the scope of this oral RfD summary sheet to describe each of these studies, but the two chronic (2 year) inhalation studies are summarized briefly below.

In a 2-year inhalation study by NTP (1989), F344 rats (60/sex/group) were exposed to 0, 600, or 1200 ppm toluene and B6C3F1 mice (60/sex/group) to 0, 120, 600, or 1200 ppm toluene for 6.5 hours/day, 5 days/week. Ten animals/group (except male mice) were removed at 15 months for toxicologic evaluation. At 15 months, there was an increased incidence and severity of nonneoplastic lesions of the nasal cavity of exposed rats. Minimal hyperplasia of the bronchial epithelium was seen in 4/10 female mice at 1200 ppm. There were no significant differences in survival among any group of animals during the 2-year study. Mean body weights were generally similar for all groups throughout the study. Nephropathy was seen in almost all rats with the severity somewhat increased in exposed rats. There were also effects on the olfactory and respiratory epithelia of exposed rats. No biologically important lesions were seen in any groups of mice. There was no evidence of carcinogenicity for any group of animals in this study.

A chronic inhalation study in rats performed by CIIT (1980) failed to produce an adverse effect. Groups of 40 F344 rats/sex were exposed to 30, 100, or 300 ppm toluene for 6 hours/day, 5 days/week for 24 months. An unexposed group of

120 rats/sex served as a control. Clinical chemistry, hematology, and urinalysis testing were conducted at 18 and 24 months. All parameters measured at the termination of the study were normal except for a dose-related reduction in hematocrit values in females exposed to 100 and 300 ppm toluene. The highest dose of 300 ppm was considered to be a NOAEL.

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o ORAL RFD CONFIDENCE :

Study: High
Data Base: Medium
RfD: Medium

Confidence in the principal study is high because a sufficient number of animals/sex were tested in each of six dose groups (including vehicle controls) and many parameters were studied. The same protocol was tested in both mice and rats, with rats being identified as the more sensitive species. The data base is rated medium because it is supported by a 6-month oral study. It is not higher than medium because there is no reproductive study. Also, the oral studies are all subchronic, with the critical study being only 13 weeks in duration. Medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- None

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o REVIEW DATES : 05/20/85, 08/05/85, 08/05/86, 05/17/90, 06/20/90
o VERIFICATION DATE : 06/20/90
o EPA CONTACTS :

Harlal Choudhury / OHEA -- (513)569-7553

Krishan Khanna / OST -- (202)260-7588

RDI -
o INHALATION RFD SUMMARY :

Critical Effect	Exposures*	UF	MF	RfC
Neurological effects	NOAEL: None	300	1	4E-1
		mg/cu.m		
Occupational Study	LOAEL: 332 mg/cu.m (88 ppm)			
	LOAEL(ADJ): 119 mg/cu.m			

Foo et al., 1990	LOAEL(HEC): 119 mg/cu.m
Degeneration of nasal epithelium	NOAEL: None
	LOAEL: 2261 mg/cu.m (600 ppm)
2-Year Rat Chronic Inhalation Study	LOAEL(ADJ): 437 mg/cu.m
	LOAEL(HEC): 79 mg/cu.m
NTP, 1990	

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*Conversion Factors: MW = 92.15.

Foo et al., 1990: Assuming 25 C and 760 mmHg, LOAEL (mg/cu.m) = 88 ppm x 92.15/24.45 = 332 mg/cu.m. This is an extrarespiratory effect of a soluble vapor. The LOAEL is based on an 8-hour TWA occupational exposure. MVho = 10 cu.m/day, MVh = 20 cu.m/day. LOAEL(HEC) = LOAEL(ADJ) = 332 x MVho/MVh x 5 days/7 days = 119 mg/cu.m.

NTP, 1990: Assuming 25 C and 760 mmHg, LOAEL (mg/cu.m) = 600 ppm x 92.15/24.45 = 2261 mg/cu.m. LOAEL(ADJ) = LOAEL (mg/cu.m) x 6.5 hours/24 hours x 5 days/7 days = 437 mg/cu.m. The LOAEL(HEC) was calculated for a gas:respiratory effect in the extrathoracic region. MVa = 0.24 cu.m/day, MVh = 20 cu.m/day, Sa (ET) = 11.6 sq.cm, Sh (ET) = 177 sq.cm. RGDR = (MVa/Sa) / (MVh/Sh) = 0.18. LOAEL(HEC) = 437 x RGDR = 79 mg/cu.m.

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o INHALATION RFD STUDIES :

Foo, S., J. Jeyaratnam and D. Koh. 1990. Chronic neurobehavioral effects of toluene. Br. J. Ind. Med. 47(7): 480-484.

NTP (National Toxicology Program). 1990. Toxicology and carcinogenesis studies of toluene in F344/N rats and B6C3F1 mice (inhalation studies). NTP-TR-371. 253 p.

In humans, toluene is a known respiratory irritant with central nervous system (CNS) effects. Because available studies could not provide subthreshold (NOAEL) concentrations for either of these effects, the LOAELs for both effects need to be considered in developing the RfC. Consequently, the study of Foo et al. (1990) was used for the CNS effects, and that of the National Toxicology Program (NTP, 1990) for the irritant effects. Because the CNS effect was judged to be a more severe and relevant endpoint, the LOAEL for this effect was used for deriving the RfC. Further, this effect is supported by a number of other occupational studies that show effects around 100 ppm.

Foo et al. (1990) conducted a cross-sectional study involving 30 exposed female workers employed at an electronic assembly plant where toluene was emitted from glue. Toluene levels reported in the study were from personal sample monitoring and reported as an 8-hour TWA, although the number of samples taken and the actual sampling period were not given. No historical

exposure values were given. Co-exposure to other solvents was not addressed in the study. The exposed and control cohorts were matched for age, ethnicity, and use of medications. Members of these cohorts did not use alcohol and were nonsmokers. Medical histories were taken to eliminate any histories of central or peripheral nervous system disorders. The average number of years (+/- SD) worked by the exposed population was 5.7 +/- 3.2 and by the controls was 2.5 +/- 2.7. Exposed workers breathed toluene air levels of 88 ppm (332 mg/cu.m) as a TWA and control workers 13 ppm (49 mg/cu.m) (TWA); both of which are averages of the individual personal samples. A battery of eight neurobehavioral tests were administered to all exposed and control workers. The tests were performed midweek, before the workers reported to their stations for the day. Group means revealed statistically significant differences in 6/8 tests; all tests showed that the exposed workers performed poorly compared with the control cohort. When individual test results were linearly regressed against personal exposure concentrations, poor concentration-response relationships resulted for the six tests, with correlation coefficients ranging from 0.44 to 0.30. Irritation effects were not evaluated in this study, and no clinical signs or symptoms were reported. The paucity of exposure information, coupled with the small size of the cohort, limits the interpretation of this study, although the results were essentially confirmed in a clinical study in which the toluene concentrations were carefully controlled (Echeverria et al., 1989) at levels bracketing 88 ppm. Although the data in Echeverria et al. (1989) were generated from short-term exposures (3-7 hours over a period of 142 days), the results may be considered relevant to longer-term exposures as several studies indicate the absence of a duration-response relationship in toluene-induced symptomatology. Fornazzari et al. (1983) noted the absence of a duration-effect relationship among toluene abusers when they were segregated into neurologically impaired vs. unimpaired ($p = 0.65$). The human studies of Iregren (1982), Cherry et al. (1985), Baelum et al. (1985), and the principal study of Foo et al. (1990) all report this lack of a duration-response relationship and confirm the occurrence of CNS effects. Foo et al. (1990) indicate a LOAEL of 88 ppm toluene (332 mg/cu.m) for neurobehavioral changes from chronic exposure to toluene.

In a 2-year bioassay, Fischer 344 rats (60/sex/group) were exposed to 0, 600, or 1200 ppm (0, 2261, or 4523 mg/cu.m, respectively) toluene vapors, 6.5 hours/day, 5 days/week (duration-adjusted to 0, 437, and 875 mg/cu.m, respectively) for 103 weeks (NTP, 1990). To generate toluene vapor, the liquid material was heated, and the vapor diluted with nitrogen and mixed with the chamber ventilation air. An interim sacrifice was carried out at 15 months on control and 1200-ppm groups (10/sex/group) to conduct hematology and histopathology of the brain, liver, and kidney. Body weights were measured throughout the study. Gross necropsy and micropathology examinations were performed at the end of the study on all major organs including the nasal passage tissues (three sections), lungs, and mainstem bronchi. Mean body weights in both exposed groups were not different from controls for either sex. No exposure-related clinical signs were reported, and survival rate was similar for all groups. At the interim sacrifice, there was a mild-to-moderate degeneration in the olfactory and respiratory epithelium of the nasal

cavity in 39/40 rats of the 600- and 1200-ppm groups compared with 7/20 controls. At the end of 2 years, there was a significant ($p<0.05$) increase in the incidence of erosion of the olfactory epithelium (males: 0/50, 3/50, and 8/49; females: 2/49, 11/50, and 10/50; at 0, 600, and 1200 ppm, respectively) and of degeneration of the respiratory epithelium (males: 15/50, 37/50, and 31/49; females: 29/49, 45/50, and 39/50; at 0, 600, and 1200 ppm, respectively) in the exposed animals. The females exposed to 600 and 1200 ppm also exhibited a significant increase in inflammation of the nasal mucosa (27/49, 42/50, and 41/50 at 0, 600, and 1200 ppm, respectively) and respiratory metaplasia of the olfactory epithelium (0/49, 2/50, and 6/50 at 0, 600, and 1200 ppm, respectively). A LOAEL of 600 ppm toluene was determined for the concentration-dependent increase in erosion of the olfactory epithelium in male rats and the degeneration of the respiratory epithelium in both sexes. No NOAEL could be derived from this study.

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o INHALATION RFD UNCERTAINTY :

UF -- An uncertainty factor of 10 is used to account for intraspecies variability and another factor of 10 for the use of a LOAEL. An additional factor of 3 is applied for data base deficiencies, including the lack of data and well-characterized laboratory animal exposures evaluating neurotoxicity and respiratory irritation.

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o INHALATION RFD MODIFYING :

MF -- None
FACTOR

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o INHALATION RFD COMMENTS :

Toluene-induced neurotoxicity has been documented in humans over a broad spectrum of severity that correlates well with concentration. Numerous case studies on chronic toluene abusers [repeatedly exposed to greater than 30,000 ppm (113,000 mg/cu.m)] have demonstrated functional deficits of the CNS accompanied by abnormal morphology of cerebellar and cortical areas of the brain. Under acute exposure conditions [short exposures to greater than 10,000 ppm (37,690 mg/cu.m)], toluene produces CNS narcosis [American Conference of Governmental Industrial Hygienists (ACGIH), 1991]. Lower concentrations, i.e., 800-400 ppm (3015-1508 mg/cu.m), have been associated with worker complaints of CNS-related effects (ACGIH, 1991). Clinical studies using controlled exposure to toluene have demonstrated concentration-related occurrence of complaints such as drowsiness, ataxia, visual impairment, and headache. A number of occupational studies indicate that these same effects are present in exposed worker populations at concentrations lower than 400 ppm (1508 mg/cu.m) although deficiencies in most of these studies preclude confirming this finding unequivocally. Descriptions of a number of these studies follow. The preponderence of the literature showing CNS effects and the well-known proclivity for solvents to affect CNS processes in humans leave little doubt that the brain is a principal target organ for toluene toxicity in humans.

In cases of inhalation abuse of toluene, Rosenberg et al. (1988) demonstrated diffuse cerebral, cerebellar, and brainstem atrophy in 3 of 11 toluene abusers who also had neurological abnormalities. Filley et al. (1990) were able to correlate neuropsychological impairment with the degree of white matter abnormality ($p<0.01$). Cerebellar and cortical functions were classified as impaired in 15/24 individuals who had abused toluene daily (425 +/- 366 mg/day) for extended periods (6.3 +/- 3.9 years) (Fornazzari et al., 1983). In a limited case study, Metrick and Brenner (1982) demonstrated brainstem atrophy through computerized tomographic scans and abnormal brainstem auditory-evoked potentials in 2/2 chronic toluene abusers (12-16 years of admitted, continuous abuse). These studies confirm the occurrence of severe CNS damage in response to highly abusive concentrations of toluene.

Several studies that have investigated the occurrence of neurotoxicity at lesser concentrations, such as occupational situations, have not demonstrated significant neurological or other effects. Hanninen et al. (1987) performed a battery of 11 psychological tests on 43 printing workers who had been occupationally exposed to approximately 117 ppm (441 mg/cu.m) toluene for an average of 22 years and found only mildly adverse effects in 2/11 tests. The control and exposed cohorts in this study were, however, mismatched in several areas, most notably alcohol use. Iregren (1982) examined the psychological performance of 38 printers who had been occupationally exposed to 50-150 ppm (188-565 mg/cu.m) toluene for an average of 16.3 years (range 3-32 years). No effects were seen, although the cohorts in this study were apparently matched only by age. In a cohort study, Cherry et al. (1985) attempted to better match the control and exposed cohorts and considered alcohol use. Although no differences between the cohorts were statistically significant, the exposed workers performed worse than the nonexposed workers on 10/13 psychological tests. The 52 workers in this study were not, however, rigorously matched, and the concentrations listed in the study ranged up to greater than 500 ppm (1884 mg/cu.m). The cohorts in the study of Foo et al. (1990) were well matched for a number of confounders, including alcohol use, and statistically significant psychological effects were seen.

In the occupational study conducted by Yin et al. (1987), 94 solvent workers (38 men and 56 women; average employment duration, 6.8 years) and 138 controls (48 men and 90 women) were examined for exposure using diffusion dosimeters, subjective symptoms by questionnaire, hematology, and urinalysis. Exposure concentration (7-hour mean TWA) in the workers was estimated at 42.8 ppm (161 mg/cu.m) toluene with a maximum measurement of 123 ppm (464 mg/cu.m). Workers were co-exposed to 1.3 ppm benzene. No exposure-related effects were noted in any of the biochemical tests examined. In considering the prevalence of subjective symptoms (sore throat, headaches, and dizziness) workers were subgrouped into low (6-39 ppm, $n = 28$) and high (40-123 ppm, $n = 29$) categories. Although the prevalence of subjective symptoms was significantly higher in the exposed workers compared with the control cohort ($p<0.01$), a concentration-response relationship was not discernable among the groups. No other treatment-related effects were reported. The study was limited because the exposed and unexposed groups were not matched to control for confounding

effects (e.g., age, smoking, alcohol consumption, exposure duration). Based on these results, exposure to an average of approximately 42.8 ppm toluene produced no biochemical abnormalities, although neither respiratory irritation nor psychological performance was directly evaluated in these workers.

In the occupational study by Lee et al. (1988), prevalence of subjective symptoms was categorized with respect to exposure levels. The study population (193 women and 65 controls) completed a questionnaire. The exposures were reported as 8-hour TWAs, and workers were grouped in exposure categories of nonexposed, 1-50 ppm, 51-100 ppm, 101-150 ppm, and more than 151 ppm (duration of exposures was not reported). A concentration-dependent increase in prevalence was reported for 25/67 symptoms with increases in complaints over controls occurring at around 100 ppm (348 mg/cu.m). Similar to the Yin et al. study (1987) reported above, symptomatology included headaches, sore throats, and dizziness. Although an effect level in humans of around 100 ppm is indicated by this study, no objective measures of toxicity were examined.

A number of acute human studies have focused on toluene effects. In general, these studies corroborate subjective CNS effects such as headaches and dizziness reported in other longer-term occupational studies (Yin et al., 1987; Lee et al., 1988) and also document irritation effects. The study of Echeverria et al. (1989) correlates the occurrence of these subjective effects with substantial neurological symptoms.

Forty-two college students (21 female and 21 male) were exposed to 0, 74 ppm (279 mg/cu.m), or 151 ppm (569 mg/cu.m) toluene for 7 hours over 3 days (Echeverria et al., 1989). This exposure sequence was repeated for a total of 42 exposures over a 3-month period. The odor of toluene was masked. A battery of performance tests was administered to each participant prior to starting the exposures and again at 4 and 7 hours during the exposure; the initial test served as a control for those tests performed during the exposure. A 5-10% decrement in performance was considered significant if consistent with a linear trend. Test results for visual perception differed from control values for both exposure levels. Results of a manual dexterity test differed from control values at the higher but not the lower exposure level. Psychomotor test results were unaffected by toluene exposure. Subjective symptomatology increased with exposure with increasing numbers of complaints of eye irritation, headache, and somnolence. A NOAEL of 74 ppm (279 mg/cu.m) is indicated for these results. The duration-adjusted value is 122 mg/cu.m for these acute effects.

Andersen et al. (1983) exposed 16 subjects (average age of 24 years) to 0, 10, 40, or 100 ppm (0, 38, 151, or 377 mg/cu.m) toluene for 6 hours on each of 4 consecutive days. Individuals were tested for nasal mucous flow, lung function, subjective response, and psychometric performance. At 100 ppm, irritation was experienced in the eyes and nose, but no effect on nasal mucous flow or lung function was observed. The subjects frequently reported headaches, dizziness, and a feeling of intoxication. These effects were not reported by the 10- or 40-ppm exposure groups. No effects were seen in

performance tests. This study indicates an effect level of 100 ppm, and a NOAEL of 40 ppm (151 mg/cu.m).

The acute study by Baelum et al. (1990) evaluated 32 males and 39 females exposed to 0 or 100 ppm (0 or 377 mg/cu.m), or to varying exposures of 50-300 ppm (188-1131 mg/cu.m) (TWA = 102 ppm), for 7 hours. Volunteers exercised on an ergometer cycle for 3 periods of 15 minutes each during the exposure. No significant differences were found in the performances between the exposed and control groups in a battery of tests for performance, visual attention, and reaction times. Exposed subjects reported an increase over nonexposed subjects ($p<0.1$) in nose and lower respiratory irritation, feelings of intoxication, dizziness, increased coughing, and headaches. Differences were not noted between the group exposed to a constant level (100 ppm) and the group exposed to the same TWA, but with peaks of up to 300 ppm.

Baelum et al. (1985) investigated the effects of a 6.5-hour toluene exposure to 43 printers with a long-term occupational exposure to a mixture of solvents including toluene and 43 controls with no history of exposure to solvents or other chemicals. The duration of employment for the workers ranged from 9-25 years. Each individual was exposed only once to either 0 or 100 ppm (0 or 377 mg/cu.m) toluene during a 6.5-hour exposure period, preceded by a 1-hour acclimatization period. These subjects were then subgrouped into printers exposed to toluene (n = 20), printers exposed to air (n = 23), controls exposed to toluene (n = 21), and controls exposed to air (n = 22). All subjects carried out a battery of tests for psychometric performance, visual perception, and vigilance evaluation. Both printers and controls complained of nasal and eye irritation, unacceptable air quality, and unacceptable odor level during the toluene exposure. Signs of neurotoxicity, including moderate fatigue, sleepiness, headaches, and a feeling of intoxication, were likewise similarly reported for both groups. A significant decrease in performance was found for the pegboard visual motor function test in the exposed printers, but not in the controls exposed to 100 ppm toluene. A decrease in psychometric performance, primarily in visual perception and accuracy, was observed in toluene-exposed individuals. Acute exposure to toluene resulted in a lower performance in 4/10 tests conducted, 3 of these 4 evaluated visual perception. The most profound difference between subjects exposed to 100 ppm toluene and those exposed to clean air was observed in the color discrimination test; this difference was seen in both exposed vs. nonexposed printers and exposed vs. nonexposed controls. This study indicates that little tolerance develops to the irritative and central effects in humans exposed to toluene and that 100 ppm (377 mg/cu.m) is the effect level for these symptoms.

Von Oettingen et al. (1942) exposed 3 humans to 100 or 200 ppm (377 or 754 mg/cu.m) toluene vapors for 8 hours. At 200 ppm, the subjects experienced muscular weakness, confusion, impaired coordination, and dilated pupils, with after-effects including fatigue, general confusion, and moderate insomnia. In 1 subject exposed to 100 ppm toluene, moderate fatigue, sleepiness, and headaches were reported.

Hepatotoxicity has also been examined as a toxicologic endpoint of toluene exposure in humans. Fornazzari et al. (1983) described moderate elevation of serum AP levels in 13/24 (and SGOT in 7/24) toluene abusers upon admission to a clinic. These elevated levels were normal after 2 weeks of solvent abstinence, although the accompanying CNS effects were only minimally improved. In a cross-sectional study of 181 printing workers in which toluene exposures were less than 200 mg/cu.m, no adverse effects were apparent as judged from serum liver enzymes (Boewer et al., 1988). In another cross-sectional occupational study conducted by Guzelian et al. (1988) that involved 289 printing factory employees, 8 workers were found who had an increase described as "marked" in the ratio of ALT/AST enzyme serum activity. Biopsies revealed mild pericentral fatty livers in each of the eight cases. Based on environmental data (probably area monitors) the levels of toluene to which these workers were exposed was less than 200 mg/cu.m., 2-8 hours/day.

Fischer 344 rats (120/sex/group) inhaled 0, 30, 100, or 300 ppm (0, 113, 377, or 1130 mg/cu.m, respectively) toluene (99.9% purity), 6 hours/day, 5 days/week (duration-adjusted to 0, 20, 67, or 202 mg/cu.m, respectively) for 106 weeks (CIIT, 1980; Gibson and Hardisty, 1983). Vapor, generated by bubbling clean air through toluene, was passed through the air supply duct and mixed with air by turbulent flow to produce the desired concentration. Hematology, blood chemistry, and urinalysis were conducted in all groups at 6 (5/sex), 17 (5/sex), 18 (10-20/sex), and 24 months (10/sex). Histopathology was evaluated only in the control and 300-ppm groups at 6 (5/sex), 12 (5/sex), and 18 months (20/sex). At 24 months, histopathological examinations were conducted in organs of all surviving animals, including the respiratory system and sections through the nasal turbinates (number not indicated). No treatment-related non-neoplastic effects were observed in the exposed animals. Although the male rats exposed to 300 ppm had a significant increase in body weight compared to controls, no concentration-response was evident. At the end of the exposure period, the female rats exposed to 100 or 300 ppm exhibited a slight but significant reduction in hematocrit; an increase in the mean corpuscular hemoglobin concentration was also noted but only in the females exposed to 300 ppm. The highest concentration examined in this study, 300 ppm, is designated as a NOAEL for toxicity remote from the respiratory tract in rats. CIIT (1980) reported that the technical and raw data were not audited by their quality assurance group during the study period, although CIIT did conduct a quality assessment procedure to review the data. The available pathology reports containing these data indicate that at least the lower respiratory tract was examined. Communication with the testing sponsor has provided information indicating that only one section was examined from the nasal cavity of these test animals. It is not clear whether this single section would have been sufficient to elucidate the areas of lesions noted in the NTP (1990) study. Consequently, the designation of the 300-ppm exposure level as a NOAEL for respiratory lesions (see NTP,1990) is problematic.

Fischer 344/N rats (10/sex/group) were exposed to toluene vapors at 0, 100, 625, 1250, 2500, and 3000 ppm (0, 377, 2355, 4711, 9422, and 11,307 mg/cu.m, respectively) 6.5 hours/day, 5 days/week (duration-adjusted to 0, 73, 455, 911, 1823, and 2187 mg/cu.m, respectively) for 15 weeks (NTP, 1990).

Organ weights were measured and histological examinations were performed only on controls, 2500- and 3000-ppm groups, and animals that died before the end of the study. Eight of 10 males exposed to 3000 ppm died, all during the 2nd exposure week. No females died at any exposure level. Compared to the controls, final body weights were 15 and 25% lower in the males and 15 and 14% lower in the females of the 2500- and 3000-ppm groups, respectively. There was a concentration-related increase in the relative liver weight, significant at 1250, 2500, and 3000 ppm in males and at 2500 and 3000 ppm in females. The relative weights of the heart, lung, kidney, and right testis were also significantly elevated in the 2500- and 3000-ppm animals compared to those of the controls, although no histopathology was observed in any exposure group. Toxic effects noted in a concurrently conducted gavage study (urinary bladder hemorrhages in the two highest exposure groups) were not noted in this subchronic inhalation study. A LOAEL of 2500 ppm [LOAEL(HEC) = 1823 mg/cu.m] was determined for the decrease in body weight gain in both males and females, and the NOAEL for this effect was 1250 ppm [NOAEL(HEC) = 911 mg/cu.m].

Toluene has been suspected to cause congenital defects in infants born to mothers who were exposed to or who abused toluene during pregnancy. In a case report study, Hersh et al. (1985) describes clinical and morphometric characteristics common to 3 children whose mothers had abused toluene (but apparently not alcohol or any other substance) for a period of 4-5 years including during their pregnancies with the affected children. Clinical findings common to these three children included microcephaly, CNS dysfunction, attention deficits, and developmental delay/mental deficiency. Phenotypic similarities included a small midface, deep-set eyes, micrognathia (smallness of the jaws), and blunting of the fingertips. A retrospective cohort study was conducted by McDonald et al. (1987) who examined the history of exposure to chemicals of 301 women who had recently given birth to an infant with an important congenital defect. An identical number of women (referents) who had given birth to normal children were matched with respect to age, employment (hours/week), date of delivery, and educational level. In initial matched-pair analysis, chemical exposure was higher in the cases than in the referents (63 cases:47 referents) due to excess cardiac and miscellaneous defects. In further analysis by chemical categories, only exposure to aromatic solvents showed a clear excess of defects, mostly in the urinary tract. Details of these cases (n = 19) showed that toluene was identified as the solvent in 11 of these cases.

Hudak and Ungvary (1978) exposed three groups of pregnant CFY rats to toluene during different periods of gestation and for different durations of exposure. Two of the groups had their own control group exposed to air only and matched for period and daily duration. The first of these (n = 19) was exposed to 1500 mg/cu.m for 24 hours/day during gestational days 9 to 14. Two dams died during these exposures. No details on the deaths are given but no other maternal toxicity was observed. Fetotoxicity was also in evidence as sternebral alterations (6% vs. 1% in controls), extra ribs (22% vs. 0% in controls), and the presence of fetuses with missing tails (2/213, none observed in 315 controls) were recorded. Under these exposure conditions, 1500 mg/cu.m is a LOAEL for fetotoxicity and a frank effect level (FEL) for maternal

toxicity. The second group (n = 14) received this same concentration continuously but on days 1-8 of gestation. Five dams died under these exposure conditions although toxicity parameters of the surviving dams were identical with the controls from the first group (gestational days 9-14). Slight hydrocephaly was noted in 4 fetuses (all from the same litter), and 17% growth retardation was noted vs. 7% in the controls. Thus these exposure conditions are a FEL for maternal toxicity and a LOAEL for fetotoxicity. A third group was exposed to 1000 mg/cu.m for 8 hours/day from the 1st to the 21st day of gestation. No maternal deaths or toxicity occurred. Minor skeletal retardation was present in the exposed fetuses at a higher incidence rate (25%) than in concurrent controls (0%). These results indicate that 1000 mg/cu.m is a LOAEL for developmental effects under these exposure conditions. This concentration is also a NOAEL for maternal effects. These workers also exposed groups of pregnant CFLP mice (n = 11-15) to either air or 1500 or 500 mg/cu.m toluene continuously during days 6-13 of pregnancy. All mice exposed to the high concentration died within 24 hours of the beginning of exposure. No dams died in the lower exposure group. In this group, the average fetal weight decreased to 0.96 g from the average control weight of 1.07 g, and the percentage of weight-retarded fetuses (less than 0.9 g) increased to 27.6% from 6.5% in the controls. No difference in incidence of skeletal malformations or anomalies was noted between these and control fetuses. For mice, 1500 mg/cu.m is an FEL and 500 mg/cu.m is a mild LOAEL. Since duration adjustment is not performed for developmental effects, this concentration is also the LOAEL(HEC).

B6C3F1 mice (60/sex/group) were exposed to 0, 120, 600, or 1200 ppm (0, 452, 2261, or 4523 mg/cu.m, respectively) toluene 6.5 hours/day, 5 days/week (duration-adjusted to 0, 87, 47, and 875 mg/cu.m, respectively) for 2 years (NTP, 1990). Mean body weights were not significantly different among groups and no treatment-related clinical signs were observed. Deaths (moribund and natural) occurred in all exposure groups but were not related to exposure and were not greater than the control rates. An excess incidence of non-neoplastic inflammatory lesions of the urinary and genital system was observed in all the groups of male mice. At the 15-month interim sacrifice, minimal hyperplasia in the bronchial epithelium was observed in 4/10 females exposed to 1200 ppm. At the end of the study, there was a concentration-dependent increase in the incidence of splenic pigmentation in the exposed males (9/60, 11/60, and 18/59 at 120, 600, and 1200 ppm, respectively) compared to controls (4/60). In the females, the incidence was 37/50, 33/50, 34/49, and 28/47 at 0, 120, 600, and 1200 ppm, respectively. The occurrence of endometrial hyperplasia was present in 14% of the animals exposed to the highest concentration but only in 4% in the low-exposure groups and controls. No differences were noted between the exposed and control mice of either sex in the incidence of degeneration of either the olfactory or respiratory epithelium. No other non-neoplastic lesions were observed in exposed mice. As no adverse effects were noted in this study, the highest concentration, 1200 ppm was designated as a NOAEL in mice for this chronic study [NOAEL(HEC) = 875 mg/cu.m].

Sprague-Dawley rats (15/sex/group) were exposed to cumulative mean exposures of 0, 100, or 1481 ppm (0, 377, or 5653 mg/cu.m) toluene vapors, 6

hours/day, 5 days/week (duration-adjusted to 0, 67, and 1009 mg/cu.m, respectively) for 26 weeks (API, 1981). On weeks 9, 18, and 27, neurohistopathological examinations were conducted in 3-5 rats/sex/group. Hematology, clinical chemistry, and urinalysis parameters were evaluated after 13 and 26 weeks of exposure. Body weights were measured weekly. No significant treatment-related effects were reported. Therefore, a NOAEL of 1481 ppm [NOAEL(HEC) = 1009 mg/cu.m] toluene was determined for systemic effects in rats. The study was limited because there were no other neurohistopathological examinations or organ weight measurements conducted on the animals.

Inhalation exposure to toluene has been shown to result in irreversible high-frequency hearing loss in rats. Pryor et al. (1984) exposed young male Fischer 344 rats to a variety of exposure concentrations and durations. Hearing loss was evaluated by a behavioral technique (avoidance response elicited to an auditory signal) or brainstem auditory-evoked responses (elicited by tone pips of differing loudness and frequency and detected by subdural scalp electrodes). Hearing loss, as measured by both techniques, was observed after as few as 2 weeks exposure to 1000 ppm toluene for 14 hours/day. Lower concentrations of 700 ppm for 14 hours/day were without effect after 16 weeks of exposure. Intermittent exposure to 3000 ppm for 30 minutes/hour for 8 hours/day caused hearing loss within 2 weeks, whereas a similar exposure schedule for only 4 hours/day was without effect after 9 weeks. These data define a NOAEL for hearing loss in rats of 700 ppm [NOAEL(HEC) = 2638 mg/cu.m]. The duration-adjusted HEC (assumed 5 days/week) would be 14/24 hours x 5/7 days = 1100 mg/cu.m. Although these results clearly document hearing loss in young adult rats, their direct significance to humans remains unclear. Among chronic toluene abusers there is only a single report of adverse effects on hearing; Metrick and Brenner (1982) claimed that the abnormal auditory-evoked potentials recorded in two chronic toluene abusers was evidence of brainstem abnormalities.

Pregnant Wistar rats and hamsters (group size not indicated) inhaled 0 or 800 mg/cu.m toluene vapors 6 hours/day on gestational days 14-20 (rats) or gestational days 6 to 11 (hamsters) (DaSilva et al., 1990). In the exposed rats, there was a significant ($p<0.05$) increase in the number of litters with one or more low birth weight pups (less than 4.9 g), from 10% in the controls to 54% in the exposed dams. A decrease ($p<0.05$) in the number of live pups at birth was also noted in the litters of exposed dams. No evaluation of malformations or anomalies was performed. The neurobehavioral development of the offspring of the exposed rats was assessed using tests of spontaneous alternation, rim escape, and avoidance responses. The only effect noted in the rats, a shortened first trial latency in choosing one side of a maze, was minimal and its significance unclear. No comparable reproductive deficits occurred in the exposed hamsters. The only effect noted in the neurobehavioral tests of the hamster offspring was an equivocal effect in rota-rod performance. No neurobehavioral effect levels were designated from this study, although it appears that the rat developmental processes are more sensitive than those of the hamster, exhibiting adverse effects at 800 mg/cu.m.

Ungvary and Tatrai (1985) exposed New Zealand rabbits (8-10/group) to 0, 500, or 1000 mg/cu.m toluene, 24 hours/day, on gestational days 7-20, and CFLP mice (15 females/group) to 0, 500, 1000, or 1500 mg/cu.m toluene, also continuously, on gestational days 6-15. The control groups consisted of 115 mice and 60 rabbits. All the female mice exposed to 1500 mg/cu.m died. In the mice exposed to 1000 mg/cu.m, there was an increase in fetuses with retarded weight (29%, level of retardation not indicated) and in fetuses with skeletal retardation (12%) compared to 7% and 5%, respectively, in the controls, which did not differ from the animals exposed to 500 mg/cu.m. Of the 8 pregnant rabbits exposed to 1000 mg/cu.m, 2 died, 4 had spontaneous abortions, and the remaining 2 had total litter resorption. No deaths occurred in the 10 rabbits exposed to 500 mg/cu.m but 1/10 rabbits had a spontaneous abortion (as compared to 0/60 reported for the controls). A NOAEL(HEC) of 500 mg/cu.m toluene was determined for reproductive effects in mice. For rabbits, the 500 mg/cu.m concentration is designated as a LOAEL. These results indicate that pregnant mice may be a sensitive population to the effects of toluene.

Pregnant Charles River CD-1 mice (15-16 females/group) inhaled filtered air or 200 or 400 ppm (754 and 1508 mg/cu.m) toluene 7 hours/day on gestational days 7-16 (Courtney et al., 1986). The relative liver weight in the exposed dams was reported to be significantly lower in the two exposed groups compared to the controls, although no data were presented. A statistically significant increase in lactate dehydrogenase activity in the brain of the dams exposed to 400 ppm was also reported. The exposed pregnant mice did not exhibit any significant differences in the number of implantation sites, number of live fetuses, fetal deaths, or fetal body weight compared to the control values. A statistically significant increase over controls in the incidence (both per litter and per fetus) of enlarged renal pelves was noted in dams exposed to 200 ppm but not 400 ppm. A statistically significant alteration from controls in the rib profile (percentage of fetuses with 1 or 2 additional/fewer ribs) was reported for fetuses from dams exposed to 400 ppm but not 200 ppm. The toxicological significance of this finding is not clear. As no clearly significant toxicological effects were observed, the highest concentration used, 400 ppm [NOAEL(HEC) = 1508 mg/cu.m] is designated as a NOAEL for reproductive and developmental effects in mice.

A 2-generation inhalation reproductive study was conducted in CD rats (10-40 males, 20-80 females/group) (API, 1985). Animals were exposed by whole-body inhalation to toluene at 0, 100, 500, or 2000 ppm (0, 377, 1885, or 7538 mg/cu.m, respectively) 6 hours/day, 7 days/week for 80 days and a 15-day mating period. The mated females were then exposed to the same concentrations during days 1-20 of gestation and days 5-20 of lactation. After weaning, the pups in this generation (F1) were exposed 80 times and then randomly mated with members of the same exposure group (2 females/1 male) to produce the second generation (F2). Mean male body weights were slightly reduced (maximum of 10%) in the first 2 weeks of the exposure in the animals exposed to 500 and 2000 ppm, although the size of the reduction was not related to exposure. No differences were observed in male or female fertility indices, length of gestation, mean numbers of viable and nonviable pups at birth, or pup survival

indices during lactation. No abnormal histopathology was noted in organs examined. A significant decrease ($p<0.05$) in weight relative to controls was observed in the first generation offspring. The decrease was maintained throughout the lactation period in the pups from dams exposed to the highest exposure and in those from the ancillary group in which females exposed to the 2000 ppm concentration were mated with males having no exposure. No data were available in the report about the F2 generation. Based on the effects on the pups of the first generation (F1), a LOAEL of 2000 ppm [LOAEL(HEC) = 7538 mg/cu.m] is designated, the NOAEL being 500 ppm [NOAEL(HEC) = 1885 mg/cu.m].

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o INHALATION RFD CONFIDENCE : Study -- Medium Data Base -- Medium RfC -- Medium The study of Foo et al. (1990) indicates adverse neurological effects of toluene in a small worker population. These effects are consistent with more severe CNS effects occurring at abusive concentrations of toluene and could not have been confounded by alcohol as the control and exposed populations did not use alcohol. However, the paucity of exposure information and identification of only a LOAEL is not sufficient to warrant a higher confidence than medium for this study. Other studies indicate that irritation may occur at around the same concentration, 100 ppm (Baelum et al., 1985; Echeverria et al., 1989). In regard to this effect, the NTP (1990) rat chronic inhalation study was well conducted, established the rat as the most sensitive species, examined an adequate number of animals, and performed histopathology on all major organs, including the brain and the respiratory tract. The sensitive endpoint was the concentration-dependent degeneration of the nasal epithelium characterized by the erosion of the olfactory epithelium and degeneration of the respiratory epithelium in male rats. The NTP study is also given medium confidence, however, as it did not establish a NOAEL. Although this data base has a complement of chronic laboratory animal studies, long-term data in humans are not available for either the neurotoxicity or irritation endpoints. The reproductive/developmental studies in three species were not comprehensive in endpoint evaluation but

do identify the rabbit as the most sensitive species. The data base is thus given a medium confidence rating. A medium confidence rating for the RfC follows.

o INHALATION RFD SOURCE :

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- U.S. EPA, 1984, 1985
DOCUMENT

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o REVIEW DATES : 04/21/88, 05/26/88, 02/16/89, 03/21/89, 05/18/89, 08/15/91, 12/11/91
o VERIFICATION DATE : 05/18/89, 12/11/91
o EPA CONTACTS :

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CAREV-
o CLASSIFICATION : D; not classified
o BASIS FOR CLASSIFICATION : No human data and inadequate animal data. Toluene did not produce positive results in the majority of genotoxic assays.
o HUMAN CARCINOGENICITY DATA :

None.

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o ANIMAL CARCINOGENICITY DATA :

A chronic (106-week) bioassay of toluene in F344 rats of both sexes reported no carcinogenic responses (CIIT, 1980). A total of 960 rats were exposed by inhalation for 6 hours/day, 5 days/week to toluene at 0, 30, 100, or 300 ppm. Groups of 20/sex/dose were sacrificed at 18 months. Gross and microscopic examination of tissues and organs identified no increase in neoplastic tissue or tumor masses among treated rats when compared with controls. The study is considered inadequate because the highest dose administered was well below the MTD for toluene and because of the high incidence of lesions and pathological changes in the control animals.

Several studies have examined the carcinogenicity of toluene following repeated dermal applications. Toluene (dose not reported) applied to shaved interscapular skin of 54 male mice (strains A/He, C3HeB, SWR) throughout their lifetime (3 times weekly) produced no carcinogen1c response (Poel, 1963). One drop of toluene (about 6 mL) applied to the dorsal skin of 20 random-bred albino mice twice weekly for 50 weeks caused no skin papillomas or carcinomas after a 1-year latency period was allowed (Coombs et al., 1973). No increase

in the incidence of skin or systemic tumors was demonstrated in male or female mice of three strains (CF, C3H, or CBaH) when toluene was applied to the back of 25 mice of each sex of each strain at 0.05-0.1 mL/mouse, twice weekly for 56 weeks (Doak et al., 1976). One skin papilloma and a single skin carcinoma were reported among a group of 30 mice treated dermally with one drop of 0.2% (w/v) solution toluene twice weekly, administered from droppers delivering 16-20 uL per drop for 72 weeks (Lijinsky and Garcia, 1972). It is not reported whether evaporation of toluene from the skin was prevented during these studies.

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o SUPPORTING DATA :

Toluene was found to be nonmutagenic in reverse mutation assays with S. typhimurium (Mortelmans and Riccio, 1980; Nestmann et al., 1980; Bos et al., 1981; Litton Bionetics, Inc., 1981; Snow et al., 1981) and E. coli (Mortelmans and Riccio, 1980), with and without metabolic activation. Toluene did not induce mitotic gene conversion (Litton Bionetics, Inc., 1981; Mortelmans and Riccio, 1980) or mitotic crossing over (Mortelmans and Riccio, 1980) in S. cerevisiae. Although Litton Bionetics, Inc. (1981) reported that toluene did not cause increased chromosomal aberrations in bone marrow cells, several Russian studies (Dobrokhotov, 1972; Lyapkalo, 1973) report toluene as effective in causing chromosal damage in bone marrow cells of rats. There was no evidence of chromosomal aberrations in blood lymphocytes of workers exposed to toluene only (Maki-Paakkanen et al., 1980; Forni et al., 1971), although a slight increase was noted in workers exposed to toluene and benzene (Forni et al., 1971; Funes-Craviota et al., 1977). This finding is supported by studies of cultured human lymphocytes exposed to toluene in vitro; no elevation of chromosomal aberrations or sister chromatid exchanges was observed (Gerner-Smidt and Friedrich, 1978).

CARDR-
o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1987

The values in the 1987 Drinking Water Criteria Document for Toluene have received peer and administrative review.
DOCUMENT

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o REVIEW DATES : 09/15/87
o VERIFICATION DATE : 09/15/87
o EPA CONTACTS :

Dharm V. Singh / OHEA -- (202)260-5958

Robert E. McGaughy / OHEA -- (202)260-5889

HAONE-

One-day HA -- 2E+1 mg/L

NOAEL -- 21.5 mg/kg/day
UF -- 10 (allows for intrahuman variability with the use of a NOAEL from a human study)
Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Gamberale and Hultengren, 1972

This study reported that a 20-minute exposure to 100 ppm toluene was a no-effect level when determined by perceptual speed and reaction time tests in human volunteers. At 200 ppm, toluene was noted as clearly causing toxic effects such as incoordination, exhilaration, and prolonged reaction time. These and other data support the selection of 100 ppm (377 mg/cu.m) toluene as the NOAEL in humans exposed for up to 8 hours. Based on the conditions of exposure and an assumed absorption rate of 60%, this level is equivalent to 21.5 mg/kg/day.

HATEN-

No information was found in the available literature that was suitable for determination of a Ten-day HA value. It is, therefore, recommended that the DWEL, adjusted for a 10-kg child (3 mg/L) be used as the Ten-day HA value.

HALTC-

No information was found in the available literature that was suitable for determination of a Longer-term HA value. It is, therefore, recommended that the DWEL, adjusted for a 10-kg child (3 mg/L) be used as the Longer-term HA value for a child.

HALTA-

No information was found in the available literature that was suitable for determination of a Longer-term HA value. It is, therefore, recommended that the DWEL, adjusted for a 70-kg adult (10 mg/L) be used as the Longer-term HA value for an adult.

HALIF-

Drinking Water Equivalent Level (DWEL) -- 7E-0 mg/L

Assumptions -- 2 L/day water consumption for a 70-kg adult

RfD Verification Date -- 06/20/90

Lifetime HA -- 1E-0 mg/L

Assumptions -- 20% exposure by drinking water

Principal Study -- NTP, 1989 (This study was used in the derivation of the chronic oral RfD; see RDO)

OLEP -

Taste threshold in water is reported as 0.04 and 1 mg/L. Odor threshold in water is reported as 0.04 and 1 mg/L.

ALAB -

Analysis of toluene is by a purge-and-trap gas chromatographic procedure used for the determination of volatile aromatic and unsaturated organic compounds in water.

TREAT-

Treatment options for removing toluene form drinking water sources include air stripping and adsorption onto granular activated carbon.

HADR -

o HEALTH ADVISORY SOURCE :

U.S. EPA. 1990. Final Draft of the Drinking Water Criteria Document for Toluene. Office of Drinking Water, Washington, DC.
DOCUMENT

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o HEALTH ADVISORY REVIEW :

EPA review of HAs in 1986.

Public review of HAs in 1987.

Science Advisory Board review to be determined.

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o EPA DRINKING WATER CONTACT :

Krishan Khanna / OST -- (202)260-9568

Edward V. Ohanian / OST -- (202)260-7571

WQCHU-

Water and Fish Consumption: 1.43E+4 ug/L

Fish Consumption Only: 4.24E+5 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of 1.43E+4 ug/L is based on consumption of contaminated aquatic organisms and water. A WQC of 4.24E+5 ug/L has also been established based on consumption of contaminated aquatic organisms alone.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC -- 1.75E+4 ug/L
Chronic LEC -- none

Marine:

Acute LEC -- 6.3E+3 ug/L
Chronic LEC -- 5.0E+3 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria,but are the lowest effect levels found in the literature. LEC's are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 1 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- EPA has set a MCLG for toluene based on its potential adverse effects reported in a 13-week oral study in rats. The MCLG is based upon a DWEL of 7 mg/L and an assumed drinking water contribution of 20 percent.

Reference -- 54 FR 22062 (05/22/89)

EPA Contact -- Health and Ecological Criteria Division / OST / (202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 1 mg/L (Final, 1991)

Considers technological or economic feasibility? -- YES

Monitoring requirements -- All systems initially monitored for four consecutive quarters; repeat monitoring dependent upon detection, vulnerability status and system size.

Analytical methodology -- Gas chromatography (EPA 502.2, 503.1); gas chromatography/mass spectrometry (EPA 524.1, 524.2): PQL= 0.005 mg/L.

Best available technology -- Granular activated carbon; packed tower aeration

Reference -- 56 FR 3526 (01/30/91); 56 FR 30266 (07/01/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

Value -- 0.04 mg/L (Proposed, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- SMCLs are non-enforceable and establish limits for contaminants which may affect the aesthetic qualities (e.g. taste and odor) of drinking water. It is recommended that systems monitor for these contaminants every three years. More frequent monitoring for contaminants such as pH, color, odor or others may be appropriate under certain circumstances. The SCML for toluene is based on odor detection. Promulgation deferred following public comment

(56 FR 3526).

Reference -- 54 FR 22062 (05/22/89); 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

CERC -

Value (status) -- 1000 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ is based on aquatic toxicity, as established under Section 311(b)(4) of the Clean Water Act, ignitability, and chronic toxicity. Available data indicate that the aquatic 96-Hour Median Threshold Limit for Toluene is between 10 and 100 ppm. Its closed-cup flash point is less than 100F and its boiling point is >100F. RQ assignments based on chronic toxicity reflect two primary attributes of the hazardous substance, the minimum effective dose (MED) levels for chronic exposure (mg/day for a 70-kg person) and the type of effect (liver necrosis, teratogenicity, etc). A composite score is determined from an evaluation of these two attributes. Toluene was determined to have a composite score between 6 and 20, corresponding to a chronic toxicity RQ of 1000 pounds.

Reference -- 50 FR 13456 (04/04/85); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - CIIT (Chemical Industry Institute of Technology). 1980. A 24-month inhalation toxicology study in Fischer-344 rats exposed to atmospheric toluene. CIIT, Research Triangle Park, NC.

OREF - Kostas, J. and J. Hotchin. 1981. Behavioral effects of low-level perinatal exposure to toluene in mice. Neurobehav. Toxicol. Teratol. 3: 467-469.

OREF - Nawrot, P.S. and R.E. Staples. 1979. Embryo-fetal toxicity and teratogenicity of benzene and toluene in the mouse. Teratology. 19: 41A (abstr.)

OREF - NTP (National Toxicology Program). 1989. Toxicology and carcinogenesis studies of toluene (CAS No. 108-88-3) in F344/N rats and B5C3F1 mice (inhalation studies). Technical Report Series No. 371. Research Triangle Park, NC.

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

IREF - ACGIH (American Conference of Governmental Industrial Hygienists). 1991. Notice of intended changes - toluene, trimethylamine, and vinyl acetate. Appl. Occup. Environ. Hyg. 6(11): 966-977.

IREF - Andersen, I., G.R. Lundqvist, L. Molhave et al. 1983. Human response to controlled levels of toluene in six-hour exposures. Scand. J. Work Environ. Health. 9: 405-418.

IREF - API (American Petroleum Institute). 1981. 26-Week inhalation toxicity study of toluene in the rat. Conducted by Bio/dynamics Inc. and Institute of Neurotoxicity, Albert Einstein College of Medicine for API, Washington, DC.

IREF - API (American Petroleum Institute). 1985. Two-generation inhalation reproduction/fertility study on a petroleum-derived hydrocarbon. Doc. ID FYI- AX-0284-0294 IN. Microfiche No. 0294.

IREF - Baelum, J., I. Andersen, G.R. Lundqvist et al. 1985. Response of solvent- exposed printers and unexposed controls to six-hour toluene exposure. Scand. J. Work Environ. Health. 11: 271-280.

IREF - Baelum, J., G. Lundqvist, L. Molhave and N.T. Andersen. 1990. Human response to varying concentrations of toluene. Int. Arch. Occup. Environ. Health. 62(1): 65-71.

IREF - Boewer, C., G. Enderlein, U. Wollgast, S. Nawka, H. Palowski, and R. Bleiber. 1988. Epidemiological study on the hepatotoxicity of occupational toluene exposure. Int. Arch. Occup. Environ. Health. 60: 181-186.

IREF - Cherry, N., H. Hutchins, T. Pace and H.A. Waldron. 1985.

Neurobehavioral effects of repeated occupational exposure to toluene and paint solvents. Br. J. Ind. Med. 42(5): 291-300.

IREF - CIIT (Chemical Industry Institute of Toxicology). 1980. A twenty-four month inhalation toxicology study in Fischer-344 rats exposed to atmospheric toluene. Conducted by Industrial Bio-Test Laboratories, Inc., Decatur, IL, and Experimental Pathology Laboratories, Inc., Raleigh, NC, for CIIT, Research Triangle Park, NC. October 15, 1980.

IREF - Courtney, K.D., J.E. Andrews, J. Springer et al. 1986. A perinatal study of toluene in CD-1 mice. Fund. Appl. Toxicol. 6: 145-154.

IREF - DaSilva, V.A., L.R. Malheiros and F.M.R. Bueno. 1990. Effects of toluene exposure during gestation on neurobehavioral development of rats and hamsters. Brazil J. Med. Biol. Res. 23: 533-537.

IREF - Echeverria, D., L. Fine, G. Langolf, A. Schork and C. Sampio. 1989. Acute neurobehavioral effects of toluene. Br. J. Ind. Med. 46(7): 483-495.

IREF - Filley, C.M., R.K. Heaton and N.L. Rosenberg. 1990. White matter dementia in chronic toluene abuse. Neurology. 40: 532-534.

IREF - Foo, S.C., J. Jeyaratnam and D. Koh. 1990. Chronic neurobehavioral effects of toluene. Br. J. Ind. Med. 47(7): 480-484.

IREF - Fornazzari, L., D.A. Wilkinson, B.M. Kapur and P.L. Carlen. 1983. Cerebellar, cortical and functional impairment in toluene abusers. Acta Neurol. Scand. 67: 319-329.

IREF - Gibson, J.E. and J.F. Hardisty. 1983. Chronic toxicity and oncogenicity bioassay of inhaled toluene in Fischer-344 rats. Fund. Appl. Toxicol. 3: 315-319.

IREF - Guzelian, P., S. Mills and H.J. Fallon. 1988. Liver structure and function in print workers exposed to toluene. J. Occup. Med. 30(10): 791-796.

IREF - Hanninen, H., M. Antti-Poika and P. Savolainen. 1987. Psychological performance, toluene exposure and alcohol consumption in rotogravure printers. Int. Arch. Occup. Environ. Health. 59(5): 475-483.

IREF - Hersh, J.H., P.E. Podruch, G. Rogers and B. Weisskopf. 1985. Toluene embryopathy. J. Pediatr. 106: 922-927.

IREF - Hudak, A. and G. Ungvary. 1978. Embryotoxic effects of benzene and its methyl derivatives: Toluene, xylene. Toxicology. 11: 55-63.

IREF - Iregren, A. 1982. Effects on psychological test performance of workers exposed to a single solvent (toluene) - a comparison with effects of exposure to a mixture of organic solvents. Neurobehav. Toxicol. Teratol. 4(6): 695- 701.

IREF - Lee, B., S. Lee, K. Lee et al. 1988. Dose-dependent increase in subjective symptom prevalence among toluene-exposed workers. Ind. Health. 26(1): 11-23.

IREF - McDonald, J.C., J. Lavoie, R. Cote and A.D. McDonald. 1987. Chemical exposures at work in early pregnancy and congenital defect: A case-referent study. Br. J. Ind. Medicine. 44: 527-533.

IREF - Metrick, S.A. and R.P. Brenner. 1982. Abnormal brainstem auditory evoked potentials in chronic paint sniffers. Ann. Neurol. 12: 553-556.

IREF - NTP (National Toxicology Program). 1990. Toxicology and carcinogenesis studies of toluene (CAS No. 108-88-3) in F344/N rats and B6C3F1 mice (inhalation studies). NTP-TR-371.
IREF - Pryor, G.T., C.S. Rebert, J. Dickinson and E.M. Feeney. 1984. Factors affecting toluene-induced ototoxicity in rats. Neurobehav. Toxicol. Teratol. 6: 223-238.
IREF - Rosenberg, N.L., M.C. Spitz, C.M. Filley, K.A. Davis, and H.H. Schaumburg. 1988. Central nervous system effects of chronic toluene abuse - clinical, brainstem evoked response and magnetic resonance imaging studies. Neurotoxicol. Teratol. 10: 489-495.
IREF - Ungvary, G. and E. Tatrai. 1985. On the embryotoxic effects of benzene and its alkyl derivatives in mice, rats, and rabbits. Arch. Toxicol. Suppl. 8: 425-430.
IREF - U.S. EPA. 1984. Health Effects Assessment for Toluene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Emergency and Remedial Response, Washington, DC. EPA-600/X-84-188.
IREF - U.S. EPA. 1985. Drinking Water Criteria Document for Toluene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Drinking Water, Washington, DC. EPA/540/1-86-033.
IREF - Von Oettingen, W.F., P.A. Neal, D.D. Donahue et al. 1942. The toxicity and potential dangers of toluene, with special reference to its maximal permissible concentration. U.S. Public Health Service, Public Health Bulletin No. 279: 50.
IREF - Yin, S., G. Li, Y. Hu et al. 1987. Symptoms and signs of workers exposed to benzene, toluene or the combination. Ind. Health. 25(3): 113-130.
CREF - Bos, R.P., R.M.E. Brouns, R. van Doorn, J.L.G. Theuws and P.Th. Henderson. 1981. Non-mutagenicity of toluene, o-, m- and p-xylene, o-methylbenzylalcohol and o-methylbenzylsulfate in the Ames assay. Mutat. Res. 88(3): 273-279.
CREF - CIIT (Chemical Industry Institute of Toxicology). 1980. A twenty-four-month inhalation toxicology study in Fischer-344 rats exposed to atmospheric toluene. Executive Summary and Data Tables, October 15. CIIT, Research Triangle Park, NC.
CREF - Coombs, M.M., T.S. Bhatt and C.J. Croft. 1973. Correlation between carcinogenicity and chemical structure in cyclopenta(a)phenanthrenes. Cancer Res. 33(4): 832-837.
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CREF - Forni, A., E. Pacifico and A. Limonta. 1971. Chromosome studies in workers exposed to benzene or toluene or both. Arch. Environ. Health. 22(3): 373-378.
CREF - Funes-Craviota, F., B. Kolmodin-hedman, J. Lindsten, et al. 1977.

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CREF - Maki-Paakkanen, J., K. Husgafvel-Pursiainen, P.L. Kalliomaki, J. Tuominen and M. Sorsa. 1980. Toluene-exposed workers and chromosome aberrations. J. Toxicol. Environ. Health. 6: 775-781.

CREF - Mortelmans, K.E. and E.S. Riccio. 1980. In vitro microbiological genotoxicity assays of toluene. Prepared by SRI International, Menlo Park, CA, under Contract No. 68-02-2947 for the U.S. EPA, Research Triangle Park, NC. p. 25.

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[IRIS] SS 11 /cf?
USER:

79-01-6
    Search in progress

SS (11) PSTG (1)

[IRIS] SS 12 p/cf?
USER:
prt dl ncar, car continuous
    Search in progress

NP (PPRT DL NCAR, CAR CONTINUOUS (IRIS))
*NONE-

[IRIS] SS 12 /cf?
USER:
prt dl ncar, car continuous

1   - IRIS
IRSN - 196
DATE - 940706
UPDT - 07/06/94, 1 field
STAT - Oral RfD Assessment (RDO) pending 08/01/92
STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) withdrawn 07/01/94
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 04/01/92
IRH - 03/01/88 CARO Text revised
IRH - 03/01/88 CARO Confidence statement revised
IRH - 03/01/88 CARI Text added
IRH - 03/01/88 CARI Confidence statement revised
IRH - 03/01/88 ? Documentation corrected
IRH - 05/01/89 CAR Carcinogen assessment summary noted as pending change
IRH - 06/01/89 CARDR Primary contact changed
IRH - 07/01/89 CAR Withdrawn; new assessment verified (in preparation)
IRH - 12/01/89 RDI Inhalation RfD now under review
IRH - 06/01/90 CAA Area code for EPA contact corrected
IRH - 06/01/90 RCRA EPA contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 04/01/92 CAA CAA regulatory action withdrawn
IRH - 07/01/92 CAR EPA contact changed; work group review dates added
IRH - 08/01/92 RDO Oral RfD now under review
IRH - 11/01/93 CAR Work group review date added
IRH - 07/01/94 CAR Work group review date added
RLEN - 6626
NAME - Trichloroethylene
RN   - 79-01-6
SY   - ACETYLENE TRICHLORIDE
SY   - ALGYLEN
SY   - ANAMENTH
SY   - BENZINOL

SY - BLACOSOLV
SY - BLANCOSOLV
SY - CECOLENE
SY - CHLORILEN
SY - 1-CHLORO-2,2-DICHLOROETHYLENE
SY - CHLORYLEA
SY - CHLORYLEN
SY - CHORYLEN
SY - CIRCOSOLV
SY - CRAWHASPOL
SY - DENSINFLUAT
SY - 1,1-DICHLORO-2-CHLOROETHYLENE
SY - DOW-TRI
SY - DUKERON
SY - ETHINYL TRICHLORIDE
SY - ETHYLENE TRICHLORIDE
SY - ETHYLENE, TRICHLORO-
SY - FLECK-FLIP
SY - FLOCK FLIP
SY - FLUATE
SY - GEMALGENE
SY - GERMALGENE
SY - LANADIN
SY - LETHURIN
SY - NARCOGEN
SY - NARKOGEN
SY - NARKOSOID
SY - NCI-C04546
SY - NIALK
SY - PERM-A-CHLOR
SY - PERM-A-CLOR
SY - PETZINOL
SY - PHILEX
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SY - TRETHYLENE
SY - TRI
SY - TRIAD
SY - TRIAL
SY - TRIASOL
SY - TRICHLOORETHEEN
SY - TRICHLOORETHYLEEN, TRI
SY - TRICHLORAETHEN
SY - TRICHLORAETHYLEN, TRI
SY - TRICHLORAN
SY - TRICHLOREN
SY - TRICHLORETHENE
SY - TRICHLORETHYLENE
SY - TRICHLORETHYLENE, TRI
SY - TRICHLOROETHENE
SY - Trichloroethylene
SY - 1,1,2-TRICHLOROETHYLENE

SY - 1,2,2-TRICHLOROETHYLENE
SY - TRI-CLENE
SY - TRICLORETENE
SY - TRICLOROETILENE
SY - TRIELENE
SY - TRIELIN
SY - TRIELINA
SY - TRIKLONE
SY - TRILEN
SY - TRILENE
SY - TRILINE
SY - TRIMAR
SY - TRIOL
SY - TRI-PLUS
SY - TRI-PLUS M
SY - UN 1710
SY - VESTROL
SY - VITRAN
SY - WESTROSOL

RDO -
o ORAL RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work
group.

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o REVIEW DATES : 06/24/85, 07/08/85, 07/22/85, 06/23/92

RDI -
o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work
group.

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o REVIEW DATES : 04/21/88, 11/17/89, 02/22/90
CAREV-
o CLASSIFICATION : The carcinogen assessment summary for

this substance has been withdrawn following further review. A new carcinogen summary is in preparation by

the CRAVE Work Group. Agency Work Group

Review -- 12/04/86, 04/06/89,

05/30/89,

09/22/93, 06/09/94 EPA Contacts:

Charles

Ris / OHEA -- 202/260-5898

WQCHU-

Water and Fish Consumption -- 2.7E+0 ug/L

Fish Consumption Only -- 8.07E+1 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criteria represents a E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS (202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC -- 4.5E+4 ug/L
Chronic LEC -- 2.19E+4 ug/L

Marine:

Acute LEC -- 2.0E+3 ug/L
Chronic LEC -- None

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 79318 (11/28/80); Quality Criteria for Water, EPA 440/5-86-001 (5/87).

EPA Contact -- Criteria and Standards Division / OWRS (202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 0 mg/L (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0 mg/L for trichloroethylene is proposed based on carcinogenic effects. Significant increases in the incidence of liver tumors have been reported in B6C3F1 mice of both sexes. Malignant lymphomas and pulmonary adenocarcinomas were also reported in mice. EPA has classified trichloroethylene in Group B2: sufficient evidence in animals and inadequate evidence in humans.

Reference -- 50 FR 46880 (11/13/85)

EPA Contact -- Health and Ecological Criteria Division / OST / (202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value (status) -- 0.005 mg/L (Final, 1987)

Considers technological or economic feasibility? -- YES

Discussion -- EPA has set an MCL based on detection limits.

Monitoring requirements -- All systems to be monitored for four consecutive quarters; repeat monitoring dependent upon detection and

vulnerability status
and system size.

Analytical methodology -- Gas chromatography (EPA 502.1, 502.2, 503.1):
gas chromatographic/mass spectrometry (EPA 524.1, 524.2).

Best available technology -- Packed tower aeration; granular activated
carbon.

Reference -- 52 FR 25690 (07/08/87); 56 FR 30266 (07/01/91).

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

CERC -

Value (status) -- 100 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for trichloroethylene is 100 pounds, based on potential
carcinogenicity. The available data indicate a hazard ranking of low, based
on a potency factor of 0.070 (mg/kg/day)-1 and weight-of-evidence classification B2, which corresponds to an RQ of 100 pounds.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline

(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

___IV.E.1. TSCA, SECTION 6

Status -- Advance Notice of Proposed Rulemaking (ANPR) (1985)

Discussion -- EPA is developing a comprehensive and integrated strategy for
a regulatory investigation of six solvents, including trichloroethylene.

Reference: 50 FR 42005 (10/17/85); 40 CFR 754

EPA Contact -- Chemical Control Division / OTS (202)260-3749 / FTS 260-3749

[IRIS] SS 12 /cf?
USER:

USER:
prt dl ncar, car continuous

1 - IRIS
IRSN - 520
DATE - 940706
STAT - Oral RfD Assessment (RDO) no data
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) pending 07/01/94
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) no data
IRH - 08/01/91 CAR Carcinogenicity assessment now under review
IRH - 07/01/94 CAR Work group review date added
RLEN - 774
NAME - Vinyl chloride
RN - 75-01-4
SY - CHLORETHENE
SY - CHLORETHYLENE
SY - CHLOROETHENE
SY - CHLOROETHYLENE
SY - CHLORURE DE VINYLE [FRENCH]
SY - CLORURO DE VINILO [SPANISH]
SY - CLORURO DI VINILE [ITALIAN]
SY - ETHENE, CHLORO-
SY - ETHYLENE, CHLORO-
SY - ETHYLENE MONOCHLORIDE
SY - HSDB 169
SY - MONOCHLOROETHENE
SY - MONOCHLOROETHYLENE
SY - RCRA WASTE NUMBER U043
SY - UN 1086
SY - VC
SY - VCM
SY - VINILE (CLORURO DI) [ITALIAN]
SY - VINYL CHLORIDE
SY - VINYL CHLORIDE, INHIBITED
SY - VINYL CHLORIDE MONOMER
SY - VINYLCHLORID [GERMAN]
SY - VINYL C MONOMER
SY - VINYLE(CHLORURE DE) [FRENCH]
SY - WINYLU CHLOREK [POLISH]

[IRIS] SS 19 /cf?
USER:
105-67-9
Search in progress

SS (19) PSTG (1)

108-38-3
Search in progress

SS (4) PSTG (1)

[IRIS] SS 5 /cf?
USER:
prt dl ncar ^H, car continuous

1 - IRIS
IRSN - 264
DATE - 920122
STAT - Oral RfD Assessment (RDO) on-line 09/30/87
STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) on-line 03/01/91
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 09/26/88 CAR Carcinogen summary on-line
IRH - 07/01/89 RDI Inhalation RfD now under review
IRH - 07/01/89 REFS Bibliography on-line
IRH - 03/01/91 CARDR Primary contact changed
IRH - 03/01/91 RCRA EPA contact changed
IRH - 01/01/92 RDO Secondary contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
RLEN - 17557
NAME - Xylenes
RN - 1330-20-7
SY - 108-38-3
SY - 1330-20-7
SY - 2106-42-3
SY - 95-47-6
SY - dimethylbenzene
SY - 1,2-dimethylbenzene
SY - 1,3-dimethylbenzene
SY - 1,4-dimethylbenzene
SY - mixed xylenes
SY - m-xylene
SY - meta-xylene
SY - o-xylene
SY - ortho-xylene
SY - p-xylene
SY - para-xylene
SY - Xylenes

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD

Hyperactivity, decreased body weight and increased mortality (males) Chronic Rat Gavage Study	NOAEL: 250 mg/kg/day (converted to 179 mg/kg/day) FEL: 500 mg/kg/day (converted to 357 mg/kg/day)	100	1	2E+0 mg/kg/day

NTP, 1986

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*Conversion Factors: Dose adjusted for gavage schedule (5\days/week).

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o ORAL RFD STUDIES :

NTP (National Toxicology Program). 1986. NTP Technical Report on the Toxicology and Carcinogenesis of Xylenes (mixed) (60.2% m-xylene, 13,6% p-xylene, 17.0 ethylbenzene and 9.1% 0-xylene) (CAS No. 1330-20-7) in F344/N rats and B6C3F1 mice (gavage studies). U.S. DHHS, PHS, NIH, NTP, Research Triangle Park, NC. NTP TR 327, NIH Publ. No. 86-2583.

Groups of 50 male and 50 female Fischer 344 rats and 50 male and 50 female B6C3F1 mice were given gavage doses of 0, 250, or 500 mg/kg/day (rats) and 0, 500, or 1000 mg/kg/day (mice) for 5 days/week for 103 weeks. The animals were observed for clinical signs of toxicity, body weight gain, and mortality. All animals that died or were killed at sacrifice were given gross necropsy and comprehensive histologic examinations. There was a dose-related increased mortality in male rats, and the increase was significantly greater in the high-dose group compared with controls. Although increased mortality was observed at 250 mg/kg/day, the increase was not significant. Although many of the early deaths were caused by gavage error, NTP (1986) did not rule out the possibility that the rats were resisting gavage dosing because of the behavioral effects of xylene. Mice given the high dose exhibited hyper-activity, a manifestation of CNS toxicity. There were no compound-related histopathologic lesions in any of the treated rats or mice. Therefore, the high dose is a FEL and the low dose a NOAEL.

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o ORAL RFD UNCERTAINTY :

UF = 100. An uncertainty factor of 100 was chosen: 10 for species-to-species extrapolation and 10 to protect sensitive individuals.

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o ORAL RFD MODIFYING FACTOR :

MF = 1.

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o ORAL RFD COMMENTS :

U.S. EPA (1984) reported an RfD of 0.01 mg/kg/day, based on a rat dietary NOAEL of 200 ppm or 10 mg/kg/day as defined by Bowers et al. (1982) in a 6-month study. This NOAEL was divided by an uncertainty factor of 1000. U.S.

EPA (1985, 1986) noted that this study used aged rats, loss of xylene from volatilization was not controlled, only one exposure level was used, and histopathologic examination was incomplete. An RfD of 4.31 mg/day (about 0.06 mg/kg/day) based on an inhalation study (Jenkins et al., 1970) using rats, guinea pigs, monkeys, and dogs exposed to o-xylene at 3358 mg/cu.m, 8 hours/day, 5 days/ week for 6 weeks or at 337 mg/cu.m continuously for 90 days was derived by U.S. EPA (1985). Deaths in rats and monkeys, and tremors in dogs occurred at the highest dose, whereas no effects were observed in the 337 mg/cu.m continuous exposure group. The RfD based on the NTP (1986) study is preferable because it is based on a chronic exposure in two species by a relevant route of administration, and comprehensive histology was performed. Xylene is fetotoxic and teratogenic in mice at high oral doses (Nawrot and Staples, 1981; Marks et al., 1982), but the RfD as calculated should be protective of these effects.

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o ORAL RFD CONFIDENCE :

Study: Medium
Data Base: Medium
RfD: Medium

The NTP (1986) study was given a medium confidence level because it was a well-designed study in which adequately sized groups of two species were tested over a substantial portion of their lifespan, comprehensive histology was performed, and a NOAEL was defined; but clinical chemistries, blood enzymes, and urinalysis were not performed. The data base was given a medium confidence level because, although supporting data exist for mice and teratogenicity and fetotoxicity data are available with positive results at high oral doses, a LOAEL for chronic oral exposure has not been defined. Medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

U.S. EPA. 1986. Health and Environmental Effects Profile for Xylenes (o-, m-, p-). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH and the Environmental Criteria and Assessment Office, Research Triangle Park, NC for the Office of Solid Waste and Emergency Response and the Office of Air Quality Planning and Standards, Office of Air and Radiation, Washington, DC.

Limited peer review and extensive agency-wide review, 1986.

U.S. EPA. 1985. Drinking Water Criteria Document For Xylenes. Prepared by Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

Extensive peer review agency-wide review.

U.S. EPA. 1984. Health Effects Assessment for Xylene. Prepared by the

Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC.

ECAO internal review and limited agency review.

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o REVIEW DATES : 12/05/85, 03/19/87
o VERIFICATION DATE : 03/19/87
o EPA CONTACTS :

Harlal Choudhury / ORD -- (513)569-7536 / FTS 684-7536

Kenneth Poirier / ORD -- (513)569-7553 / FTS 684-7553

RDI -
o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

CAREV-
o CLASSIFICATION : D; not classifiable as to human carcinogenicity.
o BASIS FOR CLASSIFICATION : Orally administered technical xylene mixtures did not result in significant increases in incidences in tumor responses in rats or mice of both sexes.
o HUMAN CARCINOGENICITY DATA :

None.

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o ANIMAL CARCINOGENICITY DATA :

Inadequate. In an NTP (1986) study, 50 male and 50 female F344/N rats were treated by gavage with mixed xylenes in corn oil (60% m-xylene, 14% p-xylene, 9% o-xylene and 17% ethylbenzene) at dosages of 0, 250 or 500 mg/kg/day, 5 days/week for 103 weeks. Similarly, 50 male and 50 female B6C3F1 mice were treated with the same xylene mixture at dosages of 0, 500 or 1000 mg/kg/day. Animals were killed and examined histologically when moribund or after 104-105 weeks. An apparent dose-related increased mortality was observed in male rats, but this difference was statistically significant for the high dose group, only. No other differences in survival between dosage groups of either sex were observed. Interstitial cell tumors of the testes could not be attributed to administration of the test compound observed in male rats (43/50 control, 38/50 low-dose and 41/49 high-dose). NTP (1986) reported that there were no significant changes in the incidence of neoplastic

or nonneoplastic lesions in either the rats or mice that could be considered related to the mixed xylene treatment, and concluded that under the conditions of these 2-year gavage studies, there was "no evidence of carcinogenicity" of xylene (mixed) for rats or mice of either sex at any dosage tested.

Maltoni et al. (1985), in a limited study, reported higher incidences (compared with controls) of malignant tumors in male and female Sprague-Dawley rats treated by gavage with xylene in olive oil at 500 mg/kg/day, 4 or 5 days/week for 104 weeks. This study did not report survival rates or specific tumor types; therefore, the results cannot be interpreted.

Berenblum (1941) reported that "undiluted" xylene applied at weekly intervals produced one tumor-bearing animal out of 40 after 25 weeks in skin-painting experiments in mice. No control groups were described. Pound (1970) reported negative results in initiation-promotion experiments with xylene as the initiator and croton oil as the promotor.

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o SUPPORTING DATA :

The frequency of sister chromatid exchanges and chromosomal aberrations were nearly identical between a group of 17 paint industry workers exposed to xylene and their respective referents (Haglund et al., 1980). In vitro, xylene caused no increase in the number of sister chromatid exchanges in human lymphocytes (Gerner-Smidt and Friedrich, 1978). Studies indicate that xylene isomers, technical grade xylene or mixed xylene are not mutagenic in tests with Salmonella typhimurium (Florin et al., 1980; NTP, 1986; Bos et al., 1981) nor in mutant reversion assays with Escherichia coli (McCarroll et al., 1981). Technical grade xylene, but not o- and m-xylene, was weakly mutagenic in Drosophila recessive lethal tests. Chromosomal aberrations were not increased in bone marrow cells of rats exposed to xylenes by inhalation (Donner et al., 1980).

CARDR-
o CARCINOGENICITY SOURCE :

U.S. EPA. 1987. Drinking Water Criteria Document for Xylene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. ECAO-CIN-416. Final.

The Drinking Water Criteria Document for Xylene has received Agency and external review.
DOCUMENT

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o REVIEW DATES : 12/02/87
o VERIFICATION DATE : 12/02/87
o EPA CONTACTS :

Bruce Mintz / ODW -- (202)260-9569 / FTS 260-9569

W. Bruce Peirano / ORD -- (513)569-7540 / FTS 684-7540

MCLG -

Value (status) -- 10.0 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- The EPA has promulgated a MCLG of 10.0 mg/L based upon potential adverse effects reported in a chronic oral study in rats. Cancer information on xylenes was reviewed and found to be inadequate for determining potential human carcinogenicity.

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Health and Ecological Criteria Division / OST / (202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 10.0 mg/L (Final, 1991)

Considers technological or economic feasibility? -- YES

Discussion -- The EPA has promulgated a MCL equal to the MCLG of 10.0 mg/L.

Monitoring requirements -- All systems initially monitored for four consecutive quarters; repeat monitoring dependent upon detection, vulnerability status and system size.

Analytical methodology -- Purge and trap capillary gas chromatography (EPA 502.2); gas chromatographic/mass spectrometry (EPA 524.2); purge and trap gas chromatography (EPA 503.1); gas chromatography/mass spectrometry (EPA 524.1); PQL= 0.005 mg/L.

Best available technology -- Granular activated carbon; packed tower aeration.

Reference -- 56 FR 3526 (01/30/91); 56 FR 30266 (07/01/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

Value -- 0.02 mg/L (Proposed, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- SMCLs are non-enforceable and establish limits for contaminants which may affect the aesthetic qualities (e.g. taste and odor) of drinking water. It is recommended that systems monitor for these contaminants every three years. More frequent monitoring for contaminants such as pH, color, odor or others may be appropriate under certain circumstances. The SMCL for xylenes is based on odor qualities. Promulgation has been deferred following public comment (56 FR 3526).

Reference -- 54 FR 22062 (05/22/89); 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

FISTD-

Status -- List "C" Pesticide (1989)

Reference -- 54 FR 30846 (07/24/89)

EPA Contact -- Registration Branch / OPP
(703)557-7760 / FTS 557-7760

FIREV-

No data available

CERC -

Value (status) -- 1000 pounds (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ is based on ignitability and aquatic toxicity as established for xylene under Section 311(b)(4) of the Clean Water Act (40 CFR 117.3). The available data indicate the aquatic 96-hour Median Threshold Limit for xylene is between 10 and 100 ppm, corresponding to an RQ of 1000 pounds. The ignitibility RQ of 1000 pounds is based on a flash point of 81 to 90F.

Reference -- 50 FR 13456 (04/04/85); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Bowers, D.E. Jr., M.S. Cannon and D.H. Jones. 1982. Ultrastructural changes in liver of young and aging rats exposed to methylated benzenes. Am. J. Vet. Res. 43(4): 679-683.

OREF - Jenkins, L.J. Jr., R.A. Jones and J. Siegel. 1970. Long-term inhalation studies on benzene, toluene, o-xylene and cumene on experimental animals. Toxicol. Appl. Pharmacol. 16: 818.

OREF - Marks, T.A., T.A. Ledous and J.A. Moore. 1982. Teratogenicity of a commercial xylene mixture in the mouse. J. Toxicol. Environ. Health. 9: 97-105.

OREF - Nawrot, P.S. and R.E. Staples. 1981. Embryofetal toxicity and teratogenicity of isomer of xylene in the mouse. Toxicologist. 1: A22.

OREF - NTP (National Toxicology Program). 1986. NTP Technical Report on the Toxicology and Carcinogenesis of Xylenes (mixed) (60.2% m-xylene, 13.6% p-xylene, 17.0% ethylbenzene and 9.1% o-xylene) in F344/N rats and B6C3F1 mice (gauge studies). U.S. DHHS, PHS, NIH, Research Triangle Park, NC. NTP TR 327, NIH Publ. No. 86-2583.

OREF - U.S. EPA. 1984. Health Effects Assessment for Xylene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC.

OREF - U.S. EPA. 1985. Drinking Water Criteria Document for Xylenes. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

OREF - U.S. EPA. 1986. Health and Environmental Effects Profile for Xylene (o-,m-,p-). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH and the Environmental Criteria and Assessment Office, Research Triangle Park, NC for the Office of Solid Waste and Emergency Respo

IREF - None

CREF - Berenblum, I. 1941. The cocarcinogenic action of croton resin. Cancer Res. 1: 44-48.

CREF - Bos, R.P., R.M.E. Brouns, R. Van Doorn, J.L.G. Theuws and P.Th. Henderson. 1981. Non-mutagenicity of toluene, o-, m- and p-xylene, o-methylbenzylalcohol and o-methylbenzylsulfate in the Ames assay. Mutat. Res. 88: 273-280.

CREF - Donner, M., J. Maki-Paakkanen, H. Norppa, M. Sorsa and H. Vainio. 1980. Genetic toxicology of xylenes. Mutat. Res. 74: 171-172.

CREF - Florin, I., L. Rutberg, M. Curvall and C.R. Enzell. 1980. Screening of tobacco smoke constituents for mutagenicity using the Ames' test. Toxicology. 15: 219-232.

CREF - Gerner-Smidt, P. and U. Friedrich. 1978. The mutagenic effect of benzene, toluene and xylene studied by the SCE technique. Mutat. Res. 58: 313-316.

CREF - Haglund, U., I. Lundberg and L. Zech. 1980. Chromosome aberrations and sister chromatid exchanges in Swedish paint industry workers. Scand. J. Work Environ. Health. 6: 291-298.

CREF - Maltoni, C., B. Conti, G. Cotti and F. Belpoggi. 1985. Experimental studies on benzene carcinogenicity at the Bologna Institute of Oncology: Current results and ongoing research. Am. J. Ind. Med. 7: 415-446.

CREF - McCarroll, N.E., C.E. Piper and B.H. Keech. 1981. An E. coli microsuspension assay for the detection of DNA damage induced by direct-acting and promutagens. Environ. Mutagen. 3: 429-444.

CREF - NTP (National Toxicology Program). 1986. Toxicology and carcinogenesis studies of xylenes (mixed) in F344/N rats and B6C3F1 mice. (Gavage studies). NTP TR 327. NIH PB No. 86-2583.

CREF - Pound, A.W. 1970. Induced cell proliferation and the initiation of skin tumor formation in mice by ultraviolet light. Pathology. 2: 269-275.

CREF - U.S. EPA. 1987. Drinking Water Criteria Document for Xylene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. ECAO-CIN-416. Final.
HAREF- None

[IRIS] SS 5 /cf?
USER:

79-01-6
Search in progress

SS (11) PSTG (1)

[IRIS] SS 12 p/cf?
USER:
prt dl ncar, car continuous
Search in progress

NP (PPRT DL NCAR, CAR CONTINUOUS (IRIS))
*NONE-

[IRIS] SS 12 /cf?
USER:
prt dl ncar, car continuous

1 - IRIS
IRSN - 196
DATE - 940706
UPDT - 07/06/94, 1 field
STAT - Oral RfD Assessment (RDO) pending 08/01/92
STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) withdrawn 07/01/94
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 04/01/92
IRH - 03/01/88 CARO Text revised
IRH - 03/01/88 CARO Confidence statement revised
IRH - 03/01/88 CARI Text added
IRH - 03/01/88 CARI Confidence statement revised
IRH - 03/01/88 ? Documentation corrected
IRH - 05/01/89 CAR Carcinogen assessment summary noted as pending change
IRH - 06/01/89 CARDR Primary contact changed
IRH - 07/01/89 CAR Withdrawn; new assessment verified (in preparation)
IRH - 12/01/89 RDI Inhalation RfD now under review
IRH - 06/01/90 CAA Area code for EPA contact corrected
IRH - 06/01/90 RCRA EPA contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 04/01/92 CAA CAA regulatory action withdrawn
IRH - 07/01/92 CAR EPA contact changed; work group review dates added
IRH - 08/01/92 RDO Oral RfD now under review
IRH - 11/01/93 CAR Work group review date added
IRH - 07/01/94 CAR Work group review date added
RLEN - 6626
NAME - Trichloroethylene
RN - 79-01-6
SY - ACETYLENE TRICHLORIDE
SY - ALGYLEN
SY - ANAMENTH

SY - BENZINOL
SY - BLACOSOLV
SY - BLANCOSOLV
SY - CECOLENE
SY - CHLORILEN
SY - 1-CHLORO-2,2-DICHLOROETHYLENE
SY - CHLORYLEA
SY - CHLORYLEN
SY - CHORYLEN
SY - CIRCOSOLV
SY - CRAWHASPOL
SY - DENSINFLUAT
SY - 1,1-DICHLORO-2-CHLOROETHYLENE
SY - DOW-TRI
SY - DUKERON
SY - ETHINYL TRICHLORIDE
SY - ETHYLENE TRICHLORIDE
SY - ETHYLENE, TRICHLORO-
SY - FLECK-FLIP
SY - FLOCK FLIP
SY - FLUATE
SY - GEMALGENE
SY - GERMALGENE
SY - LANADIN
SY - LETHURIN
SY - NARCOGEN
SY - NARKOGEN
SY - NARKOSOID
SY - NCI-C04546
SY - NIALK
SY - PERM-A-CHLOR
SY - PERM-A-CLOR
SY - PETZINOL
SY - PHILEX
SY - RCRA WASTE NUMBER U228
SY - TCE
SY - THRETHYLEN
SY - THRETHYLENE
SY - TRETHYLENE
SY - TRI
SY - TRIAD
SY - TRIAL
SY - TRIASOL
SY - TRICHLOORETHEEN
SY - TRICHLOORETHYLEEN, TRI
SY - TRICHLORAETHEN
SY - TRICHLORAETHYLEN, TRI
SY - TRICHLORAN
SY - TRICHLOREN
SY - TRICHLORETHENE

SY - TRICHLORETHYLENE
SY - TRICHLORETHYLENE, TRI
SY - TRICHLOROETHENE
SY - Trichloroethylene
SY - 1,1,2-TRICHLOROETHYLENE
SY - 1,2,2-TRICHLOROETHYLENE
SY - TRI-CLENE
SY - TRICLORETENE
SY - TRICLOROETILENE
SY - TRIELENE
SY - TRIELIN
SY - TRIELINA
SY - TRIKLONE
SY - TRILEN
SY - TRILENE
SY - TRILINE
SY - TRIMAR
SY - TRIOL
SY - TRI-PLUS
SY - TRI-PLUS M
SY - UN 1710
SY - VESTROL
SY - VITRAN
SY - WESTROSOL

RDO -
o ORAL RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

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o REVIEW DATES : 06/24/85, 07/08/85, 07/22/85, 06/23/92

RDI -
o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

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o REVIEW DATES : 04/21/88, 11/17/89, 02/22/90
CAREV-
o CLASSIFICATION : The carcinogen assessment summary for this substance has been withdrawn following further review. A new carcinogen summary is in preparation by the CRAVE Work Group. Agency Work Group Review -- 12/04/86, 04/06/89, 05/30/89, 09/22/93, 06/09/94 EPA Contacts: Charles Ris / OHEA -- 202/260-5898

WQCHU-

Water and Fish Consumption -- 2.7E+0 ug/L

Fish Consumption Only -- 8.07E+1 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criteria represents a E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC -- 4.5E+4 ug/L
Chronic LEC -- 2.19E+4 ug/L

Marine:

Acute LEC -- 2.0E+3 ug/L
Chronic LEC -- None

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 79318 (11/28/80); Quality Criteria for Water, EPA 440/5-86-001 (5/87).

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 0 mg/L (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0 mg/L for trichloroethylene is proposed based on carcinogenic effects. Significant increases in the incidence of liver tumors have been reported in B6C3F1 mice of both sexes. Malignant lymphomas and pulmonary adenocarcinomas were also reported in mice. EPA has classified trichloroethylene in Group B2: sufficient evidence in animals and inadequate evidence in humans.

Reference -- 50 FR 46880 (11/13/85)

EPA Contact -- Health and Ecological Criteria Division / OST / (202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value (status) -- 0.005 mg/L (Final, 1987)

Considers technological or economic feasibility? -- YES

Discussion -- EPA has set an MCL based on detection limits.

Monitoring requirements -- All systems to be monitored for four consecutive quarters; repeat monitoring dependent upon detection and vulnerability status and system size.

Analytical methodology -- Gas chromatography (EPA 502.1, 502.2, 503.1): gas chromatographic/mass spectrometry (EPA 524.1, 524.2).

Best available technology -- Packed tower aeration; granular activated carbon.

Reference -- 52 FR 25690 (07/08/87); 56 FR 30266 (07/01/91).

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

CERC -

Value (status) -- 100 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for trichloroethylene is 100 pounds, based on potential carcinogenicity. The available data indicate a hazard ranking of low, based on a potency factor of 0.070 (mg/kg/day)-1 and weight-of-evidence classification B2, which corresponds to an RQ of 100 pounds.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

___IV.E.1. TSCA, SECTION 6

Status -- Advance Notice of Proposed Rulemaking (ANPR) (1985)

Discussion -- EPA is developing a comprehensive and integrated strategy for a regulatory investigation of six solvents, including trichloroethylene.

Reference: 50 FR 42005 (10/17/85); 40 CFR 754

EPA Contact -- Chemical Control Division / OTS (202)260-3749 / FTS 260-3749

1 - IRIS
IRSN - 159
DATE - 930701
UPDT - 07/01/93, 2 fields
STAT - Oral RfD Assessment (RDO) no data
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 07/01/93
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 03/01/88 CAREV Text clarified
IRH - 03/01/88 CARO Confidence statement revised
IRH - 03/01/88 CARI Confidence statement revised
IRH - 01/01/91 CAR Text edited
IRH - 01/01/91 CARI Inhalation slope factor removed (global change)
IRH - 04/01/91 CAR Text edited
IRH - 04/01/91 REFS Bibliography on-line
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 07/01/93 CARDR Secondary contact's phone number changed
RLEN - 12239
NAME - alpha-Hexachlorocyclohexane (alpha-HCH)
RN - 319-84-6
SY - alpha-BENZENEHEXACHLORIDE
SY - BENZENE HEXACHLORIDE-alpha-isomer
SY - alpha-BHC
SY - CYCLOHEXANE, 1,2,3,4,5,6-HEXACHLORO-, alpha-
SY - CYCLOHEXANE, alpha-1,2,3,4,5,6-HEXACHLORO-
SY - CYCLOHEXANE, 1,2,3,4,5,6-HEXACHLORO-, alpha-isomer
SY - ENT 9,232
SY - alpha-HCH
SY - alpha-HEXACHLORAN
SY - alpha-HEXACHLORANE
SY - HEXACHLORCYCLOHEXAN
SY - alpha-HEXACHLORCYCLOHEXANE
SY - 1-alpha,2-alpha,3-beta,4-alpha,5-beta,6-beta-HEXACHLOROCYCLOHEXANE
SY - Hexachlorocyclohexane, alpha-
SY - alpha-1,2,3,4,5,6-HEXACHLOROCYCLOHEXANE
SY - alpha-LINDANE
CAREV-
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Dietary alpha-HCH has been shown to cause increased incidence of liver tumors in five mouse strains and in Wistar rats.
o HUMAN CARCINOGENICITY DATA :

Inadequate. One case report of a Japanese sanitation employee with acute leukemia was associated with occupational exposure to HCH and DDT (Hoshizaki et al., 1970).

---

o ANIMAL CARCINOGENICITY DATA :

Dietary alpha-HCH has been shown to cause increased incidences of liver tumors in five mouse strains and in Wistar rats (Ito et al., 1973a,b, 1976; Nagasaki et al., 1972, 1975; Hanada et al., 1973; Goto et al., 1972; Schulte-Hermann and Parzefall, 1981).

Ito et al. (1973a) treated groups of 20-40 male dd mice with 100, 250, or 500 ppm alpha-HCH in the diet for 24 weeks. They observed liver nodules and hepatocellular carcinomas in the two upper dose groups. In a subsequent study, Ito et al. (1976) maintained male DDY mice on a diet containing 500 ppm alpha-HCH for 16, 20, 24, or 36 weeks. This was followed by basal diet for 4, 8, 12, 16, 24, or 36 weeks, respectively. Incidence of liver tumors increased with continuous alpha-HCH administration. Incidence decreased, however, with recovery time. At 24 weeks most lesions observed were nodules, but by 60 or 72 weeks the tumors were primarily hepatocellular carcinomas.

Schulte-Hermann and Parzefall (1981) noted an increased incidence of hepatic nodules and hepatocellular carcinomas in female Wistar rats treated with approximately 20 mg/kg/day alpha-HCH for their lifetime. Male Wistar rats (18-24 animals/group) were fed alpha-HCH in the diet at 500, 1000, or 1500 ppm for 24, 48, or 72 weeks. Liver nodules and carcinomas were observed in rats fed the two highest doses for 72 weeks. Liver nodules only developed in animals fed 1000 ppm for 48 weeks (Nagasaki et al., 1975). Nagasaki et al. (1972) observed liver nodules and tumor formation in male dd mice fed 250 or 500 ppm for 24 weeks, but not in those consuming 100 ppm alpha-HCH. Both males and females of the dd strain responded in a dose-dependent fashion with liver nodules and hepatomas when fed 100, 300, or 600 ppm dietary alpha-HCH for 32 weeks, followed by 5-6 weeks basal diet (Hanada et al., 1973). In a feeding study using male ICR-JCL mice, alpha-HCH produced hepatomas in 100% of the animals (Goto et al., 1972). Liver tumors have been observed as early as 24-26 weeks (Sugihara et al., 1975).

---

o SUPPORTING DATA :

No data on the genetic toxicology of alpha-HCH are available. Alpha-HCH produces carcinogenic effects similar to that of t-HCH, which is 65% alpha-HCH. Shulte-Hermann and Parzefall (1981) reported that alpha-HCH may promote carcinogenic lesions initiated by diethylnitrosamine.

CARO -

o CLASSIFICATION : B2; probable human carcinogen

o BASIS FOR CLASSIFICATION : Dietary alpha-HCH has been shown to cause increased incidence of liver tumors in

five mouse strains and in Wistar rats.
o ORAL SLOPE FACTOR : 6.3E+0 per (mg/kg)/day
o DRINKING WATER UNIT RISK : 1.8E-4 per (ug/L)
o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	6E-1 ug/L
E-5 (1 in 100,000)	6E-2 ug/L
E-6 (1 in 1,000,000)	6E-3 ug/L

---

o ORAL DOSE-RESPONSE DATA :

Tumor Type -- hepatic nodules and hepatocellular carcinomas
Test Animals -- mouse/dd, male
Route -- diet
Reference -- Ito et al., 1973a

Administered Dose ppm	Administered Dose (mg/kg)/day	Human Equivalent Dose (mg/kg)/day	Tumor Incidence
0	0	0	0/20
100	13.0	0.012	0/20
250	37.5	0.035	30/38
500	65.0	0.060	20/20

---

o ADDITIONAL COMMENTS :

Animal doses were obtained by multiplying dietary ppm by a food consumption factor of 0.13. The authors classified 10/38 of the mid-dose tumors and 17/20 of the high-dose tumors as carcinomas. The slope factor includes an increase of (104/24)**-3 to adjust for the short duration of the experiment. The human equivalent dose was calculated by multiplying the transformed dose by (0.03/70)**1/3 for body weight adjustment and (24/104)**3 to adjust the length of the experiment to the lifespan of the animal. Mice dying during the experiment were excluded.

The unit risk should not be used if the water concentration exceeds 60 ug/L, since above this concentration the unit risk may not be appropriate.

---

o DISCUSSION OF CONFIDENCE :

Relatively few animals were treated, and the treatment time was not

considered adequate for the development of spontaneous tumors. A slope factor based on data of Nagasaki et al. (1972) was calculated to be 4.7 per (mg/kg)/day, and one based on Schulte-Hermann and Parzefall was determined to be 1.3 per (mg/kg)/day. An estimate based on the Ito et al. (1976) data was calculated to be 2.7 per (mg/kg)/day (U.S. EPA, 1980). A slope factor for t-HCH, which contains 65% alpha-HCH, was calculated to be 1.8 per (mg/kg)/day based on data of Munir et al., 1983. These estimates are supportive of the slope factor based on the Ito et al. (1973a) study.

CARI -
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Dietary alpha-HCH has been shown to cause increased incidence of liver tumors in five mouse strains and in Wistar rats.
o INHALATION UNIT RISK : 1.8E-3 per (ug/cu.m)
o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	6E-2 ug/cu.m
E-5 (1 in 100,000)	6E-3 ug/cu.m
E-6 (1 in 1,000,000)	6E-4 ug/cu.m

---

o INHALATION DOSE-RESPONSE DATA :

The inhalation risk estimates were calculated from the oral data presented in CARO.

---

o ADDITIONAL COMMENTS :

The unit risk should not be used if the air concentration exceeds 6 ug/cu.m, since above this concentration the unit risk may not be appropriate.

---

o DISCUSSION OF CONFIDENCE :

See CARO.

CARDR-
o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1986

The 1986 Health and Environmental Effects Profile received Agency review.
DOCUMENT

---

o REVIEW DATES : 12/17/86
o VERIFICATION DATE : 12/17/86
o EPA CONTACTS :

James W. Holder / OHEA -- (202)260-5721

Jim Cogliano / OHEA -- (202)260-3814

WQCHU-

Water and Fish Consumption -- 9.2E-3 ug/L

Fish Consumption Only -- 3.1E-2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criteria represents a E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC -- 1.0E+2 ug/L
Chronic LEC -- None

Marine:

Acute LEC -- 3.4E-1 ug/L
Chronic LEC -- None

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the

minimum data required to derive water quality criteria are not available. The values given are for a mixture of isomers. Criteria for lindane (gamma isomer) are also available.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

CERC -

Value (status) -- 1 pounds (Statutory, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The statutory RQ for all isomers of hexachlorocyclohexane is 1 pound, based on the chemical structural similarity to the gamma isomer, commonly known as lindane. An RQ of 1 pound, based on aquatic toxicity, was established for lindane under the Clean Water Act (Section 311 (40 CFR 117.3). The available data indicate the aquatic 96-hour Median Threshold Limit for lindane is less than 0.1 ppm, which corresponds to an RQ of 1 pound (50 FR 13465).

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - None
IREF - None
CREF - Goto, M., M. Hattori, T. Miyagawa and E. Enomoto. 1972. Contribution on ecological chemistry. II. Formation of hepatoma in mice after ingest of HCH isomers in high doses. Chemosphere. 6: 279-282.
CREF - Hanada, M., C. Yutani and T. Miyaji. 1973. Induction of hepatoma in mice by benzene hexachloride. Gann. 64: 511-513.
CREF - Hoshizaki, H., Y. Niki, H. Tajima, Y. Terada and A. Kasahara. 1969. A case of leukemia following exposure to insecticide. Acta Haematol. Japon. 32(4): 672-677.
CREF - Ito, N., H. Nagasaki, M. Arai, S. Sugihara and S. Makiura. 1973a. Histologic and ultrastructural studies on the hepatocarcinogenicity of benzene hexachloride in mice. J. Natl. Cancer Inst. 51(3): 817-826.
CREF - Ito, N., H. Nagasaki, M. Arai, S. Makiura, S. Sugihara and K. Hirao. 1973b. Histopathologic studies on liver tumerigenesis induced in mice by technical polychlorinated biphenyls and its promoting effect on liver tumors induced by benzene hexachloride. J. Natl. Cancer Inst. 51(5): 1637-1642.
CREF - Ito, N., M. Hananouchi, S. Sugihara, et al. 1976. Reversibility and irreversibility of liver tumors in mice induced by the alpha isomer of 1,2,3,4,5,6-hexachlorocyclohexane. Cancer Res. 36: 2227-2234.
CREF - Munir, K.Md., C.S. Soman and S.Y. Bhide. 1983. Hexaclorocyclohexane-induced tumorigenicity in mice under different experimental conditions. Tumori. 69: 383-386.
CREF - Nagasaki, H., S. Tomii, T. Mega, M. Marugami and N. Ito. 1972. Hepatocarcinogenic effect of alpha-, beta-, gamma-, and delta-isomers of benzene hexachloride in mice. Gann. 63(3): 393.
CREF - Nagasaki, H., H. Kawabata, Y. Miyata, et al. 1975. Effect of various factors on induction of liver tumors in animals by the alpha-isomer of benzene hexachloride. Gann. 66(2): 185-191.
CREF - Schulte-Hermann, R. and W. Parzefall. 1981. Failure to determine initiation from promotion of liver tumors in a long-term study with the phenobarbital- type inducer alpha-hexachlorocyclohexane and the role of sustained stimulation of hepatic growth and monooxygenases. Cancer Res. 41: 4140-4146.
CREF - Sugihara, S., K. Hirao, M. Hananouchi and N. Ito. 1975. Ultrastructural studies on hepatoma induced by benzene hexachloride (BHC). J. Electron. Microsc. 24: 192.
CREF - U.S. EPA. 1986. Health and Environmental Effects Profile for Hexachloro- cyclohexanes. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.
HAREF- None

117-81-7
Search in progress

SS (9) PSTG (1)

[IRIS] SS 10 /cf?
USER:
prt dl ncar, car continuous

1 - IRIS
IRSN - 13
DATE - 930201
UPDT - 02/01/93, 3 fields
STAT - Oral RfD Assessment (RDO) on-line 05/01/91
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 02/01/93
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 03/01/88 RDO Text added to paragraph 1
IRH - 09/07/88 CAR Carcinogen summary on-line
IRH - 02/01/89 CAREV Study description revised
IRH - 02/01/89 CARDR Primary contact's phone number corrected
IRH - 07/01/89 REFS Bibliography on-line
IRH - 08/01/89 RDO Text revised
IRH - 05/01/90 CAREV Text revised
IRH - 05/01/90 CREF Kozumbo et al., 1982 citation added
IRH - 05/01/91 RDO Corrected principal study title
IRH - 05/01/91 RDO 2nd para, line 3 units corrected from g/kg to mg/kg
IRH - 08/01/91 CARDR Primary and secondary contacts changed
IRH - 08/01/91 RCRA EPA contact changed
IRH - 01/01/92 RDO Secondary contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 02/01/93 CARDR Primary contact changed
RLEN - 20331
NAME - Di(2-ethylhexyl)phthalate (DEHP)
RN - 117-81-7
SY - BEHP
SY - Bis(2-ethylhexyl)-1,2-benzene-dicarboxylate
SY - Bis(2-ethylhexyl)phthalate
SY - Bisoflex 81
SY - Bisoflex DOP
SY - Compound 889
SY - DAF 68
SY - DEHP
SY - Di(2-ethylhexyl)orthophthalate
SY - Di(2-ethylhexyl)phthalate
SY - Dioctyl phthalate
SY - Di-sec-octyl phthalate
SY - DOP

SY - Ergoplast FDO
SY - Ethylhexyl phthalate
SY - 2-Ethylhexyl phthalate
SY - Eviplast 80
SY - Eviplast 81
SY - Fleximel
SY - Flexol DOP
SY - Flexol plasticizer DOP
SY - Good-Rite GP 264
SY - Hatcol DOP
SY - Hercoflex 260
SY - Kodaflex DOP
SY - Mollan O
SY - NCI- C52733
SY - Nuoplaz DOP
SY - Octoil
SY - Octyl phthalate
SY - Palatinol AH
SY - Phthalic acid, Bis(2-ethylhexyl) ester
SY - Phthalic acid, dioctyl ester
SY - Pittsburgh PX-138
SY - Platinol DOP
SY - RC Plasticizer DOP
SY - RCRA waste number U028
SY - Reomol D 79P
SY - Reomol DOP
SY - Sicol 150
SY - Staflex DOP
SY - Truflex DOP
SY - Vestinol AH
SY - Vinicizer 80
SY - Witcizer 312

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Increased relative liver weight	NOAEL: none LOAEL: 0.04% of diet (19 mg/kg bw/day)	1000	1	2E-2 mg/kg/day

Guinea Pig Sub-chronic-to-Chronic Oral Bioassay

Carpenter et al., 1953

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*Conversion Factors: none

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o ORAL RFD STUDIES :

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

The following numbers of guinea pigs were fed diets containing DEHP for a period of 1 year: 24 males and 23 females consumed feed containing 0.13% DEHP; 23 males and 23 females consumed feed containing 0.04% DEHP; and 24 males and 22 females were fed the control diet. These dietary levels corresponded to 64 or 19 mg/kg bw/day based on measured food consumption. No treatment-related effects were observed on mortality, body weight, kidney weight, or gross pathology and histopathology of kidney, liver, lung, spleen, or testes. Statistically significant increases in relative liver weights were observed in both groups of treated females (64 and 19 mg/kg bw/day).

Groups of 32 male and 32 female Sherman rats were maintained for 2 years on diets containing either 0.04, 0.13 or 0.4% DEHP (equivalent to 20, 60, and about 195 mg/kg bw/day based on measured food consumption). An F1 group of 80 animals was fed the 0.04% diet for 1 year. Mortality in the F1 treated and control groups was high; 46.2 and 42.7%, respectively, survived to 1 year. There was, however, no effect of treatment on either parental or F1 group mortality, life expectancy, hematology, or histopathology of organs. Both parental and F1 rats receiving the 0.4% DEHP diet were retarded in growth and had increased kidney and liver weights.

It appears that guinea pigs offer the more sensitive animal model for DEHP toxicity. A LOAEL in this species is determined to be 19 mg/kg/day.

---

o ORAL RFD UNCERTAINTY :

UF -- Factors of 10 each were used for interspecies variation and for protection of sensitive human subpopulations. An additional factor of 10 was used since the guinea pig exposure was longer than subchronic but less than lifetime, and because, while the RfD is set on a LOAEL, the effect observed was considered to be minimally adverse.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

Dietary levels of 0, 0.01, 0.1, and 0.3% DEHP (greater than 99% pure) were administered to male and female CD-1 mice that were examined for adverse fertility and reproductive effects using a continuous breeding protocol. DEHP was a reproductive toxicant in both sexes significantly decreasing fertility and the proportion of pups born alive per litter at the 0.3% level, and inducing damage to the seminiferous tubules. DEHP has been observed to be both fetotoxic and teratogenic (Singhe, 1972; Shiot and Nishimura, 1982).

---

o ORAL RFD CONFIDENCE :

Study -- Medium
Data Base -- Medium
RfD -- Medium

The study by Carpenter et al. (1953) utilized sufficient numbers of guinea pigs and measured multiple endpoints. The fact that there were only two concentrations of DEHP tested precludes a rating higher than medium. Since there are corroborating chronic animal bioassays, the data base is likewise rated medium. Medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

The RfD has been reviewed by the RfD Work Group. Documentation may be found in the meeting notes of 01/22/86.

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o REVIEW DATES : 01/22/86
o VERIFICATION DATE : 01/22/86
o EPA CONTACTS :

Michael L. Dourson / OHEA -- (513)569-7533

W. Bruce Peirano / OHEA -- (513)569-7553

CAREV-
o CLASSIFICATION : B2; probable human carcinogen.
o BASIS FOR CLASSIFICATION : Orally administered DEHP produced significant dose-related increases in liver tumor responses in rats and mice of both sexes.
o HUMAN CARCINOGENICITY DATA :

Inadequate. Thiess et al. (1978) conducted a mortality study of 221 DEHP production workers exposed to unknown concentrations of DEHP for 3 months to 24 years. Workers were followed for a minimum of 5 to 10 years (mean follow-up time was 11.5 years). Eight deaths were reported in the exposed population. Deaths attributable to pancreatic carcinoma (1 case) and uremia (1 case in which the workers also had urethral and bladder papillomas) were significantly elevated in workers exposed for >15 years when compared to the corresponding age groups in the general population. The study is limited by a short follow-up period and unquantified worker exposure. Results are considered inadequate for evidence of a causal association.

---

o ANIMAL CARCINOGENICITY DATA :

Sufficient. In an NTP (1982) study, 50 male and 50 female fisher 344 rats

per group were fed diets containing 0, 6000 or 12,000 ppm DEHP for 103 weeks. Similarly, groups of 50 male and 50 female B6C3F1 mice were given 0, 3000 or 6000 ppm DEHP in the diet for 103 weeks. Animals were killed and examined histologically when morbund or after 105 weeks. No clinical signs of toxicity were observed in either rats or mice. A statistically significant increase in the incidence of hepatocellular carcinomas and combined incidence of carcinomas and adenoma were observed in female rats and both sexes of mice. The combined incidence of neoplastic nodules and hepatocellular carcinomas was statistically significantly increased in the high-dose male rats. A positive dose response trend was also noted.

Carpenter et al. (1953) found no malignant tumors in treated groups of 32 male and 32 female Sherman rats. Animals were given 400, 1300 or 4000 ppm DEHP in the diet for 1 year and reduced to a maximum of 8 males and 8 females and treated for another year. Controls, F1 and 4000 ppm groups were sacrificed after being maintained on control or 4000 ppm diets for 1 year. Only 40 to 47% of the animals in each group, including F1 animals, survived 1 year. Thus, an insufficient number of animals were available for a lifetime evaluation.

Carpenter et al. (1953) did not find a carcinogenic effect in guinea pigs and dogs exposed to 1300 or 4000 ppm DEHP. Both guinea pigs and dogs were terminated after 1 year of exposure. The treatment and survival periods for these animals were considerably below their lifetimes.

---

o SUPPORTING DATA :

Studies indicate that DEHP is not a direct acting mutagen in either a forward mutation assay in Salmonella typhimurium (Seed, 1982) or the rec assay in Bacillus subtilis (Tomita et al., 1982). DEHP did not induce mutations in a modified reverse mutation plate incorporation assay in Salmonella strains TA100 and TA98 at concentrations up to 1000 ug/plate in the presence or absence of S9 hepatic homogenate (Kozumbo et al., 1982). MEHP, the monoester form of DEHP and a metabolite is positive in the rec assay and in the reverse mutation assay in Salmonella. In the absence of exogenous metabolism MEHP produced chromosomal aberrations and sister chromatid exchanges in V79 cells. Both DEHP and MEHP induced chromosomal aberrations and morphological transformation in cultured fetal Syrian hamster cells exposed in utero (Tomita et al., 1982). Chromosomal effects were not found in CHO mammalian cells (Phillips et al., 1982) exposed to DEHP. DEHP was weakly positive with metabolic activation in only one of several studies testing for mutagenic activity at the thymidine kinase locus in L5178Y mouse lymphoma cells (Ashby et al., 1985). DEHP is a potent inducer of hepatic peroxisomal enzyme activity (Ganning et al., 1984).

CARO -

o CLASSIFICATION : B2; probable human carcinogen.

o BASIS FOR CLASSIFICATION : Orally administered DEHP produced significant dose-related increases in liver tumor responses in rats and mice of both sexes.
o ORAL SLOPE FACTOR : 1.4E-2/mg/kg/day
o DRINKING WATER UNIT RISK : 4.0E-7 per (ug/L)
o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	3E+2 ug/L
E-5 (1 in 100,000)	3E+1 ug/L
E-6 (1 in 1,000,000)	3E+0 ug/L

---

o ORAL DOSE-RESPONSE DATA :

Tumor Type -- Mouse/B6C3FI, male
Test Animals -- hepatocellular carcinoma and adenoma
Route -- diet
Reference -- NTP, 1982

Dose (Administered (ppm))	Dose (Human Equivalent (mg/kg)/day)	Tumor Incidence
0	0	14/50
3000	32	25/48
6000	65	29/50

---

o ADDITIONAL COMMENTS :

In this study powdered rodent meal was provided in such a way that measured food consumption could include significant waste and spillage rather than true food intake. For this reason a standard food consumption rate of 13% mouse body weight was used in the dose conversion.

DEHP is hydrolyzed to monoesters including MEHP (Pollack et al., 1985; Lhuguenot et al., 1985; Kluwe, 1982). Although several species of animals have been determined to excrete glucuronide conjugates of monoethylhexyl phthalate (MEHP) upon exposure to DEHP, rats do not (Tanaka et al., 1975; Williams and Blanchfield, 1975; Albro et al., 1982).

Slope factors based on combined hepatocellular carcinoma and neoplastic nodule incidences were 4.5E-3/mg/kg/day for female rats, 3.2E-3/mg/kg/day for male rats. A slope factor based on hepatocellular adenomas or carcinomas in

female mice is 1.0E-2/mg/kg/day.

The unit risk should not be used if the water concentration exceeds 4E+4 ug/L, since above this concentration the slope factor may differ from that stated.

---

o DISCUSSION OF CONFIDENCE :

An adequate number of animals was observed and a statistically significant increase in incidence of liver tumors was seen in both sexes and were dose dependent in both sexes of mice and female rats. A potential source of variability in the NTP study is the possibility of feed scattering. The above calculations are based on standard food consumption rates for mice (13% of body weight) and rats (5% of body weight).

CARDR-
o CARCINOGENICITY SOURCE :

U.S. EPA. 1988. Drinking Water Criteria Document for Phthalic Acid Esters. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (External Review Draft).

The values in the 1988 Drinking Water Criteria Document for Phthalic Acid Esters (External Review Draft) have received Agency review.
DOCUMENT

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o REVIEW DATES : 08/26/87; 10/07/87
o VERIFICATION DATE : 10/07/87
o EPA CONTACTS :

Brian J. Commons / OST -- (202)260-7589

Linda R. Papa / OHEA -- (513)569-7587

WQCHU-

Water and Fish Consumption: 1.5E+4 ug/L

Fish Consumption Only: 5E+4 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of 1.5E+4 ug/L is based on consumption of contaminated aquatic organisms and water. A WQC of 5E+4 ug/L has also been established based on consumption of contaminated aquatic organisms alone.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 4.0E+2 ug/L
Chronic -- 3.6E+2 ug/L

Marine:

Acute -- 4.0E+2 ug/L
Chronic -- 3.6E+2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. EPA is currently considering withdrawing some or all the values.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value -- 0 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- NO

Discussion -- The proposed MCLG for di(2-ethylhexyl)phthalate is zero based on the evidence of carcinogenic potential (B2)

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 0.004 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- YES

Discussion -- The MCL is based on 10x the MDL and is associated with a maximum lifetime individual risk of 1 E-6.

Monitoring requirements -- Community and non-transient water system monitoring based on state vulnerability assessment; vulnerable systems to be monitored quarterly for one year; repeat monitoring dependent upon detection and size of system.

Analytical methodology -- Photoionization/gas chromatography (EPA 502.2); gas chromatographic/mass spectrometry (EPA 524.1, 524.2): PQL= 0.004 mg/L.

Best available technology -- Granular activated carbon

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

Status -- Listed (Final, 1991)

Discussion -- "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement -- All systems to be monitored unless a vulnerability assessment determines the system is not vulnerable.

Analytical methodology -- Gas chromatography (EPA 506); gas chromatography/ mass spectrometry (EPA 525).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

CERC -

Value (status) -- 100 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The 100-pound RQ is based on assessment for potential carcinogenicity. Available data indicate a hazard ranking of low based on a potency factor of 0.015/mg/kg/day and weight-of-evidence group B2, which corresponds to an RQ of 100 pounds.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

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

OREF - NTP (National Toxicology Program). 1984. Di(2-ethylhexyl)phthalate: Reproduction and fertility assessment in CD-1 mice when

administered by gavage. Final Report. NTP-84-079. NTP, Research Triangle Park, NC.
OREF - Shiota, K. and H. Nishimura. 1982. Teratogenicity of di-2-ethylhexyl phthalate and di-n-butyl phthalate in mice. Environ. Health Perspect. 45(0): 65-70.
OREF - Singhe, A.R., W.H. Lawrence and J. Autian. 1972. Teratogenicity of phthalate esters in rats. J. Pharmacol. Sci. 61: 51.
IREF - None
CREF - Albro, P.W., J.T. Corbett, J.L. Schroeder, et al. 1982. Pharmacokinetics, interactions with macromolecules and species differences in metabolism of DEHP. Environ. Health Perspect. 45: 19-25.
CREF - Ashby, J., F.J. de Serres, M. Draper, et al. 1985. Evaluation of short-term tests for carcinogens. Report of the International Programme on Chemical Safety's Collaborative Study on In Vitro Assays. Elsevier Science Publishers, Amsterdam.
CREF - Carpenter, C.P., C.S. Weil and H.F. Smith, Jr. 1953. Chronic oral toxicity of di-(2-ethylhexyl)phthalate for rats, guinea pigs and dogs. AMA Arch. Ind. Hyg. Occup. Med. 8: 219-226.
CREF - Ganning, A.E., V. Brunk and G. Dallner. 1984. Phthalate esters and their effect on the liver. Hepatology. 4(3): 541-547.
CREF - Kluwe, W.M. 1982. Overview of phthalate ester pharmacokinetics in mammalian species. Environ. Health Perspect. 45: 3-10.
CREF - Kozumbo, W.J., R. Kroll and R.J. Rubin. 1982. Assessment of the mutagenicity of phthalate esters. Environ. Health Perspect. 45: 103-109.
CREF - Lhuguenot, J.C., A.M. Mitchell, G. Milner, E.A. Lock and C.R. Elcombe. 1985. The metabolism of di-(2-ethylhexyl)phthalate (DEHP) and mono-(2-ethylhexyl)phthalate (MEHP) in rats: In vivo and in vitro dose and time dependency of metabolism. Toxicol. Appl. Pharmacol. 80: 11-22.
CREF - NTP (National Toxicology Program). 1982. Carcinogenesis bioassay of di-(2-ethylhexyl)phthalate (CAS No. 117-81-7) in F344 rats and B6C3F, mice (feed study). NTP Tech. Rep. Ser. TR No. 217, NTP, Research Triangle Park, NC.
CREF - Phillips, B.J., T.E.B. James and S.D. Gangolli. 1982. Genotoxicity studies of di-(2-ethylhexyl)phthalate and its metabolites in CHO cells. Mutat. Res. 102: 297-304.
CREF - Pollack, G.M., R.C. Li, J.C. Ermer and D.D. Shen. 1985. Effects of route of administration and repetitive dosing on the disposition kinetics of di-(2-ethylhexyl)phthalate and its mono-de-esterified metabolite in rats. Toxicol. Appl. Pharmacol. 79: 246-256.
CREF - Seed, J.L. 1982. Mutagenic activity of phthalate esters in bacterial liquid suspension assays. Environ. Health Perspect. 45: 111-114.
CREF - Tanaka, A., T. Adachi, T. Takahashi and T. Yamaha. 1975. Biochemical studies on phthalic esters. I. Elimination, distribution and metabolism of di-(2-ethylhexyl)phthalate in rats. Toxicology. 4: 253-264.
CREF - Thiess, A.M., R. Frentzel-Beyme and R. Wieland. 1978. Mortality

study in workers exposed to di-(2-ethylhexyl)phthalate (DOP). In: Moglichkerten und Grenzen des Biological Monitoring. Arbeitsmedizinische Probleme des Dienstleistungsqewerbes. Arbeitsmedizinische kolloquium [Possibilities and Limits of Biological Monitoring. Problems of Occupational Medicine in Small Industries. Colloquim in Occupational Medicine], Frankfurt/M., May 1978. Stuttgart, A.W. Gentner, p. 155-164. (Ger.)

CREF - Tomita, I., Y. Nakamura, N. Aoki and N. Inui. 1982. Mutagenic/carcinogenic potential of DEHP and MEHP. Environ. Health Perspect. 45: 119-125.

CREF - Williams, D.T. and B.J. Blanchfield. 1975. The retention, distribution, excretion and metabolism of dibutylphthalate-7-14C in the rat. J. Agric. Food Chem. 23: 854-857.

CREF - U.S. EPA. 1988. Drinking Water Criteria Document for Phthalic Acid Esters. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (External Review Draft).

HAREF- None

[IRIS] SS 10 /cf?
USER:

prt dl ncar car c^H^H^H^H^H^H^H[C^H^Hr, car continuous

1 - IRIS
IRSN - 441
DATE - 920122
STAT - Oral RfD Assessment (RDO) on-line 11/01/90
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) no data
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 11/01/90 RDO Oral RfD summary on-line
IRH - 11/01/90 REFS Bibliography on-line
IRH - 01/01/92 EXSR Regulatory Action section on-line
RLEN - 7054
NAME - 2,4-Dimethylphenol
RN - 105-67-9
SY - Phenol, 2,4-dimethyl-
SY - Caswell No. 907A
SY - EPA Pesticide Chemical Code 086804
SY - HSDB 4253
SY - m-XYLENOL
SY - NSC 3829
SY - RCRA WASTE NUMBER U101
SY - 1-HYDROXY-2,4-DIMETHYLBENZENE
SY - 2,4-dimethylphenol
SY - 2,4-Xylenol
SY - 4-HYDROXY-1,3-DIMETHYLBENZENE
SY - 4,6-DIMETHYLPHENOL

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Clinical signs (lethargy, prostration, and ataxia) and hematological changes	NOAEL: 50 mg/kg/day LOAEL: 250 mg/kg/day	3000	1	2E-2 mg/kg/day
Mouse Subchronic Oral Gavage				
U.S. EPA, 1989				

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*Conversion Factors: None

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o ORAL RFD STUDIES :

U.S. EPA. 1989. Ninety-day gavage study in Albino mice using 2,4-dimethylphenol. Study No. 410-2831, prepared by Dynamac Corporation, Rockville, MD, for the Office of Solid Waste and Emergency Response, Washington, DC.

2,4-Dimethylphenol was administered daily to male and female albino mice by gavage. The animals (30/sex/group) were dosed for 90 days with 5.0, 50.0, or 250 mg 2,4-dimethylphenol/kg/day. Two control groups, untreated and vehicle (corn oil), of similar size were also established. Effects examined included mortality, clinical signs, body weights, food consumption, opthalmology, hematology and clinical chemistry, organ weights, and gross histopathology. Although 15 deaths occurred during this study (mostly because of errors in technical procedure), only one was considered as possibly treatment-related: a male in the 5 mg/kg/day-dose group died during the first 30 days of the experiment. No significant differences were found between treated and vehicle control groups in mean body weight, body weight gains, food consumption, or eye examinations at any dosage. Toxicologically relevant clinical signs observed only after week 6 in the high-dose groups of both genders included: squinting, lethargy, prostration, and ataxia, with onset shortly after dosing. Statistically significant hematological changes ($p<0.05$) included lower mean corpuscular volume and mean corpuscular hemoglobin concentration in females at terminal, but not interim, sacrifice.

At interim sacrifice in female mid- and high-dose groups, blood urea nitrogen (BUN) levels were significantly below vehicle controls; whereas at final sacrifice in the female mid-dose group, BUN levels were significantly higher than vehicle controls. Low-dose males at interim sacrifice had significantly higher cholesterol levels. Significant differences were not found in gross necropsy or histopathological evaluations, or in organ weights, except for an increase in adrenal weights of low-dose females. The LOAEL and NOAEL for this study were 250 and 50 mg/kg/day, respectively.

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o ORAL RFD UNCERTAINTY :

UF = 3000. An uncertainty factor of 3000 was established: 10 each for inter- and intraspecies variability and 30 for lack of chronic toxicity data, data in a second species and reproductive/developmental studies.

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o ORAL RFD MODIFYING FACTOR :

MF = 1.

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o ORAL RFD COMMENTS :

A 14-day gavage study with 2,4-dimethylphenol conducted by the same laboratory that conducted the principal study, revealed lethargy, prostration, and ataxia in males and females in the 250 mg/kg/day-dose group, the same dose at which effects were found in the principal study (U.S. EPA, 1987).

No other long-term toxicity, reproductive, or developmental studies of

2,4-dimethylphenol were found in the data bases searched. Literature concerning 2,6-dimethylphenol was identified, but an SAR-based RfD is considered inappropriate when a valid long-term toxicity study for 2,4-dimethylphenol is available.

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o ORAL RFD CONFIDENCE :

Study: Medium
Data Base: Low
RfD: Low

Confidence in the study is medium, since it examined appropriate endpoints and identified both a LOAEL and a NOAEL. The results of this study are consistent with those of a 14-day gavage study. The data base provides no information on chronic and reproductive studies. Low confidence in both the data base and oral RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- None

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o REVIEW DATES : 02/21/90
o VERIFICATION DATE : 02/21/90
o EPA CONTACTS :

Harlal Choudhury / ORD -- (513)569-7633 / FTS 684-7633

Kenneth A. Poirier / ORD -- (513)569-7462 / FTS 684-7462

WQCHU-

Water and Fish Consumption: None

Fish Consumption Only: None

Considers technological or economic feasibility? -- NO

Discussion -- To control undesirable taste and odor quality of ambient water, the estimated level is 400 ug/L. There is no demonstrated relationship between organoleptic endpoints and adverse human health effects.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 2.12E+3 ug/L
Chronic -- None

Marine: None

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. Requirements and methods are covered in the reference to the federal register.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

CERC -

Value (status) -- 100 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ for 2,4-dimethylphenol is based on aquatic toxicity. The available data indicate that the aquatic 96-Hour Median Threshold Limit is between 1-10 ppm, which corresponds to an RQ of 100 pounds.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - U.S. EPA. 1987. Fourteen-day gavage study in Albino mice using 2,4-dimethylphenol. Study No. 410-2830, prepared by Dynamac Corporation, Rockville, MD for the Office of Solid Waste and Emergency Response, Washington, DC.
OREF - U.S. EPA. 1989. Ninety-day gavage study in Albino mice using 2,4-dimethylphenol. Study No. 410-2831, prepared by Dynamac Corporation, Rockville, MD, for the Office of Solid Waste and Emergency Response, Washington, DC.
IREF - None
CREF - None
HAREF- None

[IRIS] SS 20 /cf?
USER:
7440-43-9
Search in progress

SS (20) PSTG (1)

[IRIS] SS 21 /cf?
USER:

50-29-3
Search in progress

SS (5) PSTG (1)

[IRIS] SS 6 /cf?
USER:
prt dl ncar, car continuous

1 - IRIS
IRSN - 144
DATE - 920212
UPDT - 02/12/92, 52 fields
STAT - Oral RfD Assessment (RDO) on-line 09/30/87
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 05/01/91
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 09/30/87 RDO Documentation changed
IRH - 08/22/88 CAR Carcinogen summary on-line
IRH - 01/01/91 CAR Text edited
IRH - 01/01/91 CARI Inhalation slope factor removed (global change)
IRH - 05/01/91 CAREV Change Lehman, 1952 to '1951'
IRH - 05/01/91 REFS Bibliography on-line
IRH - 01/01/92 RDO Secondary contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
RLEN - 24124
NAME - p,p'-Dichlorodiphenyltrichloroethane (DDT)
RN - 50-29-3
SY - AGRITAN
SY - ANOFEX
SY - ARKOTINE
SY - AZOTOX
SY - BENZENE, 1,1'-(2,2,2-TRICHLOROETHYLIDENE)BIS(4-CHLORO-)
SY - alpha,alpha-BIS(p-CHLOROPHENYL)-beta,beta,beta-TRICHLORETHANE
SY - 1,1-BIS-(p-CHLOROPHENYL)-2,2,2-TRICHLOROETHANE
SY - 2,2-BIS(p-CHLOROPHENYL)-1,1,1-TRICHLOROETHANE
SY - BOSAN SUPRA
SY - BOVIDERMOL
SY - CHLOROPHENOTHAN
SY - CHLOROPHENOTHANE
SY - CHLOROPHENOTOXUM
SY - CITOX
SY - CLOFENOTANE
SY - DDT
SY - p,p'-DDT
SY - DEDELO
SY - DEOVAL
SY - DETOX

SY - DETOXAN
SY - DIBOVAN
SY - DICHLORODIPHENYLTRICHLOROETHANE
SY - 4,4'-DICHLORODIPHENYLTRICHLOROETHANE
SY - Dichlorodiphenyltrichloroethane, p,p'-
SY - DICOPHANE
SY - DIDIGAM
SY - DIDIMAC
SY - DIPHENYLTRICHLOROETHANE
SY - DODAT
SY - DYKOL
SY - ENT 1,506
SY - ESTONATE
SY - ETHANE, 1,1,1-TRICHLORO-2,2-BIS(p-CHLOROPHENYL)-
SY - GENITOX
SY - GESAFID
SY - GESAPON
SY - GESAREX
SY - GESAROL
SY - GUESAPON
SY - GUESAROL
SY - GYRON
SY - HAVERO-EXTRA
SY - HILDIT
SY - IVORAN
SY - IXODEX
SY - KOPSOL
SY - MICRO DDT 75
SY - MUTOXIN
SY - NA 2761
SY - NCI-C00464
SY - NEOCID
SY - PARACHLOROCIDUM
SY - PEB1
SY - PENTACHLORIN
SY - PENTECH
SY - PPZEIDAN
SY - R50
SY - RCRA WASTE NUMBER U061
SY - RUKSEAM
SY - SANTOBANE
SY - TECH DDT
SY - 1,1,1-TRICHLOOR-2,2-BIS(4-CHLOOR FENYL)-ETHAAN
SY - 1,1,1-TRICHLOR-2,2-BIS(4-CHLOR-PHENYL)-AETHAN
SY - 1,1,1-TRICHLORO-2,2-BIS(p-CHLOROPHENYL)ETHANE
SY - TRICHLOROBIS(4-CHLOROPHENYL)ETHANE
SY - 1,1,1-TRICHLORO-2,2-DI(4-CHLOROPHENYL)-ETHANE
SY - 1,1,1-TRICLORO-2,2-BIS(4-CLORO-FENIL)-ETANO
SY - ZEIDANE
SY - ZERDANE

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Liver lesions	NOEL: 1 ppm diet (0.05 mg/kg bw/day)	100	1	5E-4 mg/kg/day
27-Week Rat Feeding Study	LOAEL: 5 ppm			

Laug et al., 1950

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*Conversion Factors: Food consumption = 5% bw/day

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o ORAL RFD STUDIES :

Laug, E.P., A.A. Nelson, O.G. Fitzhugh and F.M. Kunze. 1950. Liver cell alteration and DDT storage in the fat of the rat induced by dietary levels of 1-50 ppm DDT. J. Pharmacol. Exp. Therap. 98: 268-273.

Weanling rats (25/sex/group) were fed commercial DDT (81% P,P isomer and 19% O,P isomer) at levels of 0, 1, 5, 10 or 50 ppm for 15-27 weeks. The diet was prepared by mixing appropriate amounts of DDT in corn oil solution with powdered chow. No interference with growth was noted at any level. Females stored more DDT in peripheral fat than did males, but pathologic changes were seen to a greater degree in males. Increasing hepatocellular hypertrophy, especially centrilobularly, increased cytoplasmic oxyphilia, and peripheral basophilic cytoplasmic granules (based on H and E paraffin sections) were observed at dose levels of 5 ppm and above. The effect was minimal at 5 ppm (LOAEL) and more pronounced at higher doses. No effects were reported at 1 ppm, the NOEL level used as the basis for the RfD calculation. The authors believe the effect seen at 5 ppm "represents the smallest detectable morphologic effect, based on extensive observations of the rat liver as affected by a variety of chemicals."

DDT fed to rats for 2 years (Fitzhugh, 1948) caused liver lesions at all dose levels (10-800 ppm of diet). A LOAEL of 0.5 mg/kg bw/day was established. Application of a factor of 10 each for uncertainty of estimating a NOEL from a LOAEL, as well as for interspecies conversion and protection of sensitive human subpopulations (1000 total) results in the same RfD level as that calculated from the critical study. DDT-induced liver effects were observed in mice, hamsters and dogs as well.

The Laug et al. (1950) study was chosen for the RfD calculation because: 1) male rats appear to be the most sensitive animals to DDT exposure; 2) the study was of sufficient length to observe toxic effects; and 3) several doses were administered in the diet over the range of the dose-response curve. This study also established a LOAEL and a NOEL, with the LOAEL (0.25 mg/kg/day) being the lowest of any observed for this compound.

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o ORAL RFD UNCERTAINTY :

UF = 100. A factor of 10 each was applied for the uncertainty of interspecies conversion and to protect sensitive human subpopulations. An uncertainty factor for subchronic to chronic conversion was not included because of the corroborating chronic study in the data base.

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o ORAL RFD MODIFYING FACTOR :

MF = 1.

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o ORAL RFD COMMENTS :

In one 3-generation rat reproduction study (Treon and Cleveland, 1955), offspring mortality increased at all dose levels, the lowest of which corresponds to about 0.2 mg/kg bw/day. Three other reproduction studies (rat and mouse) show no reproductive effects at much higher dose levels.

---

o ORAL RFD CONFIDENCE :

Study: Medium
Data Base: Medium
RfD: Medium

The principal study appears to be adequate, but of shorter duration than that desired; therefore, confidence in the study can be considered medium to low. The data base is only moderately supportive of both the critical effect and the magnitude, and lacks a clear NOEL for reproductive effects; therefore, confidence in the data base can also be considered medium to low. Medium to low confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

The only U.S. EPA documentation at present is on IRIS.

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o REVIEW DATES : 12/18/85
o VERIFICATION DATE : 12/18/85
o EPA CONTACTS :

Michael L. Dourson / ORD -- (513)569-7544 / FTS 684-7544

Moiz Mumtaz / ORD -- (513)569-7553 / FTS 684-7553

CAREV-
o CLASSIFICATION : B2; probable human carcinogen.
o BASIS FOR CLASSIFICATION : Observation of tumors (generally of the

liver) in seven studies in various mouse strains and three studies in rats. DDT is structurally similar to other probable carcinogens, such as DDD and DDE.

o HUMAN CARCINOGENICITY DATA :

Inadequate. The existing epidemiological data are inadequate. Autopsy studies relating tissue levels of DDT to cancer incidence have yielded conflicting results. Three studies reported that tissue levels of DDT and DDE were higher in cancer victims than in those dying of other diseases (Casarett et al., 1968; Dacre and Jennings, 1970; Wasserman et al., 1976). In other studies no such relationship was seen (Maier-Bode, 1960; Robinson et al., 1965; Hoffman et al., 1967). Studies of occupationally exposed workers and volunteers have been of insufficient duration to be useful in assessment of the carcinogenicity of DDT to humans.

---

o ANIMAL CARCINOGENICITY DATA :

Sufficient. Twenty-five animal carcinogenicity assays have been reviewed for DDT. Nine feeding studies, including two multigenerational studies, have been conducted in the following mouse strains: BALB/C, CF-1, A strain, Swiss/Bombay and (C57B1)x(C3HxAkR). Only one of these studies, conducted for 78 weeks, showed no indication of DDT tumorigenicity (NCI, 1978). Both hepatocellular adenomas and carcinomas were observed in six mouse liver tumor studies (Walker et al., 1973; Thorpe and Walker, 1973; Kashyap et al., 1977; Innes et al., 1969; Terracini et al., 1973; Turusov et al., 1973). Both benign and malignant lung tumors were observed in two studies wherein mice were exposed both in utero and throughout their lifetime (Shabad et al., 1973; Tarjan and Kemeny, 1969). Doses producing increased tumor incidence ranged from 0.15-37.5 mg/kg/day.

Three studies using Wistar, MRC Porton and Osborne-Mendel rats and doses from 25-40 mg/kg/day produced increased incidence of benign liver tumors (Rossi et al., 1977; Cabral et al., 1982; Fitzhugh and Nelson, 1946). Another study wherein Osborne-Mendel rats were exposed in this dietary dose range for 78 weeks was negative (NCI, 1978) as were three additional assays in which lower doses were given.

Tests of DDT in hamsters have not resulted in increased tumor incidence. Unlike mice and humans, hamsters accumulate DDT in tissue but do not metabolize it to DDD or DDE. Studies of DDT in dogs (Lehman, 1951, 1965) and monkeys (Adamson and Sieber, 1979, 1983) have not shown a carcinogenic effect. However, the length of these studies (approximately 30% of the animals' lifetimes) was insufficient to assess the carcinogenicity of DDT. DDT has been shown to produce hepatomas in trout (Halver, 1967).

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o SUPPORTING DATA :

DDT has been shown to act as a liver tumor promoter in rats initiated with 2-acetylaminofluorene, 2-acetamidophenanthrene or trans-4-acetylaminostilbene (Peraino et al., 1975; Scribner and Mottet, 1981; Hilpert et al., 1983).

DDT has produced both negative and positive responses in tests for genotoxicity. Positive responses have been noted in V79 mutation assays, for chromosome aberrations in cultured human lymphocytes, and for sister chromatid exchanges in V79 and CHO cells (Bradley et al., 1981; Rabello et al., 1975; Preston et al., 1981; Ray-Chaudhuri et al., 1982). In one study, DDT was reported to interact directly with DNA; this result was not confirmed in the absence of a metabolizing system (Kubinski et al., 1981; Griffin and Hill, 1978).

DDT is structurally related to the following chemicals which produce liver tumors in mice: DDE, DDD, dicofol and chlorobenzilate.

CARO -
o CLASSIFICATION : B2; probable human carcinogen.
o BASIS FOR CLASSIFICATION : Observation of tumors (generally of the liver) in seven studies in various mouse strains and three studies in rats. DDT is structurally similar to other probable carcinogens, such as DDD and DDE.
o ORAL SLOPE FACTOR : 3.4E-1 per (mg/kg)/day
o DRINKING WATER UNIT RISK : 9.7E-6 per (ug/L)
o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk
o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	1E+1 ug/L
E-5 (1 in 100,000)	1E+0 ug/L
E-6 (1 in 1,000,000)	1E-1 ug/L

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o ORAL DOSE-RESPONSE DATA :

Tumor Type -- Liver
Test Animals -- mouse/CF-1, mouse/BALB/C, rat/MRC Porton, rat/Wistar
Route -- oral (diet)
Reference -- Turusov et al., 1973; Terracini et al., 1973; Thorpe and Walker, 1973; Tomatis and Turusov, 1975; Cabral et al., 1982; Rossi et al., 1977

Slope Factor

Species/Strain Tumor Type	Male	Female	Reference
Mouse/CF-1, Benign	0.80	0.42	Turusov et al., 1973
Mouse/BALB/C, Benign	0.082		Terracini et al., 1973
Mouse/CF-1, Benign, Malignant	0.52	0.81	Thorpe and Walker, 1973
Mouse/CF-1, Benign	1.04	0.49	Tomatis and Turusov, 1975
Rat/MRC Porton		0.084	Cabral et al., 1982
Rat/Wistar, Benign	0.16	0.27	Rossi et al., 1977

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o ADDITIONAL COMMENTS :

The estimate of the slope factor did not increase in the multigeneration feeding studies (Terracini et al., 1973; Turusov et al., 1973) but remained the same from generation to generation. A geometric mean of the above slope factors was used for the overall slope factor of 3.4E-1. This was done in order to avoid excluding relevant data (note that the appropriateness of this procedure is currently under study by U.S. EPA). All tumors were of the liver; there were no metastases. A few malignancies were observed in the Turusov study; possible neoplasms were indicated in the Terracini and Tomatis studies. The Turusov study was carried out over six generations, the Terracini assay for two. The slope factor derived from data of Tarjan and Kemeny (1969) was not included in the calculation of the geometric mean because the tumors developed at different sites than in any other studies. In addition, there was a problem in this study with possible DDT contamination of the feed.

DDT is known to be absorbed by humans in direct proportion to dietary exposure; t(1/2) for clearance is 10-20 years.

The unit risk should not be used if the water concentraion exceeds 1E+3 ug/L, since above this concentration the unit risk may not be appropriate.

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o DISCUSSION OF CONFIDENCE :

Ten slope factors derived from six studies were within a 13-fold range. The slope factor derived from the mouse data alone was 4.8E-1 while that derived from the rat data alone was 1.5E-1. There was no apparent difference in slope factor as a function of sex of the animals. The geometric mean of the slope factors from the mouse and rat data combined was identical for the same tumor site as that for DDE [3.4E-1 per (mg/kg)/day], a structural analog.

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o CLASSIFICATION : B2; probable human carcinogen.
o BASIS FOR CLASSIFICATION : Observation of tumors (generally of the liver) in seven studies in various mouse strains and three studies in rats. DDT is structurally similar to other probable carcinogens, such as DDD and DDE.
o INHALATION UNIT RISK : 9.7E-5 (ug/cu.m)
o DOSE EXTRAPOLATION METHOD : Linear multistage procedure, extra risk
o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	1E+0 ug/cu.m
E-5 (1 in 100,000)	1E-1 ug/cu.m
E-6 (1 in 1,000,000)	1E-2 ug/cu.m

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o INHALATION DOSE-RESPONSE DATA :

The inhalation risk estimates were calculated from the oral data presented in CARO.

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o ADDITIONAL COMMENTS :

The unit risk should not be used if the air concentration exceeds 1E+2 ug/cu.m, since above this concentration the unit risk may not be appropriate.

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o DISCUSSION OF CONFIDENCE :

This inhalation risk estimate was calculated from the oral data presented in CARO.

CARDR-
o CARCINOGENICITY SOURCE :

U.S. EPA. 1985. The Carcinogenic Assessment Groups Calculation of the Carcinogenicity of Dicofol (Kelthane), DDT, DDE and DDD (TDE). Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC for the Hazard Evaluation Division, Office of Toxic Substances, Washington, DC.

The U.S. EPA risk assessment document on DDT is an internal report and has not received external review.

DOCUMENT

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o REVIEW DATES : 10/29/86, 11/12/86, 06/24/87
o VERIFICATION DATE : 06/24/87
o EPA CONTACTS :

James W. Holder / ORD -- (202)260-5721 / FTS 260-5721

Chao W. Chen / ORD -- (202)260-5898 / FTS 260-5898

WQCHU-

Water and Fish Consumption -- 2.4E-5 ug/L

Fish Consumption Only -- 2.4E-5 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criteria represents a E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 1.1E+0 ug/L (at any time)
Chronic -- 1.0E-3 ug/L (24-hour average)

Marine:

Acute -- 1.3E-1 ug/L (at any time)
Chronic -- 1.0E-3 ug/L (24-hour average)

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

FISTD-

Status -- List "B" Pesticide (1989)

Reference -- 54 FR 22706 (05/25/89)

EPA Contact -- Registration Branch / OPP
(703)557-7760 / FTS 557-7760

FIREV-

Action -- Most uses canceled (1972)

Considers technological or economic feasibility? -- YES

Summary of regulatory action -- Canceled, all products, except the following list of uses: 1) the U.S. Public Health Service and other Health Service Officials for control of vector diseases, 2) the USDA or military for health quarantine, 3) in drugs, for controlling body lice (to be dispensed only by a physician), 4) in the formulation of prescription drugs for controlling body lice. PR Notice 71-1 (January 15, 1971) and 37 FR 13369 (July 7, 1972). Criterion of concern: carcinogenicity, bio-accumulation, wildlife hazard and other chronic effects.

Reference -- 37 FR 13369 (07/07/72)

EPA Contact -- Special Review Branch / OPP
(703)557-7400 / FTS 557-7400

CERC -

Value (status) -- 1 pound (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for DDT is I pound, based on the aquatic toxicity, as established under CWA Section 311 (40 CFR 117.3). The available data indicate

the aquatic 96-hour Median Threshold Limit for DDT is less than 0.1 ppm. This corresponds to an RQ of 1 pound. DDT has also been found to bioaccumulate in the tissues of aquatic and marine organisms, and has the potential to concentrate in the food chain.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Fitzhugh, O.G. 1948. Use of DDT insecticides on food products. Ind. Eng. Chem. 40(4): 704-705.
OREF - Laug, E.P., A.A. Nelson, O.G. Fitzhugh and F.M. Kunze. 1950. Liver cell alteration and DDT storage in the fat of the rat induced by dietary levels of 1-50 ppm DDT. J. Pharmacol. Exp. Therap. 98: 268-273.
OREF - Treon, J.F. and F.P. Cleveland. 1955. Toxicity of certain chlorinated hydrocarbon insecticides for laboratory animals, with special reference to aldrin and dieldrin. J. Agric. Food Chem. 3(5): 402-408.
IREF - None
CREF - Adamson, R.H. and S.M. Sieber. 1979. The use of nonhuman primates for chemical carcinogenisis studies. Ecotoxicol. Environ. Qual. 2: 275-296.
CREF - Adamson, R.H. and S.M. Sieber. 1983. Chemical carcinogenesis studies in nonhuman primates. Basic Life Sci. 24: 129-156.
CREF - Bradley, M.O., B. Bhuyan, M.C. Francis, R. Langenbach, A. Peterson and E. Huberman. 1981. Mutagenesis by chemical agents in V79 Chinese hamster cells: A review and analysis of the literature.

Mutat. Res. 87: 81-142.
CREF - Cabral, J.R.P., R.K. Hall, L. Rossi, S.A. Bronczyk and P. Shubik. 1982. Effects of long-term intake of DDT on rats. Tumorigenesis. 68: 11-17.
CREF - Casarett, L.J., G.C. Fryer, W.L. Yauger, Jr. and H.W. Klemmer. 1968. Organochlorine pesticide residues in human tissue--Hawaii. Arch. Environ. Health. 17: 306-311.
CREF - Dacre, J.C. and R.W. Jennings. 1970. Organochlorine insecticides in normal and carcinogenic human lung tissues. Toxicol. Appl. Pharmacol. 17: 277.
CREF - Fitzhugh, O.G. and A.A. Nelson. 1946. The chronic oral toxicity of DDT [2,2- bis(p-chlorophenyl-1,1,1-trichloroethane)]. J. Pharmacol. 89: 18-30.
CREF - Griffin, D.E. and W.E. Hill. 1978. In vitro breakage of plasmid DNA by mutagens and pesticides. Mutat. Res. 52: 161-169.
CREF - Halver, J.E. 1967. Crystalline aflatoxin and other vectors for trout hepatoma. In: J.E. Halver and I.A. Mitchell, Ed. Trout Hepatoma Research Conference Papers. Bureau of Sport Fisheries and Wildlife Research Rep. No. 70. Dept. of the Interior, Washington, DC: p. 78-102.
CREF - Hilpert, D., W. Romen and H-G. Neumann. 1983. The role of partial hepatectomy and of promoters in the formation of tumors in non-target tissues of trans-4-acetylaminostilbene in rats. Carcinogenesis. 4(12): 1519-1525.
CREF - Hoffman, W.S., H. Adler, W.I. Fishbein and F.C. Bauer. 1967. Relation of pesticide concentrations in fat to pathological changes in tissues. Arch. Environ. Health. 15: 758-765.
CREF - Innes, J.R.M., B.M. Ulland, M.G. Valerio, et al. 1969. Bioassay of pesticides and industrial chemicals for tumorgenicity in mice: A preliminary note. J. Natl. Cancer Inst. 42(6): 1101-1114.
CREF - Kashyap, S.K., S.K. Nigam, A.B. Karnik, R.C. Gupta and S.K. Chatterjee. 1977. Carcinogenicity of DDT (dichlorodiphenyl trichloroethane) in pure inbred Swiss mice. Int. J. Cancer. 19: 725-729.
CREF - Kubinski, H., G.E. Gutzke and Z.O. Kubinski. 1981. DNA-cell-binding (DCB) assay for suspected carcinogens and mutagens. Mutat. Res. 89: 95-136.
CREF - Lehman, A.J. 1951. Chemicals in Foods: A Report to the Association of Food and Drug Officials on Current Developments. Part II, Pesticides. Section V. Pathology, Q. Bull. Assoc. Food Drug Office, U.S. 15(4): 126-132.
CREF - Lehman, A.J. 1965. Summaries of pesticide toxicity. Association of Food and Drug Officials of the United States, Topeka, Kansas.
CREF - Maier-Bode, H. 1960. Zur Frage der Herkunft des DDT im Koperfett des Menschen. Med. Exp. 3: 284-286. (Ger.)
CREF - NCI (National Cancer Institute). 1978. Bioassays of DDT, TDE and p,p'-DDE for possible carcinogenicity (CAS No. 50-29-3, 72-54-8, 72-55-9). NCI Report No. 131. DHEW Publ. No. (NIH) 78-1386.
CREF - Peraino, C., R.J.M. Fry, E. Staffeldt and J. P. Christopher. 1975. Comparative enhancing effects of phenobarbital, amobarbital,

diphenylhydantoin, and dichlorodiphenyltrichloroethane of 2-acetylaminofluorene-induced hepatic tumorgenesis in the rat. Cancer Res. 35: 2884-2890.
CREF - Preston, R.J., W. Au, M.A. Bender, et al. 1981. Mammalian in vivo and in vitro cytogenetic assays: A report of the U.S. EPA's Gene-Tox Program. Mutat. Res. 87: 143-188.
CREF - Rabello, M.N., W. Becak, W.F. DeAlmeida, et al. 1975. Cytogenetic study on individuals occupationally exposed to DDT. Mutat. Res. 28: 449-454.
CREF - Ray-Chaudhuri, R., M. Currens and P.T. Iype. 1982. Enhancement of sister- chromatid exchanges by tumor promoters. Br. J. Cancer. 45: 769-777.
CREF - Robinson, J., A. Richardson, C.G. Hunter, A.N. Crabtree and H.S. Rees. 1965. Organo-chlorine insecticide content of human adipose tissue in south-eastern England. Br. J. Ind. Med. 22: 220-229.
CREF - Rossi, L., M. Ravera, G. Repetti and L. Santi. 1977. Long-term administration of DDT or phenobarbital-Na in Wistar rats. Int. J. Cancer. 19: 179-185.
CREF - Scribner, J.D. and N.K. Mottet. 1981. DDT acceleration of mammary gland tumors induced in the male Sprague-Dawley rat by 2-acetomidophenanthrene. Carcinogenesis. 2(12): 1235-1239.
CREF - Shabad, L.M., T.S. Kolesnichenko and T.V. Nikonova. 1973. Transplacental and combined long-term effect of DDT in five generations of A-strain mice. Int. J. Cancer. 11: 688-693.
CREF - Tarjan, R. and T. Kemeny. 1969. Multigeneration studies on DDT in mice. Food Cosmet. Toxicol. 7: 215-222.
CREF - Terracini, B., M.C. Testa, J.R. Cabral and N. Day. 1973. The effects of long-term feeding of DDT to BALB/c mice. Int. J. Cancer. 11: 747-764.
CREF - Thorpe, E. and A.I.T. Walker. 1973. The toxicology of dieldrin (HEOD). II. Comparative long-term oral toxicity studies in mice with dieldrin, DDT, phenobarbitone, beta-BHC and gamma-BHC. Food Cosmet. Toxicol. 11: 433-442.
CREF - Tomatis, L. and V. Turusov. 1975. Studies on the carcinogenicity of DDT. Gann Monograph Cancer Res. 17: 219-241.
CREF - Turusov, V.S., N.E. Day, L. Tomatis, E. Gati and R.T. Charles. 1973. Tumors in CF-1 mice exposed for six consecutive generations to DDT. J. Natl. Cancer Inst. 51: 983-998.
CREF - U.S. EPA. 1985. The Carcinogenic Assessment Groups Calculation of the Carcinogenicity of Dicofol (Kelthane), DDT, DDE and DDD (TDE). Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC for the Hazard Evaluation Division, Office of Pesticide Programs, Office of Pesticides and Toxic Substances, Washington, DC.
CREF - Walker, A.I.T., E. Thorpe and D.E. Stevenson. 1973. The toxicology of dieldrin (HEOD). I. Long-term oral toxicity studies in mice. Food Cosmet. Toxicol. 11: 415-432.
CREF - Wasserman, M., D.P. Nogueira, L. Tomatis, et al. 1976. Organochlorine compounds in neoplastic and adjacent apparently normal breast tissue. Bull. Environ. Contam. Toxicol. 15(4):

115-29-7
Search in progress

SS (8) PSTG (1)

[IRIS] SS 9 /cf?
USER:
prt dl ncar, car continuous

1 - IRIS
IRSN - 230
DATE - 940706
UPDT - 07/06/94, 1 field
STAT - Oral RfD Assessment (RDO) withdrawn 05/01/93
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) pending 07/01/94
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 07/01/89 RDO MRID numbers added to principal study
IRH - 07/01/89 RDO Citations added
IRH - 07/01/89 REFS Bibliography on-line
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 12/01/92 RDO Withdrawn; new Oral RfD verified (in preparation)
IRH - 12/01/92 OREF Oral RfD references withdrawn
IRH - 05/01/93 RDO Work group review date added
IRH - 07/01/94 CAR Carcinogenicity assessment now under review
RLEN - 3871
NAME - Endosulfan
RN - 115-29-7
SY - BENZOEPIN
SY - BEOSIT
SY - BIO 5,462
SY - CHLORTHIEPIN
SY - CRISULFAN
SY - CYCLODAN
SY - DEVISULPHAN
SY - ENDOCEL
SY - ENDOSOL
SY - Endosulfan
SY - ENDOSULPHAN
SY - ENSURE
SY - ENT 23,979
SY - FMC 5462
SY - 1,2,3,4,7,7-HEXACHLOROBICYCLO(2.2.1)HEPTEN-5,6-BIOXYMETHYLENESULFITE
SY - alpha,beta-1,2,3,4,7,7-HEXACHLOROBICYCLO(2.2.1)-2-HEPTENE-5,6-BISOXYMETHYLENE
SY - SULFITE
SY - HEXACHLOROHEXAHYDROMETHANO 2,4,3-BENZODIOXATHIEPIN-3-OXIDE

SY - 6,7,8,9,10,10-HEXACHLORO-1,5,5a,6,9,9a-HEXAHYDRO-6,9-METHANO-2,4,3-
SY - BENZODIOXATHIEPIN-3-OXIDE
SY - 1,4,5,6,7,7-HEXACHLORO-5-NORBORNENE-2,3-DIMETHANOL cyclic SULFITE
SY - HILDAN
SY - HOE 2,671
SY - INSECTOPHENE
SY - KOP-THIODAN
SY - MALIX
SY - NA 2761
SY - NCI-C00566
SY - NIA 5462
SY - NIAGARA 5,462
SY - 5-NORBORNENE-2,3-DIMETHANOL, 1,4,5,6,7,7-HEXACHLORO-, CYCLIC SULFITE
SY - OMS 570
SY - RCRA WASTE NUMBER P050
SY - THIFOR
SY - THIMUL
SY - THIODAN
SY - THIOFOR
SY - THIOMUL
SY - THIONEX
SY - THIOSULFAN
SY - THIOSULFAN TIONEL
SY - TIOVEL

RDO -
o ORAL RFD SUMMARY :

The Oral RfD for endosulfan has been withdrawn on 12/01/92 as a result of further review. A new RfD summary is in preparation by the RfD/RfC Work Group.

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o REVIEW DATES : 05/20/85, 05/31/85, 11/21/85, 02/05/86, 06/11/86, 03/18/87, 11/04/92, 03/31/93

WQCHU-

Water and Fish Consumption -- 7.4E+1 ug/L

Fish Consumption Only -- 1.59E+2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC for the protection of human health is based on an ADI of 0.28 mg/day, which was derived from a 78-week mouse study from the National Cancer Institute. The bioconcentration factor was established to be 270.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 2.2E-1 ug/L (at any time)
Chronic -- 5.6E-2 ug/L (24 hour average)

Marine:

Acute -- 3.4E-2 ug/L (at any time)
Chronic -- 8.7E-3 ug/L (24 hour average)

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

FISTD-

Status -- Issued (1982)

Reference -- Endosulfan Pesticide Registration Standard. April, 1982 (NTIS No. PB82-243999).

EPA Contact -- Registration Branch / OPP
(703)557-7760 / FTS 557-7760

FIREV-

No data available

CERC -

Value (status) -- 1 pound (Final, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ is based on aquatic toxicity as established under CWA Section 311 (40 CFR 117.3). The available data indicate that the 96-Hour Median Threshold Limit for endosulfan is less than 0.1 ppm. Endosulfan is known to have a chronic effect but, since its RQ is set at the lowest possible level based on aquatic toxicity, no further evaluation has been carried out.

Reference -- 50 FR 13456 (04/04/85); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

[IRIS] SS 9 /cf?
USER:

r^Hprt dl cn^H^Hncar, car coni^Htinuous

1 - IRIS
IRSN - 63
DATE - 920706
UPDT - 07/06/92, 52 fields
STAT - Oral RfD Assessment (RDO) on-line 03/01/88
STAT - Inhalation RfC Assessment (RDI) pending 07/01/92
STAT - Carcinogenicity Assessment (CAR) pending
STAT - Drinking Water Health Advisories (DWHA) on-line 08/01/90
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 03/31/87 CAA Additional pesticide AI data added
IRH - 03/01/88 RDO Principal study citation corrected
IRH - 03/01/88 RDO Dose conversion corrected
IRH - 03/01/88 RDO Text revised
IRH - 03/01/88 HADV Health Advisory added
IRH - 08/01/90 HADR Primary contact changed
IRH - 08/01/90 RCRA EPA contact changed
IRH - 08/01/91 REFS Bibliography on-line
IRH - 01/01/92 RDO Secondary contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 07/01/92 RDI Inhalation RfC now under review
RLEN - 17109
NAME - gamma-Hexachlorocyclohexane (gamma-HCH)
RN - 58-89-9
SY - AALINDAN
SY - AFICIDE
SY - AGRISOL G-20
SY - AGRONEXIT
SY - AMEISENATOD
SY - AMEISENMITTEL MERCK
SY - APARASIN
SY - APHTIRIA
SY - APLIDAL
SY - ARBITEX
SY - BBH
SY - BEN-HEX
SY - BENTOX 10
SY - gamma-BENZENE HEXACHLORIDE
SY - BENZENE HEXACHLORIDE-gamma-isomer
SY - BEXOL
SY - BHC
SY - gamma-BHC
SY - CELANEX
SY - CHLORESENE
SY - CODECHINE
SY - CYCLOHEXANE, 1,2,3,4,5,6-HEXACHLORO-, gamma-isomer
SY - DBH
SY - DETMOL-EXTRAKT

SY - DETOX 25
SY - DEVORAN
SY - DOL GRANULE
SY - DRILL TOX-SPEZIAL AGLUKON
SY - ENT 7,796
SY - ENTOMOXAN
SY - EXAGAMA
SY - FORLIN
SY - GALLOGAMA
SY - GAMACARBATOX
SY - GAMACID
SY - GAMAPHEX
SY - GAMENE
SY - GAMISO
SY - GAMMA-COL
SY - GAMMAHEXA
SY - GAMMAHEXANE
SY - GAMMALIN
SY - GAMMALIN 20
SY - GAMMATERR
SY - GAMMEX
SY - GAMMEXANE
SY - GAMMOPAZ
SY - GEXANE
SY - HCCH
SY - HCH
SY - gamma-HCH
SY - HECLOTOX
SY - HEXA
SY - HEXACHLORAN
SY - HEXACHLORANE
SY - gamma-HEXACHLORANE
SY - gamma-HEXACHLORAN
SY - gamma-HEXACHLOR
SY - gamma-HEXACHLOROBENZENE
SY - 1,2,3,4,5,6-HEXACHLOROCYCLOHEXANE
SY - 1-alpha,2-alpha,3-beta,4-alpha,5-alpha,6-beta-HEXACHLOROCYCLOHEXANE
SY - Hexachlorocyclohexane, gamma-
SY - gamma-1,2,3,4,5,6-HEXACHLOROCYCLOHEXANE
SY - 1,2,3,4,5,6-HEXACHLOROCYCLOHEXANE, gamma-ISOMER
SY - HEXACHLOROCYCLOHEXANE, gamma-ISOMER
SY - HEXATOX
SY - HEXAVERM
SY - HEXICIDE
SY - HEXYCLAN
SY - HGI
SY - HORTEX
SY - INEXIT
SY - ISOTOX
SY - JACUTIN

SY - KOKOTINE
SY - KWELL
SY - LENDINE
SY - LENTOX
SY - LIDENAL
SY - LINDAFOR
SY - LINDAGAM
SY - LINDAGRAIN
SY - LINDAGRANOX
SY - Lindane
SY - gamma-LINDANE
SY - LINDAPOUDRE
SY - LINDATOX
SY - LINDOSEP
SY - LINTOX
SY - LOREXANE
SY - MILBOL 49
SY - MSZYCOL
SY - NA 2761
SY - NCI-C00204
SY - NEO-SCABICIDOL
SY - NEXEN FB
SY - NEXIT
SY - NEXIT-STARK
SY - NEXOL-E
SY - NICOCHLORAN
SY - NOVIGAM
SY - OMNITOX
SY - OWADZIAK
SY - PEDRACZAK
SY - PFLANZOL
SY - QUELLADA
SY - RCRA WASTE NUMBER U129
SY - SANG gamma
SY - SILVANOL
SY - SPRITZ-RAPIDIN
SY - SPRUEHPFLANZOL
SY - STREUNEX
SY - TAP 85
SY - TRI-6
SY - VITON

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Liver and kidney toxicity	NOAEL: 4 ppm diet [0.33 mg/kg/day (females)]	1000	1	3E-4 mg/kg/day

Rat, Subchronic Oral Bioassay Zoecon Corp., 1983 — LOAEL: 20 ppm diet [1.55 mg/kg/day (males)]

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*Conversion Factor: Converted dose calculated from actual food consumption data

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o ORAL RFD STUDIES :

Zoecon Corporation. 1983. MRID No. 00128356. Available from EPA. Write to FOI, EPA, Washington D.C. 20460.

Twenty male and 20 female Wistar KFM-Han (outbred) SPF rats/treatment group were administered 0, 0.2, 0.8, 4, 20, or 100 ppm lindane (99.85%) in the diet. After 12 weeks, 15 animals/sex/group were sacrificed. The remaining rats were fed the control diet for an additional 6 weeks before sacrifice. No treatment-related effects were noted on mortality, hematology, clinical chemistry, or urinalysis. Rats receiving 20 and 100 ppm lindane were observed to have greater-than-control incidence of the following: liver hypertrophy, kidney tubular degeneration, hyaline droplets, tubular distension, interstitial nephritis, and basophilic tubules. Since these effects were mild or rare in animals receiving 4 ppm, this represents a NOAEL. The reviewers of the study calculated the dose to be 0.29 mg/kg/day for males and 0.33 mg/kg/day for females, based on measured food intake.

In a 2-year feeding study (Fitzhugh, 1950), 10 Wistar rats/sex/group were exposed to 5, 10, 50, 100, 400, 800, or 1600 ppm lindane. Slight liver and kidney damage and increased liver weights were noted at the 100 ppm level. If a food intake equal to 5% body weight is assumed, a NOAEL of 2.5 mg/kg bw/day (50 ppm) can be determined from this assay. In a 2-year bioassay (Rivett et al., 1978), four beagle dogs/sex/group were administered 0, 25, 50, or 100 ppm lindane in the diet. Treatment-related effects noted in the animals of the 100 ppm group were increased serum alkaline phosphatase and enlarged dark friable livers. A NOAEL was determined to be 50 ppm (1.6 mg/kg bw/day).

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o ORAL RFD UNCERTAINTY :

UF = 1000. A factor of 10 each was employed for use of a subchronic vs. a lifetime assay, to account for interspecies variation and to protect sensitive human subpopulations.

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o ORAL RFD MODIFYING FACTOR :

MF = 1.

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o ORAL RFD COMMENTS :

Data on reproductive effects of lindane are inconclusive. Most reports

indicate that hexachlorocyclohexane isomers are nonteratogenic.

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o ORAL RFD CONFIDENCE :

Study: Medium
Data Base: Medium
RfD: Medium

The principal study used an adequate number of animals and measured mul- tiple endpoints. Since there are other reported chronic and subchronic studies, confidence in the data base is medium. Medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

U.S. EPA. 1985. Drinking Water Criteria Document for Lindane. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

The RfD in the Drinking Water Criteria Document has been extensively reviewed by U.S. EPA scientists and selected outside experts.

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o REVIEW DATES : 01/22/86
o VERIFICATION DATE : 01/22/86
o EPA CONTACTS :

Michael L. Dourson / OHEA -- (513)569-7533

W. Bruce Peirano / OHEA -- (513)569-7553

RDI -
o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

HAONE-

Appropriate data for calculating a One-day HA are not available. It is recommended that the Ten-day HA of 1.2 mg/L (rounded to 1 mg/L) be used as the One-day HA.

HATEN-

Ten-day HA -- 1.2E+0 mg/L

NOAEL -- 12.3 mg/kg/day
UF -- 100 (allows for interspecies and intrahuman variability)
Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Muller et al., 1981

Rats were fed lindane at daily doses of 1.3, 12.3, or 25.4 mg/kg bw in the diet for 30 days. Nerve conduction delay was observed in the animals fed a daily dose of 25.4 mg/kg but was not observed at dose levels of 12.3 or 1.3 mg/kg. A NOAEL of 12.3 mg/kg/day was identified.

HALTC-

Longer-term (Child) HA -- 3.3E-2 mg/L

NOAEL -- 0.33 mg/kg/day
UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)
Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Zoecon Corporation, 1983

Male and female rats were fed lindane at dietary levels of 0, 0.2, 0.8, 4, 20, or 100 ppm for 84 consecutive days. Liver hypertrophy, kidney tubular degeneration, hyaline droplets, tubular casts, tubular distension, interstitial nephritis, and basophilic tubules were observed in the 20 and 100 ppm groups. Effects were rare and very mild when noted at 4 ppm. The NOAEL was considered to be 4 ppm in this study. Based upon measured food consumption, the daily intake of lindane at 4 ppm in the diet was 0.29 mg/kg in males and 0.33 mg/kg in females. The dose of 0.33 mg/kg is identified as the NOAEL.

HALTA-

Longer-term (Adult) HA -- 1.2E-1 mg/L

NOAEL -- 0.33 mg/kg/day
UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)
Assumptions -- 2 L/day water consumption for a 70-kg adult

Principal Study -- Zoecon Corporation, 1983 (study described in HALTC)

HALIF-

Drinking Water Equivalent Level (DWEL) -- 1E-2 mg/L

RfD Verification Date -- 01/22/86 (see the RfD Section of this file)

Assumptions -- 2 L/day water consumption for a 70-kg adult

Lifetime HA -- 2E-4 mg/L

Assumptions -- 20% exposure by drinking water

Principal Study -- Zoecon Corporation, 1983

This study was used in the derivation of the oral chronic RfD; see the RfD Section for a description. NOTE: A safety factor of 10 was used in the derivation of this HA, in addition to the UF of 1000 for the RfD, to account for the possible carcinogenicity of this substance. The assessment for the potential human carcinogenicity of lindane is currently under review.

OLEP -

No data available

ALAB -

Determination of lindane is by a liquid-liquid extraction gas chromatographic procedure.

TREAT-

Treatment techniques capable of removing lindane from drinking water include adsorption on activated carbon, air stripping, reverse osmosis, and oxidation.

HADR -

o HEALTH ADVISORY SOURCE :

U.S. EPA. 1985. Final Draft of the Drinking Water Criteria Document on Lindane. Office of Drinking Water, Washington, DC.
DOCUMENT

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o HEALTH ADVISORY REVIEW :

EPA review of HAs in 1985.

Public review of HAs following notification of availability in October, 1985.

Scientific Advisory Panel review of HAs in June, 1986.

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o EPA DRINKING WATER CONTACT :

Jennifer Orme / OST -- (202)260-7586

Edward V. Ohanian / OST -- (202)260-7571

WQCHU-

Water and Fish Consumption: 1.86E-2 ug/L

Fish Consumption Only: 6.25E-2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient concentration should be zero. However, zero may not be attainable at this time so the criteria given represents a E-6 incremental increase in cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 2.0E+0 ug/L
Chronic -- 8.0E-2 ug/L

Marine:

Acute -- 1.6E-1 ug/L
Chronic -- None

Considers technological or economic feasibility? -- NO

Discussion -- Water quality criteria for the protection of aquatic life are derived from a minimum data base of acute and chronic tests on a variety of aquatic organisms. The data are assumed to be statistically representative and are used to calculate concentrations which will not have significant short- or long-term effects on 95% of the organisms exposed. Recent criteria (1985 and later) contain duration and frequency stipulations: the acute

criteria maximum concentration is a 1-hour average and the chronic criteria continuous concentration is a 4-day average which are not to be exceeded more than once every 3 years, on the average (see Stephen et al., 1985). Earlier criteria (1980-1984) contained instantaneous acute and 24-hour average chronic concentrations which were not to be exceeded. The freshwater chronic WQC is a 24-hour average.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 0.0002 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0.0002 mg/L for lindane is promulgated based upon potential adverse effects reported in a dietary study in rats. The MCLG is based upon a DWEL of 0.01 mg/L and an assumed drinking water contribution of 20 percent. An additional uncertainty factor of 10 was applies since lindane was classed a category II contaminant (limited evidence of carcinogenicity via drinking water ingestion).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value (status) -- 0.0002 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- EPA has promulgated an MCL equal to the MCLG and PQL of 0.0002 mg/L.

Monitoring requirements -- All systems monitored for four consecutive quarters every three years; repeat monitoring dependent upon detection, vulnerability status and system size.

Analytical methodology -- Microextraction/gas chromatography (EPA 505); electron-capture/gas chromatography (EPA 508P; gas chromatographic/mass

spectrometry (EPA 525): PQL= 0.0002 mg/L.

Best available technology -- Granular activated carbon

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

FISTD-

Status -- Issued (1985)

Reference -- Lindane Pesticide Registration Standard. September, 1985
(NTIS No. PB86-175114).

EPA Contact -- Registration Branch / OPP
(703)557-7760 / FTS 557-7760

FIREV-

Action -- Final regulatory decision - PD4 (1984)

Considers technological or economic feasibility? -- YES

Summary of regulatory action -- Negotiated settlements have been made for Lindane in dog dips [49 FR 26282 (06/27/84)] and in smoke bombs [50 FR 5424 (02/08/85)].

Reference -- 45 FR 48513 (10/19/83); 49 FR 26282 (06/27/84)

EPA Contact -- Special Review Branch / OPP
(703)557-7400 / FTS 557-7400

CERC -

Value (status) -- 1 pound (Statutory, 1987)

Considers technological or economic feasibility? -- NO

Discussion -- The 1-pound RQ for lindane is based on aquatic toxicity as assigned by Section 311(b)(4) of the Clean Water Act (40 CFR 117.3). Available data indicate a 96-hour Median Threshold Limit of less than 0.1 ppm, which corresponds to an RQ of 1 pound.

Reference -- 52 FR 8140 (03/16/87); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Fitzhugh, O.G., A.A. Nelson and J.P. Frawley. 1950. The chronic toxicities of technical benzene hexachloride and its alpha, beta and gamma isomers. J. Pharmacol. Exp. Ther. 100: 59-66.

OREF - Muller, D., H. Klepel, R.M. Macholz, H.J. Lewerenz and R. Engst. 1981. Electroneurophysiological studies on neurotoxic effects of hexachlorocyclo- hexane isomers and gamma-pentachlorocyclohexene. Bull. Environ. Contam. Toxicol. 27(5): 704-706.

OREF - Rivett, K.F., H. Chesterman, D.N. Kellett, A.J. Newman, and A.N.

Worden. 1978. Effects of feeding lindane to dogs for periods of up to 2 years. Toxicology. 9: 273-289.
OREF - U.S. EPA. 1985. Drinking Water Criteria Document for Lindane. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.
OREF - Zoecon Corporation. 1983. MRID No. 00128356. Available from EPA. Write to FOI, EPA, Washington, DC 20460.
IREF - None
CREF - None
HAREF- Muller, D., H. Klepel, R.M. Macholz, H.J. Lewerenz and R. Engst. 1981. Electroneurophysiological studies on neurotoxic effects of hexachlorocyclo- hexane isomers and gamma-pentachlorocyclohexene. Bull. Environ. Contam. Toxicol. 27(5): 704-706.
HAREF- U.S. EPA. 1985. Final Draft of the Drinking Water Criteria Document on Lindane. Office of Drinking Water, Washington, DC.
HAREF- Zoecon Corporation. 1983. MRID No. 00128356. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

[IRIS] SS 7 /cf?
USER:

prt dl ncar, car continuous

1 - IRIS
IRSN - 85
DATE - 920406
UPDT - 04/06/92, 2 fields
STAT - Oral RfD Assessment (RDO) on-line 02/01/90
STAT - Inhalation RfC Assessment (RDI) message 03/01/91
STAT - Carcinogenicity Assessment (CAR) on-line 11/01/90
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 04/01/92
IRH - 03/01/88 RDO Review date added
IRH - 12/01/88 RDO Withdrawn; RfD verified (in preparation)
IRH - 06/01/89 RDO Oral RfD summary replaced; RfD changed
IRH - 06/01/89 REFS Bibliography on-line
IRH - 09/01/89 CAR Carcinogen assessment now under review
IRH - 10/01/89 RDI Inhalation RfD now under review
IRH - 02/01/90 RDO Text edited
IRH - 02/01/90 RDO Text edited
IRH - 06/01/90 RDI Data judged inadequate for derivation of inhalation RfD
IRH - 06/01/90 CAA Area code for EPA contact corrected
IRH - 06/01/90 RCRA EPA contact changed
IRH - 07/01/90 RDI Not verified; data inadequate
IRH - 11/01/90 CAR Carcinogen assessment on-line
IRH - 11/01/90 CREF Carcinogen references added
IRH - 03/01/91 RDI Inhalation RfC message on-line
IRH - 03/01/91 IREF Inhalation RfC references added
IRH - 01/01/92 RDO Secondary contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 04/01/92 CAA CAA regulatory action withdrawn
RLEN - 23761
NAME - Phenol
RN - 108-95-2
SY - Benzenol
SY - Carbolic Acid
SY - Hydroxybenzene
SY - Izal
SY - Monohydroxybenzene
SY - Monophenol
SY - NCI-C50124
SY - Oxybenzene
SY - Phenic Acid
SY - Phenol
SY - Phenyl Alcohol
SY - Phenyl Hydrate
SY - Phenyl Hydroxide
SY - Phenylic Acid
SY - Phenylic Alcohol

RDO -

o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Reduced fetal body weight in rats	NOAEL: 60 mg/kg/day LOAEL: 120 mg/kg/day	100 mg/kg/day	1	6E-1

Rat Oral Developmental Study

NTP, 1983

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*Conversion Factors: none

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o ORAL RFD STUDIES :

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

Developmental effects of phenol were evaluated in timed-pregnant CD rats. Phenol was administered by gavage at 0, 30, 60, and 120 mg/kg/day in distilled water on gestational days 6 to 15. Females were weighed daily during treatment and observed for clinical signs of toxicity. A total of 20 to 22 females/group were confirmed to be pregnant at sacrifice on gestational day 20. Detailed teratological evaluations were conducted at sacrifice. Results of this study did not show any dose-related signs of maternal toxicity or any clinical symptoms of toxicity related to phenol treatment. The number of implantation sites per litter was approximately the same in all groups, as was the number of live fetuses per litter. However, since implantations in this strain take place prior to gestational day 6 (prior to dosing), no relationships between treatment and number of implantation sites can be established. The most important finding, however, was a highly significant reduction in fetal body weights in the high-dose group. The highest fetal NOAEL in this study was 60 mg/kg/day.

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o ORAL RFD UNCERTAINTY :

UF = 100. Uncertainty factor included 10 for interspecies extrapolation and 10 for sensitive human population.

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o ORAL RFD MODIFYING FACTOR :

MF = 1.

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o ORAL RFD COMMENTS :

In NCI (1980) rat and mice 90-day subchronic studies, 10 animals/sex/ group were exposed to 0, 100, 300, 1000, 3000, or 10,000 ppm phenol in water. Decreased water intake and body weight gain were noted for both sexes of rats and mice and rats exposed to the high dose (780 mg/kg/day for rats and 1700 mg/kg/day for mice). Lower doses of phenol exposure did not cause any adverse effects in either rats or mice (234 and 510 mg/kg/day, respectively). The LOAEL for this study was 10,000 ppm.

In a subchronic oral study (Dow, 1945), 10 rats/group were gavaged 5 days/week with 0, 50, or 100 mg/kg (0, 35.7 or 71.4 mg/kg/day) phenol until 135 or 136 doses were administered. Rats in the high-dose group showed a more marked drop in body weight gain than did other groups, but the group rapidly recovered. Rats in both dosage groups showed some degree of unspecific kidney damage yielding a LOAEL of 50 mg/kg, or 5000 ppm, for this study. This difference between the LOAELs of the NCI (1980) and Dow (1945) studies may be attributed to differences in mode of administration, with the Dow gavage study showing the lower LOAEL (possibly explained as a bolus dosage effect).

The Dow research also indicates that the 100% lethal acute dose of phenol is 700 mg/kg (Dow, 1945). In contrast, in a well-designed dose selection study (NCI, 1980) conducted prior to the 2-year bioassay, all rats exposed to 10,000 ppm (780 mg/kg/day) phenol in the drinking water survived a 90-day exposure period. The Dow (1945) study contained several deficiencies, such as limited sample size, lack of details of pertinent experimental design, incomplete histopathological evaluations and unspecific high mortality rate in control and exposed rats during early stages of the study. Therefore, the Dow (1945) study is not considered the best available study for risk assessment.

Other studies indicate no effects on water consumption and weight gain at phenol concentrations as high as 1600 mg/L (1600 ppm) (Deichmann and Oesper, 1940).

In a chronic drinking water study conducted by NCI (1980), rats (F344) and mice (B6C3F1) were dosed with 0, 2500, and 5000 ppm phenol (rats: 0, 153, 344 mg/kg/day; mice: 0, 313, 500 mg/kg/day) in the drinking water for 103 weeks. All the animals were sacrificed 2 weeks after dosing ceased; detailed histopathological and carcinogenic evaluations of target organs were conducted. Results of this bioassay indicated a dose-related depression in mean body weight gain in both sexes of mice and rats. Animals exposed to both dose levels of phenol showed a significant drop in water consumption (water consumption in mice was severely depressed) resulting in significant body weight depression in the high-dose animals. This study also reported an increased incidence of chronic kidney inflammation in all dosed female rats and in the 5000-ppm male rats. The incidence of this lesion in females was: 7/50 (control); 13/50 (2500 ppm); 37/50 (5000 ppm), whereas in male rats the incidence was: 37/50 (control); 37/50 (2500 ppm) and 48/50 (5000 ppm). However, historical control data (Armed Forces Institute of Pathology, 1980) in the F344 rat indicated nephropathy that approaches an incidence of 100%. These rats were the same (comparable) age as the rats killed at the completion of this 2-year NCI (1980) study. In the absence of other toxicological

parameters, such as mortality, percent survival, clinical signs of toxicity, and morphological alterations in target organs, the reduction in body weight in both high-dose mice and rats could be related to depressed water intake resulting from phenol exposure. Based on the body weight depression in both exposed mice and rats, the LOAELs in mice and rats, respectively, were 313 and 344 mg/kg/day and the NOAEL in rats was 153 mg/kg/day. A NOAEL for mice was not observed.

Heller and Pursell (1938) reported normal growth and reproduction at phenol concentrations up to 5000 mg/L (400 mg/kg/day) in a multi-generation rat reproduction study.

In a mouse developmental toxicity study (NTP, 1983). phenol was administered by gavage at 0, 70, 140, or 280 mg/kg/day on gestational days 6 to 15. At the highest dose, 4/36 mice died; no deaths occurred in any other groups. Average maternal body weight gain and weight gain in survivors also were significantly reduced at the highest dose; significant clinical signs of toxicity (tremors) also were seen at that dose level. As in the rat study, there was a highly significant dose-related for reduced fetal body weight, statistically different from controls at the highest dose level. An increased incidence of cleft palate was also reported at the highest dose level. The highest NOAEL in this study was 140 mg/kg/day.

In an unpublished developmental toxicity study, Kavlock (1987) gavaged SD rats with phenol at doses of 0, 667, and 1000 mg/kg on gestational day 11; the females were allowed to deliver and postnatal weight, viability, and function were evaluated. Pup body weights at weaning was decreased in the 1000 mg/kg/day group; kidney weight decreased only in female pups at weaning (667 and 1000 mg/kg groups). On days 8 and 9 postnatally, pup kidney weights were increased at both dosages of phenol, while urine osmolality was decreased and urine volume was increased at 1000 mg/kg. The most striking findings were limb abnormalities (paralysis and palsy) produced by phenol (667 and 1000 mg/kg groups) that were evident 10-14 days after birth. The LOAEL in this study was 667 mg/kg/day.

In summary, the evaluations of subchronic, chronic and reproductive/ developmental studies indicated that phenol administered to pregnant rats at 120 mg/kg/day caused significant depression in fetal body weights, establishing this endpoint as the critical effect. Therefore, it is inappropriate to use NOAELs of 140 mg/kg/day for mice (NTP, 1983) or 153 mg/kg/day for rats (NCI, 1980). The LOAEL for fetotoxicity was established at 120 mg/kg/day and the highest NOAEL at 60 mg/kg/day (NTP, 1983).

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o ORAL RFD CONFIDENCE :

Study: Low
Data Base: Medium
RfD: Low

Confidence in the study is low because of the gavage nature of the dose

administration. The data base contains several supporting studies (subchronic, chronic, and reproductive/developmental); thus, a medium confidence is recommended. Low-to-medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

U.S. EPA. 1985. Health and Environmental Effects Profile for Phenol. Errata, 1986. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

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o REVIEW DATES : 08/05/85, 10/28/86, 11/16/88, 03/22/89
o VERIFICATION DATE : 11/16/88
o EPA CONTACTS :

Harlal Choudhury / ORD -- (513)569-7536 / FTS 684-7536

Chris Cubbison / ORD -- (513)569-7553 / FTS 684-7553

RDI -
o INHALATION RFD SUMMARY :

The health effects data for phenol have been reviewed by the U.S. EPA RfD/RfC Work Group and determined to be inadequate for derivation of an inhalation RfC. The verification status of this chemical is currently not verifiable. For additional information on the health effects of this chemical, interested parties are referred to the EPA documentation listed below.

U.S. EPA. 1986. Summary Review of the Health Effects Associated with Phenol: Health Issue Assessment. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA/600/8-86/003F.

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o REVIEW DATES : 02/22/90
CAREV-
o CLASSIFICATION : D; not classifiable as to human carcinogencity
o BASIS FOR CLASSIFICATION : Based on no human carcinogenicity data and inadequate animal data.
o HUMAN CARCINOGENICITY DATA :

None.

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o ANIMAL CARCINOGENICITY DATA :

Inadequate. In carcinogenicity bioassays conducted by the National Cancer Institute (NCI, 1980), B6C3F1 mice (50/sex/dose) and F344 rats (50/sex/dose) were administered analytical grade phenol (approximately 98.5% pure) in the drinking water at concentrations of 0, 2500 or 5000 ppm for 103 weeks. Dose-related decreases in weight gain in treated mice were attributed to decreased water consumption. No other clinical signs of toxicity were observed, and mortality rates (approximately 14%) were comparable between experimental and control groups. Histopathological examination and statistical analyses revealed no phenol-related toxic or carcinogenic effects in mice.

At the end of the study the survival rate of male rats was comparable among the three groups (approximately 52%) and the survival rate among the female rat groups was comparable (approximately 76%). No trends in cancer incidence were seen when compared with controls, however, low-dose male rats had, by pair-wise comparison, a statistically significant increase in the incidences of pheochromocytomas of the adrenal medulla (13/50, 22/50 and 9/50 in the control, low-, and high-dose groups, respectively), interstitial cell tumors of the testes (42/48, 49/50 and 47/50), and leukemias or lymphomas (18/50, 31/50 and 25/50). There was no significant increase in tumor incidence in any tissue in female rats. Based on a high spontaneous tumor rate in matched controls, comparable survival patterns with no major fall off, and the lack of a positive association between phenol administration and tumor incidence in high-dose male rats, NCI concluded that, under these conditions, phenol was not carcinogenic in mice or rats (NCI, 1980).

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o SUPPORTING DATA :

Studies indicate that phenol may be a promoter and/or weak skin carcinogen in specially inbred sensitive mouse strains. Boutwell and Bosch (1959) demonstrated that repeated dermal applications of phenol promoted the development of skin papillomas and carcinomas in Sutter, Holtzman, CHF1, and C3H mouse strains exposed to a single dermal application of an initiator, 7,12-dimethylbenz[a]anthracene (DMBA, 75 ug). In this series of studies, groups of 23 to 30 mice/sex were treated twice a week for up to 72 weeks with equivalent volumes of benzene- or acetone-based solutions containing 10% phenol. Housing conditions were not described. Papillomas first appeared at 6 weeks and a 95% response had occurred by week 13; carcinomas first appeared at 19 weeks with a 73% response by week 42. In mice receiving only the 10% phenol treatments (no initiator), 4% of the mice had papillomas at week 12 and 36% had papillomas at week 32. The incidence of carcinomas was not reported. In the same series of studies, groups of 30 female mice/dose received twice-weekly dermal applications of 5, 10 or 20% phenol in benzene after an initial treatment of benzene (control) or benzene with 75 ug DMBA. In the noninitiated groups (those receiving only the dermal phenol applications) the percentage of mice bearing papillomas was 74, 100 and 100% in the 5, 10 and 20% phenol treatment groups, respectively, and in the groups receiving the initial DMBA application, 56, 95 and 90% of the mice bore papillomas in the 5, 10 and 20% treatment groups, respectively. Papillomas occurred in 11% of the mice treated with benzene alone. The percentage of mice bearing carcinomas (between weeks 38 and 40) in the noninitiated groups was 26, 93 and 70% in the

5, 10 and 20% phenol groups. In the groups receiving the initial DMBA application, the percentage of mice bearing carcinomas was 12, 68 and 65% in the 5, 10 and 20% phenol groups. No carcinomas were reported in the group receiving only benzene.

Similar results were obtained by Salaman and Glendenning (1957). "S" strain albino mice (20 mice/group) showed strong tumor-promoting activity after initiation with 0.15% DMBA and subsequent, repeated weekly applications of 5 or 20% phenol (w/v in acetone) for 24 to 32 weeks. At the 20% level, phenol induced ulceration of the skin and had a strong promoting effect on tumor induction. At the 0.5% level, no ulceration was found; phenol had a moderate promoting effect but did not act as an initiator. Housing conditions of the animals were not indicated.

Analytical grade phenol (99.9% pure) (up to 10 mg/plate) was not mutagenic in Salmonella typhumurium strains TA98, TA100, TA1535, TA1537, or TA1538 with or without addition of rat liver homogenates (Florin et al., 1980; Pool and Lin, 1982; Haworth et al., 1983). However, Gocke et al. (1981) reported that phenol was mutagenic in TA98 with hepatic homogenates. Phenol was not mutagenic in Neurospora crassa (Dickey et al., 1949) and was not positive in the micronucleus test on mouse bone marrow from male and female NMRI mice treated in vivo (Gocke et al., 1981). In a study by Demerec et al. (1951), phenol exhibited mutagenic activity in Escherichia coli but only at highly toxic concentrations (0.1-0.2%).

CARDR-
o CARCINOGENICITY SOURCE :

U.S. EPA. 1988. Updated Health Effects Assessment for Phenol. Prepared by the Office of Health and Environment Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

The 1988 Health Effects Assessment for Phenol has received Agency review.
DOCUMENT

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o REVIEW DATES : 08/02/89
o VERIFICATION DATE : 08/02/89
o EPA CONTACTS :

Charli Hiremath / ORD -- (202)260-5725 / FTS 260-5725

WQCHU-

Water and Fish Consumption: 3.5E+3 ug/L

Fish Consumption Only: None

Considers technological or economic feasibility? -- NO

Discussion -- To control undesirable taste and odor qualities of ambient water, the estimated concentration is 3.0E+2 ug/L. There is no demonstrated relationship between organoleptic endpoints and adverse health effects. If there is significant chlorination of water containing phenol, reference should be made to the criteria for 2-chlorophenol, and 2,4-dichlorophenol.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC -- 1.02E+4 ug/L
Chronic LEC -- 2.56E+3 ug/L

Marine:

Acute LEC -- 5.8E+3 ug/L
Chronic -- None

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

FISTD-

Status -- List "D" Pesticide (1989)

Reference -- 54 FR 43388 (10/24/89)

EPA Contact -- Registration Branch / OPP
(703)557-7760 / FTS 557-7760

FIREV-

No data available

CERC -

Value (status) -- 1000 pounds (Final, 1986)

Considers technological or economic feasibility? -- NO

Discussion-- The final RQ takes into account the natural biodegradation and photolysis of this hazardous substance. The biological oxygen demand in 5 days (BOD5) is between 58-83% of the theoretical oxygen demand. The lowest primary RQ adjustment criteria for phenol (100 pounds based on chronic toxicity composite score of 35) has been adjusted upward one RQ level.

Reference -- 51 FR 34534 (09/29/86); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Deichmann, W. and P. Oesper. 1940. Ingestion of phenol-effects on the albino rat. Ind. Med. 9: 296.
OREF - Dow Chemical Co. 1945. The toxicity of phenol. Biochem. Res. Lab. Unpublished report dated 04/12/45.
OREF - Heller, V.G. and L. Pursell. 1938. J. Pharmacol. Exp. Ther. 63: 99. (Cited in Deichmann and Oesper, 1940)
OREF - Kavlock, R.J. 1987. Interim Report on Structure-Activity Relationships in the Developmental Toxicity of Substituted Phenols. Health Effects Research Laboratory, Research Triangle Park, NC.
OREF - NCI (National Cancer Institute). 1980. Bioassay of phenol for possible carcinogenicity in F344 rats and B6C3F1 mice. NIH Publ. No. 80-1759. August 1980.
OREF - NTP (National Toxicology Program). 1983. Teratologic evaluation of phenol in CD rats and mice. Report prepared by Research Triangle Institute, Research Triangle Park, NC. NTIS PB83-247726. Gov. Rep. Announce. Index. 83(25): 6247.
OREF - U.S. EPA. 1985. Health and Environmental Effects Profile for Phenol. Errata, 1986. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.
IREF - U.S. EPA. 1986. Summary Review of the Health Effects Associated with Phenol: Health Issue Assessment. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA/600/8-86/003F.
CREF - Boutwell, R.K. and D.K. Bosch. 1959. The tumor-promoting action of phenol and related compounds for mouse skin. Cancer Res. 19: 413-424.
CREF - Demerec, M., G. Bertani and J. Flint. 1951. A survey of chemicals for mutagenic action on E. coli. Am. Natur. 85(821): 119-135.
CREF - Dickey, F.H., G.H. Cleland and C. Lotz. 1949. The role of organic peroxides in the induction of mutations. Proc. Natl. Acad. Sci. 35: 581-586.
CREF - Florin, I., L. Rutberg, M. Curvall and C.R. Enzell. 1980. Screening of tobacco smoke constituents for mutagenicity using the Ames test. Toxicology. 18: 219-232.
CREF - Gocke, E., M.-T. King, K. Eckhardt and D. Wild. 1981. Mutagenicity of cosmetics ingredients licensed by the European communities. Mutat. Res. 90: 91-109.
CREF - Haworth, S., T. Lawlor, K. Mortelmans, W. Speck and E. Zeiger. 1983. Salmonella mutagenicity test results for 250 chemicals. Environ. Mutagen.1Suppl. 1: 3-142.
CREF - NCI (National Cancer Institute). 1980. Bioassay of phenol for possible carcinogenicity. Prepared by the National Cancer Institute, Bethesda, MD for the National Toxicology Program, Research Triangle Park, NC. NCI-CG-TR-203, DHHS/PUB/NIH80-1759.
CREF - Pool, B.L. and P.Z. Lin. 1982. Mutagenicity testing in the Salmonella typhimurium assay of phenolic compounds and phenolic

fractions obtained from smokehouse smoke condensates. Food Chem. Toxicol. 20: 383-391.
CREF - Salaman, M.H. and O.M. Glendenning. 1957. Tumor promotion in mouse skin by sclerosing agents. Br. J. Cancer. 11: 434-444.
CREF - U.S. EPA. 1988. Updated Health Effects Assessment for Phenol. Prepared by the Office of Health and Environment Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.
HAREF- None

[IRIS] SS 11 /cf?
USER:
470-90-6
Search in progress

NP (470-90-6 (IRIS))
*NONE-

[IRIS] SS 11 /cf?
USER:
121-82-4
Search in progress

SS (11) PSTG (1)

[IRIS] SS 12 /cf?
USER:

prt dl ncar, car continuous

1 - IRIS
IRSN - 5
DATE - 920120
UPDT - 01/20/92, 52 fields
STAT - Oral RfD Assessment (RDO) on-line 02/01/91
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) no data
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 08/01/89 RDO Gross et al. (1955) citation clarified
IRH - 08/01/89 REFS Bibliography on-line
IRH - 02/01/91 RDO Conversion factor revised
IRH - 02/01/91 RDO Text added
IRH - 02/01/91 RDO Paragraph 2 added
IRH - 02/01/91 OREF Dunn, 1928 and Monier-Williams, 1934 added
IRH - 01/01/92 RDO Primary contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
RLEN - 13562
NAME - Antimony
RN - 7440-36-0
SY - Antimony
SY - ANTIMONY BLACK
SY - ANTIMONY POWDER
SY - ANTIMONY, REGULUS
SY - ANTYMON
SY - C.I. 77050
SY - STIBIUM
SY - UN 2871

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Longevity, blood glucose, and cholesterol	NOEL: none LOAEL: 0.35 mg/kg bw/day	1000	1	4E-4 mg/kg/day

Rat Chronic Oral

Bioassay

Schroeder et al., 1970

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*Conversion Factors: 5 mg/L (5 ppm) given as 0.350 mg/kg/day in the discussion section of the critical study

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o ORAL RFD STUDIES :

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

An experimental group of 50 male and 50 female rats was administered 5 ppm potassium antimony tartrate in water. Over the period of study, growth rates of treated animals were not affected, but male rats survived 106 and females 107 fewer days than did controls at median lifespans. Nonfasting blood glucose levels were decreased in treated males, and cholesterol levels were altered in both sexes. Since there was only one level of antimony administered, a NOEL was not established in this study. A decrease in mean heart weight for the males was noted. No increase in tumors was seen as a result of treatment. Although not precisely stated, the concentration of 5 ppm antimony was expressed as an exposure of 0.35 mg/kg/day by the authors.

---

o ORAL RFD UNCERTAINTY :

UF = 1000. An uncertainty factor of 1000 (10 for interspecies conversion, 10 to protect sensitive individuals, and 10 because the effect level was a LOAEL and no NOEL was established) was applied to the LOAEL of 0.35 mg/kg bw/day.

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o ORAL RFD MODIFYING FACTOR :

MF = 1

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o ORAL RFD COMMENTS :

In a similar study (Kanisawa and Schroeder, 1969), groups of CD-1 mice (54/sex) were given potassium antimony tartrate in drinking water at 0 or 5 mg/L (5 ppm) for 540 days (18 months). Lifespans were significantly reduced in both males and females, but the degree of antimony toxicity was less severe in mice than rats. Bradley and Fredrick (1941) and Browning (1969) reported disturbances in glucose and cholesterol metabolism in rats ingesting 5 mg/L antimony, but no signs of injury to the heart were observed in rats receiving doses up to 100 mg/kg/day. Substantially higher doses of antimony trioxide were tolerated by rats in studies by Sunagawa (1981) and Gross et al. (1955a,b), suggesting a NOAEL of 500 mg/kg, but these studies are of inadequate duration to assess adverse effects on toxicity.

Seventy people became acutely ill after drinking lemonade containing 0.013% antimony (Dunn, 1928 and Monier-Williams, 1934). The lemonade had been prepared and left overnight in buckets coated with an enamel containing 2.88% antimony trioxide. Fifty-six people were taken to the hospital with burning stomach pains, colic, nausea and vomiting. Most recovered within 3 hours, but in some cases recovery was not complete for several days. It is estimated that a person consuming 300 mL of lemonade would have received a dose of approximately 36 mg antimony, or approximately 0.5 mg/kg for a 70-kg adult.

According to U.S. EPA (1980), multimedia antimony exposures are essentially negligible by comparison to occupational exposures at which discrete clinical health effects have been observed. Myocardial effects are among the best-characterized human health effects associated with antimony exposure. Studies by Brieger et al. (1954) suggest an inhalation NOEL for myocardial damage to be approximately 0.5 mg/cu.m. This exposure

is
approximately equivalent to an oral reference dose of 0.003 mg/kg bw/day
(i.e., 0.5 mg/cu.m x 10 cu.m/day x 0.5 / 1.0 x 5 days/7 days / 70 kg / 10).
Parallel studies in rats and rabbits resulted in observation of EKG

alterations following exposure to 3.1-5.6 mg/cu.m. There are, however, no
adequate data on oral exposure to antimony which permit reasonable estimate of
no effect levels regarding heart damage.

One study (Belyaeva, 1967) indicated that women workers exposed in an
antimony plant experienced a greater incidence of spontaneous abortions than
did a control group of nonexposed working women. A high rate of premature
deliveries among women workers in antimony smelting and processing was also
observed (Aiello, 1955).

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o ORAL RFD CONFIDENCE :

Study: Low
Data Base: Low
RfD: Low

Confidence in the chosen study is rated as low because only one species
was used, only one dose level was used, no NOEL was determined, and gross
pathology and histopathology were not well described. Confidence in the data
base is low due to lack of adequate oral exposure investigations. Low
confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

U.S. EPA. 1980. Ambient Water Quality Criteria Document for Antimony.
Prepared by the Office of Health and Environmental Assessment, Environmental
Criteria and Assessment Office, Cincinnati, OH for the Office of Water
Regulations and Standards, Washington, DC. EPA-440/5-80-020. NTIS PB 81-
117319.

The ADI in the 1980 Ambient Water Quality Criteria Document was extensively
reviewed by the Agency and was reviewed by the public.

U.S. EPA. 1985. Health and Environmental Effects Profile for Antimony
Oxides. Prepared by the Office of Health and Environmental Assessment,
Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of
Solid Waste and Emergency Response, Washington, DC.

Limited peer review and extensive Agency-wide review, 1985.

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o REVIEW DATES : 11/06/85
o VERIFICATION DATE : 11/06/85
o EPA CONTACTS :

Harlal Choudhury / ORD -- (513)569-7553 / FTS 684-7553

Michael L. Dourson / ORD -- (513)569-7544 / FTS 684-7544

WQCHU-

Water and Fish Consumption: 1.46E+2 ug/L

Fish Consumption Only: 4.5E+4 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of 1.46E+2 ug/L is based on consumption of contaminated
aquatic organisms and water. A WQC of 4.5E+4 ug/L has also been established
based
on consumption of contaminated aquatic organisms alone.

Reference -- 45 FR 79315 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 8.8E+1 ug/L
Chronic -- 3.0E+1 ug/L

Marine:

Acute -- 1.5E+3 ug/L
Chronic -- 5.0E+2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register. The proposed values are based on studies of antimony (III).

Reference -- 55 FR 19986 (05/14/89)

EPA Contact -- Criteria and Standards Division / OWRS (202)260-1315 / FTS 260-1315

MCLG -

Value -- 0.003 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- NO

Discussion -- EPA is proposing to regulate antimony based on its potential adverse effects (decreased longevity and altered blood cholesterol and glucose) reported in a lifetime oral exposure study in rats. The MCLG is based upon a DWEL of 0.015 mg/L and an assumed drinking water contribution of 20 percent.

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Health and Ecological Criteria Division / OST / (202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 0.01 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- YES

Discussion -- EPA proposes an MCL of 0.01 mg/L based upon a PQL of 10x the MDL. EPA also proposes as an alternative option an MCL of 0.005 based on a PQL of 5x the MDL.

Monitoring requirements -- Ground water systems every 3 years; surface water systems annually; will allow monitoring at up to 10-year intervals after the system completes 3 rounds of sampling at <50% of the MCL.

Analytical methodology -- Atomic absorption/furnace technique (EPA 204.2; SM 304); ICP mass spectrometry (EPA 200.8); hydride-atomic absorption spectrometry (ASTM D-3697): PQL= 0.01 / 0.005 mg/L.

Best available technology -- Coagulation/filtration; reverse osmosis.

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

Status -- Listed (Final, 1991)

Discussion -- "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement -- All systems to be monitored unless a vulnerability assessment determines the system is not vulnerable.

Analytical methodology -- Atomic absorption/furnace technique (EPA 502.2; SM 304); inductively coupled plasma (EPA 200.8).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

CERC -

Value (status) -- 5000 lbs (Final, 1986)

Considers technological or economic feasibility? -- NO

Discussion -- No data have been found to permit the ranking of this hazardous substance. The available data for acute hazards may lie above the upper limit for the 5000-pound RQ, but since it is a designated hazardous substance, the largest assignable RQ is 5000 pounds. This chemical is currently being assessed for chronic toxicity and is subject to change in future rulemaking.

Reference -- 51 FR 34534 (09/29/86); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline (800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Aiello, G. 1955. Pathology of antimony. Folia Med. (Naples). 38:
100. (Ital.)
OREF - Belyaeva, A.P. 1967. The effect of antimony on reproduction. Gig.
Truda Prof. Zabol. 11: 32.
OREF - Bradley, W.R. and W.G. Frederick. 1941. The toxicity of
antimony--animal studies. Ind. Med. 10 Ind. Hyg. Sec. 2: 15-22.
OREF - Brieger, H., C.W. Semisch, III, J. Stasney and D.A. Platnek. 1954.
Industrial antimony poisoning. Ind. Med. Surg. 23: 521.
OREF - Browning, E. 1969. Antimony. In: Toxicity of Industrial Metals, 2nd
ed. Appleton-Century-Craft, New York. p. 23-38.
OREF - Dunn, J.T. 1928. A curious case of antimony poisoning. Analyst. 53:
532-533.
OREF - Gross, P., J.H.V. Brown, M.L. Westrick, R.P.Srsic, N.L. Butler and
T.F. Hatch. 1955a. A toxicological study of calcium halophosphate
phospher and antimony trioxide. I. Acute and chronic toxicity and

some pharmacological aspects. Arch. Ind. Health. 11: 473-479.
OREF - Gross, P., M.L. Westrick, J.H.V. Brown, R.P. Srsic, H.H. Schrenk
and T.F. Hatch. 1955b. Toxicologic study of calcium halophosphate
phosphors and antimony trioxide. II. Pulmonary studies. Arch. Ind.
Health. 11: 479-486.
OREF - Kanisawa, M. and H.A. Schroeder. 1969. Life term studies on the
effect of trace elements on spontaneous tumor in mice and rats.
Cancer Res. 29: 892-895.
OREF - Monier-Williams, G.W. 1934. Antimony in enamelled hollow-ware.
Report on Public Health and Medical Subjects, No. 73, Ministry of
Health, London. p. 18. (Cited in U.S. EPA, 1985)
OREF - Schroeder, H.A., M. Mitchner and A.P. Nasor. 1970. Zirconium,
niobium, antimony, vanadium and lead in rats: Life term studies. J.
Nutr. 100(1): 59-68.
OREF - Sunagawa, S. 1981. Experimental studies on antimony poisoning.
Igaku Kenkyu. 51(3): 129-142. (Jap.) (CA 096/080942D)
OREF - U.S. EPA. 1980. Ambient Water Quality Criteria Document for
Antimony. Prepared by the Office of Health and Environmental
Assessment, Environmental Criteria and Assessment Office,
Cincinnati, OH for the Office of Air Quality Planning and
Standards, Washington, DC. EPA 440/5-80-020.
OREF - U.S. EPA. 1985. Health and Environmental Effects Profile for
Antimony Oxides. Prepared by the Office of Health and Environmental
Assessment, Environmental Criteria and Assessment, Cincinnati, OH
for the Office of Solid Waste and Emergency Response, Washington,
DC.
IREF - None
CREF - None
HAREF- None

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7440-38-2
Search in progress

SS (13) PSTG (1)

[IRIS] SS 14 /cf?
USER:

prt dl ncar, car continuous

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STAT - Oral RfD Assessment (RDO) on-line 02/01/93
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 06/01/94
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 06/30/88 CARO Revised last paragraph
IRH - 06/30/88 CARI Inhalation slope factor changed
IRH - 06/30/88 CARI Paragraph 2 added
IRH - 09/07/88 CARO Major text changes
IRH - 12/01/88 CAREV Mabuchi et al. citation year corrected
IRH - 12/01/88 CAREV Pershagen et al. citation year corrected
IRH - 09/01/89 CARI Citations added to anacondor smelter
IRH - 09/01/89 REFS Bibliography on-line
IRH - 06/01/90 CAREV 2nd & 3rd paragraph - Text revised
IRH - 06/01/90 CAREV Text corrected
IRH - 06/01/90 CARI Inhalation slope factor removed (format change)
IRH - 06/01/90 RCRA EPA contact changed
IRH - 06/01/90 CREF References added
IRH - 12/01/90 CARO Changed slope factor to "unit risk", 2nd para, 1st sen
IRH - 02/01/91 CARI Text edited
IRH - 09/01/91 RDO Oral RfD summary now on-line
IRH - 09/01/91 RDO Oral RfD bibliography added
IRH - 10/01/91 RDO Conversion factor text clarified
IRH - 10/01/91 MCLG MCLG noted as pending change
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 08/01/92 CAR Note added to indicate text in oral quant. estimate
IRH - 10/01/92 CREF Missing reference added to bibliography
IRH - 02/01/93 RDO Citations added to second paragraph
IRH - 02/01/93 OREF References added to bibliography
IRH - 03/01/93 OREF Corrections to references
IRH - 03/01/94 CARDR Work group review date added
IRH - 06/01/94 CAR Carcinogen assessment noted as pending change
RLEN - 39437
NAME - Arsenic, inorganic
RN - 7440-38-2
SY - Arsenic
SY - Arsenic, inorganic
SY - gray-arsenic

RDO -
o ORAL RFD SUMMARY :

NOTE: There was not a clear consensus among Agency scientists on the oral RfD. Applying the Agency's RfD methodology, strong scientific arguments can be made for various values within a factor of 2 or 3 of the currently recommended RfD value, i.e., 0.1 to 0.8 ug/kg/day. It should be noted, however, that the RfD methodology, by definition, yields a number with inherent uncertainty spanning perhaps an order of magnitude. New data that possibly impact on the recommended RfD for arsenic will be evaluated by the Work Group as it becomes available. Risk managers should recognize the considerable flexibility afforded them in formulating regulatory decisions when uncertainty and lack of clear consensus are taken into account.

Critical Effect	Experimental Doses*	UF	MF	RfD
Hyperpigmentation, keratosis and possible vascular complications Human chronic oral exposure	NOAEL: 0.009 mg/L converted to 0.0008 mg/kg-day LOAEL: 0.17 mg/L converted to 0.014 mg/kg-day	3	1	3E-4 mg/kg-day

Tseng, 1977;
Tseng et al., 1968

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*Conversion Factors: NOAEL was based on an arithmetic mean of 0.009 mg/L in a range of arsenic concentration of 0.001 to 0.017 mg/L. This NOAEL also included estimation of arsenic from food. Since experimental data were missing, arsenic concentrations in sweet potatoes and rice were estimated as 0.002 mg/day. Other assumptions included consumption of 4.5 L water/day and 55 kg bw (Abernathy et al., 1989). NOAEL = [(0.009 mg/L x 4.5 L/day) + 0.002 mg/day] / 55 kg = 0.0008 mg/kg-day. The LOAEL dose was estimated using the same assumptions as the NOAEL starting with an arithmetic mean water concentration from Tseng (1977) of 0.17 mg/L. LOAEL = [(0.17 mg/L x 4.5 L/day) + 0.002 mg/day] / 55 kg = 0.014 mg/kg-day.

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o ORAL RFD STUDIES :

Tseng, W.P. 1977. Effects and dose-response relationships of skin cancer and blackfoot disease with arsenic. Environ. Health Perspect. 19: 109-119.

Tseng, W.P., H.M. Chu, S.W. How, J.M. Fong, C.S. Lin and S. Yeh. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. J. Natl. Cancer Inst. 40: 453-463.

The data reported in Tseng (1977) show an increased incidence of blackfoot disease that increases with age and dose. Blackfoot disease is a significant adverse effect. The prevalences (males and females combined) at the low dose are 4.6 per 1000 for the 20-39 year group, 10.5 per 1000 for the 40-59 year group, and 20.3 per 1000 for the >60 year group. Moreover, the prevalence of

blackfoot disease in each age group increases with increasing dose. However, a recent report indicates that it may not be strictly due to arsenic exposure (Lu, 1990). The data in Tseng et al. (1968) also show increased incidences of hyperpigmentation and keratosis with age. The overall prevalences of hyperpigmentation and keratosis in the exposed groups are 184 and 71 per 1000, respectively. The text states that the incidence increases with dose, but data for the individual doses are not shown. These data show that the skin lesions are the more sensitive endpoint. The low dose in the Tseng (1977) study is considered a LOAEL.

The control group described in Tseng et al. (1968; Table 3) shows no evidence of skin lesions and presumably blackfoot disease, although this latter point is not explicitly stated. This group is considered a NOAEL.

The arithmetic mean of the arsenic concentration in the wells used by the individuals in the NOAEL group is 9 ug/L (range: 1-17 ug/L) (Abernathy et al., 1989). The arithmetic mean of the arsenic concentration in the wells used by the individuals in the LOAEL group is 170 ug/L (Tseng, 1977; Figure 4). Using estimates provided by Abernathy et al. (1989), the NOAEL and LOAEL doses for both food and water are as follows: LOAEL - [170 ug/L x 4.5 L/day + 2 ug/day (contribution of food)] x (1/55 kg) = 14 ug/kg/day; NOAEL - [9 ug/L x 4.5 L/day + 2 ug/day (contribution of food)] x (1/55 kg) = 0.8 ug/kg/day.

Although the control group contained 2552 individuals, only 957 (approximately 38%) were older than 20, and only 431 (approximately 17%) were older than 40. The incidence of skin lesions increases sharply in individuals above 20; the incidence of blackfoot disease increases sharply in individuals above 40 (Tseng, 1968; Figures 5, 6 and 7). This study is less powerful than it appears at first glance. However, it is certainly the most powerful study available on arsenic exposure to people.

This study shows an increase in skin lesions, 22% (64/296) at the high dose vs. 2.2% (7/318) at the low dose. The average arsenic concentration in the wells at the high dose is 410 ug/L and at the low dose is 5 ug/L (Cebrian et al., 1983; Figure 2 and Table 1) or 7 ug/L (cited in the abstract). The average water consumption is 3.5 L/day for males and 2.5 L/day for females. There were about an equal number of males and females in the study. For the dose estimates given below we therefore assume an average of 3 L/day. No data are given on the arsenic exposure from food or the body weight of the participants (we therefore assume 55 kg). The paper states that exposure times are directly related to chronological age in 75% of the cases. Approximately 35% of the participants in the study are more than 20 years old (Figure 1).

Exposure estimates (water only) are: high dose - 410 ug/L x 3 L/day x (1/55 kg) = 22 ug/kg/day; low dose - 5-7 ug/L x 3 L/day x (1/55 kg) = 0.3-0.4 ug/kg/day.

The high-dose group shows a clear increase in skin lesions and is therefore designated a LOAEL. There is some question whether the low dose is a NOAEL or

a LOAEL since there is no way of knowing what the incidence of skin lesions would be in a group where the exposure to arsenic is zero. The 2.2% incidence of skin lesions in the low-dose group is higher than that reported in the Tseng et al. (1968) control group, but the dose is lower (0.4 vs. 0.8 ug/kg/day).

The Southwick et al. (1983) study shows a marginally increased incidence of a variety of skin lesions (palmar and plantar keratosis, diffuse palmar or plantar hyperkeratosis, diffuse pigmentation, and arterial insufficiency) in the individuals exposed to arsenic. The incidences are 2.9% (3/105) in the control group and 6.3% (9/144) in the exposed group. There is a slight, but not statistically significant increase in the percent of exposed individuals that have abnormal nerve conduction (8/67 vs. 13/83, or 12% vs. 16% (Southwick et al., 1983; Table 8). The investigators excluded all individuals older than 47 from the nerve conduction portion of the study. These are the individuals most likely to have the longest exposure to arsenic.

Although neither the increased incidence of skin lesions nor the increase in abnormal nerve conduction is statistically significant, these effects may be biologically significant because the same abnormalities occur at higher doses in other studies. The number of subjects in this study was insufficient to establish statistical significance.

Table 3 (Southwick et al., 1983) shows the annual arsenic exposure from drinking water. No data are given on arsenic exposure from food or the body weight (assume 70 kg). Exposure times are not clearly defined, but are >5 years, and dose groups are ranges of exposure.

Exposure estimates (water only) are: dosed group - 152.4 mg/year x 1 year/365 days x (1/70) kg = 6 ug/kg/day; control group - 24.2 mg/year x year/365 days x (1/70) kg = 0.9 ug/kg/day.

Again because there are no data for a group not exposed to arsenic, there is some question if the control group is a NOAEL or a LOAEL. The incidence of skin lesions in this group is about the same as in the low-dose group from the Cebrian et al. (1983) study; the incidence of abnormal nerve conduction in the control group is higher than that from the low-dose group in the Hindmarsh et al. (1977) study described below. The control dose is comparable to the dose to the control group in the Tseng et al. (1968) and Hindmarsh et al. (1977) studies. The dosed group may or may not be a LOAEL, since it is does not report statisically significant effects when compared to the control.

This study shows an increased incidence of abnormal clinical findings and abnormal electromyographic findings with increasing dose of arsenic (Hindmarsh et al., 1977; Tables III and VI). However, the sample size is extremely small. Percentages of abnormal clinical signs possibly attributed to As were 10, 16, and 40% at the low, mid and high doses, respectively. Abnormal EMG were 0, 17 and 53% in the same three groups.

The exact doses are not given in the Hindmarsh et al. (1977) paper; however,

some well data are reported in Table V. The arithmetic mean of the arsenic concentration in the high-dose and mid-dose wells is 680 and 70 ug/L, respectively. Figure 1 (Hindmarsh et al., 1977) shows that the average arsenic concentration of the low-dose wells is about 25 ug/L. No data are given on arsenic exposure from food. We assume daily water consumption of 2 liters and body weight of 70 kg. Exposure times are not clearly stated.

Exposure estimates (water only) are: low - 25 ug/L x 2 L/day x (1/70) kg = 0.7 ug/kg/day; mid - 70 ug/L x 2 L/day x (1/70) kg = 2 ug/kg/day; high - 680 ug/L x 2 L/day x (1/70) kg = 19 ug/kg/day.

The low dose is a no-effect level for abnormal EMG findings. However, because there is no information on the background incidence of abnormal clinical findings in a population with zero exposure to arsenic, there is no way of knowing if the low dose is a no-effect level or another marginal effect level for abnormal clinical findings. The low dose is comparable to the dose received by the control group in the Tseng (1977) and Southwick et al. (1983) studies.

The responses at the mid dose do not show a statistically significant increase but are part of a statistically significant trend and are biologically significant. This dose is an equivocal NOAEL/LOAEL. The high dose is a clear LOAEL for both responses.

As discussed previously there is no way of knowing whether the low doses in the Cebrian et al. (1983), Southwick et al. (1983) and Hindmarsh et al. (1977) studies are NOAELs for skin lesions and/or abnormal nerve conduction. However, because the next higher dose in the Southwick and Hindmarsh studies only shows marginal effects at doses 3-7 times higher, the Agency feels comfortable in assigning the low doses in these studies as NOAELs.

The Tseng (1977) and Tseng et al. (1968) studies are therefore considered superior for the purposes of developing an RfD and show a NOAEL for a sensitive endpoint. Even discounting the people <20 years of age, the control group consisted of 957 people that had a lengthy exposure to arsenic with no evidence of skin lesions.

The following is a summary of the defined doses in mg/kg-day from the principal and supporting studies:

1) Tseng (1977): NOAEL = 8E-4; LOAEL = 1.4E-2

2) Cebrian et al. (1983): NOAEL = 4E-4; LOAEL = 2.2E-2

3) Southwick et al. (1983): NOAEL = 9E-4; LOAEL = none (equivocal effects at 6E-3)

4) Hindmarsh et al., 1977: NOAEL = 7E-4; LOAEL = 1.9E-2 (equivocal effects at 2E-3)

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o ORAL RFD UNCERTAINTY :

UF -- The UF of 3 is to account for both the lack of data to preclude reproductive toxicity as a critical effect and to account for some uncertainty in whether the NOAEL of the critical study accounts for all sensitive individuals.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

Ferm and Carpenter (1968) produced malformations in 15-day hamster fetuses via intravenous injections of sodium arsenate into pregnant dams on day 8 of gestation at dose levels of 15, 17.5, or 20 mg/kg bw. Exencephaly, encephaloceles, skeletal defects and genitourinary systems defects were produced. These and other terata were produced in mice and rats all at levels around 20 mg/kg bw. Minimal effects or no effects on fetal development have been observed in studies on chronic oral exposure of pregnant rats or mice to relatively low levels of arsenic via drinking water (Schroeder and Mitchner, 1971). Nadeenko et al. (1978) reported that intubation of rats with arsenic solution at a dose level of 25 ug/kg/day for a period of 7 months, including pregnancy, produced no significant embryotoxic effects and only infrequent slight expansion of ventricles of the cerebrum, renal pelves and urinary bladder. Hood et al. (1977) reported that very high single oral doses of arsenate solutions (120 mg/kg) to pregnant mice were necessary to cause prenatal fetal toxicity, while multiple doses of 60 mg/kg on 3 days had little effect.

Extensive human pharmacokinetic, metabolic, enzymic and long-term information is known about arsenic and its metabolism. Valentine et al. (1987) established that human blood arsenic levels did not increase until daily water ingestion of arsenic exceeded approximately 250 ug/day (approximately 120 ug of arsenic/L. Methylated species of arsenic are successively 1 order of magnitude less toxic and less teratogenic (Marcus and Rispin, 1988). Some evidence suggests that inorganic arsenic is an essential nutrient in goats, chicks, minipigs and rats (NRC, 1989). No comparable data are available for humans.

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o ORAL RFD CONFIDENCE :

Study -- Medium
Data Base -- Medium
RfD -- Medium

Confidence in the chosen study is considered medium. An extremely large number of people were included in the assessment (>40,000) but the doses were not well-characterized and other contaminants were present. The supporting human toxicity data base is extensive but somewhat flawed. Problems exist

with all of the epidemiological studies. For example, the Tseng studies do not look at potential exposure from food or other source. A similar criticism can be made of the Cebrian et al. (1983) study. The U.S. studies are too small in number to resolve several issues. However, the data base does support the choice of NOAEL. It garners medium confidence. Medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- This assessment is not presented in any existing U.S. EPA document.

This analysis has been reviewed by EPA's Risk Assessment Council on 11/15/90. This assessment was discussed by the Risk Assessment Council of EPA on 11/15/90 and verified through a series of meetings during the 1st, 2nd and 3rd quarters of FY91.

Other EPA Documentation -- U.S. EPA, 1984, 1988

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o REVIEW DATES : 03/24/88, 05/25/88, 03/21/89, 09/19/89, 08/22/90, 09/20/90
o VERIFICATION DATE : 11/15/90
o EPA CONTACTS :

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CAREV-
o CLASSIFICATION : A; human carcinogen
o BASIS FOR CLASSIFICATION : based on observation of increased lung cancer mortality in populations exposed primarily through inhalation and on increased skin cancer incidence in several populations consuming drinking water with high arsenic concentrations. NOTE: The carcinogenicity assessment for arsenic may change in the near future pending the outcome of a further review now being conducted by the Carcinogen Risk Assessment Verification Endeavor Work Group.
o HUMAN CARCINOGENICITY DATA :

Studies of smelter worker populations (Tacoma, WA; Magma, UT; Anaconda, MT; Ronnskar, Sweden; Saganoseki-Machii, Japan) have all found an association between occupational arsenic exposure and lung cancer mortality

(Enterline and Marsh, 1982; Lee-Feldstein, 1983; Axelson et al., 1978; Tokudome and Kuratsune, 1976; Rencher et al., 1977). Both proportionate mortality and cohort studies of pesticide manufacturing workers have shown an excess of lung cancer deaths among exposed persons (Ott et al., 1974; Mabuchi et al., 1979). One study of a population residing near a pesticide manufacturing plant revealed that these residents were also at an excess risk of lung cancer (Matanoski et al., 1981). Case reports of arsenical pesticide applicators have also demonstrated an association between arsenic exposure and lung cancer (Roth, 1958).

A cross-sectional study of 40,000 Taiwanese exposed to arsenic in drinking water found significant excess skin cancer prevalence by comparison to 7500 residents of Taiwan and Matsu who consumed relatively arsenic-free water (Tseng et al., 1968). This study design limited its usefulness in risk estimation. Arsenic-induced skin cancer has also been attributed to water supplies in Chile, Argentina and Mexico (Borgono and Greiber, 1972; Bergoglio, 1964; Cebrian et al., 1983). No excess skin cancer incidence has been observed in U.S. residents consuming relatively high levels of arsenic in drinking water (Morton et al., 1976; Southwick et al., 1981). The results of these U.S. studies, however, are not necessarily inconsistent with the existing findings from the foreign populations. The statistical powers of the U.S. studies are considered to be inadequate because of the small sample size.

A follow-up study (Tseng, 1977) of the population living in the same area of Taiwan, where arsenic contamination of the water supply was endemic, found significantly elevated standard mortality ratios for cancer of the bladder, lung, liver, kidney, skin and colon. This study of bladder, liver and lung cancer cases in the endemic area found a significant association with arsenic exposure that was dose-related. The association of arsenic ingestion and cancer of various internal organs has also been cited in a number of case reports (Chen et al., 1985, 1986). Persons treated with arsenic-containing medicinals have also been shown to be at a risk of skin cancer (Sommers and McManus, 1953).

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o ANIMAL CARCINOGENICITY DATA :

None. There has not been consistent demonstration of arsenic carcinogenicity in test animals for various chemical forms administered by different routes to several species (IARC, 1980). There are some data to indicate that arsenic may produce animal tumors if retention time in the lung can be increased (Pershagen et al., 1982, 1984).

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o SUPPORTING DATA :

Sodium arsenate has been shown to transform Syrian hamster embryo cells (Dipaolo and Casto, 1979) and to produce sister-chromatid-exchange in DON cells, CHO cells and human peripheral lymphocytes exposed in vitro (Wan et al., 1982; Ohno et al., 1982; Larramendy et al., 1981; Andersen, 1983; Crossen, 1983). While arsenic compounds have not been shown to mutate bacterial strains, it produces preferential killing of repair deficient

strains (Rossman, 1981).

CARO -
o CLASSIFICATION : A; human carcinogen
o BASIS FOR CLASSIFICATION : based on observation of increased lung cancer mortality in populations exposed primarily through inhalation and on increased skin cancer incidence in several populations consuming drinking water with high arsenic concentrations. NOTE: The carcinogenicity assessment for arsenic may change in the near future pending the outcome of a further review now being conducted by the Carcinogen Risk Assessment Verification Endeavor Work Group.
o ORAL DOSE-RESPONSE DATA :

The Risk Assessment Forum has completed a reassessment of the carcinogenicity risk associated with ingestion of inorganic arsenic. This report, which has been extensively peer-reviewed by outside reviewers (including SAB review) concluded that the most appropriate basis for an oral quantitative estimate was the study by Tseng et al. (1977), which reported increased prevalence of skin cancers in humans as a consequence of arsenic exposure in drinking water. Based on this study a unit risk of 5E-5/ug/L was proposed.

A recent memorandum by the Administrator of the EPA recommended that the above unit risk be adopted. The memorandum further counsels that "in reaching risk management decisions in a specific situation, risk managers must recognize and consider the qualities and uncertainties of risk estimates. The uncertainties associated with ingested inorganic arsenic are such that estimates could be modified downwards as much as an order of magnitude, relative to risk estimates associated with most other carcinogens. In such instances, the management document must clearly articulate this fact and state the factors that influenced such a decision."

CARI -
o CLASSIFICATION : A; human carcinogen
o BASIS FOR CLASSIFICATION : based on observation of increased lung cancer mortality in populations exposed primarily through inhalation and on increased skin cancer incidence in several populations consuming drinking

water with high arsenic concentrations. NOTE: The carcinogenicity assessment for arsenic may change in the near future pending the outcome of a further review now being conducted by the Carcinogen Risk Assessment Verification Endeavor Work Group.

o INHALATION UNIT RISK : 4.3E-3/ug/cu.m
o DOSE EXTRAPOLATION METHOD : absolute-risk linear model
o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-2 ug/cu.m
E-5 (1 in 100,000)	2E-3 ug/cu.m
E-6 (1 in 1,000,000)	2E-4 ug/cu.m

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o INHALATION DOSE-RESPONSE DATA :

Tumor Type -- lung cancer
Test Animals -- human, male
Route -- inhalation, occupational exposure
Reference -- Brown and Chu, 1983a,b,c; Lee-Feldstein, 1983; Higgins, 1982; Enterline and Marsh, 1982

Ambient Unit Risk Estimates

Exposure Source	Study	Unit Risk	Geometric Mean Unit Risk	Final Estimates Unit Risk
Anaconda smelter	Brown and Chu, 1983a,b,c	1.25 E-3		
	Lee-Feldstein, 1983	2.80 E-3	2.56 E-3	
	Higgins, 1982; Higgins et al., 1982; Welch et al., 1982	4.90 E-3		4.29 E-3
ASARCO smelter	Enterline and Marsh, 1982	6.81 E-3	7.19 E-3	
		7.60 E-3		

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o ADDITIONAL COMMENTS :

A geometric mean was obtained for data sets obtained within distinct exposed populations (U.S. EPA, 1984). The final estimate is the geometric mean of those two values. It was assumed that the increase in age-specific mortality rate of lung cancer was a function only of cumulative exposures.

The unit risk should not be used if the air concentration exceeds 2

ug/cu.m, since above this concentration the unit risk may not be appropriate.

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o DISCUSSION OF CONFIDENCE :

Overall a large study population was observed. Exposure assessments included air measurements for the Anaconda smelter and both air measurements and urinary arsenic for the ASARCO smelter. Observed lung cancer incidence was significantly increased over expected values. The range of the estimates derived from data from two different exposure areas was within a factor of 6.

CARDR-

o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1984

The 1984 Health Assessment Document for Inorganic Arsenic received Agency and external review including a review by SAB.

DOCUMENT

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o REVIEW DATES : 01/13/88, 12/07/89, 02/03/94
o VERIFICATION DATE : 01/13/88
o EPA CONTACTS :

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Chao W. Chen / OHEA -- (202)260-5898

WQCHU-

Water and Fish Consumption -- 2.2E-3 ug/L

Fish Consumption Only -- 1.75E-2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criteria represents a E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 3.6E+2 ug/L (Arsenic III)
Chronic -- 1.9E+2 ug/L (Arsenic III)

Marine:

Acute -- 6.9E+1 ug/L (Arsenic III)
Chronic -- 3.6E+1 ug/L (Arsenic III)

Considers technological or economic feasibility? -- NO

Discussion -- The criteria given are for Arsenic III. Much less data are available on the effects of Arsenic V to aquatic organisms, but the toxicity seems to be less. A complete discussion may be found in the referenced notice.

Reference -- 50 FR 30784 (07/29/85)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 0.05 mg/L (Proposed, 1985)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0.05 mg/L for arsenic is proposed based on the current MCL of 0.05 mg/L. Even though arsenic is potentially carcinogenic in humans by inhalation and ingestion, its potential essential nutrient value was considered in determination of an MCLG. The basis for this evaluation is nutritional requirements by NAS (NAS, 1983, Vol. 5, Drinking Water and Health, National Academy of Sciences Press, Washington, DC.)

Reference -- 50 FR 46936 (11/13/85)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value (status) -- 0.05 mg/L (Interim, 1980)

Considers technological or economic feasibility? -- YES

Discussion -- As an interim measure the U.S. EPA is using the value previously derived by the Public Health Service.

Monitoring requirements -- Ground water systems every three years; surface water systems annually.

Analytical methodology -- Atomic absorption/furnace technique (EPA 206.2; SM 304); atomic absorption/gaseous hydride (EPA 206.3; SM 303E; ASTM D-2972-78B)

Best available technology -- No data available.

Reference -- 45 FR 57332 (08/27/80); 50 FR 46936 (11/13/85)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

FISTD-

Status -- Issued (1988)

Reference -- Arsenic, Chromium and Chromated Arsenical Compounds Pesticide Registration Standard. June, 1988. [NTIS# PB89-102842]

EPA Contact -- Registration Branch / OPP (703)557-7760 / FTS 557-7760

FIREV-

Action -- Final regulatory decision - PD4 (1988)

Considers technological or economic feasibility? -- NO

Summary of regulatory action -- Cancellation of specified non-wood uses. Registrant of lead arsenate voluntarily canceled 09/87. Registrant of calcium arsenate voluntarily canceled 02/14/89. Use of sodium arsenate as ant bait canceled on 07/26/89. Criterion of concern: oncogenicity, mutagenicity and teratogenicity. Previous actions: 1) Voluntary cancellation of sodium arsenite (1978). Voluntary cancellation of two products. Criterion of concern: oncogenicity, mutagenicity and teratogenicity; 2) PD4 (1984). Requires label changes for wood use including a restricted use classification. Criterion of concern: oncogenicity, mutagenicity and teratogenicity; 3) Voluntary cancellation of copper arsenate (1977). Criterion of concern: oncogenicity.

Reference -- 53 FR 24787 (06/30/88); 43 FR 48267 (10/18/78); 42 FR 18422 (04/07/77); 49 FR 28666 (07/13/84) [NTIS# PB84-241538]; 49 FR 43772 (10/31/84); 50 FR 4269 (01/30/85)

EPA Contact -- Special Review Branch / OPP
(703)557-7400 / FTS 557-7400

CERC -

Value (status) -- 1 pound (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The 1-pound RQ for arsenic is based on its potential carcinogenicity. Available data indicate a hazard ranking of high based on a potency factor of 142.31/mg/kg/day and a weight-of-evidence group A, which corresponds to an RQ of 1 pound. Evidence found in "Water-Related Environmental Fate of 129 Priority Pollutants" (EPA 440/4-79-029a) also indicates that this material, or a constituent of this material, is bioaccumulated to toxic levels in the tissue of aquatic and marine organisms, and has the potential to concentrate in the food chain. Reporting of releases of massive forms of this hazardous substance is not required if the diameter of the pieces released exceeds 100 micrometers (0.004 inches).

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

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OREF - Cebrian, M.E., A. Albores, M. Aguilar and E. Blakely. 1983. Chronic arsenic poisoning in the north of Mexico. Human Toxicol. 2: 121-133.

OREF - Ferm, V.H. and S.J. Carpenter. 1968. Malformations induced by sodium arsenate. J. Reprod. Fert. 17: 199-201.

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OREF - Schroeder, H.A. and M. Mitchner. 1971. Toxic effects of trace elements on the reproduction of mice and rats. Arch. Environ. Health. 23(2): 102-106.

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OREF - Valentine, J.L., L.S. Reisbord, H.K. Kang and M.D Schluchter. 1987. Arsenic effects on population health histories. In: Trace Elements in Man and Animals - TEMA 5, C.F. Mills, I. Bremner and J.K. Chesters, eds. Commonwealth Agricultural Bureaux, Aberdeen, Scotland.

OREF - U.S. EPA. 1984. Health Assessment Document for Inorganic Arsenic. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA 600/8-83-021F.

OREF - U.S. EPA. 1988. Quantitative Toxicological Evaluation of Ingested Arsenic. Office of Drinking Water, Washington, DC. (Draft)

IREF - None

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CREF - Axelson, O., E. Dahlgren, C.D. Jansson and S.O. Rehnlund. 1978. Arsenic exposure and mortality: A case referent study from a Swedish copper smelter. Br. J. Ind. Med. 35: 8-15.

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J. Natl. Cancer Inst. 70: 455-463.
CREF - Brown, C.C. and K.C. Chu. 1983c. A new method for the analysis of cohort studies, implications of the multistage theory of carcinogenesis applied to occupational arsenic exposure. Environ. Health Perspect. 50: 293-308.
CREF - Cebrian, M.E., A. Albores, M. Aguilar and E. Blakely. 1983. Chronic arsenic poisoning in the north of Mexico. Human Toxicol. 2: 121-133.
CREF - Crossen, P.E. 1983. Arsenic and SCE in human lymphocytes. Mutat. Res. 119: 415-419.
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CREF - Higgins, I., K. Welch and C. Burchfield. 1982. Mortality of Anaconda smelter workers in relation to arsenic and other exposures. University of Michigan, Dept. Epidemiology, Ann Arbor, MI.
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CREF - Pershagen, G., B. Lind and N.E. Bjorkund. 1982. Lung retention and toxicity of some inorganic arsenic compounds. Environ. Res. 29: 425-434.
CREF - Pershagen, G., G. Nordberg and N.E. Bjorklund. 1984. Carcinomas of the respiratory tract in hamsters given arsenic trioxide and/or benzo(a)pyrene by the pulmonary route. Environ. Res. 34: 227-241.
CREF - Rencher, A.C., M.W. Carter and D.W. McKee. 1978. A retrospective epidemiological study of mortality at a large western copper smelter. J. Occup. Med. 19: 754-758.
CREF - Rossman, T.G. 1981. Enhancement of UV-mutagenesis by low concentrations of arsenite in E. Coli. Mutat. Res. 91: 207-211.
CREF - Roth, F. 1958. Uber den Bronchialkrebs Arsengeschodigter Winzer. Virchows Arch. 331: 119-137.
CREF - Sommers, S.C. and R.G. McManus. 1953. Multible arsenical cancers of the skin and internal organs. Cancer. 6: 347-359.
CREF - Southwick, J., A. Western, M. Beck, T. Whitley, R. Isaacs, J. Petajan and C. Hansen. 1981. Community health associated with arsenic in drinking water in Millard County, Utah. Health Effects Research Laboratory, Cincinnati, OH, EPA-600/1-81-064.
CREF - Tokudome, S. and M. Kuratsune. 1976. A cohort study on mortality from cancer and other causes among workers at a metal refinery. Int. J. Cancer. 17: 310-317.
CREF - Tseng, W.P. 1977. Effects and dose response relationships of skin cancer and blackfoot disease with arsenic. Environ. Health Perspect. 19: 109-119.
CREF - U.S. EPA. 1984. Health Assessment Document for Inorganic Arsenic. Prepared by Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA/600/8-83/021F.
CREF - Wan, B., R.T. Christian and S.W. Sookup. 1982. Studies of cytogenetic effects of sodium arsenicals on mammalian cells in vitro. Environ. Mutag. 4: 493-498.
CREF - Welch, K., I. Higgins, M. Oh and C. Burchfield. 1982. Arsenic exposure, smoking, and respiratory cancer in copper smelter workers. Arch. Environ. Health. 37: 325-335.
HAREF- None

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74-87-3
Search in progress

SS (14) PSTG (1)

[IRIS] SS 15 /cf?
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UPDT - 01/20/92, 52 fields
STAT - Oral RfD Assessment (RDO) on-line 08/01/90
STAT - Inhalation RfC Assessment (RDI) pending 12/01/91
STAT - Carcinogenicity Assessment (CAR) no data
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 09/30/87 EXSR Regulatory Action section added
IRH - 03/01/88 RDO Dose conversion clarified
IRH - 03/01/88 RDO Text changed
IRH - 03/01/88 RDO Secondary contact changed
IRH - 06/30/88 RDO Contacts switched
IRH - 08/01/89 REFS Bibliography on-line
IRH - 06/01/90 RCRA EPA contact changed
IRH - 07/01/90 RDO Withdrawn; new RfD verified (in preparation)
IRH - 07/01/90 REFS Bibliography withdrawn
IRH - 08/01/90 RDO Oral RfD summary replaced; RfD changed
IRH - 08/01/90 REFS Bibliography replaced
IRH - 12/01/91 RDI Inhalation RfC now under review
IRH - 01/01/92 EXSR Regulatory actions updated
RLEN - 14502
NAME - Barium
RN - 7440-39-3
SY - Barium
SY - UN 1399
SY - UN 1400
SY - UN 1854

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Increased blood pressure	NOAEL: 10 mg/L (0.21 mg/kg/day)	3	1	7E-2 mg/kg/day

Subchronic to Chronic LOAEL: None
Human Drinking Water
Studies

Wones et al., 1990;
Brenniman and Levy, 1984

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*Conversion Factors: 10 mg/L x 1.5 L/day/70 kg = 0.21 mg/kg/day

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o ORAL RFD STUDIES :

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

Brenniman, G.R. and P.S. Levy. 1984. High barium levels in public drinking water and its association with elevated blood pressure. In: Advances in Modern Toxicology IX, E.J. Calabrese, Ed. Princeton Scientific Publications, Princeton NJ. p. 231-249.

No single study considered alone is appropriate to calculate a lifetime RfD for barium. The RfD must be based rather on a weight of evidence approach which takes into account recent findings of the Wones et al. (1990) and Brenniman and Levy (1984) epidemiologic studies as well as the various rodent studies that have been conducted (Perry et al., 1983; McCauley et al., 1985; Schroeder and Mitchener, 1975a,b; Tardiff et al., 1980). Because of the number of studies involved, the complete reference citations are given in the Section VI.

Wones et al. (1990) administered barium (as barium chloride) in the drinking water of 11 healthy male volunteers. Subjects ranged in age from 27 to 61 years and had no previous history of diabetes, hypertension, or cardiovascular disease. Diets were strictly controlled throughout the 10-week study. Subjects were given 1.5 L/day of distilled and charcoal-filtered water containing 0 mg/L barium for weeks 0 to 2; 5 mg/L for weeks 3 to 6, and 10 mg/L for weeks 7 to 10. Blood and urine samples, as well as morning and evening blood pressures, were taken. Electrocardiograms and 24-hour continuous electrocardiographic monitoring were also performed.

There were no changes in systolic or diastolic blood pressures, or serum chemistry, especially total cholesterol, HDL, LDL, triglycerides, potassium or glucose levels. There was an increase in serum calcium levels that was attributed to a decrease in serum albumin levels. This increase, although statistically significant, was considered borderline and not clinically significant. There were also no changes in cardiac cycle as noted by electrocardiograms and no significant arrhythmias. A NOAEL of 10 mg/L was identified in this study which corresponds to 0.21 mg/kg/day, based on an actual consumption rate of 1.5 L/day and a 70-kg body weight.

Brenniman and Levy (1984) conducted a retrospective epidemiology study which compared human mortality and morbidity rates in populations ingesting elevated barium levels (2 to 10 mg/L) in their drinking water to populations ingesting very little or no barium (less than or equal to 0.2 mg/L). Mortality rates for cardiovascular diseases were determined for the years 1971-1975 and were age-adjusted. For the morbidity study, 1175 adult males and 1203 adult females were selected from communities in which the average drinking water concentration was 7.3 mg/L. Differences in mortality rates from all cardiovascular diseases were significantly higher ($p<0.05$) in the communities with elevated barium. However, these differences were largely in

the 65 and over age group and did not account for confounding variables such as population mobility, or use of water softeners or medication.

Differences in blood pressure, prevalance of hypertension, stroke, and heart and renal disease were also measured between the individuals in the two communities. Data were analyzed using signed ranked test for age-specific rates, the weighted Z test for prevalence rates, and analysis of variance for blood pressures. No significant differences were found in mean systolic and diastolic pressures between the two communities. No significant differences were found when the total populations were broken down by duration (10 years or more), medication, or use of water softeners. Also, the prevalence rates for hypertension, stroke, and heart and kidney disease were not significantly different between the communities.

A concentration of 7.3 mg/L corresponds to a dose of 0.20 mg/kg/day (assuming a 70-kg adult drinks 2 L/day).

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o ORAL RFD UNCERTAINTY :

UF = 3. According to U.S. EPA guidelines, an uncertainty factor of 10 is applied when a NOAEL from a subchronic human study is employed. However, data are available from chronic human studies which support this NOAEL, as well as several oral chronic animal studies. Therefore, this UF is not considered necessary. In addition, another factor of 10 is used with a human study to protect sensitive individuals. However, the data base supports the finding that the critical effect is hypertension which results from long exposure durations, and that the population most at risk is the adult male. Furthermore, the chosen study is a careful observation of this critical effect in adult males. Because of both the critical study's unique focus and the supporting studies, a 3-fold UF, instead of a 10-fold UF, was chosen as most appropriate to protect for sensitive individuals within that population.

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o ORAL RFD MODIFYING FACTOR :

MF = 1.

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o ORAL RFD COMMENTS :

Occupational studies of workers exposed to barium dust have shown that workers develop "baritosis." Affected workers showed no symptoms, no abnormal physical signs, no loss of vital capacity or interference with function, although they had a significantly higher incidence of hypertension.

McCauley et al. (1985) studied the histologic and cardiovascular effects of drinking water containing 0, 10, 100, or 250 mg/L barium for 36 weeks; 0, 1, 10, 100, or 1000 mg/L barium for 16 weeks, or 0, 10, 100, or 250 mg/L (0, 1.4, 14, 35, or 140 mg/kg Ba) barium for 68 weeks on male Sprague-Dawley rats (6/group). Females were exposed to 0 or 250 mg/L for 46 weeks. No significant histologic, carcinogenic, or cardiovascular (including hypertension) effects were observed. No changes were reported in body weight,

or food and water consumption in any of the treated animals. Animals treated at the highest dose (1000 mg/L) did exhibit ultrastructural changes in the kidney glomeruli and the presence of myelin figures. No other effects were reported at any dose level for males or females.

Perry et al. (1983) exposed weanling rats to barium at 1, 10, or 100 ppm in drinking water for up to 16 months (average daily barium doses of 0.051, 0.51, and 5.1 mg/kg, respectively). There were no signs of toxicity at any barium dose level. Systolic blood pressure measurements revealed no increase in animals exposed to 1 ppm for 16 months, an increase of 4 mm Hg ($p<0.01$) in animals exposed to 10 ppm barium for 16 months, and an increase of 16 mm Hg ($p<0.001$) in animals exposed to 100 ppm barium for 16 months. The animals in this study were maintained in a special contaminant-free environment and fed a diet designed to reduce exposure to trace metals. It is possible that the restricted intake of certain beneficial metals (e.g., calcium and potassium) may have predisposed the test animals to the hypertensive effects of barium (U.S. EPA, 1985).

Schroeder and Mitchener (1975a,b) exposed rats and mice to 5 mg/L barium in drinking water for a lifetime (approximately 0.25 mg/kg/day for rats and 0.825 mg/kg/day for mice). No adverse effects were observed; however, blood pressure was not measured.

Tardiff et al. (1980) exposed rats to barium at 0, 10, 50, or 250 ppm in drinking water for 4, 8, and 13 weeks. The barium concentrations were approximately 0, 2.75, 13.7, and 66.25 mg/kg/day at the beginning of the study and 0, 1.7, 6.6, and 31.5 mg/kg/day at the end of the study. Although the barium body burden increased with increasing barium dosage, no conclusive signs of barium toxicity were observed in these animals. Blood pressure was not measured.

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o ORAL RFD CONFIDENCE :

Study: Medium
Data Base: Medium
RfD: Medium

As previously stated, EPA does not believe that any single study, considered alone, is adequate to calculate an RfD for barium. However, EPA believes that medium confidence can be placed in the total data base used to determine the RfD.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- U.S. EPA. 1985. Draft Drinking Water Health Effects Criteria Document on Barium. Office of Drinking Water, Washington, DC. NTIS PB 86-118031/AS.

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o REVIEW DATES : 07/08/85, 07/22/85, 12/15/87, 05/17/90, 06/21/90

o VERIFICATION DATE : 06/21/90
o EPA CONTACTS :

Kenneth L. Bailey / ODW -- (202)260-5535 / FTS 260-5535

Linda R. Papa / ODW -- (513)569-7587 / FTS 684-7587

RDI -
o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

WQCHU-

Water and Fish Consumption: 1E+3 ug/L

Fish Consumption Only: None

Considers technological or economic feasibility? -- NO

Discussion -- The criteria is the same as the existing standard for drinking water (1 mg/L).

Reference -- Quality Criteria for Water (7/76) EPA 440/9-76-023 [NTIS No. PB-263943].

EPA Contact -- Criteria and Standards Division / OWRS (202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- none
Chronic -- none

Marine:

Acute -- none
Chronic -- none

Considers technological or economic feasibility? -- NO

Discussion -- It is generally believed that the physical and chemical properties of barium will preclude the existence of toxic soluble forms under usual marine and fresh water conditions and thus a restrictive criterion for aquatic life is considered unwarranted.

Reference -- Quality Criteria for Water, July, 1976, PB-263943

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 2 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- The MCLG of 2 mg/L for barium is based on potential adverse effects reported in humans and animal studies.

Reference -- 56 FR 3600 (01/30/91); 56 FR 30266 (07/01/91)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 2 mg/L (Final, 1991)

Considers technological or economic feasibility? -- YES

Discussion -- EPA has set the MCL equal to the MCLG of 2 mg/L.

Monitoring requirements -- Ground water systems monitored every three years; surface water systems monitored annually; systems out of compliance must begin monitoring quarterly until system is reliably and consistently below MCL.

Analytical methodology -- Atomic absorption/furnace technique (EPA 208.2; SM 304); atomic absorption/direct aspiration (EPA 208.1; SM 303C); inductively coupled plasma (EPA 200.7A): PQL= 0.15 mg/L.

Best available technology -- Ion exchange; lime softening; reverse osmosis; electrodialysis.

Reference -- 56 FR 3526 (01/30/91); 56 FR 3600 (01/30/91); 56 FR 30266 (07/01/91).

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Brenniman, G.R. and P.S. Levy. 1984. Epidemiological study of barium in Illinois drinking water supplies. In: Advances in Modern Environmental Toxicology IX, E.J. Calabrese, R.W. Tuthill and L. Condie, Ed. Princeton Scientific Publications, Princeton NJ. p. 231-240.

OREF - McCauley, P.T., B.H. Douglas, R.D. Laurie and R.J. Bull. 1985. Investigations into the effect of drinking water barium on rats. Environ. Health Perspect. Vol. IX, E.J. Calabrese, Ed. Princeton Scientific Publications, Princeton, NJ. p. 197-210.

OREF - Perry, H.M., S.J. Kopp, M.W. Erlanger and E.F. Perry. 1983.

Cardiovascular effects of chronic barium ingestion. In: Trace Substances in Environmental Health, XVII, D.D. Hemphill, Ed. Proc. Univ. Missouri's 17th Ann. Conf. on Trace Substances in Environmental Health. University of Missouri Press, Columbia, MO.

OREF - Schroeder, H.A. and M. Mitchener. 1975a. Life-term effects of mercury, methyl mercury and nine other trace metals on mice. J. Nutr. 105: 452-458.

OREF - Schroeder, H.A. and M. Mitchener. 1975b. Life-term studies in rats: Effects of aluminum, barium, beryllium and tungsten. J. Nutr. 105: 421-427.

OREF - Tardiff, R.G., M. Robinson and N.S. Ulmer. 1980. Subchronic oral toxicity of BaCl2 in rats. J. Environ. Pathol. Toxicol. 4: 267-275.

OREF - U.S. EPA. 1985. Draft Drinking Water Health Effects Criteria Document on Barium. Office of Drinking Water, Washington, DC. NTIS PB 86-118031/AS.

OREF - Wones, R.G., B.L. Stadler and L.A. Frohman. 1990. Lack of effect of drinking water barium on cardiovascular risk factor. Environ. Health Perspect. 85: 1- 13.

IREF - None

CREF - None

HAREF- None

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7440-41-7
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SS (26) PSTG (1)

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prt dl ncar, car continuous

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STAT - Oral RfD Assessment (RDO) on-line 02/01/93
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 09/01/92
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 03/01/88 RDO Reference dose table clarified
IRH - 03/01/88 RDO Text added
IRH - 09/07/88 CAR Carcinogen summary on-line
IRH - 01/01/90 CAREV References clarified
IRH - 01/01/90 CAREV Text revised
IRH - 01/01/90 CARO Quantitative estimate for oral exposure section added
IRH - 01/01/90 CARI Text revised
IRH - 01/01/90 CARDR Work group review dates and verification date added
IRH - 01/01/90 REFS Bibliography on-line
IRH - 02/01/90 OREF Puzanova et al. 1978 citation corrected
IRH - 02/01/90 CREF Wagner et al. 1969 citation corrected
IRH - 09/01/90 RDO Morgareidge ref. now Cox (same study-authors reversed)
IRH - 09/01/90 RCRA EPA contact changed
IRH - 09/01/90 OREF Morgareidge ref. now Cox (same study-authors reversed)
IRH - 01/01/91 CAR Text edited
IRH - 01/01/91 CARI Inhalation slope factor removed (global change)
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 09/01/92 CAREV U.S. EPA citation year corrected, paragraph 3
IRH - 09/01/92 CARDR Source document year corrected
IRH - 09/01/92 CARDR Review statement revised
IRH - 09/01/92 CREF U.S. EPA reference year corrected
IRH - 02/01/93 RDO Primary contact changed
RLEN - 27537
NAME - Beryllium
RN - 7440-41-7
SY - Beryllium
SY - Beryllium-9
SY - Glucinum
SY - RCRA waste number P015
SY - UN 1567

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
No adverse effects	NOAEL: 5 ppm in	100	1	5E-3

	drinking water (0.54	mg/kg/day
Rat, Chronic Oral Bioassay	mg/kg bw/day)	
Schroeder and Mitchner, 1975	LOAEL: none	

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*Conversion Factors: 5 ppm (5 mg/L) x 0.035 L/day / 0.325 kg bw = 0.54 mg/kg bw/day

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o ORAL RFD STUDIES :

Schroeder, H.A. and M. Mitchner. 1975. Life-term studies in rats: Effects of aluminum, barium, beryllium and tungsten. J. Nutr. 105: 421-427.

Fifty-two weanling Long-Evans rats of each sex received 0 or 5 ppm beryllium (as BeSO4, beryllium sulfate) in drinking water. Exposure was for the lifetime of the animals. At natural death the rats were dissected and gross and microscopic changes were noted in heart, kidney, liver, and spleen. There were no effects of treatment on these organs or on lifespan, urinalysis, serum glucose, cholesterol, and uric acid, or on numbers of tumors. Male rats experienced decreased growth rates from 2 to 6 months of age.

Similar studies were carried out on Swiss (CD strain) mice in groups of 54/sex at doses of approximately 0.95 mg/kg/day (Schroeder and Mitchner, 1975). Female animals showed decreased body weight compared with untreated mice at 6 of 8 intervals. Male mice exhibited slight increases in body weight. These effects were not considered adverse, therefore, 0.95 mg/kg/day is considered a NOAEL.

An unpublished investigation by Cox et al. (1975) indicates a much higher dose level (approximately 25 mg/kg/day) in the diet may be a NOEL.

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o ORAL RFD UNCERTAINTY :

UF -- The uncertainty factor of 100 reflects a factor of 10 each for interspecies conversion and for the protection of sensitive human subpopulations.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

This RfD is limited to soluble beryllium salts. Data on the terato- genicity or reproductive effects of beryllium are limited. It has been reported to produce embryolethality and terata in chick embryos (Puzanova et al., 1978).

---

o ORAL RFD CONFIDENCE :

Study -- Low
Data Base -- Low
RfD -- Low

Confidence in the study is rated as low because only one dose level was administered. Although numerous inhalation investigations and a supporting chronic oral bioassay in mice exist, along with the work by Cox et al. (1975) which indicates that a higher dose level might be a NOEL, these studies are considered as low to medium quality; thus, the data base is given a low confidence rating. The overall confidence in the RfD is low, reflecting the need for more toxicity data by the oral route.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- U.S. EPA, 1985

The 1985 Drinking Water Criteria Document for Beryllium is currently undergoing Agency review.

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o REVIEW DATES : 12/02/85
o VERIFICATION DATE : 12/02/85
o EPA CONTACTS :

Linda R. Papa / OHEA -- (513)569-7587

Krishan Khanna / OST -- (202)260-7588

CAREV-
o CLASSIFICATION : B2; probable human carcinogen.
o BASIS FOR CLASSIFICATION : Beryllium has been shown to induce lung cancer via inhalation in rats and monkeys and to induce osteosarcomas in rabbits via intravenous or intramedullary injection. Human epidemiology studies are considered to be inadequate.
o HUMAN CARCINOGENICITY DATA :

Inadequate. Reported increases, while apparently associated with exposure, did not take a variety of possible confounding factors into account. Wagoner et al. (1980) observed 47 deaths from cancer among 3055 white males employed in beryllium-processing with a median duration of employment of 7.2 months. Among the 2068 followed for 25 years or more, 20 lung cancer deaths were observed. These increased incidences were statistically significant. When lung cancer mortality data became available for 1968-1975, the number of expected deaths was recalculated and the increased incidence was statistically

significant only among workers followed 25 years or more (Bayliss, 1980; MacMahon, 1977, 1978). When the number of expected deaths was adjusted for smoking, the increased incidence was no longer significant (U.S. EPA, 1986).

An earlier study of workers from this same beryllium processing plant, and several studies of workers from this plant combined with workers from other beryllium plants, have reported a statistically significant increased incidence of lung cancer (Bayliss and Wagoner, 1977; Mancuso, 1970, 1979, 1980). No adjustment was made for smoking in these studies, and all were limited in their ability to detect a possible increased incidence of lung cancer because of methodological constraints and deficiencies.

---

o ANIMAL CARCINOGENICITY DATA :

Sufficient. Based on the evidence for induction of tumors by a variety of beryllium compounds in male and female monkeys and in several strains of rats of both sexes, via inhalation and intratracheal instillation, and the induction of osteosarcomas in rabbits by intravenous or intramedullary injection in multiple studies.

Slight increases in cancer incidence (not statistically significant in comparison with controls) were reported in Long-Evans rats (52/sex/group) administered 5 ppm beryllium sulfate in the drinking water for a lifetime. The authors reported a slight excess of grossly observed tumors in the 5 ppm group (9/33) over controls (4/26) in the male rats. The power of this test to detect a carcinogenic effect was reduced by high mortality (approximately 60% survived a pneumonia epidemic at 20 months) (Schroeder and Mitchener, 1975a). Schroeder and Mitchener (1975b) administered 5 ppm beryllium sulfate in drinking water to Swiss mice (54/sex/group) over a lifetime. A non-statistically significant increase in incidence of lymphoma leukemias were reported in the females (9/52) relative to controls (3/47).

An increase in reticulum cell sarcomas of the lungs was seen in male, but not female Wistar-derived rats administered beryllium sulfate in the diet at 5 and 50 ppm, but not at 500 ppm (Morgareidge et al., 1977). The incidence in males equaled 10/49, 17/35, 16/40 and 12/39 for the control, low, intermediate and high dose groups, respectively. Since the results were published only as an abstract, and since no response was seen at the highest dose, these results are considered to be only suggestive for the induction of cancer via this route.

Osteogenic sarcomas were induced in rabbits by intravenous injection of beryllium compounds in at least 12 different studies and by intramedullary injection in at least four studies (U.S. EPA, 1991). Bone tumors were induced by beryllium oxide, zinc beryllium silicate, beryllium phosphate, beryllium silicate and beryllium metal. No bone tumors were reported to be induced by intravenous injection of beryllium oxide or zinc beryllium silicate in rats or guinea pigs (Gardner and Heslington, 1946). Positive results, however, were reported in mice injected with zinc beryllium silicate, although the numbers were not listed (Cloudman et al., 1949). The sarcomas were generally reported

to be quite malignant and metastasized to other organs.

Lung tumors, primarily adenomas and adenocarcinomas, have been induced via the inhalation route in both male and female Sprague-Dawley rats during exposure periods of up to 72 weeks by beryllium sulfate (Reeves et al., 1967), in both male and female Sherman and Wistar rats by beryllium phosphate, beryllium fluoride and zinc beryllium silicate (Schepers, 1961), in male Charles River CR-CD rats by beryl ore (Wagner et al., 1969) and in both male and female rhesus monkeys by beryllium sulfate (Vorwald, 1968). Positive results were seen in rats exposed to beryllium sulfate at concentrations as low as 2 ug/cu.m (Vorwald, 1968).

Tumors were also induced by intratracheal instillation of metallic beryllium, beryllium-aluminum alloys and beryllium oxide in both Wistar rats and rhesus monkeys. Adenomas, adenocarcinomas and malignant lymphomas were seen in the lungs, with lymphosarcomas and fibrosarcomas present at extrapulmonary sites (Groth et al., 1980; Ishinishi et al., 1980).

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o SUPPORTING DATA :

Beryllium sufate and beryllium chloride have been shown to be nonmutagenic in bacterial and yeast gene mutation assays (Simmon et al., 1979). In contrast, gene mutation studies in Chinese hamster V79 and CHO cells were positive (Miyaki et al., 1979; Hsie et al., 1979). Chromosomal aberrations and sister chromatid exchange were also induced by beryllium in cultured human lymphocytes and Syrain hamster embryo cells (Larramendy et al., 1981).

CARO -

o CLASSIFICATION : B2; probable human carcinogen.

o BASIS FOR CLASSIFICATION : Beryllium has been shown to induce lung cancer via inhalation in rats and monkeys and to induce osteosarcomas in rabbits via intravenous or intramedullary injection. Human epidemiology studies are considered to be inadequate.

o ORAL SLOPE FACTOR : 4.3 per(mg/kg)/day

o DRINKING WATER UNIT RISK : 1.2E-4 per(ug/L)

o DOSE EXTRAPOLATION METHOD : Linearized multistage procedure, extra risk

o RISK/WATER CONCENTRATIONS :

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	8.3E-1 ug/L

E-5 (1 in 100,000) 8.3E-2 ug/L
E-6 (1 in 1,000,000) 8.3E-3 ug/L

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o ORAL DOSE-RESPONSE DATA :

Tumor Type -- gross tumors, all sites combined
Test Animals -- rat/Long-Evans, male
Route -- drinking water
Reference -- Schroeder and Mitchener, 1975a

Administered ppm	Dose (mg/kg)/day	Human Equivalent Dose (mg/kg/day)	Tumor Incidence
0	0	0	4/26
5	0.54	0.09	9/33

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o ADDITIONAL COMMENTS :

The solubility and speciation of beryllium in air and water media vary, with ambient air characterized by relatively insoluble beryllium compounds such as beryllium oxide and metallic beryllium, and water characterized by more soluble forms. Carcinogenic potency varies according to the form of beryllium present.

Human equivalent doses were calculated using a human body weight of 70 kg, an animal weight of 0.325 kg and length of exposure, experiment and lifespan of 1126 days for treated and control animals.

The unit risk should not be used if the water concentration exceeds 8.3E+1 ug/L, since above this concentration the unit risk may not be appropriate.

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o DISCUSSION OF CONFIDENCE :

The estimate is derived from a study which did not show a significant increase in tumorigenic response. While this study is limited by use of only one non-zero dose group, the occurrence of high mortality and unspecified type and site of the tumors, it was used as the basis of the quantitative estimate because exposure occurred via the most relevant route. Oral risk estimates derived by extrapolation from studies in other species/strains for the intravenous and inhalation routes (also highly uncertain) are within an order of magnitude.

CARI -
o CLASSIFICATION : B2; probable human carcinogen.

o BASIS FOR CLASSIFICATION : Beryllium has been shown to induce lung cancer via inhalation in rats and monkeys and to induce osteosarcomas in rabbits via intravenous or intramedullary injection. Human epidemiology studies are considered to be inadequate.
o INHALATION UNIT RISK : 2.4E-3 per (ug/cu.m)
o DOSE EXTRAPOLATION METHOD : Relative risk
o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	4E-2 ug/cu.m
E-5 (1 in 100,000)	4E-3 ug/cu.m
E-6 (1 in 1,000,000)	4E-4 ug/cu.m

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o INHALATION DOSE-RESPONSE DATA :

Tumor Type --
Test Animals -- humans
Route -- inhalation, occupational exposure
Reference --

Beryllium Concentration in Workplace (ug/cu.m)	Fraction of Lifetime	Effective dose (ug/cu.m)	95 percent Upper-bound Estimate of Relative Risk	Unit Risk /ug/cu.m
100	1.00	21.92	1.98	1.61E-3
			2.09	1.79E-3
	0.25	5.48	1.98	6.44E-3
			2.09	7.16E-3
1000	1.00	219.18	1.98	1.61E-4
			2.09	1.79E-4
	0.25	54.79	1.98	6.44E-4
			2.09	7.16E-4

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o ADDITIONAL COMMENTS :

Human data were used for the inhalation exposure quantitation despite limitations in the study. Humans are most likely to be exposed by inhalation to beryllium oxide, rather than other beryllium salts. Animal studies by inhalation of beryllium oxide have utilized intratracheal instillation, rather than general inhalation exposure.

Effective dose was determined by adjusting for duration of daily (8/24 hours) and annual (240/365 days) exposure, and the fraction of the lifetime at risk (i.e., time from onset of employment to termination of

follow-up). The risk estimates were based on the data of Wagoner et al. (1980) in which the smoking adjusted, expected lung cancer deaths were found to range from 13.91 to 14.67, in comparison to 20 observed. Relative risk estimates of 1.36 and 1.44 were derived and the 95% confidence limits of these estimates, 1.98 and 2.09, respectively, were used to estimate the lifetime cancer risk. Note that all of the above estimates are based on one data set using a range of estimated exposure and exposure times. Because of uncertainties regarding workplace beryllium concentration and exposure duration, unit risks were derived using two estimates each of concentration, fraction of lifetime exposed and relative risk. The recommended value is the arithmetic mean of the 8 derived unit risks.

The unit risk should not be used if the air concentration exceeds 4 ug/cu.m, since above this concentration the unit risk may not be appropriate.

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o DISCUSSION OF CONFIDENCE :

The estimate of risk for inhalation exposure was based upon an epidemiologic study having several confounding variables. The estimates of exposure levels and duration were also somewhat uncertain. While a quantitative assessment based on several animal studies resulted in a similar estimate of risk (which increases the confidence somewhat), the quality of the available studies was poor (that is, they were conducted at single dose levels or lacked control groups).

CARDR-
o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1986, 1991

Source Document Review -- The values in 1986 Health Assessment Document for Beryllium and the 1991 Drinking Water Criteria Document for Beryllium received Agency and external review.

Other EPA Documentation -- None
DOCUMENT

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o REVIEW DATES : 05/04/88, 02/01/89, 12/07/89
o VERIFICATION DATE : 05/04/88 (inhalation); 02/01/89 (oral)
o EPA CONTACTS :

William Pepelko / OHEA -- (202)260-5904

David Bayliss / OHEA -- (202)260-5726

CAA -

Considers technological or economic feasibility? -- YES

Discussion -- Beryllium was listed as a hazardous air pollutant under section 112 of the CAA in 1971 on the basis that it can cause the chronic lung disease berylliosis. Emission standards promulgated for extraction, ceramic, and propellant plants, foundries, incinerators, and machine shops are 10 g/24 hr or attainment of an ambient concentration near the source of 0.01 ug/cu.m, 30 day average. This ambient concentration was judged adequate to protect the public health with an ample margin of safety. More complex standards were also promulgated for beryllium rocket motor firing. The NESHAPs are now under review, and will consider new health evidence that beryllium may be a carcinogen. Reporting of releases of massive forms of this hazardous substance is not required if the diameter of the pieces released exceeds 100 micrometers (0.004 inches).

Reference -- 40 CFR Part 61, Subparts C & D

EPA Contact -- Emissions Standards Division, OAQPS
(917)541-5571 / FTS 629-5571

WQCHU-

Water and Fish Consumption: 6.8E-3 ug/L

Fish Consumption Only: 1.17E-1 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- For the maximum protection from the potential carcinogenic properties of this chemical, the ambient water concentration should be zero. However, zero may not be attainable at this time, so the recommended criterion represent a E-6 estimated incremental increase of cancer risk over a lifetime.

Reference -- 45 FR 79318 (11/28/80); Quality Criteria for Water, EPA 440/5-86-001 (5/87).

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute LEC -- 1.3E+2 ug/L
Chronic LEC -- 5.3E+0 ug/L

Marine: None

Considers technological or economic feasibility? -- NO

Discussion -- The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LECs are given when the minimum data required to derive water quality criteria are not available. Hardness has a substantial effect on acute toxicity.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value -- 0 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- NO

Discussion -- The proposed MCLG for beryllium is zero based on the evidence of carcinogenic potential (B2).

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 0.001 mg/L (Proposed, 1990)

Considers technological or economic feasibility? -- YES

Discussion -- The MCL is based on 5x the MDL, which is associated with a maximum lifetime individual risk of 1 E-4.

Monitoring requirements -- Ground water systems every 3 years; surface water systems annually; will allow monitoring at up to 10-year intervals after the system completes 3 rounds of sampling at <50% of the MCL.

Analytical methodology -- Atomic absorption/furnace technique (EPA 210.2;

ASTM D-3645; SM 304); inductively-coupled plasma (EPA 200.7; SM 305); ICP mass spectrometry (EPA 200.8): PQL= 0.001 mg/L.

Best available technology -- Activated alumina;ion exchange; reverse osmosis; lime softening; coagulation/filtration.

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

Status -- Listed (Final, 1991)

Discussion -- "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement -- All systems to be monitored unless a vulnerability assessment determines the system is not vulnerable.

Analytical methodology -- Atomic absorption/furnace technique (EPA 210.2; SM 304; ASTM D-3645); inductively coupled plasma (EPA 200.7; SM 305); spectrophotometric
(EPA 200.8).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

CERC -

Value (status) -- 10 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for beryllium is based on potential carcinogenicity. Available data indicate a hazard ranking of medium based on a potency factor of 79.70/mg/kg/day and a weight-of-evidence group B2, which correspond to an RQ of 10 pounds. Reporting of releases of massive forms of this hazardous substance is not required if the diameter of the pieces released exceeds 100 micrometers (0.004 inches).

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Cox, G.E., D.E. Bailey and K. Morgareidge. 1975. Chronic feeding studies with beryllium sulfate in rats. Unpublished report submitted by the Food and Drug Research Laboratories, Inc., to the Aluminum Company of America, Pittsburgh, PA.

OREF - Puzanova, L., M. Doskocil and A. Doubkova. 1978. Disturbances of the development of chick embryos after the administration of beryllium chloride at early stages of embryogenesis. Folia. Morphologica. 26(3): 228-231.

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

OREF - U.S. EPA. 1985. Drinking Water Criteria Document for Beryllium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.

IREF - None

CREF - Bayliss, D.L. 1980. U.S. EPA, Washington, DC. Letter to William H. Foege, M.D., Center for Disease Control, Atlanta, GA. November 12.
CREF - Bayliss, D.L. and J.K. Wagoner. 1977. Bronchogenic cancer and cardio- respiratory disease mortality among white males employed in a beryllium production facility. OSHA Beryllium Hearing, 1977, Exhibit 13.F.
CREF - Cloudman, A.M., D. Vining, S. Barkulis and J.J. Nickson. 1949. Bone changes following intravenous injections of beryllium. Am. J. Pathol. 25: 810-811.
CREF - Gardner, L.U. and H.F. Heslington. 1946. Osteo-sarcoma from intravenous beryllium compounds in rabbits. Fed. Proc. 5: 221. (Cited in U.S. EPA, 1987)
CREF - Groth, D.H., C. Kommineni and G.R. Mackay. 1980. Carcinogenicity of beryllium hydroxide and alloys. Environ. Res. 21(1): 63-84.
CREF - Hsie, A.W., J.P. O'Neill, J.R. San Sebastian, et al. 1979. Quantitative mammalian cell genetic toxicology: Study of the cytotoxicity and mutagenicity of seventy individual environmental agents related to energy technologies and three subfractions of crude synthetic oil in the CHO/HGPRT system. Environ. Sci. Res. 15: 219-315.
CREF - Ishinishi, N., M. Mizunoe, T. Inamasu and A. Hisanga. 1980. Experimental study on carcinogenicity of beryllium oxide and arsenic trioxide to the lung of rats by an intratracheal instillation. Fukuoka Igaku Zasshi. 71(1): 19-26. (Jap. with Eng. abstract)
CREF - Larramendy, M.L., N.C. Popescu and J.A. DiPaola. 1981. Induction by inorganic metal salts of sister chromatid exchanges and chromosome aberrations in human and Syrian hamster cell strains. Environ. Mutagen. 3: 597-606.
CREF - MacMahon, B. 1977. Evaluation of epidemiological materials. January 10, 1978. Brush Wellman, Cleveland, OH. OSHA Beryllium Hearings: 5.
CREF - MacMahon, B. 1978. OSHA Beryllium Hearings, comment on recent post-hearing submissions. Docket No. H005, February 9, 1979.
CREF - Mancuso, T.F. 1970. Relation of duration of employment and prior respiratory illness to respiratory cancer among beryllium workers. Environ. Res. 3: 251-275.
CREF - Mancuso, T.F. 1979. Occupational lung cancer among beryllium workers in dusts and disease. In: Proc. Conference on Occupational Exposure to Fibrous and Particulate Dust and Their Extension into the Environment, R. Lemen and J. Dement, Ed. Pathrotox Publishers, Inc.
CREF - Mancuso, T.F. 1980. Mortality study of beryllium industry workers' occupational lung cancer. Environ. Res. 21: 48-55.
CREF - Miyaki, M., N. Akamatsu, T. Ono, H. Koyama. 1979. Mutagenicity of metal cations in cultured cells from chinese hamster. Mutat. Res. 68: 259-263.
CREF - Morgareidge, K., G.E. Cox, D.E. Bailey and M.A. Gallo. 1977. Chronic oral toxicity of beryllium in the rat. Toxicol. Appl. Pharmocol. 41(1): 204-205.
CREF - Reeves, A.L., D. Deitch, and A.J. Vorwald. 1967. Beryllium

carcinogenesis: I. Inhalation exposure of rats to beryllium sulfate aerosol. Cancer Res. 27(1): 439-445.
CREF - Schepers, G.W.H. 1961. Neoplasia experimentally induced by beryllium compounds. Prog. Exp. Tumor Res. 2: 203-244.
CREF - Schroeder, H.A. and M. Mitchener. 1975a. Life-term studies in rats: Effects of aluminum, barium, beryllium and tungsten. J. Nutr. 105: 421-427.
CREF - Schroeder, H.A. and M. Mitchener. 1975b. Life-term effects of mercury, methyl mercury and nine other trace metals on mice. J. Nutr. 105: 452-458.
CREF - Simmon, V.F., H.S. Rosenkranz, E. Zeiger and L.A. Poirier. 1979. Mutagenic activity of chemical carcinogens and related compounds in the intraperitoneal host-mediated assay. J. Natl. Cancer Inst. 62(4): 911-918.
CREF - U.S. EPA. 1986. Health Assessment Document for Beryllium. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA 600/8-84-026F.
CREF - U.S. EPA. 1991. Drinking Water Criteria Document for Beryllium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.
CREF - Vorwald, A.J. 1968. Biologic manifestations of toxic inhalants in monkeys. In: Use of Nonhuman Primates in Drug Evaluation, H. Vagtborg, Ed. University of Texas Press, Austin, TX. p. 222-228.
CREF - Wagner, W.D., D.H. Groth, J.L. Holtz, G.E. Madden and H.E. Stokinger. 1969. Comparative chronic inhalation toxicity of beryllium ores, bertrandite and beryl, with production of pulmonary tumors by beryl. Toxicol. Appl. Pharmacol. 15: 10-29.
CREF - Wagoner, J.K., P.F. Infante and D.L. Bayliss. 1980. Beryllium: An etiologic agent in the induction of lung cancer, nonneoplastic respiratory disease, and heart disease among industrially exposed workers. Environ. Res. 21: 15-34.
HAREF- None

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STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) pending 05/01/93
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 10/01/89 RDO Oral RfD summary on-line
IRH - 10/01/89 REFS Bibliography on-line
IRH - 11/01/89 RDO Work group review date corrected
IRH - 08/01/90 RDO Text edited
IRH - 01/01/92 EXSR Regulatory Action section on-line
IRH - 01/01/93 CAR Carcinogenicity assessment now under review
IRH - 05/01/93 CAR Work group review date added
IRH - 07/01/93 RDO Oral RfD noted as pending change
IRH - 07/01/93 RDO Work grp. rev. date added; mtg. & verif. date corrected
RLEN - 7821
NAME - Boron (Boron and Borates only)
RN - 7440-42-8
SY - BORON

RDO -
o ORAL RFD SUMMARY :

NOTE: The Oral RfD for boron may change in the near future pending the outcome of a further review now being conducted by the RfD/RfC Work Group.

Critical Effect	Experimental Doses*	UF	MF	RfD
Testicular atrophy, spermatogenic arrest	NOAEL: 350 ppm (8.8 mg/kg/day)	100	1	9E-2 mg/kg/day

2-Year Dog Study
Oral Exposure (diet) LOAEL: 1170 ppm
(29 mg/kg/day)
Weir and Fisher, 1972

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*Conversion Factors: 1 ppm = 0.025 mg/kg/day (assumed dog food consumption). Author converted borax and boric acid doses to boron equivalents.

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o ORAL RFD STUDIES :

Weir, R.J., Jr. and R.S. Fisher. 1972. Toxicological studies on borax and

boric acid. Toxicol. Appl. Pharmacol. 23: 351-364.

Groups of 4 male and 4 female dogs were fed borax and boric acid in the diet for 2 years. The NOAEL was established at 350 ppm of boron equivalents (8.8 mg/kg/day), highest dose tested. In an additional study, dogs were fed 1170 ppm (29 mg/kg/day) for 38 weeks. At this dose, severe testicular atrophy and spermatogenic arrest occurred.

Groups of 35 male and 35 female rats were fed borax and boric acid in the diet for 2 years at boron-equivalent doses of 117, 350, and 1170 ppm (5.9, 17.5 or 58.5 mg B/kg/day). No treatment-related effects were seen at 5.9 or 17.5 mg/kg/day, so the highest NOAEL is 17.5 mg/kg/day. The LOAEL is 58.5 mg/kg/day, based on the following: significantly decreased testes weights and testes-to-body weight ratios; atrophied seminiferous epithelium; and decreased tubular size in the testes. Brain and brain-to-body weight ratios were also significantly decreased.

Schroeder and Mitchener (1975) reported a lifetime study in which mice were administered boron in drinking water at 5 mg/L (equivalent to 8.1 mg B/kg/day). No effects were observed with regard to body weight, longevity or survival. The NOAEL in this study was 8.1 mg/kg/day.

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o ORAL RFD UNCERTAINTY :

UF -- Used in accordance with Agency guidelines for a NOAEL from a lifetime animal study.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

The two principal studies indicate that the dog is more sensitive than the rat, with more severe testicular effects occurring at half the dose level in the dog vs. the rat (29 mg/kg/day vs. 58 mg/kg/day).

Other studies reviewed:

1) 70-Day Study - rats: Groups of 15 rats were exposed to boron in drinking water. LOAEL=23.7 mg/kg/day (150 mg B/L) (LDT: decreased body weights; decreased weights of testes, seminal vesicles, spleens, and right femurs; lower fat content of bones; and lower plasma triglycerides and protein concentrations). At 300 mg B/L (44.7 mg/kg/day), spermatogenesis was impaired. There was no NOAEL in this study (Seal and Weeth, 1980).

2) 13-Week Study - mice: Male and female mice were fed boric acid. LOAEL= 34-47 mg/kg/day (1200 ppm) (LDT: extramedullary hematopoiesis of spleen of minimal to mild severity was observed in all groups). At the higher doses, 68 mg/kg/day (2500 ppm) to 544 mg/kg/day (20,000 ppm), degeneration or atrophy of

seminiferous tubules was observed in males (NTP, 1987).

3) 90-Day Study - rats: Boric acid and borax were administered in the diet at 52.5, 175, 525, 1750, and 5250 ppm (2.6, 8.8, 26, 88, and 260 mg B/kg/day. The low dose caused an increase in the weight of the brain, spleen, kidneys, liver, and ovaries in females. Increased kidney weight occurred at 175 ppm in males. No organ weight changes were seen at 525 ppm in either sex. Severe effects in both sexes were seen at 1750 ppm and above (organ and body weight decreases) (Weir and Fisher, 1972).

4) 90-Day Feeding Study - dogs: Fed boron at levels of 17.5, 175, and 1750 ppm (0.44, 4.4, or 44 mg/kg/day). The lowest dose resulted in decreased spleen/body weight ratio in male dogs; 175 ppm resulted in decreased testes/body weight ratio; the highest dose produced severe testicular atrophy. No changes in female organ weights were observed at 17.5 or 175 ppm. No histologic changes were seen in dogs fed 175 ppm or below. Severe testicular atrophy seen at highest dose (Weir and Fisher, 1972).

5) Multigeneration Reproductive Study - rats: Dosed at 117, 350, and 1170 ppm (5.9, 17.5, 58.5 mg/kg/day). NOAEL=17.5 mg/kg/day; LOAEL=58.5 mg/kg/day; (HDT: males showed lack of spermatazoa in atrophied testes; females showed decreased ovulation) (Weir and Fisher, 1972).

6) 90-Day Reproductive Study - rats: Males were dosed with 0.3, 1.0, or 6.0 mg B/L (0.02, 0.072, or 0.426 mg/kg/day). NOAEL=0.426 mg/kg/day (HDT) (Dixon et al., 1976).

7) 60-Day Reproductive Study - rats: Doses were 0, 500, 1000, or 2000 mg/kg diet (equivalent to 25, 50, or 100 mg/kg/day). NOAEL=25 mg/kg/day; LOAEL=50 mg/kg/day; (decreased weights in liver, testes, and epididymis, and reduced fertility) (Dixon et al., 1979).

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o ORAL RFD CONFIDENCE :

Study -- Medium
Data Base -- Medium
RfD -- Medium

The referenced lifetime dog study provides both a NOAEL and a LOAEL and examines many biological endpoints, but has a limited number of experimental animals; it rates a medium confidence. Several sub-chronic, chronic and reproductive toxicity studies provide supportive data, but developmental data do not exist; hence the data base rates a medium level of confidence. Medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- None

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o REVIEW DATES : 06/23/88, 07/20/89, 06/15/93
o VERIFICATION DATE : 07/20/89
o EPA CONTACTS :

Robert Cantilli / OST -- (202)260-5546

Charles Abernathy / OST -- (202)260-7571

WQCHU-

No data available

WQCAQ-

No data available

* A criterion of 7.5E+2 ug/L for Boron in water has been recommended based on long-term irrigation on sensitive crops.

TSCA -

No data available

OREF - Dixon, R.L., I.P. Lee and R.J. Sherins. 1976. Methods to assess reproductive effects of environmental chemicals - Studies of cadmium and boron administered orally. Environ. Health Perspec. 13: 59-67.

OREF - Dixon, R.L., R.J. Sherins, and I.P. Lee. 1979. Assessment of environmental factors affecting male fertility. Environ. Health Perspec. 30: 53-68.

OREF - Seal, B.S. and H.J. Weeth. 1980. Effect of boron in drinking water on the male laboratory rat. Bull. Environ. Contam. Toxicol. 25: 782-789.

OREF - Schroeder, H.A. and M. Mitchener. 1975. Life-term effects of mercury, methyl mercury and nine other trace metals in mice. J. Nutr. 105: 452-458.

OREF - NTP (National Toxicology Program). 1987. Toxicology and carcinogenesis studies of boric acid in B6C3F1 mice (feed studies). NTP Technical Report Series No. 324. Research Triangle Park, NC.
OREF - Weir, R.J., Jr. and R.S. Fisher. 1972. Toxicologic studies on borax and boric acid. Toxicol. Appl. Pharmacol. 23: 351-364.
IREF - None
CREF - None
HAREF- None

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STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) on-line 06/01/92
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 04/01/92
IRH - 05/21/87 CARI Slope factor corrected
IRH - 03/01/88 CAREV Text added
IRH - 03/01/88 CARI Text revised
IRH - 03/01/88 CARI Confidence statement revised
IRH - 03/01/88 CARDR Secondary contact changed
IRH - 01/01/89 WQCHU Water quality human health criteria added
IRH - 01/01/89 WQCAQ Corrected marine acute criterion
IRH - 08/01/89 REFS Bibliography on-line
IRH - 10/01/89 RDO Oral RfD summary on-line
IRH - 10/01/89 OREF Oral RfD references added
IRH - 12/01/89 RDI Inhalation RfD now under review
IRH - 06/01/90 CAA Area code for EPA contact corrected
IRH - 06/01/90 RCRA EPA contact changed
IRH - 08/01/90 CAREV Basis statement revised
IRH - 08/01/90 CAREV Text revised, paragraph 1
IRH - 08/01/90 CARO Text revised
IRH - 01/01/91 CAR Text edited
IRH - 01/01/91 CARI Inhalation slope factor removed (global change)
IRH - 03/01/91 CAREV Text revised
IRH - 03/01/91 CARO Text revised
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 04/01/92 CAA CAA regulatory action withdrawn
IRH - 05/01/92 CARI Number correction in data table
IRH - 06/01/92 CAREV Text revised, paragraph 1
IRH - 06/01/92 CAREV Text clarified
IRH - 02/01/94 RDO Secondary contact changed
RLEN - 20060
NAME - Cadmium
RN - 7440-43-9
SY - C.I. 77180
SY - Cadmium
SY - KADMIUM

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD

Significant proteinuria	NOAEL (water): 0.005 mg/kg/day	10	1	5E-4 mg/kg/day (water)
Human studies involving chronic exposures	NOAEL (food): 0.01 mg/kg/day	10	1	1E-3 mg/kg/day (food)

U.S. EPA, 1985

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*Conversion Factors: See text for discussion

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o ORAL RFD STUDIES :

U.S. EPA. 1985. Drinking Water Criteria Document on Cadmium. Office of Drinking Water, Washington, DC. (Final draft)

A concentration of 200 ug cadmium (Cd)/gm wet human renal cortex is the highest renal level not associated with significant proteinuria (U.S. EPA, 1985). A toxicokinetic model is available to determine the level of chronic human oral exposure (NOAEL) which results in 200 ug Cd/gm wet human renal cortex; the model assumes that 0.01% day of the Cd body burden is eliminated per day (U.S. EPA, 1985). Assuming 2.5% absorption of Cd from food or 5% from water, the toxicokinetic model predicts that the NOAEL for chronic Cd exposure is 0.005 and 0.01 mg Cd/kg/day from water and food, respectively (i.e., levels which would result in 200 ug Cd/gm wet weight human renal cortex). Thus, based on an estimated NOAEL of 0.005 mg Cd/kg/day for Cd in drinking water and an UF of 10, an RfD of 0.0005 mg Cd/kg/day (water) was calculated; an equivalent RfD for Cd in food is 0.001 mg Cd/kg/day (see Section VI.A. for references).

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o ORAL RFD UNCERTAINTY :

UF -- This uncertainty factor is used to account for intrahuman variability to the toxicity of this chemical in the absence of specific data on sensitive individuals.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

Cd is unusual in relation to most, if not all, of the substances for which an oral RfD has been determined in that a vast quantity of both human and animal toxicity data are available. The RfD is based on the highest level of Cd in the human renal cortex (i.e., the critical level) not associated with significant proteinuria (i.e., the critical effect). A toxicokinetic model has been used to determine the highest level of exposure associated with the lack of a critical effect. Since the fraction of ingested Cd that is absorbed appears to vary with the source (e.g., food vs. drinking water), it is

necessary to allow for this difference in absorption when using the toxicokinetic model to determine an RfD.

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o ORAL RFD CONFIDENCE :

Study -- Not applicable
Data Base -- High
RfD -- High

The choice of NOAEL does not reflect the information from any single study. Rather, it reflects the data obtained from many studies on the toxicity of cadmium in both humans and animals. These data also permit calculation of pharmacokinetic parameters of cadmium absorption, distribution, metabolism and elimination. All of this information considered together gives high confidence in the data base. High confidence in either RfD follows as well.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- U.S. EPA, 1985

Other EPA Documentation -- None

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o REVIEW DATES : 05/15/86, 08/19/86, 09/17/87, 12/15/87, 01/20/88, 05/25/88
o VERIFICATION DATE : 05/25/88
o EPA CONTACTS :

Ken Bailey / OST -- (202)260-5535

Yogi Patel / OST -- (202)260-5849

RDI -
o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

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o REVIEW DATES : 11/16/89
CAREV-
o CLASSIFICATION : B1; probable human carcinogen
o BASIS FOR CLASSIFICATION : Limited evidence from occupational epidemiologic studies of cadmium is consistent across investigators and study populations. There is sufficient evidence of carcinogenicity in rats and mice by inhalation and intramuscular and

subcutaneous injection. Seven studies in rats and mice wherein cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of carcinogenic response.

o HUMAN CARCINOGENICITY DATA :

Limited. A 2-fold excess risk of lung cancer was observed in cadmium smelter workers. The cohort consisted of 602 white males who had been employed in production work a minimum of 6 months during the years 1940-1969. The population was followed to the end of 1978. Urine cadmium data available for 261 workers employed after 1960 suggested a highly exposed population. The authors were able to ascertain that the increased lung cancer risk was probably not due to the presence of arsenic or to smoking (Thun et al., 1985). An evaluation by the Carcinogen Assessment Group of these possible confounding factors has indicated that the assumptions and methods used in accounting for them appear to be valid. As the SMRs observed were low and there is a lack of clear cut evidence of a causal relationship of the cadmium exposure only, this study is considered to supply limited evidence of human carcinogenicity.

An excess lung cancer risk was also observed in three other studies which were, however, compromised by the presence of other carcinogens (arsenic, smoking) in the exposure or by a small population (Varner, 1983; Sorahan and Waterhouse, 1983; Armstrong and Kazantzis, 1983).

Four studies of workers exposed to cadmium dust or fumes provided evidence of a statistically significant positive association with prostate cancer (Kipling and Waterhouse, 1967; Lemen et al., 1976; Holden, 1980; Sorahan and Waterhouse, 1983), but the total number of cases was small in each study. The Thun et al. (1985) study is an update of an earlier study (Lemen et al., 1976) and does not show excess prostate cancer risk in these workers. Studies of human ingestion of cadmium are inadequate to assess carcinogenicity.

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o ANIMAL CARCINOGENICITY DATA :

Exposure of Wistar rats by inhalation to cadmium as cadmium chloride at concentrations of 12.5, 25 and 50 ug/cu.m for 18 months, with an additional 13-month observation period, resulted in significant increases in lung tumors (Takenaka et al., 1983). Intratracheal instillation of cadmium oxide did not produce lung tumors in Fischer 344 rats but rather mammary tumors in males and tumors at multiple sites in males (Sanders and Mahaffey, 1984). Injection site tumors and distant site tumors (for example, testicular) have been reported by a number of authors as a consequence of intramuscular or subcutaneous administration of cadmium metal and chloride, sulfate, oxide and sulfide compounds of cadmium to rats and mice (U.S. EPA, 1985). Seven studies in rats and mice where cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of a carcinogenic response.

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o SUPPORTING DATA :

Results of mutagenicity tests in bacteria and yeast have been inconclusive. Positive responses have been obtained in mutation assays in Chinese hamster cells (Dom and V79 lines) and in mouse lymphoma cells (Casto, 1976; Ochi and Ohsawa, 1983; 0berly et al., 1982).

Conflicting results have been obtained in assays of chromosomal aberrations in human lymphocytes treated in vitro or obtained from exposed workers. Cadmium treatment in vivo or in vitro appears to interfere with spindle formation and to result in aneuploidy in germ cells of mice and hamsters (Shimada et al., 1976; Watanabe et al., 1979; Gilliavod and Leonard, 1975).

CARO -

o CLASSIFICATION : B1; probable human carcinogen

o BASIS FOR CLASSIFICATION : Limited evidence from occupational epidemiologic studies of cadmium is consistent across investigators and study populations. There is sufficient evidence of carcinogenicity in rats and mice by inhalation and intramuscular and subcutaneous injection. Seven studies in rats and mice wherein cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of carcinogenic response.

o ORAL DOSE-RESPONSE DATA :

Not available. There are no positive studies of orally ingested cadmium suitable for quantitation.

CARI -

o CLASSIFICATION : B1; probable human carcinogen

o BASIS FOR CLASSIFICATION : Limited evidence from occupational epidemiologic studies of cadmium is consistent across investigators and study populations. There is sufficient evidence of carcinogenicity in rats and mice by inhalation and intramuscular and subcutaneous injection. Seven studies in rats and mice wherein cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of carcinogenic response.

o INHALATION UNIT RISK : 1.8E-3 per (ug/cu.m)

o DOSE EXTRAPOLATION METHOD : Two stage; only first affected by exposure; extra risk

o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	6E-2 ug/cu.m
E-5 (1 in 100,000)	6E-3 ug/cu.m
E-6 (1 in 1,000,000)	6E-4 ug/cu.m

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o INHALATION DOSE-RESPONSE DATA :

Tumor Type -- lung, trachea, bronchus cancer deaths
Test Animals -- human/white male
Route -- inhalation, exposure in the workplace
Reference -- Thun et al., 1985

Cumulative Exposure (mg/day/cu.m)	Median Observation	24 hour/ ug/cu.m Equivalent	No. of Expected Lung, Trachea and Bronchus Cancers Assuming No Cadmium Effect	Observed No. of Deaths (lung, trachea, bronchus cancers)
less than or equal to 584	280	168	3.77	2
585-2920	1210	727	4.61	7
greater than or equal to 2921	4200	2522	2.50	7

The 24-hour equivalent = median observation x IE+3 x 8/24 x 1/365 x 240/365.

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o ADDITIONAL COMMENTS :

The unit risk should not be used if the air concentration exceeds 6 ug/cu.m, since above this concentration the unit risk may not be appropriate.

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o DISCUSSION OF CONFIDENCE :

The data were derived from a relatively large cohort. Effects of arsenic and smoking were accounted for in the quantitative analysis for cadmium effects.

An inhalation unit risk for cadmium based on the Takenaka et al. (1983) analysis is 9.2E-2 per (ug/cu.m). While this estimate is higher than that

derived from human data [1.8E-3 per (ug/cu.m)] and thus more conservative, it was felt that the use of available human data was more reliable because of species variations in response and the type of exposure (cadmium salt vs. cadmium fume and cadmium oxide).

CARDR-
o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1985

The Addendum to the Cadmium Health Assessment has received both Agency and external review.
DOCUMENT

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o REVIEW DATES : 11/12/86
o VERIFICATION DATE : 11/12/86
o EPA CONTACTS :

William E. Pepelko / OHEA -- (202)260-5904

David Bayliss / OHEA -- (202)260-5726

WQCHU-

Water and Fish Consumption: 1E+1 ug/L

Fish Consumption Only: None

Considers technological or economic feasibility? -- NO

Discussion -- The criteria is the same as the existing standard for drinking water.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 3.9E+0 ug/L (1-hour average)
Chronic -- 1.1E+0 ug/L (4-day average)

Marine:

Acute -- 4.3E+1 ug/L (1-hour average)
Chronic -- 9.3E+0 ug/L (4-day average)

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. The freshwater criteria are hardness dependent. Values given here are calculated at a hardness of 100 mg/L CaCO3. A complete discussion can be found in the referenced notice.

Reference -- 50 FR 30784 (07/29/85)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 0.005 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- Cadmium has been classed as a Category III contaminant with an MCLG of 0.005 mg/L based upon reports of renal toxicity in humans. The MCLG is based upon a DWEL of 0.018 mg/L and an assumed drinking water contribution (plus aquatic organisms) of 25 percent. An uncertainty factor of 10 was also applied.

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value (status) -- 0.005 mg/L (Final, 1991)

Considers technological or economic feasibility? -- YES

Discussion -- EPA has promulgated an MCL equal to the established MCLG of 0.005 mg/L.

Monitoring requirements -- Ground water systems monitored every three years; surface water systems monitored annually; systems out of compliance must begin

monitoring quarterly until system is reliably and consistently below MCL.

Analytical methodology -- Atomic absorption/ furnace technique (EPA 213.2; SM 304); inductively coupled plasma (200.7): PQL= 0.002 mg/L.

Best available technology -- Coagulation/filtration; ion exchange; lime softening;
and reverse osmosis.

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

FISTD-

Status -- Voluntary Cancellation [cadmium chloride] (1990)

Reference -- 55 FR 31227 (08/01/90)

EPA Contact -- Registration Branch / OPP
(703)557-7760 / FTS 557-7760

FIREV-

Action -- Termination of Special Review (1991)

Considers technological or economic feasibility? -- YES

Summary of regulatory action -- All uses of cadmium pesticides cancelled. Criterion of concern: oncogenicity, mutagenicity, teratogenicity, and fetotoxicity.

Reference -- 56 FR 14522 (04/10/91)

EPA Contact -- Special Review Branch / OPP
(703)557-7400 / FTS 557-7400

CERC -

Value (status) -- 10 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The RQ for cadmium is 10 pounds, based on potential carcinogenicity. Available data indicate a hazard ranking of medium, based on a potency factor of 57.87/mg/kg/day and weight-of-evidence group B1, which corresponds to an RQ of 10 pounds. Cadmium has also been found to bioaccumulate in the tissues of aquatic and marine organisms, and has the potential to concentrate in the food chain. Reporting of releases of massive forms of this hazardous substance is not required if the diameter of the pieces released exceeds 100 micrometers (0.004 inches).

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Foulkes, E.C. 1986. Absorption of cadmium. In: Handbook of

Experimental Pharmacology, E.C. Foulkes, Ed. Springer Verlag, Berlin. Vol. 80, p. 75-100.
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OREF - Shaikh, Z.A. and J.C. Smith. 1980. Metabolism of orally ingested cadmium in humans. In: Mechanisms of Toxicity and Hazard Evaluation, B. Holmstedt et al., Ed. Elsevier Publishing Co., Amsterdam. p. 569-574.
OREF - U.S. EPA. 1985. Drinking Water Criteria Document on Cadmium. Office of Drinking Water, Washington, DC. (Final draft)
OREF - WHO (World Health Organization). 1972. Evaluation of certain food additives and the contaminants mercury, lead, and cadmium. Sixteenth Report of the Joint FAO/WHO Expert Committee on Food Additives. WHO Technical Report Series No. 505, FAO Nutrition Meetings Report Series No. 51. Geneva, Switzerland.
OREF - WHO (World Health Organization). 1984. Guidelines for drinking water quality -- recommendations. Vol. 1. Geneva, Switzerland.
IREF - None
CREF - Armstrong, B.G. and G. Kazantzis. 1983. The mortality of cadmium workers. Lancet. June 25, 1983: 1425-1427.
CREF - Casto, B. 1976. Letter to Richard Troast, U.S. EPA. Enclosing mutagenicity data on cadmium chloride and cadmium acetate.
CREF - Gilliavod, N. and A. Leonard. 1975. Mutagenicity tests with cadmium in the mouse. Toxicology. 5: 43-47.
CREF - Holden, H. 1980. Further mortality studies on workers exposed to cadmium fumes. Presented at Seminar on Occupational Exposure to Cadmium, March 20, 1980, London, England.
CREF - Kipling, M.D. and J.A.H. Waterhouse. 1967. Cadmium and prostatic carcinoma. Lancet. 1: 730.
CREF - Lemen, R.A., J.S. Lee, J.K. Wagoner and H.P. Blejer. 1976. Cancer mortality among cadmium production workers. Ann. N.Y. Acad. Sci. 271: 273.
CREF - Oberly, T., C.E. Piper and D.S. McDonald. 1982. Mutagenicty of metal salts in the L5178 Y mouse lymphoma assay. J. Toxicol. Environ. Health. 9: 367-376.
CREF - Ochi, T. and M. Ohsawa. 1983. Induction of 6-thioguanine-resistant mutants and single-strand scission DNA by cadmium chloride in cultured Chinese hamster cells. Mutat. Res. 111: 69-78.
CREF - Sanders, C.L. and J.A. Mahaffey. 1984. Carcinogenicity of single and multiple intratracheal instillations of cadmium oxide in the rat. Environ. Res. 33: 227-233.
CREF - Shimada, T., T. Watanabe and A. Endo. 1976. Potential mutagenicity of cadmium in mammalian oocytes. Mutat. Res. 40: 389-396.
CREF - Sorahan, T. and J.A.H. Waterhouse. 1983. Mortality study of nickel-cadmium battery workers by the method of regression models in life tables. Br. J. Ind. Med. 40: 293-300.
CREF - Takenaka, S., H. Oldiges, H. Konig, D. Hochrainer and G. Oberdoerster. 1983. Carcinogenicity of cadmium aerosols in Wistar rats. J. Natl. Cancer Inst. 70: 367-373.

CREF - Thun, M.J., T.M. Schnorr, A.B. Smith and W.E. Halperin. 1985. Mortality among a cohort of U.S. cadmium production workers: An update. J. Natl. Cancer Inst. 74(2): 325-333.
CREF - U.S. EPA. 1985. Updated Mutagenicity and Carcinogenicity Assessment of Cadmium. Addendum to the Health Assessment Document for Cadmium (EPA 600/B- B1-023). EPA 600/B-83-025F.
CREF - Varner, M.O. 1983. Updated epidemiologic study of cadmium smelter workers. Presented at the Fourth International Cadmium Conference. Unpublished.
CREF - Watanabe, T., T. Shimada and A. Endo. 1979. Mutagenic effects of cadmium on mammalian oocyte chromosomes. Mutat. Res. 67: 349-356.
HAREF- None

[IRIS] SS 21 /cf?
USER:
7440-47-3
Search in progress

SS (21) PSTG (2)

[IRIS] SS 22 /cf?
USER:

prt dl ncar, car continuous

1 - IRIS
IRSN - 27
DATE - 920604
UPDT - 06/04/92, 52 fields
STAT - Oral RfD Assessment (RDO) on-line 03/01/88
STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) pending 05/01/92
STAT - Drinking Water Health Advisories (DWHA) on-line 11/01/90
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 03/01/88 RDO Critical effect added
IRH - 03/01/88 HADV Health Advisory added
IRH - 08/01/89 REFS Bibliography on-line
IRH - 08/01/90 RCRA EPA contact changed
IRH - 10/01/90 RDI Inhalation RfC now under review
IRH - 11/01/90 HADV Full Health Advisory summary added
IRH - 01/01/92 RDO Secondary contact changed
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 05/01/92 CAR Carcinogenicity assessment now under review
RLEN - 15079
NAME - Chromium(III)
RN - 16065-83-1
SY - 7440-47-3
SY - CHROMIC ION
SY - CHROMIUM
SY - Chromium(III)
SY - CHROMIUM (III) ION
SY - CHROMIUM, ION

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
No effects observed Rat Chronic Feeding Study Ivankovic and Preussmann, 1975	NOEL: 5% Cr2O3 in diet 5 days/week for 600 feedings (1800 g/kg bw average total dose) LOAEL: none	100	10	1E+0 mg/kg/day (as an insoluble salt)

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*Dose Conversion Factors & Assumptions: 1800 g Cr2O3/kg bw x 1000 mg/g x 0.6849 Cr/g Cr2O3 / 600 feeding days x 5 feeding days/7 days = 1468 mg/kg/day

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o ORAL RFD STUDIES :

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

Groups of 60 male and female rats were fed chromic oxide (Cr2O3) baked in bread at dietary levels of 0, 1, 2, or 5%, 5 days/week for 600 feedings (840 total days). The primary purpose of this study was to assess the carcinogenic potential of Cr2O3. Body weight and food consumption were monitored. The average total amounts of ingested Cr2O3 were given as 360, 720, and 1800 g/kg bw for the 1, 2, and 5% treatment groups, respectively. The animals were maintained on control diets following termination of exposure until they became moribund or died. All major organs were examined histologically. Other toxicologic parameters were not mentioned explicitly, but may have included some or all of those described for the accompanying subchronic study (see below). No effects due to Cr2O3 treatment were observed at any dose level.

Ivankovic and Preussmann (1975) also treated rats (both sexes, 12-19 rats/group) at dietary levels of 0, 2, or 5% Cr2O3 in bread, 5 days/week for 90 days. Food consumption and body weight were monitored. Toxicologic parameters included serum protein, bilirubin, hematology, urinalysis, organ weights, and histopathology. The only effects observed were reductions (12-37%) in the absolute weights of the livers and spleens of animals in the high-dose group. Organ weights relative to body weight were not reported. The high dose is equivalent to 1400 mg/kg/day (dose converted using reported data).

Other subchronic oral studies show no indication of adverse effects attributable to trivalent chromium compounds, but dose levels were considerably lower.

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o ORAL RFD UNCERTAINTY :

UF = 100. The factor of 100 represents two 10-fold decreases in mg/kg bw/day dose that account for both the expected interhuman and interspecies variability to the toxicity of the chemical in lieu of specific data.

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o ORAL RFD MODIFYING FACTOR :

MF = 10. The additional modifying factor of 10 is adopted to reflect uncertainty in the NOEL because: 1) the effects observed in the 90-day study were not explicitly addressed in the 2-year study and, thus, the highest NOAEL in the 2-year study may be a LOAEL; 2) the absorption of chromium is low (<1%) and is influenced by a number of factors; thus, a considerable potential variation in absorption exists; and 3) animals were allowed to die naturally after feeding stopped (2 years) and only then was histology performed.

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o ORAL RFD COMMENTS :

This RfD is limited to metallic chromium (III) of insoluble salts. Examples of insoluble salts include chromic III oxide (Cr203) and chromium III sulfate [Cr2(SO4)3].

Very limited data suggest that Cr III may have respiratory effects on humans. No data on chronic or subchronic effects of inhaled Cr III in ani- mals can be found. Adequate teratology data do not exist, but reproductive effects are not seen at dietary levels of 5% Cr2O3.

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o ORAL RFD CONFIDENCE :

Study: Low
Data Base: Low
RfD: Low

The principal study is rated low because of the lack of explicit detail on study protocol and results. Low confidence in the data base reflects the lack of high-dose supporting data. The low confidence in the RfD reflects the foregoing, but also reflects the lack of an observed effect level. Thus, the RfD, as given, should be considered conservative, since the MF addresses only those factors which might lower the RfD.

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o ORAL RFD SOURCE DOCUMENT :

U.S. EPA. 1984. Health Effects Assessment for Trivalent Chromium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, OHEA for the Office of Solid Waste and Emergency Response.

The ADI in the 1984 Health Effects Assessment document received an Agency review with the help of two external scientists.

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o REVIEW DATES : 11/21/85, 02/05/86
o VERIFICATION DATE : 11/21/85
o EPA CONTACTS :

Michael L. Dourson / ORD -- (513)569-7544 / FTS 684-7544

Robert Bruce / ORD -- (513)569-7553 / FTS 684-7553

RDI -
o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

HAONE-

NOTE: All chromium HAs are based on total chromium (III and VI).

Appropriate data for calculating a One-day HA are not available. It is recommended that the Ten-day HA of 1.4 mg/L be used as the One-day HA.

HATEN-

NOTE: All chromium HAs are based on total chromium (III and VI).

Ten-day HA -- 1.4E+0 mg/L

NOAEL -- 14.4 mg/kg/day
UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)
Assumptions -- 1 L/day water consumption for a 10-kg child

Principal Study -- Gross and Heller, 1946

Rats were exposed to drinking water containing Cr(VI) (K_2CrO_4) at levels of 80 or 134 mg Cr(VI)/L for 60 days (8.3 or 14.4 mg Cr(VI)/kg/day, respectively) without adverse effects. Therefore, a NOAEL of 14.4 mg/kg/day is identified.

HALTC-

NOTE: All chromium HAs are based on total chromium (III and VI).

Longer-term (Child) HA -- 2.4E-1 mg/L

NOAEL -- 2.4 mg/kg/day
UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)
Assumptions -- 1 L/day water consumption for a 10-kg child

Principal study -- MacKenzie et al., 1958

In a 1-year drinking water study, consumption of water containing either Cr(III) ($CrCl_3$) or Cr(VI) (K_2CrO_4) (0 to 1.87 mg/kg/day for male rats and 0 to 2.41 mg/kg/day for female rats) produced no significant differences in weight gain, appearance, or pathological changes in the blood or other tissue. Therefore, a NOAEL of 2.41 mg/kg/day is identified.

HALTA-

NOTE: All chromium HAs are based on total chromium (III and VI).

Longer-term (Adult) HA -- 8.4E-1 mg/L

NOAEL -- 2.4 mg/kg/day
UF -- 100 (allows for interspecies and intrahuman variability with the use of a NOAEL from an animal study)
Assumptions -- 2 L/day water consumption for a 70-kg adult

Principal study -- MacKenzie et al., 1958 (study described in HALTC)

HALIF-

NOTE: All chromium HAs are based on total chromium (III and VI).

Drinking Water Equivalent Level (DWEL) -- 1.7E-1 mg/L

Assumptions -- 2 L/day water consumption for a 70-kg adult

RfD Verification Date = 02/05/86 (see RDO)

Lifetime HA -- 1.2E-1 mg/L

Assumptions -- 71% exposure by drinking water

Principal study -- MacKenzie et al., 1958 (This study was used in the derivation of the chronic oral RfD; see RDO)

OLEP -

No data available

ALAB -

Determination of chromium is by an atomic absorption technique using either direct aspiration into a flame or a furnace.

TREAT-

The treatment technologies that are available to remove chromium from water include coagulation/filtration, lime softening, ion exchange, and reverse osmosis.

HADR -
o HEALTH ADVISORY SOURCE :

U.S. EPA. 1985. Draft of the Drinking Water Criteria Document on Chromium. Office of Drinking Water, Washington, DC.
DOCUMENT

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o HEALTH ADVISORY REVIEW :

EPA review of HAs in 1985.

Public review of HAs following notification of availability in October, 1985.

Scientific Advisory Panel review of HAs in January, 1986.

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o EPA DRINKING WATER CONTACT :

Kenneth Bailey / ODW -- (202)260-5535 / FTS 260-5535

Edward V. Ohanian / ODW -- (202)260-7571 / FTS 260-7571

WQCHU-

Water and Fish Consumption: 1.7E+5 ug/L

Fish Consumption Only: 3.433E+6 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of 1.7E+5 ug/L is based on consumption of contaminated aquatic organisms and water. A WQC of 3.433E+6 ug/L has also been established based on consumption of contaminated aquatic organisms alone.

Reference -- 45 FR 79318 (11/28/80); 50 FR 30784 (07/29/85)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 9.8E+2 ug/L (hardness dependent)
Chronic -- 1.2E+2 ug/L (hardness dependent)

Marine:

Acute LEC -- 1.03 E+ 4 ug/L
Chronic LEC -- none

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. For freshwater aquatic life the concentration (in ug/L) of total recoverable trivalent chromium should not exceed the numerical value given by the equations "e**(0.8190 [ln (hardness)]+3.688)" for acute exposure and "e**(0.8190 [ln (hardness)]+1.561)" for chronic exposure (** indicates exponentiation; hardness is in mg/L). For example, at a hardness of 50 mg/L, the acute and chronic WQC would be 980 and 120 ug/L, respectively. The values that are indicated as "LEC" are not criteria, but are the lowest effect levels found in the literature. LEC's are given when the minimum data required to derive water quality criteria are not available.

Reference -- 50 FR 30784 (07/29/85)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 0.1 mg/L [total chromium] (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- An MCLG of 0.1 mg/L for total chromium (Cr III and Cr VI) is based on EPA's RfD methodology for Cr VI, the more toxic chromium species. The MCLG is based upon a DWEL of 0.17 mg/L calculated from available human and animal data and an assumed drinking water contribution of 20 percent. An uncertainty factor of 500 was applied. The MCLG also falls into the safe and adequate daily dietary intake range of 50 to 200 mg/day for Cr III established by the National Research Council in the National Academy of Sciences (NAS, 1989).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value (status) -- 0.1 mg/L [total chromium] (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- The EPA has established an MCL equal to the MCLG of 0.1 mg/L.

Monitoring requirements -- Ground water systems monitored every three years; surface water systems monitored annually; systems out of compliance must begin monitoring quarterly until system is reliably and consistently below MCL.

Analytical methodology -- Atomic absorption/furnace technique (EPA 218.2; SM 304); inductively coupled plasma (EPA 200.7): PQL= 0.01 mg/L.

Best available technology -- Coagulation/filtration; ion exchange; lime softening; and reverse osmosis.

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

CERC -

Value (status) -- 1 pound (Statutory, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- Though "Chromium (III), insoluble salts" is not specifically designated as a CERCLA hazardous substance, insoluble chromium (III) salts would be considered hazardous substances under the CERCLA broad generic listing for "Chromium and Compounds." There is no corresponding reportable quantity (RQ) for this generic class of compounds. However, the releaser is still liable for cleanup costs if the designated Federal On-Scene Coordinator (OSC) decides to take response action with respect to the release of an insoluble chromium (III) salt that is not otherwise specifically listed as a

CERCLA hazardous substance. There are two chromium (III) salts which are specifically listed as CERCLA hazardous substances, chromic acetate and chromic sulfate. Both have been assigned final RQs of 1000 pounds based on aquatic toxicity (as established under section 311(b)(4) of the Clean Water Act). Metallic chromium has been assigned a final RQ of 5000 pounds.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed (total chromium)

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

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

OREF - U.S. EPA. 1984. Health Effects Assessment for Trivalent Chromium. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH. OHEA for the Office of Solid Waste and Emergency Response, Washington, DC.

IREF - None

CREF - None

HAREF- Gross, W.G., and V.G. Heller. 1946. Chromates in animal nutrition. J. Ind. Hyg. Toxicol. 28: 52-56.

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

HAREF- U.S. EPA. 1985. Draft of the Drinking Water Criteria Document on Chromium. Office of Drinking Water, Washington, DC.

prt dl ncar, car continuous

1 - IRIS
IRSN - 613
DATE - 920604
UPDT - 06/04/92, 52 fields
STAT - Oral RfD Assessment (RDO) pending 05/01/92
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) no data
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) no data
IRH - 05/01/92 RDO Oral reference dose now under review
IRH - 06/01/92 SY Synonyms added
RLEN - 613
NAME - Cobalt
RN - 7440-48-4
SY - Cobalt
SY - C.I. 77320
SY - CCRIS 1575
SY - COBALT-59
SY - HSDB 519
SY - Kobalt [German, Polish]
SY - NCI-C60311

RDO -
o ORAL RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

[IRIS] SS 29 /cf?
USER:
7440-50-8
Search in progress

SS (29) PSTG (1)

[IRIS] SS 30 /cf?
USER:

prt dl ncar, car continuous

1 - IRIS
IRSN - 367
DATE - 920122
STAT - Oral RfD Assessment (RDO) no data
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 08/01/91
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 09/07/88 CAR Carcinogen summary on-line
IRH - 08/01/91 CAREV Law citation year corrected
IRH - 08/01/91 REFS Bibliography on-line
IRH - 01/01/92 EXSR Regulatory Action section on-line
RLEN - 11298
NAME - Copper
RN - 7440-50-8
SY - Copper
CAREV-
o CLASSIFICATION : D; not classified
o BASIS FOR CLASSIFICATION : There are no human data, inadequate animal data from assays of copper compounds, and equivocal mutagenicity data.
o HUMAN CARCINOGENICITY DATA :

None.

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o ANIMAL CARCINOGENICITY DATA :

Inadequate. Bionetics Research Labs (1968) studied the carcinogenicity of a copper-containing compound, copper hydroxyquinoline, in two strains of mice (B6C3F1 and B6AKF1). Groups of 18 male and 18 female 7-day-old mice were administered 1000 mg copper hydroxyquinoline/kg bw (180.6 mg Cu/kg) suspended in 0.5% gelatin daily until they were 28 days old, after which they were administered 2800 ppm (505.6 ppm Cu) in the feed for 50 additional weeks. No statistically significant increases in tumor incidence were observed in the treated 78-week-old animals.

In the same study, Bionetics Research Labs (1968) administered a single subcutaneous injection of gelatin (control) or 1000 mg of copper hydroxyquinoline/kg bw (180.6 mg Cu/kg) suspended in 0.5% gelatin to groups of 28-day-old mice of both strains. After 50 days of observation, the male B6C3F1 had an increased incidence of reticulum cell sarcomas compared with controls. No tumors were observed in the treated male B6AKF1 mice, and a low incidence of reticulum cell sarcomas was observed in the treated female mice of both strains.

Gilman (1962) administered intramuscular injections containing 20 mg of

cupric oxide (16 mg Cu), cupric sulfide (13.3 mg Cu), and cuprous sulfide (16 mg Cu) into the left and right thighs of 2- to 3-month-old Wistar rats. After 20 months of observations, no injection-site tumors were observed in any animals, but other tumors were observed at very low incidence in the animals receiving cupric sulfide (2/30) and cuprous sulfide (1/30). As the relevance of the organic copper compound to the observation of sarcoma induction is uncertain and the incidence of tumors in rats treated i.m. with inorganic copper was very low, data are considered inadequate for classification.

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o SUPPORTING DATA :

Moriya et al. (1983) reported no increase in mutations in E. coli and S. typhimurium strains TA98, TA1535, TA1537 and TA1538 incubated with up to 5 mg copper quinolinolate/plate and in S. typhimurium TA98 and TA100 incubated with up to 5 mg copper sulfate/plate. Demerec et al. (1951) reported dose-related mutagenic effects in E. coli with 2 to 10 ppm copper sulfate in a reverse mutation assay. Negative results were obtained with copper sulfate or copper chloride in assays using S. cerevisiae (Singh, 1983) and Bacillus subtilis (Nishioka, 1975, Matsui, 1980, Kanematsu et al., 1980). Errors in DNA synthesis from poly(c)templates have been induced in viruses incubated with copper chloride or copper acetate (Sirover and Loeb, 1976). Chromosomal aberrations were induced in isolated rat hepatocytes when incubated with copper sulfate (Sina et al., 1983). Casto et al. (1979) showed enhanced cell transformation in Syrian hamster embryo cells infected with simian adenovirus with the addition of cuprous sulfide and copper sulfate. High concentrations of copper compounds have been reported to induce mitosis in rat ascites cells and recessive lethals in Drosophila melanogaster. Law (1938) reported increases in the percent lethals observed in Drosophila larvae and eggs when exposed to copper by microinjection (0.1% copper sulfate) or immersion (concentrated aqueous copper sulfate), respectively.

CARDR-
o CARCINOGENICITY SOURCE :

U.S. EPA. 1987. Drinking Water Criteria Document for Copper. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. ECAO-CIN 417.

The values in the 1987 Drinking Water Criteria Document for Copper have received peer and administrative review.
DOCUMENT

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o REVIEW DATES : 09/15/87
o VERIFICATION DATE : 09/15/87
o EPA CONTACTS :

David J. Reisman / ORD -- (513)569-7588 / FTS 684-7588

W. Bruce Peirano / ORD -- (513)569-7540 / FTS 684-7540

WQCHU-

No data available

WQCAQ-

Freshwater:

Acute -- 9.2E+0 ug/L (hardness dependent)
Chronic -- 6.5E+0 ug/L (hardness dependent)

Marine:

Acute -- 2.9E+0 ug/L
Chronic -- None

Considers technological or economic feasibility? -- NO

Discussion -- For freshwater aquatic life the concentration (in ug/L) of total recoverable copper should not exceed the numerical value given by the equations "e**(0.9422 [ln (hardness)]-1.464)" for acute exposure and "e**(0.8545[ln (hardness)]-1.465)" for chronic exposure (** indicates exponentiation; hardness is in mg/L). For example at a hardness of 50 mg/L, the acute and chronic WQC would be 9.2 and 6.5 ug/L, respectively.

Reference -- 45 FR 79318 (11/28/80); 50 FR 30784 (07/29/85)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value -- 1.3 mg/L (Final,1991)

Considers technological or economic feasibility? -- NO

Discussion -- The MCLG of 1.3 mg/L for copper is based on potential adverse effects (gastrointestinal) reported in human studies.

Reference -- 53 FR 31516 (08/18/88); 56 FR 26460 (06/07/91); 56 FR 32112 (07/15/91)

EPA Contact -- Health and Ecological Criteria Division / OST / (202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- None (Final, 1991)

Considers technological or economic feasibility? -- YES

Discussion -- EPA concluded that setting an MCL for copper is not feasible and believes that the treatment approach contained in the final rule (corrosion control, source water reduction, public education and copper service line replacement) will achieve the public health goals of the SDWA without the problems associated with establishing MCL's.

Monitoring requirements -- Ground water systems monitored annually; surface water systems monitored quarterly; repeat monitoring dependent upon detection and compliance history with a minimum of 5 years between sampling; community and non-transient non-community water systems to have different monitoring requirements for determining compliance with corrosion control treatment techniques.

Analytical methodology -- Atomic absorption/furnace technique (EPA 220.2; ASTM D1688-90C; SM 3113); atomic absorption/direct aspiration (EPA 220.1; ASTM D1688-90A; SM 3111-B); inductively-coupled plasma (EPA 200.7; SM 3120); inductively-coupled plasma/mass spectrometry (EPA 200.8); atomic absorption/ platform furnace (EPA 200.9).

Best available technology -- Coagulation/filtration; ion exchange; lime softening; reverse osmosis.

Reference -- 53 FR 31516 (08/18/88); 56 FR (06/07/91); 56 FR 32112 (07/15/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

Value -- 1 mg/L (Proposed, 1988)

Considers technological or economic feasibility? -- NO

Discussion -- SMCLs are non-enforceable and establish limits for contaminants which may affect the aesthetic qualities (e.g. taste and odor) of drinking water It is recommended that systems monitor for these contaminants every three years. More frequent monitoring for contaminants such as pH, color, odor or others may be appropriate under certain circumstances.

Reference -- 53 FR 31516 (08/18/88)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

FISTD-

Status -- Issued (1987)

Reference -- Copper Compounds Pesticide Registration Standard. April, 1987. (NTIS No. PB87-190344)

EPA Contact -- Registration Branch / OPP (703)557-7760 / FTS 557-7760

FIREV-

No data available

CERC -

Value -- 5000 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- No data have been found to permit the ranking copper. Since it is designated a hazardous substance, the largest assignable RQ is 5000 pounds. Copper is currently being assessed for chronic

toxicity and is subject to change in future rulemaking. Reporting of releases of massive forms of this substance is not required if the diameter of the pieces released exceeds 100-micrometers (0.004 inches).

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800) 424-9346 / (202) 260-3000 / FTS 260-3000

TSCA -

No data available

OREF - None
IREF - None
CREF - Casto, B.C., J. Meyers and J.A. DiPaolo. 1979. Enhancement of viral transformation for evaluation of the carcinogenic or mutagenic potential of inorganic metal salts. Cancer Res. 39: 193-198.
CREF - Demerec, M., G. Bertani and J. Flint. 1951. A survey of chemicals for mutagenic action on E. coli. Am. Natur. 85(821): 119-136.
CREF - Gilman, J.P.W. 1962. Metal carcinogenesis. II. A study on the carcinogenic activity of cobalt, copper, iron and nickel compounds. Cancer Res. 22: 158-166.
CREF - Kanematsu, N., M. Hara and T. Kada. 1980. Rec assay and mutagenicity studies on metal compounds. Mutat. Res. 77: 109-116.
CREF - Law, L.W. 1938. The effects of chemicals on the lethal mutation rate in drosophilia melanogaster. Proc. Natl. Acad. Sci., USA. 24: 546-550.
CREF - Matsui, S. 1980. Evaluation of a Bacillus subtilis rec-assay for the detection of mutagens which may occur in water environments. Water Res. 14(11): 1613-1619.
CREF - Moriya, M., T. Ohta, K. Watanabe, T. Miyazawa, K. Kato and Y. Shirasu. 1983. Further mutagenicity studies on pesticides in bacterial reversion assay systems. Mutat. Res. 116(3-4): 185-216.
CREF - NCI (National Cancer Institute). 1968. Evaluation of carcinogenic, teratogenic and mutagenic activities of selected pesticides and industrial chemicals. Vol. I. NCI-DCCP-CG-1973-1-1.
CREF - Nishioka, H. 1975. Mutagenic activities of metal compounds in bacteria. Mutat. Res. 31: 185-189.
CREF - Sina, J.F., C.L. Bean, G.R. Dysart, V.I. Taylor and M.O. Bradley. 1983. Evaluation of the alkaline elution/rat hepatocyte assay as a predictor of carcinogenic/mutagenic potential. Mutat. Res. 113(5): 357-391.
CREF - Singh, I. 1983. Induction of reverse mutation and mitotic gene conversion by some metal compounds in Saccharomyces cerevisiae.

Mutat. Res. 117(1-2): 149-152.
CREF - Sirover, M.A. and L.A. Loeb. 1976. Infidelity of DNA synthesis in vitro: Screening for potential metal mutagens or carcinogens. Science. 194: 1434-1436.
CREF - U.S. EPA. 1987. Drinking Water Criteria Document for Copper. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.
HAREF- None

[IRIS] SS 30 /cf?
USER:
57-12-5
Search in progress

SS (30) PSTG (1)

[IRIS] SS 31 /cf?
USER:

pr ^Ht dl ncar, car continuous

1 - IRIS
IRSN - 271
DATE - 931101
UPDT - 11/01/93, 2 fields
STAT - Oral RfD Assessment (RDO) message 02/01/91
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 11/01/93
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 06/01/92
IRH - 09/26/88 CAR Carcinogen summary on-line
IRH - 02/01/89 MCLG Effect level corrected in discussion
IRH - 06/01/89 CARDR Primary contact changed
IRH - 06/01/89 CAA Reference corrected - changed number for part in CFR
IRH - 12/01/89 CAREV Last paragraph - Correct Van Esch 1969 citation
IRH - 12/01/89 REFS Bibliography on-line
IRH - 07/01/90 RDO Changed contact J. Cohen's office and telephone number
IRH - 07/01/90 RCRA EPA contact changed
IRH - 02/01/91 RDO Message revised to include new EPA document
IRH - 02/01/91 RDO EPA contacts changed
IRH - 05/01/91 CAREV Text edited
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 06/01/92 MCL MCL monitoring reqs. and BAT corrected
IRH - 07/01/93 CARDR Secondary contact's phone number changed
IRH - 07/01/93 CREF References alphabetized correctly
IRH - 11/01/93 CARDR U.S. EPA 1987 replaced with 1989; rev. state. revised
IRH - 11/01/93 CREF U.S. EPA 1987 deleted; U.S. EPA 1989 added
RLEN - 18690
NAME - Lead and compounds (inorganic)
RN - 7439-92-1
SY - Lead
SY - Lead and compounds
SY - plumbum

RDO -
o ORAL RFD SUMMARY :

A great deal of information on the health effects of lead has been obtained through decades of medical observation and scientific research. This information has been assessed in the development of air and water quality criteria by the Agency's Office of Health and Environmental Assessment (OHEA) in support of regulatory decision-making by the Office of Air Quality Planning and Standards (OAQPS) and by the Office of Drinking Water (ODW). By comparison to most other environmental toxicants, the degree of uncertainty about the health effects of lead is quite low. It appears that some of these effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead

levels so low as to be essentially without a threshold. The Agency's RfD Work Group discussed inorganic lead (and lead compounds) at two meetings (07/08/85 and 07/22/85) and considered it inappropriate to develop an RfD for inorganic lead. For additional information, interested parties are referred to the 1986 Air Quality Criteria for Lead (EPA-600/8-83/028a-dF) and its 1990 Supplement (EPA/600/8-89/049F) or the following Agency scientists:

Harlal Choudhury / OHEA -- (513)569-7536

J. Michael Davis / OHEA -- (919)541-4162

Jeff Cohen / OST -- (202)260-5456

John Haines / OAQPS -- (919)541-5533

CAREV-
o CLASSIFICATION : B2; probable human carcinogen
o BASIS FOR CLASSIFICATION : Sufficient animal evidence. Ten rat bioassays and one mouse assay have shown statistically significant increases in renal tumors with dietary and subcutaneous exposure to several soluble lead salts. Animal assays provide reproducible results in several laboratories, in multiple rat strains with some evidence of multiple tumor sites. Short term studies show that lead affects gene expression. Human evidence is inadequate.
o HUMAN CARCINOGENICITY DATA :

Inadequate. There are four epidemiologic studies of occupational cohorts exposed to lead and lead compounds. Two studies (Dingwall-Fordyce and Lane, 1963; Nelson et al., 1982) did not find any association between exposure and cancer mortality. Selevan et al. (1985), in their retrospective cohort mortality study of primary lead smelter workers, found a slight decrease in the total cancer mortality (SMR=95). Apparent excesses were observed for respiratory cancer (SMR=111, obs=41, $p>0.05$) and kidney cancer (SMR=204, obs=6, $p>0.05$). Cooper and Gaffey (1975) and Cooper (1985 update) performed a cohort mortality study of battery plant workers and lead smelter workers. They found statistically significant excesses for total cancer mortality (SMR=113, obs=344), stomach cancer (SMR=168, obs=34), and lung cancer (SMR=124, obs=109) in the battery plant workers. Although similar excesses were observed in the smelter workers, they were not statistically significant. Cooper and Gaffey (1975) felt it was possible that individual subjects were monitored primarily on the basis of obvious signs of lead exposure, while

others who showed no symptoms of lead poisoning were not monitored.

All of the available studies lacked quantitative exposure information, as well as information on the possible contribution from smoking. All studies also included exposures to other metals such as arsenic, cadmium, and zinc for which no adjustment was done. The cancer excesses observed in the lung and stomach were relatively small (<200). There was no consistency of site among the various studies, and no study showed any dose-response relationship. Thus, the available human evidence is considered to be inadequate to refute or demonstrate any potential carcinogenicity for humans from lead exposure.

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o ANIMAL CARCINOGENICITY DATA :

Sufficient. The carcinogenic potential of lead salts (primarily phosphates and acetates) administered via the oral route or by injection has been demonstrated in rats and mice by more than 10 investigators. The most characteristic cancer response is bilateral renal carcinoma. Rats given lead acetate or subacetate orally have developed gliomas, and lead subacetate also produced lung adenomas in mice after i.p. adminstration. Most of these investigations found a carcinogenic response only at the highest dose. The lead compounds tested in animals are almost all soluble salts. Metallic lead, lead oxide and lead tetralkyls have not been tested adequately. Studies of inhalation exposure have not been located in the literature.

Azar et al. (1973) administerd 10, 50, 100, and 500 ppm lead as lead acetate in dietary concentrations to 50 rats/sex/group for 2 years. Control rats (100/sex) received the basal laboratory diet. In a second 2-year feeding study, 20 rats/group were given diets containing 0, 1000, and 2000 ppm lead as lead acetate. No renal tumors were reported in the control groups or in treated animals of either sex receiving 10 to 100 ppm. Male rats fed 500, 1000, and 2000 ppm lead acetate had an increased renal tumor incidence of 5/50, 10/20, and 16/20, while 7/20 females in the 2000-ppm group developed renal tumors.

The Azar et al. (1973) study is limited by the lack of experimental detail. The possibility of environmental contamination from lead in the air or drinking water was not mentioned. The strains of rats used were not specified in the study, but the Health Effects Assessment for Lead (U.S. EPA, 1984) indicates the rats were Wistar strain. The weight gain at 1000 and 2000 ppm was reported to be depressed, but details were not given.

Kasprzak et al. (1985), in investigating the interaction of dietary calcium on lead carcinogenicity, fed 1% lead subacetate (8500 ppm Pb) to male Sprague-Dawley rats in the diet for 79 weeks. Of the rats surviving (29/30) in this treatment group beyond 58 weeks, 44.8% had renal tumors. Four rats had adenocarcinomas; the remainaing nine had adenomas. Bilateral tumors were noted. No renal tumors were noted among the controls.

As part of a study to determine interactions between sodium nitrite, ethyl urea and lead, male Sprague-Dawley rats were given lead acetate in their

drinking water for 76 weeks (Koller et al., 1986). The concentration of lead was 2600 ppm. No kidney tumors were detected among the 10 control rats. Thirteen of 16 (81%) lead-treated rats had renal tubular carcinoma; three tumors were detected at 72 weeks and the remainder detected at the termination of the study.

Van Esch and Kroes (1969) fed basic lead acetate at 0, 0.1%, and 1.0% in the diet to 25 Swiss mice/sex/group for 2 years. No renal tumors developed in the control group, but 6/25 male mice of 0.1% basic lead acetate group had renal tumors (adenomas and carcinomas combined). In the 1.0% group, one female had a renal tumor. The authors thought that the low incidence in the 1.0% group was due to early mortality.

Hamsters given lead subacetate at 0.5% and 1% in the diet had no significant renal tumor response (Van Esch and Kroes, 1969).

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o SUPPORTING DATA :

Lead acetate induces cell transformation in Syrian hamster embryo cells (DiPaolo et al., 1978) and also enhances the incidence of simian adenovirus induction. Lead oxide showed similar enhanced adenovirus induction (Casto et al., 1979).

Under certain conditions lead compounds are capable of inducing chromosomal aberrations in vivo and in tissue cultures. Grandjean et al. (1983) showed a relationship between SCE and lead exposure in exposed workers. Lead has been shown, in a number of DNA structure and function assays, to affect the molecular processes associated with the regulation of gene expression (U.S. EPA, 1986).

CARO -

o CLASSIFICATION : B2; probable human carcinogen

o BASIS FOR CLASSIFICATION : Sufficient animal evidence. Ten rat bioassays and one mouse assay have shown statistically significant increases in renal tumors with dietary and subcutaneous exposure to several soluble lead salts. Animal assays provide reproducible results in several laboratories, in multiple rat strains with some evidence of multiple tumor sites. Short term studies show that lead affects gene expression. Human evidence is inadequate.

o ORAL DOSE-RESPONSE DATA :

Not available.

Quantifying lead's cancer risk involves many uncertainties, some of which may be unique to lead. Age, health, nutritional state, body burden, and exposure duration influence the absorption, release, and excretion of lead. In addition, current knowledge of lead pharmacokinetics indicates that an estimate derived by standard procedures would not truly describe the potential risk. Thus, the Carcinogen Assessment Group recommends that a numerical estimate not be used.

CARDR-
o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1984, 1986, 1989

U.S. EPA, 1989 has received OHEA and SAB review.

The 1986 Air Quality Criteria Document for Lead has received Agency and External Review.
DOCUMENT

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o REVIEW DATES : 05/04/88
o VERIFICATION DATE : 05/04/88
o EPA CONTACTS :

William Pepelko / OHEA -- (202)260-5898

Jim Cogliano / OHEA -- (202)260-3814

CAA -

Considers technological or economic feasibility? -- No

Discussion -- Under Section 109 of the CAA, EPA has set a primary (health-based) NAAQS for lead of 1.5 ug/cu.m, calendar quarter average not to be exceeded (43 FR 41258, 10/05/78). The secondary (welfare-based) NAAQS is identical to the primary standard. EPA is currently reviewing these standards to determine if changes are warranted.

Reference -- 40 CFR 50.12

U.S. EPA Contact -- Air Quality Management Division / OAQPS / (919)541-5656 / FTS 629-5656

WQCHU-

Water and Fish Consumption -- 5.0E+1 ug/L

Fish Consumption Only -- None

Considers technological or economic feasibility? -- NO

Discussion -- The criterion was set at the existing drinking water standard in 1980.

Reference -- 45 FR 79318 (11/28/80)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 8.2E+1 ug/L (1-hour average)
Chronic -- 3.2E+0 ug/L (4-day average)

Marine:

Acute -- 1.40E+2 ug/L (1-hour average)
Chronic -- 5.6E+0 ug/L (4-day average)

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. The toxicity of this compound in freshwater is hardness dependent. The values given are for a hardness of 100 mg/L CaCO3. For a more complete discussion, see the referenced notice.

Reference -- 50 FR 30784 (07/29/85)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value (status) -- 0 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- The MCLG for lead is zero based on (1) occurrence of low level effects and difficulties in identifying clear threshold levels, (2) the overall Agency goal of reducing total lead exposures, and (3) the classification of lead as a group B2 carcinogen.

Reference -- 56 FR 26460 (06/07/91); 56 FR 32112 (07/15/91)

EPA Contact -- Health and Ecological Criteria Division / OST / (202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- None (Final, 1991)

Considers technological or economic feasibility? -- YES

Discussion -- EPA concluded that setting an MCL for lead is not feasible and believes that the treatment approach contained in the final rule (corrosion control, source water reduction, public education and lead service line problems associated with establishing MCL's.

Monitoring requirements -- Tap water monitoring for lead and copper to determine whether a system is subject to the treatment technique requirements. Water quality parameter sampling to determine the effectiveness of optional corrosion control treatment. Source water monitoring for lead and copper to determine source water's contribution to total tap water lead and copper levels, and the need for treatment. Monitoring schedules vary by system size and type of monitoring.

Analytical methodology -- Atomic absorption/furnace technique (EPA 239.2; ASTM D-3559-85D; SM 3113); inductively-coupled plasma/mass spectrometry (EPA 200.8); atomic absorption/platform furnace technique (EPA 200.9).

Best available technology --

Optimal corrosion control treatment: pH/akalinity adjustment, calcium adjustment; addition of corrosion inhibitor.

Source water treatment: Coagulation/filtration; ion exchange; lime softening; reverse osmosis.

Public education.

Lead service line replacement.

Reference -- 45 FR 57332 (08/27/80); 53 FR 31517 (08/18/88); 56 FR 26460 (06/07/91); 56 FR 32112 (07/15/91).

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

CERC -

Value (status) -- 1 pound (Statutory, 1987)

Considers technological or economic feasibility? -- NO

Discussion -- The statutory 1-pound RQ for lead is retained pending assessment of its potential carcinogenicity and may be adjusted in a future notice of proposed rulemaking when the evaluation of available data is completed. Lead was evaluated for chronic toxicity, but was not ranked for toxicity because of insufficient data.

Reference -- 52 FR 8140 (03/16/87); 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed (total lead)

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - None
IREF - None
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HAREF- None

[IRIS] SS 32 /cf?
USER:

AUTOMATIC LOG-OFF IN 2 MINUTES IF NO INPUT IS RECEIVED!!!
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[IRIS] SS 32 /cf?
USER:
7439-96^H5-4
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NP (7439-95-4 (IRIS))
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7439-96-5
Search in progress

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1 - IRIS
IRSN - 372
DATE - 940406
UPDT - 04/06/94, 2 fields
STAT - Oral RfD Assessment (RDO) on-line 04/01/94
STAT - Inhalation RfC Assessment (RDI) on-line 12/01/93
STAT - Carcinogenicity Assessment (CAR) on-line 03/01/94
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 09/26/88 CAR Carcinogen summary on-line
IRH - 09/01/89 REFS Bibliography on-line
IRH - 06/01/90 RDO Oral RfD now under review
IRH - 08/01/90 RDO Oral RfD summary on-line
IRH - 08/01/90 CAR Text edited
IRH - 08/01/90 REFS Oral RfD references added
IRH - 09/01/90 RDI Inhalation RfC now under review
IRH - 12/06/90 RDI Inhalation RfC on-line
IRH - 12/06/90 IREF Inhalation RfC references added
IRH - 01/01/92 EXSR Regulatory Action section on-line
IRH - 06/01/92 IREF Iregren, 1990 and Nishiyama et al., 1975 pages corrected
IRH - 08/01/92 RDO Oral RfD noted as pending change
IRH - 08/01/92 RDO Work group review date added
IRH - 10/01/92 RDO Oral RfD withdrawn; new summary in preparation
IRH - 10/01/92 RDO Work group review date added
IRH - 10/01/92 OREF Oral RfD references withdrawn
IRH - 01/01/93 RDO Oral RfD replaced (RfD changed)
IRH - 01/01/93 OREF Oral RfD references replaced
IRH - 05/01/93 RDO Work group review date added
IRH - 07/01/93 CAREV 'Inadequate' added to 1st paragraph
IRH - 07/01/93 CARDR EPA Documentation clarified
IRH - 11/01/93 RDI Inhalation RfC noted as pending changed
IRH - 11/01/93 RDI Work group review date added
IRH - 12/01/93 RDI Inhalation RfC replaced; RfC changed
IRH - 12/01/93 IREF Inhalation RfC references replaced
IRH - 01/01/94 RDO Oral RfD noted as pending change
IRH - 01/01/94 RDO Work group review date added
IRH - 03/01/94 CARDR Primary contact changed
IRH - 04/01/94 RDO Text revised
IRH - 04/01/94 RDO Text revised
IRH - 04/01/94 RDO Text revised
IRH - 04/01/94 RDO Text revised
IRH - 04/01/94 OREF Oral RfD references revised
RLEN - 108265
NAME - Manganese
RN - 7439-96-5
SY - COLLOIDAL MANGANESE

SY - MAGNACAT
SY - MANGAN
SY - Manganese
SY - MANGAN NITRIDOVANY
SY - TRONAMANG

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
CNS effects Human Chronic Ingestion Data Kondakis et al., 1989	NOAEL (water): 0.005 mg/kg-day LOAEL (water): 0.06 mg/kg-day	1	1	5E-3 mg/kg-day (water)
CNS effects Human Chronic Ingestion Data WHO, 1973; Schroeder et al., 1966; NRC, 1989	NOAEL (food): 0.14 mg/kg-day LOAEL (food): None	1	1	1.4E-1 mg/kg-day (food)

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*Conversion Factors and Assumptions: The arithmetic mean of the range of manganese concentrations for the NOAEL and LOAEL are 167 ug/L and 1950 ug/L, respectively. Assuming a water consumption of 2 L/day and a body weight of 70 kg, these are equivalent to 0.005 mg/kg-day and 0.06 mg/kg-day, respectively. The water RfD assumes a separate dietary intake of manganese, as this essential element is found in varying amounts in all diets. The NOAEL of 10 mg/day (0.14 mg/kg-day for 70 kg adult) for chronic human consumption of manganese in the diet is based on a composite of data from all three references. WHO (1973) reported no adverse effects in humans consuming supplements of 8-9 mg Mn/day (0.11-0.13 mg/kg-day). Schroeder et al. (1966) reported a chronic human NOAEL of 11.5 mg Mn/day (0.16 mg/kg-day).

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o ORAL RFD STUDIES :

WHO (World Health Organization). 1973. Trace Elements in Human Nutrition: Manganese. Report of a WHO Expert Committee. Technical Report Service, 532, WHO, Geneva, Switzerland. p. 34-36.

Schroeder, H.A., J.J. Balassa and I.H. Tipton. 1966. Essential trace metals in man: Manganese, a study in homeostasis. J. Chron. Dis. 19: 545-571.

NRC (National Research Council). 1989. Recommended Dietary Allowances, 10th

ed. Food and Nutrition Board, National Research Council, National Academy Press, Washington, DC. p. 230-235.

Kondakis, X.G., N. Makris, M. Leotsinidis, M. Prinou and T. Papapetropoulos. 1989. Possible health effects of high manganese concentration in drinking water. Arch. Environ. Health. 44: 175-178.

The World Health Organization (WHO) reviewed several investigations of adult diets and reported the average daily consumption of manganese to range from 2.0-8.8 mg Mn/day. Higher manganese intakes are associated with diets high in whole-grain cereals, nuts, green leafy vegetables, and tea. From manganese balance studies, the WHO concluded that 2-3 mg/day is adequate for adults and 8-9 mg/day is "perfectly safe."

Evaluations of standard diets from the United States, England, and Holland reveal average daily intakes of 2.3-8.8 mg Mn/day. Depending on individual diets, however, a normal intake may be well over 10 mg Mn/day, especially from a vegetarian diet. While the actual intake is higher, the bioavailability of manganese from a vegetarian diet is lower, thereby decreasing the actual absorbed dose. This is discussed in more detail in the Additional Studies / Comments Section.

No signs of toxicity were reported in patients (number not specified) given 30 mg manganese citrate (9 mg Mn/day) for many months. Assuming the patients also consumed 2.5 mg Mn/day in food, the total manganese intake would be approximately 11.5 mg Mn/day.

The Food and Nutrition Board of the National Research Council (NRC, 1989) determined an "adequate and safe" intake of manganese to be 2-5 mg/day for adults. This level was chosen because it includes an "extra margin of safety" from the level of 10 mg/day, which the NRC considered to be safe for an occasional intake.

There is one epidemiologic study of manganese in drinking water performed by Kondakis et al. (1989). Three areas in northwest Greece were chosen for this study, with manganese concentrations in natural well water of 3.6-14.6 ug/L in area A, 81.6-252.6 ug/L in area B, and 1600-2300 ug/L in area C. The total population of the three areas studied ranged from 3200 to 4350 people. The study included only individuals over the age of 50 drawn from a random sample of 10% of all households (n=62, 49, and 77 for areas A, B, and C, respectively). The authors reported that "all areas were similar with respect to social and dietary characteristics," but few details were reported. The three areas are located within a 200-square km region. Although the amount of manganese in the diet was not reported, the authors indicated that most of the food was purchased from markets and is expected to be comparable for all three areas. Chemicals other than manganese in the well water were reported to be within Economic Community (EC) standards, except for hardness (120-130 mg calcium carbonate per liter). The individuals chosen were submitted to a neurologic examination, the score of which represents a composite of the presence and severity of 33 symptoms (e.g., weakness/fatigue, gait

disturbances, tremors, dystonia). Whole blood and hair manganese concentrations also were determined. The mean concentration of manganese in hair was 3.51, 4.49, and 10.99 ug/g dry weight for areas A, B, and C, respectively ($p<0.0001$ for area C versus A). The concentration of manganese in whole blood did not differ between the three areas, but this is not considered to be a reliable indicator of manganese exposure. The mean (x) and range (r) of neurologic scores were as follows: Area A (males: x=2.4, r=0-21; females: x=3.0, r=0-18; both x=2.7, r=0-21); Area B (males x=1.6, r=0-6; females: x=5.7 r=0-43; both: x=3.9, r=0-43); and Area C (males: x=4.9, r=0-29; females: x=5.5, r=0-21; both x=5.2, r=0-29). The authors indicate that the difference in mean scores for area C versus A was significantly increased (Mann-Whitney z=3.16, p=0.002 for both sexes combined). In a subsequent analysis, logistic regression indicated that there is a significant difference between areas A and C even when both age and sex are taken into account (Kondakis, 1990). Therefore, the LOAEL for this study is defined by Area C (1600-2300 ug/L) and NOAEL by Area B (81.6-252.6 ug/L). Using the arithmetic means of the ranges of manganese provided (1950 ug/L for Area C and 167 ug/L for Area B), and assuming a water consumption of 2 L/day and a body weight of 70 kg, the LOAEL is equivalent to 0.06 mg Mn/kg-day and the NOAEL is equivalent to 0.005 mg Mn/kg-day.

The individuals examined in the Greek epidemiologic study also had exposure to manganese in their diet. This was roughly estimated to be 10-15 mg/day because of the high intake of vegetables (Kondakis, 1990). In a subsequent correspondence from the investigator, a lower dietary estimate of 5-6 mg Mn/day was stated (Kondakis, 1993), but data have not been supplied to substantiate either estimate. Because of the uncertainty in the amount of manganese in the diet, it is difficult to estimate a total oral intake. The lack of dietary data is recognized as a source of significant uncertainty in this assessment.

The use of the Kondakis et al. (1989) study in supporting the derivation of a separate "water RfD" assumes that the manganese exposure from drinking water is in addition to that found in the diet. This assumption is made primarily because the differences in the bioavailability of manganese in food as compared with that of manganese in water may be such that it is inappropriate to add these intakes together. Unfortunately, while it is agreed that the bioavailability of manganese may vary substantially, relatively few data are available to quantitate these differences, and the number of variables that may affect the uptake of manganese are such that to determine a single value for the absorption of manganese from any medium is not appropriate. These issues are discussed further in the Additional Studies / Comments Section. If the water RfD is used to support a drinking water standard for manganese, no relative source contribution needs to be applied because dietary intake of manganese is already assumed. An RfD of 0.005 mg/kg-day for manganese in water is equivalent to a drinking water standard of 0.2 mg/L.

A report by Kawamura et al. (1941) is the only epidemiologic study describing toxicologic responses in humans consuming large amounts of

manganese dissolved in drinking water. The manganese came from about 400 dry-cell batteries buried near a drinking water well resulting in high levels of both manganese and zinc in the water. Twenty-five cases of manganese poisoning were reported, with symptoms including lethargy, increased muscle tonus, tremor and mental disturbances. The most severe symptoms were seen in elderly people, while children appeared to be unaffected. Three individuals died, one from suicide. The cause of death for the other two was not reported, but the autopsy of one individual revealed manganese concentration in the liver to be 2-3 times higher than in control autopsies. Zinc levels also were increased in the liver. The well water was not analyzed until 1 month after the outbreak, at which time it was found to contain approximately 14 mg Mn/L. When re-analyzed 1 month later, however, the levels were decreased by about half. Therefore, by retrospective extrapolation, the concentration of manganese at the time of exposure may have been as high as 28 mg Mn/L. Consumption of 2 liters of water per day containing 28 mg/L of manganese approximates an intake of 0.8 mg/kg-day for a 70-kg adult. The severe effects seen in this study support the LOAEL estimated from the 1989 Kondakis et al. study of 0.06 mg/kg-day, which was associated with less severe effects.

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o ORAL RFD UNCERTAINTY :

UF -- The information used to determine the RfD for manganese in food was taken from many large populations consuming normal diets over an extended period of time with no adverse health effects. As long as physiologic systems are not overwhelmed, humans exert an efficient homeostatic control over manganese such that body burdens are kept constant with variations in diet. It is recognized that manganese is an essential element, being required for normal human growth and maintenance of health. It has also been suggested that children are less susceptible to manganese intoxication and may require slightly higher levels of manganese than adults. This is not true for infants, who may represent a more sensitive subpopulation (see discussion in the Additional Studies / Comments Section). The available information providing a chronic NOAEL in many cross-sections of human populations, taken in conjunction with the essentiality of manganese, warrants an uncertainty factor of 1. For the drinking water RfD, the study by Kondakis et al. (1989) describes a population of older adults (over 50 years) who had consumed well water containing various concentrations of manganese for a lifetime. The study group is considered to represent a sensitive subpopulation, hence an uncertainty factor of 1 is applied.

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o ORAL RFD MODIFYING FACTOR :

MF -- The modifying factors are 1 for both the dietary RfD and the drinking water RfD.

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o ORAL RFD COMMENTS :

While the Greek epidemiologic study (Kondakis et al., 1989) is considered to be acceptable for deriving a separate water RfD for manganese, it is also

recognized as being limited in scope. Arguments for using the Greek study are strengthened when it is reviewed in the context of additional information. In addition to the Greek and Japanese studies of humans ingesting manganese in drinking water, one limited oral study has been performed in a group of four Rhesus monkeys (Gupta et al., 1980). Muscular weakness and rigidity of the lower limbs developed after 18 months of exposure to 6.9 mg Mn/kg-day (as MnC12.4H2O). Histologic analysis showed degenerated neurons in the substantia nigra and scanty neuromelanin granules in some other pigmented cells.

A pattern is seen when the two human studies are viewed in conjunction with the study in Rhesus monkeys. Kondakis et al. (1989) identified a NOAEL for humans ingesting manganese in drinking water of 0.005 mg/kg-day and a LOAEL associated with minor neurologic effects of 0.06 mg/kg-day. Kawamura et al. (1941) reported severe effects in a small group of people ingesting about 0.8 mg/kg-day of manganese in water. Some subpopulations (e.g., children) appeared to be unaffected. Finally, Gupta et al. (1980) reported severe effects confirmed by histopathologic analysis in monkeys receiving 6.9 mg/kg manganese.

A couple of case studies have also pointed to the potential for manganese poisoning by routes other than inhalation. One involved a 59-year-old male who was admitted to the hospital with symptoms of classical manganese poisoning, including dementia and a generalized extrapyramidal syndrome (Banta and Markesbery, 1977). The patient's serum, hair, urine, feces, and brain were found to have manganese "elevated beyond toxic levels," perhaps a result of his consumption of "large doses of vitamins and minerals for 4 to 5 years." Unfortunately, no quantitative data were reported.

Another case study of manganese intoxication involved a 62-year-old male who had been receiving total parenteral nutrition that provided 2.2 mg of manganese (form not stated) daily for 23 months (Ejima et al., 1992). The patient's whole blood manganese was found to be elevated, and he was diagnosed as having parkinsonism, with dysarthria, mild rigidity, hypokinesia with masked face, a halting gait, and severely impaired postural reflexes. To be able to compare the manganese load in this individual with that corresponding to an oral intake, the difference between the direct intravenous exposure and the relatively low level of absorption of manganese from the gastrointestinal tract must be taken into account. Assuming an average absorption of roughly 5% of an oral dose, the intravenous dose of 2.2 mg Mn/day would be approximately equivalent to an oral intake of 40 mg Mn/day. This is consistent with the severe neurological effects reported in the Japanese drinking water poisoning (Kawamura et al., 1941) in which it is estimated that the individuals were exposed to about 58 mg Mn/day.

Several oral studies have been performed in rodents, also demonstrating biochemical changes in the brain following administration of 1 mg MnC12.4H2O/mL in drinking water (approximately 38.9 mg Mn/kg-day) (Lai et al., 1981, 1982; Leung et al., 1981; Chandra and Shukla, 1981). However, rodents do not exhibit the same neurologic deficits that humans do following exposure to manganese; thus the relevance of these biochemical changes has been

challenged. The problem with using rodents is exemplified by the disease of parkinsonism, which is characterized by effects very similar to those seen in manganese poisoning. Marsden and Jenner (1987) hypothesize that the ability of certain drugs to induce parkinsonism in primates but not in rodents is due to the relative lack of neuromelanin in rodents. Because manganese selectively accumulates in pigmented regions of the brain (e.g., the substantia nigra), this species difference is fundamentally important.

It is also important to recognize the substantial difference in species' requirements for manganese. For rats, the estimated requirement is 50 mg Mn/kg diet (Rogers, 1979). Assuming a food consumption equivalent to 5% of body weight, this corresponds to a requirement of about 2.5 mg Mn/kg-day. Although the dietary requirement for manganese has not been determined quantitatively for humans, the "Estimated Safe and Adequate Daily Dietary Intake" is 2-5 mg/day, or about 0.03-0.07 mg Mn/kg-day, assuming a reference body weight of 70 kg. The dietary requirement for manganese in rats then, is about 50-fold higher than the estimated safe and adequate intake for humans, suggesting that data derived from rodent studies may not be appropriate for use in deriving quantitative risk estimates for manganese in humans.

Another issue of great importance to consider in the risk assessment for manganese concerns the bioavailability of different forms of manganese consumed under different exposure conditions. The separate RfDs for food and water indicate a potentially higher bioavailability of manganese from drinking water. In considering dietary sources, it is also important to recognize that various dietary factors as well as the form of manganese can have a significant bearing on the dose absorbed from the gastrointestinal tract. Many constituents of a vegetarian diet (e.g., tannins, oxalates, phytates, fiber, calcium, and phosphorus) have been found to inhibit manganese absorption presumably by forming insoluble complexes in the gut. Individuals who are deficient in iron demonstrate an increase in manganese absorption. It is also recognized that manganese uptake and elimination are under homeostatic control, generally allowing for a wide range of dietary intakes considered to be safe. These factors and others are described in a review by Kies (1987). In addition to the influence of extrinsic variables, significant interindividual differences in manganese absorption and retention have been reported. In humans administered a dose of radiolabeled manganese in an infant formula, the mean absorption was 5.9 +/- 4.8%, but the range was 0.8-16%, a 20-fold difference (Davidsson et al., 1989). Retention at day 10 was 2.9 +/- 1.8%, but the range was 0.6-9.2%, again indicating substantial differences between individuals.

In a 100-day dietary study in 6-week-old male mice, Komura and Sakamoto (1991) demonstrated significant differences in tissue levels of manganese in mice fed equivalent amounts of manganese as MnCl2.4H2O, Mn(Ac)2.4H2O, MnCO3 and MnO2. Mice receiving the two soluble forms of manganese (the chloride and acetate salts) were found to gain significantly less weight than controls, while mice consuming the insoluble forms of manganese (the carbonate and dioxide salts) appeared to actually gain slightly more weight than controls. However, the acetate and carbonate groups had significantly higher manganese

levels in the liver and kidney compared with the chloride and dioxide groups, both of which were elevated above control levels. Reduced locomotor activity in the carbonate and acetate groups was also reported, perhaps related to the higher tissue levels of manganese. This study points out a need for understanding the effects of the various forms of manganese, of which relatively little is known. More information on manganese speciation can be found in the RfC file on IRIS.

It is also recognized that neonates may be at increased risk of toxicity resulting from exposure to manganese because of a higher level of uptake from the gastrointestinal tract. The uptake and retention of manganese have been reviewed by Lonnerdal et al. (1987). In rats, manganese absorption decreased dramatically as the animals matured. While 24-hour retention values are as high as 80% in 14-day-old pups, this value drops to about 30% by day 18. Low levels of manganese absorption (about 3-4%) have also been reported for mature humans, but few data are available for infants.

Collipp et al. (1983) found that hair manganese levels in newborn infants was found to increase significantly from birth (0.19 ug/g) to 6 weeks of age (0.865 ug/g) and 4 months of age (0.685 ug/g) when the infants were given formula, but that the increase was not significant in babies who were breast-fed (0.330 ug/g at 4 months). While human breast milk is relatively low in manganese (7-15 ug/L), levels in infant formulas are 3-100 times higher. It was further reported in this study that the level of manganese in the hair of learning disabled children (0.434 ug/g) was significantly increased in comparison with that of normal children (0.268 ug/g). Other investigators also have reported an association between elevated levels of manganese in hair and learning disabilities in children (Barlow and Kapel, 1979; Pihl and Parkes, 1977). Although no causal relationship has been determined for learning disabilities and manganese intake, further research in this area is warranted. High levels of manganese in infant formulas may be of concern because of the increased absorption and retention of manganese that has been reported in neonatal animals (Lonnerdal et al., 1987). Also, manganese has been shown to cross the blood-brain barrier, with the rate of penetration in animal experiments being 4 times higher in neonates than in adults (Mena, 1974). It was suggested by Dieter et al. (1992) that "if there were a toxicological limit to manganese according to the principles of preventive health care, then it would have to be set at 0.2 mg/L of manganese for infants as a group at risk."

Although conclusive evidence is lacking, some investigators have also linked increased intakes of manganese with violent behavior. Gottschalk et al. (1991) found statistically significantly elevated levels of manganese in the hair of convicted felons (1.62 +/- 0.173 ppm in prisoners compared with 0.35 +/- 0.020 ppm in controls). The authors suggest that "a combination of cofactors, such as the abuse of alcohol or other chemical substances, as well as psychosocial factors, acting in concert with mild manganese toxicity may promote violent behavior." Caution should be exercised to prevent reading too much into these data, but support for this hypothesis is provided by studies of a population of Aborigines in Groote Eylandt. Several clinical symptoms

consistent with manganese intoxication are present in about 1% of the inhabitants of this Australian island, and it may not be coincidental that the proportion of arrests in this native population is the highest in Australia (Cawte and Florence, 1989; Kilburn, 1987). The soil in this region is very high in manganese (40,000-50,000 ppm), and the fruits and vegetables grown in the region also are reported to be high in manganese. Quantitative data on oral intakes have not been reported, but elevated concentrations of manganese have been determined in the blood and hair of the Aborigines (Stauber et al., 1987). In addition to the high levels of environmental manganese, other factors common to this population may further increase the propensity for manganism: high alcohol intake, anemia, and a diet deficient in zinc and several vitamins (Florence and Stauber, 1989).

In addition to the toxicity information available by the oral route, several inhalation toxicity studies on manganese also have been performed in laboratory animals, demonstrating an effect on both the brain and lungs. An in-depth discussion of the psychologic and neurologic effects associated with inhalation of manganese can be found in the RfC file on IRIS.

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o ORAL RFD CONFIDENCE :

Study -- Not applicable
Data Base -- Not applicable
RfD -- Not applicable

Many studies have reported similar findings with regard to the normal dietary intake of manganese by humans. These data are considered to be superior to any data obtained from animal toxicity studies, especially as the physiologic requirements for manganese vary quite a bit among different species, with man requiring less than rodents (Schroeder et al., 1966). There is no one study used to derive the dietary RfD for manganese. While several studies have determined average levels of manganese in various diets, no quantitative information is available to indicate toxic levels of manganese in the diet of humans. Because of the homeostatic control humans maintain over manganese, it is generally not considered to be very toxic when ingested with the diet. It is important to recognize that while the RfD process involves the determination of a point estimate of an oral intake, it is also stated that this estimate is associated "with uncertainty spanning perhaps an order of magnitude." Numerous factors, both environmental factors (e.g., the presence or absence of many dietary constituents) and biological or host factors (e.g., age, alcohol consumption, anemia, and general nutritional status) can significantly influence an individual's uptake of manganese from the diet. As discussed in the Additional Studies / Comments Section, there is significant variability in the absorption of manganese by humans. The determination of a single intake of manganese in the diet must be recognized as a process that is limited in its ability to reflect the variable nature of manganese toxicity. Such a determination may both over- and underestimate the risk, depending on the specific combination of environmental and individual circumstances. Confidence in the data base is medium and confidence in the dietary RfD for manganese is also medium.

It is again emphasized that this oral RfD is based on a total dietary intake of manganese and is not intended to be applied directly to drinking water situation. Because of the greater bioavailability of manganese from water, a separate RfD for water is proposed. This is based on the Greek epidemiologic study by Kondakis et al. (1989). The major advantage of this study is that it examined a sensitive subpopulation of humans exposed to varying concentrations of manganese in the drinking water for a lifetime. A significant weakness of this study is that the actual manganese content in the diet was not measured. The author did indicate, however, that the people in the three areas were age-matched and had similar social, economic and educational backgrounds, and that the food consumed by these subjects was purchased at the marketplace and was not expected to vary much in manganese content. The confidence in the critical study can be considered low-to-medium. It is not higher primarily because of the lack of data on dietary manganese in the three populations under study. Also, many of the endpoints scored in the neurological exam are not specific for manganese poisoning and are, in fact, associated with the normal process of aging. Confidence in the data base also can be considered medium-to-low. The Greek study is supported by the more severe effects occurring at higher levels in the Japanese study (Kawamura et al., 1941) and the study in rhesus monkeys (Gupta et al., 1980). Overall confidence in the drinking water RfD can be considered medium-to-low. While the RfD process involves the determination of a single point estimate of an oral intake, it must be recognized that this estimate is associated with several sources of uncertainty. Available data indicate that numerous factors, both environmental factors (e.g., the presence of high or low levels of other inorganics in drinking water) and biological or host factors (e.g., age, alcohol consumption, and nutritional status, particularly anemia) can significantly influence the uptake of manganese by an individual. The determination of a single concentration of manganese in drinking water, then, must be recognized as a process that is limited in its ability to reflect the variable nature of manganese toxicity.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- U.S. EPA, 1993

The Drinking Water Criteria Document for Manganese has received Agency review.

Other EPA Documentation -- U.S. EPA, 1984

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o REVIEW DATES : 05/17/90, 06/21/90, 06/24/92, 09/22/92, 03/31/93, 12/14/93

o VERIFICATION DATE : 09/22/92

o EPA CONTACTS :

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RDI -

o INHALATION RFD SUMMARY :

Critical Effect	Exposures*	UF	MF	RfC
Impairment of neuro-behavioral function	NOAEL: None LOAEL: 0.15 mg/cu.m	1000	1	5E-5 mg/cu.m
Occupational exposure to manganese dioxide	LOAEL(ADJ): 0.05 mg/cu.m LOAEL(HEC): 0.05 mg/cu.m			

Roels et al., 1992

Impairment of neuro-behavioral function	NOAEL: None LOAEL: 0.97 mg/cu.m			
Occupational exposure to manganese oxides and salts	LOAEL(ADJ): 0.34 mg/cu.m LOAEL(HEC): 0.34 mg/cu.m			

Roels et al., 1987

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*Conversion Factors and Assumptions: Roels et al., 1992: The LOAEL is derived from an occupational-lifetime integrated respirable dust (IRD) concentration of manganese dioxide (MnO_2) (based on 8-hour TWA occupational exposure multiplied by individual work histories in years) expressed as mg manganese (Mn)/cu.m x years. The IRD concentrations ranged from 0.040 to 4.433 mg Mn/cu.m x years, with a geometric mean of 0.793 mg Mn/cu.m x years and a geometric standard deviation of 2.907. The geometric mean concentration (0.793 mg/cu.m x years) was divided by the average duration of MnO_2 exposure (5.3 years) to obtain a LOAEL TWA of 0.15 mg/cu.m. The LOAEL refers to an extrarespiratory effect of particulate exposure and is based on an 8-hour TWA occupational exposure. MVho = 10 cu.m/day, MVh = 20 cu.m/day. LOAEL(HEC) = 0.15 mg/cu.m x (MVho/MVh) x 5 days/7 days = 0.05 mg/cu.m.

Roels et al., 1987: The LOAEL is based on an 8-hour TWA occupational exposure. The TWA of total airborne manganese dust ranged from 0.07 to 8.61 mg/cu.m, and the median was 0.97 mg/cu.m. This is an extrarespiratory effect of a particulate exposure. MVho = 10 cu.m/day, MVh = 20 cu.m/day. LOAEL(HEC) = 0.97 mg/cu.m x (MVho/MVh) x 5 days/7 days = 0.34 mg/cu.m.

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o INHALATION RFD STUDIES :

Roels, H., R. Lauwerys, J.-P. Buchet et al. 1987. Epidemiological survey among workers exposed to manganese: Effects on lung, central nervous system,

and some biological indices. Am. J. Ind. Med. 11: 307-327.

Roels, H.A., P. Ghyselen, J.P. Buchet, E. Ceulemans, and R.R. Lauwerys. 1992. Assessment of the permissible exposure level to manganese in workers exposed to manganese dioxide dust. Br. J. Ind. Med. 49: 25-34.

Roels et al. (1992) conducted a cross-sectional study of 92 male workers exposed to manganese dioxide (MnO_2) dust in a Belgian alkaline battery plant. A control group of 101 male workers was matched for age, height, weight, work schedule, coffee and alcohol consumption, and smoking; educational level was slightly higher in the control group ($p = 0.046$ by chi square test).

The manganese (Mn)-exposed group had been exposed to MnO_2 for an average of 5.3 years (range: 0.2-17.7 years). The geometric means of the workers' TWA airborne Mn concentrations, as determined by personal sampler monitoring at the breathing zone, were 0.215 mg Mn/cu.m for respirable dust and 0.948 mg Mn/cu.m for total dust. No data on particle size or purity were presented, but the median cut point for the respirable dust fraction was 5 um according to information provided by Roels et al. (1992) and Roels (1993). Total and respirable dust concentrations were highly correlated ($r = 0.90$, $p < 0.001$), with the Mn content of the respirable fraction representing on average 25% of the Mn content in the total dust. The authors noted that the personal monitoring data were representative of the usual exposure of the workers because work practices had not changed during the last 15 years of the operation of the plant.

Occupational-lifetime integrated exposure to Mn was estimated for each worker by multiplying the current airborne Mn concentration for the worker's job classification by the number of years for which that classification was held and adding the resulting (arithmetic) products for each job position a worker had held. The geometric mean occupational-lifetime integrated respirable dust (IRD) concentration was 0.793 mg Mn/cu.m x years (range: 0.040-4.433 mg Mn/cu.m x years), with a geometric standard deviation of 2.907 mg Mn/cu.m x years, based on information provided by Roels (1993). The geometric mean occupational-lifetime integrated total dust (ITD) concentration was 3.505 mg Mn/cu.m x years (range: 0.191-27.465 mg Mn/cu.m x years). Geometric mean concentrations of blood Mn (MnB) (0.81 ug/dL) and urinary Mn (MnU) (0.84 ug/g creatinine) were significantly higher in the Mn-exposed group than in the control group, but on an individual basis no significant correlation was found between either MnB or MnU and various external exposure parameters. Current respirable and total Mn dust concentrations correlated significantly with geometric mean MnU on a group basis (Spearman $r = 0.83$, $p < 0.05$).

A self-administered questionnaire focused on occupational and medical history, neurological complaints, and respiratory symptoms. Lung function was evaluated by standard spirographic measures. Neurobehavioral function was evaluated by tests of audio-verbal short-term memory, visual simple reaction time, hand steadiness, and eye-hand coordination. Blood samples were assayed for several hematological parameters (erythrocyte count, leukocyte count,

hemoglobin, hematocrit, mean corpuscular volume, mean corpuscular hemoglobin, platelets, and differential leukocyte count); Mn; lead; zinc protoporphyrin; and serum levels of calcium, iron, follicle stimulating hormone (FSH), luteinizing hormone (LH), and prolactin. Urinary Mn, cadmium, and mercury concentrations were also determined.

Responses to the questionnaire indicated no significant differences between groups in either respiratory or neurological symptoms, nor were spirometric, hormonal, or calcium metabolism measurements significantly different for the two groups. In addition, a separate report (Gennart et al., 1992) indicated no significant difference in the fertility of 70 of these workers, in contrast to earlier findings in 85 workers exposed not only to MnO2 but also to other Mn oxides and salts at higher concentrations (Lauwerys et al., 1985). Erythropoietic parameters and serum iron concentrations were consistently and significantly lower in the Mn-exposed workers, albeit within the normal range of values.

Of particular note, Mn workers performed worse than controls on several measures of neurobehavioral function. Visual reaction time was consistently and significantly slower in the Mn-exposed workers measured in four 2-minute periods, with more pronounced slowing over the total 8-minute period and significantly greater variability in reaction times for the Mn-exposed group. Abnormal values for mean reaction times (defined as greater than or equal to the 95th percentile of the control group) also were significantly more prevalent in the Mn-exposed group during three of four 2-minute intervals of the 8-minute testing period.

Five measures of eye-hand coordination (precision, percent precision, imprecision, percent imprecision, and uncertainty) reflected more erratic control of fine hand-forearm movement in the Mn-exposed group than in the controls, with mean scores on all five measures being highly significantly different for the two groups. There was also a significantly greater prevalence of abnormal values for these five measures in the Mn-exposed group. The hole tremormeter test of hand steadiness indicated a consistently greater amount of tremor in the Mn-exposed workers, with performance for two of the five hole sizes showing statistically significant impairment.

Roels et al. (1992) performed an exposure-response analysis by classifying IRD values into three groups (<0.6, 0.6-1.2, and >1.2 mg Mn/cu.m x years) and comparing the prevalence of abnormal scores for visual reaction time, hand steadiness, and eye-hand coordination with controls. This analysis indicated that the prevalence of abnormal eye-hand coordination values was significantly greater in workers whose IRD levels were less than 0.6 mg Mn/cu.m x years. However, the relationship between exposure and response was not linear across groups. Visual reaction time and hand steadiness showed linear exposure-related trends but did not achieve statistical significance except at levels of >1.2 mg Mn/cu.m x years. As noted by the authors, "analysis of the data on a group basis ... does not permit us to identify a threshold effect level for airborne Mn." Although suggestive of a LOAEL of <0.6 mg Mn/cu.m x years, the exposure-response analysis by Roels et al. (1992) possibly could reflect the

small disparity in educational level between exposed and control workers that was noted above with regard to the matching criteria for this study. If educational level were in fact a covariate of exposure as well as neurobehavioral performance, it could confound the exposure-response analysis. Although it is not clear that such was the case, the possibility of confounding suggests that the LOAEL should not be based on the results of the exposure-response analysis until these results can be confirmed by other studies. Also, statistical correction for multiple comparisons should be included in the exposure response analysis.

A LOAEL may be derived from the Roels et al. (1992) study by using the IRD concentration of MnO2, expressed as mg Mn/cu.m x years (based on 8-hour TWA occupational exposures for various job classifications, multiplied by individual work histories in years). Dividing the geometric mean IRD concentration (0.793 mg/cu.m x years) by the average duration of the workers' exposure to MnO2 (5.3 years) yields a LOAEL of 0.15 mg/cu.m. The LOAEL(HEC) is 0.05 mg/cu.m.

Roels et al. (1987) conducted a cross-sectional study in 141 male workers exposed to MnO2, manganese tetroxide (Mn3O4), and various Mn salts (sulfate, carbonate, and nitrate). A matched group of 104 male workers was selected as a control group. The two groups were matched for socioeconomic status and background environmental factors; in addition, both groups had comparable work-load and work-shift characteristics.

The TWA of total airborne Mn dust ranged from 0.07 to 8.61 mg/cu.m, with an overall arithmetic mean of 1.33 mg/cu.m, a median of 0.97 mg/cu.m, and a geometric mean of 0.94 mg/cu.m. The duration of employment ranged from 1 to 19 years, with a mean of 7.1 years. The particle size and purity of the dust were not reported. Neurological examination, neurobehavioral function tests (simple reaction time, short-term memory, eye-hand coordination, and hand tremor), spirographic measurements, blood and urine tests, and a self-administered questionnaire were used to assess possible toxic effects of Mn exposure. The questionnaire was designed to detect CNS and respiratory symptoms.

Significant differences in mean scores between Mn-exposed and reference subjects were found for objective measures of visual reaction time, eye-hand coordination, hand steadiness, and audio-verbal short-term memory. The prevalence of abnormal scores on eye-hand coordination and hand steadiness tests showed a dose-response relationship with blood Mn levels; short-term memory scores were related to years of Mn exposure but not to blood Mn levels. The prevalence of subjective symptoms was greater in the exposed group than in controls for 20 of 25 items on the questionnaire, with four items being statistically significant: fatigue, tinnitus, trembling of fingers, and irritability.

A significantly greater prevalence of coughs during the cold season, dyspnea during exercise, and recent episodes of acute bronchitis was self-reported in the exposed group. Lung function parameters were only slightly

(<10%) lower in the Mn-exposed workers, with the only significant alterations evident in Mn-exposed smokers. These mild changes in Mn-exposed workers (apart from the effects of smoking) and the absence of respiratory effects in the more recent study by Roels et al. (1992) suggest that the nervous system is a more sensitive target for Mn toxicity.

Based upon the findings of impaired neurobehavioral function in workers whose average Mn exposure was estimated by the geometric mean TWA of total airborne Mn dust at the time of the study, a LOAEL of 0.97 mg/cu.m was identified, with a LOAEL(HEC) of 0.34 mg/cu.m. Note that this LOAEL(HEC) is based on total Mn dust of mixed forms, whereas the LOAEL(HEC) from the more recent Roels et al. (1992) study is based on the measured respirable dust fraction of MnO2 only. However, the geometric mean total dust concentrations in the 1987 and 1992 studies by Roels et al. were approximately the same (0.94 and 0.95 mg/cu.m, respectively).

The findings of Roels et al. (1987, 1992) are supported by other recent reports that provide comparable and consistent indications of neurobehavioral dysfunction in Mn-exposed workers (Mergler et al., 1993; Iregren, 1990; Wennberg et al., 1991, 1992).

Mergler et al. (1993) conducted a cross-sectional study of 115 male ferromanganese and silicomanganese alloy workers in southwest Quebec. A matched-pair design was employed because of presumptively high environmental pollutant levels; 74 pairs of workers and referents were matched on age, educational level, smoking status, number of children, and length of residency in the region.

Air concentrations of respirable and total dust were sampled by stationary monitors during silicomanganese production. The geometric mean of a series of 8-hour TWAs was 0.035 mg Mn/cu.m (range: 0.001-1.273 mg Mn/cu.m) for respirable dust and 0.225 mg Mn/cu.m (range: 0.014-11.480 mg Mn/cu.m) for total dust. The authors noted that past dust levels at certain job sites had been considerably higher. The mean duration of the workers' Mn exposure was 16.7 years and included Mn fumes as well as mixed oxides and salts of Mn. Geometric mean MnB was significantly higher in the Mn alloy workers, but MnU did not differ significantly between exposed workers and controls.

The number of discordant pairs, in which workers reported undesirable symptoms on a self-administered questionnaire but their matched pairs did not, was statistically significant for 33 of 46 items, including the following: fatigue; emotional state; memory, attention, and concentration difficulties; nightmares; sweating in the absence of physical exertion; sexual dysfunction; lower back pain; joint pain; and tinnitus. Workers did not report symptoms typical of advanced Mn poisoning (e.g., hand tremor, changes in handwriting, loss of balance when turning, difficulty in reaching a fixed point) significantly more than referents, which suggests that the other reported symptoms were probably not due to bias on the part of the workers.

The greatest differences in neurobehavioral function were evident in tests

of motor function, especially tests requiring coordinated alternating and/or rapid movements. Workers performed significantly worse on the motor scale of a neuropsychological test battery both in overall score and in eight subscales of rapid sequential or alternating movements. Worker performance also was significantly worse on tests of hand steadiness, parallel-line drawing performance, and ability to rapidly identify and mark specified alphabetic characters within strings of letters. Performance on a variety of other tests of psychomotor function, including simple reaction time, was worse in Mn-exposed workers but marginally significant ($0.05 < p < 0.10$). In addition, Mn alloy workers differed significantly from referents on measures of cognitive flexibility and emotional state. Olfactory perception also was significantly enhanced in the Mn-alloy workers.

The matched-pair design of Mergler et al. (1993) helped reduce differences between exposed and referent subjects that might otherwise have confounded the study. However, to the extent that the referents also may have had significant exposure to Mn in the ambient atmosphere, such exposure may have reduced the differences in neurobehavioral performance between workers and referents. This possibility is supported by the fact that the finger-tapping speed of both workers and referents on a computerized test was slower than that of Mn-exposed workers assessed on the same test by Iregren (1990) in Sweden. In the absence of a NOAEL, the LOAEL from the study of Mergler et al. (1993) is based on the geometric mean respirable dust level (0.035 mg Mn/cu.m), with a LOAEL(HEC) of approximately 0.01 mg/cu.m, which is about five-fold lower than the LOAEL(HEC) identified in the study by Roels et al. (1992).

Workers exposed to Mn in two Swedish foundries (15 from each plant) were evaluated in a study first reported by Iregren (1990). The exposure to Mn varied from 0.02 to 1.40 mg/cu.m (mean = 0.25 mg/cu.m; median = 0.14 mg/cu.m) for 1-35 years (mean = 9.9 years). Earlier monitoring measurements made in both factories suggested that essentially no changes in exposure had occurred in either factory for the preceding 18 years. Each exposed worker was matched for age, geographical area, and type of work to two workers not exposed to Mn in other industries. Neurobehavioral function was assessed by eight computerized tests and two manual dexterity tests. There were significant differences between exposed and control groups for simple reaction time, the standard deviation of reaction time, and finger-tapping speed of the dominant hand. In addition, digit-span short-term memory, speed of mental addition, and verbal (vocabulary) understanding differed significantly between exposed and control groups. The difference in verbal understanding suggested that the two groups were not well matched for general cognitive abilities. With verbal performance used as an additional matching criterion, differences between the groups in simple reaction time, the standard deviation of reaction time, and finger-tapping speed remained statistically significant, despite a decrease in statistical power due to reducing the size of the reference group to 30 workers. Further analyses using verbal test scores as a covariate also indicated that these same three measures of neurobehavioral function were statistically different in exposed and control workers. No significant correlation was found within the exposed group to establish a concentration-

response relationship.

Additional reports of neurobehavioral and electrophysiological evaluations of these same workers have been published by Wennberg et al. (1991, 1992). Although none of the latter results achieved statistical significance at p = 0.05, increased frequency of self-reported health symptoms, increased prevalence of abnormal electroencephalograms, slower brain-stem auditory-evoked potential latencies, and slower diadochokinesometric performance were found in the exposed workers. Diadochokinesis refers to the ability to perform rapidly alternating movements such as supination and pronation of the forearm, and is an indicator of extrapyramidal function (see Additonal Comments/Studies). Also, an increase in P-300 latency, as suggested by these results, has been observed in patients with parkinsonism according to Wennberg et al. (1991), who viewed these results in Mn-exposed workers as early (preclinical) signs of disturbances similar to parkinsonism. Based on the impairments in reaction time and finger-tapping speed reported in the Swedish studies (Iregren, 1990; Wennberg et al., 1991, 1992), the LOAEL(HEC) is calculated to be 0.05 mg/cu.m. Although numerically the same value as that derived from Roels et al. (1992), the Swedish study measured total dust. However, Wennberg et al. (1991) stated that the respirable dust level was 20-80% of total dust, which implies that the LOAEL(HEC) from the Swedish studies could be somewhat lower than that from Roels et al. (1992).

All of the above studies taken together provide a consistent pattern of evidence indicating that neurotoxicity is associated with low-level occupational Mn exposure. The fact that the speed and coordination of motor function are especially impaired is consistent with other epidemiological, clinical, and experimental animal evidence of Mn intoxication (see Additional Comments/Studies). Moreover, the LOAEL(HEC)s obtained from these studies are not appreciably different. Nevertheless, some differences between the studies are evident in the durations of exposure and forms of Mn to which workers were exposed. In the Roels et al. (1992) study, the mean period of exposure was 5.3 years (range: 0.2-17.7 years), and exposure was limited to MnO2. In the other studies, mixed forms of Mn were present, and the mean durations of exposure were longer: 7.1 years in Roels et al. (1987), 9.9 years in Iregren (1990), and 16.7 years in Mergler et al. (1993). The findings of Mergler et al. (1993) suggest that the LOAEL(HEC) could be at least as low as approximately 0.01 mg/cu.m. However, the variable concentrations and mixed compounds of Mn to which workers were exposed make it difficult to rely primarily upon the findings of Mergler et al. (1993) in deriving the RfC. Nevertheless, their results provide support for the findings of Roels et al. (1992) and suggest that the longer period of exposure (16.7 years in Mergler et al. (1993) vs. 5.3 years in Roels et al., 1992) may have contributed to the apparent differences in sensitivity. Although analyses by Roels et al. (1987, 1992) and Iregren (1990) generally did not indicate that duration of exposure correlated significantly with neurobehavioral outcomes, none of these studies involved average exposures as long as those in the Mergler et al. (1993) study. Also, the oldest worker in the Roels et al. (1992) study was less than 50 years old, and the average age in that study was only 31.3 years vs. 34.3 years in Roels et al. (1987), 43.4 years in Mergler et al. (1993), and 46.4 in

Iregren (1990). These points suggest that chronic exposure to Mn and/or interactions with aging could result in effects at lower concentrations than would be detected after shorter periods of exposure and/or in younger workers.

Based on the findings of neurobehavioral impairment by Roels et al. (1987, 1992), with supporting evidence from Mergler et al. (1993) and the Swedish reports (Iregren, 1990; Wennberg et al., 1991, 1992), the LOAEL for derivation of the RfC is 0.15 mg/cu.m, and the LOAEL(HEC) is 0.05 mg/cu.m.

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o INHALATION RFD UNCERTAINTY :

UF -- An uncertainty factor of 1000 reflects 10 to protect sensitive individuals, 10 for use of a LOAEL, and 10 for database limitations reflecting both the less-than-chronic periods of exposure and the lack of developmental data, as well as potential but unquantified differences in the toxicity of different forms of Mn.

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o INHALATION RFD MODIFYING :

MF -- None
FACTOR

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o INHALATION RFD COMMENTS :

Manganese toxicity varies depending upon the route of exposure. When ingested, Mn is considered to be among the least toxic of the trace elements. In the normal adult, between 3 and 10% of dietary Mn is absorbed. Total body stores normally are controlled by a complex homeostatic mechanism regulating absorption and excretion. As detailed in the Uncertainty Factor Text and the Additonal Comments/Studies for the oral RfD, toxicity from ingested Mn is rarely observed. However, deficiencies of calcium and iron have been noted to increase Mn absorption (Mena et al., 1969; Murphy et al., 1991). Also, Mena et al. (1969) found that anemic subjects absorbed 7.5% of ingested Mn, whereas normal subjects absorbed 3%. Interestingly, manganism patients absorbed 4%, and apparently healthy Mn miners absorbed only 3%. These differences suggest that certain subpopulations, such as children, pregnant women, elderly persons, iron- or calcium-deficient individuals, and individuals with liver impairment, may have an increased potential for excessive Mn body burdens due to increased absorption or altered clearance mechanisms, which may be of particular importance for those exposed to Mn by multiple routes.

As a route of Mn exposure, the respiratory tract is the most important portal of entry. The inhalation toxicity of Mn is in part a function of particle dosimetry and subsequent pharmacokinetic events. Particle size determines the site of deposition in the respiratory tract. Generally, in humans, fine mode particles (<2.5 um) preferentially deposit in the pulmonary region, and coarse mode particles (>2.5 um) deposit in the tracheobronchial and extrathoracic regions. Those particles depositing in the extrathoracic and tracheobronchial regions are predominantly cleared by the mucociliary escalator into the gastrointestinal tract where absorption is quite low (about

3%). Particles deposited in the pulmonary region are cleared predominantly to the systemic compartment by absorption into the blood and lymph circulation. Disregarding the possibility of counteracting mechanisms, 100% absorption of particles deposited in the pulmonary region is assumed. Another possible route of exposure may exist. Studies such as those of Perl and Good (1987) and Evans and Hastings (1992) have indicated that neurotoxic metals such as aluminum and cadmium can be directly transported to the brain olfactory bulbs via nasal olfactory pathways (i.e., from extrathoracic deposition). The alteration in olfactory perception that Mergler et al. (1993) found in Mn-exposed workers lends support to the speculation that this pathway may also operate for Mn, which would further complicate understanding of target-site dosimetry.

The human health effects data base on Mn does not include quantitative inhalation pharmacokinetics information on the major oxides of Mn. Two of the studies described in the Principal and Support Studies (Roels et al., 1992; Mergler et al., 1993) measured respirable as well as total Mn dust, and one study (Roels et al., 1992) dealt with workers exposed to only one form of Mn, namely MnO2. However, this information does not allow quantitative determinations of the dose delivered to the respiratory tract or estimates of target-site doses. After absorption via the respiratory tract, Mn is transported through the blood stream directly to the brain, bypassing the liver and the opportunity for first-pass hepatic clearance. This direct path from the respiratory tract to the brain is the primary reason for the differential toxicity of inhaled and ingested Mn. Pharmacokinetic analyses based on inhalation of manganese chloride (MnCl2) by macaque monkeys (Newland et al., 1987) indicated that clearance from the brain was slower than from the respiratory tract and that the rate of clearance depended on the route of exposure. Brain half-times were 223-267 days after inhalation vs. 53 days following subcutaneous administration (Newland et al., 1987) or 54 days in humans given Mn intravenously (Cotzias et al., 1968). These long half-times were thought to reflect both slower clearance of brain stores and replenishment from other organs, particularly the respiratory tract. In rats, Drown et al. (1986) also observed slower clearance of labeled Mn from brain than from the respiratory tract. Several occupational physicians have reported large individual differences in workers' susceptibility to Mn intoxication, which Rodier (1955) speculated might be due in part to differences in the ability to clear particulate Mn from the lung.

The bioavailability of different forms of Mn is another matter for consideration. Roels et al. (1992) noted that geometric mean blood and urinary Mn levels of workers exposed only to MnO2 in their 1992 report were lower (MnB: 0.81 ug/dL; MnU: 0.84 ug/g creatinine) than those of workers exposed to mixed oxides and salts in their 1987 report (MnB: 1.22 ug/dL; MnU: 1.59 ug/g creatine), even though airborne total dust levels were approximately the same (geometric means of 0.94 and 0.95 mg/cu.m, respectively). Mena et al. (1969) observed no difference between the absorption of 1 um particles of MnCl2 and manganese sesquioxide (Mn2O3) in healthy adults. Drown et al. (1986) found that following intratracheal instillation of MnCl2 and Mn3O4 in rats, the soluble chloride cleared four times faster than the insoluble oxide

from the respiratory tract. However, despite this initial difference, after 2 weeks the amounts of labeled Mn in the respiratory tract were similar for the two compounds. Recent work by Komura and Sakamoto (1993) comparing different forms of Mn in mouse diet suggested that less soluble forms such as MnO2 were taken up to a significantly greater degree in cerebral cortex than the more soluble forms of MnCl2 and manganese acetate [Mn(CH3COO)2]; however, the corpus striatal binding characteristics of the +4 valence state of Mn in MnO2 were not substantially different from those of the divalent forms in MnCl2, Mn(CH3COO)2, and manganese carbonate. Different oxidation states of certain metals (e.g., chromium, nickel, mercury) are known to have different toxicities, and some researchers have suggested that endogenous Mn can have quite different roles in Mn neurotoxicity depending on its oxidation state (e.g., Archibald and Tyree, 1987; Donaldson et al., 1982). There have been unsubstantiated claims that the higher valence states of Mn (Mn+3, Mn+4) and the higher oxides in ores (Mn2O3 and Mn3O4) are more toxic (Oberdoerster and Cherian, 1988). At present, however, insufficient information exists by which to determine the relative toxicities of different forms of Mn, and thus, for the purpose of deriving an RfC for Mn, no distinction is made among various compounds of Mn.

Because Mn is an essential element and is commonly ingested in diet, total Mn exposure is an issue. It would be desirable to know the effective target-site doses and apportion the dose to both the inhalation and oral routes of exposure. However, given the lack of data regarding oral and inhalation pharmacokinetics under environmental conditions, such quantitative apportionment is not possible at present.

Among the primary effects associated with Mn toxicity from inhalation exposure in humans are signs and symptoms of CNS toxicity. The first medical description of chronic Mn neurotoxicity (manganism) in workers is generally credited to Couper in the 1830s (NAS, 1973). Although the course and degree of Mn intoxication can vary greatly among individuals, manganism is generally considered to consist of two or three phases (Rodier, 1955). The first is the psychiatric aspect, which includes disturbances such as excessive weeping and laughing, sleep disturbance, irritability, apathy, and anorexia. These symptoms can occur independently of the second phase, neurological signs. The latter may include gait disturbances, dysarthria, clumsiness, muscle cramps, tremor, and mask-like facial expression. In addition, there may be a final stage of Mn intoxication involving symptoms of irreversible dystonia and hyperflexion of muscles that may not appear until many years after the onset of exposure (Cotzias et al., 1968). Cotzias et al. (1976) noted a parallel between these stages of symptoms and the biphasic pattern of dopamine levels over time in the Mn-exposed individuals noted above. Indeed, various specific features of Mn toxicity show biphasic patterns in which there is generally first an increase then a decrease in performance (e.g., a notable increase in libido followed by impotence, or excitement followed by somnolence) (Rodier, 1955).

In addition to studies described in the Principal and Supporting Studies, numerous investigators have reported CNS effects in workers exposed to Mn dust

or fumes (Sjoegren et al., 1990; Huang et al., 1989; Wang et al., 1989; Badawy and Shakour, 1984; Siegl and Bergert, 1982; Chandra et al., 1981; Saric et al., 1977; Cook et al., 1974; Smyth et al., 1973; Emara et al., 1971; Tanaka and Lieben, 1969; Schuler et al., 1957; Rodier, 1955; Flinn et al., 1941). Limitations in these studies generally preclude describing a quantitative concentration-response relationship. Exposure information is often quite limited, with inadequate information on the historical pattern of Mn concentrations or on the chemical composition and particle size distribution of the dust. In addition, exposure to other chemicals in the workplace is often not adequately characterized. Despite these limitations, such studies collectively point to neurobehavioral dysfunction as a primary endpoint for Mn toxicity.

The neuropathological bases for manganism have been investigated by many researchers and have indicated the involvement of the corpus striatum and the extrapyramidal motor system (e.g., Archibald and Tyree, 1987; Donaldson and Barbeau, 1985; Donaldson et al., 1982; Eriksson et al., 1987, 1992). Neuropathological lesions have generally been associated with the basal ganglia, specifically involving neuronal degeneration in the putamen and globus pallidus (e.g., Newland et al., 1987). Brain imaging studies (e.g., Wolters et al., 1989; Nelson et al., 1993) more recently have begun to provide additional insight into the brain structures involved in Mn toxicity.

In terms of the neurochemistry of Mn toxicity, several studies have shown that dopamine levels are affected by Mn exposure in humans, monkeys, and rodents, with various indications of an initial increase in dopamine followed by a longer term decrease (e.g., Cotzias et al., 1976; Bird et al., 1984; Barbeau, 1984; Brouillet et al., 1993). Some theories of Mn neurotoxicity have focused on the role of excessive Mn in the oxidation of dopamine resulting in free radicals and cytotoxicity (e.g., Donaldson et al., 1982; Barbeau, 1984). In addition, the fundamental role of mitochondrial energy metabolism in Mn toxicity has been indicated by the studies of Aschner and Aschner (1991), Gavin et al. (1992), and others. Brouillet et al. (1993) have suggested that the mitochondrial dysfunctional effects of Mn result in various oxidative stresses to cellular defense mechanisms (e.g., glutathione) and, secondarily, free radical damage to mitochondrial DNA. In view of the slow release of Mn from mitochondria (Gavin et al., 1992), such an indirect effect would help account for a progressive loss of function in the absence of ongoing Mn exposure (Brouillet et al., 1993), as Mn toxicity has been known to continue to progress in humans despite the termination of exposure (Cotzias et al., 1968; Rodier, 1955).

Because of the involvement of the dopaminergic system and extrapyramidal motor system in both Parkinson's disease and manganism, symptoms of the two diseases are somewhat similar, and several writers have suggested the possibility of a common etiology; however, many neurological specialists make a clear distinction in the etiologies and clinical features of Parkinson's disease and manganism (Barbeau, 1984; Langston et al., 1987).

Another primary endpoint of Mn toxicity has been male reproductive

dysfunction, often manifesting in symptoms such as loss of libido, impotence, and similar complaints (e.g., Rodier, 1955; Cook et al., 1974). Some neuropathological evidence suggests that the hypothalamus is a site of Mn accumulation (Donaldson et al., 1973); thus, disturbance of the hypothalamic-pituitary-gonadal axis hormones might be expected (Deskin et al., 1981) and has been examined in a few occupational studies. Lauwerys et al. (1985) reported the results of a fertility questionnaire administered to male factory workers (n = 85) exposed to Mn dust. This study involved the same population of workers for which Roels et al. (1987) reported neurobehavioral disturbances. The range of Mn levels in the breathing zone was 0.07-8.61 mg/cu.m, with a median concentration of 0.97 mg/cu.m. Average length of exposure was 7.9 years (range of 1-19 years). A group of workers (n = 81) with a similar workload was used as a control group. The number of births expected during different age intervals of the workers (16-25, 26-35, and 36-45 years) was calculated on the basis of the reproductive experience of the control employees during the same period. A decrease in the number of children born to workers exposed to Mn dust during the ages of 16-25 and 26-35 was observed. No difference in the sex ratio of the children was found. The same apparent LOAEL(HEC) (0.34 mg/cu.m) that was identified in Roels et al. (1987) for neurobehavioral effects is identified in this study for human reproductive effects.

However, a more recent report from the same group of investigators (Gennart et al., 1992), based on 70 of the alkaline battery plant workers evaluated by Roels et al. (1992), indicated that the probability of live birth was not different between the Mn-exposed and control workers. Also, in the study by Roels et al. (1992), serum levels of certain hormones related to reproductive function (FSH, LH, and prolactin) were not significantly different for the full group of 92 Mn workers vs. 102 controls. The latter results are partially supported by a preliminary report by Alessio et al. (1989), who found that serum FSH and LH levels were not significantly different in 14 workers generally exposed to <1 mg Mn/cu.m compared to controls, although prolactin and cortisol levels were significantly higher in the Mn-exposed workers. It is possible that differences in the forms of Mn to which workers were exposed in these studies may have contributed to the similarities and differences in the results, but insufficient information exists to substantiate this speculation.

Average concentrations of airborne Mn differed slightly in the reports of Gennart et al. (1992) and Roels et al. (1992), evidently because only a subset of Mn workers, presumably with different job functions, was used in the Gennart et al. (1992) analysis. The median respirable dust concentration was 0.18 mg/cu.m, and the median total dust concentration (comparable to Roels et al., 1987, and Lauwerys et al., 1985) was 0.71 mg/cu.m. Thus, if 0.34 mg/cu.m is identified as a LOAEL(HEC) based on the reports of Lauwerys et al. (1985) and Roels et al. (1987), 0.25 mg/cu.m total dust is the NOAEL(HEC) for reproductive effects based on the report of negative findings by Gennart et al. (1992).

The respiratory system is another primary target for Mn toxicity; numerous

reports of Mn pneumonitis and other effects on the respiratory system have appeared in the literature, dating back to 1921 (NAS, 1973). In their cross-sectional study of workers exposed to mixed Mn oxides and salts (described in the Principal and Supporting Studies), Roels et al. (1987) found that significantly greater prevalences of coughs during the cold season, dyspnea during exercise, and recent episodes of acute bronchitis were reported in the exposed group on a self-administered questionnaire. However, objectively measured lung function parameters were only slightly altered and only in Mn-exposed smokers (also see Saric and Lucic-Palaic, 1977, regarding a possible synergism between Mn and smoking in producing respiratory symptoms). In their more recent study, Roels et al. (1992) found no significant differences between MnO2-exposed and control workers in responses to a questionnaire regarding respiratory symptoms. Nor were objective spirometric measurements significantly different for the two groups. The LOAEL(HEC) for respiratory effects is 0.34 mg/cu.m total dust, based on the Roels et al. (1987) study, and the NOAEL(HEC) is 0.05 mg/cu.m respirable dust, based on the Roels et al. (1992) study. In view of the near equivalence of the geometric mean total dust concentrations in the 1987 and 1992 studies by Roels et al. (0.94 and 0.95 mg/cu.m, respectively), there in fact may be little difference between the LOAEL(HEC) and the NOAEL(HEC) in terms of air concentrations; however, differences in the forms of Mn (MnO2 vs. mixed Mn oxides and salts) to which the workers in the two studies were exposed make it difficult to compare these values quantitatively.

Nogawa et al. (1973) investigated an association between atmospheric Mn levels and respiratory endpoints in junior high school students. A questionnaire focusing on eye, nose, and throat symptoms and pulmonary function tests were given to students attending junior high schools that were 100 m (enrollment = 1258) and 7 km (enrollment = 648) from a ferromanganese plant. Approximately 97-99% of the students participated. Based on measurements obtained at another time by a government agency, the 5-day average atmospheric Mn level 300 m from the plant was reported to be 0.0067 mg/cu.m.

Significant increases in past history of pneumonia, eye problems, clogged nose, nose colds, throat swelling and soreness, and other symptoms were noted among the students in the school 100 m from the plant. Those living closest to the plant reported more throat symptoms and past history of pneumonia than did students living farther away. Pulmonary function tests revealed statistically significant decreases in maximum expiratory flow, forced vital capacity (FVC), forced expiratory volume at 1 second (FEV-1), and the FVC:FEV-1 ratio in the students attending the school closer to the plant, with some measures suggesting a relationship between performance and distance of residence from the plant.

Although the results from the study of Nogawa et al. (1973) suggest an association between ambient Mn exposure and respiratory problems, limitations in the study make it difficult to interpret. No direct measurements were made of atmospheric Mn levels either in the schools or homes, and exposure levels were inferred from the distance from the plant and other indirect measures of

Mn in the environment. Also, the authors did not note whether socioeconomic variables were controlled, and this factor could well be confounded with both distance from the plant and health problems. A follow-up study by Kagamimori et al. (1973) suggested that, following reductions in Mn emissions (with apparently no reduction in sulfur dioxide or total dust) from the ferromanganese plant, students nearest the plant showed improvements in subjective symptoms and pulmonary function tests. As before, however, exposure levels were not adequately characterized to allow clear-cut conclusions.

Lloyd-Davies (1946) reported an increased incidence of pneumonia in men employed at a potassium permanganate manufacturing facility over an 8-year period. During that period, the number of workers in the facility varied from 40 to 124. Dust measurements were well described in terms of collection conditions and particle size and composition, but actual exposure levels were not evaluated. Air concentrations ranged from 9.6 to 83.4 mg/cu.m as MnO2, which constituted 41-66% of the dust. The incidence of pneumonia in the workers was 26 per 1000, compared to an average of 0.73 per 1000 in a reference group of over 5000 workers. Workers also complained of bronchitis and nasal irritation. In a continuation of this study, Lloyd-Davies and Harding (1949) reported the results of sputum and nasopharynx cultures for four men diagnosed as having lobar- or bronchopneumonia. With the exception of one of these cases, they concluded that Mn dust, without the presence of bacterial infection or other factors, caused the observed pneumonitis.

Evidence from several laboratory animal studies supports findings in Mn-exposed humans. For example, inhaled Mn has been shown to produce significant alterations in dopamine levels in the caudate and globus pallidus of Rhesus monkeys (Bird et al., 1984) and behavioral changes in mice (Morganti et al., 1985). However, species differences may complicate interpretation of certain neurobehavioral findings in laboratory animals. Unlike primates, rodents do not have pigmented substantia nigra, which is a brain region of relatively high Mn uptake and consequent involvement in neurobehavioral dysfunction. Nevertheless, rodent and primate studies show various neurochemical, neuropathological, and neurobehavioral effects resulting from Mn exposure. However, because most laboratory animal studies of Mn neurotoxicity involve exposure by routes other than inhalation, they are not described here.

Other endpoints of Mn toxicity also have been investigated with laboratory animal models of inhalation exposure. Experimental animal data qualitatively support human study findings of respiratory effects in that Mn exposure results in increased incidence of pneumonia in rats exposed to 68-219 mg/cu.m MnO2 for 2 weeks (Shiotsuka, 1984), pulmonary emphysema in monkeys exposed to 0.7-3.0 mg/cu.m MnO2 for 10 months (Suzuki et al., 1978), and bronchiolar lesions in rats and hamsters exposed to 0.117 mg/cu.m Mn3O4 for 56 days (Moore et al., 1975). Also, Lloyd-Davies and Harding (1949) induced bronchiolar epithelium inflammation, widespread pneumonia, and granulomatous reactions in rats administered 10 mg MnO2 by intratracheal injection, and pulmonary edema in rats administered 5-50 mg MnCl2 in the same fashion. However, no significant pulmonary effects were detected in other studies of rats and

monkeys exposed to as much as 1.15 mg Mn/cu.m as Mn3O4 for 9 months (Ulrich et al., 1979a,b,c) or rabbits exposed to as much as 3.9 mg Mn/cu.m as MnCl2 for 4-6 weeks (Camner et al., 1985).

Laboratory animal studies also have shown that inhaled Mn may increase susceptibility to infectious agents such as Streptococcus pyogenes in mice (Adkins et al., 1980), Enterobacter cloacae in guinea pigs (Bergstrom, 1977), Klebsiella pneumoniae in mice (Maigetter et al., 1976), and Streptococcus hemolyticus in mice (Lloyd-Davies, 1946). In general, Mn concentrations were relatively high (>10 mg/cu.m) in these studies. However, Adkins et al. (1980) concluded that, based on the regression line of the relationship between concentration and mortality in Mn-exposed mice, exposure to <0.62 mg/cu.m would result in a mortality rate that differed from controls by at least 10%.

The developmental effects of Mn have been investigated primarily from the viewpoint of the nutritional role of this element and therefore have generally involved oral exposure. Some studies indicate that neonates of various species have a greater body burden of Mn than mature individuals have, possibly because neonates do not develop the ability to eliminate Mn--and thereby maintain Mn homeostasis--until some time after birth (Miller et al., 1975; Cotzias et al., 1976; Wilson et al., 1991). Moreover, some evidence suggests that the neonate's inability to maintain Mn homeostasis is due to a limitation in the elimination of Mn rather than in its gastrointestinal absorption (Bell et al., 1989), which would suggest a potentially greater vulnerability of young individuals to excessive Mn exposure regardless of the route of exposure.

Several studies have demonstrated neurochemical alterations in young rats and mice exposed postnatally to Mn by routes other than inhalation (e.g., Kontur and Fechter, 1988; Seth and Chandra, 1984; Deskin et al., 1981; Cotzias et al., 1976). The only inhalation study of the developmental toxicity of Mn appears to be that of Lown et al. (1984). Female mice were exposed to MnO2 7 hours/day, 5 days/week for 16 weeks prior to conception and for 17 days following conception (i.e., gestational days 1-18). For the first 12 weeks, the air concentration was 49.1 mg Mn/cu.m; all later exposures were at 85.3 mg Mn/cu.m. To separate prenatal and postnatal exposure effects, a cross-fostering design was used. Although mothers exposed to MnO2 prior to conception produced significantly worse pups per litter, prenatally exposed offspring showed reduced scores on various activity measures (open field, roto-rod, and exploration) and retarded growth that persisted into adulthood. A decrease in roto-rod performance was also observed in the offspring of nonexposed mice that were fostered to Mn-exposed females during lactation. Thus, balance and coordination were affected by either gestational or postpartum exposure to MnO2.

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o INHALATION RFD CONFIDENCE : Study -- Medium Data Base -- Medium RfC -- Medium Confidence in the principal studies (Roels et al., 1987, 1992) is medium. Neither of the principal studies identified a NOAEL for neurobehavioral

effects, nor did either study directly measure particle size or provide information on the particle size distribution. The 1992 study by Roels et al. did provide respirable and total dust measurements, but the 1987 study measured only total dust. These limitations of the studies are mitigated by the fact that the principal studies found similar indications of neurobehavioral dysfunction, and these findings were consistent with the results of other human studies (Mergler et al., 1993; Iregren, 1990; Wennberg et al., 1991, 1992; as well as various clinical studies). In addition, the exposure history of the workers in the 1992 study by Roels et al. was well characterized and essentially had not changed over the preceding 15 years, thereby allowing calculation of integrated exposure levels for individual workers. However, individual integrated exposures were not established in the 1987 study of Roels et al. Confidence in the data base is medium. The duration of exposure was relatively limited in all of the principal and supporting studies, ranging from means of 5.3 and 7.1 years in the co-principal studies by Roels et al. (1992 and 1987, respectively) to a maximum of 16.7 years in the study by Mergler et al. (1993). Moreover, the workers were relatively young, ranging from means of 31.3 and 34.3 years in the co-principal studies (Roels et al., 1992 and 1987, respectively) to a maximum of 46.4 years (Iregren, 1990). These temporal limitations raise concerns that longer durations of exposure and/or interactions with aging might result in the detection of effects at lower concentrations, as suggested by results from studies involving longer exposure durations and lower concentrations (Mergler et al., 1993; Iregren, 1990). In addition, except for the 1992 study by Roels et al., in which Mn exposure was limited to MnO2, the other principal and supporting studies did not specify the

species of Mn and the proportions of the different compounds of Mn to which workers were exposed. It is not clear whether certain compounds or oxidation states of Mn are more toxic than others. Thus, it is not possible to distinguish the relative toxicity of different Mn compounds in these studies, despite some indications in the literature regarding the differential toxicity of various oxidation states of Mn. Although the primary neurotoxicological effects of exposure to airborne Mn have been qualitatively well characterized by the general consistency of effects across studies, the exposure-effect relationship remains to be well quantified, and a no-effect level for neurotoxicity has not been identified in any of these studies thus far. Finally, the effects of Mn on development and reproduction have not been studied adequately. Insufficient information on the developmental toxicity of Mn by inhalation exposure exists, and the same is true for information on female reproductive function. The study of the reproductive toxicity of inhaled Mn in males also needs to be characterized more fully. Reflecting medium confidence in the principal studies and medium confidence in the data base, confidence in the inhalation RfC is medium.

o INHALATION RFD SOURCE :

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- U.S. EPA, 1984
DOCUMENT

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o REVIEW DATES : 08/23/90, 09/19/90, 09/23/93
o VERIFICATION DATE : 09/23/93
o EPA CONTACTS :

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CAREV-

o CLASSIFICATION : D; not classifiable as to human carcinogenicity

o BASIS FOR CLASSIFICATION : Existing studies are inadequate to assess the carcinogenicity of manganese. NOTE: Manganese is an element considered essential to human health.

o HUMAN CARCINOGENICITY DATA :

None.

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o ANIMAL CARCINOGENICITY DATA :

Inadequate. DiPaolo (1964) subcutaneously or intraperitoneally injected DBA/1 mice with 0.1 mL of an aqueous of solution 1% manganese chloride twice weekly for 6 months. A larger percentage of the mice exposed subcutaneously (24/36; 67%) and intraperitoneally (16/39; 41%) to manganese developed lymphosarcomas compared with controls injected with water (16/66; 24%). In addition, tumors appeared earlier in the exposed groups than in the control groups. The incidence of tumors other than lymphosarcomas (i.e., mammary adenocarcinomas, leukemias, injection site tumors) did not differ significantly between the exposed groups and controls. A thorough evaluation of the results of this study was not possible because the results were published in abstract form.

Stoner et al. (1976) tested manganous sulfate in a mouse lung adenoma screening bioassay. Groups of strain A/Strong mice (10/sex), 6-8 weeks old, were exposed by intraperitoneal injection to 0, 6, 15 or 30 mg/kg manganous sulfate 3 times/week for 7 weeks (a total of 21 injections). The animals were observed for an additional 22 weeks after the dosing period, before sacrifice at 30 weeks. Lung tumors were observed in 12/20, 7/20, and 7/20 animals in the high, medium, and low dosage groups, respectively. The percentage of mice with tumors was elevated, but not significantly, at the highest dose level (Fisher Exact test) compared with that observed in the vehicle controls. In addition, there was an apparent increase in the average number of pulmonary adenomas per mouse both at the mid and high doses, as compared with the vehicle controls (10 mice/sex), but the increase was significant only at the high dose (Student's t-test, $p<0.05$).

In the mouse lung adenoma bioassay, certain specific criteria should be met in order for a response to be considered positive (Shimkin and Stoner, 1975). Among these criteria are an increase in the mean number of tumors per mouse and an evident dose-response relationship. While the results of this study are suggestive of carcinogenicity, the data cannot be considered conclusive since the mean number of tumors per mouse was significantly increased at only one dose, and the evidence for a dose-response relationship was marginal.

Furst (1978) exposed groups of F344 rats (25/sex) intramuscularly or by gavage to manganese powder, manganese dioxide, and manganese (II) acetylacetonate (MAA). Treatment consisted of either 9 i.m. doses of 10 mg

each of manganese powder or manganese dioxide, 24 doses of 10 mg manganese powder by gavage, or 6 i.m. doses of 50 mg of MAA. In addition, female swiss mice (25/group) were exposed intramuscularly to manganese powder (single 10 mg dose) and manganese dioxide (6 doses of 3 or 5 mg each). There was an increased incidence of fibrosarcomas at the injection site in male (40%) and female (24%) rats exposed intramuscularly to MAA compared with vehicle controls (4% male, 4% female). EPA (1984) determined that these increases were statistically significant and noted that the study results regarding MAA, an organic manganese compound, cannot necessarily be extrapolated to pure manganese or other inorganic manganese compounds. No difference in tumor incidence was found between rats and mice exposed to manganese powder and manganese dioxide and controls.

Sunderman et al. (1974, 1976) exposed male 344 rats to 0.5 to 4.4 mg manganese dust intramuscularly and found that no tumors were induced at the injection site. It was further observed that co-administration of manganese with nickel subsulfide resulted in decreased sarcoma production by comparison to nickel subsulfide alone. Subsequent studies by Sunderman et al. (1980) suggest that manganese dust may inhibit local sarcoma induction by benzo(a)pyrene.

Witschi et al. (1981) exposed female A/J mice intraperitoneally to 80 mg/kg methylcyclopentadienyl manganese tricarbonyl (MMT) and found that although cell proliferation was produced in the lungs, lung tumor incidence did not increase.

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o SUPPORTING DATA :

None.

CARDR-
o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1984, 1988

The Drinking Water Criteria Document for Manganese has received OHEA review.
DOCUMENT

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o REVIEW DATES : 05/25/88
o VERIFICATION DATE : 05/25/88
o EPA CONTACTS :

Susan Velazquez-Tutt / OHEA -- (513)569-7571

Julie Du / OST -- (202)260-7583

WQCHU-

Water and Fish Consumption: 1.0E+2 ug/L

Fish Consumption Only: None

Considers technological or economic feasibility? -- NO

Discussion -- A criterion for domestic water supplies of 5.0E+1 ug/L should minimize the objectionable qualities, such as staining of laundry and undesirable taste. There is no demonstrated relationship between organoleptic endpoints and adverse health effects.

Reference -- Quality Criteria for Water, July 1976 (PB-263943).

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

No data available

TSCA -

No data available

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de Louvain, Clos Chapelle-aux-Champs 30, BTE 3054, 1200 Bruxelles, Belgium, to J. Michael Davis, Environmental Criteria and Assessment Office (MD-52), U.S. EPA, Research Triangle Park, NC 27711, October 19.

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Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. ECAO-CIN-D008. (External Review Draft).

CREF - Witschi, H.P., P.J. Hakkinen and J.P. Kehrer. 1981. Modification of lung tumor development in A/J mice. Toxicology. 21: 37-45.

HAREF- None

[IRIS] SS 33 /cf?
USER:
7439-97-6
Search in progress

SS (33) PSTG (1)

[IRIS] SS 34 /cf?
USER:

prt dl ncar, car continuous

1 - IRIS
IRSN - 369
DATE - 940406
UPDT - 04/06/94, 4 fields
STAT - Oral RfD Assessment (RDO) pending
STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) on-line 04/01/94
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 09/07/88 CAR Carcinogen summary on-line
IRH - 09/01/89 REFS Bibliography on-line
IRH - 12/01/89 RDI Inhalation RfD now under review
IRH - 05/01/91 CAREV Text edited
IRH - 01/01/92 EXSR Regulatory Action section on-line
IRH - 04/01/94 CAR Carcinogenicity assessment noted as pending change
IRH - 04/01/94 CARDR Work group review date added
RLEN - 7224
NAME - Mercury (Inorganic)
RN - 7439-97-6
SY - hydragyrum
SY - Mercury

RDO -
o ORAL RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

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o REVIEW DATES : 08/05/85, 02/05/86, 08/19/86, 11/16/88

RDI -
o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

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o REVIEW DATES : 11/16/89, 03/22/90, 04/19/90
CAREV-
o CLASSIFICATION : D; not classifiable as to human carcinogenicity
o BASIS FOR CLASSIFICATION : No human data are available. Animal and supporting data are inadequate. NOTE: The carcinogenicity assessment for mercury (inorganic) may change in the near future pending the outcome of a further review now being conducted by the Carcinogen Risk Assessment Verification Endeavor

Work Group.

o HUMAN CARCINOGENICITY DATA :

None.

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o ANIMAL CARCINOGENICITY DATA :

When 39 BD III and BD IV rats were injected i.p. over 2 weeks with 0.1 ml metallic mercury and observed for their lifetimes, sarcomas were seen only in those tissues that had been in direct contact with the metal (Druckrey et al., 1957). No concurrent controls were reported.

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o SUPPORTING DATA :

Mitsumori et al. (1981) fed groups of 60 male and 60 female SPF ICR mice 0, 15 or 30 ppm methyl mercury chloride in the diet for up to 78 weeks. The majority of the 30 ppm groups died from neurotoxicity by week 26. Histopathology on kidney tissue from all animals surviving after 53 weeks revealed renal tumors in 13/16 males in the 15 ppm group (2 adenomas, 11 adenocarcinomas). One adenoma was detected among 37 controls surviving to week 53 or beyond, and no tumors were seen in either control or exposed females. The possible presence of tumors at other sites was not reported in this preliminary communication.

Methyl mercury hydroxide administered in the diet to Drosophila melanogaster at 5 mg/L induced chromosomal nondisjunction. Methyl and phenyl mercury produced small increases in the rate of point mutations (Ramel, 1972).

The relevance of data from studies of organic mercury to the possible carcinogenicity of inorganic mercury is uncertain.

CARDR-

o CARCINOGENICITY SOURCE :

Source Document -- U.S. EPA, 1987

The 1987 Drinking Water Criteria Document for Mercury has received Agency and external review.

DOCUMENT

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o REVIEW DATES : 01/13/88, 03/03/94

o VERIFICATION DATE : 01/13/88

o EPA CONTACTS :

W. Bruce Peirano / OHEA -- (513)569-7540

Krishan Khanna / OST -- (202)260-7588

WQCHU-

Water and Fish Consumption: 1.44E-1 ug/L

Fish Consumption Only: 1.46E-1 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of 1.44E-1 ug/L is based on consumption of contaminated aquatic organisms and water. A WQC of 1.46E-1 ug/L has also been established based on consumption of contaminated aquatic organisms alone.

Reference -- 45 FR 79318 (11/28/80); 50 FR 30784 (07/29/85)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 2.4E+0 ug/L (1-hour average)
Chronic -- 1.2E-2 ug/L (4-day average)

Marine:

Acute -- 2.1E+0 ug/L (1-hour average)
Chronic -- 2.5E-2 ug/L (4-day average)
Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute tests on a variety of species. Requirements and methods are covered in the reference to the Federal Register. The Agency recommends an exceedence frequency of no more than 3 years.

Reference -- 45 FR 79318 (11/28/80); 50 FR 30784 (07/29/85)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value -- 0.002 mg/L (Final, 1991)

Considers technological or economic feasibility? -- NO

Discussion -- EPA has promulgated a MCLG of 0.002 mg/L based on potential adverse effects (renal toxicity) in three major studies. The MCLG is based upon a DWEL of 0.01 mg/L and an assumed drinking water contribution of 20 percent.

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Health and Ecological Criteria Division / OST / (202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 0.002 mg/L (Final, 1991)

Considers technological or economic feasibility? -- YES

Discussion -- EPA has set an MCL equal to the MCLG of 0.002 mg/L.

Monitoring requirements -- Ground water systems monitored every three years; surface water systems monitored annually; systems out of compliance must begin monitoring quarterly until system is reliably and consistently below MCL.

Analytical methodology -- Manual cold vapor technique (EPA 245.1; ASTM D3223-80; SM 303F); automated cold vapor technique (EPA 245.2): PQL=0.0005 mg/L.

Best available technology -- Coagulation/filtration; Lime softening; Reverse osmosis; Granular activated carbon.

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

CERC -

Value (status) -- 1 pound (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The final RQ for mercury is based on aquatic toxicity. The available data indicate that the aquatic 96-Hour Median Threshold Limit is less than 0.1 ppm, which corresponds to an RQ of 1 pound.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed (total mercury)

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - None
IREF - None
CREF - Druckrey, H., H. Hamperl and D. Schmahl. 1957. Carcinogenic action of metallic mercury after intraperitoneal administration in rats. Z. Krebs- forsch. 61: 511-519.
CREF - Mitsumori, K., K. Maita, T. Saito, S. Tsuda and Y. Shikasu. 1981. Carcinogenicity of methylmercury chloride in ICR mice: Preliminary note on renal carcinogens. Cancer Lett. 12: 305-310.
CREF - Ramel, C. 1972. Genetic effects. In: Mercury in the Environment -- An Epidemiological and Toxicological Appraisal, L. Friberg and J. Vostal, Ed. CRC Press, Cleveland, OH. p. 169-181.

CREF - U.S. EPA. 1987. Drinking Water Criteria Document for Mercury. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC.
HAREF- None

[IRIS] SS 34 /cf?
USER:
7723-14-0
Search in progress

SS (34) PSTG (1)

[IRIS] SS 35 /cf?
USER:

prt dl ncar, car continuous

1 - IRIS
IRSN - 501
DATE - 930802
UPDT - 08/02/93, 1 field
STAT - Oral RfD Assessment (RDO) on-line 08/01/93
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) no data
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) no data
IRH - 08/01/91 RDO Oral RfD now under review
IRH - 11/01/92 RDO Oral RfD summary on-line
IRH - 11/01/92 OREF Oral RfD references on-line
IRH - 08/01/93 RDO Transposed number corrected in para 1, line 13
RLEN - 18931
NAME - Molybdenum
RN - 7439-98-7
SY - Molybdenum
SY - HSDB 5032
SY - MCHVL
SY - TSM1

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD
Increased uric acid levels	NOAEL: None LOAEL: 0.14 mg/kg-day	30 mg/kg-day	1	5E-3

Human 6-year to Lifetime Dietary Exposure Study

Koval'skiy et al., 1961

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*Conversion Factors: Dose determined from study: molybdenum (Mo) concentration in diet is 10-15 mg/day. Assumed body weight of adult male is 70 kg; 10 mg molybdenum/70-kg body weight = 0.14 mg/kg-day.

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o ORAL RFD STUDIES :

Koval'skiy, V.V., G.A. Yarovaya and D.M. Shmavonyan. 1961. Changes of purine metabolism in man and animals under conditions of molybdenum biogeochemical provinces. Zh. Obshch. Biol. 22:179-191. (Russian trans.)

In a cross-sectional epidemiology study in a Morich geoprovince of

Armenia, Koval'skiy et al. (1961) correlated the dietary intake of molybdenum with serum uric acid levels, several biochemical endpoints, and with a gout-like sickness affecting the adult population in two settlements, Ankava village and a smaller adjoining settlement. Ankava village is a large settlement over 100 years old, while the adjoining settlement (the control) is smaller and was established in the 6-year period prior to the study. This particular region was selected for two reasons: high molybdenum content in the soil and plants (38 and 190 times that of the control area) and low content of copper (Cu). Based on these figures and dietary estimates, the average adult person in the Ankava settlement received 10-15 mg of molybdenum and 5-10 mg of copper. This intake corresponds to molybdenum doses of 0.14-0.21 mg/kg-day for a 70-kg adult. These values compare with control area values of 1-2 mg of molybdenum and 10-15 mg of copper. Three hundred villagers (184 of whom were age 18 or older) from Ankava and 100 villagers (78 adults) from the adjoining settlement underwent medical examinations. Only limited data on length of residency were reported. The results from the medical exam indicated that 57 Ankava adults (31% of the adult population) and 14 adults of the new settlement (17.9% of the adult population) had gout-like symptoms as compared with 1-4% as an overall average rate. This condition was characterized by pain, swelling, inflammation and deformities of the joints, and, in all cases, an increase in the uric acid content of the blood. In a number of cases (exact number not reported), this condition was accompanied by illnesses of the GI tract, liver, and kidneys. Fifty-two adults from Ankava and five from the adjoining settlement (controls) underwent a more detailed examination in which blood copper, molybdenum, uric acid, and xanthine oxidase concentrations in blood and molybdenum, copper, and uric acid concentrations in urine were measured. The average uric acid content in blood of the 52 Ankara adults was 6.2 mg as compared with 3.8 mg, the average of the five controls. Above normal blood uric acid content (>5.5 mg) was found in 29 of the 52 adults examined; at least 17 of these 29 had gout-like symptoms. When the 52 inhabitants were segregated as to whether they were sick with gout symptoms or not, the average concentration of uric acid in blood increased to 8.1 mg (n=17) for those sick and to 5.3 mg (n=35) for those healthy. Both serum molybdenum and serum xanthine oxidase (a molybdenum-containing enzyme that converts purines to uric acid) activity were positively correlated with serum uric acid levels. Increasing urinary excretion of copper was inversely related to increasing serum levels of molybdenum. Among the group of 52 adults from Ankara, blood uric acid levels increased with increasing residency time in the region; they increased from 3.75 mg for up to 1 year, to 6.4 mg after 1-5 years, and to 6.8 mg for 5 years or more. Based on these results, a molybdenum intake of 0.14 mg/kg-day may result in serum uric acid levels elevated above the average range of the adult population (2-6 mg; White et al., 1973). This level is designated as a LOAEL.

The effect of dietary molybdenum on uric acid and copper excretion was also observed in experiments with four adult men given diets based on sorghum varieties differing widely in molybdenum content for 10 days (Deosthale and Gopalan, 1974). The urinary excretion of uric acid was unaltered at molybdenum intake levels up to 1540 ug/day (approximately 0.022 mg/kg-day). The urinary excretion of copper increased in direct proportion to dietary molybdenum intake; molybdenum intakes of 0.002 or 0.022 mg/kg-day resulted in

the urinary excretion of copper at 24 or 77 ug/day, respectively. Normal urinary copper excretion is less than 40 ug/day.

The effects of human ingestion of molybdenum in drinking water were investigated in two Colorado cities over a 2-year period (U.S. EPA, 1979). Urinary levels of molybdenum and copper and serum levels of ceruloplasmin and uric acid were compared in individuals consuming city drinking water over a 2-year period. The low-molybdenum group consisted of 42 individuals from Denver, Colorado where the molybdenum concentration in the drinking water ranged from 2 to 50 ug/L. The high-molybdenum group consisted of 13 college students from Golden, Colorado where the drinking water molybdenum concentrations were equal to or greater than 200 ug/L.

Among subjects consuming water containing up to 50 ug molybdenum/L, plasma molybdenum levels were within the normal range. No adverse health effects were observed. While higher daily urinary molybdenum was associated with higher molybdenum intake, no adverse biochemical or systemic effects were noted. The Denver subjects had a mean urinary molybdenum value of 87 +/- 18 ug/day as compared with a value of 187 +/- 34 ug/day for the Golden subjects. Higher mean serum ceruloplasmin (40.31 mg/100 mL vs. 30.41 mg/100 mL) and lower mean serum uric acid (4.35 mg/100 mL vs. 5.34 mg/100 mL) were also associated with the higher molybdenum intake. The average dietary intake of molybdenum was 180 ug/day (estimated from foods purchased at Denver area grocery stores) (Tsongas et al., 1980). When the dietary molybdenum was added to the molybdenum from the drinking water, the NOAEL for the Denver subjects was 4 ug/kg-day and 8 ug/kg-day for Golden subjects, assuming a 2-L/day water consumption and a 70-kg body weight.

When these three studies are viewed collectively, the increased serum ceruloplasmin and urinary excretion of copper observed in human studies indicates that high levels of ingested molybdenum may be associated with potential mineral imbalance. Excretion of sufficient quantities of this element may put individuals at risk for the hypochromic microcytic anemia associated with a dietary copper deficiency. Although increased copper excretion and elevated serum ceruloplasmin are not definitive adverse effects, and as presented here are associated with no frank adverse effects in a human population, the potential for mineral imbalance must be weighed in developing an RfD. Laboratory animal studies discussed below demonstrate that the effects of molybdenum on growth and melanin synthesis are more pronounced under situations where dietary copper intake is low. For this reason, the RfD was derived with the Estimated Safe and Adequate Daily Intake (ESAADI) in mind. It is important to note that the average level of copper intake in the American population from 1982 to 1986 was less than the lower limit of the ESAADI recommendation for all age and sex groups studied in the Food and Drug Administration (FDA) Total Diet Study (Pennington et al., 1989).

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o ORAL RFD UNCERTAINTY :

UF -- An uncertainty factor of 3 is used for protection of sensitive human populations and a factor of 10 for the use of a LOAEL, rather than a NOAEL,

from a long-term study in a human population. A full factor of 10 is not used for the protection of sensitive human populations because the study was conducted in a relatively large human population. The data base does not contain studies on reproductive and developmental toxicity. However, an additional uncertainty factor for these deficiencies is not considered necessary because the RfD is only slightly above the ESAADI which was derived from the molybdenum content of the average U.S. diet.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

Molybdenum is an essential dietary nutrient which is a constituent of several mammalian enzymes including xanthine oxidase, sulfite oxidase and aldehyde oxidase (NRC, 1989). The Food and Nutrition Board of the Subcommittee on the Tenth Edition of the RDAs has established ESAADI values for molybdenum of 15-40 ug/day (2.5-4.45 ug/kg-day) for infants, 25-150 ug/day (1.95-5.36 ug/kg-day) for children, and 75-250 ug/day (1.5-3.6 ug/kg-day) for adolescents and adults (NRC, 1989). These values were derived from the reported molybdenum intake of adults and older children with average American diets (ug/kg-day values are derived from the Second National Health and Nutrition Examination Survey (NHANES II). Values for infants and children were extrapolated from the adult values on the basis of body weight. The dietary intake range reported by Tsongas (1980) from foods purchased in the Denver area was 120-240 ug/day with a mean of 180 ug/day. In the 1984 FDA Total Diet Study, the molybdenum intakes of older children and adults ranged from 74-126 ug/day (Pennington and Jones, 1987). Food for this assay was purchased from grocery stores in several northeastern locations. The data from these dietary surveys support the ESAADI recommendations.

Miller et al. (1956) administered diets to groups of Holtzman rats (21 days old; 4/dosage group). The basal diet (which contained 4 mg copper/kg and 0.2 mg molybdenum/kg) was supplemented with hydrogen molybdate at 75 and 300 ppm (approximately 7.5 and 30 mg molybdenum/kg/day, respectively). Some of the groups also received 2200 ppm sulfate (as a 1:1 mixture of sodium sulfate and potassium sulfate) for 6 weeks. Molybdenum alone exerted a significant (p value not reported) growth inhibition at the 75- and 300-ppm levels (50% and 78% reduction in weight gain, respectively). The addition of sulfate reversed this inhibition at molybdenum levels of 75 ppm and reduced it at 300 ppm. The addition of molybdenum alone increased liver copper and molybdenum concentrations. These increases were reduced by sulfate supplementation. An enlargement of the femoro-tibial joint and a thickening of the epiphysis of the femur and tibia were observed in the rats receiving 75 and 300 ppm molybdenum without sulfate and in the rats receiving 2200 ppm molybdenum with sulfate. Histological examination of the femurs indicated a chondrodystrophy of the epiphyseal cartilage. The femurs in the groups receiving lower molybdenum levels were normal. This study suggested a LOAEL of 7.5 mg molybdenum/kg/day based on body weight loss and bone deformities.

Jeter and Davis (1954) tested the effects of dietary molybdenum and copper on Long-Evans rats (4 or 8 pairs/group). The rats received either the basal diet (1.78 mg copper/kg as CuSO45H2O and <1 mg molybdenum/kg as NaMoO42H2O) or the basal diet supplemented with molybdenum at approximately 2, 8 or 14 mg/kg, ad libitum daily for 13 weeks. Each diet contained 0.5 mg copper/kg. Two groups of animals also received <1 or 8 mg molybdenum/kg with 2 mg copper/kg. The weight gain of male rats given 2, 8 or 14 mg molybdenum/kg/day at the lower copper level (0.5 mg copper/kg/day) was retarded, while that of females was retarded only at the two higher molybdenum levels. Hemoglobin concentrations were not affected by any diet. Achromotrichia (depigmentation of the hair) followed by varying degrees of alopecia (balding) was observed in some but not all rats in the groups receiving 8 or 15 mg/kg-day of molybdenum. Depigmentation was occasionally observed in rats receiving approximately 2 mg molybdenum/kg/day. The change in hair coloration may be explained by the fact that a copper-containing, mixed function oxidase catalyzes the initial reaction in the synthesis of the melanin hair pigments. The 2-3 mg molybdenum/kg/day dose represents a LOAEL in this study.

The effect of excess dietary molybdenum (added as sodium molybdate) was tested in guinea pigs of unspecified strain (Arthur, 1965). In the first experiment, groups of five guinea pigs were maintained for 8 weeks on diets with varying molybdenum content. The basal diet contained 8.9 mg copper/kg, 0.3 mg molybdenum/kg, and 0.25% sulfate. Molybdenum was increased to 8000 mg molybdenum/kg in increments of 1000 mg molybdenum/kg. Assuming a body weight of 0.75 kg and food consumption of 30 g/day for guinea pigs, a dietary level of 8000 mg molybdenum/kg corresponds to 320 mg molybdenum/kg/day. Weight gains decreased as molybdenum was increased from 40-160 mg molybdenum/kg/day, and weight loss occurred above 160 mg molybdenum/kg/day. The color of the hair of the black guinea pigs changed to gray when the dose was higher than 40 mg molybdenum/kg/day. Some fatalities were reported at 200 mg molybdenum/kg/day, and approximately 75% of the animals receiving 240-320 mg molybdenum/kg/day died.

In the second part of the Arthur study (1965), the levels of copper and molybdenum were both varied with either 0, 10 or 20 mg copper/kg and 0 or 2000 mg molybdenum/kg added to the diet. All of the animals at dietary levels of 2000 mg/kg added molybdenum (80 mg molybdenum/kg/day) and either 0 or 0.4 mg copper/kg/day developed gray hair. The inclusion of 0.8 mg copper/kg/day, however, reversed this effect. All animals receiving added molybdenum accumulated molybdenum in the liver. The animals on 80 mg molybdenum/kg/day had the smallest weight gain. The failure to gain weight was only partially alleviated by the addition of copper.

In the third part of the study, three weanling guinea pigs were supplied a low-copper basal diet (5.6 mg copper/kg and 1.8 mg molybdenum/kg) with dietary additions of 0, 200, 500, 1000 or 2000 mg molybdenum (equivalent to 8, 20, 40 or 80 mg/kg-day) for 8 weeks (Arthur, 1965). Molybdenum in the blood, liver and kidneys increased with dietary molybdenum levels. An increase in copper was observed in the blood and kidneys with increasing molybdenum intake. At

40 and 80 mg molybdenum/kg/day, liver copper concentrations decreased. Guinea pigs appeared to be somewhat less sensitive than rats or rabbits to molybdenum toxicity. The level of 40 mg molybdenum/kg/day represents a LOAEL in this study based on loss of copper.

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o ORAL RFD CONFIDENCE :

Study -- Medium
Data Base -- Medium
RfD -- Medium

The level of confidence in the oral RfD for molybdenum is medium. It is based on the results of a study that examined only gross physical effects of a gout-like disease and examined some blood chemistry parameters normally associated with gout. An exhaustive analysis of blood chemistry and individual dietary habits was not done. Therefore, the results are clearly generalized for a large population. Studies in human and animals suggest that molybdenum has an adverse effect on copper homeostasis, making the changes in serum ceruloplasmin a matter of possible concern. A study that monitored a broader spectrum of hematological or clinical chemistry parameters, especially those related to copper distribution and copper metalloenzyme function, would have helped to characterize the copper-molybdenum interaction, which appears critical to the development of gout-like symptoms at very high levels of molybdenum. The proposed RfD satisfies molybdenum nutrient requirements for all healthy members of the population, based on a comparison with the ESAADI. Dietary studies conducted by Tsongas et al. (1980) and Pennington and Jones (1987) indicate that people in the U.S. are receiving between 76 and 240 ug/day (1.1-3.4 ug/kg-day, based on a 70-kg adult) in their diets. Much of these data served as the basis for the ESAADI.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- U.S. EPA, 1990

The Drinking Water Health Advisory for Molybdenum has received Agency review.

Other EPA Documentation -- None

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o REVIEW DATES : 09/21/89, 08/15/91, 09/11/91, 11/06/91, 12/12/91
o VERIFICATION DATE : 11/06/91
o EPA CONTACTS :

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IREF - None
CREF - None
HAREF- None

[IRIS] SS 23 /cf?
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7440-31-5
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*NONE-

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7440-02-0
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7440-02-0 APPEARS IN THE FOLLOWING FIELDS IN IRIS:

#	FIELD	POSTINGS
1	RN	1
2	NAME/SY	1

SPECIFY NUMBERS, ALL, OR NONE-
USER:
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[IRIS] SS 24 /cf?
USER:
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1 - IRIS
IRSN - 265
DATE - 940801
STAT - Oral RfD Assessment (RDO) on-line 01/01/92
STAT - Inhalation RfC Assessment (RDI) pending
STAT - Carcinogenicity Assessment (CAR) message 08/01/94
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 03/01/88 RDO Text clarified paragraph 1
IRH - 12/01/89 RDI Inhalation RfD now under review
IRH - 04/01/90 RDO ABC, 1986 corrected to U.S. EPA, 1986
IRH - 04/01/90 REFS Bibliography on-line
IRH - 06/01/90 RDO Oral RfD summary noted as pending change
IRH - 06/01/90 RCRA EPA contact changed
IRH - 09/01/91 RDO Oral RfD no longer pending change; no changes made
IRH - 12/01/91 RDO Text revised; additional study added
IRH - 12/01/91 RDO Text significantly revised; additional studies added
IRH - 12/01/91 OREF Oral RfD references revised to reflect new text
IRH - 01/01/92 RDO Citation year corrected for Schnegg and Kirchgessner
IRH - 01/01/92 EXSR Regulatory actions updated
IRH - 01/01/92 OREF Citation year corrected for Schnegg and Kirchgessner
IRH - 03/01/94 CAR Carcinogenicity assessment now under review
IRH - 08/01/94 CAR Message added
IRH - 08/01/94 CAR Work group review date added
RLEN - 27287
NAME - Nickel, soluble salts
RN - 7440-02-0
SY - C.I. 77775
SY - NICHEL
SY - Nickel
SY - Nickel, soluble salts

RDO -
o ORAL RFD SUMMARY :

Critical Effect	Experimental Doses*	UF	MF	RfD

Decreased body and organ weights	NOAEL: 100 ppm diet (5 mg/kg/day)	300 mg/kg/day	1	2E-2
Rat Chronic Oral Study	LOAEL: 1000 ppm diet (50 mg/kg/day)			
Ambrose et al., 1976				

*Conversion Factors: 1 ppm = 0.05 mg/kg/day (assumed rat consumption)

o ORAL RFD STUDIES :

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

Ambrose et al. (1976) reported the results of a 2-year feeding study using rats given 0, 100, 1000 or 2500 ppm nickel (estimated as 0, 5, 50 and 125 mg Ni/kg bw) in the diet. Body weights in the high-dose male and female rats were significantly decreased compared with controls. Body weight was also reduced at 1000 ppm. This reduction was significant for females at week 6 and from weeks 26 through 104, whereas males showed body weight reduction only at 52 weeks. Groups of female rats on the 1000 or 2500 ppm nickel diets (50 and 125 mg Ni/kg bw) had significantly higher heart-to-body weight ratios and lower liver-to-body weight ratios than controls. No significant effects were reported at 100 ppm (5 mg Ni/kg bw). The dose of 1000 ppm (50 mg Ni/kg bw) represents a LOAEL for this study, while the dose of 100 ppm (5 mg Ni/kg bw) is a NOAEL. In this study, 2-year survival was poor, particularly in control rats of both sexes (death: 44/50), raising some concern about the interpretation of the results of this study. A subchronic study conducted by American Biogenics Corp. (ABC, 1986) also found 5 mg/kg/day to be a NOAEL, which supports the Ambrose et al. (1976) chronic NOAEL of 5 mg/kg/day.

Dietary exposure of dogs to 2500 ppm Ni (about 63 mg/kg/day) resulted in depressed body weight gain; no effects were seen at either 100 ppm (about 2.5 mg/kg/day) or 1000 ppm Ni (about 25 mg/kg/day) in the diet (Ambrose et al., 1976). This study demonstrates that rats are the more sensitive of the two species.

ABC (1986) conducted the 90-day study with nickel chloride in water (0, 5, 35 and 100 mg/kg/day) administered by gavage to both male and female CD rats (30 animals/sex/group). The data generated in this study included clinical pathology, ophthalmological evaluations, serum biochemistry, body and organ weight changes and histopathological evaluations of selected organs (heart, kidney, liver).

The body weight and food consumption values were consistently lower than those of controls for the 35 and 100 mg/kg/day dosed males. Female rats in both high-dose groups had lower body weights than controls, but food consumption

was unaffected by the test article. Clinical signs of toxicity, such as lethargy, ataxia, irregular breathing, cool body temperature, salivation and discolored extremities, were seen primarily in the 100 mg/kg/day group; these signs were less severe in the 35 mg/kg/day group. The 5 mg/kg/day group did not show any significant clinical signs of toxicity. There was 100% mortality in the high-dose group; 6/30 males and 8/30 females died in the mid-dose group (35 mg/kg/day). Histopathologic evaluation indicated that deaths of 3/6 males and 5/8 females in the mid-dose group were due to gavage errors. At sacrifice, kidney, liver and spleen weights for 35 mg/kg/day treated males and right kidney weights for 35 mg/kg/day treated females were significantly lower than controls. Based on the results obtained in this study, the 5 mg/kg/day nickel dose was a NOAEL, whereas 35 mg/kg/day was a LOAEL for decreased body and organ weights.

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o ORAL RFD UNCERTAINTY :

UF -- An uncertainty factor of 10 is used for interspecies extrapolation and 10 to protect sensitive populations. An additional uncertainty factor of 3 is used to account for inadequacies in the reproductive studies (RTI, 1987; Ambrose et al., 1976; Smith et al., 1990) (see Additional Comments section). During the gestation and postnatal development of F1b litters in the RTI (1987) study, temperatures were about 10 degrees F higher than normal at certain times, which makes evaluation of this part of the reproductive study impossible. In the Ambrose et al. (1976) study, statistical design limitations included small sample size and use of pups rather than litters as the unit for comparison. There were also problems with the statistical analysis of the Smith et al. (1990) study.

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o ORAL RFD MODIFYING FACTOR :

MF -- None

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o ORAL RFD COMMENTS :

In addition to the effects on organ weights described in the critical study, two other sensitive endpoints exist: neonatal mortality and dermatotoxicity. While no reproductive effects have been associated with nickel exposure to humans, several studies in laboratory animals have demonstrated fetotoxicity. These studies are described below.

Following the reproductive studies is a discussion of nickel-induced dermatotoxicity in hypersensitive humans. While nickel has long been recognized as a contact irritant, many studies have also demonstrated dermal effects in sensitive humans resulting from ingested nickel. The weight-of-evidence from these studies indicates that ingested nickel may invoke an eruption or worsening of eczema; however, a dose-response relationship is difficult to establish. A few representative studies and review articles are cited below.

While the systemic toxicity data (as manifested in organ weight changes) was

used as the critical study for the RfD determination, the reproductive/fetotoxicity and the dermatotoxicity were both considered as possible endpoints upon which to base the quantitative risk assessment of nickel. The data for effects on the latter two endpoints do not demonstrate consistent dose-response relationships, and in both cases the available studies are sufficiently flawed so as to prevent their selection as the basis for the oral RfD. It is noted, however, that the RfD based on the Ambrose et al. (1976) study is considered to be protective of all endpoints with the possible exception of hypersensitive individuals as described below.

In addition to the 2-year feeding study used as the basis for the RfD, Ambrose et al. (1976) also reported reproductive toxicity of nickel. The study had some statistical design limitations including small sample size and use of pups rather than litters as the unit for comparison. Furthermore, the results were equivocal and did not clearly define a NOAEL or LOAEL. Because nickel was administered in a laboratory chow diet rather than drinking water, quantifying analogous nickel exposure via drinking water was problematic.

In a 2-generation study (RTI, 1987) nickel chloride was administered in drinking water to male and female CD rats (30/sex/dose) at dose levels of 0, 50, 250 and 500 ppm (0, 7.3, 30.8 and 51.6 mg/kg/day, estimated) for 90 days before breeding (10 rats/sex/group comprised a satellite subchronic nonbreeder group). At the 500 ppm dose level there was a significant decrease in the Po maternal body weight, along with absolute and relative liver weights. Thus, 250 ppm (30.8 mg/kg/day) was a NOAEL for Po breeders. Histopathology was performed for liver, kidney, lungs, heart, pituitary, adrenals and reproductive organs to make this assessment. This NOAEL is higher than the NOAEL derived from the chronic Ambrose et al. (1976) and subchronic gavage (ABC, 1986) assays.

In the RTI (1987) F1a generation (postnatal days 1-4) at the 500 ppm dose level the number of live pups/litter was significantly decreased, pup mortality was significantly increased, and average pup body weight was significantly decreased in comparison with controls. Similar effects were seen with F1b litters of Po dams exposed to 500 ppm nickel. In the 50 and 250 ppm dose groups increased pup mortality and decreased live litter size was observed in the F1b litters. However, these effects seen with F1b litters are questionable because the room temperature tended to be 10 degrees F higher than normal at certain times (gestation-postnatal days) along with much lower levels of humidity. As evidenced in the literature, temperatures that are 10 degrees F above normal during fetal development cause adverse effects (Edwards, 1986). Therefore, the above results seen at 50 and 250 ppm cannot be considered to be genuine adverse effects.

F1b males and females of the RTI (1987) study were randomly mated on postnatal day 70 and their offspring (F2a and F2b) were evaluated through postnatal day 21. This phase included teratological evaluations of F2b fetuses. Evaluation of the data indicated that the 500 ppm dose caused significant body weight depression of both mothers and pups, and increased neonatal mortality during the postnatal development period. The intermediate dose, 250 ppm nickel,

produced transient depression of maternal weight gain and water intake during gestation of the F2b litters. The 50 ppm nickel exposure caused a significant increase in short ribs (11%). However, since this effect was not seen in both the higher dose groups, the reported incidence of short ribs in the 50 ppm group is not considered to be biologically significant.

Schroeder and Mitchener (1971) conducted a 3-generation study in which 5 mating pairs of rats were provided drinking water containing 5 mg Ni/L (estimated as 0.43 mg/kg bw). Results of this study indicated significant increases in neonatal mortality and in the number of runts born to exposed rats compared with controls. The major weakness of this study, however, is that the end result is based on a total of five matings. The matings were not randomized and the males were not rotated. The Schroeder and Mitchener (1971) study was conducted in an environmentally controlled facility where rats had access to food and water containing minimal levels of essential trace metals. Because of the interactions of nickel with other trace metals, the restricted exposure to trace metals (chromium was estimated as inadequate) may have contributed to the toxicity of nickel.

Smith et al. (1990) also studied the reproductive and fetotoxic effects of nickel. Four groups of 34 female Long-Evans rats were given drinking water containing nickel chloride in the following concentrations of nickel: 0, 10, 50 or 250 ppm (0, 1.3, 6.8 or 31.6 mg/kg/day) for 11 weeks prior to mating and during two successive gestation periods (G1, G2) and lactation periods (L1, L2). Maternal body weight gain was reduced during G1 in mid- and high-dose females. The reproductive performance of the exposed rats was not affected. Pup birth weight was unaltered by treatment, and weight gain was reduced only in male pups exposed to 50 ppm nickel during L1. The most significant toxicological finding was the increased incidence of perinatal mortality. The proportion of dead pups per litter was elevated at the high dose in L1 and at 10 and 250 ppm in L2. While the perinatal mortality reported in this study is consistent with other reproductive studies on nickel, it is hard to define a NOAEL and LOAEL because of the absence of a clear dose-response trend at the lower doses.

Many studies have been published regarding nickel sensitivity in humans. Of the general population, approximately 8-10% of women and 1-2% of men demonstrate a sensitivity to nickel as determined by a patch test (North American Contact Dermatitis Group, 1973; Prystowsky et al., 1979). Initial sensitization to nickel is believed to result from dermal contact, but recurring flares of eczema, particularly of the hands, may be triggered by ingestion.

The human studies described below are difficult to interpret for several reasons: very small numbers of subjects (mostly women already determined to be sensitive to nickel by a patch test) were used in the studies; many investigators reported a placebo effect; many studies were not conducted in a double-blind manner, thereby introducing investigator bias; and it was often not specified whether subjects had been fasted overnight or whether there were other dietary restrictions. It is important to note that the way in which

nickel is consumed may greatly affect its bioavailability. Sunderman et al. (1989) demonstrated that 27+/-17% of the nickel in drinking water was absorbed by healthy humans whereas only 0.7+/-0.4% of the same dose of nickel ingested in food was absorbed (a 40-fold difference). One final point to bear in mind in interpreting these studies is that the subjects were generally given a bolus dose of nickel. The absorption and biokinetics following such an exposure may be quite different from an exposure which is given incrementally throughout the day.

Following an overnight fast, groups of 5 nickel-sensitive women were given 100 mL of water along with one oral dose of nickel sulfate containing 0.6, 1.25 or 2.5 mg nickel (Cronin et al., 1980). The clinical response was observed for the next 24 hours. Worsening of hand eczema was reported in 2/5 female subjects that received 0.6 mg, 3/5 at 1.25 mg and 5/5 at 2.5 mg. Erythema was observed in 1/5 (0.6 mg), 4/5 (1.25 mg) and 4/5 (2.5 mg) women. While there appears to be a good dose-response relationship, this study did not report controls. The response observed at the lowest dose may well be within background levels.

Numerous other studies have been conducted to attempt to establish the relationships between nickel exposure and dermal irritation. Kaaber et al. (1978, 1979) reported worsening of eczema following an oral challenge with 2.5 mg nickel. In the 1978 study, 17/28 subjects experienced aggravation of dermatitis following nickel ingestion. Nine of the 17 that experienced adverse effects from the nickel found that their condition improved when they adopted a low nickel diet. In the 1979 study 9/14 subjects responded negatively to nickel treatment.

Studies conducted by Gawrodger et al. (1986), Burrows et al. (1981) and Jordan and King (1979) offer different results. Jordan and King's double blind, placebo controlled investigation suggested that 0.5 mg supplement to a normal diet was safe with the possible exception of extremely sensitive individuals. Gawrodger et al. (1986) reported that 5/10 women responded to both the 0.4 and 2.5 mg doses of nickel, but 10/26 also reacted to a placebo. They determined the LOAEL of their experiment to be 5.6 mg of nickel, a dose at which 100% of the women responded. Burrows et al. (1981) administered 0.5 mg nickel twice a day on two consecutive days to 22 patients, each of whom served as her own control. There was no significant difference between the number of individuals responding to a placebo as compared to nickel. However, the placebo response was high (12/22). The authors concluded that there is probably no connection between nickel in an ordinary diet and exacerbation of dermatitis but that a higher level may aggravate dermatitis in some individuals.

Nielsen (1989) describes a study in which 12 nickel-sensitive women were challenged for a 4-day period with a diet providing 490 ug Ni/day. No changes were observed before the start of the nickel challenge to day 0 (start of challenge). On day 4, the eczema of 6 patients was considered to be worse according to both the patients' impressions and a dermatologist's evaluation. The delayed reaction in this study may be attributed to the fact that the dose

of nickel was ingested in the diet throughout the day as opposed to studies which employed a bolus dose. This difference may greatly affect the pharmacokinetics of ingested nickel.

While the previous studies on humans with a hypersensitivity to nickel were considered in developing the RfD, none of them were adequate to serve as the basis for the quantitative risk assessment. The RfD is believed to be set at a level which would not cause individuals to become sensitized to nickel; however, those who have already developed a hypersensitivity (e.g., from a dermal exposure) may not be fully protected.

One final point to bear in mind in establishing an RfD for nickel is that nickel has been shown to be an essential trace element for several animal species. Rats deprived of nickel exhibit retarded growth and low hemoglobin levels (Schnegg and Kirchgessner, 1977). A requirement for nickel has not been conclusively demonstrated in humans, but nickel is considered to be a normal constituent of the diet. Typical daily intake of nickel ranges from 100-300 ug/day.

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o ORAL RFD CONFIDENCE :

Study -- Low
Data Base -- Medium
RfD -- Medium

The chronic study (Ambrose et al., 1976) was properly designed and provided adequate toxicological endpoints; however, high mortality occurred in the controls (44/50). Therefore, a low confidence is recommended for the study. The data base provided adequate supporting subchronic studies, one by gavage and the other in drinking water (Po animals of the RTI subchronic study, 1986). A medium confidence level in the data base is recommended since there are inadequacies in the remaining reproduction data.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- U.S. EPA, 1986, 1991

The information contained in the Quantification of Toxicologic Effects for Nickel was reviewed by the Science Advisory Board in August 1990.

Other EPA Documentation -- None

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o REVIEW DATES : 04/16/87, 05/20/87, 07/16/87, 05/17/90, 08/14/91
o VERIFICATION DATE : 07/16/87
o EPA CONTACTS :

Sue Velazquez / OHEA -- (513)569-7571

Jennifer Orme / OST -- (202)260-7586

RDI -
o INHALATION RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

---

o REVIEW DATES : 11/16/89

WQCHU-

Water and Fish Consumption: 1.34E+1 ug/L

Fish Consumption Only: 1.0E+2 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of 1.34E+1 ug/L is based on consumption of contaminated aquatic organisms and water. A WQC of 1.0E+2 ug/L has also been established based on consumption of contaminated aquatic organisms alone.

Reference -- 51 FR 43665 (12/03/86)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 1.4E+3 ug/L
Chronic -- 1.6E+2 ug/L

Marine:

Acute -- 7.5E+1 ug/L
Chronic -- 8.3E+0 ug/L

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. The freshwater criteria are hardness dependent. Values given here are calculated at a hardness of 100 mg/L CaCO3. A complete discussion can be found in the referenced notice.

Reference -- 51 FR 43665 (12/03/86)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

Value -- 0.1 mg/L (nickel) (Proposed, 1990)

Considers technological or economic feasibility? -- NO

Discussion -- EPA is proposing to regulate nickel based on its potential adverse effects (reduced body and liver weights) reported in a two-year dietary study in rats. The MCLG is based upon a DWEL of 0.58 mg/L and an assumed drinking water contribution of 20 percent.

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Health and Ecological Criteria Division / OST /
(202) 260-7571 / FTS 260-7571; or Safe Drinking Water Hotline / (800) 426-4791

MCL -

Value -- 0.1 mg/L (nickel) (Proposed, 1990)

Considers technological or economic feasibility? -- YES

Discussion -- EPA is proposing an MCL equal to the proposed MCLG of 0.1 mg/L.

Monitoring requirements -- Ground water systems every 3 years; surface water systems annually; will allow monitoring at up to 10-year intervals after the system completes 3 rounds of sampling at <50% of the MCL.

Analytical methodology -- Atomic absorption (EPA 249.2; SM 304); inductively-coupled plasma (EPA 200.7; SM 305); ICP mass spectrometry (EPA 200.8): PQL= 0.050 mg/L.

Best available technology -- Ion exchange; reverse osmosis; lime softening.

Reference -- 55 FR 30370 (07/25/90)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

No data available

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

Status -- Listed (nickel) (Final, 1991)

Discussion -- "Unregulated" contaminants are those contaminants for which EPA establishes a monitoring requirement but which do not have an associated final MCLG, MCL, or treatment technique. EPA may regulate these contaminants in the future.

Monitoring requirement -- All systems to be monitored unless a vulnerability assessment determines the system is not vulnerable.

Analytical methodology -- Atomic absorption (EPA 249.2; SM 304); inductively coupled plasma (EPA 200.7; SM 305).

Reference -- 56 FR 3526 (01/30/91)

EPA Contact -- Drinking Water Standards Division / OGWDW / (202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

CERC -

Value -- 100 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- The proposed RQ for soluble nickel salts is 100 pounds, based on potential carcinogenicity. The available data indicate a hazard ranking of low and a weight-of-evidence classification of C, which corresponds to an RQ of 100 pounds.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800) 424-9346 / (202) 260-3000 / FTS 260-3000

RCRA -

Status -- Listed (total nickel)

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - Ambrose, A.M., P.S. Larson, J.R. Borzelleca and G.R. Hennigar, Jr. 1976. Long-term toxicologic assessment of nickel in rats and dogs. J. Food Sci. Technol. 13: 181-187.

OREF - ABC (American Biogenics Corp.). 1986. Ninety-day gavage study in albino rats using nickel. Draft Final Report submitted to Research Triangle Institute, P.O. Box 12194, Research Triangle Park, NC 27709.

OREF - Burrows, D., S. Creswell and J.D. Merrett. 1981. Nickel, hands and hip prostheses. Br. J. Dermatol. 105: 437-444.

OREF - Cronin, E., A. Di Michiel and S.S. Brown. 1980. Oral nickel challenge in nickel-sensitive women with hand eczema. In: Nickel Toxicology, S.S. Brown and F.W. Sunderman Jr., Ed. Academic Press, New York. p. 149-152.

OREF - Edwards, M.J. 1986. Hyperthermia as a teratogen: A review of experimental studies and their clinical significance. Terat. Carcin. Mutagen. 6: 563-582.

OREF - Gawkrodger, D.J., S.W. Cook, G.S. Fell and J.A.A. Hunter. 1986. Nickel dermatitis: The reaction to oral nickel challenge. Br. J. Dermatol. 115: 33-38.

OREF - Jordan, W.P. and S.E. King. 1979. Nickel feeding in nickel-sensitive patients with hand eczema. J. Am. Acad. Dermatol. 1(6): 506-508.

OREF - Kaaber, K., N.K. Veien and J.C. Tjell. 1978. Low nickel diet in the treatment of patients with chronic nickel dermatitis. Br. J. Derm. 98: 197-201.

OREF - Kaaber, K., T. Menne, J.C. Tjell and N. Veien. 1979. Antabuse treatment of nickel dermatitis. Chelation - a new principle in the treatment of nickel dermatitis. Contact Derm. 5: 221-228.

OREF - Nielsen, G.D. 1989. Oral challenge of nickel-allergic patients with hand eczema. In: Nickel and Human Health: Current Perspectives. Advances in Environmental Science and Technology, E. Nieboer and A.

Aitio, Ed. John Wiley and Sons, Inc., New York, NY. Chapter 16: 1.
OREF - North American Contact Dermatitis Group. 1973. Epidemiology of contact dermatitis in North America: 1972. Arch. Dermatol. 108: 537-540.
OREF - Prystowsky, S.D., A.M. Allen, R.W. Smith, J.H. Nonomura, R.B. Odom and W.A. Akers. 1979. Allergic contact hypersensitivity to nickel, neomycin, ethylenediamine and benzocaine. Relationships between age, sex, history, exposure and reactivity to standard patch tests and use tests in a general population. Arch. Dermatol. 115(8): 959-962.
OREF - RTI (Research Triangle Institute). 1987. Two generation reproduction and fertility study of nickel chloride administered to CD rats in drinking water: Fertility and reproductive performance of the Po generation (Part II of III) and F1 generation (Part III of III). Final study report. Report submitted to Office of Solid Waste Management, U.S. EPA, Washington, DC.
OREF - Schnegg, A. and M. Kirchgessner. 1977. Ni deficiency and its effect on metabolism. In: Trace Element Metabolism in Man and Animals, Vol. 3, M. Kirchgessner, Ed. Freising-Weihenstephan: Tech. Univ. Munich West Germany. p. 236-243.
OREF - Schroeder, H.A. and M. Mitchener. 1971. Toxic effects of trace elements on the reproduction of mice and rats. Arch. Environ. Health. 23: 102-106.
OREF - Schroeder, H.A., J.J. Balassa and I.H. Tipton. 1962. Abnormal trace elements in man - nickel. J. Chronic Dis. 15: 51.
OREF - Smith, M.K., J.A. George, H.F. Stober and G.L. Kimmel. 1990. Perinatal toxicity associated with nickel chloride exposure. Fund. Appl. Toxicol. Preliminary unpublished draft.
OREF - Sunderman Jr., F.W., S.M. Hopfer, K.R. Sweeney, A.H. Marcus, B.M. Most and J. Creason. 1989. Nickel absorption and kinetics in human volunteers. Proc. Soc. Exp. Biol. Med. 191: 5-11.
OREF - U.S. EPA. 1986. Health Assessment Document for Nickel. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. (Final Report). EPA/600/8- 83/012FF.
OREF - U.S. EPA. 1991. Quantification of Toxicologic Effects for Nickel. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water, Office of Science and Technology, Washington, DC.
IREF - None
CREF - None
HAREF- None

2 - IRIS
IRSN - 266
DATE - 920122
STAT - Oral RfD Assessment (RDO) no data
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 01/01/91

STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 06/01/90 CAREV Text revised
IRH - 06/01/90 REFS Bibliography on-line
IRH - 01/01/91 CAR Text edited
IRH - 01/01/91 CARI Inhalation slope factor removed (global change)
IRH - 01/01/92 EXSR Regulatory Action section on-line
RLEN - 15930
NAME - Nickel refinery dust
RN - NO CAS RN
SY - 7440-02-0
SY - NICKEL DUST
SY - NICKEL PARTICLES
SY - Nickel Refinery Dust
CAREV-
o CLASSIFICATION : A; human carcinogen
o BASIS FOR CLASSIFICATION : Human data in which exposure to nickel refinery dust caused lung and nasal tumors in sulfide nickel matte refinery workers in several epidemiologic studies in different countries, and on animal data in which carcinomas were produced in rats by inhalation and injection
o HUMAN CARCINOGENICITY DATA :

Sufficient. Nickel refinery dust from pyrometallurgical sulfide nickel matte refineries is considered a human carcinogen when inhaled. Evidence of carcinogenicity includes a consistency of findings across different countries (Clydach, Wales; Copper Cliff, Ontario; Port Colborne, Ontario; Kristiansand, Norway; and Huntington, WV) in several epidemiologic studies, specificity of tumor site (lung and nose), high relative risks, particularly for nasal cancer, and a dose-response relationship by length of exposure. Excess risks are greatest in the dustier areas of the respective refineries. At Port Colborne, Roberts et al. (1983) reported high risks of lung (SMR = 298) and nasal (SMR = 9412) cancer among men "ever exposed" to calcining, leaching, and sintering, the dustier areas of the refinery. Similar exposures and high risks of lung and nasal cancer were observed in the calcining sheds at Clydach (lung SMR = 510, nasal SMR = 26,667) (Peto et al., 1984), the sintering furnaces at Copper Cliff (lung SMR = 424, nasal SMR = 1583) (Roberts and Julian, 1982), and the roasting/smelting (lung SMR = 360, nasal SMR = 4000) and electrolysis (lung SMR = 550, nasal SMR = 2700) furnaces at Kristiansand, Norway (Magnus et al., 1982). In the study of refinery and nonrefinery workers at a nickel refinery in West Virginia, nasal cancer was exclusive to the refinery workers, with an SMR of 2443 (Enterline and Marsh, 1982). No large excess of lung cancer was observed in either refinery (SMR = 118) or nonrefinery (SMR = 107.6) employees. The data do show a dose-response relationship between cumulative nickel exposure and lung cancer response (allowing for a 20-year latent period). The dose-response relationship is consistent with findings at nickel refineries in Clydach, Wales (Peto et al., 1984) and Copper Cliff, Ontario (Chovil et al., 1981). While the dust levels

and lung cancer relative risks were much higher in the two latter refineries, all dose-response relationships appear linear, and the tumor type and sites are the same, indicating that the functional relationship spans a broad range of nickel exposures.

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o ANIMAL CARCINOGENICITY DATA :

Animal studies indicate that some nickel refinery dusts are potentially carcinogenic. Nickel refinery flue dust (20% nickel sulfate, 59% nickel subsulfide, and 6.3% nickel oxide) from Port Colborne, Canada was tested for carcinogenic potential (Gilman and Ruckerbauer, 1962) by intramuscular injection. It was found to be a strong inducer of injection-site sarcomas in Hooded (52/66) and Wistar (8/20) rats after injection of 20 or 30 mg in one or both thighs and in mice (23/40) after injection of 10 mg/thigh. Fisher et al. (1971), as reviewed by Rigaut (1983), tested nickel refinery dust (20% nickel sulfate, 59% nickel subsulfide, and 6.3% nickel oxide) by inhalation. The refinery dust was one of six types of dust exposures administered to 348 rats at 5 to 15 mg/cu.m. The combined tumor incidence for refinery dust, synthetic dust, nickel subsulfide, and iron sulfide was 11 pulmonary tumors in the 348 rats. When Wistar rats were exposed to a combination of nickel and iron dust at concentrations of 2.1 +/- 0.2 mg Ni/cu.m. and 1.9 +/- 0.2 mg Fe/cu.m (Kim et al., 1976), one of the 60 surviving rats developed lung cancer.

An intermediate of nickel refinery dust which contains nickel subsulfide, nickel oxide, and metallic nickel (Feinstein dust) was tested in albino (nonpedigree) rats at 70 mg dust/cu.m, 5 hours/day for 6 months (Saknyn and Blohkin, 1978, as reviewed by Sunderman, 1981). Squamous-cell carcinomas were found in two of the five surviving treated rats. Saknyn and Blohkin (1978) also treated the Albino rats by intraperitoneal injection of Feinstein dust at 90 to 150 mg/rat. Six of the 39 survivors developed injection-site sarcomas.

Nickel dust from roasting (31% nickel subsulfide and 33.4% nickel oxide + silicon oxide and oxides of iron and aluminum) was tested for carcinogenicity in rats by inhalation (Belobragina and Saknyn, 1964, as reviewed by Rigaut, 1983). After exposure to 80 to 100 mg/cu.m, 5 hours/day for 12 months, no tumors were found.

Three carcinogenicity studies (Schroeder and Mitchener, 1975; Schroeder et al., 1964, 1974) of nickel acetate and an unspecified nickel salt using doses of 5 ppm of nickel in the drinking water of Long-Evans rats and Swiss mice produced negative results. Ambrose et al. (1976) administered nickel sulfate hexahydrate in the diet of Wistar-derived rats and beagle dogs for 2 years at nickel concentrations of 100 to 2500 ppm. A lack of carcinogenic response was observed in both studies. The dog study may have been inadequate to detect a carcinogenic response, since the duration was relatively short.

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o SUPPORTING DATA :

Nickel refinery dust has not been studied using in vitro short-term test systems or tests for macromolecular interactions.

CARI -
o CLASSIFICATION : A; human carcinogen
o BASIS FOR CLASSIFICATION : Human data in which exposure to nickel refinery dust caused lung and nasal tumors in sulfide nickel matte refinery workers in several epidemiologic studies in different countries, and on animal data in which carcinomas were produced in rats by inhalation and injection
o INHALATION UNIT RISK : 2.4E-4 per (ug/cu.m)
o DOSE EXTRAPOLATION METHOD : Additive and multiplicative
o RISK/AIR CONCENTRATIONS :

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	4E-1 ug/cu.m
E-5 (1 in 100,000)	4E-2 ug/cu.m
E-6 (1 in 1,000,000)	4E-3 ug/cu.m

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o INHALATION DOSE-RESPONSE DATA :

Estimates of Incremental Unit Risks for Lung Cancer due to Exposure to 1 ug Ni/cu.m for a Lifetime Based on Extrapolations from Epidemiologic Data Sets

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Study	Relative Risk Model
Huntington, WV (Enterline and Marsh, 1982) (maximum likelihood estimates only)	
Refinery workers	1.5E-5 - 3.1E-5
Nonrefinery workers	9.5E-6 - 2.1E-5
Copper Cliff, Ontario (Chovil et al., 1981)	1.1E-5 - 8.9E-5
Clydach, Wales (Peto et al., 1984)	8.1E-5 - 4.6E-4
Kristiansand, Norway (Magnus et al., 1982)	1.9E-5 - 1.9E-4
Midpoint of range for refinery workers	2.4E-4

---

o ADDITIONAL COMMENTS :

Nickel refinery dust is a mixture of many nickel moieties, and it is not

certain what the carcinogenic nickel species is in the refinery dust.

Data sets from nickel refineries in Huntington, WV (Enterline and Marsh, 1982), Copper Cliff, Ontario (Chovil et al., 1981), Clydach, Wales (Peto et al., 1984), and Kristiansand, Norway (Magnus et al., 1982) provide information available for choice of model or for separation of risk by the type of nickel exposure. The dose-response curves for nasal cancer were not used for risk estimation because nasal cancer risk from nickel is thought to be an occupational hazard associated only with the pyrometallurgical process, and these tumors are not found in the general public to the same extent as lung tumors. The same lung tumor type was found in all epidemiologic studies of occupational exposure to nickel refinery dust. The average relative risk model was applied to the Huntington, WV and Copper Cliff, Ontario data sets.

For the four data sets analyzed, both the additive and multiplicative excess risk models were fitted whenever possible. The relative risk or multiplicative model follows the assumption that the background cause-age-specific rate at any time is increased by an amount proportional to the cumulative dose up to that time. The model assumes the standardized mortality ratio (SMR) is linearly related to dose and is constant for a set cumulative exposure. Excess mortality for a set cumulative exposure is constant over time, and excess risk remains constant once exposure ceases. The relative risk model differs from the additive risk model in that the latter model assumes that the excess cause-age-specific rate is increased by an amount proportional to the cumulative exposure up to that time.

The unit risk estimates ranged from 1.1E-5 to 4.6E-4 per (ug/cu.m). The estimates from the Huntington refinery were somewhat lower, but this may be a result of only the small sample size. If the nasal cancer deaths are added to the eight lung cancer deaths, the unit risk estimate becomes 1.3E-4 per (ug/cu.m), well within the range of the other estimates. As the best estimate, the midpoint of the range, 2.4E-4 per (ug/cu.m), is taken as the incremental unit risk due to a lifetime exposure to nickel matte refinery dust. When the additive risk model is applied to the data for Huntington, WV, the estimates (2.8E-4 and 1.8E-4 for refinery and nonrefinery workers, respectively) are close to those derived by the relative risk model.

The above unit risk should not be used if the air concentration exceeds 40 ug/cu.m, since above this concentration the unit risk may not be appropriate.

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o DISCUSSION OF CONFIDENCE :

Four data sets, all from humans, offer a range of incremental unit risk estimates which are consistent with each other.

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o CARCINOGENICITY SOURCE :

U.S. EPA. 1986. Health Assessment Document for Nickel and Nickel Compounds. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA/600/8-83/012FF.

Chovil, A., R.B. Sutherland and M. Halliday. 1981. Respiratory cancer in a cohort of sinter plant workers. Br. J. Ind. Med. 38: 327-333.

Enterline, P.E. and G.M. Marsh. 1982. Mortality among workers in a nickel refinery and alloy manufacturing plant in West Virginia. J. Natl. Cancer Inst. 68: 925-933.

Magnus, K., A. Andersen and A. Hogetveit. 1982. Cancer of respiratory organs among workers at a nickel refinery in Norway. Int. J. Cancer. 30: 681-685.

Peto, J., H. Cuckle, R. Doll, C. Hermon and L.G. Morgan. 1984. Respiratory cancer mortality of Welsh nickel refinery workers. In: Nickel in the Human Environment: Proceedings of a Joint Symposium, March, 1983. IARC Scientific Publ. No. 53. International Agency for Research on Cancer, Lyon, France, p. 36-46.

The 1986 Health Assessment Document has received both Agency and external review.

DOCUMENT

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o REVIEW DATES : 04/01/87
o VERIFICATION DATE : 04/01/87
o EPA CONTACTS :

Steven P. Bayard / ORD -- (202)260-5722 / FTS 260-5722

Herman J. Gibb / ORD -- (202)260-5898 / FTS 260-5898

WQCHU-

Water and Fish Consumption: 1.34E+1 ug/L (total nickel)

Fish Consumption Only: 1.0E+2 ug/L (total nickel)

Considers technological or economic feasibility? -- NO

Discussion -- The WQC of 1.34E+1 ug/L is based on consumption of contaminated aquatic organisms and water. A WQC of 1.0E+2 ug/L has also been established

based on consumption of contaminated aquatic organisms alone.

Reference -- 51 FR 43665 (12/03/86)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

WQCAQ-

Freshwater:

Acute -- 1.4E+3 ug/L (total nickel)
Chronic -- 1.6E+2 ug/L (total nickel)

Marine:

Acute -- 7.5E+1 ug/L (total nickel)
Chronic -- 8.3E+0 ug/L (total nickel)

Considers technological or economic feasibility? -- NO

Discussion -- Criteria were derived from a minimum data base consisting of acute and chronic tests on a variety of species. The freshwater criteria are hardness dependent. Values given here are calculated at a hardness of 100 mg/L CaCO3. A complete discussion can be found in the referenced notice.

Reference -- 51 FR 43665 (12/03/86)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

TSCA -

No data available

OREF - None
IREF - None
CREF - Ambrose, A.M., P.S. Larson, J.F. Borzelleca and G.R. Hennigar. 1976. Long term toxicologic assessment of nickel in rats and dogs. J. Food Sci. Tech. 13: 181-187.
CREF - Belobragina, G.V. and A.V. Saknyn. 1964. An experimental study into the effects produced by dust in nickel industry. Gig. Tr. Prof. Zabol. 7: 8 (1963), Abst. Bull. Hyg. 39: 497.

CREF - Chovil, A., R.B. Sutherland and M. Halliday. 1981. Respiratory cancer in a cohort of sinter plant workers. Br. J. Ind. Med. 38: 327-333.
CREF - Enterline, P.E. and G.M. Marsh. 1982. Mortality among workers in a nickel refinery and alloy manufacturing plant in West Virginia. J. Natl. Cancer Inst. 68(6): 925-933.
CREF - Gilman, J.P.W. and G.M. Ruckerbauer. 1962. Metal carcinogenesis. I. Observations on the carcinogenicity of a refinery dust, cobalt oxide, and colloidal thorium dioxide. Cancer Res. 22: 152-157.
CREF - Kim, M.K., A.M. Fisher and R.J. Mackay. 1976. Pulmonary effects of metallic dusts--nickel and iron [master's thesis]. University of Toronto, Toronto, Ontario, Canada.
CREF - Magnus, K., A. Andersen and A. Hogetveit. 1982. Cancer of respiratory organs among workers at a nickel refinery in Norway. Int. J. Cancer. 30: 681-685.
CREF - Peto, J., H. Cuckle, R. Doll, C. Hermon and L.G. Morgan. 1984. Respiratory cancer mortality of Welsh nickel refinery workers. In: Nickel in the Human Environment: Proceedings of a Joint Symposium, March, 1983. IARC Scientific Publ. No. 53. International Agency for Research on Cancer, Lyon, France, p. 36-46.
CREF - Rigaut, J.P. 1983. Rapport preparatoiie sur Les criteres de Sante Pour le nickel. Doc. CCE/Lux/V/E/24/83. (Trans. Fre.)
CREF - Roberts, R.S. and J.A. Julian. 1982. Mortality study of Canadian nickel miners. In: Proceedings of the Conference on Health Issues-related to Metal and Non-metallic Mining. Wagner, W.L., W.N. Rom and J.A. Merchant Eds. Park City, UT. p. 241-260.
CREF - Roberts, R.S., J.A. Julian, D.C.F. Muir and H. Shannon. 1983. Cancer mortality associated with nickel sintering. Occupational Health Faculty of Health Sciences. McMaster University Hamilton, Ontario, Canada. Presented at the IARC Nickel Symposium March 1983, Lyon, France.
CREF - Saknyn, A.V. and V.A. Blohkin. 1978. On the development of malignamt tumors in rats exposed to nickel containing aerosols. Vopr. Onkol. 24(4): 44-48.
CREF - Schroeder, H.A., J.J. Balassa and W.H. Vinton Jr. 1964. Chromium, lead, cadmium, nickel and titanium in mice: Effect on mortality, tumors and tissue levels. J. Nutr. 83: 239-250.
CREF - Schroeder, H.A., M. Mitchener and A.P. Nason. 1974. Life-term effects of nickel in rats: Survival, tumors, interactions with trace elements and tissue levels. J. Nutr. 104: 239-243.
CREF - Schroeder, H.A. and M. Mitchener. 1975. Life-term effects of mercury, methyl mercury, and nine other trace metals on mice. J. Nutr. 105: 452-458.
CREF - Sunderman, F.W. 1981. Recent research on nickel carcinogenesis. Environ. Health Perspect. 40: 131-141.
CREF - U.S. EPA. 1986. Health Assessment Document for Nickel and Nickel Compounds. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA/600/8- 83/012FF.
HAREF- None

[IRIS] SS 24 /cf?
USER:
7440-42-8
Search in progress

SS (24) PSTG (1)

[IRIS] SS 25 /cf?
USER:

prt dl ncar, car continuous

1 - IRIS
IRSN - 499
DATE - 910805
UPDT - 08/05/91, 52 fields
STAT - Oral RfD Assessment (RDO) pending
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) no data
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) no data
IRH - 08/01/91 RDO Oral RfD now under review
RLEN - 521
NAME - Vanadium
RN - 7440-62-2
SY - HSDB 1022
SY - VANADIUM
SY - VANADIUM-51

RDO -
o ORAL RFD SUMMARY :

A risk assessment for this substance/agent is under review by an EPA work group.

[IRIS] SS 37 /cf?
USER:
7440-66-6
Search in progress

SS (37) PSTG (1)

[IRIS] SS 38 /cf?
USER:

prt dl ncar, car continuous

1 - IRIS
IRSN - 458
DATE - 921007
UPDT - 10/07/92, 2 fields
STAT - Oral RfD Assessment (RDO) on-line 10/01/92
STAT - Inhalation RfC Assessment (RDI) no data
STAT - Carcinogenicity Assessment (CAR) on-line 02/01/91
STAT - Drinking Water Health Advisories (DWHA) no data
STAT - U.S. EPA Regulatory Actions (EXSR) on-line 01/01/92
IRH - 02/01/91 CAR Carcinogenicity assessment on-line
IRH - 02/01/91 REFS Bibliography on-line
IRH - 01/01/92 EXSR Regulatory Action section on-line
IRH - 10/01/92 RDO Oral Rfd summary on-line
IRH - 10/01/92 OREF Oral RfD references on-line
RLEN - 43680
NAME - Zinc and Compounds
RN - 7440-66-6
SY - Zinc
SY - Asarco L 15
SY - Blue powder
SY - Cinc [Spanish]
SY - EMANAY ZINC DUST
SY - GRANULAR ZINC
SY - HSDB 1344
SY - JASAD
SY - Lead refinery vacuum zinc
SY - Merrillite
SY - UN 1436
SY - Zinc
SY - ZINC DUST
SY - ZINC POWDER
SY - ZINC, ashes
SY - ZINC, powder or dust, non-pyrophoric
SY - ZINC, powder or dust, pyrophoric

RDO -
o ORAL RFD SUMMARY :

NOTE: This RfD for the soluble salts of zinc supplies adequate zinc to meet the requirements in adolescents and adults over a lifetime without any concurrent physiological impairment. It does not supply the Recommended Daily Allowance (RDA) to those members of the population who have greater requirements for a short, less-than-lifetime duration, for example, infants, pre-adolescent children, or, possibly, lactating women. For short-term requirements in infants, pre-adolescent children, and lactating females, refer to the RDAs (NRC, 1989).

At a Workshop on the "Risk Assessment of Essential Elements" (Herndon, VA; March 10-12, 1992), several nutritionists commented on the derivation of the zinc RfD. The most relevant comment raised the issue of zinc bioavailability from various media. Dr. Harold Sandstead (1992) summarized this viewpoint and suggested the following values for zinc RfDs from various media: zinc supplements - 0.25 mg/kg/day; "omnivores" - 0.7 mg/kg/day; and vegetarians - 1.7 mg/kg/day. The proposed RfD for individuals consuming supplements, which is roughly comparable to soluble salts of zinc, is quite similar to the RfD verified by EPA's RfD/RfC Work Group. This agreement between the nutritionists and the toxicologists gives the EPA greater confidence in the verified RfD.

Critical Effect	Experimental Doses*	UF	MF	RfD
47% Decrease in erythrocyte superoxide dismutase (ESOD) concentration in adult females after 10 weeks of zinc exposure	NOAEL: None LOAEL = 59.72 mg/day (1.0 mg/kg/day)	3	1	3E-1 mg/kg/day

Human Diet Supplement Study

Yadrick et al., 1989

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*Conversion Factors: The dose conversion factors were based on a 60-kg reference female body weight. Total dose was derived from estimations from the FDA Total Diet Study for 1982-1986, plus reported supplemental dose. For example, for the Yadrick et al., 1989 study, the dose is 1.0 mg/kg-day based on 50 mg zinc supplement plus 9.72 mg/day zinc from the diet (total of 60), divided by the assumed average body weight of the participants (60 kg).

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o ORAL RFD STUDIES :

Yadrick, M.K., M.A. Kenney and E.A. Winterfeldt. 1989. Iron, copper, and zinc status: Response to supplementation with zinc or zinc and iron in adult females. Am. J. Clin. Nutr. 49: 145-150.

The oral RfD is based on a clinical study which investigated the effects of oral zinc supplements on copper and iron balance. This study is supported by several other studies which indicate that zinc supplementation can alter copper balance. The effects on copper and iron biochemistry are considered of concern since long-term iron or copper deficiency could result in significant adverse effects. For example, zinc supplementation therapy with megadoses of up to 5 g/day, as well as smaller amounts of 150 mg/day, taken for 1 to 2 years have produced copper deficiency anemia (Fischer et al., 1984). In addition, several studies have investigated the effects of zinc supplementation on the high-density lipoprotein (HDL) levels of adult males. These have been added as supporting studies because the observed change in HDL

values in males may be significant since a sustained decrease in HDL concentrations may be associated with increased risk of coronary artery disease when combined with a parallel increase in low-density lipoprotein (LDL) cholesterol.

A 10-week study of zinc supplementation in 18 healthy women given zinc gluconate supplements twice daily (50 mg zinc/day, or 1.0 mg/kg-day, see below) resulted in a decrease of erythrocyte superoxide dismutase (ESOD) activity (Yadrick et al., 1989). ESOD concentrations declined over the 10-week supplementation period and at 10 weeks were significantly different ($p<0.05$) from values during the pretreatment period. By 10 weeks, ESOD activity had declined to 53% of pretreatment levels. Change in enzyme activity is considered a better indicator of altered copper status than a measure of metal concentration in tissue or plasma. This has been documented by studies in rats fed copper-deficient or high-zinc diets, in which copper metalloenzyme activity is greater and precedes changes in plasma or tissue levels of copper (L'Abbe and Fischer, 1984a,b). Ceruloplasmin concentrations were not altered. Serum zinc was significantly increased. There was also a significant decline in serum ferritin and hematocrit values at 10 weeks. Such a decrease could pose a significant risk to the iron status of women.

No measurements were made of dietary zinc or copper in this study. However, a level of dietary zinc can be estimated at 9.72 mg/day for females (20- to 30-years old) from the results of the FDA Total Diet Study for 1982-1986 (Pennington et al., 1989). The LOAEL of 1.0 mg/kg-day was calculated from the sum of these dietary estimates and the supplemental zinc dose using an assumed body weight of 60 kg for adult females, as shown in the conversion factor section.

Support for considering the intake of 50 mg/kg-day supplemental zinc as a threshold LOAEL is provided by Fischer et al. (1984) which also suggests that zinc affects copper balance at doses of 0.95 mg/kg-day in males. Healthy men given 25 mg of zinc as gluconate twice daily for a 6-week period displayed a significant decrease ($p <0.05$) in erythrocyte superoxide dismutase (ESOD) activity at the end of 6 weeks exposure. There were no differences between serum copper levels or ceruloplasmin activity in the 13 members of the supplement group compared with controls. Serum zinc levels were significantly increased in the supplement group after 2 weeks.

Prasad et al. (1978) fed a patient with sickle cell anemia supplements of 150 to 200 mg zinc/day for 2 years. The supplement resulted in copper deficiency; serum copper and plasma ceruloplasmin levels were decreased. When copper was administered, the plasma ceruloplasmin levels became normal. In a follow-up study, of 13 patients on zinc therapy (similar treatment levels assumed), 7 patients had ceruloplasmin levels at the lower limit of normal after 24 weeks of dosing.

In a 9-week study, Festa et al. (1985) fed nine male students diets containing 2.6 mg copper/day and 1.8-20.7 mg zinc/day for 1- to 2-week periods. This study indicated that fecal copper excretion was influenced by

the amount of zinc in the diet and the length of time it was administered. Typically, after 1-2 weeks at 18.5 mg/day (just 3.5 mg/day higher than the adult RDA), subjects lost significantly more copper in the feces. Plasma copper concentrations were unchanged.

Groups of 9, 13 or 9 healthy white men were administered 0, 50, or 75 mg/kg-day zinc as zinc gluconate, respectively, for 12 weeks (Black et al., 1988). The subjects were given instructions to avoid foods high in calcium, fiber and phytic acid, dietary constituents that have a negative impact on zinc absorption. Subjects were also told to restrict their intake of zinc-rich foods in order to minimize the variation in daily dietary zinc. Three-day dietary records were collected on a biweekly basis. These records indicated that the dietary zinc intakes of the three treatment groups were 12.5, 14.0, and 9.5 mg/day for the groups receiving 0, 50, and 75 mg/kg-day supplement, respectively. Based on the average body weights for each treatment group, these doses correspond to a total zinc intake of 0.16, 0.85, and 1.10 mg/kg-day.

Biweekly blood samples were collected from all subjects and analyzed for total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, zinc, and copper. Urinary zinc and copper values were also determined. There was a general decline in the mean serum HDL-cholesterol for the 75-mg supplement group between weeks 6 and 12. HDL values for this group were significantly lower than those for the placebo group at weeks 6 and 12 ($p < 0.05$). When the mean HDL-cholesterol level of these subjects was compared to population percentile norms, there was a decline from the 92nd to the 77th percentile (Simko et al., 1984) in 6 weeks, followed by a relative stabilization of HDL values for the remaining 6-week test period. There was also a decline in the HDL values for the 50-mg group between weeks 8 through 12; however, this decline was not significantly different (p 0.05) from that for the controls until the 12th week of treatment. Over the 12-week period the HDL values for the 50-mg group declined from the 90th to the 77th population percentile norms. Serum zinc, copper, total cholesterol, LDL-cholesterol and triglycerides did not appear to be affected by treatment. While it is not absolutely certain that the 50-mg zinc/day supplement represents a clearly biologically significant endpoint, this level, when viewed collectively with other studies investigating effects on HDL-cholesterol, may signify the beginning of the dose-response trend. The significance of this change is unknown in light of an absence of increase in LDLs.

Zinc supplementation (160 mg as zinc sulfate) was found to lower HDL-cholesterol values in 11 healthy men when administered over 5 weeks (Hooper et al., 1980). A control group of eight subjects received a placebo. Fasting cholesterol, HDL-cholesterol, and triglycerides were determined on a weekly basis for 7 weeks and again 11 weeks after the end of supplementation. Dietary zinc levels were not measured; however, in the FDA Total Diet Study, adult males consumed an average of 16.41 mg/day during 1982-1987 (Pennington et al., 1989). Based on a 70-kg average body weight and 16.41 mg/day dietary zinc, the average dietary zinc intake for those receiving a supplement was 2.52 mg/kg-day.

After an initial HDL increase during the first 2 weeks of supplementation, HDL levels were significantly lower than those for the controls during weeks 4 through 7 ($p = 0.002$ to 0.0001). HDL levels returned to normal 11 weeks after supplementation had ended. The 11 subjects of this study had initial mean HDL values below average for their age category (23-35 years old). During the first 7 weeks of monitoring, their HDL percentile values fell from the 36th to the 8th population percentile norm. Percentile standings lower than 10 are associated with cardiovascular risk. Serum cholesterol, LDL-cholesterol, and triglycerides did not change significantly during the study; serum zinc levels increased during the supplementation period. Serum cholesterol values were normal.

A third study of the effects of zinc supplementation was conducted by Chandra (1984) in 11 adult men (ages not given). Zinc sulfate tablets were administered twice daily for a total zinc supplement intake of 300 mg/day. Average dietary zinc during the supplementation period was 10.1 mg/day, based on 24-hour recall data and 11.2 mg/day in the pre-test period. Thus, the daily zinc intake was 4.43 mg/kg-day for a 70-kg male during supplementation. Fasting serum cholesterol, HDL-cholesterol, LDL-cholesterol, and triglycerides were measured biweekly for 6 weeks; a final measurement of these parameters was conducted at 16 weeks. Total lymphocytes, T-lymphocytes, and B-lymphocytes were also measured. Lymphocyte activity was monitored through polymorphonuclear migration response to chemotactic phytohemagglutinin (PHA) stimulation and phagocytosis of opsonized bacteria.

There was a significant decrease in serum HDL values during weeks 4 and 6 ($p<0.1$ and $p<0.01$, respectively) with a return to baseline levels at week 16 (Chandra, 1984). LDL-cholesterol levels were significantly increased ($p<0.05$) at week 6, but there were no significant changes in serum cholesterol and triglycerides. During the 6-week supplement administration period, the HDL percentile values fell from the 43rd to the 6th percentile, as estimated from the population percentile norms for 30- to 35-year-old males (Simko et al., 1984).

There were no significant changes in lymphocyte counts during the period of zinc supplementation, but polymorphonuclear response to PHA stimulation (chemotactic migration) and phagocytosis were impaired (Chandra, 1984). Plasma zinc values increased during the supplement administration.

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o ORAL RFD UNCERTAINTY :

UF -- An uncertainty factor of 3 was used, based on a minimal LOAEL from a moderate-duration study of the most sensitive humans and consideration of a substance that is an essential dietary nutrient.

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o ORAL RFD MODIFYING FACTOR :

MF -- 1.

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o ORAL RFD COMMENTS :

Zinc is an essential nutrient with RDA values ranging from 5 to 15 mg/day for different age and sex categories (NRC, 1989). The RDA is an estimate of the zinc needed for growth, development, metabolism and tissue maintenance for over 98% of the healthy American population. For 79% of a 70-year lifetime (55 years), the proposed RfD of 0.3 mg/kg-day supplies adequate zinc to meet these requirements in adolescents and adults without any concurrent physiological impairment. It does not supply the RDA for infants, preadolescent children or, possibly, for lactating women.

The RfD of 0.3 mg/kg-day is expected to be without adverse effects when consumed on a daily basis over an extended period of time. It neither induces a nutritional deficiency in healthy, non-pregnant, adult humans consuming the average American diet nor causes undesirable inhibition of normal lipid transport.

When the three studies monitoring HDL-cholesterol are considered as a group, they show a consistent lowering of HDL-cholesterol levels in response to the addition of zinc to the diet, an effect which is reversed with cessation of the zinc supplementation. The data of Black et al. (1988) indicate that the depressed HDL values can persist for up to 12 weeks. Data are available from all 3 studies at 6 weeks. However, in the Hooper et al. (1980) study, the 6-week data represent HDL status 1 week after supplement administration ended. Additional data will be needed to clarify whether or not this change is significant with longer exposure.

Supplemental zinc does not appear to have the same effect on females that it has on males. Healthy adult females were given supplemental zinc doses of 0, 15, 50 or 100 mg/day zinc as zinc acetate for 60 days (Freeland-Graves et al., 1982). Plasma cholesterol, HDL-cholesterol, and zinc were monitored at biweekly intervals. A transitory decrease in HDL values was noted at 4 weeks, but only in the group receiving the 100-mg/day supplement (1.8 mg/kg-day based on a 60-kg body weight and 8.1 mg/day zinc in the diet [from diet records]). This decrease in HDL values was not apparent at 6 and 8 weeks. Serum zinc levels were also highest in these subjects at 4 weeks.

A very slight but statistically significant ($p = 0.04$) 2-mg/dL increase in HDL cholesterol was seen in a group of 22 elderly male and female subjects (sex ratio unknown) 8 weeks after they ceased using zinc supplements (Goodwin et al., 1985). Serum zinc values fell from 92 to 86 g/dL during the same period. The average supplement intake was 29.1 mg/day with a range of 17.5 to 52.2 mg/day. The increase in HDL value seemed to be greatest for the subjects with the highest ratings for physical activity. Although the data in this study are far from conclusive with regard to the relationship between zinc and HDL values, they do add to the weight of evidence which suggests that the impact of supplemental zinc on HDL levels is real.

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o ORAL RFD CONFIDENCE :

Study -- Medium
Data Base -- Medium
RfD -- Medium

The level of confidence in the studies is medium since they are well-conducted clinical studies with many biochemical parameters investigated but only few numbers of humans were tested. The confidence in the overall database is medium since these studies are all of short duration. Medium confidence in the RfD follows.

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o ORAL RFD SOURCE DOCUMENT :

Source Document -- U.S. EPA, 1990

The Drinking Water Health Advisory for Zinc has received internal Office of Water review.

Other EPA Documentation -- None

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o REVIEW DATES : 09/21/89, 08/15/91, 09/11/91, 11/06/91
o VERIFICATION DATE : 11/06/91
o EPA CONTACTS :

Robert Cantilli / OST -- (202)260-7571

Edward V. Ohanian / OST -- (202)260-7571

CAREV-
o CLASSIFICATION : D; not classifiable as to human carcinogenicity
o BASIS FOR CLASSIFICATION : Based on inadequate evidence in humans and animals.
o HUMAN CARCINOGENICITY DATA :

Inadequate. There are no reports on the possible carcinogenicity of zinc and compounds per se in humans. Case studies have been used to evaluate the effects of zinc administered for therapeutic reasons. There are reports which compare zinc levels in normal and cancerous tissue. Studies of occupational exposure to zinc compounds have also been conducted, but have limited value because they do not correlate exposure with cancer risk.

Case reports of chronic therapeutic exposure for approximately 2 years of two patients, a 59-year-old female and a 26-year-old homozygous sickle-cell male, to 100-150 mg/day zinc as zinc sulfate or zinc acetate, respectively, have reported a profound anemia associated with hypoceruloplasminemia and hypocupremia (Porter et al., 1977; Prasad et al., 1978). The conditions were corrected by copper supplementation and, in one case, withdrawal of zinc.

Habib et al. (1976) reported that average zinc concentrations in normal and hypertrophic prostate tissues were similar, approximately 6.8 umol/g, but the average zinc concentration was lower in carcinomatous prostate tissues (2.6 umol/g). These tissue samples were obtained as follows: normal prostate tissues were obtained at autopsy from 9 men 25-58 years old (average age 36); and both hyperplastic and carcinomatous prostate tissues were obtained from the biopsies of 23 men 58-87 years old (average age 70) and from 9 men 64-91 years old (average age 73), respectively. Several other studies have also shown lower average zinc concentrations in cancerous vs. normal or hypotrophic prostate tissue (U.S. EPA, 1987). NRC (1978) and U.S. EPA (1987) have reviewed other studies which have noted both high and low zinc levels in other cancerous and noncancerous tissues with no definite pattern. From these studies it could not be concluded whether zinc was a carcinogen.

Several occupational studies have been conducted on workers exposed to zinc compounds (Batchelor et al., 1926; Chmielewski et al., 1974a,b; Bobrishchev-Pushkin et al., 1977). No increase in the incidence of cancer was noted; however, the studies were designed to evaluate other endpoints and did not specifically address cancer. Other symptoms such as slight leukocytosis, occurrences of metal fume fever, respiratory disease and hypocalcemia were some of the findings noted in exposed workers. Batchelor et al. (1926) extensively investigated workers exposed to zinc in a smelter. A total of 24 workers whose exposure ranged from 2-35.5 years were selected. In most work areas the mean zinc concentrations were generally below 35 mg/cu.m, except in the zinc dust plant where concentrations of up to 130 mg/cu.m were measured. The average level of zinc in whole blood of the 24 exposed workers was 458 ug/100 mL, compared with 387 ug/100 mL in 10 control measurements. No information was given about the control subjects. Klucik and Koprda (1979) found that exposure levels to zinc oxide dust in a zinc oxide factory were on average 0.5 mg/cu.m for zinc melters and 2.44-7.15 mg/cu.m for zinc oxide packers; it was not indicated how these values were obtained. Chmielewski et al. (1974a,b) examined a group of workers who were exposed to zinc oxide in a shipyard; this included 20 ship smiths, 20 electric welders, 20 ship's pipeline fitters, and 20 zincifying workers. High concentrations of zinc oxide were found at the stands of the electric welders, who worked in containers (maximum 58 mg/cu.m, mean 18 mg/cu.m), and the ship smiths, who worked in a superstructure (maximum 50 mg/cu.m, mean 12 mg/cu.m). These workers were also exposed to other hazardous compounds, such as nitrogen oxides. Bobrishchev-Pushkin et al. (1977) studied 1018 workers in the casting shops of three copper alloy production facilities in the USSR. Four hundred and fifty-one workers from the rolling shops were used as controls. The average level of zinc oxide exposure in the casting shop was 2.1 mg/cu.m (range of 0.2-5.1 mg/cu.m), well below the USSR's maximally allowable concentration of 6 mg/cu.m. Workers were also exposed to other metals such as copper, lead and nickel.

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o ANIMAL CARCINOGENICITY DATA :

Inadequate. In a 1-year study, an unspecified number of newborn Chester

Beatty stock mice (sex not reported) were administered 0, 1000, or 5000 ppm zinc (approximately 0, 170, or 850 mg/kg/day) as zinc sulfate in drinking water (Walters and Roe, 1965). A separate group of mice received zinc oleate in the diet at an initial dose of 5000 ppm zinc; this dose was reduced to 2500 ppm after 3 months and to 1250 ppm after an additional 3 months because of mortality due to anemia. An epidemic of ectromelia caused the deaths of several mice during the first 8 weeks; consequently, additional control and test-diet groups were established. There was no difference in body weight gain between control and treated groups, except the dietary zinc group which became anemic. Survival was not reported in treated compared with control groups.

An apparent increase in the incidence of hepatomas was observed in treated mice surviving for 45 weeks or longer relative to controls (original and replacement mice pooled). The hepatoma incidence in the control, low-dose drinking water, high-dose drinking water, and test-diet group was 3/24 (12.5%), 3/28 (10.7%), 3/22 (13.6%), and 7/23 (30.4%), respectively. Incidence of malignant lymphoma in the control, low-dose drinking water, high-dose drinking water, and test-diet groups was 3/24 (12.5%), 4/28 (14.3%), 2/22 (9%), and 2/23 (8.7%), respectively. Incidence of lung adenoma in the control, low-dose drinking water, high-dose drinking water, and test-diet groups was 10/24 (41.7%), 9/28 (32.1%), 5/22 (22.7%), and 9/23 (39.1%), respectively. None of these were significantly elevated in a statistical analysis of this data performed by the EPA. In a 14-month study conducted with 150 C3H mice (sex not reported), administration of 500 mg/L zinc sulfate (approximately 100 mg/kg/day) in the drinking water resulted in hypertrophy of the adrenal cortex and pancreatic islets (Aughey et al., 1977). No tumors were noted; however, only the adrenal, pancreas and adenohypohysis were examined. Accurate consumption data could not be obtained due to spillage during drinking. No instances of adrenal or pancreatic hypertrophy were seen in a control group (number of animals not stated) that received only distilled water.

After an intratesticular injection of zinc, Guthrie observed seasonally-related testicular tumors in fowl (Guthrie, 1964) but no tumors in rats (Guthrie, 1956). Guthrie (1964) administered zinc chloride, zinc acetate or zinc stearate to groups of white leghorn chickens by intratesticular injection (approximately 0.01 g/injection); groups of chickens were sacrificed from 3 weeks to 11 months. Eight of the 111 chickens injected with zinc chloride in January and February developed testicular testoma, while none of the 48 chickens injected with zinc chloride in March developed tumors. None of the 36 chickens injected with zinc acetate in March and none of the 14 chickens injected with zinc stearate in January and February developed tumors; no conclusions about the carcinogenicity of these two compounds could be made because an insufficient number of chickens were tested. No control group was described.

Guthrie injected 0.15-0.20 mL of 10% zinc sulfate into the testis of nineteen 4-month-old rats and 0.15 mL of 5% zinc chloride into the testis of twenty-nine 3-month-old rats (strain not specified) (Guthrie 1956). No

testicular tumors were observed in either group at sacrifice 15 months after injection. No controls were described. Riviere et al. (1959) injected 5% zinc chloride in distilled water into the testicles of 100 Wistar rats. The rats were subdivided into several groups; some rats were unilaterally castrated and some rats received an injection of 200 units serum gonadotrophin and a subcutaneous implantation of a 25 mg pellet of distilbene or 100 mg testosterone. The number of rats in each of the four groups (unilateral castration +/- hormone treatment and untreated +/- hormone treatment was not stated. No control group was described. Testicular tumors (including interstitial tumors, a seminoma and an embryoma) became apparent 15 months after inoculation (tumor incidence not specified). There are no specific data on the effects of hormones in this experiment.

Halme (1961) exposed tumor-resistant and tumor-susceptible strains of mice to zinc in drinking water. In a 3-year, five-generation study, zinc chloride was added to the water of tumor-resistant mice (strain not specified); the groups received 0, 10, 20, 50, 100, or 200 mg Zn/L. The spontaneous tumor frequency for this strain of mice was 0.0004%. The tumor frequencies in the generations were: F0=0.8%, F1=3.5%, F1 and F2=7.6% and F3 and F4=25.7%. Most of the tumors occurred in the 10 and 20 mg Zn dose groups. No statistical analyses and no individual tumor-type data were reported. In the tumor-susceptible mice, strains C3H and A/Sn received 10-29 mg Zn/L in their drinking water for 2 years; 33/76 tumors were observed in the C3H strain (31 in females) and 24/74 tumors were observed in the A/Sn strain (20 in females). Most of the tumors were adenocarcinomas. The numbers of specific tumor types were not reported. The tumor frequencies (43.4% for C3H and 32.4% for A/Sn both sexes combined) were higher than the spontaneous frequency (15% for each strain), although no statistical analyses were reported.

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o SUPPORTING DATA :

In a short-term, in vivo assay, Stoner et al. (1976) injected strain A/Strong mice (20/sex/dose) intraperitoneally with zinc acetate 3 times/week for a total of 24 injections (total doses were 72, 180, or 360 mg/kg). Controls (20/sex/group) consisted of an untreated group, a vehicle control group administered 24 injections of saline and a positive control group administered a single injection of urethan (20 mg/mouse). Mice were sacrificed 30 weeks after the first injection; survival was comparable for all groups. There was no increase in number of lung tumors per mouse in treated animals relative to the pooled controls. While four thymomas were observed in zinc acetate-treated groups and none in controls, the occurrence of these tumors was not statistically significantly elevated.

Urine samples from subjects occupationally exposed in the rubber industry to a variety of compounds, including zinc oxide, were not found to be mutagenic in the microtitre fluctuation assay with Salmonella typhimurium strains TA1535, TA98 and TA100 (Crebelli et al., 1985).

The results of short-term genotoxicity assays for zinc are equivocal. Zinc acetate and/or zinc 2,4-pentanedione have been analyzed in four short-

term mutagenicity assays (Thompson et al., 1989). In the Salmonella assay (with or without hepatic homogenates), zinc acetate was not mutagenic over a dose range of 50-7200 ug/plate but zinc 2,4-pentanedione was mutagenic to strains TA1538 and TA98 at 400 ug/plate. The addition of hepatic homogenates diminished this response in a dose-dependent manner. In the mouse lymphoma assay, zinc acetate gave a dose-dependent positive response with or without metabolic activation; the mutation frequency doubled at 10 ug/mL. In the CHO in vitro cytogenetic assay, zinc acetate gave a dose-dependent positive response with or without metabolic activation, but the presence of hepatic homogenates decreased the clastogenic effect. Neither zinc acetate nor zinc 2,4-pentanedione were positive in the unscheduled DNA synthesis assay in rat hepatocytes over a dose range of 10-1000 ug/mL.

Zinc chloride is reported to be positive in the Salmonella assay (Kalinina et al., 1977), negative in the mouse lymphoma assay (Amacher and Paillet, 1980), and a weak clastogen in cultured human lymphocytes (Deknudt and Deminatti, 1978). Zinc sulfate is reported to be not mutagenic in the Salmonella assay (Gocke et al., 1981), and zinc acetate is reported to not induce chromosomal abberations in cultured human lymphocytes (Gasiorek and Bauchinger, 1981). Crebelli et al. (1985) found zinc oxide (99% purity) (1000-5000 ug/plate) to be not mutagenic for Salmonella in the reversion assay.

Responses in mutagenicity assays are thought to depend on the form (e.g., inorganic or organic salt) of the zinc tested. For example, inorganic salts tend to dissociate and the zinc becomes bound with culture media constituents. Salts that dissociate less readily tend to be transported into the cell and are postulated to cause a positive response (Thompson et al., 1989). Zinc is an essential trace element involved in numerous biological functions including growth, taste and spermatogenesis. It is a cofactor for several enzymes such as those involved in the metabolism of proteins and nucleic acids. Zinc may be a modifier of the carcinogenic response; zinc deficiency or excessively high levels of zinc may enhance susceptibility to carcinogenesis, whereas supplementation with low to moderate levels of zinc may offer protection (Woo et al., 1988). Zinc deficiency enhanced carcinomas of the esophagus induced by methylbenzylnitrosoamine (Fong et al., 1978) but retarded the development of cancer of the oral cavity induced by 4-nitroquinoline-N-oxide (Wallenius et al., 1979). In a study that examined both zinc deficiency and supplementation, Mathur (1979) found that animals with a deficient diet (5.9 mg/kg) and animals diet supplemented with excessively high levels of zinc in the diet (200-260 mg/kg) had fully developed carcinomas of the palatial mucosa. While the rats were on the specific diets, the palatial mucosa was painted with 4 nitroquinoline 3 times/week for 20 weeks. In the zinc deficient group 2/25 rats developed cancer of the palatial mucosa; 2/25 rats in the excessive zinc group also developed this form of cancer. Animals supplemented with moderate levels of zinc in the diet (50 mg/kg) developed only moderate dysplasia. Thus, zinc's modifying effect on carcinogenesis may be dose-dependent.

CARDR-
o CARCINOGENICITY SOURCE :

U.S. EPA. 1980. Ambient Water Quality Criteria for Zinc. Prepared by the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-079.

U.S. EPA. 1984. Health Effects Assessment for Zinc (and Compounds). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC.

U.S. EPA. 1987. Summary Review of the Health Effects Associated with Zinc and Zinc Oxide. Health Issue Assessment. Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA/600/8-87/022F.

U.S. EPA. 1988. Ambient Water Quality Criteria Document Addendum for Zinc. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC.

The 1984 Health Effects Assessment for Zinc (and compounds), the 1987 Health Issue Assessment and the 1980 abd 1988 Ambient Water Quality Criteria Documents have received Office of Health Effects Assessment review.
DOCUMENT

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o REVIEW DATES : 11/08/89, 06/15/90
o VERIFICATION DATE : 06/15/90
o EPA CONTACTS :

Rita S. Schoeny / ORD -- (513)569-7544 / FTS 684-7544

WQCHU-

No data available

WQCAQ-

Freshwater:

Acute -- 1.2E+2 ug/L (1-hour average)
Chronic -- 1.1E+2 ug/L (4-hour average)

Marine:

Acute -- 9.5E+1 ug/L (1-hour average)
Chronic -- 8.6E+1 ug/L (4-hour average)

Considers technological or economic feasibility? -- NO

Discussion -- The freshwater criteria are hardness dependent. Values given here are calculated at a hardness of 100 mg/L CaCO3. A complete discussion can be found in the referenced Federal Register notice.

Reference -- 52 FR 6213 (03/02/87)

EPA Contact -- Criteria and Standards Division / OWRS
(202)260-1315 / FTS 260-1315

MCLG -

No data available

MCL -

No data available

___IV.B.3. SECONDARY MAXIMUM CONTAMINANT LEVEL (SMCL) for Drinking Water

Value -- 5 mg/L [zinc] (Proposed, 1989)

Considers technological or economic feasibility? --

Discussion -- SMCLs are non-enforceable and establish limits for contaminants which may affect the aesthetic qualities (e.g. taste and odor) of drinking water It is recommended that systems monitor for these contaminants every three years. More frequent monitoring for contaminants such as pH, color, odor or others may be appropriate under certain circumstances.

Reference -- 54 FR 22062 (05/22/89)

EPA Contact -- Drinking Water Standards Division / OGWDW /
(202) 260-7575 / FTS 260-7575; or Safe Drinking Water Hotline / (800) 426-4791

___IV.B.4. REQUIRED MONITORING OF "UNREGULATED" CONTAMINANTS

No data available

CERC -

Value (status) -- 1000 pounds (Final, 1989)

Considers technological or economic feasibility? -- NO

Discussion -- Finely divided zinc metal (particles 100 microns or less in diameter) has an RQ of 1000 pounds based on chronic toxicity.

Reference -- 54 FR 33418 (08/14/89)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

RCRA -

Status -- Listed

Reference -- 52 FR 25942 (07/09/87)

EPA Contact -- RCRA/Superfund Hotline
(800)424-9346 / (202)260-3000 / FTS 260-3000

TSCA -

No data available

OREF - L'Abbe, M.R., and P.W.F. Fischer. 1984a. The effects of high dietary zinc and copper deficiency on the activity of copper-requiring metalloenzymes in the growing rat. J. Nutr. 114: 813-822.

OREF - L'Abbe, M.R., and P.W.F. Fischer. 1984b. The effects of dietary zinc on the activity of copper-requiring metalloenzymes in the rat. J. Nutr. 114: 823-828.

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HAREF- None

[IRIS] SS 38 /cf?
USER:
13494-80-9

APPENDIX R
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Lead Uptake/Biokinetic Model Results A-1

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1.0 INTRODUCTION

This appendix provides a summary of the total combined risks and hazards for the two risk assessments performed for the LBAD site. The scope of the first risk assessment (April, 1994) was limited to determine the potential risks to human populations posed by exposure to chemicals in soil, sediment, and surface water. The groundwater aquifer was treated as a separate operable unit and was therefore, evaluated in a separate risk assessment (October, 1994).

The baseline risk assessments centered upon the determination of the media-specific exposure concentrations, exposure pathways, exposure estimates, and relative noncancer hazard and carcinogenic risk of metals, semivolatile organic compounds (including pesticides and PCBs), and volatile organic compounds.

Exposures to the chemicals of concern in soil, sediment, and surface water were investigated for human receptor populations identifiable for the LBAD under both existing and future use scenarios. An LBAD worker was the only receptor identified for the existing land use scenario. Three receptor groups which were identified for the evaluation of the hypothetical future land use scenarios included a future worker, future residential adult and child receptors, and future recreational receptors. Receptor groups evaluated for exposure to groundwater include the hypothetical future worker and hypothetical future residential adult and child.

Total risks and hazards determined from the evaluation of exposure to all media of concern at the LBAD site (soil, sediment, surface water, and groundwater) are summarized on a SWMU- or Area of Concern-specific basis in Tables R-1 through R-36.

Also provided in this appendix are the results of the U.S. EPA's lead biokinetic/uptake model that were utilized to evaluate SWMUs and Areas of Concern where lead was detected in soil and groundwater above background levels.

ATTACHMENT 1

LEAD UPTAKE BIOKINETIC MODEL RESULTS

TABLE R-1
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 4 (OLD LANDFILL)
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	8.37E-06	6.65E-01
	Dermal	4.78E-05	4.65E+00
	Inhalation	1.80E-06	1.54E-02
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Surface Water	Dermal	ND	2.14E-03
Total		1.96E-04	6.15E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.02E-05	9.86E-01
	Dermal	2.87E-04	4.15E+00
	Inhalation	1.08E-05	9.25E-02
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Surface Water	Dermal	ND	2.14E-03
Total		9.42E-04	9.13E+01
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.32E-05	8.63E+00
	Dermal	1.09E-04	3.35E+01
	Inhalation	8.56E-06	7.31E-02
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Surface Water	Dermal	ND	8.70E-03
Total		5.06E-04	1.64E+02

ND – Not Determined

TABLE R-1 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 4 (OLD LANDFILL)
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.36E-06	6.65E-01
	Dermal	2.76E-05	4.65E+00
	Inhalation	1.80E-06	1.54E-02
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Surface Water	Dermal	ND	2.14E-03
Total		1.74E-04	6.15E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.82E-05	9.86E-01
	Dermal	1.66E-04	4.15E+00
	Inhalation	1.08E-05	9.25E-02
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Surface Water	Dermal	ND	2.14E-03
Total		8.09E-04	9.13E+01
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.57E-05	8.63E+00
	Dermal	6.31E-05	3.35E+01
	Inhalation	8.56E-06	7.31E-02
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Surface Water	Dermal	ND	8.70E-03
Total		4.43E-04	1.64E+02

ND – Not Determined

TABLE R-2
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 2, 5, 6 AND 7
(INDUSTRIAL AND SANITARY WASTE DISPOSAL LAND)
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.48E-04	2.39E-01
	Dermal	1.49E-03	9.72E+00
	Inhalation	2.11E-07	1.71E+01
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Sediment	Dermal	7.80E-06	3.41E-02
Total		1.78E-03	8.32E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	8.89E-04	6.39E-01
	Dermal	8.96E-03	2.21E+01
	Inhalation	1.26E-06	1.71E+01
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Sediment	Dermal	4.68E-05	4.44E-02
Total		1.05E-02	1.26E+02

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.30E-03	5.59E+00
	Dermal	3.41E-03	5.06E+01
	Inhalation	9.99E-07	8.09E+01
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Sediment	Dermal	3.16E-05	1.80E-01
Total		5.06E-03	2.59E+02

ND – Not Determined

TABLE R-2 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 2, 5, 6 AND 7
(INDUSTRIAL AND SANITARY WASTE DISPOSAL LAND)
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.26E-05	2.39E-01
	Dermal	3.28E-04	9.72E+00
	Inhalation	2.11E-07	1.71E+01
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Sediment	Dermal	1.73E-06	3.41E-02
Total		5.01E-04	8.32E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.96E-04	6.39E-01
	Dermal	1.97E-03	2.21E+01
	Inhalation	1.26E-06	1.71E+01
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Sediment	Dermal	1.04E-05	4.44E-02
Total		2.77E-03	1.26E+02
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.85E-04	5.59E+00
	Dermal	7.50E-04	5.06E+01
	Inhalation	9.99E-07	8.09E+01
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Sediment	Dermal	7.02E-06	1.80E-01
Total		1.36E-03	2.59E+02

ND – Not Determined

TABLE R-3
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 1 (NEW LANDFILL)
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Futute Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.60E-08	9.96E-02
	Dermal	5.65E-07	5.46E+00
	Inhalation	3.09E-08	1.15E+01
Groundwater	Ingestion	7.26E-05	1.61E+01
	Dermal	6.80E-07	4.92E-01
	Inhalation	1.85E-07	2.89E-04
Sediment	Dermal	2.41E-07	4.66E-05
Total		7.44E-05	3.37E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.36E-07	5.31E-01
	Dermal	3.39E-06	1.88E+01
	Inhalation	1.85E-07	1.15E+01
Groundwater	Ingestion	3.11E-04	6.19E+01
	Dermal	2.91E-06	1.21E+00
	Inhalation	7.93E-07	3.05E-02
Sediment	Dermal	1.44E-06	5.08E-05
Total		3.20E-04	9.40E+01
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.90E-07	4.65E+00
	Dermal	1.29E-06	4.29E+01
	Inhalation	1.46E-07	5.44E+01
Groundwater	Ingestion	1.59E-04	3.51E+01
	Dermal	1.12E-06	8.08E-01
	Inhalation	8.09E-07	1.24E-03
Sediment	Dermal	9.77E-07	2.06E-04
Total		1.64E-04	1.38E+02

ND – Not Determined

TABLE R-3 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 1 (NEW LANDFILL)
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Futute Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.65E-09	9.96E-02
	Dermal	3.67E-08	5.46E+00
	Inhalation	3.09E-08	1.15E+01
Groundwater	Ingestion	7.26E-05	1.61E+01
	Dermal	6.80E-07	4.92E-01
	Inhalation	1.85E-07	2.84E-04
Sediment	Dermal	1.92E-08	4.66E-05
Total		7.36E-05	3.37E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.19E-08	5.31E-01
	Dermal	2.20E-07	1.88E+01
	Inhalation	1.85E-07	1.15E+01
Groundwater	Ingestion	3.11E-04	6.19E+01
	Dermal	2.91E-06	1.21E+00
	Inhalation	7.93E-07	3.05E-02
Sediment	Dermal	1.15E-07	5.08E-05
Total		3.15E-04	9.40E+01
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.19E-08	4.65E+00
	Dermal	8.39E-08	4.29E+01
	Inhalation	1.46E-07	5.44E+01
Groundwater	Ingestion	1.59E-04	3.51E+01
	Dermal	1.12E-06	8.08E-01
	Inhalation	8.09E-07	1.24E-03
Sediment	Dermal	7.80E-08	2.06E-04
Total		1.61E-04	1.38E+02

ND – Not Determined

TABLE R-4
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR AREA A (SEPTIC TANK)
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.94E-09	7.94E-02
	Dermal	4.77E-08	6.08E+00
	Inhalation	ND	9.51E+00
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Total		1.38E-04	7.18E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.17E-08	7.94E-02
	Dermal	2.86E-07	6.08E+00
	Inhalation	ND	9.51E+00
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Total		5.94E-04	1.02E+02
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.70E-08	6.94E-01
	Dermal	1.09E-07	1.39E+01
	Inhalation	ND	4.51E+01
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Total		3.15E-04	1.81E+02

ND – Not Determined

TABLE R-4 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR AREA A (SEPTIC TANK)
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.94E-09	7.94E-02
	Dermal	4.77E-08	6.08E+00
	Inhalation	ND	9.51E+00
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Total		1.38E-04	7.18E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.17E-08	7.94E-02
	Dermal	2.86E-07	6.08E+00
	Inhalation	ND	9.51E+00
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-06	2.14E-02
Total		5.71E-04	1.02E+02
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.70E-08	6.94E-01
	Dermal	1.09E-07	1.39E+01
	Inhalation	ND	4.51E+01
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Total		3.15E-04	1.81E+02

ND – Not Determined

TABLE R-5
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR AREA B (DRAINAGE PATH NEAR WATER TOWER)
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology)
Future Residential Adult – Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	1.54E-03	7.16E-03
	Dermal	1.55E-02	7.66E-02
	Inhalation	3.36E-10	1.95E-05
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Sediment	Dermal	1.61E-05	1.02E-01
Total		1.72E-02	5.63E+01

Future Residential Adult – Long Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	9.26E-03	5.69E-02
	Dermal	9.33E-02	5.85E-01
	Inhalation	2.02E-09	1.95E-05
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Sediment	Dermal	9.66E-05	1.04E-01
Total		1.03E-01	8.68E+01

Future Residential Child – Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	1.35E-02	4.98E-01
	Dermal	3.55E-02	1.34E+00
	Inhalation	1.59E-09	9.25E-05
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Sediment	Dermal	6.53E-05	4.22E-01
Total		4.94E-02	1.24E+02

ND – Not Determined

TABLE R-5 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR AREA B (DRAINAGE PATH NEAR WATER TOWER)
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology)
Future Residential Adult – Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	1.38E-04	7.16E-03
	Dermal	1.39E-03	7.66E-02
	Inhalation	3.36E-10	1.95E-05
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Sediment	Dermal	2.42E-06	1.02E-01
Total		1.67E-03	5.63E+01

Future Residential Adult – Long Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	8.29E-04	5.69E-02
	Dermal	8.36E-03	5.85E-01
	Inhalation	2.02E-09	1.95E-05
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Sediment	Dermal	1.45E-05	1.04E-01
Total		9.80E-03	8.68E+01

Future Residential Child – Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	1.21E-03	4.98E-01
	Dermal	3.18E-03	1.34E+00
	Inhalation	1.59E-09	9.25E-05
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Sediment	Dermal	9.84E-06	4.22E-01
Total		4.72E-03	1.24E+02

ND – Not Determined

TABLE R–6
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR AREA C
LEXINGTON–BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	7.66E–02
	Dermal	ND	4.69E–01
	Inhalation	ND	1.08E–02
Groundwater	Ingestion	7.26E–05	1.61E+01
	Dermal	6.80E–07	4.92E–01
	Inhalation	1.85E–07	2.84E–04
Sediment	Dermal	3.10E–06	1.87E–01
Total		7.66E–05	1.73E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	7.66E–02
	Dermal	ND	4.69E–01
	Inhalation	ND	1.08E–02
Groundwater	Ingestion	3.11E–04	6.19E+01
	Dermal	2.91E–06	1.21E+00
	Inhalation	7.93E–07	3.05E–02
Sediment	Dermal	1.86E–05	1.87E–01
Total		3.33E–04	6.39E+01

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	6.70E–01
	Dermal	ND	1.07E+00
	Inhalation	ND	5.10E–02
Groundwater	Ingestion	1.59E–04	3.51E+01
	Dermal	1.12E–06	8.08E–01
	Inhalation	8.09E–07	1.24E–03
Sediment	Dermal	1.26E–05	7.59E–01
Total		1.74E–04	3.85E+01

ND – Not Determined

TABLE R-6 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR AREA C
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	7.66E-02
	Dermal	ND	4.69E-01
	Inhalation	ND	1.08E-02
Groundwater	Ingestion	7.26E-05	1.61E+01
	Dermal	6.80E-07	4.92E-01
	Inhalation	1.85E-07	2.84E-04
Sediment	Dermal	3.02E-07	1.87E-01
Total		7.38E-05	1.73E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	7.66E-02
	Dermal	ND	4.69E-01
	Inhalation	ND	1.08E-02
Groundwater	Ingestion	3.11E-04	6.19E+01
	Dermal	2.91E-06	1.21E+00
	Inhalation	7.93E-07	3.05E-02
Sediment	Dermal	1.81E-06	1.87E-01
Total		3.17E-04	6.39E+01

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	6.70E-01
	Dermal	ND	1.07E+00
	Inhalation	ND	5.10E-02
Groundwater	Ingestion	1.59E-04	3.51E+01
	Dermal	1.12E-06	8.08E-01
	Inhalation	8.09E-07	1.24E-03
Sediment	Dermal	1.23E-06	7.59E-01
Total		1.62E-04	3.85E+01

ND – Not Determined

TABLE R–7
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 3 (INDUSTRIAL WASTE LAGOONS)
LEXINGTON–BLUEGRASS ARMY DEPOT

(BaP Methodology) Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.61E–08	4.15E–04
	Dermal	4.48E–07	1.89E–02
	Inhalation	8.66E–08	4.69E–06
Total		5.61E–07	1.93E–02
Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.30E–07	9.14E–02
	Dermal	2.24E–06	7.97E+00
	Inhalation	4.33E–07	4.69E–06
Total		2.80E–06	8.06E+00
Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.65E–08	8.17E–05
	Dermal	8.56E–07	4.69E–03
	Inhalation	7.28E–09	ND
Groundwater	Ingestion	4.66E–05	1.93E+01
Total		4.75E–05	1.93E+01
Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.32E–07	8.06E–03
	Dermal	4.28E–06	6.77E–01
	Inhalation	3.64E–08	ND
Groundwater	Ingestion	1.66E–04	2.98E+01
Total		1.71E–04	3.05E+01

ND – Not Determined

TABLE R-7 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 3 (INDUSTRIAL WASTE LAGOONS)
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.27E-08	4.15E-04
	Dermal	1.79E-07	1.89E-02
	Inhalation	8.66E-08	4.69E-06
Total		2.78E-07	1.93E-02
Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.37E-08	9.14E-02
	Dermal	8.96E-07	7.97E+00
	Inhalation	4.33E-07	4.69E-06
Total		1.39E-06	8.06E+00
Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.41E-08	8.17E-05
	Dermal	2.04E-07	4.69E-03
	Inhalation	7.28E-09	ND
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		4.68E-05	1.93E+01
Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.04E-08	8.06E-03
	Dermal	1.02E-06	6.77E-01
	Inhalation	3.64E-08	ND
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.67E-04	3.05E+01

ND – Not Determined

TABLE R-7 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 3 (INDUSTRIAL WASTE LAGOONS)
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.30E-07	2.29E-04
	Dermal	1.20E-06	6.56E-03
	Inhalation	3.06E-08	ND
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Surface Water	Dermal	2.86E-07	1.48E-02
Seeps	Dermal	ND	3.55E-03
Sediment	Dermal	1.77E-05	1.90E-01
Total		1.58E-04	5.63E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.80E-07	2.26E-02
	Dermal	7.19E-06	9.48E-01
	Inhalation	1.83E-07	ND
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Surface Water	Dermal	1.71E-06	7.26E-02
Seeps	Dermal	ND	1.75E-02
Sediment	Dermal	1.06E-04	5.49E+00
Total		7.10E-04	9.26E+01
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.14E-06	1.97E-01
	Dermal	2.74E-06	2.16E+00
	Inhalation	1.45E-07	ND
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Surface Water	Dermal	1.16E-06	2.95E-01
Seeps	Dermal	ND	7.12E-02
Sediment	Dermal	7.19E-05	2.23E+01
Total		3.92E-04	1.46E+02

ND – Not Determined

TABLE R-7 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 3 (INDUSTRIAL WASTE LAGOONS)
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.94E-08	2.29E-04
	Dermal	2.85E-07	9.48E-01
	Inhalation	3.06E-08	ND
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Surface Water	Dermal	2.86E-07	1.48E-02
Seeps	Dermal	ND	3.55E-03
Sediment	Dermal	1.53E-05	1.90E-01
Total		1.54E-04	5.73E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.36E-07	2.26E-02
	Dermal	1.71E-06	9.48E-01
	Inhalation	1.83E-07	ND
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Surface Water	Dermal	1.71E-06	7.26E-02
Seeps	Dermal	ND	1.75E-02
Sediment	Dermal	9.20E-05	5.49E+00
Total		6.90E-04	9.26E+01
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.44E-07	1.97E-01
	Dermal	6.51E-07	2.16E+00
	Inhalation	1.45E-07	ND
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Surface Water	Dermal	1.16E-06	2.95E-01
Seeps	Dermal	ND	7.12E-02
Sediment	Dermal	6.22E-05	2.23E+01
Total		3.80E-04	1.46E+02

ND – Not Determined

TABLE R-8
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 24
(SCRAP WOOD PILE/FIRE TRAINING AREA)
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.58E-06	1.47E-01
	Dermal	7.52E-05	1.23E+00
	Inhalation	4.27E-07	4.08E-04
Total		8.22E-05	1.38E+00
Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.29E-05	1.57E-01
	Dermal	3.76E-04	2.11E+00
	Inhalation	2.13E-06	4.08E-04
Total		4.11E-04	2.27E+00
(TEF Methodology) Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.35E-06	1.47E-01
	Dermal	3.03E-05	1.23E+00
	Inhalation	4.27E-07	4.08E-04
Total		3.51E-05	1.38E+00
Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.18E-05	1.57E-01
	Dermal	1.52E-04	2.11E+00
	Inhalation	2.13E-06	4.08E-04
Total		1.76E-04	2.27E+00

ND – Not Determined

TABLE R-8 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 24
(SCRAP WOOD PILE/FIRE TRAINING AREA)
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology)			
Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.58E-06	1.47E-01
	Dermal	7.52E-05	1.23E+00
	Inhalation	4.27E-07	4.08E-04
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		1.29E-04	2.07E+01
Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.29E-05	1.57E-01
	Dermal	3.76E-04	2.11E+00
	Inhalation	2.13E-06	4.08E-04
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		5.77E-04	3.21E+01
(TEF Methodology)			
Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.35E-06	1.47E-01
	Dermal	3.03E-05	1.23E+00
	Inhalation	4.27E-07	4.08E-04
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		8.17E-05	2.07E+01
Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.18E-05	1.57E-01
	Dermal	1.52E-04	2.11E+00
	Inhalation	2.13E-06	4.08E-04
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		3.42E-04	3.21E+01

ND – Not Determined

TABLE R–8 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 24
(SCRAP WOOD PILE/FIRE TRAINING AREA)
LEXINGTON–BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.84E–05	4.38E–01
	Dermal	1.05E–04	1.73E+00
	Inhalation	1.79E–06	1.71E–03
Groundwater	Ingestion	1.30E–04	5.41E+01
	Dermal	2.01E–06	2.02E+00
	Inhalation	6.17E–06	1.38E–03
Vegetable	Ingestion	7.00E–07	4.00E–02
Total		2.64E–04	5.83E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.11E–04	4.38E–01
	Dermal	6.32E–04	2.95E+00
	Inhalation	1.08E–05	1.71E–03
Groundwater	Ingestion	5.59E–04	8.35E+01
	Dermal	8.61E–06	2.50E+00
	Inhalation	2.65E–05	2.14E–02
Vegetable	Ingestion	4.00E–06	2.20E–01
Total		1.35E–03	8.96E+01
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.61E–04	3.83E+00
	Dermal	2.41E–04	6.74E+00
	Inhalation	8.50E–06	8.13E–03
Groundwater	Ingestion	2.85E–04	1.18E+02
	Dermal	3.30E–06	3.31E+00
	Inhalation	2.70E–05	6.02E–03
Vegetable	Ingestion	3.00E–06	1.60E–01
Total		7.29E–04	1.32E+02

ND – Not Determined

TABLE R-8 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 24
(SCRAP WOOD PILE/FIRE TRAINING AREA)
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.22E-05	4.38E-01
	Dermal	4.24E-05	1.73E+00
	Inhalation	1.79E-06	1.71E-03
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	7.00E-07	4.00E-02
Total		1.95E-04	5.83E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.31E-05	4.38E-01
	Dermal	2.55E-04	2.95E+00
	Inhalation	1.08E-05	1.71E-03
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	4.00E-06	2.20E-01
Total		9.37E-04	8.96E+01
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.07E-04	3.83E+00
	Dermal	9.69E-05	6.74E+00
	Inhalation	8.50E-06	8.13E-03
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	3.00E-06	1.60E-01
Total		5.31E-04	1.32E+02

ND – Not Determined

TABLE R-9
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 135
LEXINGTON-BLUEGRASS ARMY DEPOT

Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	1.45E-04
	Dermal	ND	9.80E-03
	Inhalation	ND	ND
Total		ND	9.94E-03

Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	1.45E-04
	Dermal	ND	9.80E-03
	Inhalation	ND	ND
Total		ND	9.94E-03

Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	1.45E-04
	Dermal	ND	9.80E-03
	Inhalation	ND	ND
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		4.66E-05	1.93E+01

Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	1.45E-04
	Dermal	ND	9.80E-03
	Inhalation	ND	ND
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.66E-04	2.98E+01

ND - Not Determined

TABLE R-9 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 135
LEXINGTON-BLUEGRASS ARMY DEPOT

Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	4.05E-04
	Dermal	ND	1.37E-02
	Inhalation	ND	ND
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-07	1.38E-03
Total		1.33E-04	5.61E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	4.05E-04
	Dermal	ND	1.37E-02
	Inhalation	ND	ND
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Total		5.94E-04	8.60E+01

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	3.54E-03
	Dermal	ND	3.13E-02
	Inhalation	ND	ND
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Total		3.15E-04	1.21E+02

ND – Not Determined

TABLE R-10
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 147
LEXINGTON-BLUEGRASS ARMY DEPOT

Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	3.92E-04
	Dermal	ND	8.26E-03
	Inhalation	ND	ND
Total		ND	8.65E-03

Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	3.92E-04
	Dermal	ND	8.26E-03
	Inhalation	ND	ND
Total		ND	8.65E-03

Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	3.92E-04
	Dermal	ND	8.26E-03
	Inhalation	ND	ND
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		4.66E-05	1.93E+01

Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	3.92E-04
	Dermal	ND	8.26E-03
	Inhalation	ND	ND
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.66E-04	2.98E+01

ND - Not Determined

TABLE R-10 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 147
LEXINGTON-BLUEGRASS ARMY DEPOT

Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	1.10E-03
	Dermal	ND	1.16E-02
	Inhalation	ND	ND
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Total		1.38E-04	5.61E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	1.10E-03
	Dermal	ND	1.16E-02
	Inhalation	ND	ND
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Total		5.94E-04	8.60E+01
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	9.59E-03
	Dermal	ND	2.64E-02
	Inhalation	ND	ND
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Total		3.15E-04	1.21E+02

ND – Not Determined

TABLE R-11
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 3
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.62E-06	7.48E-02
	Dermal	1.13E-04	7.00E+00
	Inhalation	ND	7.69E-01
Total		1.19E-04	7.84E+00
Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.81E-05	7.48E-02
	Dermal	5.66E-04	1.08E+01
	Inhalation	ND	7.69E-01
Total		5.94E-04	1.16E+01
(TEF Methodology) Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.49E-06	7.48E-02
	Dermal	3.01E-05	7.00E+00
	Inhalation	ND	7.69E-01
Total		3.16E-05	7.84E+00
Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.47E-06	7.48E-02
	Dermal	1.50E-04	1.08E+01
	Inhalation	ND	7.69E-01
Total		1.57E-04	1.16E+01

ND - Not Determined

TABLE R-11 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 3
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.62E-06	7.48E-02
	Dermal	1.13E-04	7.00E+00
	Inhalation	ND	7.69E-01
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		1.65E-04	2.71E+01
Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.81E-05	7.48E-02
	Dermal	5.66E-04	1.08E+01
	Inhalation	ND	7.69E-01
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		7.60E-04	4.14E+01
(TEF Methodology) Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.49E-06	7.48E-02
	Dermal	3.01E-05	7.00E+00
	Inhalation	ND	7.69E-01
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		7.82E-05	2.71E+01
Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.47E-06	7.48E-02
	Dermal	1.50E-04	1.08E+01
	Inhalation	ND	7.69E-01
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		3.23E-04	4.14E+01

ND - Not Determined

TABLE R-11 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 3
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.57E-05	2.10E-01
	Dermal	1.59E-04	9.80E+00
	Inhalation	ND	3.23E+00
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Total		3.13E-04	6.94E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	9.44E-05	2.11E-01
	Dermal	9.51E-04	9.82E+00
	Inhalation	ND	3.23E+00
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Total		1.64E-03	9.93E+01
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.38E-04	1.84E+00
	Dermal	3.62E-04	2.24E+01
	Inhalation	ND	1.53E+01
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Total		8.15E-04	1.61E+02

ND – Not Determined

TABLE R-11 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 3
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.18E-06	2.10E-01
	Dermal	4.21E-05	9.80E+00
	Inhalation	ND	3.23E+00
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Total		1.84E-04	6.94E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.51E-05	2.11E-01
	Dermal	2.53E-04	9.82E+00
	Inhalation	ND	3.23E+00
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Total		8.72E-04	9.93E+01
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.66E-05	1.84E+00
	Dermal	9.62E-05	2.24E+01
	Inhalation	ND	1.53E+01
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Total		4.48E-04	1.61E+02

ND – Not Determined

TABLE R-12
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 10
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.86E-06	3.34E-02
	Dermal	6.27E-05	1.08E+01
	Inhalation	9.82E-08	3.56E-01
Total		6.57E-05	1.12E+01
Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.43E-05	3.70E-01
	Dermal	3.14E-04	1.08E+01
	Inhalation	4.91E-07	3.56E-01
Total		3.29E-04	1.15E+01
(TEF Methodology) Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.60E-06	3.34E-02
	Dermal	5.74E-05	1.08E+01
	Inhalation	9.82E-08	3.56E-01
Total		6.01E-05	1.12E+01
Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.30E-05	3.70E-01
	Dermal	2.87E-04	1.08E+01
	Inhalation	4.91E-07	3.56E-01
Total		3.00E-04	1.15E+01

ND - Not Determined

TABLE R-12 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 10
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.86E-06	3.34E-02
	Dermal	6.27E-05	1.08E+01
	Inhalation	9.82E-08	3.56E-01
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		1.12E-04	3.05E+01

Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.43E-05	3.70E-01
	Dermal	3.14E-04	1.08E+01
	Inhalation	4.91E-07	3.56E-01
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		4.95E-04	4.13E+01

(TEF Methodology) Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.60E-06	3.34E-02
	Dermal	5.74E-05	1.08E+01
	Inhalation	9.82E-08	3.56E-01
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		1.07E-04	3.05E+01

Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.30E-05	3.70E-01
	Dermal	2.87E-04	1.08E+01
	Inhalation	4.91E-07	3.56E-01
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		4.66E-04	4.13E+01

ND - Not Determined

TABLE R-12 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 10
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	8.01E-06	4.68E-01
	Dermal	8.78E-05	1.51E+01
	Inhalation	4.12E-07	1.49E+00
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Total		2.34E-04	7.32E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.81E-05	1.04E+00
	Dermal	5.27E-04	3.25E+01
	Inhalation	2.47E-06	1.49E+00
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Total		1.17E-03	1.21E+02
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.01E-05	9.07E+00
	Dermal	2.01E-04	7.42E+01
	Inhalation	1.96E-06	7.09E+00
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Total		5.88E-04	2.12E+02

ND – Not Determined

TABLE R-12 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 10
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.28E-06	4.68E-01
	Dermal	8.04E-05	1.51E+01
	Inhalation	4.12E-07	1.49E+00
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Total		2.26E-04	7.32E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.37E-05	1.04E+00
	Dermal	4.83E-04	3.25E+01
	Inhalation	2.47E-06	1.49E+00
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Total		1.12E-03	1.21E+02
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.37E-05	9.07E+00
	Dermal	1.84E-04	7.42E+01
	Inhalation	1.96E-06	7.09E+00
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Total		5.65E-04	2.12E+02

ND – Not Determined

TABLE R-13
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 19
LEXINGTON-BLUEGRASS ARMY DEPOT

Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.91E-06	8.32E-01
	Dermal	5.78E-05	5.20E+01
	Inhalation	6.34E-08	6.92E-01
Total		5.98E-05	5.35E+01

Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	9.56E-06	8.32E-01
	Dermal	2.89E-04	5.20E+01
	Inhalation	3.17E-07	6.92E-01
Total		2.99E-04	5.35E+01

Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.91E-06	8.32E-01
	Dermal	5.78E-05	5.20E+01
	Inhalation	6.34E-08	6.92E-01
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		1.06E-04	7.28E+01

Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	9.56E-06	8.32E-01
	Dermal	2.89E-04	5.20E+01
	Inhalation	3.17E-07	6.92E-01
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		4.65E-04	8.33E+01

TABLE R-13 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 19
LEXINGTON-BLUEGRASS army DEPOT

Future Residential Adult -- Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.35E-06	2.33E+00
	Dermal	8.10E-05	7.28E+01
	Inhalation	2.66E-07	2.91E+00
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Total		2.25E-04	1.34E+02

Future Residential Adult -- Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.21E-05	2.33E+00
	Dermal	4.86E-04	7.28E+01
	Inhalation	1.60E-06	2.91E+00
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Total		1.11E-03	1.64E+02

Future Residential Child -- Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.68E-05	2.04E+01
	Dermal	1.85E-04	1.66E+02
	Inhalation	1.26E-06	1.38E+01
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Total		5.48E-04	3.22E+02

TABLE R-14
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 63
LEXINGTON-BLUEGRASS army DEPOT

(BaP Methodology) Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.20E-05	1.25E-01
	Dermal	1.65E-04	1.22E+01
	Inhalation	1.73E-07	3.27E-04
Total		1.77E-04	1.23E+01

Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.01E-05	6.35E-01
	Dermal	8.27E-04	1.31E+01
	Inhalation	8.64E-07	1.64E-03
Total		8.88E-04	1.37E+01

(TEF Methodology) Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.14E-06	1.25E-01
	Dermal	4.69E-05	1.22E+01
	Inhalation	1.73E-07	3.27E-04
Total		5.32E-05	1.23E+01

Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.07E-05	6.35E-01
	Dermal	2.35E-04	1.31E+01
	Inhalation	8.64E-07	1.64E-03
Total		2.67E-04	1.37E+01

TABLE R-14 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 63
LEXINGTON-BLUEGRASS army DEPOT

(BaP Methodology) Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.20E-05	1.25E-01
	Dermal	1.65E-04	1.22E+01
	Inhalation	1.73E-07	3.27E-04
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		2.24E-04	3.16E+01

Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.01E-05	6.35E-01
	Dermal	8.27E-04	1.31E+01
	Inhalation	8.64E-07	1.64E-03
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.05E-03	4.35E+01

(TEF Methodology) Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.14E-06	1.25E-01
	Dermal	4.69E-05	1.22E+01
	Inhalation	1.73E-07	3.27E-04
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		9.98E-05	3.16E+01

Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.07E-05	6.35E-01
	Dermal	2.35E-04	1.31E+01
	Inhalation	8.64E-07	1.64E-03
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		4.33E-04	4.35E+01

TABLE R-14 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 63
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.36E-05	1.75E+00
	Dermal	2.31E-04	1.71E+01
	Inhalation	7.26E-07	6.87E-03
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	1.00E-06	2.50E-02
Total		4.05E-04	7.50E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.02E-04	1.78E+00
	Dermal	1.39E-03	1.83E+01
	Inhalation	4.36E-06	6.87E-03
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	6.00E-06	1.40E-01
Total		2.20E-03	1.06E+02
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.94E-04	1.55E+01
	Dermal	5.29E-04	4.19E+01
	Inhalation	3.44E-06	3.26E-02
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	4.00E-06	1.00E-01
Total		1.15E-03	1.79E+02

ND – Not Determined

TABLE R-14 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 63
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.72E-05	1.75E+00
	Dermal	6.57E-05	1.71E+01
	Inhalation	7.26E-07	6.87E-03
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	1.00E-06	2.50E-02
Total		2.23E-04	7.50E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.03E-04	1.78E+00
	Dermal	3.94E-04	1.83E+01
	Inhalation	4.36E-06	6.87E-03
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	6.00E-06	1.40E-01
Total		1.10E-03	1.06E+02

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.50E-04	1.55E+01
	Dermal	1.50E-04	4.19E+01
	Inhalation	3.44E-06	3.26E-02
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	4.00E-06	1.00E-01
Total		6.23E-04	1.79E+02

ND – Not Determined

TABLE R-15
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 64
LEXINGTON-BLUEGRASS ARMY DEPOT

Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.90E-09	1.57E-04
	Dermal	2.59E-08	2.13E-03
	Inhalation	1.70E-12	ND
Total		2.78E-08	2.29E-03

Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	9.51E-09	1.57E-04
	Dermal	1.29E-07	2.13E-03
	Inhalation	8.50E-12	ND
Total		1.39E-07	2.29E-03

Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.90E-09	1.57E-04
	Dermal	2.59E-08	2.13E-03
	Inhalation	1.70E-12	ND
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		4.66E-05	1.93E+01

Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	9.51E-09	1.57E-04
	Dermal	1.29E-07	2.13E-03
	Inhalation	8.50E-12	ND
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.66E-04	2.98E+01

ND - Not Determined

TABLE R-15 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 64
LEXINGTON-BLUEGRASS ARMY DEPOT

Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.32E-09	4.38E-04
	Dermal	3.63E-08	2.99E-03
	Inhalation	7.14E-12	ND
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Total		1.38E-04	5.61E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.19E-08	4.38E-04
	Dermal	2.18E-07	2.99E-03
	Inhalation	4.29E-11	ND
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Total		5.94E-04	8.60E+01

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.66E-08	3.84E-03
	Dermal	8.28E-08	6.82E-03
	Inhalation	3.39E-11	ND
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Total		3.15E-04	1.21E+02

ND – Not Determined

TABLE R-16
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 130
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	9.70E-07	1.55E-01
	Dermal	1.88E-05	6.67E+00
	Inhalation	2.34E-08	4.43E-04
Total		1.98E-05	6.83E+00

Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.85E-06	1.55E-01
	Dermal	9.41E-05	6.67E+00
	Inhalation	1.17E-07	4.43E-04
Total		9.91E-05	6.83E+00

(TEF Methodology) Existing Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.07E-07	1.55E-01
	Dermal	3.43E-06	6.67E+00
	Inhalation	2.34E-08	4.43E-04
Total		3.66E-06	6.83E+00

Existing Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.04E-06	1.55E-01
	Dermal	1.72E-05	6.67E+00
	Inhalation	1.17E-07	4.43E-04
Total		1.84E-05	6.83E+00

TABLE R-16 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 130
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	9.70E-07	1.55E-01
	Dermal	1.88E-05	6.67E+00
	Inhalation	2.34E-08	4.43E-04
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		6.64E-05	2.61E+01

Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.85E-06	1.55E-01
	Dermal	9.41E-05	6.67E+00
	Inhalation	1.17E-07	4.43E-04
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		2.65E-04	3.66E+01

(TEF Methodology) Future Adult Worker (short term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.07E-07	1.55E-01
	Dermal	3.43E-06	6.67E+00
	Inhalation	2.34E-08	4.43E-04
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		5.03E-05	2.61E+01

Future Adult Worker (long term)			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.04E-06	1.55E-01
	Dermal	1.72E-05	6.67E+00
	Inhalation	1.17E-07	4.43E-04
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.84E-04	3.66E+01

TABLE R-16 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 130
LEXINGTON-BLUEGRASS army DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.72E-06	4.34E-01
	Dermal	2.63E-05	9.33E+00
	Inhalation	9.84E-08	1.86E-03
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	1.20E-06	1.00E-02
Total		1.68E-04	6.59E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.63E-05	4.34E-01
	Dermal	1.58E-04	9.33E+00
	Inhalation	5.91E-07	1.86E-03
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	8.00E-06	2.60E-01
Total		7.77E-04	9.60E+01
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.38E-05	3.79E+00
	Dermal	6.02E-05	2.13E+01
	Inhalation	4.67E-07	8.82E-03
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	6.00E-06	1.90E-01
Total		4.06E-04	1.47E+02

TABLE R-16 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 130
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.80E-07	4.34E-01
	Dermal	4.81E-06	9.33E+00
	Inhalation	9.84E-08	1.86E-03
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	1.20E-06	1.00E-02
Total		1.45E-04	6.59E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.48E-06	4.34E-01
	Dermal	2.88E-05	9.33E+00
	Inhalation	5.91E-07	1.86E-03
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	8.00E-06	2.60E-01
Total		6.35E-04	9.60E+01
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.07E-06	3.79E+00
	Dermal	1.10E-05	2.13E+01
	Inhalation	4.67E-07	8.82E-03
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	6.00E-06	1.90E-01
Total		3.38E-04	1.47E+02

ND – Not Determined

TABLE R-17
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 141
LEXINGTON-BLUEGRASS ARMY DEPOT

Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	1.66E-03
	Dermal	ND	1.02E-01
	Inhalation	3.66E-09	ND
Total		3.66E-09	1.04E-01

Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	1.66E-03
	Dermal	ND	1.02E-01
	Inhalation	1.83E-08	ND
Total		1.83E-08	1.04E-01

Future Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	1.66E-03
	Dermal	ND	1.02E-01
	Inhalation	3.66E-09	ND
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		4.66E-05	1.94E+01

Future Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	1.66E-03
	Dermal	ND	1.02E-01
	Inhalation	1.83E-08	ND
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.66E-04	2.99E+01

ND – Not Determined

TABLE R-17 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 141
LEXINGTON-BLUEGRASS army DEPOT

Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	4.64E-03
	Dermal	ND	1.42E-01
	Inhalation	1.54E-08	ND
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Total		1.38E-04	5.63E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	4.64E-03
	Dermal	ND	1.42E-01
	Inhalation	9.22E-08	ND
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Total		5.94E-04	8.62E+01

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	4.06E-02
	Dermal	ND	3.25E-01
	Inhalation	7.29E-08	ND
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Total		3.15E-04	1.22E+02

ND – Not Determined

TABLE R-18
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 23 (BUILDING 4,5,135,139)
LEXINGTON-BLUEGRASS army DEPOT

(BaP Methodology) Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	9.34E-06	7.89E-02
	Dermal	1.88E-04	1.20E+00
	Inhalation	5.01E-07	3.25E-03
Total		1.98E-04	1.28E+00
Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.67E-05	2.41E-01
	Dermal	9.39E-04	1.54E+01
	Inhalation	2.50E-06	3.25E-03
Total		9.88E-04	1.56E+01
(TEF Methodology) Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.84E-07	7.89E-02
	Dermal	1.54E-05	1.20E+00
	Inhalation	5.01E-07	3.25E-03
Total		1.67E-05	1.28E+00
Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.92E-06	2.41E-01
	Dermal	7.69E-05	1.54E+01
	Inhalation	2.50E-06	3.25E-03
Total		8.33E-05	1.56E+01

TABLE R-18 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 23 (BUILDING 4,5,135,139)
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	9.34E-06	7.89E-02
	Dermal	1.88E-04	1.20E+00
	Inhalation	5.01E-07	3.25E-03
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		2.44E-04	2.06E+01
Future Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.67E-05	2.41E-01
	Dermal	9.39E-04	1.54E+01
	Inhalation	2.50E-06	3.25E-03
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.15E-03	4.54E+01
(TEF Methodology) Future Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.84E-07	7.89E-02
	Dermal	1.54E-05	1.20E+00
	Inhalation	5.01E-07	3.25E-03
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		6.33E-05	2.06E+01
Future Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.92E-06	2.41E-01
	Dermal	7.69E-05	1.54E+01
	Inhalation	2.50E-06	3.25E-03
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		2.49E-04	4.54E+01

TABLE R-18 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 23 (BUILDING 4,5,135,139)
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.62E-05	2.21E-01
	Dermal	2.63E-04	1.67E+00
	Inhalation	2.10E-06	1.36E-02
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	6.00E-07	1.00E-02
Total		4.30E-04	5.80E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.57E-04	6.75E-01
	Dermal	1.58E-03	2.16E+01
	Inhalation	1.26E-05	1.36E-02
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	4.00E-06	7.00E-02
Total		2.35E-03	1.08E+02
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.29E-04	5.91E+00
	Dermal	6.01E-04	4.92E+01
	Inhalation	9.98E-06	6.47E-02
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	3.00E-06	5.00E-02
Total		1.16E-03	1.77E+02

TABLE R-18 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 23 (BUILDING 4,5,135,139)
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.20E-06	2.21E-01
	Dermal	2.15E-05	1.67E+00
	Inhalation	2.10E-06	1.36E-02
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	6.00E-07	1.00E-02
Total		1.65E-04	5.80E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.32E-05	6.75E-01
	Dermal	1.29E-04	2.16E+01
	Inhalation	1.26E-05	1.36E-02
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	4.00E-06	7.00E-02
Total		7.53E-04	1.08E+02

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.92E-05	5.91E+00
	Dermal	4.92E-05	4.92E+01
	Inhalation	9.98E-06	6.47E-02
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	3.00E-06	5.00E-02
Total		3.97E-04	1.77E+02

TABLE R-19
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 18,19
LEXINGTON-BLUEGRASS ARMY DEPOT

Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	2.78E-02
	Dermal	ND	3.77E-01
	Inhalation	ND	3.96E-05
Total		ND	4.05E-01

Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	2.78E-02
	Dermal	ND	3.77E-01
	Inhalation	ND	3.96E-05
Total		ND	4.05E-01

Future Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	8.41E-04
	Dermal	ND	4.00E-02
	Inhalation	ND	8.40E-07
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		4.66E-05	1.93E+01

Future Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	8.41E-04
	Dermal	ND	4.00E-02
	Inhalation	ND	8.40E-07
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.66E-04	2.98E+01

ND – Not Determined

TABLE R-19 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 18,19
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology)
Future Residential Adult – Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	2.36E-03
	Dermal	ND	5.60E-02
	Inhalation	ND	3.53E-06
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Sediment	Dermal	4.45E-06	3.69E-02
Total		1.43E-04	5.62E+01

Future Residential Adult – Long Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	2.36E-03
	Dermal	ND	5.60E-02
	Inhalation	ND	3.53E-06
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Sediment	Dermal	2.67E-05	5.28E-02
Total		6.21E-04	8.61E+01

Future Residential Child – Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	2.06E-02
	Dermal	ND	1.28E-01
	Inhalation	ND	1.67E-05
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-06	6.02E-03
Sediment	Dermal	1.81E-05	2.14E-01
Total		3.09E-04	1.22E+02

ND – Not Determined

TABLE R-19 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 18,19
LEXINGTON-BLUEGRASS army DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	2.36E-03
	Dermal	ND	5.60E-02
	Inhalation	ND	3.53E-06
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Sediment	Dermal	2.32E-07	3.69E-02
Total		1.38E-04	5.62E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	2.36E-03
	Dermal	ND	5.60E-02
	Inhalation	ND	3.53E-06
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Sediment	Dermal	1.39E-06	5.28E-02
Total		5.96E-04	8.61E+01
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	2.06E-02
	Dermal	ND	1.28E-01
	Inhalation	ND	1.67E-05
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-06	6.02E-03
Sediment	Dermal	9.44E-07	2.14E-01
Total		2.92E-04	1.22E+02

ND – Not Determined

TABLE R-20
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 16,17,30
LEXINGTON-BLUEGRASS ARMY DEPOT

Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.82E-07	5.20E-02
	Dermal	2.62E-06	2.37E+00
	Inhalation	2.95E-09	1.44E-04
Total		2.80E-06	2.42E+00

Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	9.08E-07	6.03E-02
	Dermal	1.31E-05	2.98E+00
	Inhalation	1.48E-08	1.44E-04
Total		1.40E-05	3.04E+00

Future Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.82E-07	5.20E-02
	Dermal	2.62E-06	2.37E+00
	Inhalation	2.95E-09	1.44E-04
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		4.94E-05	2.17E+01

Future Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	9.08E-07	6.03E-02
	Dermal	1.31E-05	2.98E+00
	Inhalation	1.48E-08	1.44E-04
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.80E-04	3.28E+01

TABLE R-20 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 16,17,30
LEXINGTON-BLUEGRASS ARMY DEPOT

Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.09E-07	1.46E-01
	Dermal	3.67E-06	3.31E+00
	Inhalation	1.24E-08	6.04E-04
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	1.00E-06	ND
Total		1.43E-04	5.96E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.05E-06	1.69E-01
	Dermal	2.20E-05	4.18E+00
	Inhalation	7.44E-08	6.04E-04
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	8.00E-06	2.00E-01
Total		6.27E-04	9.06E+01

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.45E-06	1.48E+00
	Dermal	8.38E-06	9.54E+00
	Inhalation	5.89E-08	2.87E-03
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	6.00E-06	1.50E-01
Total		3.34E-04	1.32E+02

ND – Not Determined

TABLE R-21
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 9
LEXINGTON-BLUEGRASS army DEPOT

(BaP Methodology) Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.45E-04	1.56E-01
	Dermal	2.90E-03	1.99E+00
	Inhalation	1.76E-09	6.08E-04
Total		3.05E-03	2.15E+00
Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.25E-04	2.58E-01
	Dermal	1.45E-02	3.90E+00
	Inhalation	8.79E-09	6.08E-04
Total		1.52E-02	4.16E+00
(TEF Methodology) Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.24E-05	1.56E-01
	Dermal	6.30E-04	1.99E+00
	Inhalation	1.76E-09	6.08E-04
Total		6.62E-04	2.15E+00
Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.62E-04	2.58E-01
	Dermal	3.15E-03	3.90E+00
	Inhalation	8.79E-09	6.08E-04
Total		3.31E-03	4.16E+00

TABLE R-21 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 9
LEXINGTON-BLUEGRASS ARMY DEPOT

Matrix	Route	Risk	Hazard
(BaP Methodology) Future Adult Worker – Short Term			
Soil	Ingestion	1.45E-04	1.56E-01
	Dermal	2.90E-03	1.99E+00
	Inhalation	1.76E-09	6.08E-04
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		3.09E-03	2.14E+01
Future Adult Worker – Long Term			
Soil	Ingestion	7.25E-04	2.58E-01
	Dermal	1.45E-02	3.90E+00
	Inhalation	8.79E-09	6.08E-04
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.54E-02	3.40E+01
(TEF Methodology) Future Adult Worker – Short Term			
Soil	Ingestion	3.24E-05	1.56E-01
	Dermal	6.30E-04	1.99E+00
	Inhalation	1.76E-09	6.08E-04
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		7.09E-04	2.14E+01
Future Adult Worker – Long Term			
Soil	Ingestion	1.62E-04	2.58E-01
	Dermal	3.15E-03	3.90E+00
	Inhalation	8.79E-09	6.08E-04
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		3.48E-03	3.40E+01

TABLE R-21 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 9
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology)
Future Residential Adult – Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	4.06E-04	4.37E-01
	Dermal	4.06E-03	2.79E+00
	Inhalation	7.39E-09	2.55E-03
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	3.00E-05	2.00E-02
Beef	Ingestion	1.00E-05	1.00E-02
Total		4.64E-03	5.94E+01

Future Residential Adult – Long Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	2.44E-03	7.24E-01
	Dermal	2.44E-02	5.47E+00
	Inhalation	4.43E-08	2.55E-03
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	2.00E-04	8.30E-01
Beef	Ingestion	7.00E-05	3.10E-01
Total		2.77E-02	9.34E+01

Future Residential Child – Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	3.55E-03	6.33E+00
	Dermal	9.28E-03	1.25E+01
	Inhalation	3.50E-08	1.21E-02
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	1.00E-04	3.60E+00
Beef	Ingestion	5.00E-05	1.05E+00
Total		1.33E-02	1.45E+02

TABLE R-21 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 9
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	9.06E-05	4.37E-01
	Dermal	8.81E-04	2.79E+00
	Inhalation	7.39E-09	2.55E-03
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-07	1.38E-03
Vegetable	Ingestion	3.00E-05	2.00E-02
Beef	Ingestion	1.00E-05	1.00E-02
Total		1.14E-03	5.94E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.44E-04	7.24E-01
	Dermal	5.29E-03	5.47E+00
	Inhalation	4.43E-08	2.55E-03
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	2.00E-04	8.30E-01
Beef	Ingestion	7.00E-05	3.10E-01
Total		6.70E-03	9.34E+01

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.93E-04	6.33E+00
	Dermal	2.01E-03	1.25E+01
	Inhalation	3.50E-08	1.21E-02
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	1.00E-04	3.60E+00
Beef	Ingestion	5.00E-05	1.05E+00
Total		3.27E-03	1.45E+02

TABLE R-22
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 42
LEXINGTON-BLUEGRASS ARMY DEPOT

Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.11E-06	2.00E-01
	Dermal	6.47E-05	1.68E+01
	Inhalation	6.91E-08	9.79E-01
Total		6.69E-05	1.80E+01

Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.05E-05	5.80E-01
	Dermal	3.24E-04	4.00E+01
	Inhalation	3.45E-07	9.79E-01
Total		3.35E-04	4.16E+01

Future Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.11E-06	2.00E-01
	Dermal	6.47E-05	1.68E+01
	Inhalation	6.91E-08	9.79E-01
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		1.13E-04	3.73E+01

Future Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.05E-05	5.80E-01
	Dermal	3.24E-04	4.00E+01
	Inhalation	3.45E-07	9.79E-01
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		5.01E-04	7.14E+01

TABLE R-22 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 42
LEXINGTON-BLUEGRASS ARMY DEPOT

Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.91E-06	5.63E-01
	Dermal	9.06E-05	2.35E+01
	Inhalation	2.90E-07	4.11E+00
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Total		2.35E-04	8.43E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.54E-05	1.62E+00
	Dermal	5.44E-04	5.60E+01
	Inhalation	1.74E-06	4.11E+00
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Total		1.18E-03	1.48E+02

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.17E-05	1.42E+01
	Dermal	2.07E-04	1.28E+02
	Inhalation	1.38E-06	1.95E+01
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Total		5.75E-04	2.83E+02

TABLE R-23
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 20
LEXINGTON-BLUEGRASS ARMY DEPOT

Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.16E-07	1.54E-01
	Dermal	9.75E-06	6.90E+00
	Inhalation	2.66E-09	6.67E-01
Total		1.05E-05	7.72E+00

Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.58E-06	1.59E-01
	Dermal	4.88E-05	7.18E+00
	Inhalation	1.33E-08	6.67E-01
Total		5.24E-05	8.01E+00

Future Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.16E-07	1.40E-01
	Dermal	9.75E-06	6.65E+00
	Inhalation	2.17E-09	6.58E-01
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		5.71E-05	2.67E+01

Future Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.58E-06	1.44E-01
	Dermal	4.88E-05	6.83E+00
	Inhalation	1.09E-08	6.58E-01
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		2.18E-04	3.74E+01

TABLE R-23 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 20
LEXINGTON-BLUEGRASS ARMY DEPOT

Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.01E-06	3.92E-01
	Dermal	1.37E-05	9.30E+00
	Inhalation	9.13E-09	2.76E+00
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	2.90E-06	7.00E-02
Total		1.57E-04	6.86E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.20E-05	4.02E-01
	Dermal	8.19E-05	9.56E+00
	Inhalation	5.48E-08	2.76E+00
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	1.50E-05	7.00E-02
Total		7.03E-04	9.88E+01

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.75E-05	3.52E+00
	Dermal	3.12E-05	2.18E+01
	Inhalation	4.33E-08	1.31E+01
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	1.40E-05	3.00E-01
Total		3.78E-04	1.60E+02

TABLE R-24
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 25
LEXINGTON-BLUEGRASS ARMY DEPOT

Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.23E-09	2.46E-04
	Dermal	3.63E-08	4.04E-03
	Inhalation	1.94E-12	ND
Total		3.85E-08	4.29E-03

Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.11E-08	2.46E-04
	Dermal	1.81E-07	4.04E-03
	Inhalation	9.72E-12	ND
Total		1.92E-07	4.29E-03

Future Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.23E-09	2.46E-04
	Dermal	3.63E-08	4.04E-03
	Inhalation	1.94E-12	ND
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		4.66E-05	1.93E+01

Future Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.11E-08	2.46E-04
	Dermal	1.81E-07	4.04E-03
	Inhalation	9.72E-12	ND
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.66E-04	2.98E+01

ND – Not Determined

TABLE R-24 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 25
LEXINGTON-BLUEGRASS ARMY DEPOT

Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.24E-09	6.88E-04
	Dermal	5.08E-08	5.66E-03
	Inhalation	8.17E-12	ND
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Total		1.30E-04	5.41E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.74E-08	6.88E-04
	Dermal	3.05E-07	5.66E-03
	Inhalation	4.90E-11	ND
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Total		5.94E-04	8.60E+01

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.46E-08	6.02E-03
	Dermal	1.16E-07	1.29E-02
	Inhalation	3.87E-11	ND
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Total		3.15E-04	1.21E+02

ND – Not Determined

TABLE R-25
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 11
LEXINGTON-BLUEGRASS army DEPOT

Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.80E-08	6.13E-04
	Dermal	3.89E-07	8.44E-03
	Inhalation	3.03E-11	ND
Total		4.17E-07	9.05E-03

Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.40E-07	6.13E-04
	Dermal	1.94E-06	8.44E-03
	Inhalation	1.52E-10	ND
Total		2.08E-06	9.05E-03

Future Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.80E-08	6.13E-04
	Dermal	3.89E-07	8.44E-03
	Inhalation	3.03E-11	ND
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		4.70E-05	1.93E+01

Future Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.40E-07	6.13E-04
	Dermal	1.94E-06	8.44E-03
	Inhalation	1.52E-10	ND
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.68E-04	2.98E+01

ND – Not Determined

TABLE R-25 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 11
LEXINGTON-BLUEGRASS ARMY DEPOT

Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.83E-08	1.72E-03
	Dermal	5.44E-07	1.18E-02
	Inhalation	1.27E-10	ND
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Total		1.31E-04	5.41E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.70E-07	1.72E-03
	Dermal	3.26E-06	1.18E-02
	Inhalation	7.64E-10	ND
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Total		5.63E-04	8.35E+01

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.85E-07	1.50E-02
	Dermal	1.24E-06	2.70E-02
	Inhalation	6.04E-10	ND
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Total		2.87E-04	1.18E+02

ND – Not Determined

TABLE R-26
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE COAL PILE RUNOFF \ HEATING PLANT AREA
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology)
Existing Adult Worker - Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	3.34E-05	9.12E-01
	Dermal	3.09E-04	1.17E+01
	Inhalation	1.09E-06	2.61E-04
Total		3.43E-04	1.26E+01

Existing Adult Worker - Long Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	1.67E-04	1.77E+00
	Dermal	1.55E-03	6.46E+01
	Inhalation	5.44E-06	2.61E-04
Total		1.72E-03	6.64E+01

Future Adult Worker - Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	3.34E-05	9.13E-01
	Dermal	3.09E-04	1.17E+01
	Inhalation	1.09E-06	2.62E-04
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		3.90E-04	3.19E+01

Future Adult Worker - Long Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	1.67E-04	1.78E+00
	Dermal	1.55E-03	6.47E+01
	Inhalation	5.43E-06	2.62E-04
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.89E-03	9.63E+01

TABLE R-26 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE COAL PILE RUNOFF \ HEATING PLANT AREA
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology)
Existing Adult Worker -- Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	3.01E-05	9.12E-01
	Dermal	2.42E-04	1.17E+01
	Inhalation	1.09E-06	2.61E-04
Total		2.73E-04	1.26E+01

Existing Adult Worker -- Long Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	1.50E-04	1.77E+00
	Dermal	1.21E-03	6.46E+01
	Inhalation	5.44E-06	2.61E-04
Total		1.37E-03	6.64E+01

Future Adult Worker -- Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	3.01E-05	9.13E-01
	Dermal	2.42E-04	1.17E+01
	Inhalation	1.09E-06	2.62E-04
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		3.20E-04	3.19E+01

Future Adult Worker -- Long Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	1.50E-04	1.78E+00
	Dermal	1.21E-03	6.47E+01
	Inhalation	5.43E-06	2.62E-04
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.53E-03	9.63E+01

TABLE R-26 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE COAL PILE RUNOFF \ HEATING PLANT AREA
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology)
Future Residential Adult – Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	9.36E-05	2.56E+00
	Dermal	4.33E-04	1.64E+01
	Inhalation	4.57E-06	1.10E-03
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	4.00E-07	7.00E-03
Total		6.70E-04	7.51E+01

Future Residential Adult – Long Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	5.62E-04	4.97E+00
	Dermal	2.60E-03	9.05E+01
	Inhalation	2.74E-05	1.10E-03
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	3.00E-06	5.00E-02
Total		3.79E-03	1.82E+02

Future Residential Child – Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	8.19E-04	4.35E+01
	Dermal	9.88E-04	2.07E+02
	Inhalation	2.17E-05	5.22E-03
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	2.00E-06	3.00E-02
Total		2.15E-03	3.72E+02

TABLE R-26 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE COAL PILE RUNOFF \ HEATING PLANT AREA
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology)
Future Residential Adult – Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	8.42E-05	2.56E+00
	Dermal	3.38E-04	1.64E+01
	Inhalation	4.57E-06	1.10E-03
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	4.00E-07	7.00E-03
Total		5.65E-04	7.51E+01

Future Residential Adult – Long Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	5.05E-04	4.97E+00
	Dermal	2.03E-03	9.05E+01
	Inhalation	2.74E-05	1.10E-03
Groundwater	Ingestion	5.59E-02	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	3.00E-06	5.00E-02
Total		5.85E-02	1.82E+02

Future Residential Child – Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	7.37E-04	4.35E+01
	Dermal	7.73E-04	2.07E+02
	Inhalation	2.17E-05	5.22E-03
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	2.00E-06	3.00E-02
Total		1.85E-03	3.72E+02

TABLE R-27
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 10
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.24E-06	9.37E-01
	Dermal	3.45E-05	2.64E+01
	Inhalation	2.84E-06	3.69E-03
Total		4.06E-05	2.73E+01
Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.62E-05	7.35E+00
	Dermal	1.73E-04	5.89E+02
	Inhalation	1.42E-05	3.69E-03
Total		2.03E-04	5.96E+02
(TEF Methodology) Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.47E-06	9.37E-01
	Dermal	1.90E-05	2.64E+01
	Inhalation	2.84E-06	3.69E-03
Total		2.43E-05	2.73E+01
Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.23E-05	7.35E+00
	Dermal	9.50E-05	5.89E+02
	Inhalation	1.42E-05	3.69E-03
Total		1.22E-04	5.96E+02

TABLE R-27 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 10
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology)
Future Adult Worker – Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	3.24E-06	9.37E-01
	Dermal	3.45E-05	2.64E+01
	Inhalation	2.84E-06	3.69E-03
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		8.72E-05	4.66E+01

Future Adult Worker – Long Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	1.62E-05	7.35E+00
	Dermal	1.73E-04	5.89E+02
	Inhalation	1.42E-05	3.69E-03
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		3.69E-04	6.26E+02

(TEF Methodology)
Future Adult Worker – Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	2.47E-06	9.37E-01
	Dermal	1.90E-05	2.64E+01
	Inhalation	2.84E-06	3.69E-03
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		7.09E-05	4.66E+01

Future Adult Worker – Long Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	1.23E-05	7.35E+00
	Dermal	9.50E-05	5.89E+02
	Inhalation	1.42E-05	3.69E-03
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		2.87E-04	6.26E+02

TABLE R-27 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 10
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	9.07E-06	2.62E+00
	Dermal	4.84E-05	3.69E+01
	Inhalation	1.19E-05	1.55E-02
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	3.00E-06	ND
Beef	Ingestion	6.00E-06	ND
Total		2.17E-04	9.57E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.44E-05	2.06E+01
	Dermal	2.90E-04	8.25E+02
	Inhalation	7.15E-05	1.55E-02
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	2.00E-05	4.00E-01
Beef	Ingestion	4.00E-05	2.00E-01
Total		1.07E-03	9.32E+02
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.93E-05	1.80E+02
	Dermal	1.10E-04	1.88E+03
	Inhalation	5.65E-05	7.35E-02
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	1.00E-05	3.00E-01
Beef	Ingestion	3.00E-05	7.00E-01
Total		6.01E-04	2.18E+03

ND – Not Determined

TABLE R-27 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 10
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.91E-06	2.62E+00
	Dermal	2.66E-05	3.69E+01
	Inhalation	1.19E-05	1.55E-02
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	3.00E-06	ND
Beef	Ingestion	6.00E-06	ND
Total		1.93E-04	9.57E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.14E-05	2.06E+01
	Dermal	1.60E-04	8.25E+02
	Inhalation	7.15E-05	1.55E-02
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	2.00E-05	4.00E-01
Beef	Ingestion	4.00E-05	2.00E-01
Total		9.27E-04	9.32E+02

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.04E-05	1.80E+02
	Dermal	6.07E-05	1.88E+03
	Inhalation	5.65E-05	7.35E-02
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	1.00E-05	3.00E-01
Beef	Ingestion	3.00E-05	7.00E-01
Total		5.33E-04	2.18E+03

ND – Not Determined

TABLE R-28
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 303
LEXINGTON-BLUEGRASS ARMY DEPOT

Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.29E-06	7.16E-01
	Dermal	2.98E-05	4.94E+00
	Inhalation	1.28E-08	ND
Groundwater	Ingestion	7.26E-05	1.61E+01
	Dermal	6.80E-07	4.92E-01
	Inhalation	1.85E-07	2.84E-04
Vegetable	Ingestion	2.00E-05	3.02E+00
Beef	Ingestion	1.00E-06	3.40E-01
Total		1.29E-04	2.56E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.57E-05	7.16E-01
	Dermal	1.79E-04	4.95E+00
	Inhalation	7.69E-08	ND
Groundwater	Ingestion	3.11E-04	6.19E+01
	Dermal	2.91E-06	1.21E+00
	Inhalation	7.93E-07	3.05E-02
Vegetable	Ingestion	1.20E-04	3.10E+00
Beef	Ingestion	1.00E-05	3.40E-01
Total		6.49E-04	7.22E+01

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.75E-05	6.26E+00
	Dermal	6.80E-05	1.13E+01
	Inhalation	6.08E-08	ND
Groundwater	Ingestion	1.59E-04	3.51E+01
	Dermal	1.12E-06	8.08E-01
	Inhalation	8.09E-07	1.24E-03
Vegetable	Ingestion	9.30E-05	1.32E+01
Beef	Ingestion	4.00E-06	8.00E-01
Total		3.63E-04	6.75E+01

ND – Not Determined

TABLE R-29
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE OPEN STORAGE AND SHELTER AREA
LEXINGTON-BLUEGRASS army DEPOT

(BaP Methodology) Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	8.16E-05	1.57E-01
	Dermal	1.62E-03	3.20E+00
	Inhalation	4.52E-07	2.16E-03
Total		1.70E-03	3.36E+00
Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.08E-04	3.99E-01
	Dermal	8.09E-03	1.81E+01
	Inhalation	2.26E-06	2.16E-03
Total		8.50E-03	1.85E+01
(TEF Methodology) Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.18E-05	1.57E-01
	Dermal	2.12E-04	3.20E+00
	Inhalation	4.52E-07	2.16E-03
Total		2.24E-04	3.36E+00
Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.90E-05	3.99E-01
	Dermal	1.06E-03	1.81E+01
	Inhalation	2.26E-06	2.16E-03
Total		1.12E-03	1.85E+01

TABLE R-29 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE OPEN STORAGE AND SHELTER AREA
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology)			
Future Adult Worker - Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	8.16E-05	1.57E-01
	Dermal	1.62E-03	3.20E+00
	Inhalation	4.52E-07	2.16E-03
Groundwater	Ingesion	4.66E-05	1.93E+01
Total		1.75E-03	2.27E+01
Future Adult Worker - Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.08E-04	3.99E-01
	Dermal	8.09E-03	1.81E+01
	Inhalation	2.26E-06	2.16E-03
Groundwater	Ingesion	1.66E-04	2.98E+01
Total		8.67E-03	4.83E+01
(TEF Methodology)			
Future Adult Worker - Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.18E-05	1.57E-01
	Dermal	2.12E-04	3.20E+00
	Inhalation	4.52E-07	2.16E-03
Groundwater	Ingesion	4.66E-05	1.93E+01
Total		2.71E-04	2.27E+01
Future Adult Worker - Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.90E-05	3.99E-01
	Dermal	1.06E-03	1.81E+01
	Inhalation	2.26E-06	2.16E-03
Groundwater	Ingesion	1.66E-04	2.98E+01
Total		1.29E-03	4.83E+01

TABLE R-29 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE OPEN STORAGE AND SHELTER AREA
LEXINGTON-BLUEGRASS army DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.28E-04	4.38E-01
	Dermal	2.26E-03	4.47E+00
	Inhalation	1.90E-06	9.05E-03
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	1.00E-06	2.00E-02
Total		2.63E-03	6.11E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.37E-03	1.12E+00
	Dermal	1.36E-02	2.53E+01
	Inhalation	1.14E-05	9.05E-03
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	6.00E-06	2.00E-02
Total		1.56E-02	1.12E+02
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.00E-03	9.77E+00
	Dermal	5.17E-03	5.79E+01
	Inhalation	9.00E-06	4.30E-02
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	5.00E-06	8.00E-02
Total		7.50E-03	1.89E+02

TABLE R-29 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE OPEN STORAGE AND SHELTER AREA
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.30E-05	4.38E-01
	Dermal	2.97E-04	4.47E+00
	Inhalation	1.90E-06	9.05E-03
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	1.00E-06	2.00E-02
Total		4.71E-04	6.11E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.98E-04	1.12E+00
	Dermal	1.78E-03	2.53E+01
	Inhalation	1.14E-05	9.05E-03
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	6.00E-06	2.00E-02
Total		2.59E-03	1.12E+02

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.89E-04	9.77E+00
	Dermal	6.78E-04	5.79E+01
	Inhalation	9.00E-06	4.30E-02
Groundwater	Ingestion	2.85E-03	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	5.00E-06	8.00E-02
Total		3.86E-03	1.89E+02

TABLE R-30
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 12
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.85E-07	6.07E-01
	Dermal	1.64E-05	9.39E+00
	Inhalation	5.05E-07	8.87E-03
Total		1.77E-05	1.00E+01
Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.92E-06	1.03E+00
	Dermal	8.19E-05	4.66E+01
	Inhalation	2.53E-06	8.87E-03
Total		8.83E-05	4.76E+01
(TEF Methodology) Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	9.61E-08	6.07E-01
	Dermal	2.50E-06	9.39E+00
	Inhalation	5.05E-07	8.87E-03
Total		3.10E-06	1.00E+01
Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.81E-07	1.03E+00
	Dermal	1.25E-05	4.66E+01
	Inhalation	2.53E-06	8.87E-03
Total		1.55E-05	4.76E+01

TABLE R-30
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 12
LEXINGTON-BLUEGRASS army DEPOT

(BaP Methodology) Future Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.85E-07	6.07E-01
	Dermal	1.64E-05	9.39E+00
	Inhalation	5.05E-07	8.87E-03
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		6.43E-05	2.93E+01
Future Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.92E-06	1.03E+00
	Dermal	8.19E-05	4.66E+01
	Inhalation	2.53E-06	8.87E-03
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		2.54E-04	7.74E+01
(TEF Methodology) Future Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	9.61E-08	6.07E-01
	Dermal	2.50E-06	9.39E+00
	Inhalation	5.05E-07	8.87E-03
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		4.97E-05	2.93E+01
Future Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.81E-07	1.03E+00
	Dermal	1.25E-05	4.66E+01
	Inhalation	2.53E-06	8.87E-03
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.82E-04	7.74E+01

TABLE R-30 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 12
LEXINGTON-BLUEGRASS army DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.20E-06	1.70E+00
	Dermal	2.29E-05	1.31E+01
	Inhalation	2.12E-06	3.73E-02
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	1.00E-06	5.00E-02
Total		1.66E-04	7.10E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.32E-05	2.89E+00
	Dermal	1.38E-04	6.52E+01
	Inhalation	1.27E-05	3.73E-02
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	6.00E-06	5.00E-02
Total		7.64E-04	1.54E+02
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.92E-05	2.53E+01
	Dermal	5.24E-05	1.49E+02
	Inhalation	1.01E-05	4.04E-02
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	4.00E-06	2.10E-01
Total		4.01E-04	2.96E+02

TABLE R-30 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 12
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.69E-07	1.70E+00
	Dermal	3.50E-06	1.31E+01
	Inhalation	2.12E-06	3.73E-02
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	1.00E-06	5.00E-02
Total		1.45E-04	7.10E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.61E-06	2.89E+00
	Dermal	2.10E-05	6.52E+01
	Inhalation	1.27E-05	3.73E-02
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	6.00E-06	5.00E-02
Total		6.35E-04	1.54E+02

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.35E-06	2.53E+01
	Dermal	8.00E-06	1.49E+02
	Inhalation	1.01E-05	4.04E-02
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	4.00E-06	2.10E-01
Total		3.40E-04	2.96E+02

TABLE R-31
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR BUILDING 223
LEXINGTON-BLUEGRASS army DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Sediment	Dermal	5.57E-08	9.56E-04
Total		1.38E-04	5.61E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Sediment	Dermal	3.34E-07	9.56E-04
Total		5.94E-04	8.60E+01

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-06	6.02E-03
Sediment	Dermal	2.26E-07	3.88E-03
Total		2.91E-04	1.21E+02

TABLE R-32
SUMMARY OF RISK AND HAZARD CALCULATIONS
FACILITY WIDE
LEXINGTON-BLUEGRASS army DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Sediment	Dermal	5.24E-06	7.17E-01
Surface Water	Dermal	2.07E-08	2.32E-03
Total		1.43E-04	5.68E+01
Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Sediment	Dermal	3.15E-05	1.37E+00
Surface Water	Dermal	1.24E-07	2.32E-03
Total		6.26E-04	8.74E+01
Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Sediment	Dermal	2.13E-05	5.58E+00
Surface Water	Dermal	8.41E-08	9.43E-03
Total		3.37E-04	1.27E+02

TABLE R-32 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FACILITY WIDE
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult -- Short Term			
Matrix	Route	Risk	Hazard
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Sediment	Dermal	4.44E-06	7.17E-01
Surface Water	Dermal	2.07E-08	2.32E-03
Total		1.43E-04	5.68E+01
Future Residential Adult -- Long Term			
Matrix	Route	Risk	Hazard
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Sediment	Dermal	2.66E-05	1.37E+00
Surface Water	Dermal	1.24E-07	2.32E-03
Total		6.21E-04	8.74E+01
Future Residential Child -- Short Term			
Matrix	Route	Risk	Hazard
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Sediment	Dermal	1.80E-05	5.58E+00
Surface Water	Dermal	8.41E-08	9.43E-03
Total		3.33E-04	1.27E+02

TABLE R-33
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 22
LEXINGTON-BLUEGRASS army DEPOT

(BaP Methodology) Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.45E-06	3.40E-04
	Dermal	4.94E-05	1.14E-02
	Inhalation	7.29E-10	7.28E-07
Total		5.19E-05	1.17E-02

Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.22E-05	1.26E-02
	Dermal	2.47E-04	3.43E-01
	Inhalation	3.65E-09	7.28E-07
Total		2.59E-04	3.56E-01

(TEF Methodology) Existing Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.37E-07	3.40E-04
	Dermal	2.76E-06	1.14E-02
	Inhalation	7.29E-10	7.28E-07
Total		2.90E-06	1.17E-02

Existing Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.84E-07	1.26E-02
	Dermal	1.38E-05	3.43E-01
	Inhalation	3.65E-09	7.28E-07
Total		1.45E-05	3.56E-01

TABLE R-33 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 22
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.45E-06	4.60E-04
	Dermal	4.94E-05	1.23E-02
	Inhalation	7.29E-10	7.28E-07
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		9.85E-05	1.93E+01

Future Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.22E-05	4.19E-03
	Dermal	2.47E-04	3.44E-01
	Inhalation	3.65E-09	7.28E-07
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		4.25E-04	3.01E+01

(TEF Methodology) Future Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.37E-07	4.60E-04
	Dermal	2.76E-06	1.23E-02
	Inhalation	7.29E-10	7.28E-07
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		4.95E-05	1.93E+01

Future Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.84E-07	4.19E-03
	Dermal	1.38E-05	3.44E-01
	Inhalation	3.65E-09	7.28E-07
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		1.80E-04	3.01E+01

TABLE R-33 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 22
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Residential Adult -- Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.86E-06	1.37E-03
	Dermal	6.91E-05	1.73E-02
	Inhalation	3.06E-09	3.06E-06
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Sediment	Dermal	2.89E-08	1.58E-01
Surface Water	Dermal	1.22E-09	4.55E-04
Total		2.14E-04	5.63E+01

Future Residential Adult -- Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.12E-05	1.24E-02
	Dermal	4.15E-04	4.81E-01
	Inhalation	1.84E-08	3.06E-06
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Sediment	Dermal	1.73E-07	1.00E+00
Surface Water	Dermal	7.30E-09	2.10E-03
Total		1.05E-03	8.75E+01

Future Residential Child -- Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	6.00E-05	1.08E-01
	Dermal	1.58E-04	1.10E+00
	Inhalation	1.45E-08	1.45E-05
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Sediment	Dermal	1.17E-07	4.06E+00
Surface Water	Dermal	1.97E-08	3.40E-02
Total		5.33E-04	1.27E+02

TABLE R-33 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR SWMU 22
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.83E-07	1.37E-03
	Dermal	3.86E-06	1.73E-02
	Inhalation	3.06E-09	3.06E-06
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Sediment	Dermal	2.89E-08	1.58E-01
Surface Water	Dermal	1.22E-09	4.55E-04
Total		1.42E-04	5.63E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.30E-06	1.24E-02
	Dermal	2.31E-05	4.81E-01
	Inhalation	1.84E-08	3.06E-06
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Sediment	Dermal	1.73E-07	1.00E+00
Surface Water	Dermal	7.30E-09	2.10E-03
Total		6.20E-04	8.75E+01

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.35E-06	1.08E-01
	Dermal	8.81E-06	1.10E+00
	Inhalation	1.45E-08	1.45E-05
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Sediment	Dermal	1.17E-07	4.06E+00
Surface Water	Dermal	1.97E-08	3.40E-02
Total		3.28E-04	1.27E+02

TABLE R-34
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR AREA OF CONCERN 2
LEXINGTON-BLUEGRASS army DEPOT

Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	1.07E-03
	Dermal	ND	1.33E-02
	Inhalation	ND	1.20E-06
Groundwater	Ingestion	7.26E-05	1.61E+01
	Dermal	6.80E-07	4.92E-01
	Inhalation	1.85E-07	2.84E-04
Total		7.35E-05	1.66E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	1.07E-03
	Dermal	ND	1.33E-02
	Inhalation	ND	1.07E-03
Groundwater	Ingestion	3.11E-04	6.19E+01
	Dermal	2.91E-06	1.21E+00
	Inhalation	7.93E-07	3.05E-02
Total		3.15E-04	6.32E+01

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	ND	9.32E-03
	Dermal	ND	3.03E-02
	Inhalation	ND	5.71E-06
Groundwater	Ingestion	1.59E-04	3.51E+01
	Dermal	1.12E-06	8.08E-01
	Inhalation	8.09E-07	1.24E-03
Total		1.61E-04	3.59E+01

ND – Not Determined

TABLE R-35
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE GOLF COURSE
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology)
Future Residential Adult -- Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	1.18E-07	1.58E-06
	Dermal	1.19E-06	1.08E-05
	Inhalation	ND	ND
Groundwater	Ingestion	7.26E-05	1.61E+01
	Dermal	6.80E-07	4.92E-01
	Inhalation	1.85E-07	2.84E-04
Sediment	Dermal	1.72E-08	6.83E-02
Surface Water	Dermal	4.77E-08	3.00E-03
Total		7.48E-05	1.67E+01

Future Residential Adult -- Long Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	7.07E-07	1.58E-05
	Dermal	7.12E-06	1.08E-04
	Inhalation	ND	ND
Groundwater	Ingestion	3.11E-04	6.19E+01
	Dermal	2.91E-06	1.21E+00
	Inhalation	7.93E-07	3.05E-02
Sediment	Dermal	1.03E-07	6.58E-01
Surface Water	Dermal	2.86E-07	3.00E-03
Total		3.23E-04	6.38E+01

Future Residential Child -- Short Term

Matrix	Route	Risk	Hazard
Soil	Ingestion	1.03E-06	1.39E-04
	Dermal	2.71E-06	2.47E-04
	Inhalation	ND	ND
Groundwater	Ingestion	1.59E-04	3.51E+01
	Dermal	1.12E-06	8.08E-01
	Inhalation	8.09E-07	1.24E-03
Sediment	Dermal	6.97E-08	2.67E+00
Surface Water	Dermal	1.94E-07	1.22E-02
Total		1.65E-04	3.86E+01

ND -- Not Determined

TABLE R-35 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE GOLF COURSE
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.27E-09	1.58E-06
	Dermal	3.29E-08	1.08E-05
	Inhalation	ND	ND
Groundwater	Ingestion	7.26E-05	1.61E+01
	Dermal	6.80E-07	4.92E-01
	Inhalation	1.85E-07	2.84E-04
Sediment	Dermal	1.72E-08	6.83E-02
Surface Water	Dermal	4.77E-08	3.00E-03
Total		7.36E-05	1.67E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.96E-08	1.58E-05
	Dermal	1.97E-07	1.08E-04
	Inhalation	ND	ND
Groundwater	Ingestion	3.11E-04	6.19E+01
	Dermal	2.91E-06	1.21E+00
	Inhalation	7.93E-07	3.05E-02
Sediment	Dermal	1.03E-07	6.58E-01
Surface Water	Dermal	2.86E-07	3.00E-03
Total		3.15E-04	6.38E+01

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.86E-08	1.39E-04
	Dermal	7.52E-08	2.47E-04
	Inhalation	ND	ND
Groundwater	Ingestion	1.59E-04	3.51E+01
	Dermal	1.12E-06	8.08E-01
	Inhalation	8.09E-07	1.24E-03
Sediment	Dermal	6.97E-08	2.67E+00
Surface Water	Dermal	1.94E-07	1.22E-02
Total		1.61E-04	3.86E+01

ND – Not Determined

TABLE R-35 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE GOLF COURSE
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Recreational Adult Golfer – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.85E-09	3.26E-07
	Dermal	4.88E-08	2.22E-06
	Inhalation	ND	ND
Total		5.37E-08	2.55E-06
Future Recreational Adult Golfer – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.45E-07	3.26E-06
	Dermal	1.47E-06	2.22E-05
	Inhalation	ND	ND
Total		1.61E-06	2.55E-05
(TEF Methodology) Future Recreational Adult Golfer – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.34E-10	3.26E-07
	Dermal	1.35E-09	2.22E-06
	Inhalation	ND	ND
Total		1.48E-09	2.55E-06
Future Recreational Adult Golfer – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.03E-09	3.26E-06
	Dermal	4.06E-08	2.22E-05
	Inhalation	ND	ND
Total		4.46E-08	2.55E-05

ND – Not Determined

TABLE R-35 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE GOLF COURSE
LEXINGTON-BLUEGRASS ARMY DEPOT

Future Adult Swimmer – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	8.60E-08	2.54E-02
	Dermal	5.38E-07	3.39E-02
Total		6.24E-07	5.93E-02

Future Adult Swimmer – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	5.16E-07	2.54E-02
	Dermal	3.23E-06	3.39E-02
Total		3.75E-06	5.93E-02

Future Child Swimmer – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.76E-07	1.11E-01
	Dermal	1.07E-06	6.71E-02
Total		1.45E-06	1.78E-01

Future Adult Fisherman – Short Term			
Matrix	Route	Risk	Hazard
Fish	Ingestion	2.52E-05	3.01E+00
Total		2.52E-05	3.01E+00

Future Adult Fisherman – Long Term			
Matrix	Route	Risk	Hazard
Fish	Ingestion	1.51E-04	3.01E+00
Total		1.51E-04	3.01E+00

Future Child Fisherman – Short Term			
Matrix	Route	Risk	Hazard
Fish	Ingestion	4.45E-05	5.32E+00
Total		4.45E-05	5.32E+00

ND – Not Determined

TABLE R-36
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE TELEPHONE POLE STORAGE AREA
LEXINGTON-BLUEGRASS ARMY DEPOT

(Bap Methodology) Existing Adult Worker - Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.60E-06	5.49E-01
	Dermal	1.49E-05	6.91E+00
	Inhalation	1.43E-07	2.15E-03
Total		1.66E-05	7.46E+00
Existing Adult Worker - Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	8.01E-06	7.39E-01
	Dermal	7.43E-05	2.31E+01
	Inhalation	7.17E-07	2.15E-03
Total		8.30E-05	2.38E+01
(TEF Methodology) Existing Adult Worker - Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.44E-06	5.49E-01
	Dermal	1.15E-05	6.91E+00
	Inhalation	1.43E-07	2.15E-03
Total		1.31E-05	7.46E+00
Existing Adult Worker - Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.18E-06	7.39E-01
	Dermal	5.76E-05	2.31E+01
	Inhalation	7.17E-07	2.15E-03
Total		6.55E-05	2.38E+01

TABLE R-36 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE TELEPHONE POLE STORAGE AREA
LEXINGTON-BLUEGRASS army DEPOT

(Bap Methodology) Future Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.60E-06	5.49E-01
	Dermal	1.49E-05	6.91E+00
	Inhalation	1.43E-07	2.15E-03
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		6.32E-05	2.68E+01

Future Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	8.01E-06	7.39E-01
	Dermal	7.43E-05	2.31E+01
	Inhalation	7.17E-07	2.15E-03
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		2.49E-04	5.36E+01

(TEF Methodology) Future Adult Worker – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	1.44E-06	5.49E-01
	Dermal	1.15E-05	6.91E+00
	Inhalation	1.43E-07	2.15E-03
Groundwater	Ingestion	4.66E-05	1.93E+01
Total		5.97E-05	2.68E+01

Future Adult Worker – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	7.18E-06	7.39E-01
	Dermal	5.76E-05	2.31E+01
	Inhalation	7.17E-07	2.15E-03
Groundwater	Ingestion	1.66E-04	2.98E+01
Total		2.31E-04	5.36E+01

TABLE R-36 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE TELEPHONE POLE STORAGE AREA
LEXINGTON-BLUEGRASS ARMY DEPOT

(BaP Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.49E-06	1.54E+00
	Dermal	2.08E-05	9.68E+00
	Inhalation	6.03E-07	9.04E-03
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	2.50E-06	2.00E-02
Beef	Ingestion	6.00E-06	ND
Total		1.73E-04	6.74E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.69E-05	2.07E+00
	Dermal	1.25E-04	3.23E+01
	Inhalation	3.62E-06	9.04E-03
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	1.30E-05	4.70E-01
Beef	Ingestion	4.00E-05	2.00E-01
Total		8.03E-04	1.21E+02

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.92E-05	1.81E+01
	Dermal	4.75E-05	7.39E+01
	Inhalation	2.86E-06	4.29E-02
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	1.10E-05	3.40E-01
Beef	Ingestion	3.00E-05	8.00E-01
Total		4.46E-04	2.14E+02

ND – Not Determined

TABLE R-36 (continued)
SUMMARY OF RISK AND HAZARD CALCULATIONS
FOR THE TELEPHONE POLE STORAGE AREA
LEXINGTON-BLUEGRASS ARMY DEPOT

(TEF Methodology) Future Residential Adult – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	4.02E-06	1.54E+00
	Dermal	1.61E-05	9.68E+00
	Inhalation	6.03E-07	9.04E-03
Groundwater	Ingestion	1.30E-04	5.41E+01
	Dermal	2.01E-06	2.02E+00
	Inhalation	6.17E-06	1.38E-03
Vegetable	Ingestion	2.50E-06	2.00E-02
Beef	Ingestion	6.00E-06	ND
Total		1.67E-04	6.74E+01

Future Residential Adult – Long Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	2.41E-05	2.07E+00
	Dermal	9.67E-05	3.23E+01
	Inhalation	3.62E-06	9.04E-03
Groundwater	Ingestion	5.59E-04	8.35E+01
	Dermal	8.61E-06	2.50E+00
	Inhalation	2.65E-05	2.14E-02
Vegetable	Ingestion	1.30E-05	4.70E-01
Beef	Ingestion	4.00E-05	2.00E-01
Total		7.72E-04	1.21E+02

Future Residential Child – Short Term			
Matrix	Route	Risk	Hazard
Soil	Ingestion	3.52E-05	1.81E+01
	Dermal	3.68E-05	7.39E+01
	Inhalation	2.86E-06	4.29E-02
Groundwater	Ingestion	2.85E-04	1.18E+02
	Dermal	3.30E-06	3.31E+00
	Inhalation	2.70E-05	6.02E-03
Vegetable	Ingestion	1.10E-05	3.40E-01
Beef	Ingestion	3.00E-05	8.00E-01
Total		4.31E-04	2.14E+02

ND – Not Determined

TABLE R-37
SUMMARY OF RISK CHARACTERIZATI
LEXINGTON-BLUEGRASS ARMY
GROUNDWATER

STUDY AREA	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	>1.0E-05	>1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CH AS EX
NORTHERN PORTION	9	FUT RES AD-ST	YES	YES (7E-5)	NO	GROUNDWATER	INGESTION	arse
		FUT RES AD-LT	YES	YES	YES (3E-4)	GROUNDWATER	INGESTION/DERMAL	arse
		FUT RES CH	YES	YES	YES (2E-4)	GROUNDWATER	INGESTION/DERMAL	arse
		FUT WORK AD-ST	YES	YES (3E-5)	NO	GROUNDWATER	INGESTION	arse
		FUT WORK AD-LT	YES	YES (9E-5)	YES	GROUNDWATER	INGESTION	arse
SOUTHERN PORTION	10	FUT RES AD-ST	YES	YES	YES (1E-4)	GROUNDWATER	INGEST/DERM/INHAL	viny ber
		FUT RES AD-LT	YES	YES	YES (6E-4)	GROUNDWATER	INGEST/DERM/INHAL	viny viny
		FUT RES CH	YES	YES	YES (3E-4)	GROUNDWATER	INGEST/DERM/INHAL	viny viny
		FUT WORK AD-ST	YES	YES (5E-5)	NO	GROUNDWATER	INGESTION	viny
		FUT WORK AD-LT	YES	YES	YES (2E-4)	GROUNDWATER	INGESTION	viny

TABLE R-37
CHARACTERIZATION RESULTS
BLUEGRASS ARMY DEPOT
ROUNDWATER

POSURE UTE USING CESS	CHEMICALS ASSOC WITH EXCESS	TOTAL HAZARD > 1.0	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
STION	arsenic, beryllium	YES (16.6)	GROUNDWATER	INGESTION	lead, manganese,thallium
STION/DERMAL	arsenic, beryllium/beryllium	YES (63.1)	GROUNDWATER	INGESTION/DERMAL	lead, manganese,thallium sodium/thallium
STION/DERMAL	arsenic, beryllium/beryllium	YES (35.9)	GROUNDWATER	INGESTION	arsenic, lead, manganese, thallium
STION	arsenic, beryllium	YES (5.74)	GROUNDWATER	INGESTION	manganese, thallium
STION	arsenic, beryllium	YES (19.0)	GROUNDWATER	INGESTION	manganese, thallium
ST/DERM/INHAL	vinyl chloride, arsenic, beryllium/ beryllium/vinyl chloride	YES (56.1)	GROUNDWATER	INGESTION/DERMAL	antimony, manganese, thallium/manganese
ST/DERM/INHAL	vinyl chloride, arsenic, beryllium/ vinyl chloride, beryllium/vinyl chloride	YES (86.0)	GROUNDWATER	INGESTION/DERMAL	antimony, manganese, aluminum,thallium/ manganese
ST/DERM/INHAL	vinyl chloride, arsenic, beryllium/ vinyl chloride,beryllium/vinyl chloride	YES (121)	GROUNDWATER	INGESTION/DERMAL	antimony, lead, manganese, thallium/manganese
STION	vinyl chloride, arsenic, beryllium	YES (19.3)	GROUNDWATER	INGESTION	antimony, manganese, thallium
STION	vinyl chloride, arsenic, beryllium	YES (29.8)	GROUNDWATER	INGESTION	antimony, manganese, aluminum,thallium

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TABLE R-38
SUMMARY OF RISK CHARACTERIZATION RESULTS F
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	>1.0E-05	>1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
1	SWMU 4	23	FUT RES AD-ST	YES	YES (6E-5)	NO	SOIL	ING/DERM/INH	Arsenic, Indeno/B[A
									B[A]P, Indeno, Arse
			FUT RES AD-LT	YES	YES	YES (3E-4)	SOIL	ING/DERM/INH	Arsenic, B[B]F, B[A]
									Arsenic, B[A]A, B[A]
									B[K]F, Chrys, Indeno
			FUT RES CH	YES	YES	YES (2E-4)	SOIL	ING/DERM/INH	Arsenic, B[A]P, B[A]
									Indeno/Arsenic, Chr
									B[B]F, B[K]F, Chrys
2	SWMU 2, 5, 6, AND 7	24	FUT RES AD-ST	YES	YES	YES (2E-3)	SOIL/SED	ING/DERM/DERM	B[A]A, B[A]P, B[K]F
									B[A]P, B[B]F, B[K]F
									B[A]A, Chrys, Indeno
			FUT RES AD-LT	YES	YES	YES (1E-2)	SOIL/SED	ING/DERM/INH	B[A]A, B[A]P, B[K]F
								DERM	B[B]F, Dibenz, Inden
									Dibenz, B[A]A, Inden
									B[B]F, Chrys, B[A]A,
			FUT RES CH	YES	YES	YES (5E-3)	SOIL/SED	ING/DERM/DERM	B[A]A, B[A]P, B[K]F,
									B[A]A, B[A]P, B[K]F,
									B[A]A, B[A]P, B[K]F,
3	SWMU 1	25	FUT RES AD-ST	NO	NO	NO			
			FUT RES AD-LT	YES (5E-6)	NO	NO	SOIL/SED	DERM/DERM	B[A]A, Chrys/B[B]F,
			FUT RES CH	YES (3E-6)	NO	NO	SOIL	DERM	B[A]A, Chrys
4	AREA A	26	FUT RES AD-ST	YES	YES (2E-5)	NO	SED	DERM	B[A]A, B[A]P, B[B]F,
			FUT RES AD-LT	YES	YES	YES (1E-4)	SED	DERM	B[A]A, B[A]P, B[B]F,
			FUT RES CH	YES	YES (7E-5)	NO	SED	DERM	B[A]A, B[A]P, B[B]F,
5	AREA B	27	FUT RES AD-ST	YES	YES	YES (2E-2)	SOIL	ING/DERM	B[A]A, B[B]F, B[K]F,
									B[A]A, B[B]F, B[K]F,
			FUT RES AD-LT	YES	YES	YES (1E-1)	SOIL	ING/DERM	B[A]A, B[B]F, B[K]F,
									B[A]A, B[B]F, B[K]F,
			FUT RES CH	YES	YES	YES (5E-2)	SOIL	ING/DERM	B[A]A, B[B]F, B[K]F,
									B[A]A, B[B]F, B[K]F,
6	AREA C	28	FUT RES AD-ST	YES (3E-6)	NO	NO	SED	DERM	B[B]F
			FUT RES AD-LT	YES	YES (2E-5)	NO	SED	DERM	B[B]F
			FUT RES CH	YES	YES (1E-5)	NO	SED	DERM	B[B]F

R–38
CTERIZATION RESULTS FOR SOIL
JEGRASS ARMY DEPOT

POSURE UTE USING CESS	CHEMICALS ASSOC WITH EXCESS	TOTAL HAZARD > 1.0	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
ERM/INH	Arsenic, Indeno/B[A]A, B[B]F, Chrys, B[A]P, Indeno, Arsenic/Arsenic	YES (5.3)	SOIL	DERM	Lead, Thallium
ERM/INH	Arsenic, B[B]F, B[A]P, Indeno/ Arsenic, B[A]A, B[A]P, B[B]F, B[K]F, Chrys, Indeno, PCB 1260/Arsenic	YES (5.2)	SOIL	DERM	Lead, Thallium
ERM/INH	Arsenic, B[A]P, B[A]A, B[B]F, Indeno/Arsenic, Chrys, B[A]A, B[A]P, B[B]F, B[K]F, Chrys, Indeno/Arsenic	YES (42.2)	SOIL	ING/DERM	Arsenic, Lead, Thallium/Arsenic, Cadmium, Lead, Thallium
ERM/DERM	B[A]A, B[A]P, B[K]F, Chrys, B[B]F, Indeno/B[A]A, B[A]P, B[B]F, B[K]F, Chrys, Dibenz, Indeno/B[B]F B[A]A, Chrys, Indeno	YES (27.1)	SOIL	DERM/INH	Manganese, Thallium/Manganese
ERM/INH	B[A]A, B[A]P, B[K]F, Chrys B[B]F, Dibenz, Indeno/B[A]P, B[B]F, B[k]F, Chrys Dibenz, B[A]A, Indeno, PCB 1260/Cadmium, Nickel/Indeno B[B]F, Chrys, B[A]A, Arsenic, B[A]P, B[K]F	YES (40.0)	SOIL	DERM/INH	Manganese, Thallium, Cadmium/Manganese
ERM/DERM	B[A]A, B[A]P, B[K]F, Chrys, B[B]F, Dibenz, Indeno/ B[A]A, B[A]P, B[K]F, Chrys, B[B]F, Dibenz, Indeno/ B[A]A, B[A]P, B[K]F, Chrys, B[B]F, Indeno, Arsenic	YES (137)	SOIL	ING/DERM/INH	Thallium, Manganese/Cadmium, Lead, Manganese, Thallium/Manganese
		YES (17.1)	SOIL	DERM/INH	Manganese, Thallium/Manganese
/DERM	B[A]A, Chrys/B[B]F, Chrys	YES (30.8)	SOIL	DERM/INH	Manganese, Thallium/Manganese
	B[A]A, Chrys	YES (102)	SOIL	ING/DERM/INH	Thallium/Manganese, Thallium/Manganese
	B[A]A, B[A]P, B[B]F, Chrys, Indeno, B[K]F	YES (15.8)	SOIL	DERM/INH	Manganese/Manganese
	B[A]A, B[A]P, B[B]F, Chrys, Indeno, B[K]F	YES (15.8)	SOIL	DERM/INH	Manganese/Manganese
	B[A]A, B[A]P, B[B]F, Chrys, Indeno, B[K]F	YES (60.1)	SOIL	DERM/INH	Manganese/Manganese
ERM	B[A]A, B[B]F, B[K]F, Chrys, Indeno, Dibenz/ B[A]A, B[B]F, B[K]F, Dibenz, Indeno, Chrys	NO			
ERM	B[A]A, B[B]F, B[K]F, Chrys, Indeno, Dibenz/ B[A]A, B[B]F, B[K]F, Dibenz, Indeno, Chrys, Dieldrin	NO			
ERM	B[A]A, B[B]F, B[K]F, Chrys, Indeno, Dibenz/ B[A]A, B[B]F, B[K]F, Dibenz, Indeno, Chrys	YES(1.8)	SOIL	DERM	Benzo(g,h,i)perylene, Phenanthrene, Naphthalene
	B[B]F	NO			
	B[B]F	NO			
	B[B]F	YES (2.6)	SOIL	DERM	Lead

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TABLE R-38(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS F(
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	>1.0E-05	>1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
7	SWMU 3	29	EX AD WKR-ST	NO	NO	NO			
			EX AD WKR-LT	YES (3E-6)	NO	NO	SOIL	DERM	B[A]A, Chrys, PCB 1
			FUT AD WKR-ST	NO	NO	NO			
			FUT AD WKR-LT	YES (5E-6)	NO	NO	SOIL	DERM	Be[A]A, B[K]F, Chry
			FUT RES AD-ST	YES	YES (2E-5)	NO	SOIL/SED	DERM/DERM	B[A]A, B[K]F, Chrys
			FUT RES AD-LT	YES	YES	YES (1E-4)	SOIL/SED/SWAT	DERM/DERM/DERM	B[A]A, B[K]F, Chrys B[K]F, PCB 1260, Ar Bis(2-ethylhexyl)ph
			FUT RES CH	YES	YES (8E-5)	NO	SOIL/SED/SWAT	ING, DERM/DERM/ DERM	B[A]A, B[K]F, Chrys B[K]F, PCB 1260, Ar Bis(2-ethylhexyl)ph
8	SWMU 24	30	FUT AD WKER-ST	YES	YES (8E-5)	NO	SOIL	ING/DERM	B[A]A, Chrys, Arseni Chrys, Arsenic
			FUT AD WKER-LT	YES	YES	YES (4E-4)	SOIL	ING/DERM/INH	B[A]A, Chrys, Arseni Chrys, Arsenic, bis(2
			EX AD WKER-ST	YES	YES (8E-5)	NO	SOIL	ING/DERM	B[A]A, Chrys, Arseni Chrys, Arsenic
			EX AD WKER-LT	YES	YES	YES (4E-4)	SOIL	ING/DERM/INH	B[A]A, Chrys, Arseni Chrys, Arsenic, bis(2
			FUT RES AD-ST	YES	YES	YES (1E-4)	SOIL	ING/DERM/INH	B[A]A, Chrys, Arseni Chrys, Arsenic/Arser
			FUT RES AD-LT	YES	YES	YES (8E-4)	SOIL/VEG	ING/DERM/INH/ ING	B[A]A, Chrys, Arseni bis(2-ethylhexyl)pht
			FUT RES CH	YES	YES	YES (4E-4)	SOIL/VEG	ING/DERM/INH/ ING	B[A]A, Chrys, Arseni Chrys, Arsenic, bis(2
9	BUILDING 135	31	FUT AD WKER-ST	NO	NO	NO			
			FUT AD WKER-LT	NO	NO	NO			
			EX AD WKER-ST	NO	NO	NO			
			EX AD WKER-LT	NO	NO	NO			
			FUT RES AD-ST	NO	NO	NO			
			FUT RES AD-LT	NO	NO	NO			
			FUT RES CH	NO	NO	NO			
10	BUILDING 147	32	FUT AD WKER-ST	NO	NO	NO			
			FUT AD WKER-LT	NO	NO	NO			
			EX AD WKER-ST	NO	NO	NO			
			EX AD WKER-LT	NO	NO	NO			
			FUT RES AD-ST	NO	NO	NO			
			FUT RES AD-LT	NO	NO	NO			
			FUT RES CH	NO	NO	NO			

-38(cont'd)
TERIZATION RESULTS FOR SOIL
EGRASS ARMY DEPOT

OSURE TE SING ESS	CHEMICALS ASSOC WITH EXCESS	TOTAL HAZARD > 1.0	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
		NO			
	B[A]A, Chrys, PCB 1260	YES (8.1)	SOIL	DERM	Cadmium
		NO			
	Be[A]A, B[K]F, Chrys, PCB 1260	NO			
DERM	B[A]A, B[K]F, Chrys, PCB 1260/B[B]F, PCB 1260, Arsenic	NO			
ERM/DERM	B[A]A, B[K]F, Chrys, PCB 1260/ B[B]F, B[K]F, PCB 1260, Arsenic/ Bis(2-ethylhexyl)phthalate, Beryllium	YES (6.6)	SED	DERM	Cadmium
RM/DERM/	B[A]A, B[K]F, Chrys, PCB 1260/B[B]F, B[K]F, PCB 1260, Arsenic/ Bis(2-ethylhexyl)phthalate, Beryllium	YES (25)	SOIL/SED	DERM/DERM	Cadmium/PCB 1260, Cadmium
RM	B[A]A, Chrys, Arsenic/B[A]A Chrys, Arsenic	YES (1.4)	SOIL	DERM	Arsenic
RM/INH	B[A]A, Chrys, Arsenic/B[A]A, Chrys, Arsenic, bis(2-ethylhexyl)phthalate	YES (2.3)	SOIL	DERM	Cadmium, Arsenic
RM	B[A]A, Chrys, Arsenic/B[A]A Chrys, Arsenic	YES (1.4)	SOIL	DERM	Arsenic
RM/INH	B[A]A, Chrys, Arsenic/B[A]A, Chrys, Arsenic, bis(2-ethylhexyl)phthalate/Arsenic	YES (2.3)	SOIL	DERM	Cadmium, Arsenic
RM/INH	B[A]A, Chrys, Arsenic/B[A]A Chrys, Arsenic/Arsenic	YES (2.2)	SOIL	DERM	Arsenic
RM/INH/	B[A]A, Chrys, Arsenic/B[A]A, Chrys, Arsenic, bis(2-ethylhexyl)phthalate/Arsenic/DEHP	YES (3.6)	SOIL	DERM	Cadmium, Arsenic
RM/INH/	B[A]A, Chrys, Arsenic/B[A]A, Chrys, Arsenic, bis(2-ethylhexyl)phthalate/Arsenic/DEHP	YES (10.7)	SOIL	ING/DERM	Arsenic, Lead/Arsenic, Cadmium, Lead
		NO			
		NO			
		NO			
		NO			
		NO			
		NO			
		NO			
		NO			
		NO			
		NO			
		NO			
		NO			

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TABLE R-38(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS FO
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	>1.0E-05	>1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
11	BUILDING 3	33	FUT AD WKER-ST	YES	YES	YES(1E-4)	SOIL	ING/DERM	B[A]A, B[A]P, Chrys/ Chrys, Dibenz
			FUT AD WKER-LT	YES	YES	YES(6E-4)	SOIL	ING/DERM	B[A]A, B[A]P, B[B]F, B[K]F, Chrys/B[A]A,
			EX AD WKER-ST	YES	YES	YES (1E-4)	SOIL	ING/DERM	B[A]A, B[A]P, Chrys/ Chrys, Dibenz
			EX AD WKER-LT	YES	YES	YES (6E-4)	SOIL	ING/DERM	B[A]A, B[A]P, B[B]F, B[K]F, Chrys/B[A]A,
			FUT RES AD-ST	YES	YES	YES (2E-4)	SOIL	ING/DERM	B[A]A, B[A]P, B[B]F,
			FUT RES AD-LT	YES	YES	YES (1E-3)	SOIL	ING/DERM	B[A]A, B[A]P, B[B]F, B[A]A, B[A]P, B[B]F, B[A]A, B[A]P, B[B]F,
			FUT RES CH	YES	YES	YES (5E-4)	SOIL	ING/DERM	B[A]A, B[A]P, B[B]F B[K]F, Chrys, Dibenz B[A]P, B[B]F, B[K]F,
12	BUILDING 10	34	FUT AD WKER-ST	YES	YES (7E-5)	NO	SOIL	ING/DERM	Arsenic/Arsenic, Ber
			FUT AD WKER-LT	YES	YES	YES (3E-4)	SOIL	ING/DERM	Arsenic, Beryllium/Ar B[K]F, Chrys/Arsenic
			EX AD WKER-ST	YES	YES (7E-5)	NO	SOIL	ING/DERM	Arsenic/Arsenic, Ber
			EX AD WKER-LT	YES	YES	YES (3E-4)	SOIL	ING/DERM	Arsenic, Beryllium/Ar B[K]F, Chrys
			FUT RES AD-ST	YES	YES	YES (1E-4)	SOIL	ING/DERM	Arsenic, Beryllium/Ar B[K]F, Chrys
			FUT RES AD-LT	YES	YES	YES (6E-4)	SOIL	ING/DERM/INH	Arsenic, Beryllium, B Arsenic, Beryllium, B
			FUT RES CH	YES	YES	YES (3E-4)	SOIL	ING/DERM/INH	Arsenic, Beryllium, B Arsenic, Beryllium, B
13	BUILDING 19	35	FUT AD WKER-ST	YES	YES (6E-5)	NO	SOIL	ING/DERM	Arsenic, Beryllium/Ar
			FUT AD WKER-LT	YES	YES	YES (3E-4)	SOIL	ING/DERM	Arsenic, Beryllium/Ar
			EX AD WKER-ST	YES	YES (6E-5)	NO	SOIL	ING/DERM	Arsenic, Beryllium/Ar
			EX AD WKER-LT	YES	YES	YES (3E-4)	SOIL	ING/DERM	Arsenic, Beryllium/Ar
			FUT RES AD-ST	YES	YES (9E-5)	NO	SOIL	ING/DERM	Arsenic, Beryllium/Ar
			FUT RES AD-LT	YES	YES	YES (5E-4)	SOIL	ING/DERM/INH	Arsenic, Beryllium/Ar
			FUT RES CH	YES	YES	YES (2E-4)	SOIL	ING/DERM/INH	Arsenic, Beryllium/Ar

cont'd)
ATION RESULTS FOR SOIL
SS ARMY DEPOT

E	CHEMICALS ASSOC WITH EXCESS	TOTAL HAZARD > 1.0	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
	B[A]A, B[A]P, Chrys/B[A]A, B[A]P, B[B]F, B[K]F Chrys, Dibenz	YES(7.8)	SOIL	DERM	Manganese, Barium
	B[A]A, B[A]P, B[B]F, B[K]F, Chrys/B[A]A, B[A]P, B[B]F, B[K]F, Chrys	YES(11.6)	SOIL	DERM	Manganese, Barium
	B[A]A, B[A]P, Chrys/B[A]A, B[A]P, B[B]F, B[K]F Chrys, Dibenz	YES (7.8)	SOIL	DERM	Manganese, Barium
	B[A]A, B[A]P, B[B]F, B[K]F, Chrys/B[A]A, B[A]P, B[B]F, B[K]F, Chrys	YES (11.6)	SOIL	DERM	Manganese, Barium
	B[A]A, B[A]P, B[B]F, B[K]F, Chrys/Dibenz/ B[A]A, B[A]P, B[B]F, Dibenz, B[K]F, Chry	YES (13.2)	SOIL	DERM/INH	Manganese, Barium/Manganese
	B[A]A, B[A]P, B[B]F, B[K]F, Chrys/Dibenz/ B[A]A, B[A]P, B[B]F, B[K]F, Chrys, Dibenz	YES (13.3)	SOIL	DERM/INH	Manganese, Barium/Manganese
	B[A]A, B[A]P, B[B]F B[K]F, Chrys, Dibenz/B[A]A, B[A]P, B[B]F, B[K]F, Chrys, Dibenz	YES (39.5)	SOIL	ING/DERM/INH	Manganese, Barium/Manganese, Barium, Lead/Manganese
	Arsenic/Arsenic, Beryllium, B[A]A, B[K]F, Chrys	YES (11.2)	SOIL	DERM	Manganese, Thallium, Vanadium, Lead
	Arsenic, Beryllium/Arsenic, Beryllium, B[A]A, B[K]F, Chrys/Arsenic	YES (11.5)	SOIL	DERM	Manganese, Thallium, Vanadium, Lead
	Arsenic/Arsenic, Beryllium, B[A]A, B[K]F, Chrys	YES (11.2)	SOIL	DERM	Manganese, Thallium, Vanadium, Lead
	Arsenic, Beryllium/Arsenic, Beryllium, B[A]A, B[K]F, Chrys	YES (11.5)	SOIL	DERM	Manganese, Thallium, Vanadium, Lead
	Arsenic, Beryllium/Arsenic, Beryllium, B[A]A, B[K]F, Chrys	YES (17.1)	SOIL	DERM/INH	Lead, Manganese, Thallium, Vanadium/Manganese
H	Arsenic, Beryllium, B[A]A, B[K]F, Chrys/ Arsenic, Beryllium, B[A]A, B[K]F, Chrys/Arsenic	YES (35.0)	SOIL	ING/DERM/INH	Thallium/Lead, Manganese, Thallium, Vanadium/Manganese
H	Arsenic, Beryllium, B[A]A, B[K]F, Chrys/ Arsenic, Beryllium, B[A]A, B[K]F, Chrys/Arsenic	YES (90.4)	SOIL	ING/DERM/INH	Arsenic, Lead, Thallium/Arsenic, Lead, Barium, Manganese, Thallium, Vanadium/Manganese
	Arsenic, Beryllium/Arsenic, Beryllium	YES (53.5)	SOIL	DERM	Manganese, Thallium
	Arsenic, Beryllium/Arsenic, Beryllium	YES (53.5)	SOIL	DERM	Manganese, Thallium
	Arsenic, Beryllium/Arsenic, Beryllium	YES (53.5)	SOIL	DERM	Manganese, Thallium
	Arsenic, Beryllium/Arsenic, Beryllium	YES (53.5)	SOIL	DERM	Manganese, Thallium
	Arsenic, Beryllium/Arsenic, Beryllium	YES (78)	SOIL	ING/DERM/INH	Thallium/Manganese, Thallium/Manganese
H	Arsenic, Beryllium/Arsenic, Beryllium/Arsenic	YES (78)	SOIL	ING/DERM/INH	Thallium/Manganese, Thallium/Manganese
H	Arsenic, Beryllium/Arsenic, Beryllium/Arsenic	YES (200)	SOIL	ING/DERM/INH	Lead, Manganese, Thallium/Barium, Lead, Manganese, Thallium/Manganese

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TABLE R-38(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	>1.0E-05	>1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WIT EXCESS
14	BUILDING 63	36	FUT AD WKER-ST	YES	YES	YES (2E-4)	SOIL	ING/DERM	Arsenic, B[A]A, E
									Arsenic, B[A]A, E
			FUT AD WKER-LT	YES	YES	YES (9E-4)	SOIL	ING/DERM	Arsenic, B[A]A, E
									Arsenic, B[A]A, E
									Dieldrin/Arsenic,
			EX AD WKER-ST	YES	YES	YES (2E-4)	SOIL	ING/DERM	Arsenic, B[A]A, E
									Arsenic, B[A]A, E
			EX AD WKER-LT	YES	YES	YES (9E-4)	SOIL	ING/DERM	Arsenic, B[A]A, E
									Arsenic, B[A]A, E
			FUT RES AD-ST	YES	YES	YES (3E-4)	SOIL/VEG	ING/DERM/ING	Arsenic, B[A]A, E
									Arsenic, B[A]A, E
			FUT RES AD-LT	YES	YES	YES (2E-3)	SOIL/VEG	ING/DERM/INH/	Arsenic, B[A]A, E
								ING	Arsenic, B[A]A, E
									DDT, DEHP, Diel
			FUT RES CH	YES	YES	YES (8E-4)	SOIL/VEG	ING/DERM/INH/	Arsenic, B[A]A, E
								ING	Arsenic, B[A]A, E
									DEHP, Dieldrin/A
15	BUILDING 64	37	FUT AD WKER-ST	NO	NO	NO			
			FUT AD WKER-LT	NO	NO	NO			
			EX AD WKER-ST	NO	NO	NO			
			EX AD WKER-LT	NO	NO	NO			
			FUT RES AD-ST	NO	NO	NO			
			FUT RES AD-LT	NO	NO	NO			
			FUT RES CH	NO	NO	NO			
16	BUILDING 130	38	FUT AD WKER-ST	YES	YES (2E-5)	NO	SOIL	DERM	B[A]A, B[B]F, Ch
			FUT AD WKER-LT	YES	YES	YES (1E-4)	SOIL	ING/DERM	B[B]F/B[A]A, B[B
									bis(2-ethylhexyl
			EX AD WKER-ST	YES	YES (2E-5)	NO	SOIL	DERM	B[A]A, B[B]F, Ch
			EX AD WKER-LT	YES	YES	YES (1E-4)	SOIL	ING/DERM	B[B]F/B[A]A, B[B
									bis(2-ethylhexyl
			FUT RES AD-ST	YES	YES (3E-5)	NO	SOIL/VEG	ING/DERM/ING	B[B]F/B[A]A, B[B
			FUT RES AD-LT	YES	YES	YES (2E-4)	SOIL/VEG	ING/DERM/ING	B[A]A, B[B]F, Ch
									B[A]A, B[B]F, Ch
									PCB 1260, DEHP
			FUT RES CH	YES	YES (9E-5)	NO	SOIL/VEG	ING/DERM/ING	B[A]A, B[B]F, Ch
									B[A]A, B[B]F, Ch
									PCB 1260, DEHP

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ON RESULTS FOR SOIL
RMY DEPOT

CHEMICALS ASSOC WITH EXCESS	TOTAL HAZARD > 1.0	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
Arsenic, B[A]A, B[B]F, Chrys/	YES (12.3)	SOIL	DERM	Arsenic, Lead, Vanadium
Arsenic, B[A]A, B[B]F, Chrys				
Arsenic, B[A]A, B[B]F, Chrys/	YES (13.7)	SOIL	DERM	Arsenic, Cadmium, Lead, Vanadium
Arsenic, B[A]A, B[B]F, Chrys, bis(2-ethylhexyl)phthalate,				Arsenic, Cadmium, Lead, Vanadium
Dieldrin/Arsenic, B[A]A, B[B]F,Chrys				
Arsenic, B[A]A, B[B]F, Chrys/	YES (12.3)	SOIL	DERM	Arsenic, Lead, Vanadium
Arsenic, B[A]A, B[B]F, Chrys				
Arsenic, B[A]A, B[B]F, Chrys/	YES (13.7)	SOIL	DERM	Arsenic, Cadmium, Lead, Vanadium
Arsenic, B[A]A, B[B]F, Chrys, bis(2-ethylhexyl)phthalate, Dieldrin				
Arsenic, B[A]A, B[B]F, Chrys/	YES (18.9)	SOIL	ING/DERM	Lead/Arsenic, Lead,
Arsenic, B[A]A, B[B]F, Chrys/DEHP, DDT, Dieldrin				Vanadium
Arsenic, B[A]A, B[B]F, Chrys/	YES (20.2)	SOIL	ING/DERM	Lead/Arsenic, Barium, Cadmium, Lead,
Arsenic, B[A]A, B[B]F, Chrys				Vanadium
DDT, DEHP, Dieldrin/Arsenic/DEHP, DDT, Dieldrin				
Arsenic, B[A]A, B[B]F, Chrys/	YES (57.5)	SOIL	ING/DERM	Arsenic, Lead/Arsenic, Barium,
Arsenic, B[A]A, B[B]F, Chrys/				Cadmium, Lead, Vanadium
DEHP, Dieldrin/Arsenic/DEHP, DDT, Dieldrin				
	NO			
	NO			
	NO			
	NO			
	NO			
	NO			
	NO			
B[A]A, B[B]F, Chrys, PCB 1260	YES (6.8)	SOIL	DERM	Lead
B[B]F/B[A]A, B[B]F	YES (6.8)	SOIL	DERM	Cadmium, Lead
bis(2-ethylhexyl)phthalate, Chrysene, PCB 1260/B[B]F				
B[A]A, B[B]F, Chrys, PCB 1260	YES (6.8)	SOIL	DERM	Lead
B[B]F/B[A]A, B[B]F	YES (6.8)	SOIL	DERM	Cadmium, Lead
bis(2-ethylhexyl)phthalate, Chrysene, PCB 1260				
B[B]F/B[A]A, B[B]F, Chrys, PCB 1260/PCB 1260	YES (9.8)	SOIL	DERM	Lead
B[A]A, B[B]F, Chrys, PCB 1260/	YES (10.0)	SOIL	DERM	Cadmium, Lead
B[A]A, B[B]F, Chrys, PCB 1260, bis(2-ethylhexyl)phthalate/				
PCB 1260, DEHP				
B[A]A, B[B]F, Chrys, PCB 1260/	YES (25.3)	SOIL	ING/DERM	Cadmium, Lead/Cadmium, Lead
B[A]A, B[B]F, Chrys, PCB 1260, bis(2-ethylhexyl)phthalate/				
PCB 1260, DEHP				

TABLE R-38(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS F
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	>1.0E-05	>1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
17	BUILDING 141	39	FUT AD WKER-ST	NO	NO	NO			
			FUT AD WKER-LT	NO	NO	NO			
			EX AD WKER-ST	NO	NO	NO			
			EX AD WKER-LT	NO	NO	NO			
			FUT RES AD-ST	NO	NO	NO			
			FUT RES AD-LT	NO	NO	NO			
			FUT RES CH	NO	NO	NO			
18	SWMU 23	40	FUT AD WKER-ST	YES	YES	YES (2E-4)	SOIL	ING/DERM	B[A]A, B[B]F, Chry
			FUT AD WKER-LT	YES	YES	YES(1E-3)	SOIL	ING/DERM/INH	B[A]A, B[B]F, Chry
									B[A]A, B[B]F, Chry
			EX AD WKER-ST	YES	YES	YES (2E-4)	SOIL	ING/DERM	B[A]A, B[B]F, Chry
			EX AD WKER-LT	YES	YES	YES(1E-3)	SOIL	ING/DERM/INH	B[A]A, B[B]F, Chry
			FUT RES AD-ST	YES	YES	YES(3E-4)	SOIL	ING/DERM/INH	B[A]A, B[B]F, Chry
			FUT RES AD-LT	YES	YES	YES(2E-3)	SOIL/VEG	ING/DERM/INH/	B[A]A, B[B]F, Chry
								ING	Cadmium/Dieldrin
			FUT RES CH	YES	YES	YES(8E-4)	SOIL/VEG	ING/DERM/INH/	B[A]A, B[B]F, Chry
								ING	Cadmium/Dieldrin
19	SWMU 18 AND 19	41	EX AD WKER-ST	NO	NO	NO			
			EX AD WKER-LT	NO	NO	NO			
			FUT AD WKER-ST	NO	NO	NO			
			FUT AD WKER-LT	NO	NO	NO			
			FUT RES AD-ST	YES(4E-6)	NO	NO	SED	DERM	Chrys
			FUT RES AD-LT	YES	YES(3E-5)	NO	SED	DERM	B[A]A, B[B]F, B[K]
			FUT RES CH	YES	YES(2E-5)	NO	SED	DERM	B[A]A, B[B]F, B[K]
20	SWMU 16, 17, AND 30	42	FUT AD WKER-ST	YES (3E-6)	NO	NO	SOIL	DERM	PCB 1260
			FUT AD WKER-LT	YES	YES(1E-5)	NO	SOIL	DERM	PCB 1260, Chlorda
			EX AD WKER-ST	YES (3E-6)	NO	NO	SOIL	DERM	PCB 1260
			EX AD WKER-LT	YES	YES(1E-5)	NO	SOIL	DERM	PCB 1260, Chlorda
			FUT RES AD-ST	YES(5E-6)	NO	NO	SOIL/VEG	DERM/ING	PCB1260/PCB 126
			FUT RES AD-LT	YES	YES(3E-5)	NO	SOIL/VEG	ING/DERM/ING	PCB1260/DEHP, C
			FUT RES CH	YES	YES(2E-5)	NO	SOIL/VEG	ING/DERM/ING	PCB1260/PCB126

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RIZATION RESULTS FOR SOIL
RASS ARMY DEPOT

URE G S	CHEMICALS ASSOC WITH EXCESS	TOTAL HAZARD > 1.0	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
		NO			
		NO			
		NO			
		NO			
		NO			
		NO			
		NO			
	B[A]A, B[B]F, Chrys/B[A]A, B[B]F, Chrys	YES(1.3)	SOIL	DERM	Lead
INH	B[A]A, B[B]F, Chrys/B[A]A, B[B]F, Chrys, Dieldrin/Cadmium	YES(15.6)	SOIL	DERM	Cadmium, Lead
	B[A]A, B[B]F, Chrys				
	B[A]A, B[B]F, Chrys/B[A]A, B[B]F, Chrys	YES(1.3)	SOIL	DERM	Lead
INH	B[A]A, B[B]F, Chrys/B[A]A, B[B]F, Chrys, Dieldrin/Cadmium	YES(15.6)	SOIL	DERM	Cadmium, Lead
INH	B[A]A, B[B]F, Chrys/B[A]A, B[B]F, Chrys, Dieldrin/Cadmium	YES(1.9)	SOIL	DERM	Lead
INH/	B[A]A, B[B]F, Chrys, Dieldrin/B[A]A, B[B]F, Chrys, Dieldrin/ Cadmium/Dieldrin	YES(22.4)	SOIL	DERM	Cadmium, Lead
INH/	B[A]A, B[B]F, Chrys, Dieldrin/B[A]A, B[B]F, Chrys, Dieldrin/ Cadmium/Dieldrin	YES(55.2)	SOIL	ING/DERM	Cadmium, Lead/Cadmium, Lead
		NO			
		NO			
		NO			
		NO			
	Chrys	NO			
	B[A]A, B[B]F, B[K]F, Chrys	NO			
	B[A]A, B[B]F, B[K]F, Chrys	NO			
	PCB 1260	YES(2.4)	SOIL	DERM	Mercury
	PCB 1260, Chlordane	YES(3.0)	SOIL	DERM	Cadmium, Mercury
	PCB 1260	YES(2.4)	SOIL	DERM	Mercury
	PCB 1260, Chlordane	YES(3.0)	SOIL	DERM	Cadmium, Mercury
	PCB1260/PCB 1260	YES(3.5)	SOIL	DERM	Mercury
NG	PCB1260/DEHP, Chlordane, Dieldrin, PCB1260/PCB 1260	YES(4.5)	SOIL	DERM	Cadmium, Mercury
NG	PCB1260/PCB1260/PCB 1260	YES(11.2)	SOIL	ING/DERM	Cadmium, Mercury/Silver, Cadmium, Mercury

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TABLE R-38(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULT
LEXINGTON-BLUEGRASS ARMY DEPO

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	>1.0E-05	>1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WI EXCESS
21	SWMU 9	43	FUT AD WKER-ST	YES	YES	YES (3E-3)	SOIL	ING/DERM	B[A]A, B[A]P, B[B
									B[A]A, B[A]P, B[B
			FUT AD WKER-LT	YES	YES	YES(2E-2)	SOIL	ING/DERM	B[A]A, B[A]P, B[B
									B[A]A, B[A]P, B[B
									B[A]A, B[A]P, B[B
			EX AD WKER-ST	YES	YES	YES (3E-3)	SOIL	ING/DERM	B[A]A, B[A]P, B[B
									B[A]A, B[A]P, B[B
			EX AD WKER-LT	YES	YES	YES(2E-2)	SOIL	ING/DERM	B[A]A, B[A]P, B[B
									B[A]A, B[A]P, B[B
			FUT RES AD-ST	YES	YES	YES(5E-3)	SOIL/VEG/BEEF	ING/DERMING/	B[A]A, B[A]P, B[B
								ING	B[A]A, B[A]P, B[B
									PCB 1260/PCB 12
			FUT RES AD-LT	YES	YES	YES(3E-2)	SOIL/VEG/BEEF	ING/DERMING/	B[A]A, B[A]P, B[B
								ING	B[A]A, B[A]P, B[B
									PCB 1260, DDT/P
			FUT RES CH	YES	YES	YES(1E-2)	SOIL/VEG/BEEF	ING/DERMING/	B[A]A, B[A]P, B[B
								ING	B[A]A, B[A]P, B[B
									PCB 1260, DDT/P
22	BUILDING 42	44	FUT AD WKER-ST	YES	YES(7E-5)	NO	SOIL	ING/DERM	Arsenic/Arsenic,
			FUT AD WKER-LT	YES	YES	YES(3E-4)	SOIL	ING/DERM	Arsenic, Berylliu
			EX AD WKER-ST	YES	YES(7E-5)	NO	SOIL	ING/DERM	Arsenic/Arsenic,
			EX AD WKER-LT	YES	YES	YES(3E-4)	SOIL	ING/DERM	Arsenic, Berylliu
			FUT RES AD-ST	YES	YES	YES(1E-4)	SOIL	ING/DERM	Arsenic, Berylliu
			FUT RES AD-LT	YES	YES	YES(6E-4)	SOIL	ING/DERM/INH	Arsenic, Berylliu
			FUT RES CH	YES	YES	YES(3E-4)	SOIL	ING/DERM/INH	Arsenic, Berylliu
23	SWMU 20	45	EX AD WKER-ST	YES	YES(1E-5)	NO	SOIL	DERM	Aldrin
			EX AD WKER-LT	YES	YES(5E-5)	NO	SOIL	ING/DERM	Aldrin/Aldrin
			FUT AD WKER-ST	YES	YES(1E-5)	NO	SOIL	DERM	Aldrin
			FUT AD WKER-LT	YES	YES(5E-5)	NO	SOIL	ING/DERM	Aldrin/Aldrin
			FUT RES AD-ST	YES	YES(2E-5)	NO	SOIL/VEG	ING/DERM/	Aldrin/Aldrin/
								ING	Aldrin
			FUT RES AD-LT	YES	YES	YES(1E-4)	SOIL/VEG	ING/DERM/	Aldrin/Aldrin/
								ING	Aldrin, N-nitrosodi
			FUT RES CH	YES	YES(6E-5)	NO	SOIL/VEG	ING/DERM/	Aldrin/Aldrin/
								ING	Aldrin, N-nitrosodi

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TION RESULTS FOR SOIL
ARMY DEPOT

CHEMICALS ASSOC WITH EXCESS	TOTAL HAZARD > 1.0	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB 1260/ B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB 1260	YES(2.2)	SOIL	DERM	Lead
B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB 1260/ B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB 1260/ B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB 1260	YES(4.2)	SOIL	DERM	PCB 1260, Lead
B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB 1260/ B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB 1260	YES(2.2)	SOIL	DERM	Lead
B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB 1260/ B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB 1260	YES(4.2)	SOIL	DERM	PCB 1260, Lead
B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB1260/ B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB1260/ PCB 1260/PCB 1260	YES(3.3)	SOIL	DERM	Lead
B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB1260/ B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB1260, DDT/ PCB 1260, DDT/PCB 1260	YES(7.3)	SOIL/VEG	DERM	PCB 1260, Cadmium, Lead/PCB 1260
B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB1260/ B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB1260 PCB 1260, DDT/PCB 1260	YES(23.4)	SOIL/VEG/BEEF	ING/DERM	PCB 1260, Lead/PCB 1260, Cadmium Lead/PCB 1260/PCB 1260
Arsenic/Arsenic, Beryllium	YES(18.0)	SOIL	DERM	Barium, Manganese, Thallium, Vanadium
Arsenic, Beryllium/Arsenic, Beryllium	YES(41.6)	SOIL	DERM	Barium, Manganese, Thallium, Vanadium Thallium
Arsenic/Arsenic, Beryllium	YES(18.0)	SOIL	DERM	Barium, Manganese, Thallium, Vanadium
Arsenic, Beryllium/Arsenic, Beryllium	YES(41.6)	SOIL	DERM	Barium, Manganese, Thallium, Vanadium
Arsenic, Beryllium/Arsenic, Beryllium	YES(28.2)	SOIL	DERM/INH	Barium, Lead, Manganese, Thallium, Vanadium/Manganese
Arsenic, Beryllium/Arsenic, Beryllium/Arsenic	YES(61.7)	SOIL	ING/DERM/INH	Thallium/Barium, Lead, Manganese, Thallium, Vanadium/Manganese
Arsenic, Beryllium/Arsenic, Beryllium/Arsenic	YES(162)	SOIL	ING/DERM/INH	Lead, Manganese, Thallium/Barium, Lead, Manganese, Thallium, Vanadium/Manganese
Aldrin	YES(7.7)	SOIL	DERM	Lead, Manganese
Aldrin/Aldrin	YES(8.0)	SOIL	DERM	Lead, Manganese
Aldrin	YES(7.5)	SOIL	DERM	Lead, Manganese
Aldrin/Aldrin	YES(7.6)	SOIL	DERM	Lead, Manganese
Aldrin/Aldrin/ Aldrin	YES(12.5)	SOIL	DERM/INH	Lead, Manganese/Manganese
Aldrin/Aldrin/ Aldrin, N-nitrosodiphenylamine	YES(12.8)	SOIL	DERM/INH	Lead, Manganese/Manganese
Aldrin/Aldrin/ Aldrin, N-nitrosodiphenylamine	YES(38.7)	SOIL	ING/DERM/INH	Lead, Manganese/Aldrin, Barium, Lead, Manganese/Manganese

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TABLE R-38(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS F
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	>1.0E-05	>1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
24	SWMU 25	46	FUT AD WKER-ST	NO	NO	NO			
			FUT AD WKER-LT	NO	NO	NO			
			EX AD WKER-ST	NO	NO	NO			
			EX AD WKER-LT	NO	NO	NO			
			FUT RES AD-ST	NO	NO	NO			
			FUT RES AD-LT	NO	NO	NO			
			FUT RES CH	NO	NO	NO			
25	SWMU 11	47	FUT AD WKER-ST	NO	NO	NO			
			FUT AD WKER-LT	YES(2E-6)	NO	NO	SOIL	DERM	Dieldrin
			EX AD WKER-ST	NO	NO	NO			
			EX AD WKER-LT	YES(2E-6)	NO	NO	SOIL	DERM	Dieldrin
			FUT RES AD-ST	NO	NO	NO			
			FUT RES AD-LT	YES(4E-6)	NO	NO	SOIL	DERM	Dieldrin
			FUT RES CH	YES(2E-6)	NO	NO	SOIL	DERM	Dieldrin
26	COAL PILE RUNOFF/ HEATING PLANT AREA	48	FUT AD WKER-ST	YES	YES	YES(3E-4)	SOIL	ING/DERM/INH	B(b)F, Chrys, As/B(a)
			FUT AD WKER-LT	YES	YES	YES (2E-3)	SOIL	ING/DERM/INH	B(a)A, B(b)F, Chrys, Aldrin, Dieldrin, As, B
			EX AD WKER-ST	YES	YES	YES(3E-4)	SOIL	ING/DERM/INH	B(b)F, Chrys, As/B(a)
			EX AD WKER-LT	YES	YES	YES (2E-3)	SOIL	ING/DERM/INH	B(a)A, B(b)F, Chrys, Dieldrin, As, Be/As
			FUT RES AD-ST	YES	YES	YES(5E-4)	SOIL	ING/DERM/INH	B(a)A, B(b)F, Chrys, As, Be/As
			FUT RES AD-LT	YES	YES	YES(3E-3)	SOIL/VEG	ING/DERM/ING/ ING	Aldrin, B(a)A, B(b)F, B B(k)F, Chrys, Dieldrin
			FUT RES CH	YES	YES	YES(2E-3)	SOIL/VEG	ING/DERM/ING/ ING	Aldrin, B(a)A, B(b)F, B B(a)A, B(b)F, B(k)F, C

d)
N RESULTS FOR SOIL
RMY DEPOT

CHEMICALS ASSOC WITH EXCESS	TOTAL HAZARD > 1.0	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
	NO			
	NO			
	NO			
	NO			
	NO			
	NO			
	NO			
	NO			
ieldrin	NO			
	NO			
ieldrin	NO			
	NO			
ieldrin	NO			
ieldrin	NO			
(b)F, Chrys, As/B(a)A, B(b)F, B(K)F, Chrys, As, Be/As	YES(12.6)	SOIL	DERM	Arsenic, Thallium
(a)A, B(b)F, Chrys, As, Be/B(A)A, B(b)F, B(k)F, Chrys, ldrin, Dieldrin, As, Be/As	YES(66.4)	SOIL	ING/DERM	Arsenic, Thallium/Arsenic, Thallium
(b)F, Chrys, As/B(a)A, B(b)F, B(k)F, Chrys, As, Be/As	YES(12.6)	SOIL	DERM	Arsenic, Thallium
(a)A, B(b)F, Chrys, As, Be/Aldrin, B(a)A, B(b)F, B(k)F, Chrys ieldrin, As, Be/As	YES(66.5)	SOIL	ING/DERM	Arsenic, Thallium/Arsenic, Thallium
(a)A, B(b)F, Chrys, As, Be/Aldrin, B(a)A, B(b)F, B(b)F, Chrys, s, Be/As	YES(19.0)	SOIL	ING/DERM	Arsenic/Arsenic, Lead, Thallium
ldrin, B(a)A, B(b)F, B(k)F, Chrys, As, Be/Aldrin, B(a)A, B(b)F, (k)F, Chrys, Dieldrin, As, Be/As/Dieldrin	YES(95.5)	SOIL	ING/DERM	Arsenic, Thallium/Arsenic, Lead, Thallium
ldrin, B(a)A, B(b)F, B(k)F, Chrys, Dieldrin, As,Be/Aldrin, (a)A, B(b)F, B(k)F, Chrys, Dieldrin, As, Be/As/Dieldrin	YES(251)	SOIL	ING/DERM	Arsenic, Lead, Thallium/ Arsenic, Lead, Thallium

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TABLE R-38(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULT
LEXINGTON-BLUEGRASS army depo

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	>1.0E-05	>1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICAL ASSOC WI EXCESS
27	SWMU 10	49	FUT AD WKER-ST	YES	YES(4E-5)	NO	SOIL	ING/DERM/INH	As/B(a)A, B(b)F
			FUT AD WKER-LT	YES	YES	YES(2E-4)	SOIL	ING/DERM/INH	B(a)A, B(b)F, C PCB1260, As/N
			EX AD WKER-ST	YES	YES(4E-5)	NO	SOIL	ING/DERM/INH	As/B(a)A, B(b)F
			EX AD WKER-LT	YES	YES	YES(2E-4)	SOIL	ING/DERM/INH	B(a)A, B(b)F, C PCB1260, As/N
			FUT RES AD-ST	YES	YES(8E-5)	NO	SOIL/VEG/BEEF	ING/DERM/INH/ ING/ING	B(a)F, As/B(a)A PCB 1260/PCB
			FUT RES AD-LT	YES	YES	YES(5E-4)	SOIL/VEG/BEEF	ING/DERM/INH/ ING/ING	B(a)A, B(b)F, C PCB1260, As/A
			FUT RES CH	YES	YES	YES(3E-4)	SOIL/VEG/BEEF	ING/DERM/INH/ ING/ING	B(a)A, B(b)F, C PCB1260, As/A
28	BUILDING 303	50	FUT RES AD-ST	YES	YES(6E-5)	NO	SOIL/VEG/BEEF	ING/DERM/ ING/ING	Chlordane/Chlc Chlordane, Hep
			FUT RES AD-LT	YES	YES	YES(3E-4)	SOIL/VEG/BEEF	ING/DERM/ ING/ING	Chlordane, Diel Heptachlor epo Chlordane, Hep
			FUT RES CH	YES	YES	YES(2E-4)	SOIL/VEG/BEEF	ING/DERM/ ING/ING	Chlordane, Diel Heptachlor epo Chlordane, Hep
29	OPEN STORAGE AND SHELTER AREAS	51	FUT AD WKER-ST	YES	YES	YES(2E-3)	SOIL	ING/DERM	B(a)A, B(a)P, B(B(a)F, B(k)F, Ch
			FUT AD WKER-LT	YES	YES	YES(8.5E-3)	SOIL	ING/DERM/INH	B(a)A, B(b)F, B(B(a)A, B(a)F, B(Arsenic/Arsenic
			EX AD WKER-ST	YES	YES	YES(2E-3)	SOIL	ING/DERM	B(a)A, B(a)P, B(B(a)F, B(k)F, Ch
			EX AD WKER-LT	YES	YES	YES(8.5E-3)	SOIL	ING/DERM/INH	B(a)A, B(b)F, B(B(a)A, B(a)F, B(Arsenic/Arsenic
			FUT RES AD-ST	YES	YES	YES(2E-3)	SOIL/VEG	ING/DERM/INH/ ING	B(a)A, B(b)F, B(B(a)P, B(k)F, Ch Dieldrin
			FUT RES AD-LT	YES	YES	YES(2E-2)	SOIL/VEG	ING/DERM/INH/ ING	B(a)A, B(a)F, B(Arsenic/B(a)A, B Dieldrin, Arsenic
			FUT RES CH	YES	YES	YES(7E-3)	SOIL/VEG	ING/DERM/INH/ ING	B(a)A, B(a)F, B(Arsenic/B(a)F, B Dieldrin, Arsenic

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TION RESULTS FOR SOIL
ARMY DEPOT

CHEMICALS ASSOC WITH EXCESS	TOTAL HAZARD > 1.0	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
As/B(a)A, B(b)F, Chrys, PCB1260, As/Cd	YES(27.3)	SOIL	DERM	Cadmium, Chromium, Lead, Nickel, Silver
B(a)A, B(b)F, Chrys, PCB1260, As/B(a)A, B(b)F, Chrys, PCB1260, As/Ni, Cd	YES (596)	SOIL	ING/DERM	Cadmium/Cadmium, Chromium, Lead Nickel, Silver
As/B(a)A, B(b)F, Chrys, PCB1260, As/Cd	YES(27.3)	SOIL	DERM	Cadmium, Chromium, Lead, Nickel, Silver
B(a)A, B(b)F, Chrys, PCB1260, As/B(a)A, B(b)F, Chrys, PCB1260, As/Ni, Cd	YES (596)	SOIL	ING/DERM	Cadmium/Cadmium, Chromium, Lead Nickel, Silver
B(a)F, As/B(a)A, B(b)F, Chrys, PCB1260, As/Cd/ PCB 1260/PCB 1260	YES(39.5)	SOIL	ING/DERM	Lead/Barium, Chromium, Lead, Nickel, Silver
B(a)A, B(b)F, Chrys, PCB1260, As/B(a)A, B(b)F, Chrys, PCB1260, As/As, Cd, Ni/PCB 1260/PCB 1260	YES(846)	SOIL	ING/DERM	Cadmium, Lead/Barium, Cadmium, Chromium, Lead, Nickel, Silver
B(a)A, B(b)F, Chrys, PCB1260, As/B(a)A, B(b)F, Chrys, PCB1260, As/As, Cd, Ni/PCB 1260/PCB 1260	YES(2060)	SOIL	ING/DERM	Arsenic, Cadmium, Lead, Nickel, Silver/ Arsenic, Barium, Cadmium, Chromium, Lead, Nickel, Silver
Chlordane/Chlordane, Dieldrin, Heptachlor Epoxide/ Chlordane, Heptachlor epoxide/Chlordane	YES(9.0)	SOIL/VEG	DERM/ING	Chlordane/Chlordane
Chlordane, Dieldrin, Heptachlor epoxide/Chlordane, Dieldrin, Heptachlor epoxide/Chlordane, Dieldrin, Heptachlor epoxide/ Chlordane, Heptachlor epoxide	YES (9.1)	SOIL/VEG	DERM/ING	Chlordane/Chlordane
Chlordane, Dieldrin, Heptachlor epoxide/Chlordane, Dieldrin, Heptachlor epoxide/Chlordane, Dieldrin, Heptachlor epoxide/ Chlordane, Heptachlor epoxide	YES (31.2)	SOIL/VEG	ING/DERM/ING	Chlordane/Chlordane/Chlordane, Heptachlor epoxide
B(a)A, B(a)P, B(b)F, B(k)F, Chrys, Indeno, As/B(a)A, B(a)P B(a)F, B(k)F, Chrys, Dibenz, Dieldrin, Indeno, Arsenic	YES(3.4)	SOIL	DERM	Lead, Thallium
B(a)A, B(b)F, B(a)P, B(k)F, Chrys, Dibenz, Indeno, Arsenic/ B(a)A, B(a)F, B(a)P, B(k)F, Chrys, Dibenz, Indeno, Dieldrin, Arsenic/Arsenic	YES(18.5)	SOIL	DERM	Lead, Thallium
B(a)A, B(a)P, B(b)F, B(k)F, Chrys, Indeno, As/B(a)A, B(a)P B(a)F, B(k)F, Chrys, Dibenz, Dieldrin, Indeno, Arsenic	YES(3.4)	SOIL	DERM	Lead, Thallium
B(a)A, B(b)F, B(a)P, B(k)F, Chrys, Dibenz, Indeno, Arsenic/ B(a)A, B(a)F, B(a)P, B(k)F, Chrys, Dibenz, Indeno, Dieldrin, Arsenic/Arsenic	YES(18.5)	SOIL	DERM	Lead, Thallium
B(a)A, B(b)F, B(a)P, B(k)F, Chrys, Indeno, As/B(a)A, B(b)F, B(a)P, B(k)F, Chrys, Indeno, Dibenz, Dieldrin, Arsenic/Arsenic/ Dieldrin	YES(4.9)	SOIL	DERM	Lead, Thallium
B(a)A, B(a)F, B(a)P, B(k)F, Chrys, Indeno, Dibenz, Dieldrin, Arsenic/B(a)A, B(a)F, B(a)P, B(k)F, Chrys, Indeno, Dibenz, Dieldrin, Arsenic/Arsenic/Dieldrin	YES(26.4)	SOIL	ING/DERM	Thallium/Lead, Thallium
B(a)A, B(a)F, B(a)P, B(k)F, Chrys, Indeno, Dibenz, Dieldrin, Arsenic/B(a)F, B(a)A, B(a)P, B(k)F, Chrys, Indeno, Dibenz, Dieldrin, Arsenic/Arsenic/Dieldrin	YES(67.8)	SOIL	ING/DERM	Arsenic, Lead, Thallium/Cadmium, Lead, Thallium

TABLE R-38(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	>1.0E-05	>1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WIT EXCESS
30	SWMU 12	52	FUT AD WKER-ST	YES	YES(2E-5)	NO	SOIL	DERM	B(a)A
			FUT AD WKER-LT	YES	YES(9E-5)	NO	SOIL	ING/DERM/INH	B(a)A/B(a)A, DE
			EX AD WKER-ST	YES	YES(2E-5)	NO	SOIL	DERM	B(a)A
			EX AD WKER-LT	YES	YES(9E-5)	NO	SOIL	ING/DERM/INH	B(a)A/B(a)A, DE
			FUT RES AD-ST	YES	YES(3E-5)	NO	SOIL/VEG	ING/DERM/INH/ ING	B(a)A/B(a)A, DE DEHP
			FUT RES AD-LT	YES	YES	YES(2E-4)	SOIL/VEG	ING/DERM/INH/ ING	B(a)A/B(a)A, DE DEHP
			FUT RES CH	YES	YES(9E-5)	NO	SOIL/VEG	ING/DERM/INH/ ING	B(a)A/B(a)A, DE DEHP
31	BUILDING 223	53	FUT RES AD-ST	NO	NO	NO			
			FUT RES AD-LT	NO	NO	NO			
			FUT RES CH	NO	NO	NO			
32	FACILITY-WIDE ACTIONS (STREAMS)	54	FUT RES AD-ST	YES	YES(5E-6)	NO	SED	DERM	Be
			FUT RES AD-LT	YES	YES(3E-6)	NO	SED	DERM	B(a)A, Chrys, As
			FUT RES CH	YES(2E-5)	NO	NO	SED	DERM	B(a)A, Chrys, As
33	SWMU 22	55	EX AD WKER-ST	YES	YES(5E-5)	NO	SOIL	DERM	B(b)F, B(a)A, B(k
			EX AD WKER-LT	YES	YES	YES(3E-4)	SOIL	ING/DERM	B(b)F, B(a)A, B(k
			FUT AD WKER-ST	YES	YES(5E-5)	NO	SOIL	DERM	B(b)F, B(a)A, B(k
			FUT AD WKER-LT	YES	YES	YES(3E-4)	SOIL	ING/DERM	B(b)F, B(a)A, B(k
			FUT RES AD-ST	YES	YES(8E-5)	NO	SOIL	ING/DERM	B(b)F, B(a)A, B(k
			FUT RES AD-LT	YES	YES	YES(5E-4)	SOIL	ING/DERM	B(b)F, B(a)A, B(k
			FUT RES CH	YES	YES	YES(2E-4)	SOIL	ING/DERM	B(b)F, B(a)A, B(k
34	AREA OF CONCERN 2	56	FUT RES AD-ST	NO	NO	NO			
			FUT RES AD-LT	NO	NO	NO			
			FUT RES CH	NO	NO	NO			

B(cont'd)
IZATION RESULTS FOR SOIL
ASS ARMY DEPOT

RE G	CHEMICALS ASSOC WITH EXCESS	TOTAL HAZARD > 1.0	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
	B(a)A	YES(10.0)	SOIL	DERM	Lead, Silver
NH	B(a)A/B(a)A, DEHP/Cadmium	YES (47.6)	SOIL	ING/DERM	Lead, Cadmium/Cadmium, Lead, Silver
	B(a)A	YES(10.0)	SOIL	DERM	Lead, Silver
NH	B(a)A/B(a)A, DEHP/Cadmium	YES (47.6)	SOIL	ING/DERM	Lead, Cadmium/Cadmium, Lead, Silver
NH/	B(a)A/B(a)A, DEHP/Cadmium/ DEHP	YES(14.9)	SOIL	ING/DERM	Lead/Lead, Silver
NH/	B(a)A/B(a)A, DEHP/Cadmium/ DEHP	YES(68.2)	SOIL	ING/DERM	Lead, Cadmium/Cadmium, Lead, Silver
NH/	B(a)A/B(a)A, DEHP/Cadmium/ DEHP	YES(175)	SOIL	ING/DERM	Lead, Cadmium/Cadmium, Lead, Silver
		NO			
		NO			
		NO			
	Be	NO			
	B(a)A, Chrys, As, Be	YES(1.4)	SED	DERM	Thallium
	B(a)A, Chrys, As, Be	YES(5.6)	SED	DERM	Manganese, Thallium, Vanadium
	B(b)F, B(a)A, B(k)F, Chrys	NO			
	B(b)F, B(a)A, B(k)F, Chrys/B(b)F, B(a)A, B(k)F, Chrys	NO			
	B(b)F, B(a)A, B(k)F, Chrys	NO			
	B(b)F, B(a)A, B(k)F, Chrys/B(b)F, B(a)A, B(k)F, Chrys	NO			
	B(b)F, B(a)A, B(k)F, Chrys/B(b)F, B(a)A, B(k)F, Chrys	NO			
	B(b)F, B(a)A, B(k)F, Chrys/B(b)F, B(a)A, B(k)F, Chrys	YES(1.5)	SED	DERM	Cadmium
	B(b)F, B(a)A, B(k)F, Chrys/B(b)F, B(a)A, B(k)F, Chrys	YES(5.3)	SOIL/SED	DERM/DERM	Cadmium/Cadmium
		NO			
		NO			
		NO			

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TABLE R-38(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS F
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	>1.0E-05	>1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
35	GOLF COURSE	57	FUT RES AD-ST	YES(1E-6)	NO	NO	SOIL	DERM	B(a)A, B(k)F, Chrys
			FUT RES AD-LT	YES(8E-6)	NO	NO	SOIL	DERM	B(a)A, B(k)F, Chrys
			FUT RES CH	YES(4E-6)	NO	NO	SOIL	ING/DERM	B(a)A, B(k)F, Chrys
			FUT GOLFER-ST	NO	NO	NO			
			FUT GOLFER-LT	YES(1E-6)	NO	NO	SOIL	DERM	B(a)A, B(k)F, Chrys
			FUT SWIM AD-ST	NO	NO	NO			
			FUT SWIM AD-LT	YES(4E-6)	NO	NO	SWAT	DERM	Bis(2-ethylhexyl)p
			FUT SWIM CH	YES (1E-6)	NO	NO	SWAT	DERM	Bis(2-ethylhexyl)p
			FUT FISH AD-ST	YES	YES (3E-5)	NO	FISH	ING	Bis(2-ethylhexyl)p
			FUT FISH AD-LT	YES	YES	YES (2E-4)	FISH	ING	Bis(2-ethylhexyl)p
			FUT FISH CH	YES	YES (4E-5)	NO	FISH	ING	Bis(2-ethylhexyl)p
36	TELEPHONE POLE STORAGE AREA	58	FUT AD WKER-ST	YES	YES (2E-5)	NO	SOIL	ING/DERM	As/B(a)A, Chrys, P
			FUT AD WKER-LT	YES	YES(8E-5)	NO	SOIL	ING/DERM	PCB 1260, Arsenic
			EX AD WKER-ST	YES	YES (2E-5)	NO	SOIL	ING/DERM	As/B(a)A, Chrys, P
			EX AD WKER-LT	YES	YES(8E-5)	NO	SOIL	ING/DERM	PCB 1260, Arsenic
			FUT RES AD-ST	YES	YES(3E-5)	NO	SOIL/VEG/BEEF	ING/DERM/ING/ ING	As/B(a)A, Chrys, P
			FUT RES AD-LT	YES	YES	YES(2E-4)	SOIL/VEG/BEEF	ING/DERM/INH/ ING/ING	B(a)A, Chrys, PCB As/Cadmium, Nick
			FUT RES CH	YES	YES	YES(1E-4)	SOIL/VEG/BEEF	ING/DERM/INH/ ING/ING	B(a)A, Chrys, PCB As/Cadmium, Nick

* Number enclosed in parenthesis indicates the value of the risk or hazard

B[A]P - Benzo[A]Pyrene
B[B]F - Benzo[B]Fluoranthene
B[A]A - Benzo[A]Anthracene
Chrys - Chrysene
Indeno - Indeno[1,2,3-CD]Pyrene
Dibenz - Dibenz[A,H]Anthracene
B[K]F - Benzo[K]Fluoranthene
DEHP - bis(2-ethylhexyl)phthalate

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ON RESULTS FOR SOIL
ARMY DEPOT

CHEMICALS ASSOC WITH EXCESS	TOTAL HAZARD > 1.0	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
B(a)A, B(k)F, Chrys	NO			
B(a)A, B(k)F, Chrys	NO			
B(a)A, B(k)F, Chrys/B(a)A, B(k)F, Chrys	YES(2.7)	SED	DERM	Thallium
	NO			
B(a)A, B(k)F, Chrys	NO			
	NO			
Bis(2-ethylhexyl)phthalate, Beryllium	NO			
Bis(2-ethylhexyl)phthalate, Beryllium	NO			
Bis(2-ethylhexyl)phthalate, Beryllium	YES (3.0)	FISH	ING	Antimony
Bis(2-ethylhexyl)phthalate, Beryllium	YES (3.0)	FISH	ING	Antimony
Bis(2-ethylhexyl)phthalate, Beryllium	YES (5.3)	FISH	ING	Antimony
As/B(a)A, Chrys, PCB1260, As	YES(7.5)	SOIL	DERM	Lead
PCB 1260, Arsenic/B(a)A, DEHP, Chrys, PCB1260, As	YES(23.8)	SOIL	DERM	Cadmium, Lead
As/B(a)A, Chrys, PCB1260, As	YES(7.5)	SOIL	DERM	Lead
PCB 1260, Arsenic/B(a)A, DEHP, Chrys, PCB1260, As	YES(23.8)	SOIL	DERM	Cadmium, Lead
As/B(a)A, Chrys, PCB1260, As/PCB 1260/PCB 1260	YES(11.2)	SOIL	ING/DERM	Lead/Lead
B(a)A, Chrys, PCB1260, As/B(a)A, DEHP, Chrys, PCB1260, As/Cadmium, Nickel/PCB 1260, DEHP/PCB 1260	YES(35.0)	SOIL	ING/DERM	Lead/Cadmium, Lead
B(a)A, Chrys, PCB1260, As/B(a)A, DEHP, Chrys, PCB1260, As/Cadmium, Nickel/PCB 1260, DEHP/PCB 1260	YES(93.2)	SOIL/BEEF	ING/DERM/ING	Antimony, Cadmium, Lead/Antimony, Cadmium, Lead/Chlordane

TABLE R-39
SUMMARY OF RISK CHARACTERIZATION RESULTS FOR SOIL
BASED ON THE TOXICITY EQUIVALENCY FACTOR (TEF) FOR CARCINOGEN
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	TOTAL RISK >1.0E-05	TOTAL RISK >1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS
1	SWMU 4	23	FUT RES AD-ST	YES	YES (3E-5)	NO	SOIL	ING/DERM/INH
			FUT RES AD-LT	YES	YES	YES (2E-4)	SOIL	ING/DERM/INH
			FUT RES CH	YES	YES	YES (1E-4)	SOIL	ING/DERM/INH
2	SWMU 2, 5, 6, AND 7	24	FUT RES AD-ST	YES	YES	YES (4E-4)	SOIL/SED	ING/DERM/DERM
			FUT RES AD-LT	YES	YES	YES (2E-3)	SOIL/SED	ING/DERM/INH DERM
			FUT RES CH	YES	YES	YES (1E-3)	SOIL/SED	ING/DERM/DERM
3	SWMU 1	25	FUT RES AD-ST	NO	NO	NO		
			FUT RES AD-LT	NO	NO	NO		
			FUT RES CH	NO	NO	NO		
4	AREA A	26	FUT RES AD-ST	YES (3E-6)	NO	NO	SED	DERM
			FUT RES AD-LT	YES	YES (1E-5)	NO	SED	DERM
			FUT RES CH	YES	YES (1E-5)	NO	SED	DERM
5	AREA B	27	FUT RES AD-ST	YES	YES	YES (1E-3)	SOIL	ING/DERM
			FUT RES AD-LT	YES	YES	YES (1E-2)	SOIL	ING/DERM
			FUT RES CH	YES	YES	YES (4E-3)	SOIL	ING/DERM
6	AREA C	28	FUT RES AD-ST	NO	NO	NO		
			FUT RES AD-LT	YES(2E-6)	NO	NO	SED	DERM
			FUT RES CH	YES(1E-6)	NO	NO	SED	DERM

TS FOR SOIL
R CARCINOGENIC PAHs
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EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
ING/DERM/INH	Arsenic/B[A]P, Indeno, Arsenic/Arsenic
ING/DERM/INH	Arsenic, B[A]P, Indeno/
	Arsenic, B[A]P, B[B]F, Indeno, PCB 1260/Arsenic
ING/DERM/INH	Arsenic, B[A]P, Indeno/
	Arsenic, B[A]P, Indeno/Arsenic
ING/DERM/DERM	B[A]A, B[A]P, B[B]F, Indeno/B[A]A,
	B[A]P, B[B]F, Dibenz/B[B]F
	B[A]A, B[A]P, Indeno, Arsenic
ING/DERM/INH	B[A]A, B[A]P,
DERM	B[B]F, Dibenz, Indeno/B[A]P, B[B]F, B[k]F, Chrys
	Dibenz, B[A]A, Indeno, PCB 1260/Cadmium, Nickel/B[A]P,
	B[B]F, Arsenic
ING/DERM/DERM	B[A]A, B[A]P, B[B]F, Dibenz, Indeno/
	B[A]A, B[A]P, B[K]F, Chrys, B[B]F, Dibenz, Indeno/
	B[A]A, B[A]P, B[B]F, Dibenz, Indeno, Arsenic
DERM	B[A]P
DERM	B[A]A, B[A]P, B[B]F, Indeno, B[K]F
DERM	B[A]A, B[A]P, B[B]F
ING/DERM	B[A]A, B[B]F, B[K]F, Indeno, Dibenz/
	B[A]A, B[B]F, B[K]F, Dibenz, Indeno, Chrys
ING/DERM	B[A]A, B[B]F, B[K]F, Chrys, Indeno, Dibenz/
	B[A]A, B[B]F, B[K]F, Dibenz, Indeno, Chrys, Dieldrin
ING/DERM	B[A]A, B[B]F, B[K]F, Chrys, Indeno, Dibenz/
	B[A]A, B[B]F, B[K]F, Dibenz, Indeno, Chrys
DERM	B[B]F
DERM	B[B]F

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TABLE R-39(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS FOR SOIL
BASED ON THE TOXICITY EQUIVALENCY FACTOR (TEF) FOR CARCINOGENIC PAHs
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	TOTAL RISK >1.0E-05	TOTAL RISK >1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMIC ASSOC EXCESS
7	SWMU 3	29	EX AD WKR-ST	NO	NO	NO			
			EX AD WKR-LT	YES (1E-6)	NO	NO	SOIL	DERM	PCB 1260
			FUT AD WKR-ST	NO	NO	NO			
			FUT AD WKR-LT	YES (1E-6)	NO	NO	SOIL	DERM	Be[A]A, B[I
			FUT RES AD-ST	YES	YES (2E-5)	NO	SED	DERM	PCB 1260,
			FUT RES AD-LT	YES	YES	YES (1E-4)	SOIL/SED/SWAT	DERM/DERM/DERM	PCB 1260/I
									Bis(2-ethy
			FUT RES CH	YES	YES (7E-5)	NO	SED/SWAT	DERM/DERM	PCB 1260,
									Bis(2-ethy
8	SWMU 24	30	EX AD WKER-ST	YES	YES (3E-5)	NO	SOIL	ING/DERM	Arsenic/B[A
			EX AD WKER-LT	YES	YES	YES (2E-4)	SOIL	ING/DERM/INH	Arsenic/B[A
									bis(2-ethy
			FUT AD WKER-ST	YES	YES (3E-5)	NO	SOIL	ING/DERM	Arsenic/B[A
			FUT AD WKER-LT	YES	YES	YES (2E-4)	SOIL	ING/DERM/INH	Arsenic/B[A
									bis(2-ethy
			FUT RES AD-ST	YES	YES (6E-5)	NO	SOIL	ING/DERM/INH	Arsenic/B[A
			FUT RES AD-LT	YES	YES	YES (4E-4)	SOIL	ING/DERM/INH	B[A]A, Arse
									bis(2-ethy
			FUT RES CH	YES	YES	YES (2E-4)	SOIL	ING/DERM/INH	B[A]A, Arse
									Arsenic, bis
	BUILDING 3		EX AD WKER-ST	YES	YES(3E-5)	NO	SOIL	ING/DERM	B[A]P/B[A]
			EX AD WKER-LT	YES	YES	YES (2E-4)	SOIL	ING/DERM	B[A]P/B[A]
			FUT AD WKER-ST	YES	YES(3E-5)	NO	SOIL	ING/DERM	B[A]P/B[A]
			FUT AD WKER-LT	YES	YES	YES (2E-4)	SOIL	ING/DERM	B[A]P/B[A]
			FUT RES AD-ST	YES	YES(5E-5)	NO	SOIL	ING/DERM	B[A]P/B[A]
			FUT RES AD-LT	YES	YES	YES (3E-4)	SOIL	ING/DERM	B[A]A, B[A]
									B[A]A, B[A]
			FUT RES CH	YES	YES	YES (1E-4)	SOIL	ING/DERM	B[A]A, B[A]
									Dibenz/B[A
	BUILDING 63		EX AD WKER-ST	YES	YES(5E-5)	NO	SOIL	ING/DERM	Arsenic/Ars
			EX AD WKER-LT	YES	YES	YES (3E-4)	SOIL	ING/DERM	Arsenic, B[E
									Arsenic, B[A
			FUT AD WKER-ST	YES	YES(5E-5)	NO	SOIL	ING/DERM	Arsenic/Ars
			FUT AD WKER-LT	YES	YES	YES (3E-4)	SOIL	ING/DERM	Arsenic, B[E
									Arsenic, B[A
			FUT RES AD-ST	YES	YES(8E-5)	NO	SOIL	ING/DERM	Arsenic, B[A
			FUT RES AD-LT	YES	YES	YES (5E-4)	SOIL	ING/DERM/INH	Arsenic, B[A
									Arsenic, B[A
									bis(2-ethyl
			FUT RES CH	YES	YES	YES (3E-4)	SOIL	ING/DERM/INH	Arsenic, B[A
									bis(2-ethyl

TS FOR SOIL
R CARCINOGENIC PAHs
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EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
DERM	PCB 1260
DERM	Be[A]A, B[K]F, Chrys, PCB 1260
DERM	PCB 1260, Arsenic
DERM/DERM/DERM	PCB 1260/B[B]F, PCB 1260, Arsenic/ Bis(2-ethylhexyl)phthalate, Arsenic, Beryllium, B[B]F
DERM/DERM	PCB 1260, Arsenic/ Bis(2-ethylhexyl)phthalate, Beryllium
ING/DERM	Arsenic/B[A]A, Arsenic
ING/DERM/INH	Arsenic/B[A]A, Arsenic bis(2-ethylhexyl)phthalate/Arsenic
ING/DERM	Arsenic/B[A]A, Arsenic
ING/DERM/INH	Arsenic/B[A]A, Arsenic bis(2-ethylhexyl)phthalate/Arsenic
ING/DERM/INH	Arsenic/B[A]P, Arsenic/Arsenic
ING/DERM/INH	B[A]A, Arsenic/B[A]A, Arsenic, bis(2-ethylhexyl)phthalate/Arsenic
ING/DERM/INH	B[A]A, Arsenic/B[A]A, Arsenic, bis(2-ethylhexyl)phthalate/Arsenic
ING/DERM	B[A]P/B[A]A,B[A]P, B[B]F, Dibenz
ING/DERM	B[A]P/B[A]A, B[A]P, B[B]F, B[K]F, Dibenz
ING/DERM	B[A]P/B[A]A,B[A]P, B[B]F, Dibenz
ING/DERM	B[A]P/B[A]A, B[A]P, B[B]F, B[K]F, Dibenz
ING/DERM	B[A]P/B[A]A, B[A]P, B[B]F, B[K]F, Dibenz
ING/DERM	B[A]A, B[A]P, B[B]F, Dibenz/ B[A]A, B[A]P, B[B]F, B[K]F, Dibenz
ING/DERM	B[A]A, B[A]P, B[B]F Dibenz/B[A]A, B[A]P, B[B]F, Dibenz
ING/DERM	Arsenic/Arsenic, B[A]A, B[B]F
ING/DERM	Arsenic, B[B]F/ Arsenic, B[A]A, B[B]F, Chrys, bis(2-ethylhexyl)phthalate, Dieldrin
ING/DERM	Arsenic/Arsenic, B[A]A, B[B]F
ING/DERM	Arsenic, B[B]F/ Arsenic, B[A]A, B[B]F, Chrys, bis(2-ethylhexyl)phthalate, Dieldrin
ING/DERM	Arsenic, B[A]A, B[B]F/Arsenic, B[A]A, B[B]F
ING/DERM/INH	Arsenic, B[A]A, B[B]F/ Arsenic, B[A]A, B[B]F, DDT bis(2-ethylhexyl)phthalate, Dieldrin/Arsenic
ING/DERM/INH	Arsenic, B[A]A, B[B]F/Arsenic, B[A]A, B[B]F/ bis(2-ethylhexyl)phthalate, Dieldrin/Arsenic

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TABLE R-39(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS FOR SOI
BASED ON THE TOXICITY EQUIVALENCY FACTOR (TEF) FOR CARCINO
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	>1.0E-05	>1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSUF ROUTE CAUSING EXCESS
13	BUILDING 10	31	EX AD WKER-ST	YES	YES (6E-5)	NO	SOIL	ING/DERM
			EX AD WKER-LT	YES	YES	YES (3E-4)	SOIL	ING/DERM
			FUT AD WKER-ST	YES	YES (6E-5)	NO	SOIL	ING/DERM
			FUT AD WKER-LT	YES	YES	YES (3E-4)	SOIL	ING/DERM
			FUT RES AD-ST	YES	YES(9E-5)	NO	SOIL	ING/DERM
			FUT RES AD-LT	YES	YES	YES (5E-4)	SOIL	ING/DERM/IN
			FUT RES CH	YES	YES	YES (2E-4)	SOIL	ING/DERM/IN
	BUILDING 130		EX AD WKER-ST	YES(4E-6)	NO	NO	SOIL	DERM
			EX AD WKER-LT	YES	YES(2E-5)	NO	SOIL	ING/DERM
			FUT AD WKER-ST	YES(4E-6)	NO	NO	SOIL	DERM
			FUT AD WKER-LT	YES	YES(2E-5)	NO	SOIL	ING/DERM
			FUT RES AD-ST	YES(5E-6)	NO	NO	SOIL	DERM
			FUT RES AD-LT	YES	YES(3E-5)	NO	SOIL	ING/DERM
			FUT RES CH	YES	YES (2E-5)	NO	SOIL	ING/DERM
	SWMU 23		EX AD WKER-ST	YES	YES(2E-5)	NO	SOIL	DERM
			EX AD WKER-LT	YES	YES(8E-5)	NO	SOIL	ING/DERM/INH
			FUT AD WKER-ST	YES	YES(2E-5)	NO	SOIL	DERM
			FUT AD WKER-LT	YES	YES(8E-5)	NO	SOIL	ING/DERM/INH
			FUT RES AD-ST	YES	YES(3E-5)	NO	SOIL	ING/DERM/INH
			FUT RES AD-LT	YES	YES	YES(2E-4)	SOIL	ING/DERM/INH
			FUT RES CH	YES	YES(8E-5)	NO	SOIL	ING/DERM/INH
24	SWMU 18 AND 19	36	EX AD WKER-ST	NO	NO	NO		
			EX AD WKER-LT	NO	NO	NO		
			FUT AD WKER-ST	NO	NO	NO		
			FUT AD WKER-LT	NO	NO	NO		
			FUT RES AD-ST	NO	NO	NO		
			FUT RES AD-LT	YES(1E-6)	NO	NO	SED	DERM
			FUT RES CH	NO	NO	NO		

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TION RESULTS FOR SOIL
OR (TEF) FOR CARCINOGENIC PAHs
S ARMY DEPOT

MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
SOIL	ING/DERM	Arsenic/Arsenic, Beryllium
SOIL	ING/DERM	Arsenic, Beryllium/Arsenic, Beryllium
SOIL	ING/DERM	Arsenic/Arsenic, Beryllium
SOIL	ING/DERM	Arsenic, Beryllium/Arsenic, Beryllium
SOIL	ING/DERM	Arsenic, Beryllium/Arsenic, Beryllium
SOIL	ING/DERM/INH	Arsenic, Beryllium/ Arsenic, Beryllium, B[A]A/Arsenic
SOIL	ING/DERM/INH	Arsenic, Beryllium/ Arsenic, Beryllium/Arsenic
SOIL	DERM	B[B]F, PCB 1260
SOIL	ING/DERM	PCB 1260, B[B]F/B[A]A, B[B]F bis(2-ethylhexyl)phthalate, PCB 1260
SOIL	DERM	B[B]F, PCB 1260
SOIL	ING/DERM	PCB 1260, B[B]F/B[A]A, B[B]F bis(2-ethylhexyl)phthalate, PCB 1260
SOIL	DERM	B[B]F, PCB 1260
SOIL	ING/DERM	PCB 1260/ B[A]A, B[B]F, PCB 1260, bis(2-ethylhexyl)phthalate
SOIL	ING/DERM	B[B]F, PCB 1260/B[A]A, B[B]F, PCB 1260
SOIL	DERM	B[A]A, B[B]F
SOIL	ING/DERM/INH	B[A]A, B[B]F/B[A]A, B[B]F, Dieldrin/Cadmium
SOIL	DERM	B[A]A, B[B]F
SOIL	ING/DERM/INH	B[A]A, B[B]F/B[A]A, B[B]F, Dieldrin/Cadmium
SOIL	ING/DERM/INH	B[B]F,/B[A]A, B[B]F, Dieldrin/Cadmium
SOIL	ING/DERM/INH	B[A]A, B[B]F, Dieldrin/B[A]A, B[B]F, Dieldrin/Cadmium
SOIL	ING/DERM/INH	B[A]A, B[B]F, Dieldrin/B[A]A, B[B]F, Dieldrin/Cadmium
SED	DERM	B[A]A, B[B]F

TABLE R-39(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS FOR SOIL
BASED ON THE TOXICITY EQUIVALENCY FACTOR (TEF) FOR CARCINOGENIC P
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	>1.0E-05	>1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS	C A E
27	SWMU 9	38	EX AD WKER-ST	YES	YES	YES (6E-4)	SOIL	ING/DERM	B[A
			EX AD WKER-LT	YES	YES	YES(3E-3)	SOIL	ING/DERM	B[A
			FUT AD WKER-ST	YES	YES	YES (6E-4)	SOIL	ING/DERM	B[A
			FUT AD WKER-LT	YES	YES	YES(3E-3)	SOIL	ING/DERM	B[A
			FUT RES AD-ST	YES	YES	YES(1E-3)	SOIL	ING/DERM	B[A
			FUT RES AD-LT	YES	YES	YES(6E-3)	SOIL	ING/DERM	B[A
			FUT RES CH	YES	YES	YES(3E-3)	SOIL	ING/DERM	B[A
33	COAL PILE RUNOFF/ HEATING PLANT AREA	42	EX AD WKER-ST	YES	YES	YES(3E-4)	SOIL	ING/DERM/INH	Arse
			EX AD WKER-LT	YES	YES	YES (1E-3)	SOIL	ING/DERM/INH	Arse
			FUT AD WKER-ST	YES	YES	YES(3E-4)	SOIL	ING/DERM/INH	Arse
			FUT AD WKER-LT	YES	YES	YES (1E-3)	SOIL	ING/DERM/INH	Arse
			FUT RES AD-ST	YES	YES	YES(4E-4)	SOIL	ING/DERM/INH	Bery
			FUT RES AD-LT	YES	YES	YES(3E-3)	SOIL	ING/DERM/INH	Arse
			FUT RES CH	YES	YES	YES(2E-3)	SOIL	ING/DERM/INH	Arse
34	SWMU 10	43	EX AD WKER-ST	YES	YES(2E-5)	NO	SOIL	ING/DERM/INH	Arse
			EX AD WKER-LT	YES	YES	YES(1E-4)	SOIL	ING/DERM/INH	PCB
			FUT AD WKER-ST	YES	YES(2E-5)	NO	SOIL	ING/DERM/INH	Arse
			FUT AD WKER-LT	YES	YES	YES(1E-4)	SOIL	ING/DERM/INH	PCB
			FUT RES AD-ST	YES	YES(4E-5)	NO	SOIL	ING/DERM/INH	Arse
			FUT RES AD-LT	YES	YES	YES(3E-4)	SOIL	ING/DERM/INH	PCB
			FUT RES CH	YES	YES	YES(2E-4)	SOIL	ING/DERM/INH	PCB

ULTS FOR SOIL
FOR CARCINOGENIC PAHs
EPOT

SS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
	ING/DERM	B[A]A, B[A]P, B[B]F, Dibenz, Indeno, PCB 1260/
		B[A]A, B[A]P, B[B]F, Dibenz, Indeno, PCB 1260
	ING/DERM	B[A]A, B[A]P, B[B]F, Dibenz, Indeno, PCB 1260/
		B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB 1260
	ING/DERM	B[A]A, B[A]P, B[B]F, Dibenz, Indeno, PCB 1260/
		B[A]A, B[A]P, B[B]F, Dibenz, Indeno, PCB 1260
	ING/DERM	B[A]A, B[A]P, B[B]F, Dibenz, Indeno, PCB 1260/
		B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB 1260
	ING/DERM	B[A]A, B[A]P, B[B]F, Dibenz, Indeno, PCB1260/
		B[A]A, B[A]P, B[B]F, Dibenz, Indeno, PCB1260
	ING/DERM	B[A]A, B[A]P, B[B]F, Dibenz, Indeno, PCB1260/
		B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB1260, DDT
	ING/DERM	B[A]A, B[A]P, B[B]F, Dibenz, Indeno, PCB1260/
		B[A]A, B[A]P, B[B]F, Chrys, Dibenz, Indeno, PCB1260
	ING/DERM/INH	Arsenic/B(a)A, B(b)F, Arsenic, Beryllium/Arsenic
	ING/DERM/INH	Arsenic, Beryllium/B(A)A, B(b)F,
		Aldrin, Dieldrin, Arsenic, Beryllium/Arsenic
	ING/DERM/INH	Arsenic/B(a)A, B(b)F, Arsenic, Beryllium/Arsenic
	ING/DERM/INH	Arsenic, Beryllium/Aldrin, B(a)A, B(b)F,
		Dieldrin, Arsenic, Beryllium/Arsenic
	ING/DERM/INH	Beryllium/Aldrin, B(a)A, B(b)F,
		Arsenic, Beryllium/Arsenic
	ING/DERM/INH	Aldrin, B(a)A, B(b)F, Arsenic, Beryllium/Aldrin, B(a)A, B(b)F,
		Dieldrin, Arsenic, Beryllium/Arsenic
	ING/DERM/INH	Aldrin, B(a)A, B(b)F, Dieldrin, Arsenic, Beryllium/Aldrin,
		B(a)A, B(b)F, Dieldrin, Arsenic, Beryllium/Arsenic
	ING/DERM/INH	Arsenic/PCB1260, Arsenic/Cd
	ING/DERM/INH	PCB1260, Arsenic/B(a)A, B(b)F,
		PCB 1260, Arsenic/Nickel, Cadmium
	ING/DERM/INH	Arsenic/PCB1260, Arsenic/Cd
	ING/DERM/INH	PCB1260, Arsenic/B(a)A, B(b)F,
		PCB 1260, Arsenic/Nickel, Cadmium
	ING/DERM/INH	Arsenic/B(b)F, PCB 1260, Arsenic/Cadmium
	ING/DERM/INH	PCB1260, Arsenic/B(a)A, B(b)F,
		PCB 1260, Arsenic/Arsenic, Nickel, Cadmium
	ING/DERM/INH	PCB1260, Arsenic/B(a)A, B(b)F,
		PCB 1260, Arsenic/Arsenic, Nickel, Cadmium

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TABLE R-39(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS FOR SOIL
BASED ON THE TOXICITY EQUIVALENCY FACTOR (TEF) FOR CARCINOGE
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	>1.0E-05	>1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS
39	OPEN STORAGE AND SHELTER AREAS	45	EX AD WKER-ST	YES	YES	YES(2E-4)	SOIL	ING/DERM
			EX AD WKER-LT	YES	YES	YES(1E-3)	SOIL	ING/DERM/INH
			FUT AD WKER-ST	YES	YES	YES(2E-4)	SOIL	ING/DERM
			FUT AD WKER-LT	YES	YES	YES(1E-3)	SOIL	ING/DERM/INH
			FUT RES AD-ST	YES	YES	YES(3E-4)	SOIL	ING/DERM/INH
			FUT RES AD-LT	YES	YES	YES(2E-3)	SOIL	ING/DERM/INH
			FUT RES CH	YES	YES	YES(1E-3)	SOIL	ING/DERM/INH
40	SWMU 12	46	EX AD WKER-ST	YES(3E-6)	NO	NO	SOIL	DERM
			EX AD WKER-LT	YES	YES(2E-5)	NO	SOIL	DERM/INH
			FUT AD WKER-ST	YES(3E-6)	NO	NO	SOIL	DERM
			FUT AD WKER-LT	YES	YES(2E-5)	NO	SOIL	DERM/INH
			FUT RES AD-ST	YES(6E-6)	NO	NO	SOIL	DERM/INH
			FUT RES AD-LT	YES	YES(3E-5)	NO	SOIL	ING/DERM/INH
			FUT RES CH	YES	YES(2E-5)	NO	SOIL	ING/DERM/INH
45	SWMU 22	48	EX AD WKER-ST	YES(3E-6)	NO	NO	SOIL	DERM
			EX AD WKER-LT	YES	YES(2E-5)	NO	SOIL	DERM
			FUT AD WKER-ST	YES(3E-6)	NO	NO	SOIL	DERM
			FUT AD WKER-LT	YES	YES(2E-5)	NO	SOIL	DERM
			FUT RES AD-ST	YES(4E-6)	NO	NO	SOIL	DERM
			FUT RES AD-LT	YES	YES(3E-5)	NO	SOIL	ING/DERM
			FUT RES CH	YES	YES(1E-5)	NO	SOIL	ING/DERM

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RESULTS FOR SOIL
EF) FOR CARCINOGENIC PAHs
Y DEPOT

IA OC XCESS	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
	ING/DERM	B(a)A, B(a)P, B(b)F, Indeno, As/B(a)A, B(a)P B(a)F, B(k)F, Dibenz, Dieldrin, Indeno, Arsenic
	ING/DERM/INH	B(a)A, B(b)F, B(a)P, Diberz, Indeno, Arsenic/B(a)A, B(a)F, B(a)P, B[K]F, Chrys, Diberz, Indeno, Dieldrin, Arsenic/Arsenic
	ING/DERM	B(a)A, B(a)P, B(b)F, Indeno, As/B(a)A, B(a)P B(a)F, B(k)F, Dibenz, Dieldrin, Indeno, Arsenic
	ING/DERM/INH	B(a)A, B(b)F, B(a)P, Diberz, Indeno, Arsenic/B(a)A, B(a)F, B(a)P, B[K]F, Chrys, Diberz, Indeno, Dieldrin, Arsenic/Arsenic
	ING/DERM/INH	B(a)A, B(b)F, B(a)P, Indeno, As/B(a)A, B(b)F, B(a)P, B(k)F, Chrys, Indeno, Dibenz, Dieldrin, Arsenic/Arsenic
	ING/DERM/INH	B(a)A, B(a)F, B(a)P, B(k)F, Dibenz, Dieldrin, Indeno, Arsenic/ B(a)A, B(a)F, B(a)P, B(k)F, Chrys, Indeno, Dibenz, Dieldrin, Arsenic/Arsenic
	ING/DERM/INH	B(a)A, B(a)F, B(a)P, B(k)F, Dibenz, Dieldrin, Indeno, Arsenic/ B(a)A, B(a)F, B(a)P, B(k)F, Indeno, Dibenz, Dieldrin, Arsenic/Arsenic
	DERM	B(a)A
	DERM/INH	B(a)A, DEHP/Cadmium
	DERM	B(a)A
	DERM/INH	B(a)A, DEHP/Cadmium
	DERM/INH	B(a)A, DEHP/Cadmium
	ING/DERM/INH	B(a)A/B(a)A, DEHP/Cadmium
	ING/DERM/INH	B(a)A/B(a)A, DEHP/Cadmium
	DERM	B(b)F, B(a)A,
	DERM	B(b)F, B(a)A,
	DERM	B(b)F, B(a)A,
	DERM	B(b)F, B(a)A,
	DERM	B(b)F, B(a)A,
	ING/DERM	B(b)F, B(a)A,/B(b)F, B(a)A,
	ING/DERM	B(b)F, B(a)A,/B(b)F, B(a)A,

TABLE R-39(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS FOR SOIL
BASED ON THE TOXICITY EQUIVALENCY FACTOR (TEF) FOR CARCINOGENI(
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RESULTS FROM RFI TABLE #	RECEPTOR	TOTAL RISK >1.0E-06	>1.0E-05	>1.0E-04	MEDIA ASSOC W. EXCESS	EXPOSURE ROUTE CAUSING EXCESS
48	GOLF COURSE	50	FUT RES AD-ST	NO	NO	NO		
			FUT RES AD-LT	NO	NO	NO		
			FUT RES CH	NO	NO	NO		
			FUT GOLFER-ST	NO	NO	NO		
			FUT GOLFER-LT	NO	NO	NO		
			FUT SWIM AD-ST	NO	NO	NO		
			FUT SWIM AD-LT	YES(4E-6)	NO	NO	SWAT	DERM
			FUT SWIM CH	YES (1E-6)	NO	NO	SWAT	DERM
			FUT FISH AD-ST	YES	YES (3E-5)	NO	FISH	ING
			FUT FISH AD-LT	YES	YES	YES (2E-4)	FISH	ING
			FUT FISH CH	YES	YES (4E-5)	NO	FISH	ING
49	TELEPHONE POLE STORAGE AREA	51	EX AD WKER-ST	YES	YES (1E-5)	NO	SOIL	ING/DERM
			EX AD WKER-LT	YES	YES(7E-5)	NO	SOIL	ING/DERM
			FUT AD WKER-ST	YES	YES (1E-5)	NO	SOIL	ING/DERM
			FUT AD WKER-LT	YES	YES(7E-5)	NO	SOIL	ING/DERM
			FUT RES AD-ST	YES	YES(2E-5)	NO	SOIL	ING/DERM
			FUT RES AD-LT	YES	YES	YES(1E-4)	SOIL	ING/DERM/INH
			FUT RES CH	YES	YES(7E-5)	NO	SOIL	ING/DERM/INH

* Number enclosed in parenthesis indicates the value of the risk or hazard

B[A]P – Benzo[A]Pyrene
B[B]F – Benzo[B]Fluoranthene
B[A]A – Benzo[A]Anthracene
Chrys – Chrysene
Indeno – Indeno[1,2,3-CD]Pyrene
Dibenz – Dibenz[A,H]Anthracene
B[K]F – Benzo[K]Fluoranthene

LTS FOR SOIL
)R CARCINOGENIC PAHs
)OT

S	EXPOSURE ROUTE CAUSING EXCESS	CHEMICALS ASSOC WITH EXCESS
	DERM	Bis(2-ethylhexyl)phthalate, Beryllium
	DERM	Bis(2-ethylhexyl)phthalate, Beryllium
	ING	Bis(2-ethylhexyl)phthalate, Beryllium
	ING	Bis(2-ethylhexyl)phthalate, Beryllium
	ING	Bis(2-ethylhexyl)phthalate, Beryllium
	ING/DERM	Arsenic/PCB 1260, Arsenic
	ING/DERM	PCB 1260, Arsenic/bis(2-ethylhexyl)phthalate, PCB 1260, Arsenic
	ING/DERM	Arsenic/PCB 1260, Arsenic
	ING/DERM	PCB 1260, Arsenic/bis(2-ethylhexyl)phthalate, PCB 1260, Arsenic
	ING/DERM	Arsenic/PCB 1260, Arsenic
	ING/DERM/INH	PCB 1260, Arsenic/B(a)A, bis(2-ethylhexyl)phthalate, PCB 1260, Arsenic/Cadmium, Nickel
	ING/DERM/INH	PCB 1260, Arsenic/bis(2-ethylhexyl)phthalate, PCB 1260, Arsenic/Cadmium, Nickel

TABLE R-40
SUMMARY OF RISK CHARACTERIZATION RESULTS
FOR SOIL AND GROUNDWATER COMBINED
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RECEPTOR	RISK			HAZARD		
			SOIL	GROUND WATER	TOTAL	SOIL	GROUND WATER	TOTAL
1	SWMU 4	FUT RES AD-ST	6E-5	1E-4	2E-4	5	56	60
		FUT RES AD-LT	3E-4	6E-4	9E-4	5	86	85
		FUT RES CH	2E-4	3E-4	4E-4	42	121	162
2	SWMU 2, 5, 6, AND 7	FUT RES AD-ST	2E-3	1E-4	2E-3	27	56	82
		FUT RES AD-LT	1E-2	6E-4	1E-2	40	86	120
		FUT RES CH	5E-3	3E-4	5E-3	137	121	257
3	SWMU 1	FUT RES AD-ST	ND	7E-5	6E-5	17	17	33
		FUT RES AD-LT	5E-6	3E-4	4E-4	31	63	84
		FUT RES CH	3E-6	2E-4	1E-4	102	36	137
4	AREA A	FUT RES AD-ST	2E-5	1E-4	1E-4	16	56	71
		FUT RES AD-LT	1E-4	6E-4	7E-4	16	86	96
		FUT RES CH	7E-5	3E-4	3E-4	60	121	180
5	AREA B	FUT RES AD-ST	2E-2	1E-4	2E-2	ND	56	55
		FUT RES AD-LT	1E-1	6E-4	1E-1	ND	86	80
		FUT RES CH	5E-2	3E-4	5E-2	2	121	122
6	AREA C	FUT RES AD-ST	3E-6	7E-5	6E-5	1	17	17
		FUT RES AD-LT	2E-5	3E-4	4E-4	1	63	54
		FUT RES CH	1E-5	2E-4	1E-4	3	36	38
7	SWMU 3	EX AD WKR-ST	ND	NA	ND	ND	NA	
		EX AD WKR-LT	3E-6	NA	3E-6	8	NA	8
		FUT AD WKR-ST	ND	5E-5	3E-5	ND	19	19
		FUT AD WKR-LT	5E-6	2E-4	2E-4	ND	30	28
		FUT RES AD-ST	2E-5	1E-4	1E-4	ND	56	55
		FUT RES AD-LT	1E-4	6E-4	7E-4	7	86	87
		FUT RES CH	8E-5	3E-4	3E-4	25	121	145
8	SWMU 24	EX AD WKER-ST	8E-5	NA	8E-5	1	NA	1
		EX AD WKER-LT	4E-4	NA	4E-4	2	NA	2
		FUT AD WKER-ST	8E-5	5E-5	1E-4	1	19	21
		FUT AD WKER-LT	4E-4	2E-4	6E-4	2	30	30
		FUT RES AD-ST	1E-4	1E-4	2E-4	2	56	57
		FUT RES AD-LT	8E-4	6E-4	1E-3	4	86	84
		FUT RES CH	4E-4	3E-4	6E-4	11	121	131
9	BUILDING 135	EX AD WKER-ST	ND	NA	ND	ND	NA	ND
		EX AD WKER-LT	ND	NA	ND	ND	NA	ND
		FUT AD WKER-ST	ND	5E-5	3E-5	ND	19	19
		FUT AD WKER-LT	ND	2E-4	2E-4	ND	30	28
		FUT RES AD-ST	ND	1E-4	9E-5	ND	56	55
		FUT RES AD-LT	ND	6E-4	6E-4	ND	86	80
		FUT RES CH	ND	3E-4	2E-4	ND	121	120
10	BUILDING 147	EX AD WKER-ST	ND	NA	NA	ND	NA	ND
		EX AD WKER-LT	ND	NA	NA	ND	NA	ND
		FUT AD WKER-ST	ND	5E-5	3E-5	ND	19	19
		FUT AD WKER-LT	ND	2E-4	2E-4	ND	30	28
		FUT RES AD-ST	ND	1E-4	9E-5	ND	56	55
		FUT RES AD-LT	ND	6E-4	6E-4	ND	86	80
		FUT RES CH	ND	3E-4	2E-4	ND	121	120
11	BUILDING 3	EX AD WKER-ST	1E-4	NA	1E-4	8	NA	8
		EX AD WKER-LT	6E-4	NA	6E-4	12	NA	12
		FUT AD WKER-ST	1E-4	5E-5	2E-4	8	19	27
		FUT AD WKER-LT	6E-4	2E-4	8E-4	12	30	40
		FUT RES AD-ST	2E-4	1E-4	3E-4	13	56	68
		FUT RES AD-LT	1E-3	6E-4	2E-3	13	86	93
		FUT RES CH	5E-4	3E-4	7E-4	40	121	160
12	BUILDING 10	EX AD WKER-ST	7E-5	NA	7E-5	11	NA	11
		EX AD WKER-LT	3E-4	NA	3E-4	12	NA	12
		FUT AD WKER-ST	7E-5	5E-5	1E-4	11	19	30
		FUT AD WKER-LT	3E-4	2E-4	5E-4	12	30	40
		FUT RES AD-ST	1E-4	1E-4	2E-4	17	56	72
		FUT RES AD-LT	6E-4	6E-4	1E-3	35	86	115
		FUT RES CH	3E-4	3E-4	5E-4	90	121	210

TABLE R-40(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS
FOR SOIL AND GROUNDWATER COMBINED
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RECEPTOR	RISK			HAZARD		
			SOIL	GROUND WATER	TOTAL	SOIL	GROUND WATER	TOTAL
13	BUILDING 19	EX AD WKER-ST	6E-5	NA	6E-5	5	NA	5
		EX AD WKER-LT	3E-4	NA	3E-4	54	NA	54
		FUT AD WKER-ST	6E-5	5E-5	9E-5	5	19	73
		FUT AD WKER-LT	3E-4	2E-4	5E-4	54	30	82
		FUT RES AD-ST	9E-5	1E-4	2E-4	78	56	133
		FUT RES AD-LT	5E-4	6E-4	1E-3	78	86	158
		FUT RES CH	2E-4	3E-4	4E-4	200	121	320
14	BUILDING 63	EX AD WKER-ST	2E-4	NA	2E-4	12	NA	12
		EX AD WKER-LT	9E-4	NA	9E-4	14	NA	14
		FUT AD WKER-ST	2E-4	5E-5	2E-4	12	19	31
		FUT AD WKER-LT	9E-4	2E-4	1E-3	14	30	42
		FUT RES AD-ST	3E-4	1E-4	4E-4	19	56	74
		FUT RES AD-LT	2E-3	6E-4	2E-3	20	86	100
		FUT RES CH	8E-4	3E-4	1E-3	58	121	177
15	BUILDING 64	EX AD WKER-ST	ND	NA	NA	ND	NA	NA
		EX AD WKER-LT	ND	NA	NA	ND	NA	NA
		FUT AD WKER-ST	ND	5E-5	3E-5	ND	19	19
		FUT AD WKER-LT	ND	2E-4	2E-4	ND	30	28
		FUT RES AD-ST	ND	1E-4	9E-5	ND	56	55
		FUT RES AD-LT	ND	6E-4	6E-4	ND	86	80
		FUT RES CH	ND	3E-4	2E-4	ND	121	120
16	BUILDING 130	EX AD WKER-ST	2E-5	NA	2E-5	7	NA	7
		EX AD WKER-LT	1E-4	NA	1E-4	7	NA	7
		FUT AD WKER-ST	2E-5	5E-5	5E-5	7	19	26
		FUT AD WKER-LT	1E-4	2E-4	3E-4	7	30	35
		FUT RES AD-ST	3E-5	1E-4	1E-4	10	56	65
		FUT RES AD-LT	2E-4	6E-4	7E-4	10	86	90
		FUT RES CH	9E-5	3E-4	3E-4	25	121	145
17	BUILDING 141	EX AD WKER-ST	ND	NA	ND	ND	NA	ND
		EX AD WKER-LT	ND	NA	ND	ND	NA	ND
		FUT AD WKER-ST	ND	5E-5	3E-5	ND	19	19
		FUT AD WKER-LT	ND	2E-4	2E-4	ND	30	28
		FUT RES AD-ST	ND	1E-4	9E-5	ND	56	55
		FUT RES AD-LT	ND	6E-4	6E-4	ND	86	80
		FUT RES CH	ND	3E-4	2E-4	ND	121	120
18	SWMU 23	EX AD WKER-ST	2E-4	NA	2E-4	1	NA	1
		EX AD WKER-LT	1E-3	NA	1E-3	16	NA	16
		FUT AD WKER-ST	2E-4	5E-5	2E-4	1	19	20
		FUT AD WKER-LT	1E-3	2E-4	1E-3	16	30	44
		FUT RES AD-ST	3E-4	1E-4	4E-4	2	56	57
		FUT RES AD-LT	2E-3	6E-4	2E-3	22	86	102
		FUT RES CH	8E-4	3E-4	1E-3	55	121	175
19	SWMU 18 AND 19	EX AD WKER-ST	ND	NA	ND	ND	NA	ND
		EX AD WKER-LT	ND	NA	ND	ND	NA	ND
		FUT AD WKER-ST	ND	5E-5	3E-5	ND	19	19
		FUT AD WKER-LT	ND	2E-4	2E-4	ND	30	28
		FUT RES AD-ST	4E-6	1E-4	1E-4	ND	56	55
		FUT RES AD-LT	3E-5	6E-4	6E-4	ND	86	80
		FUT RES CH	2E-5	3E-4	2E-4	ND	121	120
20	SWMU 16, 17, AND 30	EX AD WKER-ST	3E-6	NA	3E-6	2	NA	2
		EX AD WKER-LT	1E-5	NA	1E-5	3	NA	3
		FUT AD WKER-ST	3E-6	5E-5	4E-5	2	19	22
		FUT AD WKER-LT	1E-5	2E-4	2E-4	3	30	31
		FUT RES AD-ST	5E-6	1E-4	1E-4	3	56	58
		FUT RES AD-LT	3E-5	6E-4	6E-4	4	86	84
		FUT RES CH	2E-5	3E-4	2E-4	11	121	131
21	SWMU 9	EX AD WKER-ST	3E-3	NA	3E-3	2	NA	2
		EX AD WKER-LT	2E-2	NA	2E-2	4	NA	4
		FUT AD WKER-ST	3E-3	5E-5	3E-3	2	19	21
		FUT AD WKER-LT	2E-2	2E-4	2E-2	4	30	32
		FUT RES AD-ST	5E-3	1E-4	5E-3	3	56	58
		FUT RES AD-LT	3E-2	6E-4	3E-2	7	86	87
		FUT RES CH	1E-2	3E-4	1E-2	23	121	143

TABLE R-40(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS
FOR SOIL AND GROUNDWATER COMBINED
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RECEPTOR	RISK			HAZARD		
			SOIL	GROUND WATER	TOTAL	SOIL	GROUND WATER	TOTAL
22	BUILDING 42	EX AD WKER-ST	7E-5	NA	7E-5	18	NA	18
		EX AD WKER-LT	3E-4	NA	3E-4	42	NA	42
		FUT AD WKER-ST	7E-5	5E-5	1E-4	18	19	37
		FUT AD WKER-LT	3E-4	2E-4	5E-4	42	30	70
		FUT RES AD-ST	1E-4	1E-4	2E-4	28	56	83
		FUT RES AD-LT	6E-4	6E-4	1E-3	62	86	142
		FUT RES CH	3E-4	3E-4	5E-4	162	121	281
23	SWMU 20	EX AD WKER-ST	1E-5	NA	1E-5	8	NA	8
		EX AD WKER-LT	5E-5	NA	5E-5	8	NA	8
		FUT AD WKER-ST	1E-5	5E-5	4E-5	8	19	27
		FUT AD WKER-LT	5E-5	2E-4	2E-4	8	30	36
		FUT RES AD-ST	2E-5	1E-4	1E-4	12	56	67
		FUT RES AD-LT	1E-4	6E-4	7E-4	13	86	93
		FUT RES CH	6E-5	3E-4	3E-4	39	121	158
24	SWMU 25	EX AD WKER-ST	ND	NA	ND	ND	NA	ND
		EX AD WKER-LT	ND	NA	ND	ND	NA	ND
		FUT AD WKER-ST	ND	5E-5	3E-5	ND	19	19
		FUT AD WKER-LT	ND	2E-4	2E-4	ND	30	28
		FUT RES AD-ST	ND	1E-4	9E-5	ND	56	55
		FUT RES AD-LT	ND	6E-4	6E-4	ND	86	80
		FUT RES CH	ND	3E-4	2E-4	ND	121	120
25	SWMU 11	EX AD WKER-ST	ND	NA	ND	ND	NA	ND
		EX AD WKER-LT	2E-6	NA	2E-6	ND	NA	ND
		FUT AD WKER-ST	ND	5E-5	3E-5	ND	19	19
		FUT AD WKER-LT	2E-6	2E-4	2E-4	ND	30	28
		FUT RES AD-ST	ND	1E-4	9E-5	ND	56	55
		FUT RES AD-LT	4E-6	6E-4	6E-4	ND	86	80
		FUT RES CH	2E-6	3E-4	2E-4	ND	121	120
26	COAL PILE RUNOFF/ HEATING PLANT AREA	EX AD WKER-ST	3E-4	NA	3E-4	13	NA	13
		EX AD WKER-LT	2E-3	NA	2E-3	66	NA	66
		FUT AD WKER-ST	3E-4	5E-5	4E-4	13	19	32
		FUT AD WKER-LT	2E-3	2E-4	2E-3	67	30	95
		FUT RES AD-ST	5E-4	1E-4	6E-4	19	56	74
		FUT RES AD-LT	3E-3	6E-4	4E-3	95	86	175
		FUT RES CH	2E-3	3E-4	2E-3	251	121	370
27	SWMU 10	EX AD WKER-ST	4E-5	NA	4E-5	27	NA	27
		EX AD WKER-LT	2E-4	NA	2E-4	596	NA	596
		FUT AD WKER-ST	4E-5	5E-5	7E-5	27	19	46
		FUT AD WKER-LT	2E-4	2E-4	4E-4	596	30	624
		FUT RES AD-ST	8E-5	1E-4	2E-4	39	56	94
		FUT RES AD-LT	5E-4	6E-4	1E-3	846	86	926
		FUT RES CH	3E-4	3E-4	5E-4	2060	121	2180
28	BUILDING 303	FUT RES AD-ST	6E-5	7E-5	1E-4	9	17	25
		FUT RES AD-LT	3E-4	3E-4	7E-4	9	63	62
		FUT RES CH	2E-4	2E-4	3E-4	31	36	67
29	OPEN STORAGE AND SHELTER AREAS	EX AD WKER-ST	2E-3	NA	2E-3	3	NA	3
		EX AD WKER-LT	8E-3	NA	8E-3	18	NA	18
		FUT AD WKER-ST	2E-3	5E-5	2E-3	3	19	23
		FUT AD WKER-LT	8E-3	2E-4	9E-3	18	30	47
		FUT RES AD-ST	2E-3	1E-4	3E-3	5	56	60
		FUT RES AD-LT	2E-2	6E-4	2E-2	26	86	106
		FUT RES CH	7E-3	3E-4	7E-3	68	121	187
30	SWMU 12	EX AD WKER-ST	2E-5	NA	2E-5	10	NA	10
		EX AD WKER-LT	9E-5	NA	9E-5	48	NA	48
		FUT AD WKER-ST	2E-5	5E-5	5E-5	10	19	29
		FUT AD WKER-LT	9E-5	2E-4	3E-4	48	30	76
		FUT RES AD-ST	3E-5	1E-4	1E-4	15	56	70
		FUT RES AD-LT	2E-4	6E-4	7E-4	68	86	148
		FUT RES CH	9E-5	3E-4	3E-4	175	121	294
31	BUILDING 223	FUT RES AD-ST	ND	1E-4	9E-5	ND	56	55
		FUT RES AD-LT	ND	6E-4	6E-4	ND	86	80
		FUT RES CH	ND	3E-4	2E-4	ND	121	120

TABLE R-40(cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS
FOR SOIL AND GROUNDWATER COMBINED
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RECEPTOR	RISK			HAZARD		
			SOIL	GROUND WATER	TOTAL	SOIL	GROUND WATER	TOTAL
32	FACILITY-WIDE ACTIONS (STREAMS)	FUT RES AD-ST	5E-6	1E-4	1E-4	ND	56	55
		FUT RES AD-LT	3E-6	6E-4	6E-4	1	86	81
		FUT RES CH	2E-5	3E-4	2E-4	6	121	125
33	SWMU 22	EX AD WKER-ST	5E-5	NA	5E-5	ND	NA	ND
		EX AD WKER-LT	3E-4	NA	3E-4	ND	NA	ND
		FUT AD WKER-ST	5E-5	5E-5	8E-5	ND	19	19
		FUT AD WKER-LT	3E-4	2E-4	4E-4	ND	30	28
		FUT RES AD-ST	8E-5	1E-4	2E-4	ND	56	55
		FUT RES AD-LT	5E-4	6E-4	1E-3	1	86	81
		FUT RES CH	2E-4	3E-4	4E-4	5	121	125
34	AREA OF CONCERN 2	FUT RES AD-ST	ND	7E-5	6E-5	ND	17	16
		FUT RES AD-LT	ND	3E-4	3E-4	ND	63	53
		FUT RES CH	ND	2E-4	1E-4	ND	36	35
35	GOLF COURSE	FUT RES AD-ST	1E-6	7E-5	6E-5	ND	17	16
		FUT RES AD-LT	8E-6	3E-4	4E-4	ND	63	53
		FUT RES CH	4E-6	2E-4	1E-4	3	36	38
36	TELEPHONE POLE STORAGE AREA	EX AD WKER-ST	2E-5	NA	2E-5	7	NA	7
		EX AD WKER-LT	8E-5	NA	8E-5	24	NA	24
		FUT AD WKER-ST	2E-5	5E-5	5E-5	7	19	27
		FUT AD WKER-LT	8E-5	2E-4	2E-4	24	30	52
		FUT RES AD-ST	3E-5	1E-4	1E-4	11	56	66
		FUT RES AD-LT	2E-4	6E-4	8E-4	35	86	115
		FUT RES CH	1E-4	3E-4	3E-4	93	121	213

NA – Not applicable; The exposure pathway was incomplete.
ND – The risk or hazard value was below the respective criterion of 1E-06 for risk, and 1 for hazard.
NOTE: The combined soil and groundwater risks or hazards are rounded values. Therefore, the individual risk and hazard values may appear to be added incorrectly due to rounding errors.

TABLE R-41 (cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS
FOR SOIL (BASED ON THE TEF METHODOLOGY) AND GROUNDWATER COMBINED
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RECEPTOR	RISK			HAZARD		
			SOIL	GROUND WATER	TOTAL	SOIL	GROUND WATER	TOAL
1	SWMU 4	FUT RES AD-ST	4E-5	1E-4	1E-4	5	56	60
		FUT RES AD-LT	2E-4	6E-4	8E-4	5	86	85
		FUT RES CH	1E-4	3E-4	3E-4	42	121	162
2	SWMU 2, 5, 6, AND 7	FUT RES AD-ST	4E-4	1E-4	5E-4	27	56	82
		FUT RES AD-LT	2E-3	6E-4	3E-3	40	86	120
		FUT RES CH	1E-3	3E-4	1E-3	137	121	257
3	SWMU 1	FUT RES AD-ST	ND	7E-5	6E-5	17	17	33
		FUT RES AD-LT	ND	3E-4	3E-4	31	31	84
		FUT RES CH	ND	2E-4	1E-4	102	102	137
4	AREA A	FUT RES AD-ST	3E-6	1E-4	1E-4	16	56	71
		FUT RES AD-LT	1E-5	6E-4	6E-4	16	86	96
		FUT RES CH	1E-5	3E-4	2E-4	60	121	180
5	AREA B	FUT RES AD-ST	1E-3	1E-4	2E-3	ND	56	55
		FUT RES AD-LT	1E-2	6E-4	1E-2	ND	86	80
		FUT RES CH	4E-3	3E-4	5E-3	2	121	122
6	AREA C	FUT RES AD-ST	ND	1E-4	6E-5	1	56	17
		FUT RES AD-LT	2E-6	6E-4	3E-4	1	86	54
		FUT RES CH	1E-6	3E-4	1E-4	3	121	38
7	SWMU 3	EX AD WKR-ST	ND	NA	ND	ND	NA	
		EX AD WKR-LT	1E-6	NA	1E-6	8	NA	8
		FUT AD WKR-ST	ND	5E-5	3E-5	ND	19	19
		FUT AD WKR-LT	1E-6	2E-4	2E-4	ND	30	28
		FUT RES AD-ST	2E-5	1E-4	1E-4	ND	56	55
		FUT RES AD-LT	1E-4	6E-4	7E-4	7	86	87
		FUT RES CH	7E-5	3E-4	3E-4	25	121	145
8	SWMU 24	EX AD WKER-ST	3E-5	NA	3E-5	1	NA	1
		EX AD WKER-LT	2E-4	NA	2E-4	2	NA	2
		FUT AD WKER-ST	3E-5	5E-5	7E-5	1	19	21
		FUT AD WKER-LT	2E-4	2E-4	3E-4	2	30	30
		FUT RES AD-ST	6E-5	1E-4	1E-4	2	56	57
		FUT RES AD-LT	4E-4	6E-4	9E-4	4	86	84
		FUT RES CH	2E-4	3E-4	4E-4	11	121	131
11	BUILDING 3	EX AD WKER-ST	3E-5	NA	3E-5	8	NA	8
		EX AD WKER-LT	2E-4	NA	2E-4	12	NA	12
		FUT AD WKER-ST	3E-5	5E-5	6E-5	8	19	27
		FUT AD WKER-LT	2E-4	2E-4	3E-4	12	30	40
		FUT RES AD-ST	5E-5	1E-4	1E-4	13	56	68
		FUT RES AD-LT	3E-4	6E-4	8E-4	13	86	93
		FUT RES CH	1E-5	3E-4	1E-4	40	121	160
12	BUILDING 10	EX AD WKER-ST	6E-5	NA	6E-5	11	NA	11
		EX AD WKER-LT	3E-4	NA	3E-4	12	NA	12
		FUT AD WKER-ST	6E-5	5E-5	9E-5	11	19	30
		FUT AD WKER-LT	3E-4	2E-4	5E-4	12	30	40
		FUT RES AD-ST	9E-5	1E-4	2E-4	17	56	72
		FUT RES AD-LT	5E-4	6E-4	1E-3	35	86	115
		FUT RES CH	2E-4	3E-4	5E-4	90	121	210
14	BUILDING 63	EX AD WKER-ST	5E-5	NA	5E-5	12	NA	12
		EX AD WKER-LT	3E-4	NA	3E-4	14	NA	14
		FUT AD WKER-ST	5E-5	5E-5	9E-5	12	19	31
		FUT AD WKER-LT	3E-4	2E-4	4E-4	14	30	42
		FUT RES AD-ST	8E-5	1E-4	2E-4	19	56	74
		FUT RES AD-LT	5E-4	6E-4	1E-3	20	86	100
		FUT RES CH	3E-4	3E-4	5E-4	58	121	177
16	BUILDING 130	EX AD WKER-ST	4E-6	NA	4E-6	7	NA	7
		EX AD WKER-LT	2E-5	NA	2E-5	7	NA	7
		FUT AD WKER-ST	4E-6	5E-5	4E-5	7	19	26
		FUT AD WKER-LT	2E-5	2E-4	2E-4	7	30	35
		FUT RES AD-ST	5E-6	1E-4	1E-4	10	56	65
		FUT RES AD-LT	3E-5	6E-4	6E-4	10	86	90
		FUT RES CH	2E-5	3E-4	2E-4	25	121	145
18	SWMU 23	EX AD WKER-ST	2E-5	NA	2E-5	1	NA	1
		EX AD WKER-LT	8E-5	NA	8E-5	16	NA	16
		FUT AD WKER-ST	2E-5	5E-5	5E-5	1	19	20
		FUT AD WKER-LT	8E-5	2E-4	2E-4	16	30	44
		FUT RES AD-ST	3E-5	1E-4	1E-4	2	56	57
		FUT RES AD-LT	2E-4	6E-4	7E-4	22	86	102
		FUT RES CH	8E-5	3E-4	3E-4	55	121	175

TABLE R-41 (cont'd)
SUMMARY OF RISK CHARACTERIZATION RESULTS
FOR SOIL (BASED ON THE TEF METHODOLOGY) AND GROUNDWATER COMBINED
LEXINGTON-BLUEGRASS ARMY DEPOT

SWMU/ AOC #	SWMU/AOC NAME	RECEPTOR	RISK			HAZARD		
			SOIL	GROUND WATER	TOTAL	SOIL	GROUND WATER	TOAL
19	SWMU 18 AND 19	EX AD WKER-ST	ND	NA	ND	ND	NA	ND
		EX AD WKER-LT	ND	NA	ND	ND	NA	ND
		FUT AD WKER-ST	ND	5E-5	3E-5	ND	19	19
		FUT AD WKER-LT	ND	2E-4	2E-4	ND	30	28
		FUT RES AD-ST	ND	1E-4	9E-5	ND	56	55
		FUT RES AD-LT	1E-6	6E-4	6E-4	ND	86	80
		FUT RES CH	ND	3E-4	2E-4	ND	121	120
21	SWMU 9	EX AD WKER-ST	6E-4	NA	7E-4	2	NA	2
		EX AD WKER-LT	3E-3	NA	3E-3	4	NA	4
		FUT AD WKER-ST	6E-4	5E-5	7E-4	2	19	21
		FUT AD WKER-LT	3E-3	2E-4	3E-3	4	30	32
		FUT RES AD-ST	1E-3	1E-4	1E-3	3	56	58
		FUT RES AD-LT	6E-3	6E-4	7E-3	7	86	87
		FUT RES CH	3E-3	3E-4	3E-3	23	121	143
26	COAL PILE RUNOFF/ HEATING PLANT AREA	EX AD WKER-ST	3E-4	NA	3E-4	13	NA	13
		EX AD WKER-LT	1E-3	NA	1E-3	66	NA	66
		FUT AD WKER-ST	3E-4	5E-5	3E-4	13	19	32
		FUT AD WKER-LT	1E-3	2E-4	1E-3	67	30	95
		FUT RES AD-ST	4E-4	1E-4	5E-4	19	56	74
		FUT RES AD-LT	3E-3	6E-4	3E-3	95	86	175
		FUT RES CH	2E-3	3E-4	2E-3	251	121	370
27	SWMU 10	EX AD WKER-ST	2E-5	NA	2E-5	27	NA	27
		EX AD WKER-LT	1E-4	NA	1E-4	596	NA	596
		FUT AD WKER-ST	2E-5	5E-5	6E-5	27	19	46
		FUT AD WKER-LT	1E-4	2E-4	3E-4	596	30	624
		FUT RES AD-ST	4E-5	1E-4	1E-4	39	56	94
		FUT RES AD-LT	3E-4	6E-4	9E-4	846	86	926
		FUT RES CH	2E-4	3E-4	4E-4	2060	121	2180
29	OPEN STORAGE AND SHELTER AREAS	EX AD WKER-ST	2E-4	NA	2E-4	3	NA	3
		EX AD WKER-LT	1E-3	NA	1E-3	18	NA	18
		FUT AD WKER-ST	2E-4	5E-5	3E-4	3	19	23
		FUT AD WKER-LT	1E-3	2E-4	1E-3	18	30	47
		FUT RES AD-ST	3E-4	1E-4	4E-4	5	56	60
		FUT RES AD-LT	2E-3	6E-4	3E-3	26	86	106
		FUT RES CH	1E-3	3E-4	1E-3	68	121	187
30	SWMU 12	EX AD WKER-ST	3E-6	NA	3E-6	10	NA	10
		EX AD WKER-LT	2E-5	NA	2E-5	48	NA	48
		FUT AD WKER-ST	3E-6	5E-5	4E-5	10	19	29
		FUT AD WKER-LT	2E-5	2E-4	2E-4	48	30	76
		FUT RES AD-ST	6E-6	1E-4	1E-4	15	56	70
		FUT RES AD-LT	3E-5	6E-4	6E-4	68	86	148
		FUT RES CH	2E-5	3E-4	2E-4	175	121	294
33	SWMU 22	EX AD WKER-ST	3E-6	NA	3E-6	ND	NA	ND
		EX AD WKER-LT	2E-5	NA	1E-5	ND	NA	ND
		FUT AD WKER-ST	3E-6	5E-5	4E-5	ND	19	19
		FUT AD WKER-LT	2E-5	2E-4	2E-4	ND	30	28
		FUT RES AD-ST	4E-6	1E-4	1E-4	ND	56	55
		FUT RES AD-LT	3E-5	6E-4	6E-4	1	86	81
		FUT RES CH	1E-5	3E-4	2E-4	5	121	125
35	GOLF COURSE	FUT RES AD-ST	ND	7E-5	6E-5	ND	17	16
		FUT RES AD-LT	ND	3E-4	3E-4	ND	31	53
		FUT RES CH	ND	2E-4	1E-4	3	102	38
36	TELEPHONE POLE STORAGE AREA	EX AD WKER-ST	1E-5	NA	1E-5	7	NA	7
		EX AD WKER-LT	7E-5	NA	7E-5	24	NA	24
		FUT AD WKER-ST	1E-5	5E-5	5E-5	7	19	27
		FUT AD WKER-LT	7E-5	2E-4	2E-4	24	30	52
		FUT RES AD-ST	2E-5	1E-4	1E-4	11	56	66
		FUT RES AD-LT	1E-4	6E-4	7E-4	35	86	115
		FUT RES CH	7E-5	3E-4	3E-4	93	121	213

NA – Not applicable; The exposure pathway was incomplete.
ND – The risk or hazard value was below the respective criterion of 1E-06 for risk, and 1 for hazard.
NOTE: The combined soil and groundwater risks or hazards are rounded values. Therefore, the individual risk and hazard values may appear to be added incorrectly due to rounding errors.

ATTACHMENT 1

LEAD UPTAKE BIOKINETIC MODEL RESULTS

LEAD UPTAKE/BIOKINETIC MODEL RESULTS

This appendix provides the output of the U.S. EPA lead uptake/biokinetic model (U.S. EPA, 1991) utilized for the SWMUs where the soil lead concentrations exceeded background concentrations (i.e., soil lead levels greater than 73 mg/kg or 66 mg/kg, respectively). U.S. EPA Region IV risk assessment guidelines (U.S. EPA, 1991c) recommended that the "background" chemical concentration for naturally occurring chemicals be determined by multiplying the arithmetic average of site-specific sampling results by a factor of two. The average background level for lead in soil at the LBAD was 36.5 mg/kg. The average lead level in background sediment was 33 mg/kg. The following SWMUs were found to have lead levels which exceeded two times the average background lead concentration in soil or sediment (i.e., 73 mg/kg for soil and 66 mg/kg for sediment):

SWMU/Area of Concern	Soil Lead Concentrations (mg/kg)	Modeled Airborne Lead Concentrations(mg/m^3)	Groundwater Lead Concentrations (ug/L)
SWMU 4	446	2.41E-5	41.9
SWMU 2,5,6,7	92	7.69E-6	41.9
Area C	78.1	1.7E-5	62.7
SWMU 24	99.4	2.68E-6	41.9
Building 10	180	2.42E-6	41.9
Building 130	250	2.92E-6	41.9
SWMU 23	210	2.14E-5	41.9
SWMU 18,19	77SS	2.59E-7	41.9
SWMU 9	430	3.97E-6	41.9
SWMU 20	280 SS 242 SB	4.32E-6 3.73E-6	41.9
Coal Pile	170 (SS & SB)	1.72E-6	41.9
SWMU 10	1800	2.42E-5	41.9
Open Storage	220	1.42E-5	41.9
SWMU 12	1430	5.79E-5	41.9
Telephone Pole	1300	1.5E-6	41.9
Building 63	1300	1.07E-5	41.9
Building 42	178	2.69E-6	41.9

The site-specific and U.S. EPA-desired default parameter values employed in the model are presented in Tables 1 through 17. Site-specific parameters including the SWMU-specific soil and associated air concentrations (based on the results of the Appendix 3 model calculations performed to predict fugitive dust emissions from the site) were utilized in the model. The model incorporates exposures through indoor and outdoor air inhalation, drinking water ingestion, ingestion of lead in soil and dust, and the default maternal blood contribution (using the default of 7.5 μg/dL lead).

Figures 1, through 34 present probability plots relative to the geometric mean blood lead level predicted by the model and also show the geometric mean blood lead concentrations which would be associated with the SWMU-specific conditions. The intersect on these figures represents the percentage of children who would be expected to have blood levels above 10 μg/dL.

Figures 1 through 34 also present the SWMU-specific probability density distribution about the geometric mean and standard deviation of the blood lead levels. The model predicts the percent of the exposed population which would be expected to have blood lead levels above 10 μg/dL for each of the SWMUs.

The uptake/biokinetic model is accepted by U.S. EPA as the most appropriate available method for evaluating potential lead exposure.

TABLE 1
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR SWMU 4

ABSORPTION METHODOLOGY: Non-Linear Active-Passive

AIR CONCENTRATION: 0.024 ug Pb/m3
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 41.90 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: Multiple Source Analysis

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	446.0	127.3
1-2	446.0	127.3
2-3	446.0	127.3
3-4	446.0	127.3
4-5	446.0	127.3
5-6	446.0	127.3
6-7	446.0	127.3

Additional Dust Sources: None DEFAULT
Soil contribution conversion factor: 0.28
Air contribution conversion factor: 100.0

PAINT Intake: 0.00 ug Pb/day DEFAULT

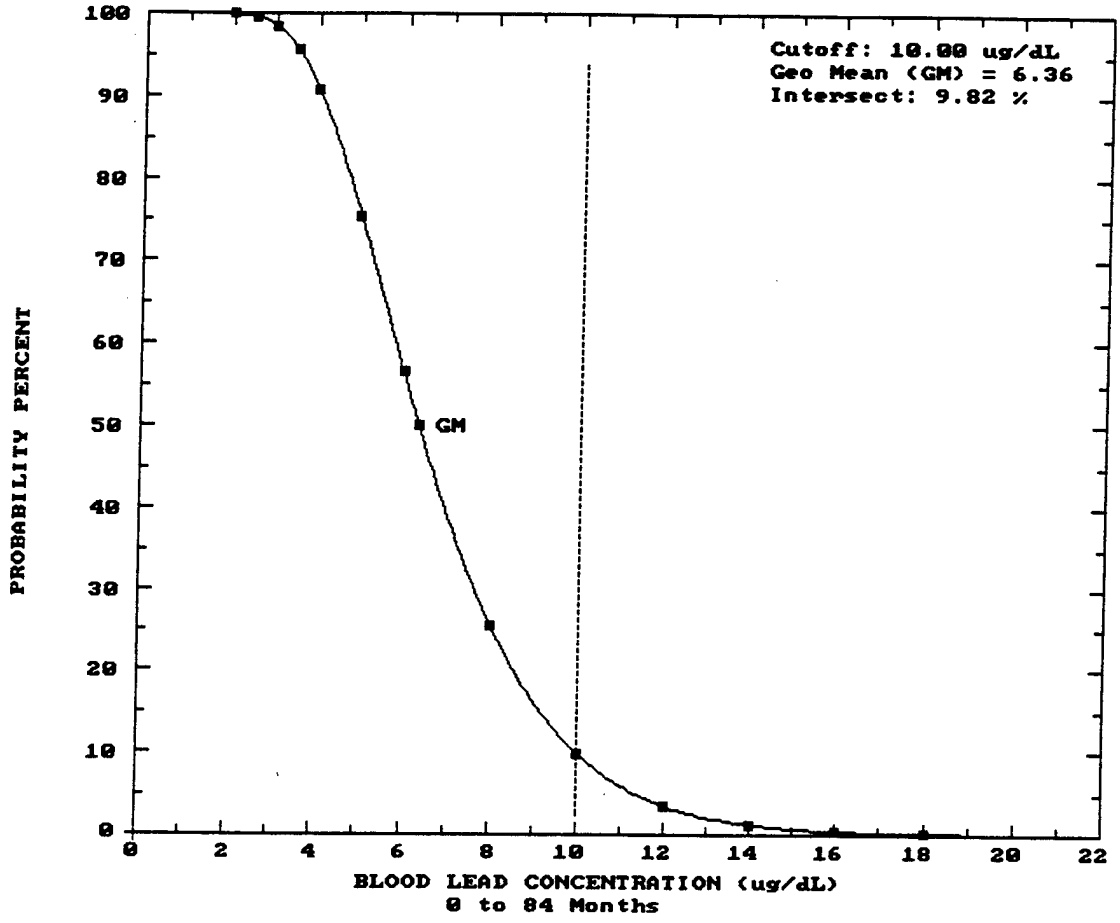
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 7.50 ug Pb/dL

TABLE 1 (cont.)
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR SWMU 4

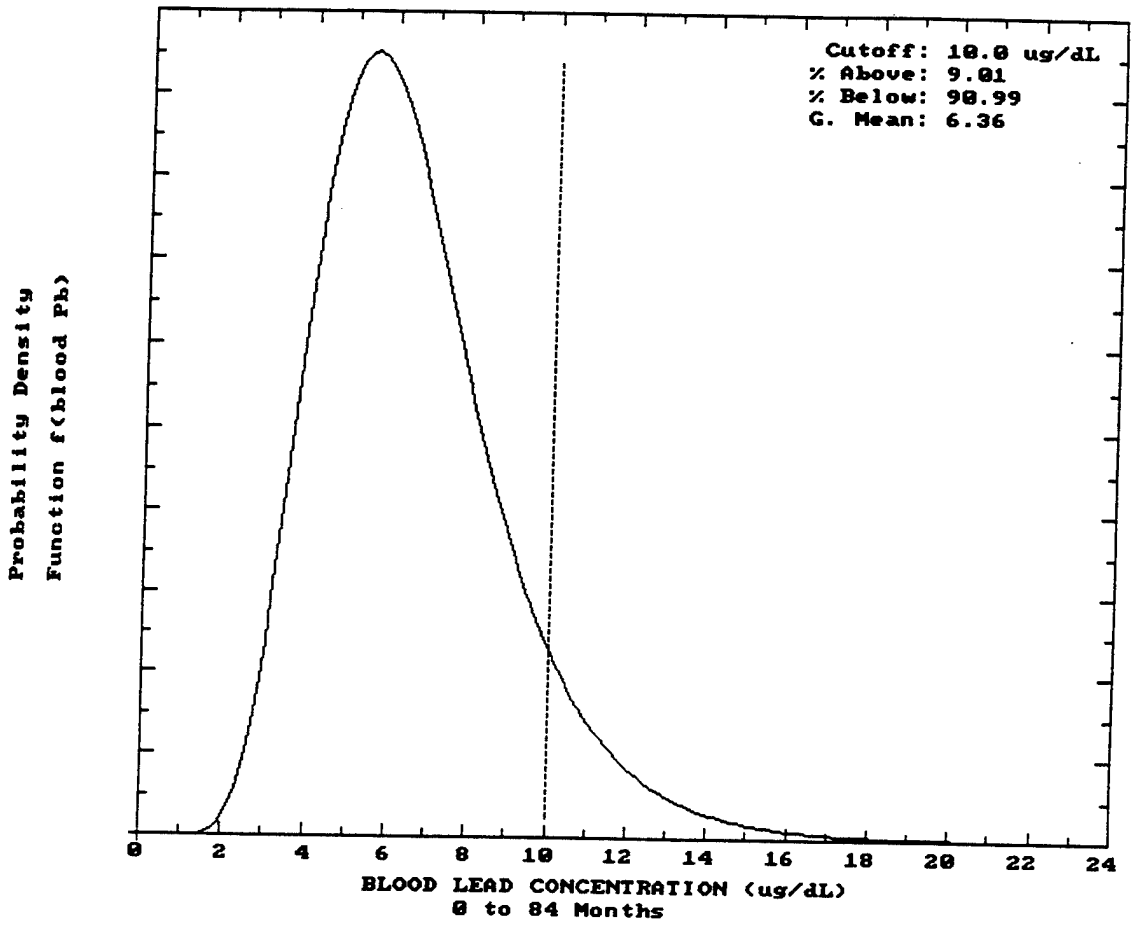
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	4.99	15.26	8.12
1-2:	5.80	21.56	8.12
2-3:	6.26	22.43	8.12
3-4:	6.47	22.53	8.12
4-5:	6.77	22.84	8.12
5-6:	6.91	23.67	8.12
6-7:	6.98	24.24	8.12

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.94	4.19	0.00	0.01
1-2:	2.96	10.47	0.00	0.01
2-3:	3.40	10.89	0.00	0.01
3-4:	3.29	11.10	0.00	0.02
4-5:	3.18	11.52	0.00	0.02
5-6:	3.38	12.15	0.00	0.02
6-7:	3.74	12.36	0.00	0.02



Metcalf & Eddy	**PROBABLITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR SWMU 4**	Project Number 012308-0005
		Figure 1



Metcalf & Eddy	**PROBABLITY DENSITY OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR SWMU 4**	Project Number 012308-0005
		Figure 2

TABLE 2
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR SWMU 2,5,6 AND 7

ABSORPTION METHODOLOGY: Non-Linear Active-Passive

AIR CONCENTRATION: 0.008 ug Pb/m3
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 41.90 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: Multiple Source Analysis

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	92.0	26.5
1-2	92.0	26.5
2-3	92.0	26.5
3-4	92.0	26.5
4-5	92.0	26.5
5-6	92.0	26.5
6-7	92.0	26.5

Additional Dust Sources: None DEFAULT
Soil contribution conversion factor: 0.28
Air contribution conversion factor: 100.0

PAINT Intake: 0.00 ug Pb/day DEFAULT

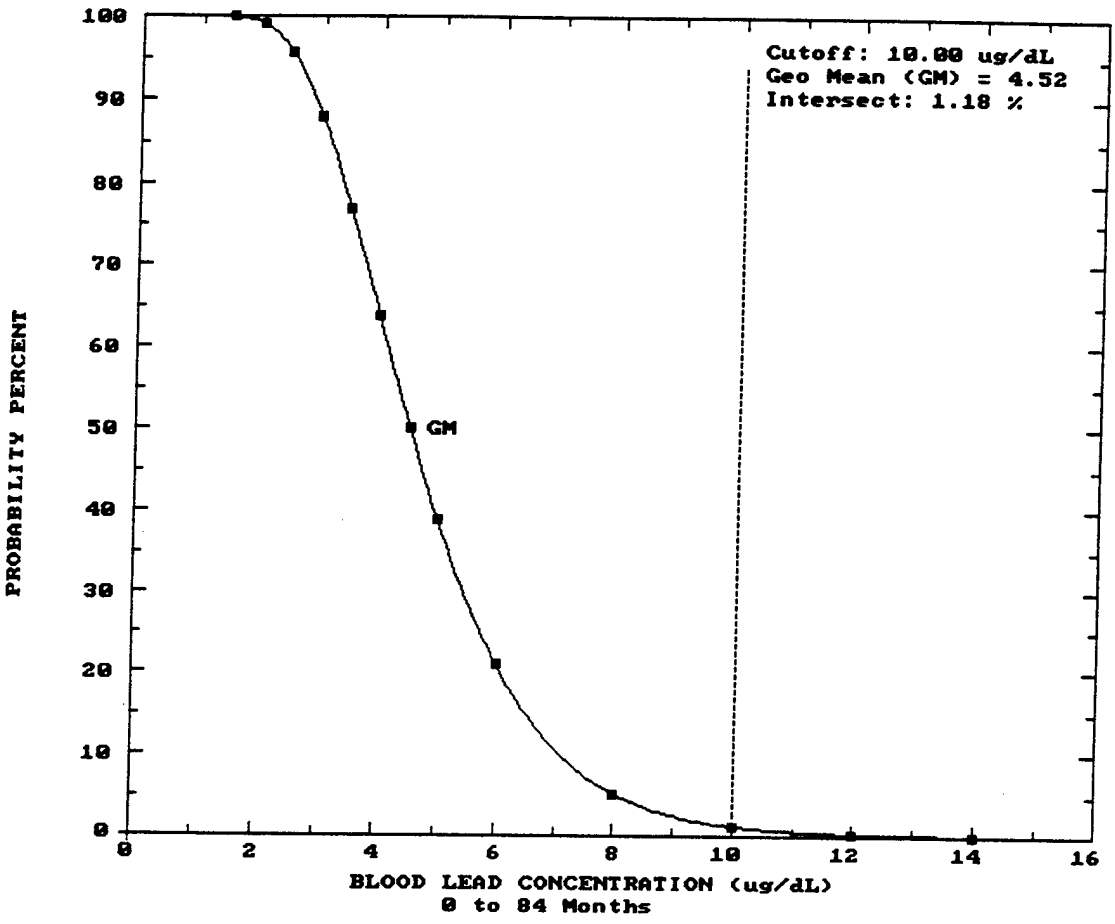
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 7.50 ug Pb/dL

TABLE 2 (cont.)
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR SWMU 2,5,6 AND 7

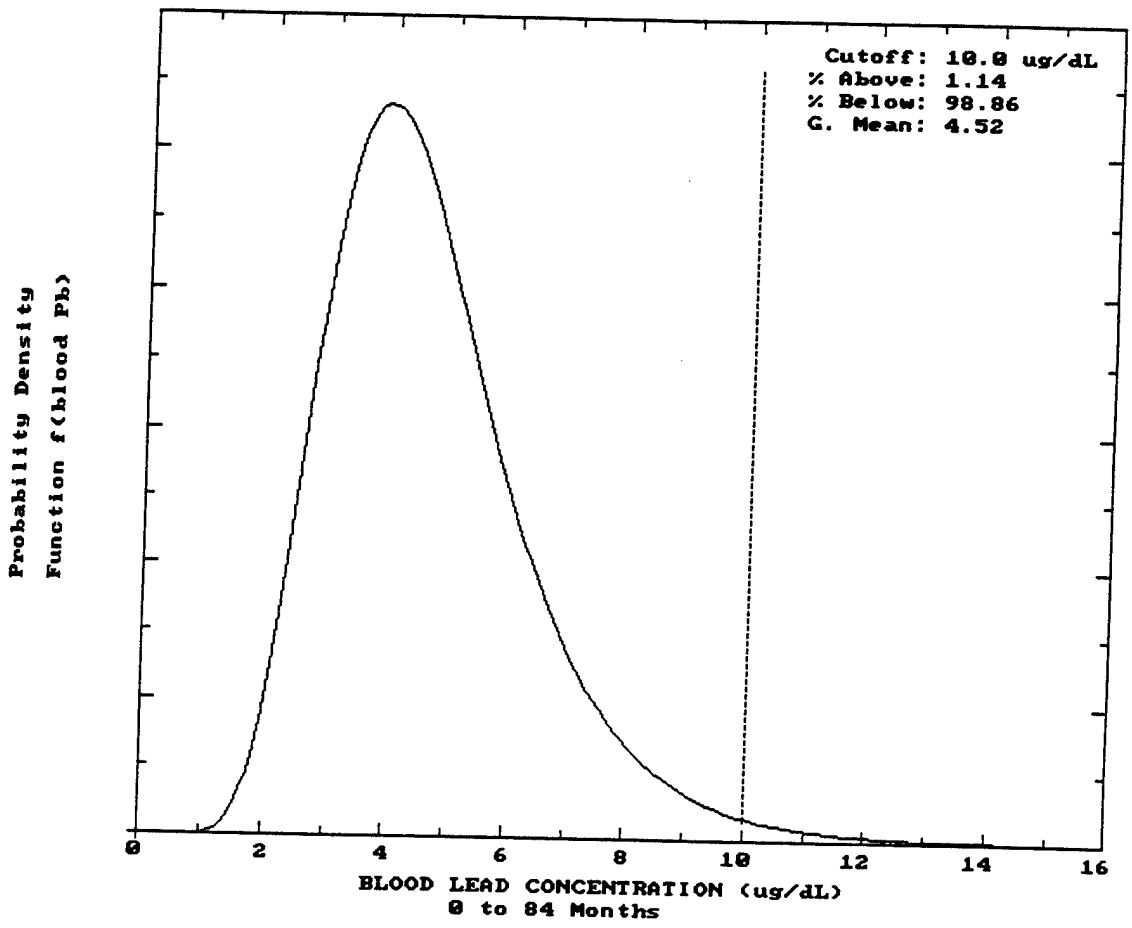
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	3.14	8.81	1.68
1-2:	3.90	15.12	1.68
2-3:	4.40	15.97	1.68
3-4:	4.60	16.07	1.68
4-5:	4.84	16.39	1.68
5-6:	4.98	17.21	1.68
6-7:	5.08	17.79	1.68

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.94	4.19	0.00	0.00
1-2:	2.96	10.47	0.00	0.00
2-3:	3.40	10.89	0.00	0.00
3-4:	3.29	11.10	0.00	0.01
4-5:	3.18	11.52	0.00	0.01
5-6:	3.38	12.15	0.00	0.01
6-7:	3.74	12.36	0.00	0.01



Metcalf & Eddy	**PROBABLITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR SWMU 2,5,6,7**	Project Number 012308-0005
		Figure 3



Metcalf & Eddy	**PROBABLITY DENSITY OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR SWMU 2,5,6,7**	Project Number 012308-0005
		Figure 4

TABLE 3
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL FOR AREA C

ABSORPTION METHODOLOGY: Non-Linear Active-Passive

AIR CONCENTRATION: 0.017 ug Pb/m3
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 62.70 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: Multiple Source Analysis

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	78.1	23.6
1-2	78.1	23.6
2-3	78.1	23.6
3-4	78.1	23.6
4-5	78.1	23.6
5-6	78.1	23.6
6-7	78.1	23.6

Additional Dust Sources: None DEFAULT
Soil contribution conversion factor: 0.28
Air contribution conversion factor: 100.0

PAINT Intake: 0.00 ug Pb/day DEFAULT

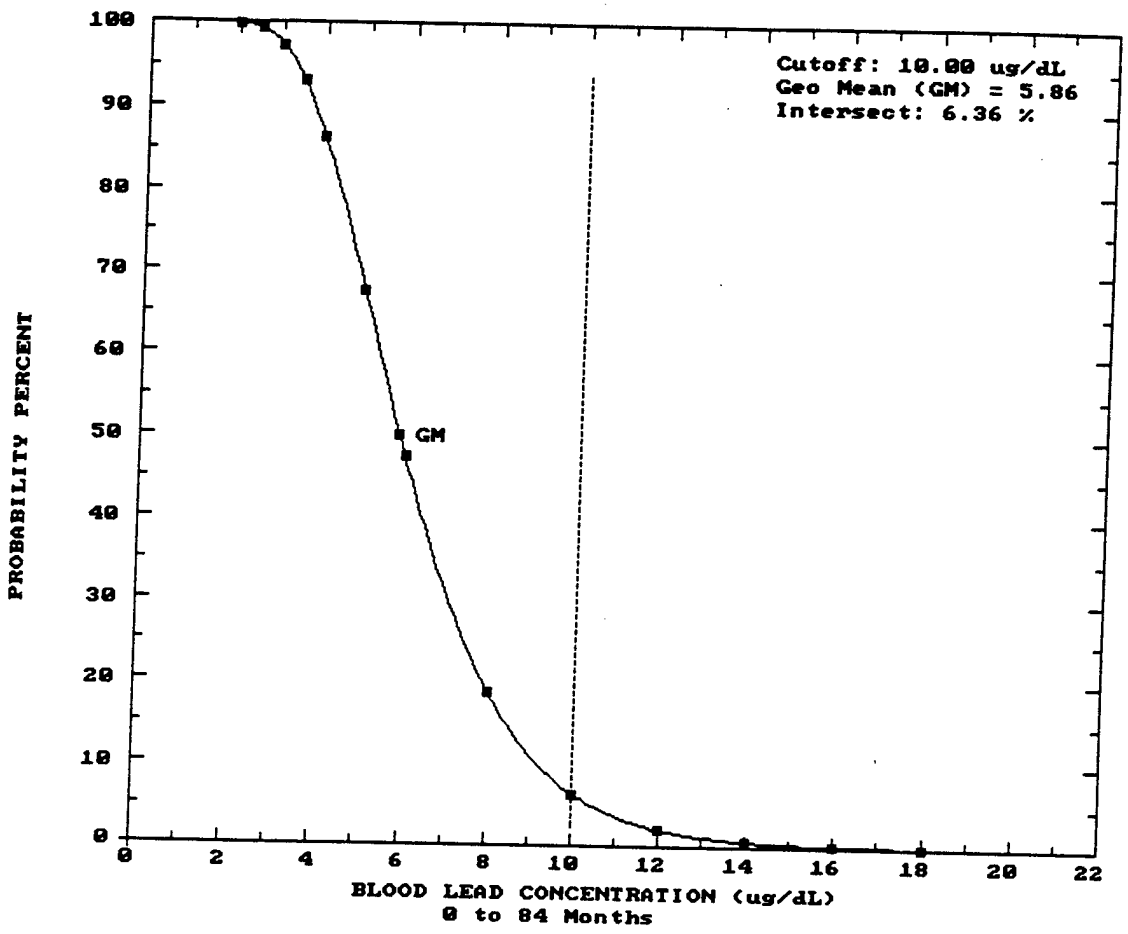
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 7.50 ug Pb/dL

TABLE 3 (cont.)
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR AREA C

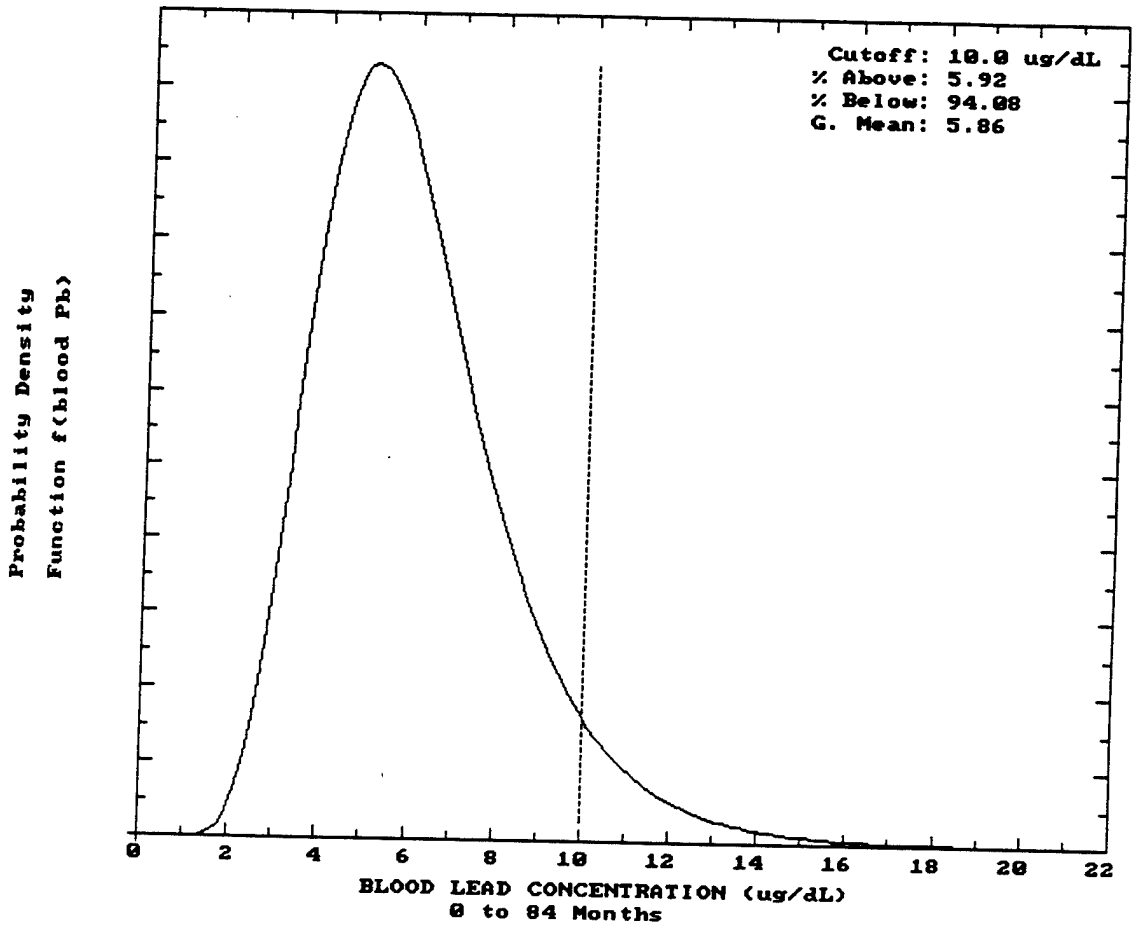
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	3.67	10.66	1.44
1-2:	5.02	20.08	1.44
2-3:	5.81	21.15	1.44
3-4:	6.09	21.36	1.44
4-5:	6.44	21.88	1.44
5-6:	6.65	23.02	1.44
6-7:	6.78	23.70	1.44

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.94	6.27	0.00	0.00
1-2:	2.96	15.68	0.00	0.01
2-3:	3.40	16.30	0.00	0.01
3-4:	3.29	16.62	0.00	0.01
4-5:	3.18	17.24	0.00	0.01
5-6:	3.38	18.18	0.00	0.02
6-7:	3.74	18.50	0.00	0.02



Metcalf & Eddy	**PROBABLITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR AREA C**	Project Number 012308-0005
		Figure 5



Metcalf & Eddy	**PROBABLITY DENSITY OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR AREA C**	Project Number 012308-0005
		Figure 6

TABLE 4
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR SWMU 24

ABSORPTION METHODOLOGY: Non-Linear Active-Passive

AIR CONCENTRATION: 0.003 ug Pb/m3
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 41.90 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: Multiple Source Analysis

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	99.4	28.1
1-2	99.4	28.1
2-3	99.4	28.1
3-4	99.4	28.1
4-5	99.4	28.1
5-6	99.4	28.1
6-7	99.4	28.1

Additional Dust Sources: None DEFAULT
Soil contribution conversion factor: 0.28
Air contribution conversion factor: 100.0

PAINT Intake: 0.00 ug Pb/day DEFAULT

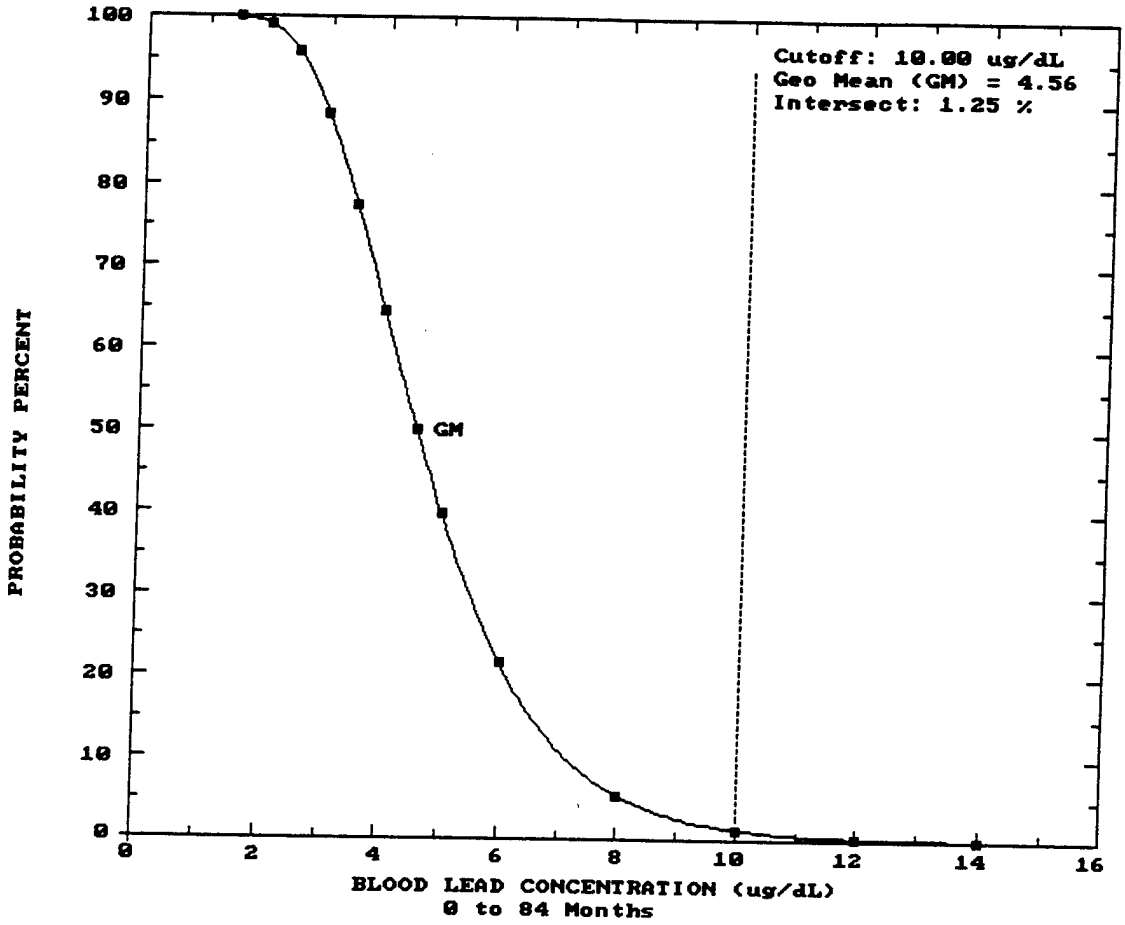
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 7.50 ug Pb/dL

TABLE 4 (cont.)
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR SWMU 24

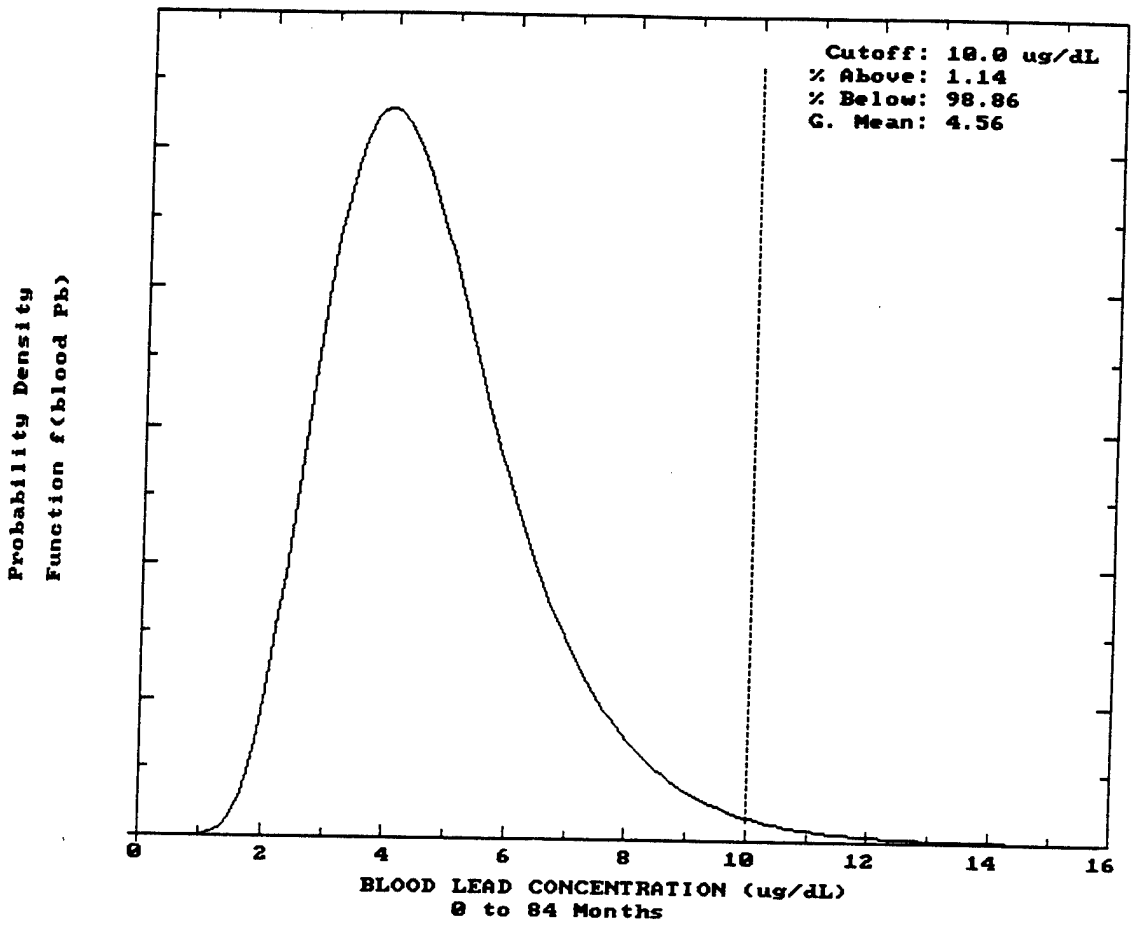
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	3.17	8.94	1.81
1-2:	3.93	15.24	1.81
2-3:	4.44	16.10	1.81
3-4:	4.63	16.20	1.81
4-5:	4.88	16.51	1.81
5-6:	5.02	17.33	1.81
6-7:	5.12	17.91	1.81

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.94	4.19	0.00	0.00
1-2:	2.96	10.47	0.00	0.00
2-3:	3.40	10.89	0.00	0.00
3-4:	3.29	11.10	0.00	0.00
4-5:	3.18	11.52	0.00	0.00
5-6:	3.38	12.15	0.00	0.00
6-7:	3.74	12.36	0.00	0.00



Metcalf & Eddy	**PROBABLITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR SWMU 24**	Project Number 012308-0005
		Figure 7



Metcalf & Eddy	**PROBABLITY DENSITY OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR SWMU 24**	Project Number 012308-0005
		Figure 8

TABLE 5
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR BUILDING 10

ABSORPTION METHODOLOGY: Non-Linear Active-Passive

AIR CONCENTRATION: 0.002 ug Pb/m3
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 41.90 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: Multiple Source Analysis

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	180.0	50.6
1-2	180.0	50.6
2-3	180.0	50.6
3-4	180.0	50.6
4-5	180.0	50.6
5-6	180.0	50.6
6-7	180.0	50.6

Additional Dust Sources: None DEFAULT
Soil contribution conversion factor: 0.28
Air contribution conversion factor: 100.0

PAINT Intake: 0.00 ug Pb/day DEFAULT

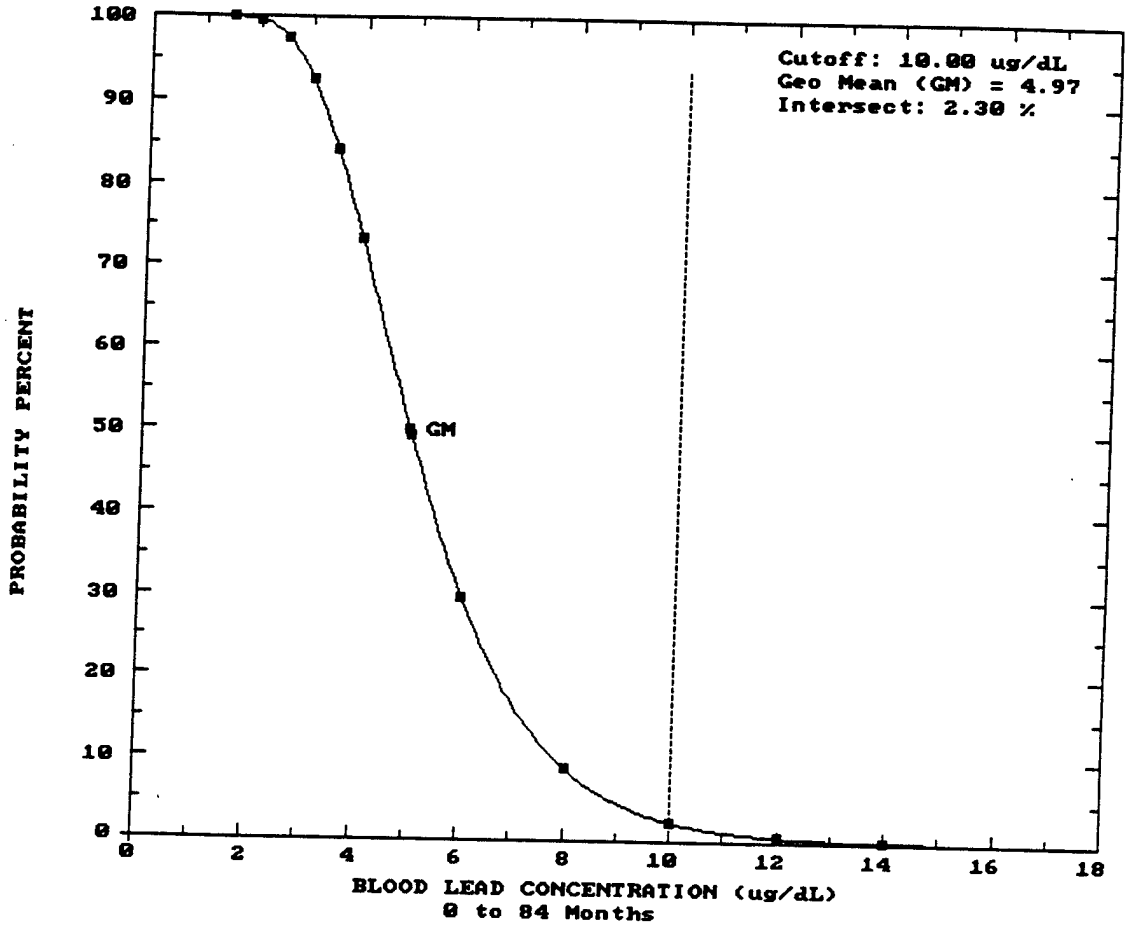
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 7.50 ug Pb/dL

TABLE 5 (cont.)
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR BUILDING 10

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	3.59	10.40	3.27
1-2:	4.36	16.70	3.27
2-3:	4.86	17.56	3.27
3-4:	5.06	17.66	3.27
4-5:	5.31	17.97	3.27
5-6:	5.45	18.79	3.27
6-7:	5.55	19.37	3.27

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.94	4.19	0.00	0.00
1-2:	2.96	10.47	0.00	0.00
2-3:	3.40	10.89	0.00	0.00
3-4:	3.29	11.10	0.00	0.00
4-5:	3.18	11.52	0.00	0.00
5-6:	3.38	12.15	0.00	0.00
6-7:	3.74	12.36	0.00	0.00



Metcalf & Eddy	**PROBABLITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR BUILDING 10**	Project Number 012308-0005
		Figure 9

TABLE 10 (cont.)
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR SWMU 20 (SOIL BORINGS)

ABSORPTION METHODOLOGY: Non-Linear Active-Passive

AIR CONCENTRATION: 0.004 ug Pb/m3
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 41.90 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: Multiple Source Analysis

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	242.0	68.2
1-2	242.0	68.2
2-3	242.0	68.2
3-4	242.0	68.2
4-5	242.0	68.2
5-6	242.0	68.2
6-7	242.0	68.2

Additional Dust Sources: None DEFAULT
Soil contribution conversion factor: 0.28
Air contribution conversion factor: 100.0

PAINT Intake: 0.00 ug Pb/day DEFAULT

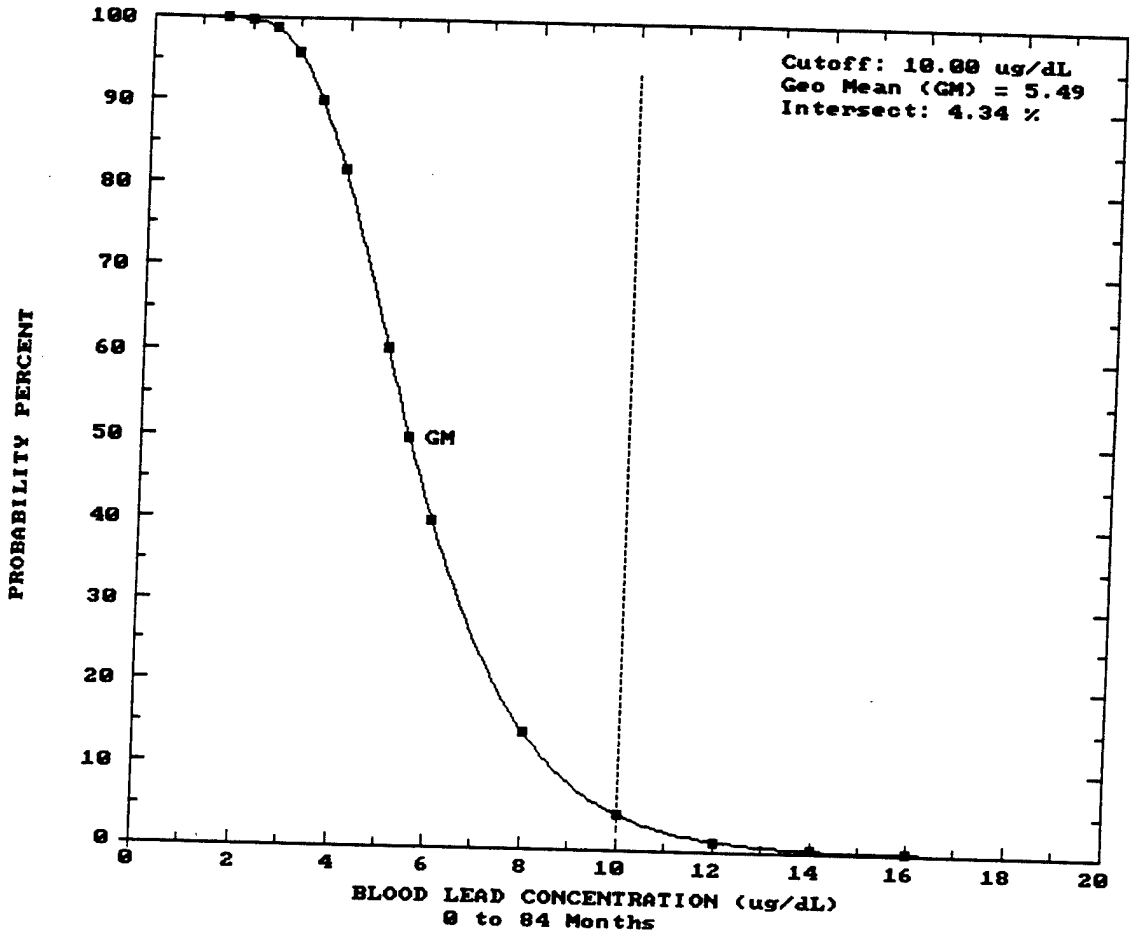
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 7.50 ug Pb/dL

TABLE 10 (cont.)
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR SWMU 20 (SOIL BORINGS)

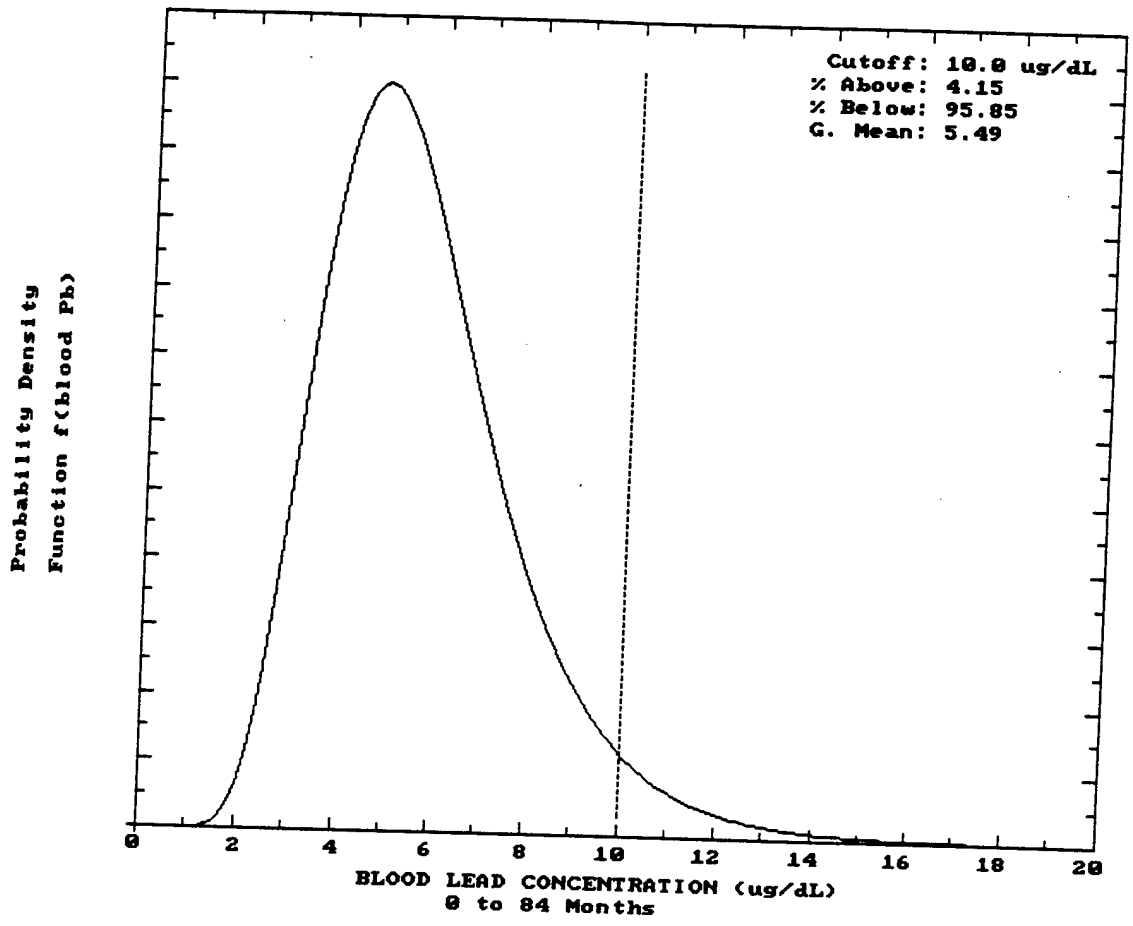
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	3.92	11.52	4.39
1-2:	4.70	17.83	4.39
2-3:	5.18	18.68	4.39
3-4:	5.38	18.78	4.39
4-5:	5.65	19.10	4.39
5-6:	5.79	19.92	4.39
6-7:	5.88	20.50	4.39

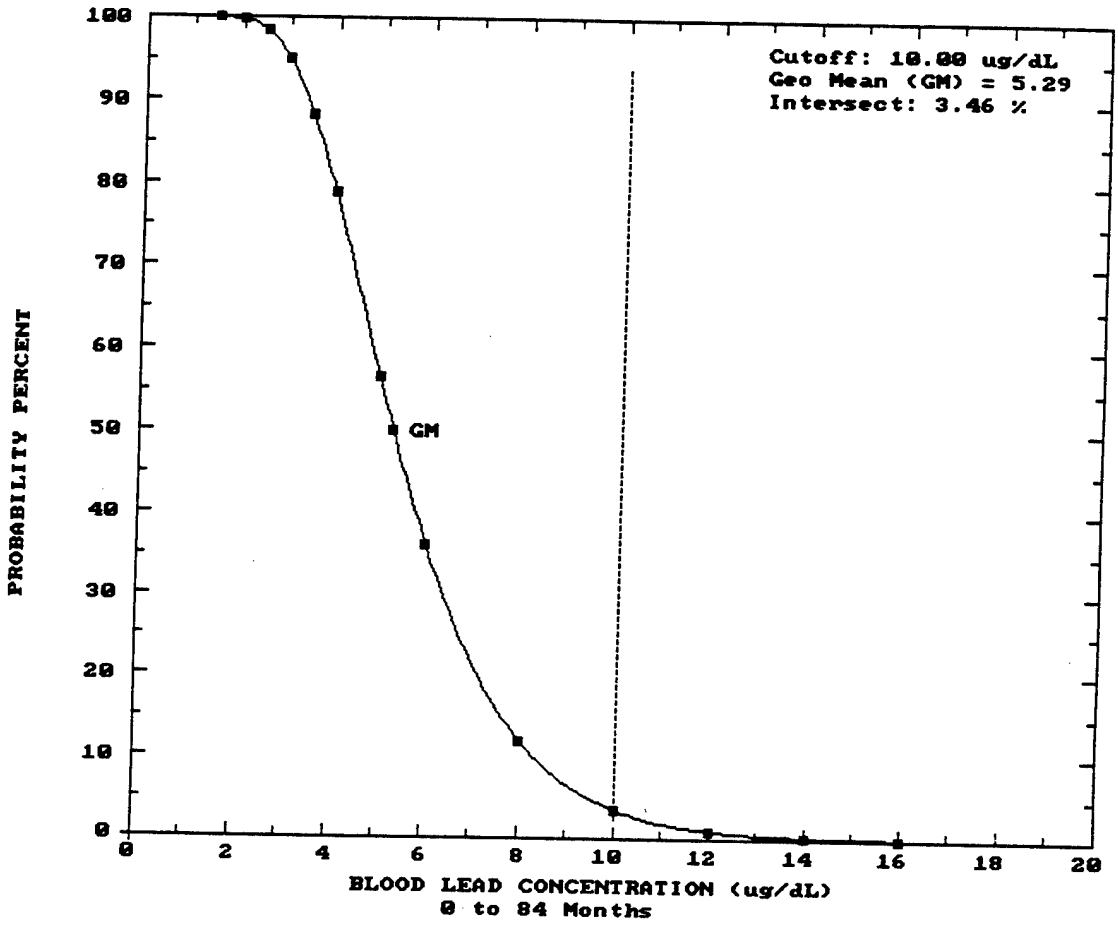
YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.94	4.19	0.00	0.00
1-2:	2.96	10.47	0.00	0.00
2-3:	3.40	10.89	0.00	0.00
3-4:	3.29	11.10	0.00	0.00
4-5:	3.18	11.52	0.00	0.00
5-6:	3.38	12.15	0.00	0.00
6-7:	3.74	12.36	0.00	0.00



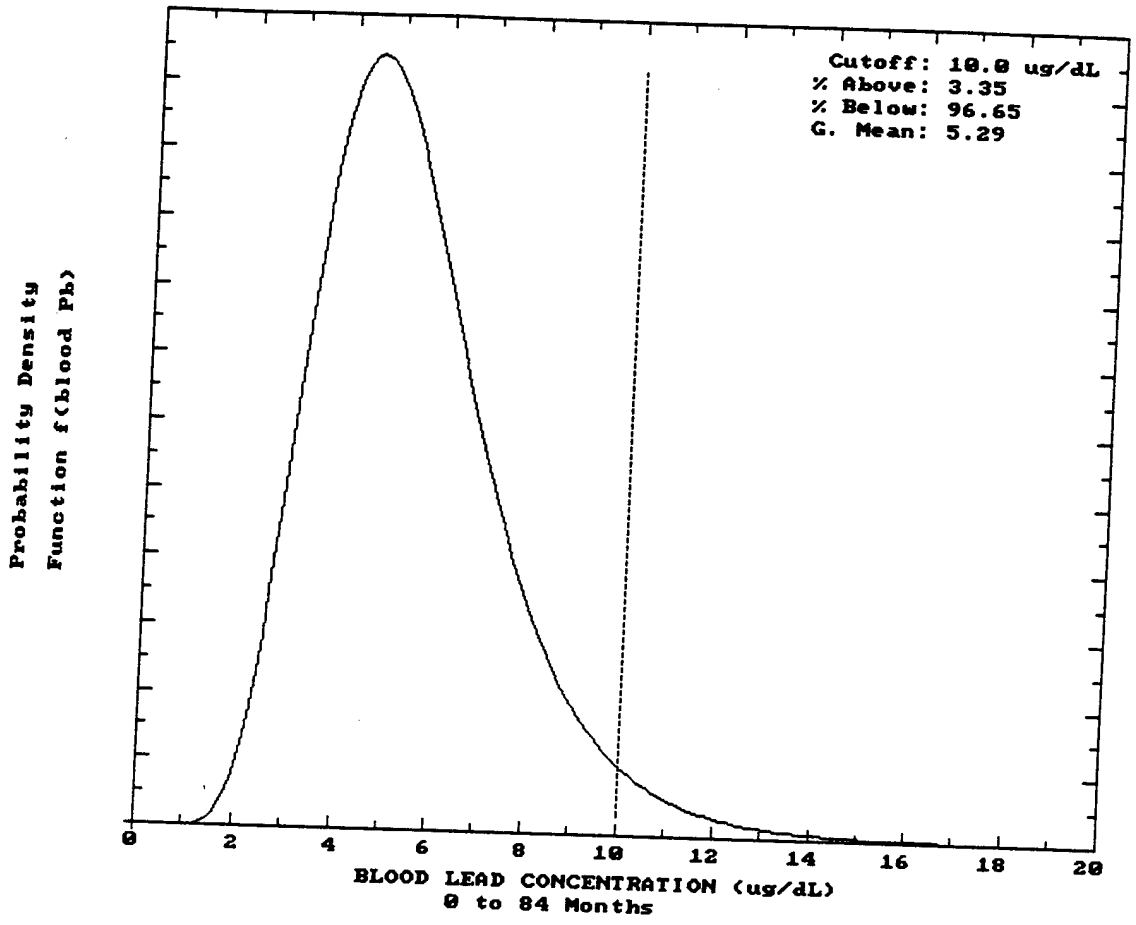
Metcalf & Eddy	**PROBABLITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR SWMU 20 SURFACE**	Project Number 012308-0005
		Figure 19



Metcalf & Eddy	**PROBABLITY DENSITY OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR SWMU 20 SURFACE**	Project Number 012308-0005
		Figure 20



Metcalf & Eddy	**PROBABLITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR SWMU 20 BORINGS**	Project Number 012308-0005
		Figure 21



Metcalf & Eddy	**PROBABLITY DENSITY OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR SWMU 20 BORINGS**	Project Number 012308-0005
		Figure 22

TABLE 11
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR COAL PILE

ABSORPTION METHODOLOGY: Non-Linear Active-Passive

AIR CONCENTRATION: 0.002 ug Pb/m3
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 41.90 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: Multiple Source Analysis

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	170.0	47.8
1-2	170.0	47.8
2-3	170.0	47.8
3-4	170.0	47.8
4-5	170.0	47.8
5-6	170.0	47.8
6-7	170.0	47.8

Additional Dust Sources: None DEFAULT
Soil contribution conversion factor: 0.28
Air contribution conversion factor: 100.0

PAINT Intake: 0.00 ug Pb/day DEFAULT

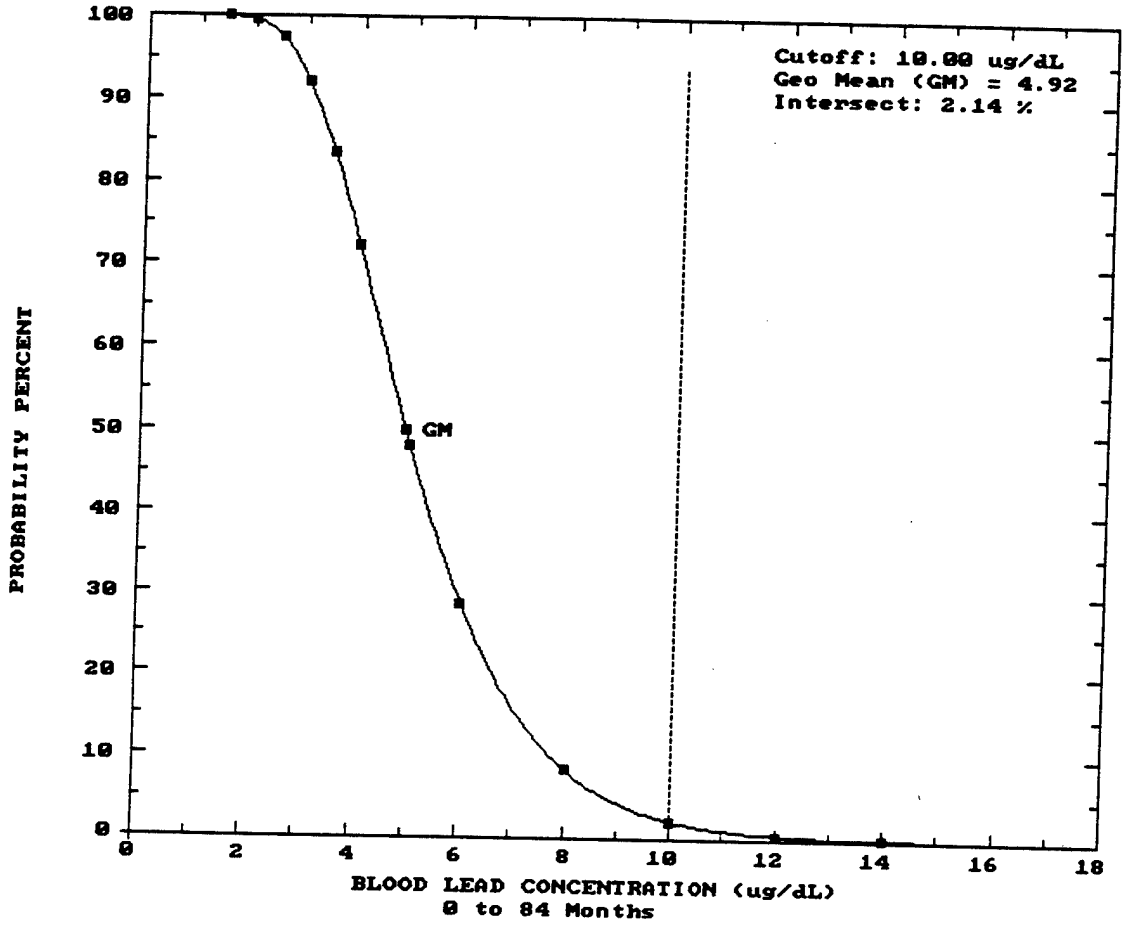
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 7.50 ug Pb/dL

TABLE 11 (cont.)
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR COAL PILE

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	3.54	10.21	3.08
1-2:	4.31	16.52	3.08
2-3:	4.80	17.37	3.08
3-4:	5.00	17.47	3.08
4-5:	5.26	17.79	3.08
5-6:	5.40	18.61	3.08
6-7:	5.49	19.19	3.08

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.94	4.19	0.00	0.00
1-2:	2.96	10.47	0.00	0.00
2-3:	3.40	10.89	0.00	0.00
3-4:	3.29	11.10	0.00	0.00
4-5:	3.18	11.52	0.00	0.00
5-6:	3.38	12.15	0.00	0.00
6-7:	3.74	12.36	0.00	0.00

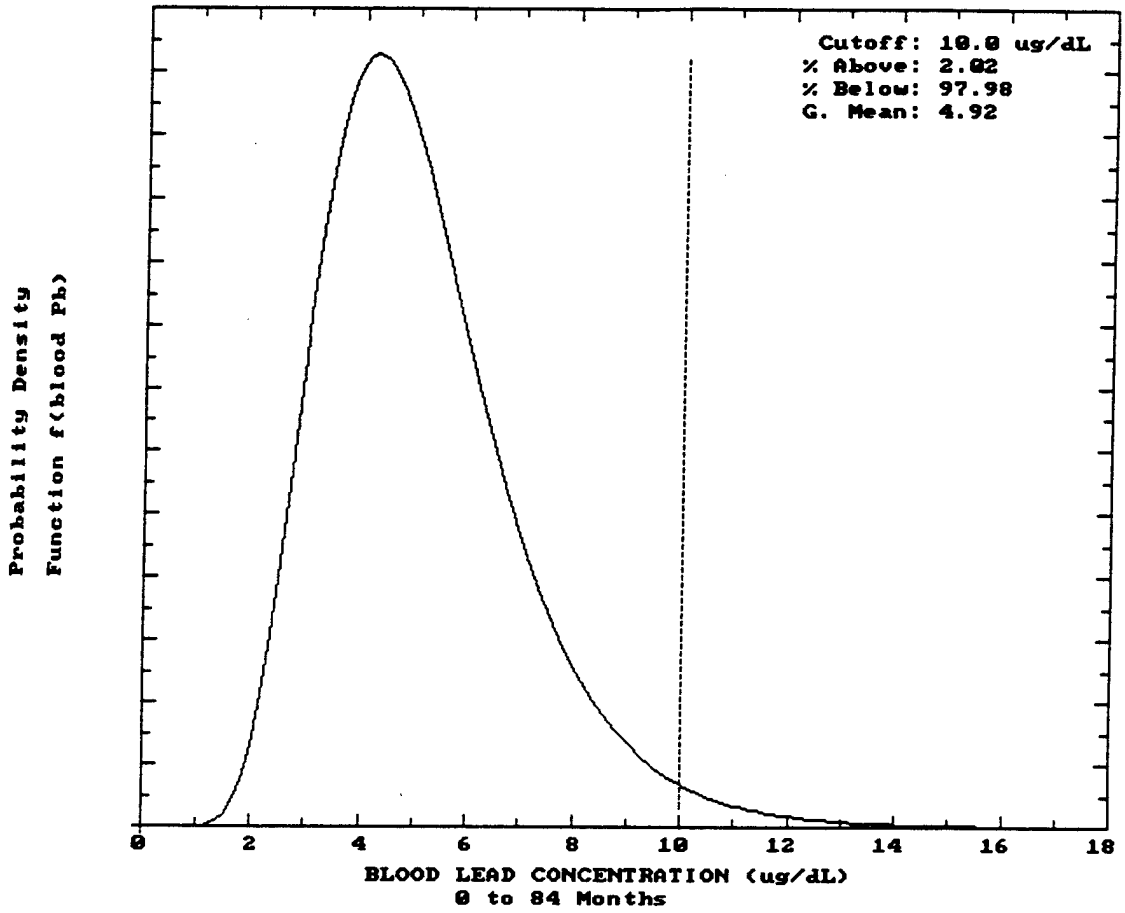


Metcalf & Eddy

PROBABLITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR COAL PILE

Project Number
012308-0005

Figure 23



Metcalf & Eddy	**PROBABLITY DENSITY OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR COAL PILE**	Project Number 012308-0005
		Figure 24

TABLE 12
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR SWMU 10

ABSORPTION METHODOLOGY: Non-Linear Active-Passive

AIR CONCENTRATION: 0.024 ug Pb/m3
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 41.90 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: Multiple Source Analysis

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	1800.0	506.4
1-2	1800.0	506.4
2-3	1800.0	506.4
3-4	1800.0	506.4
4-5	1800.0	506.4
5-6	1800.0	506.4
6-7	1800.0	506.4

Additional Dust Sources: None DEFAULT
Soil contribution conversion factor: 0.28
Air contribution conversion factor: 100.0

PAINT Intake: 0.00 ug Pb/day DEFAULT

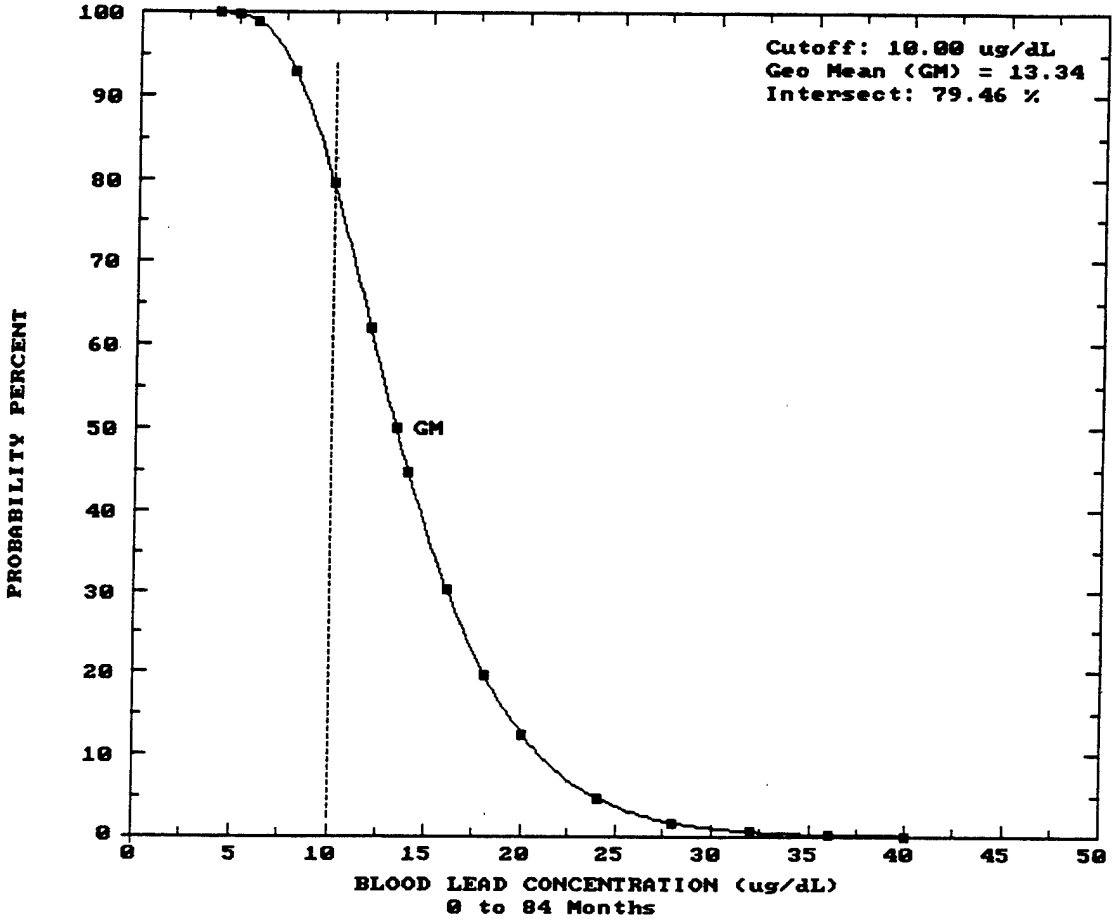
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 7.50 ug Pb/dL

TABLE 12 (cont.)
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR SWMU 10

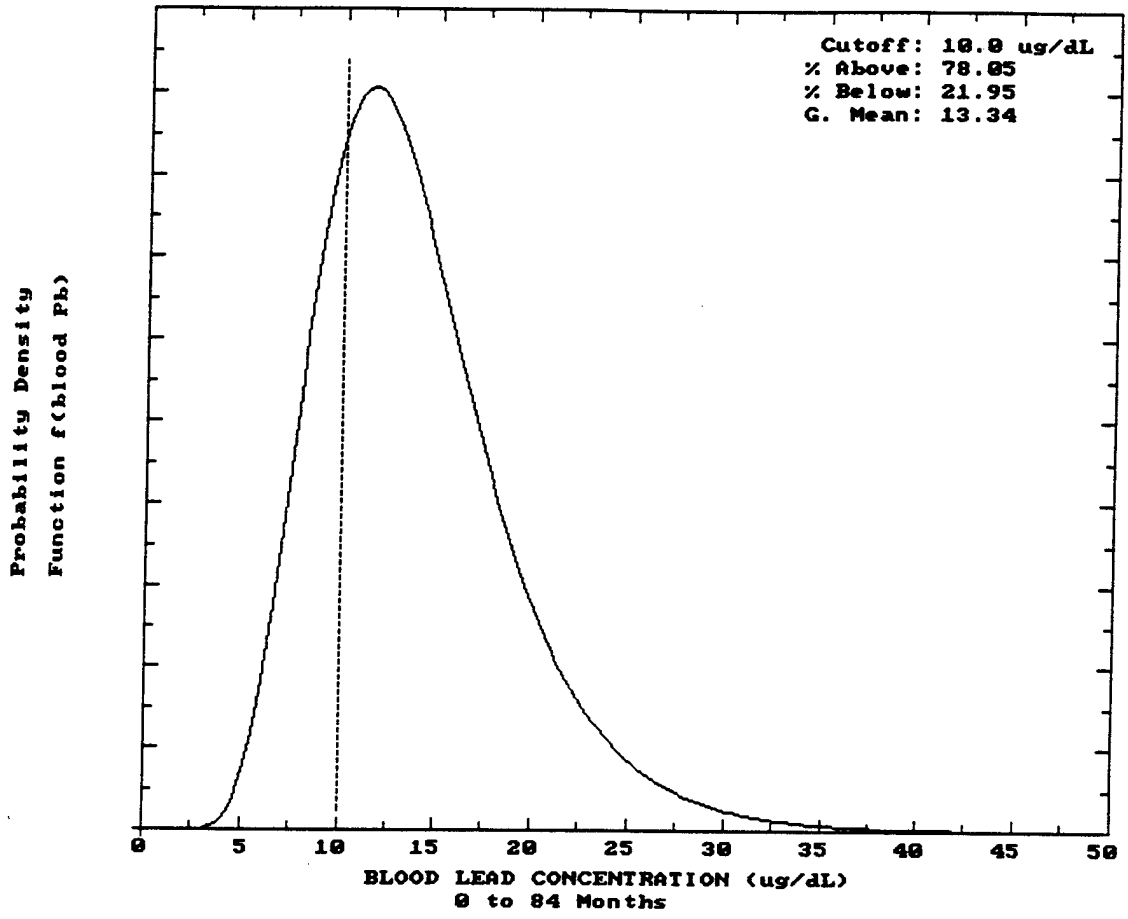
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	12.07	39.79	32.66
1-2:	13.04	46.10	32.66
2-3:	13.33	46.96	32.66
3-4:	13.61	47.06	32.66
4-5:	14.16	47.37	32.66
5-6:	14.27	48.20	32.66
6-7:	14.23	48.78	32.66

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.94	4.19	0.00	0.01
1-2:	2.96	10.47	0.00	0.01
2-3:	3.40	10.89	0.00	0.02
3-4:	3.29	11.10	0.00	0.02
4-5:	3.18	11.52	0.00	0.02
5-6:	3.38	12.15	0.00	0.02
6-7:	3.74	12.36	0.00	0.02



Metcalf & Eddy	**PROBABLITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR SWMU 10**	Project Number 012308-0005
		Figure 25



Metcalf & Eddy	**PROBABLITY DENSITY OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR SWMU 10**	Project Number 012308-0005
		Figure 26

TABLE 13
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL FOR OPEN STORAGE AND SHELTER AREA

ABSORPTION METHODOLOGY: Non-Linear Active-Passive

AIR CONCENTRATION: 0.014 ug Pb/m3
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 41.90 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: Multiple Source Analysis

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	220.0	63.0
1-2	220.0	63.0
2-3	220.0	63.0
3-4	220.0	63.0
4-5	220.0	63.0
5-6	220.0	63.0
6-7	220.0	63.0

Additional Dust Sources: None DEFAULT
Soil contribution conversion factor: 0.28
Air contribution conversion factor: 100.0

PAINT Intake: 0.00 ug Pb/day DEFAULT

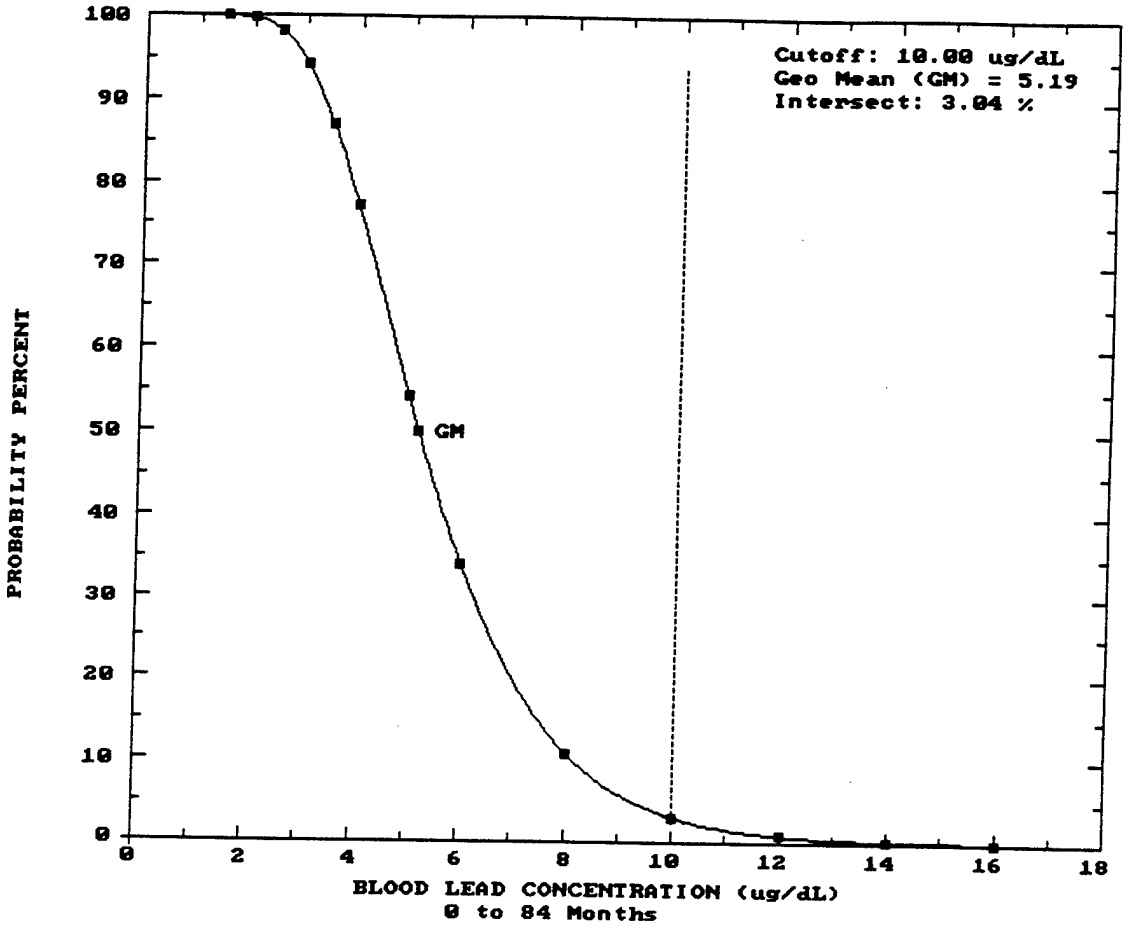
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 7.50 ug Pb/dL

TABLE 13 (cont.)
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR OPEN STORAGE AND SHELTER AREA

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	3.81	11.14	4.01
1-2:	4.58	17.45	4.01
2-3:	5.07	18.31	4.01
3-4:	5.27	18.41	4.01
4-5:	5.54	18.72	4.01
5-6:	5.68	19.55	4.01
6-7:	5.77	20.12	4.01

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.94	4.19	0.00	0.00
1-2:	2.96	10.47	0.00	0.00
2-3:	3.40	10.89	0.00	0.01
3-4:	3.29	11.10	0.00	0.01
4-5:	3.18	11.52	0.00	0.01
5-6:	3.38	12.15	0.00	0.01
6-7:	3.74	12.36	0.00	0.01

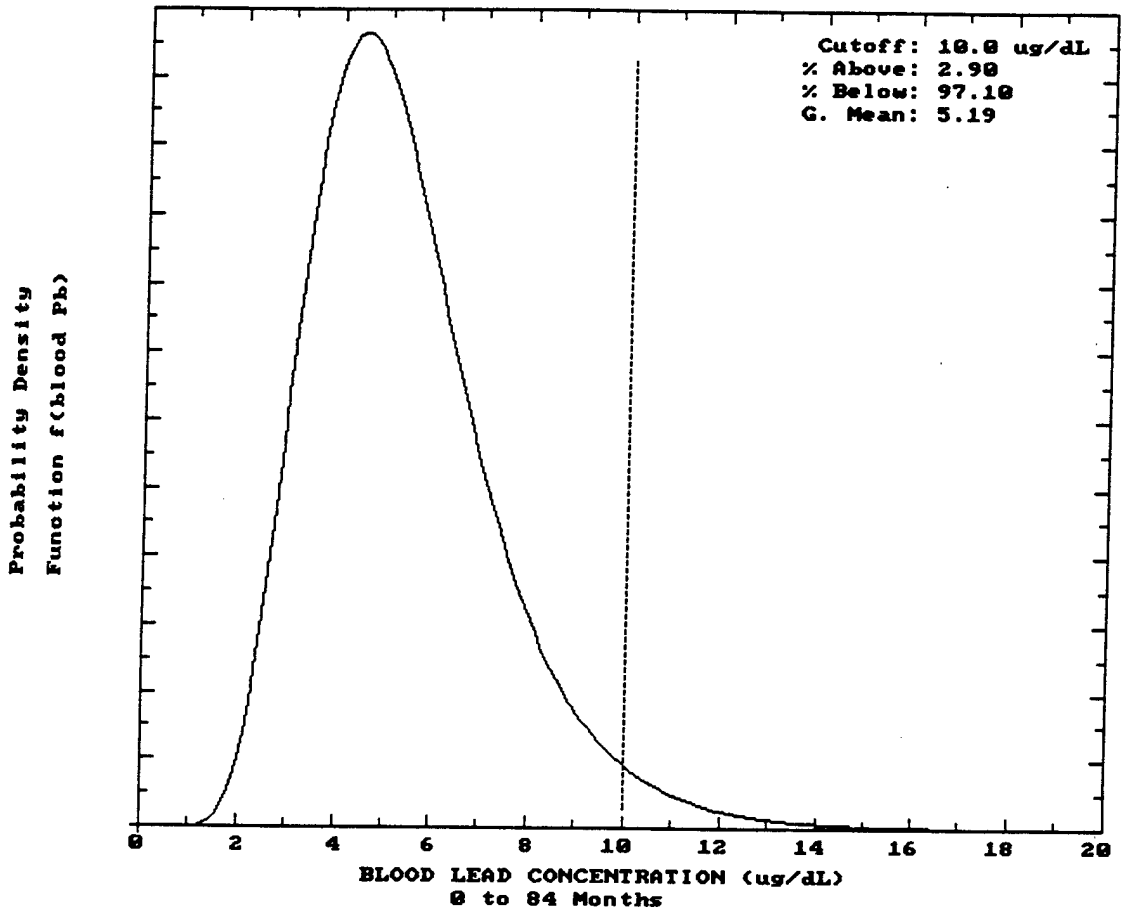


Metcalf & Eddy

PROBABLITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR OPEAN STORAGE AND SHELTER AREA

Project Number
012308-0005

Figure 27



Metcalf & Eddy	**PROBABLITY DENSITY OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR OPEAN STORAGE AND SHELTER AREA**	Project Number 012308-0005
		Figure 28

TABLE 14
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR SWMU 12

ABSORPTION METHODOLOGY: Non-Linear Active-Passive

AIR CONCENTRATION: 0.058 ug Pb/m3
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 41.90 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: Multiple Source Analysis

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	1430.0	406.2
1-2	1430.0	406.2
2-3	1430.0	406.2
3-4	1430.0	406.2
4-5	1430.0	406.2
5-6	1430.0	406.2
6-7	1430.0	406.2

Additional Dust Sources: None DEFAULT
Soil contribution conversion factor: 0.28
Air contribution conversion factor: 100.0

PAINT Intake: 0.00 ug Pb/day DEFAULT

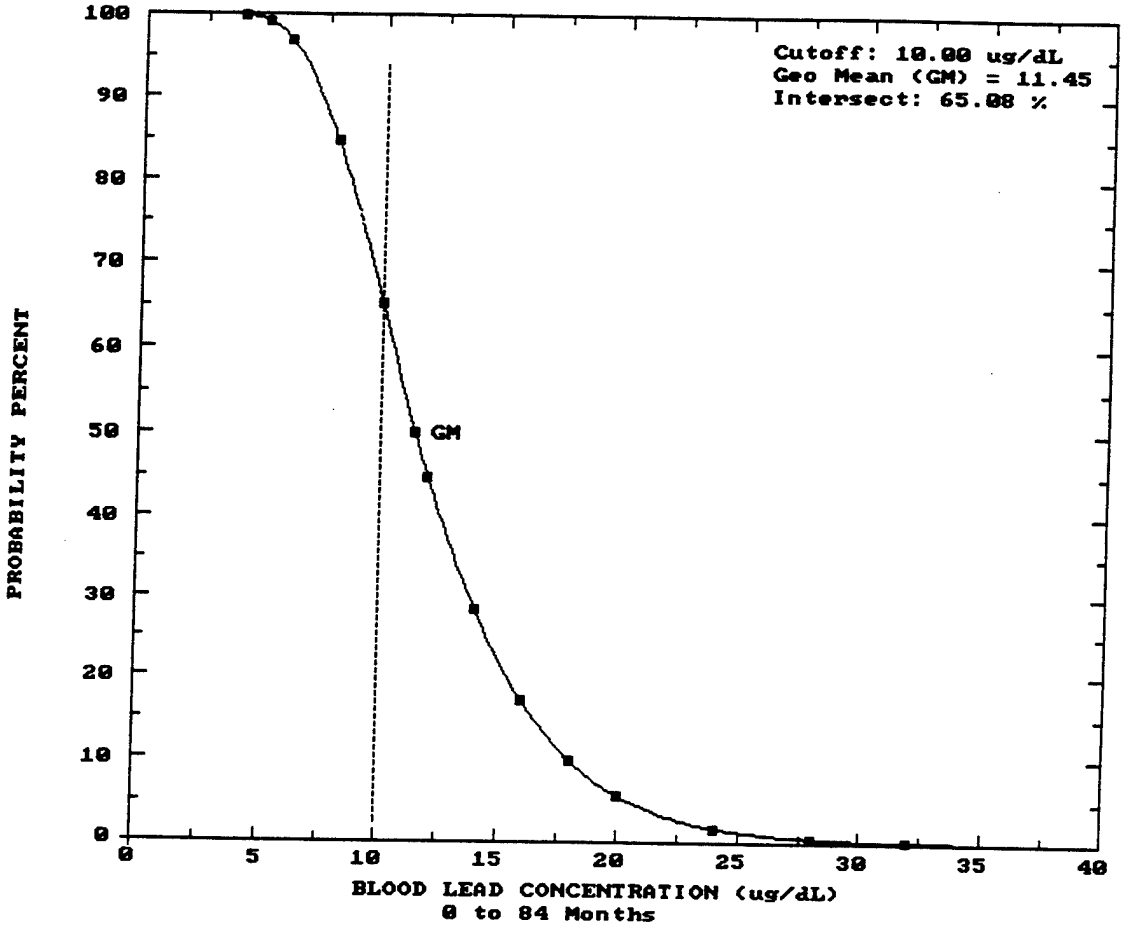
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 7.50 ug Pb/dL

TABLE 14 (cont.)
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR SWMU 12

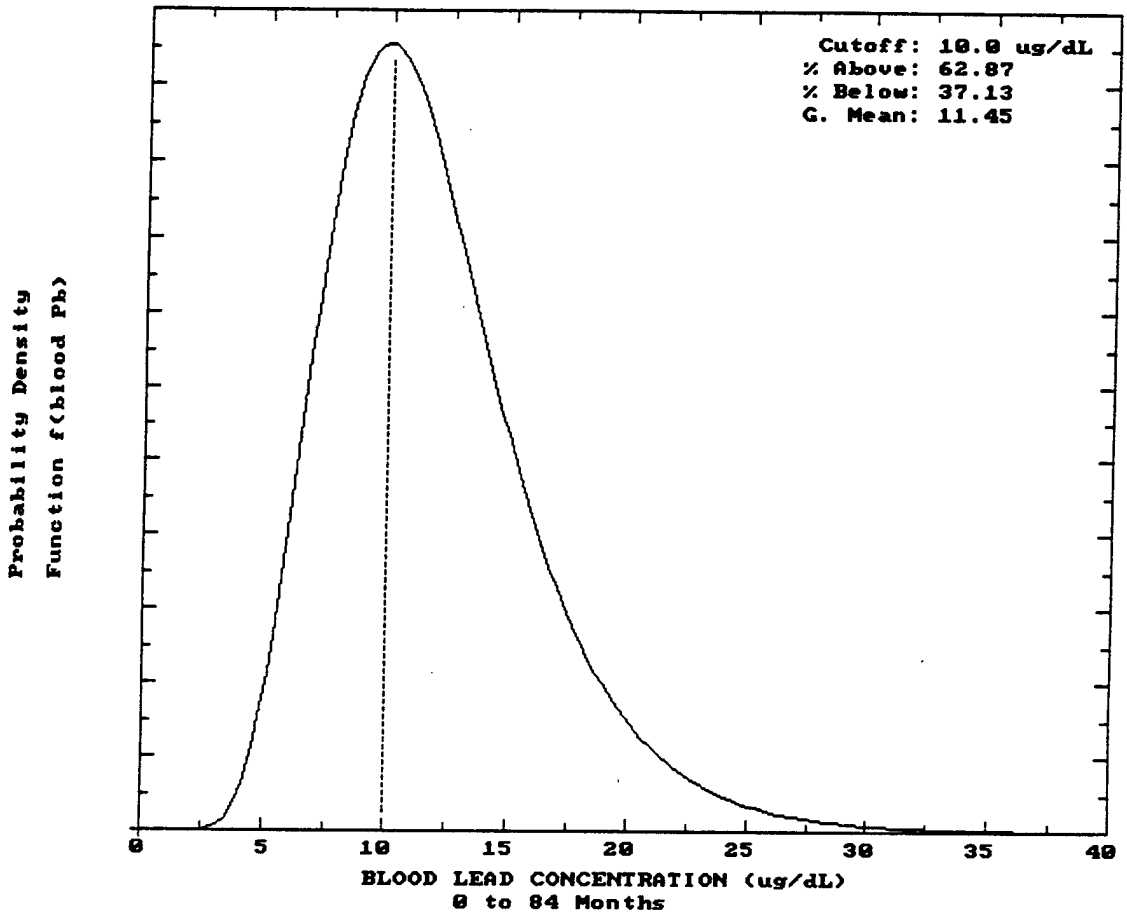
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	10.15	33.15	26.01
1-2:	11.08	39.46	26.01
2-3:	11.42	40.33	26.01
3-4:	11.67	40.43	26.01
4-5:	12.16	40.75	26.01
5-6:	12.27	41.59	26.01
6-7:	12.27	42.16	26.01

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.94	4.19	0.00	0.01
1-2:	2.96	10.47	0.00	0.02
2-3:	3.40	10.89	0.00	0.04
3-4:	3.29	11.10	0.00	0.04
4-5:	3.18	11.52	0.00	0.04
5-6:	3.38	12.15	0.00	0.05
6-7:	3.74	12.36	0.00	0.05



Metcalf & Eddy	**PROBABLITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR SWMU 12**	Project Number 012308-0005
		Figure 29



Metcalf & Eddy	**PROBABLITY DENSITY OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR SWMU 12**	Project Number 012308-0005
		Figure 30

TABLE 15
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR SWMU TELEPHONE POLE STORAGE AREA

ABSORPTION METHODOLOGY: Non-Linear Active-Passive

AIR CONCENTRATION: 0.002 ug Pb/m3
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 41.90 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: Multiple Source Analysis

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	1300.0	364.1
1-2	1300.0	364.1
2-3	1300.0	364.1
3-4	1300.0	364.1
4-5	1300.0	364.1
5-6	1300.0	364.1
6-7	1300.0	364.1

Additional Dust Sources: None DEFAULT
Soil contribution conversion factor: 0.28
Air contribution conversion factor: 100.0

PAINT Intake: 0.00 ug Pb/day DEFAULT

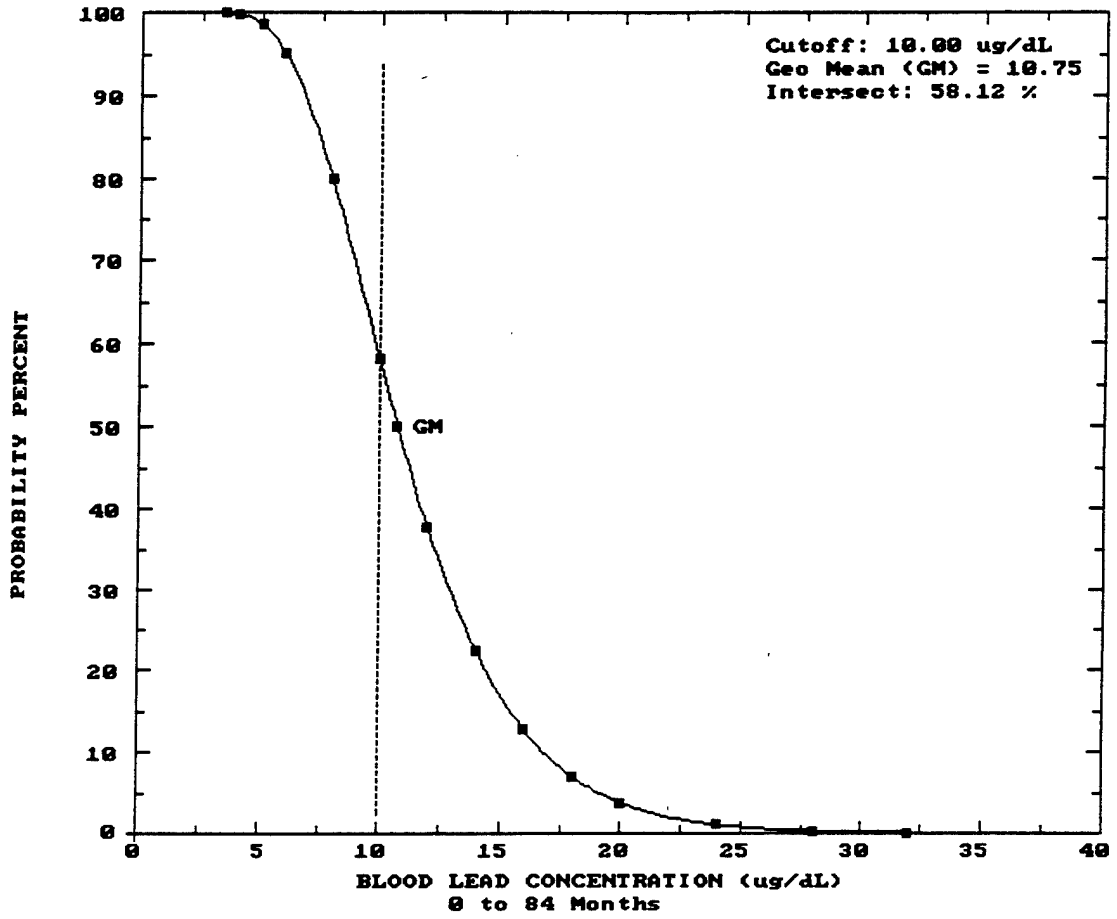
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 7.50 ug Pb/dL .

TABLE 15 (cont.)
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR SWMU TELEPHONE POLE STORAGE AREA

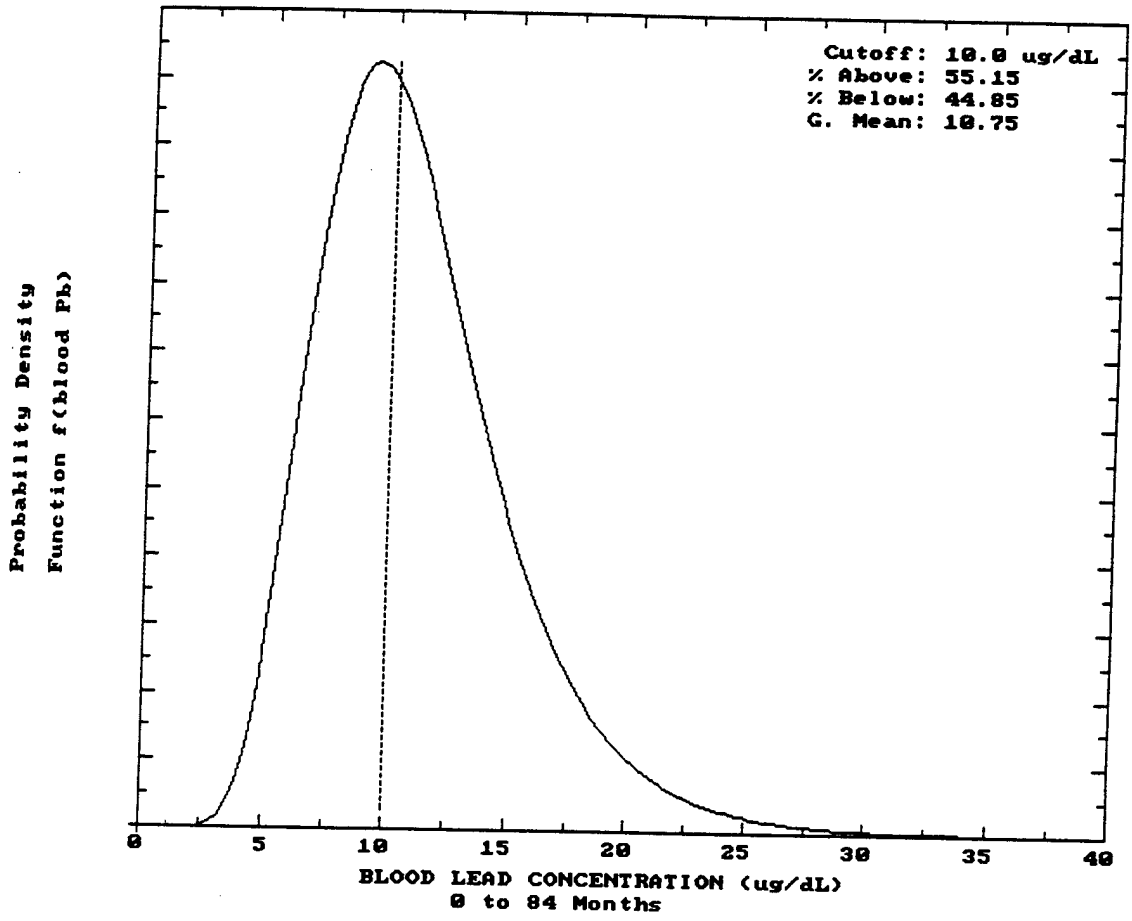
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	9.44	30.69	23.56
1-2:	10.35	36.99	23.56
2-3:	10.70	37.85	23.56
3-4:	10.95	37.95	23.56
4-5:	11.41	38.26	23.56
5-6:	11.53	39.09	23.56
6-7:	11.53	39.66	23.56

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.94	4.19	0.00	0.00
1-2:	2.96	10.47	0.00	0.00
2-3:	3.40	10.89	0.00	0.00
3-4:	3.29	11.10	0.00	0.00
4-5:	3.18	11.52	0.00	0.00
5-6:	3.38	12.15	0.00	0.00
6-7:	3.74	12.36	0.00	0.00



Metcalf & Eddy	**PROBABLITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR TELEPHONE POLE STORAGE AREA**	Project Number 012308-0005
		Figure 31



Metcalf & Eddy	**PROBABLITY DENSITY OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR TELEPHONE POLE STORAGE AREA**	Project Number 012308-0005
		Figure 32

TABLE 16
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL FOR BUILDING 63

ABSORPTION METHODOLOGY: Non-Linear Active-Passive

AIR CONCENTRATION: 0.011 ug Pb/m3
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 41.90 ug Pb/L
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: Multiple Source Analysis

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	1300.0	365.1
1-2	1300.0	365.1
2-3	1300.0	365.1
3-4	1300.0	365.1
4-5	1300.0	365.1
5-6	1300.0	365.1
6-7	1300.0	365.1

Additional Dust Sources: None DEFAULT
Soil contribution conversion factor: 0.28
Air contribution conversion factor: 100.0

PAINT Intake: 0.00 ug Pb/day DEFAULT

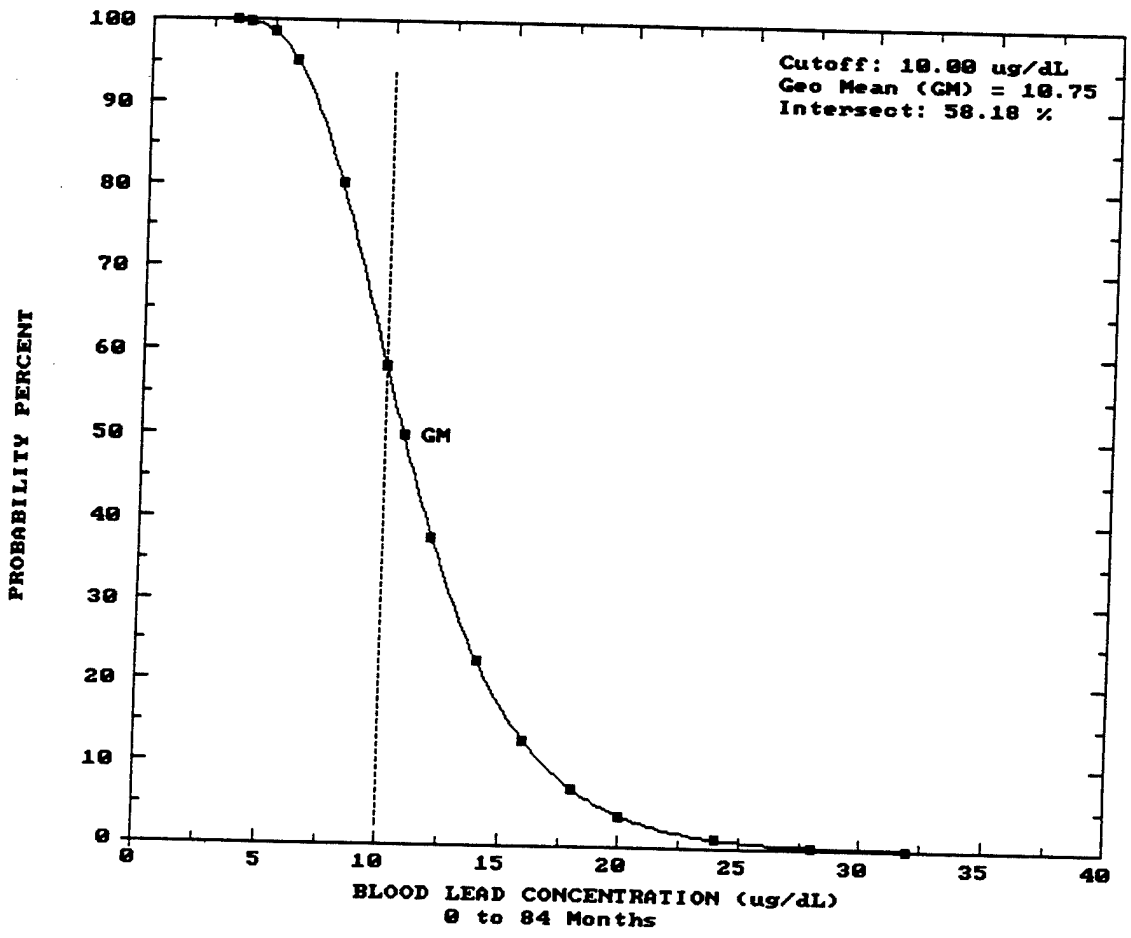
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 7.50 ug Pb/dL

TABLE 16 (cont.)
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE/BIOKINETIC MODEL
FOR BUILDING 63

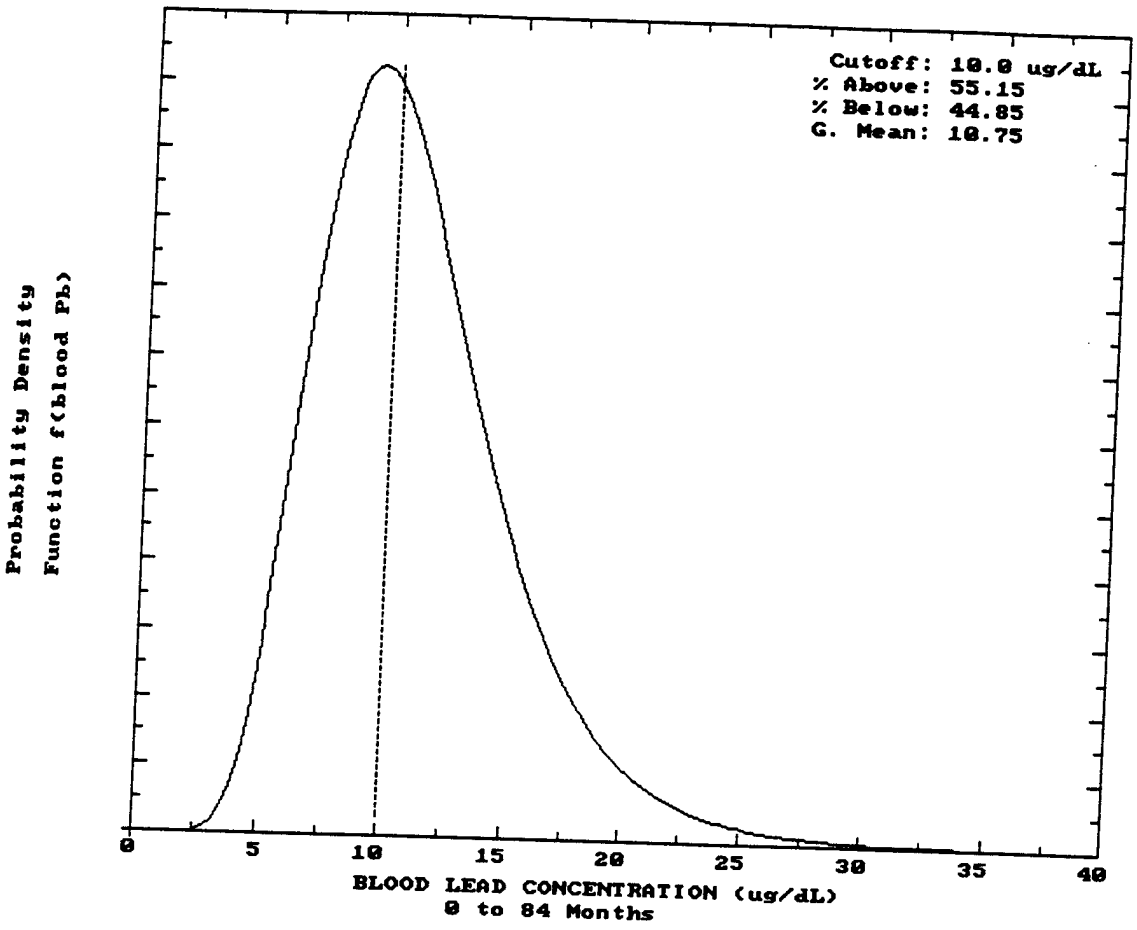
CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	9.45	30.71	23.57
1-2:	10.36	37.01	23.57
2-3:	10.71	37.87	23.57
3-4:	10.96	37.97	23.57
4-5:	11.42	38.28	23.57
5-6:	11.53	39.11	23.57
6-7:	11.54	39.68	23.57

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.94	4.19	0.00	0.00
1-2:	2.96	10.47	0.00	0.00
2-3:	3.40	10.89	0.00	0.01
3-4:	3.29	11.10	0.00	0.01
4-5:	3.18	11.52	0.00	0.01
5-6:	3.38	12.15	0.00	0.01
6-7:	3.74	12.36	0.00	0.01



Metcalf & Eddy	**PROBABLITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR BUILDING 63**	Project Number 012308-0005
		Figure 33



Metcalf & Eddy	**PROBABLITY DENSITY OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR BUILDING 63**	Project Number 012308-0005
		Figure 34

TABLE 17
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE BIOKINETIC MODEL FOR BUILDING 42

ABSORPTION METHODOLOGY: Non-Linear Active-Passive

AIR CONCENTRATION: 0.003 ug Pb/m3
Indoor AIR Pb Conc: 30.0 percent of outdoor.
Other AIR Parameters:

Age	Time Outdoors (hr)	Vent. Rate (m3/day)	Lung Abs. (%)
0-1	1.0	2.0	32.0
1-2	2.0	3.0	32.0
2-3	3.0	5.0	32.0
3-4	4.0	5.0	32.0
4-5	4.0	5.0	32.0
5-6	4.0	7.0	32.0
6-7	4.0	7.0	32.0

DIET: DEFAULT

DRINKING WATER Conc: 4.00 ug Pb/L DEFAULT
WATER Consumption: DEFAULT

SOIL & DUST:
Soil: constant conc.
Dust: Multiple Source Analysis

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
0-1	178.0	50.1
1-2	178.0	50.1
2-3	178.0	50.1
3-4	178.0	50.1
4-5	178.0	50.1
5-6	178.0	50.1
6-7	178.0	50.1

Additional Dust Sources: None DEFAULT
Soil contribution conversion factor: 0.28
Air contribution conversion factor: 100.0

PAINT Intake: 0.00 ug Pb/day DEFAULT

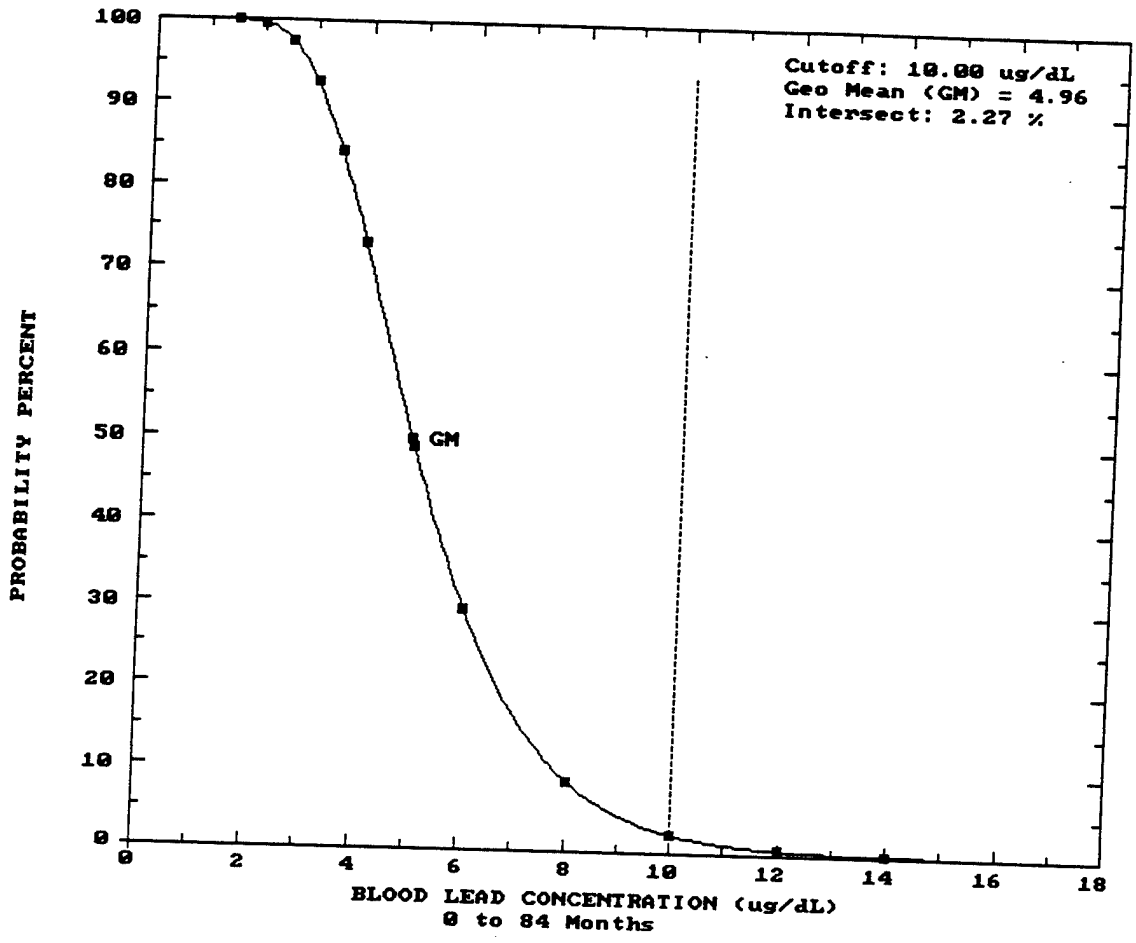
MATERNAL CONTRIBUTION: Infant Model
Maternal Blood Conc: 7.50 ug Pb/dL

TABLE 17 (cont.)
DEFAULT PARAMETER VALUES FOR LEAD UPTAKE BIOKINETIC MODEL
FOR BUILDING 42

CALCULATED BLOOD Pb and Pb UPTAKES:

YEAR	Blood Level (ug/dL)	Total Uptake (ug/day)	Soil+Dust Uptake (ug/day)
0.5-1:	2.49	6.57	3.23
1-2:	2.18	7.19	3.23
2-3:	2.16	7.67	3.23
3-4:	2.20	7.58	3.23
4-5:	2.26	7.51	3.23
5-6:	2.28	7.77	3.23
6-7:	2.32	8.15	3.23

YEAR	Diet Uptake (ug/day)	Water Uptake (ug/day)	Paint Uptake (ug/day)	Air Uptake (ug/day)
0.5-1:	2.94	0.40	0.00	0.00
1-2:	2.96	1.00	0.00	0.00
2-3:	3.40	1.04	0.00	0.00
3-4:	3.29	1.06	0.00	0.00
4-5:	3.18	1.10	0.00	0.00
5-6:	3.38	1.16	0.00	0.00
6-7:	3.74	1.18	0.00	0.00

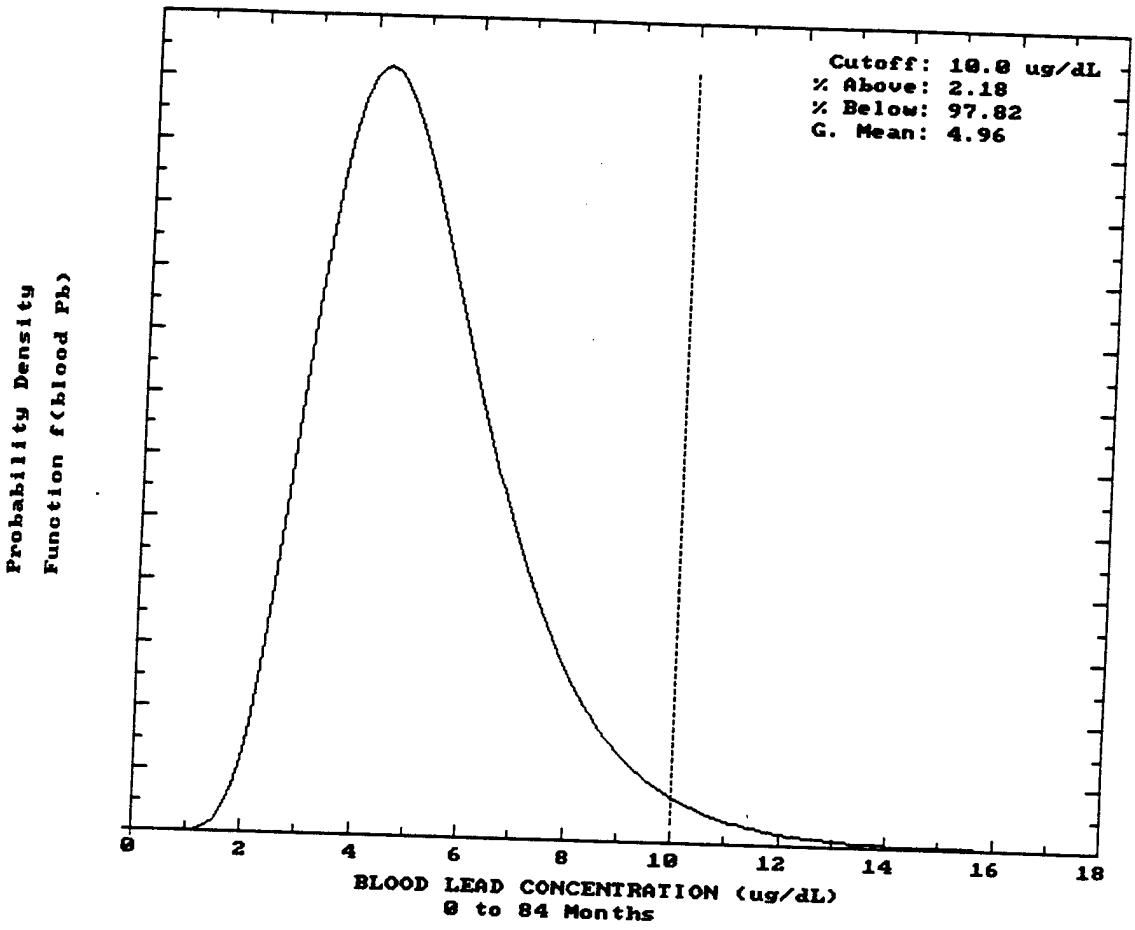


Metcalf & Eddy

PROBABLITY PLOT OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR BUILDING 42

Project Number
012308-0005

Figure 35



Metcalf & Eddy	**PROBABLITY DENSITY OF BLOOD LEAD LEVELS PREDICTED FOR CHILDREN FOR BUILDING 42**	Project Number 012308-0005
		Figure 36

APPENDIX S

REMEDIAL GOAL OPTIONS FOR CHEMICALS OF CONCERN IN GROUNDWATER

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1.0 DEVELOPMENT OF REMEDIAL GOAL OPTIONS

This section describes the methods by which human health Remedial Goal Options (RGOs) were developed for groundwater for the LBAD site. The RGOs were developed using the data that was collected during the RFI assessment. Chemicals which were considered to be of concern were included in this RGO development description. Chemicals were considered to be of concern if they contributed to carcinogenic risk above 1.0E-06 or noncarcinogenic hazard above 1.0. Guidance for the determination of the RGOs was based on the U.S. EPA's *Human Health Evaluation Manual* (HHEM), Part B: "*Development of Risk-Based Preliminary Remediation Goals*" (U.S. EPA, 1991), and U.S. EPA Region IV "*Supplemental Guidance/Development of Health Based Preliminary Remediation Goals, Remedial Goal Options, and Remediation Levels*", (U.S. EPA 1994).

Human Health Preliminary Remediation Goals

Table 1 presents the chemical-specific toxicity factors utilized in the derivation of the soil remedial goals. Groundwater RGOs were calculated for the LBAD site based on guidelines presented in the guidance cited above. Residential and occupational receptors were evaluated. However, inhalation of groundwater vapors and dermal contact with groundwater were not evaluated for the occupational receptor due to incomplete exposure routes. In addition, non-volatile chemicals were not evaluated for the residential inhalation route based on the inability of these chemicals to volatilize in a showering scenario.

Again, a residential receptor has been included as a conservative measure. Currently, groundwater is not used for potable water. However, future use of the land may include installation of wells and the use of groundwater for a potable water supply. As more information is collected concerning future land development, the assumptions for groundwater use may change. But for now, the inclusion of a residential receptor along with an occupational receptor in determining groundwater RGOs provides a range of levels that may assist in the selection of alternatives for the site.

Tables 2 and 3 present calculated chemical RGOs for occupational and residential exposure to groundwater, respectively. The RGOs for groundwater were derived on an exposure-specific and a chemical-specific basis. A target carcinogenic risk of 1E-04 to 1E-6 and hazard quotient of 0.1 to 10 were utilized for carcinogenic and non-cancer endpoints, respectively. In addition, Tables 5 through 7 provide the equations and parameter value assumptions utilized to calculate human health RGOs for residential and occupational exposure to groundwater.

TABLE 1
TOXICITY VALUES FOR CHEMICALS OF CONCERN IN GROUNDWATER
LEXINGTON BLUEGRASS ARMY DEPOT

	NONCARCINOGENIC RfDs						CANCER SLOPE FACTORS		
	ORAL RfD (MG/KG/DAY)		ADJUSTED ORAL (DERMAL) (MG/KG/DAY) (a)		INHALATION RfD (MG/KG/DAY)		ORAL SLOPE FACTOR	ADJUSTED ORAL (DERMAL) SLOPE FACTOR (b)	INHALATION SLOPE FACTOR
CHEMICAL	SUBCHRONIC	CHRONIC	SUBCHRONIC	CHRONIC	SUBCHRONIC	CHRONIC	(MG/KG/DAY)−1	(MG/KG/DAY)−1	(MG/KG/DAY)−1 (b)
Vinyl chloride	NA	NA	NA	NA	NA	NA	1.90E+00	2.40E+00	3.00E−01
Antimony	4.00E−04	4.00E−04	2.40E−04	2.40E−04	NA	NA	NA	NA	NA
Arsenic	3.00E−04	3.00E−04	2.85E−04	2.85E−04	NA	NA	1.50E+00	1.58E+00	5.00E+01
Barium	7.00E−02	7.00E−02	3.50E−03	3.50E−03	NA	NA	NA	NA	NA
Beryllium	5.00E−03	5.00E−03	5.00E−04	5.00E−04	NA	NA	4.30E+00	4.30E+01	8.40E+00
Lead(c)	1.40E−03	1.40E−03	7.00E−04	7.00E−04	4.30E−04	4.30E−04	NA	NA	NA
Manganese (water)	5.00E−03	5.00E−03	2.00E−04	2.00E−04	NA	NA	NA	NA	NA
Thallium	8.00E−04	8.00E−05	8.00E−05	8.00E−06	NA	NA	NA	NA	NA

*Sources: U.S. EPA, Integrated Risk Information System (IRIS) database accessed July 1994.
U.S. EPA Health Effects Assessment Tables (HEAST), Annual FY−1994 edition(Heast, 1994).

**NA = Not Available

(a) Adjusted oral toxicity values used for calculation of dermal hazards.
Adjustment of an administered to an absorbed dose RfD:
(Administered RfD) x (Oral Absorption Factor) = Absorbed Dose RfD.

(b) Adjusted oral toxicity values used for calculation of dermal risks.
Adjustment of an administered to an absorbed dose CSF:
(Administered CSF)−1 / (Oral Absorption Factor) = Absorbed Dose CSF
Oral absorption factors from chemical−specific Toxicological Profiles, Agency for Toxic Substances and Disease Registry, U.S. Public Health Service.

(c) The toxictiy values for lead were taken from the 1986 edition of HEAST per KDEP

TABLE 2 Risk–Based Remedial Goal Options for Occupational Exposure to Chemicals in Groundwater at the Lexington–Bluegrass Army Depot

	Toxicity Information*	
	Carcinogenic	Noncarcinogenic
CHEMICAL	SForal (mg/kg/day)–1	RFDoral (mg/kg/day)
Vinyl chloride	1.90E+00	NA
Antimony	NA	4.00E–04
Arsenic	1.50E+00	3.00E–04
Barium	NA	7.00E–02
Beryllium	4.30E+00	5.00E–03
Lead	NA	1.40E–03
Manganese (water)	NA	5.00E–03
Thallium	NA	8.00E–05

	Risk–Based Remedial Goal Options						
	Oral Exposure						
CHEMICAL	Carcin 1.0E–06 (mg/L)	Carcin 1.0E–05 (mg/L)	Carcin 1.0E–04 (mg/L)	Non–carc 0.1 (mg/L)	Non–carc 1.0 (mg/L)	Non–carc 10 (mg/L)	Maximum Contaminant Level (mg/L)
Vinyl chloride	1.51E–04	1.51E–03	1.51E–02	NA	NA	NA	0.002
Antimony	NA	NA	NA	4.09E–03	4.09E–02	4.09E–01	0.006
Arsenic	1.91E–04	1.91E–03	1.91E–02	3.07E–03	3.07E–02	3.07E–01	0.05
Barium	NA	NA	NA	7.15E–01	7.15E+00	7.15E+01	2
Beryllium	6.65E–05	6.65E–04	6.65E–03	5.11E–02	5.11E–01	5.11E+00	0.004
Lead	NA	NA	NA	1.43E–02	1.43E–01	1.43E+00	0.015**
Manganese (water)	NA	NA	NA	5.11E–02	5.11E–01	5.11E+00	0.05***
Thallium	NA	NA	NA	8.18E–04	8.18E–03	8.18E–02	0.002

NA – Not Available

* Toxicity Information Sources: Integrated Risk Information System (IRIS, Accessed July, 1994); and Health Effects Assessment Summary Tables (1993)

** – Lead action Level

*** – Secondary Maximum Contaminant Level

TABLE 3 Risk–Based Remedial Goal Options for Residential Exposure to Chemicals in Groundwater at the Lexington–Bluegrass Army Depot

	Toxicity Information*					
	Carcinogenic			Noncarcinogenic		
CHEMICAL	SForal (mg/kg/day)–1	SFdermal (mg/kg/day)–1	SFinh (mg/kg/day)–1	RFDoral (mg/kg/day)	RFDdermal (mg/kg/day)	RFDinh (mg/kg/day)
Vinyl chloride	1.90E+00	2.40E+00	3.00E–01	NA	NA	NA
Antimony	NA	NA	NA	4.00E–04	2.40E–04	NA
Arsenic	1.50E+00	1.60E+00	5.00E+01	3.00E–04	2.85E–04	NA
Barium	NA	NA	NA	7.00E–02	3.50E–03	NA
Beryllium	4.30E+00	4.30E+01	8.40E+00	5.00E–03	5.00E–04	NA
Lead	NA	NA	NA	1.40E–03	7.00E–04	4.30E–04
Manganese (water)	NA	NA	NA	5.00E–03	2.00E–04	NA
Thallium	NA	NA	NA	8.00E–05	8.00E–06	NA

	Risk–Based Remedial Goal Options						
	Oral, Dermal, and Inhalation Exposure						
CHEMICAL	Carcin 1.0E–06 (mg/L)	Carcin 1.0E–05 (mg/L)	Carcin 1.0E–04 (mg/L)	Non–carc 0.1 (mg/L)	Non–carc 1.0 (mg/L)	Non–carc 10 (mg/L)	Maximum Contaminant Level (mg/L)
Vinyl chloride	2.79E–05	2.79E–04	2.79E–03	NA	NA	NA	0.002
Antimony	NA	NA	NA	1.46E–03	1.46E–02	1.21E–01	0.006
Arsenic	5.67E–05	5.67E–04	5.67E–03	1.09E–03	1.09E–02	9.11E–02	0.05
Barium	NA	NA	NA	2.47E–01	2.47E+00	2.06E+01	2
Beryllium	1.95E–05	1.49E–02	1.95E–03	1.80E–02	1.80E–01	1.50E+00	0.004
Lead	NA	NA	NA	5.09E–03	5.09E–02	4.24E–01	0.015**
Manganese (water)	NA	NA	NA	1.75E–02	1.75E–01	1.46E+00	0.05***
Thallium	NA	NA	NA	2.87E–04	2.87E–03	2.39E–02	0.002

NA – Not Available

* Toxicity Information Sources: Integrated Risk Information System (IRIS, Accessed July, 1994); and Health Effects Assessment Summary Tables (1993)

** – Lead action Level

*** – Secondary Maximum Contaminant Level

TABLE 4
GROUNDWATER REMEDIAL GOAL OPTIONS DEVELOPMENT
RESIDENTIAL RECEPTOR
ORAL, INHALATION AND DERMAL EXPOSURE:
CHEMICAL CARCINOGENIC EFFECTS
(U.S. EPA, 1991, U.S. EPA, 1994)

$$C_W = \frac{1E\text{-}06 \ X \ BW \ X \ AT \ X \ 365}{(EF \ X \ ED) \ [(SF_0 \ X \ IR_0) + (SF_{DERM} \ X \ SA \ X \ PC \ X \ ET \ X \ CF) + (SF_{INH} \ X \ IR_{INH} \ X \ K)]}$$

Parameter	Parameter Description	Parameter Value Assumption
C_W	Chemical Concentration in Water (mg/L)	Chemical-Specific
1E-06	Target Risk Level (unitless)	1E-06 to 1E-04
BW	Body Weight (kg)	70 kg
AT	Averaging Time (years)	70 yr
365	Days/Year	365 days
EF	Exposure Frequency (days/year)	350 days/yr
ED	Exposure Duration	30 yr
SF_o	Oral Cancer Slope Factor $(mg/kg/day)^{-1}$	Chemical-Specific
SF_{DERM}	Dermal Cancer Slope Factor $(mg/kg/day)^{-1}$	Chemical-Specific
SA	Exposed Skin Surface Area (cm^2)	19,400
PC	Dermal Permeability Constant (cm/hr)	Chemical Specific
ET	Shower Exposure Time (hr/day)	0.17
CF	Conversion Factor (L/cm^3)	1.0E-3
IR_o	Oral Ingestion Rate (liters/day)	2 liters/day
SF_{INH}	Inhalation Cancer Slope Factor $(mg/kg/day)^{-1}$ (a)	Chemical-Specific
IR_{INH}	Inhalation Intake Rate (m^3/day)	15 m^3/day
K	Volatilization Factor (unitless) (b)	0.5

(a) Risk from groundwater inhalation is assumed to be relevant only for chemicals that easily volatilize.

(b) The Volatilization Factor of 0.0005 is multiplied by a conversion factor of 1000 L/m^3 so that the resulting air concentration is expressed in mg/m^3.

TABLE 5
GROUNDWATER REMEDIAL GOAL OPTIONS DEVELOPMENT
RESIDENTIAL RECEPTOR
ORAL, INHALATION AND DERMAL EXPOSURE:
NON-CARCINOGENIC EFFECTS
(U.S. EPA, 1991, U.S. EPA, 1994)

$$C_W = \frac{1.0 \; X \; BW \; X \; AT \; X \; 365}{(EF \; X \; ED) \; [((1/RfD_0) \; X \; IR_0) + ((1/RfD_{DERM}) \; X \; SA \; X \; PC \; X \; ET \; X \; (F) + ((1/RfD_{INH}) \; X \; (IR_{INH} \; X \; K)]}$$

Parameter	Parameter Description	Parameter Value Assumption
C_W	Chemical Concentration in Water (mg/L)	Chemical-Specific
1.0	Target Hazard Level (unitless)	0.1 - 10.0
BW	Body Weight (kg)	70 kg
AT	Averaging Time (years)	30 yr
365	Days/Year	365 days
EF	Exposure Frequency (days/year)	350 days/yr
ED	Exposure Duration	30 yr
RfD_o	Oral Reference Dose (mg/kg/day)	Chemical-Specific
RfD_{DERM}	Dermal Reference Dose (mg/kg/day)	Chemical-Specific
IR_o	Oral Ingestion Rate (liters/day)	2 liters/day
RfD_{INH}	Inhalation Reference Dose (mg/kg/day) (a)	Chemical-Specific
SA	Exposed Skin Surface Area (cm^2)	19,400
PC	Dermal Permeability Constant (cm/hr)	Chemical Specific
ET	Shower Exposure Time (hr/day)	0.17
CF	Conversion Facto (L/cm^3)	1.0E-3
IR_{INH}	Inhalation Intake Rate (m^3/day)	15 m^3/day
K	Volatilization Factor (unitless) (b)	0.5

(a) Risk from groundwater inhalation is assumed to be relevant only for chemicals that easily volatilize.

(b) The Volatilization Factor of 0.0005 is multiplied by a conversion factor of 1000 L/m^3 so that the resulting air concentration is expressed in mg/m^3.

TABLE 6
GROUNDWATER REMEDIAL GOAL OPTIONS DEVELOPMENT
OCCUPATIONAL RECEPTOR
ORAL EXPOSURE:
CHEMICAL CARCINOGENIC EFFECTS
(U.S. EPA, 1991, U.S. EPA 1994)

$$C_W = \frac{1E\text{-}06 \ X \ BW \ X \ AT \ X \ 365}{SF_0 \ X \ EF \ X \ ED \ X \ IR}$$

Parameter	Parameter Description	Parameter Value Assumption
C_W	Chemical Concentration in Water (mg/L)	Chemical-Specific
1E-06	Target Risk Level (unitless)	1E-06 to 1E-04
BW	Body Weight (kg)	70 kg
AT	Averaging Time (years)	70 yr
365	Days/Year	365 days
SF_0	Oral Cancer Slope Factor $(mg/kg/day)^{-1}$	Chemical-Specific
EF	Exposure Frequency (days/year)	250 days/yr
ED	Exposure Duration (years)	25 years
IR	Ingestion Rate (liters/day)	1 liter/day

TABLE 7
GROUNDWATER REMEDIAL GOAL OPTIONS DEVELOPMENT
OCCUPATIONAL RECEPTOR
ORAL EXPOSURE:
CHEMICAL NON-CARCINOGENIC EFFECTS
(U.S. EPA, 1991, U.S. EPA, 1994)

$$C_W = \frac{1.0 \ X \ BW \ X \ AT \ X \ 365}{1/RfD_0 \ X \ ED \ X \ EF \ X \ IR}$$

Parameter	Parameter Description	Parameter Value Assumption
C_w	Chemical Concentration in Water (mg/L)	Chemical-Specific
1.0	Target Hazard Level (unitless)	0.1 to 10.0
BW	Body Weight (kg)	70 kg
AT	Averaging Time (years)	25 yr
365	Days/Year	365 days
RfD_0	Oral Reference Dose (mg/kg/day)	Chemical-Specific
EF	Exposure Frequency (days/year)	250 days/yr
ED	Exposure Duration (years)	25 years
IR	Ingestion Rate (liters/day)	1 liter/day

REFERENCES

U.S. EPA. 1991. U.S. Environmental Protection Agency. Human Health Evaluation Manual Part B: "Development of Risk-Based Preliminary Remediation Goals." OSWER Directive 9285.7-01B. December 13, 1991.

U.S. EPA. 1994. U.S. Environmental Protection Agency, Region IV. Supplemental Guidance to RAGS: "Development of Health-Based Preliminary Remediation Goals, Remedial Goal Options, and Remediation Levels." October 12, 1994.

APPENDIX T
LBAD SITE ARARS

TABLE T–1
CHEMICAL–SPECIFIC POTENTIAL APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS FOR CHEMICALS DETECTED IN THE LBAD GROUNDWATER

Chemicals Detected in LBAD Groundwater	EPA Ambient Water Quality Criteria Human Health (Water and Fish Ingestion) (ug/L) I	EPA Ambient Water Quality Criteria Human Health (Fish Ingestion) (ug/L) II	EPA Ambient Water Quality Criteria Freshwater Aquatic Organisms Acute/Chronic (ug/L) III	EPA Maximum Contaminant Levels (ug/L) IV	Kentucky Water Quality Criteria		
					Domestic Water Supply Source Criteria (ug/L)	Warm Water Aquatic Habitat Acute/Chronic (a) (ug/L)	Human Health Protection from Consumption of Fish (ug/L)
1,1–Dichloroethane	NA	NA	NA	NA	NA	NA	NA
1,2–Dichloroethenes	NA	NA	NA	70	NA	NA	NA
1,3–Dimethylbenzene	NA	NA	NA	NA	NA	NA	NA
2,4–Dimethylphenol	400	2296	212/21.2	NA	3090	NA	NA
Acetone	NA	NA	NA	NA	NA	NA	NA
alpha–BHC	NA	NA	NA	NA	NA	NA	NA
alpha–Endosulfan	NA	NA	NA	NA	74	NA	NA
Benzene	1.8	71.28	530/53	5	1.2	NA	71
Bis(2–ethylhexyl)phthalate	1.76	5.92	1110/0.3	6	15000	NA	NA
Carbon tetrachloride	0.254	4.42	3520/352	5	0.4	NA	6.94
Chloromethane	NA	NA	NA	NA	NA	NA	NA
DDT	NA	NA	NA	NA	0.000024	1.1/0.001	0.000024
delta–BHC	NA	NA	NA	NA	NA	NA	NA
Ethylbenzene	NA	NA	NA	700	3100	NA	29000
Lindane	NA	NA	NA	0.2	0.019	2/0.080	NA
Methyl isobutyl ketone	NA	NA	NA	NA	NA	NA	NA
Phenol	300	4615385	1020/256	NA	3500	NA	NA
Tetrachloroethene	NA	NA	NA	5	0.8	NA	NA
Toluene	6764.8	201294	1750/175	1000	14300	NA	424000
Trichloroethene	NA	NA	NA	5	2.7	NA	NA
Vinyl chloride	NA	NA	NA	2	NA	NA	NA
Xylenes	NA	NA	NA	10000	NA	NA	NA

TABLE T–1 (Cont'd)

Chemicals Detected in LBAD Groundwater	EPA Ambient Water Quality Criteria Human Health (Water and Fish Ingestion) (ug/L) I	EPA Ambient Water Quality Criteria Human Health (Fish Ingestion) (ug/L) II	EPA Ambient Water Quality Criteria Freshwater Aquatic Organisms Acute/Chronic (ug/L) III	EPA Maximum Contaminant Levels (ug/L) IV	Kentucky Water Quality Criteria		
					Domestic Water Supply Source Criteria (ug/L)	Warm Water Aquatic Acute/Chronic (a) (ug/L)	Human Health Protection from Consumption of Fish (ug/L)
Aluminum	NA	NA	750/8.7	50–200**	NA	NA	NA
Antimony	14	4308	1300/160	6	146	NA	45000
Arsenic	0.0175	0.14	360/190	50	NA	NA/50	NA
Barium	2000	NA	NA	2000	1000	NA	NA
Beryllium	0.0077	0.132	16/0.53	4	6.8	NA/11	0.117
Boron	NA	NA	NA/750	NA	NA	NA	NA
Cadmium	5	168	1.79/0.66	5	10	3.92/1.13	NA
Calcium	NA	NA	NA	NA	NA	NA	NA
Chromium (III)	33300	673077	984/117	100 (Total)	33000; 50(Total)	1736.5/207.0	670000
Cobalt	NA	NA	NA	NA	NA	NA	NA
Copper	1000	NA	9.2/6.5	1300 *	1000	17.7/11.8	NA
Iron	300	NA	NA/1	300**	NA	4.0/1.0	NA
Lead	50	NA	33.8/1.3	15 *	50	81.6/3.18	NA
Magnesium	NA	NA	NA	NA	NA	NA	NA
Manganese	30	100	NA	50**	50	NA	NA
Mercury	0.151	0.153	2.4/0.012	2	0.144	2.4/0.012	0.146
Molybdenum	NA	NA	NA	NA	NA	NA	NA
Nickel	607	4584	789/87	100	610	1418.2/157.7	4600
Potassium	NA	NA	NA	NA	NA	NA	NA
Sodium	NA	NA	NA	NA	NA	NA	NA
Tellurium	NA	NA	NA	NA	NA	NA	NA
Thallium	13	48	140/4	2	13	NA	48
Tin	NA	NA	NA	NA	NA	NA	NA
Vanadium	NA	NA	NA	NA	NA	NA	NA
Zinc	5000	NA	65/59	5000**	NA	NA	NA

(a) Where applicable, a water hardness concentration of 100 mg/l (as CaCO3) was used to calculate the criteria values.

NA – Not Available

* – Action Level

** – Indicates the value is a secondary maximum contaminant level (SMCL)

Note: Chemicals which are shaded have no State of Kentucky or USEPA criteria concentrations available.

TABLE T-2
LOCATION-SPECIFIC POTENTIAL APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS
LEXINGTON-BLUEGRASS ARMY DEPOT

Location	Requirement	Prerequisite	Citation
Critical habitat for a threatened/endangered species	Identify activities which may impact listed species. Actions may not threaten the continued existence of a listed species or destroy critical habitat	Species/habitat listed as endangered or threatened	Endangered Species Act 50 CFR 402.02; 50 CFR 402.01
Stream or river	Protect fish or wildlife	Any diversion, channelling, etc., which modifies a stream or river, or impacts fish or wildlife	Fish and Wildlife Coordination Act 16 USC 661 et seq. 40 CFR 6.302
Historic property owned or controlled by Federal agency	Action to preserve historic properties; planning of proposed actions to minimize harm to National Historic Landmarks	Property included in or eligible for the National Register of Historic Places	National Historic Preservation Act, Section 106 (16 USC 479 et seq.); 36 CFR Part 800
Flood Plains or Wetlands	Protection of wetlands and prevention of activities in flood plains.	Construction or remediation activities performed in a flood plain or wetland.	40 CFR Part 6, Appendix A, and Executive Orders 11988 and 11990.

TABLE T-3

SELECTED FEDERAL ACTION-SPECIFIC POTENTIAL APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS a/

LEXINGTON-BLUEGRASS ARMY DEPOT

Actions	Requirement	Prerequisites for Applicability b/ c/	Citation
Closure with No Post-Closure Care (e.g., Clean Closure)	General performance standard requires elimination of need for further maintenance and control; elimination of post-closure escape of hazardous waste, hazardous constituents, leachate, contaminated run-off, or hazardous waste decomposition products.	Applicable to land-based unit containing hazardous waste. c/ Applicable to RCRA hazardous waste (listed or characteristic) placed at site after the effective date of the requirements, or placed into another unit. Not applicable to material treated, stored, or disposed only before the effective date of the requirements, or if treated in-situ, or consolidated within area of contamination. Designed for cleanup that will not require long-term management. Designed for cleanup to health-based standards.	40 CFR 264.111
	Disposal or decontamination of equipment, structures, and soils. Removal or decontamination of all residues, contaminated containment system components (e.g., liners, dikes), contaminated subsoils, and structures and equipment contaminated with waste and leachate, and management of them as hazardous waste.	May apply to surface impoundments and container or tank liners and hazardous waste residues, and to contaminated soil, including soil from dredging or soil disturbed in the course of drilling or excavation, and returned to land.	40 CFR 264.111 40 CFR 264.178 40 CFR 264.197 40 CFR 264.288(o)(1) and 40 CFR 264.258
	Meet health-based levels at unit. d/		40 CFR 244.111
Closure with Waste In Place	Eliminate free liquids by removal or solidification. Stabilization of remaining waste and waste residues to support cover	Applicable to land disposal of hazardous waste. c/ Applicable to RCRA hazardous waste (listed or characteristic) placed at site after the effective date of the requirements, or placed into another unit. Not applicable to material treated, stored, or disposed only before the effective date of the requirements, or if treated in-situ or consolidated within area of contamination	40 CFR 264.228(a)(2) 40 CFR 264.228(a)(2) 40 CFR 264.258(b)

TABLE T-3 (continued)

SELECTED FEDERAL ACTION-SPECIFIC POTENTIAL APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS a/

LEXINGTON-BLUEGRASS ARMY DEPOT

Actions	Requirement	Prerequisites for Applicability b/ c/	Citation
Closure with Waste In Place (continued)	Installation of final cover to provide long-term minimization of infiltration (see Capping).		40 CFR 264.310
	30-year post-closure care and ground water monitoring. e/		40 CFR 264.310
Consolidation between Units	With respect to the waste that is moved, see requirements in the appropriate sections: Capping, Closure with Waste in Operation and Maintenance, Tank Storage, and Treatment.	Movement of hazardous waste and placement into another unit. d/	See capping, Closure with Waste in Place, Container a New Landfill On-Site, Construction of a New Surface Impoundment On-Site, Incineration (On-Site), Land Treatment, Operation and Maintenance, Tank Storage, and Treatment in this table.
Container Storage	Containers of RCRA hazardous waste must be:	Storage of RCRA hazardous waste (listed or characteristic) not meeting small quantity generator criteria held for a temporary period greater than 90 days before treatment, disposal, or storage elsewhere (40 CFR 264.10) in a container (i.e., any portable device in which a material is stored, transported, disposed of, or handled). A generator who accumulates or stores hazardous waste on-site for 90 days or less in compliance with 40 CFR 262.34(a)(1-4) is not subject to full RCRA storage requirements. Small quantity generators are not subject to the 90 day limit (40 CFR 262.34(c), (d), and (e).	
	• Maintained in good condition;		40 CFR 264.171
	• Compatible with hazardous waste to be stored;		40 CFR 264.172
	• Closed during storage (except to add or remove waste.		40 CFR 264.173
	Inspect container storage areas weekly for deterioration		40 CFR 264.174

TABLE T-3 (continued)

SELECTED FEDERAL ACTION-SPECIFIC POTENTIAL APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS a/

LEXINGTON-BLUEGRASS ARMY DEPOT

Actions	Requirement	Prerequisites for Applicability b/ c/	Citation
Container Storage (continued)	Place containers on a sloped, crack-free base, and protect from contact with accumulated liquid. Provide containment system with a capacity of 10 percent of the volume of containers of free liquids. Remove spilled or leaked waste in a timely manner to prevent overflow of the containment system		40 CFR 264.175
	Keep containers of ignitable or reactive waste at least 50 feet from the facility's property line.		40 CFR 264.176
	Keep incompatible materials separate. Separate incompatible materials stored near each other by a dike or other barrier.		40 CFR 264.177
	At closure, remove all hazardous waste and residues from the containment system, and decontaminate or remove all containers, liners.		40 CFR 264.178
	Storage of banned waste must be in accordance with 40 CFR 268. When such storage occurs beyond one year, the owner/operator bears the burden of proving that such storage is solely for the purpose of accumulating sufficient quantity to allow for proper recovery, treatment, and disposal.		40 CFR 268.50

TABLE T-3 (continued)

SELECTED FEDERAL ACTION-SPECIFIC POTENTIAL APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS a/

LEXINGTON-BLUEGRASS ARMY DEPOT

Actions	Requirement	Prerequisites for Applicability b/ c/	Citation
Dike Stabilization	Design and operate facility to prevent overtopping due to overfilling; wind and wave action; rainfall, run-on; malfunctions of level controllers, alarms, and other equipment; and human error.	Existing surface impoundment containing hazardous waste, or creation of a new surface impoundment.	40 CFR 264.221
	Construct dikes with sufficient strength to prevent massive failure.		40 CFR 264.221
	Inspect liners and cover system during and after construction.		40 CFR 264.226
	Inspect weekly for proper operation and integrity of the containment devices.		40 CFR 264.226
	Remove surface impoundment from operation if the dike leaks or there is a sudden drop in liquid level.		40 CFR 264.227
	At closure, remove or decontaminate all waste residues and contaminated materials. Otherwise, free liquids must be removed, the remaining wastes stabilized, and the facility closed in the same manner as a landfill.		40 CFR 264.228
	Manage ignitable or reactive wastes so that it is protected from materials or conditions that may cause it to ignite or react.		40 CFR 264.227

TABLE T-3 (continued)

SELECTED FEDERAL ACTION-SPECIFIC POTENTIAL APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS a/

LEXINGTON-BLUEGRASS ARMY DEPOT

Actions	Requirement	Prerequisites for Applicability b/ c/	Citation
Discharge of Treatment System Effluent	Best Available Technology:		
	Use of best available technology (BAT) economically achievable is required to control toxic and non-conventional pollutants. Use of best conventional pollutant control technology (BCT) is required to control conventional pollutants. Technology-based limitations may be determined on a case-by-case basis.	Point Source discharge to waters of the United States. g/ h/	40 CFR 122.44(a)
	Water Quality Standards:		
	Applicable Federally approved State water quality standards must be complied with, These standards may be in addition to or more stringent than other Federal standards under the CWA. i/		40 CFR 122.44 and State regulations approved under 40 CFR 131
	Discharge limitations must be established at more stringent levels than technology-based standards for toxic pollutants.		40 CFR 122.44(e)
	Best Management Practices:		
	Develop and implement a Best Management Practice program to prevent the release of toxic constituents to surface waters.		40 CFR 125.100
	The Best Management Practices program must:	Discharge to waters of the U.S. i/	40 CFR 125.104

TABLE T-3 (continued)

SELECTED FEDERAL ACTION-SPECIFIC POTENTIAL APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS a/

LEXINGTON-BLUEGRASS ARMY DEPOT

Actions	Requirement	Prerequisites for Applicability b/ c/	Citation
Discharge of Treatment System Effluent (continued)	• Establish specific procedures for the control of toxic and hazardous pollutant spills • Include a prediction of direction, rate of flow, and total quantity of toxic pollutants where experience indicates a reasonable potential for equipment failure. • Assure proper management of solid and hazardous waste in accordance with Regulations promulgated under RCRA. <u>Monitoring Requirements</u>: Discharge must be monitored to assure compliance. Discharge will monitor: • The mass of each pollutant • The volume of effluent • Frequency of discharge and other measurements as appropriate. Approved test methods for waste constituent to be monitored must be followed. Detailed requirements for analytical procedures and quality controls are provided. Sample preservation procedures, container materials, and maximum allowable holding times are prescribed. Comply with additional substantive conditions such as:		40 CFR 122.41(i) 40 CFR 136.1-136.4 40 CFR 122.41(i)

TABLE T-3 (continued)

SELECTED FEDERAL ACTION-SPECIFIC POTENTIAL APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS a/

LEXINGTON-BLUEGRASS ARMY DEPOT

Actions	Requirement	Prerequisites for Applicability b/ c/	Citation
Discharge of Treatment System Effluent (Continued)	• Duty to mitigate any adverse effects of any discharge; and • Proper operation and maintenance of treatment system.		
Discharge to Publicly Owned Treatment Works (POTW) (off-site activity, see footnote j/)	Discharge of pollutants that pass through the POTW without treatment, interfere with POTW operation, contaminate POTW sludge, or endanger health/safety of POTW workers, is prohibited. Specific prohibitions preclude the discharge of pollutants to POTWs that: • Create a fire or explosion hazard in the POTW • Will cause corrosive structural change to POTW; • Obstruct flow resulting in interference. • Are discharged at a flow rate and/or concentration that will result in interference; and • Increase the temperature of waste-water entering the treatment plant that would result in interference, but in no case raise the POTW influent temperature above 104 | F (40 | C).	Indirect discharge through a POTW.	40 CFR 403.5

TABLE T-3 (continued)

SELECTED FEDERAL ACTION-SPECIFIC POTENTIAL APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS a/

LEXINGTON-BLUEGRASS ARMY DEPOT

Actions	Requirement	Prerequisites for Applicability b/ c/	Citation
Discharge to Publicly Owned Treatment Works (Continued)	• Discharge must comply with local POTW pretreatment program, including POTW-specific pollutants, spill prevention program requirements, and reporting and monitoring requirements.		40 CFR 403.5 and local POTW regulations
	• RCRA permit-by-rule requirements (including corrective action where the NPDES permit was issued after November 8, 1984) must be complied with for discharges of RCRA hazardous wastes to POTWs.	Transport of RCRA hazardous wastes to POTWs by truck, rail, or dedicated pipe (i.e. pipe solely dedicated for hazardous waste [as defined in 40 CFR 265] which discharges from within the boundaries of the CERCLA site to within the boundaries of the POTW).	40 CFR 270.60
Surface Water Control	Prevent run-on and control and collect run-off from a 24-hour 25-year storm (waste piles, land treatment facilities, landfills).	RCRA hazardous waste treated, stored or disposed of after the effective date of the requirements.	40 CFR 264.251(c).(d) 40 CFR 264.273(d).(d) 40 CFR 264.301(c).(d)
	Prevent over-topping of surface impoundment.		40 CFR 264.221(c)
Tank Storage (On-Site)	Tanks must have sufficient structural strength to ensure that they do not collapse, rupture, or fail.	Storage of RCRA hazardous waste (listed or characteristic) not meeting small quantity generator criteria held for a temporary period greater than 90 days before treatment, disposal, or storage elsewhere (40 CFR 264.10), in a tank (i.e., any portable device in which a material is stored, transported, disposed of, or handled). A generator who accumulates or stores hazardous waste on-site for 90 days or less in compliance with 40 CFR 262.34(a)(1-4) is not subject to full RCRA storage requirements. Small quantity generators are not subject to the 90 day limit (40 CFR 262.34(c), (d), and (e)).	40 CFR 264.190
	Waste must not be incompatible with the tank material unless the tank is protected by a liner or by other means.		40 CFR 264.191
	Tanks must be provided with secondary containment and controls to prevent overfilling, and sufficient freeboard maintained in open tanks to prevent overtopping by wave action or precipitation.		40 CFR 264.193-194
	Inspect the following: overfilling control, control equipment, monitoring data, waste level (for uncovered tanks),		40 CFR 264.195

TABLE T-3 (continued)

SELECTED FEDERAL ACTION-SPECIFIC POTENTIAL APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS a/

LEXINGTON-BLUEGRASS ARMY DEPOT

Actions	Requirement	Prerequisites for Applicability b/ c/	Citation
Tank Storage (On-Site) (Continued)	tank condition, above-ground portions of tanks (to assess their structural integrity), and the area surrounding the tank (to identify signs of leakage).		
	Repair any corrosion, crack or leak.		40 CFR 264.196
	At closure, remove all hazardous waste and hazardous waste residues from tanks, discharge control equipment, and discharge confinement structures.		40 CFR 264.197
	Store ignitable and reactive waste so as to prevent the waste from igniting or reacting. Ignitable or reactive wastes in covered tanks must comply with buffer zone requirements in "Flammable and Combustible Liquids Code," Tables 2-1 through 2-6 (National Fire Protection Association, 1976 or 1981).		40 CFR 264.198
	Storage Prohibitions:		
	Storage of banned wastes must be in accordance with 40 CFR 268. When such storage occurs beyond one year, the owner/operator bears the burden of proving that such storage is solely for the purpose of accumulating sufficient quantities to allow for proper recovery, treatment and disposal.		40 CFR 268.50
Treatment (in a unit)	Design and operating standards for unit in which hazardous waste is treated. (See citations at right for design and operating requirements for specific unit.)	Treatment of hazardous waste in a unit.	40 CFR 264.190-264.192 (Tanks) 40 CFR 264.221 (Surface Impoundments) 40 CFR 264.251 (Waste Piles)

TABLE T-3 (continued)

SELECTED FEDERAL ACTION-SPECIFIC POTENTIAL APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS a/

LEXINGTON-BLUEGRASS ARMY DEPOT

Actions	Requirement	Prerequisites for Applicability b/ c/	Citation
Treatment (in a unit) (Continued)			40 CFR 264.273 (Land Treatment Unit) 40 CFR 264.343-345 (Incinerators) 40 CFR 264.601 (Miscellaneous Treatment Units) 40 CFR 266.373 (Thermal Treatment Units)
Treatment (when Waste will be Land Disposed)	Treatment of waste subject to ban on land disposal must attain levels achievable by best demonstrated available treatment technologies (BDAT) for each hazardous constituent in each listed waste, if residual is to be land disposed. If residual is to be further treated, initial treatment and any subsequent treatment that produces residual to be treated need not be BDAT, if it does not exceed value in CCWE (Constituent Concentration in Waste Extract) Table for each applicable water. (See 51 FR 40642, November 6, 1986.)	Disposal of contaminated soil and debris resulting from CERCLA response actions or RCRA corrective action is not subject to land disposal prohibitions and/or treatment standards for solvents, dioxins, or California list wastes until November 8, 1990 (for certain first third wastes until August 8, 1990). All wastes listed as hazardous is outlined in 40 CFR part 261 as of November 8, 1984, except for spent solvent wastes and dioxin-containing wastes,have been ranked with respect to volume and intrinsic hazards, and are scheduled for land disposal prohibition and/or treatment standard predetermination as follows: Solvents and dioxins — Nov. 8, 1986 One-third of all ranked and hazardous wastes — Aug. 8, 1988 Underground injection of solvents and dioxins and California list wastes — Aug. 8, 1988 CERCLA response action and RCRA corrective action soil and debris — Nov. 8, 1988 Two-thirds of all ranked and listed hazardous wastes — Jul. 8, 1989	40 CFR 268.10 40 CFR 268.11 40 CFR 268.12 40 CFR 268.41 40 CFR 268 Subpart D 51 FR 40641 52 FR 25760

TABLE T-3 (continued)

SELECTED FEDERAL ACTION-SPECIFIC POTENTIAL APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS a/

LEXINGTON-BLUEGRASS ARMY DEPOT

Actions	Requirement	Prerequisites for Applicability b/ c/		Citation
Treatment (when Waste will be Land Disposed)(Continued)		All remaining ranked and listed hazardous wastes tic under RCRA section 3001	May 8, 1990	identified by characteris-
		Any hazards waste listed or identified under RCRA section 3001 after November 8, 1984	Within 6 mos. of the date of identification or listing.	
	BDAT standards for spent solvent wastes and dioxin-containing wastes are based on one of four technologies or combinations: for waste waters, (1) steam stripping, (2) biological treatment, or (3) carbon adsorption [alone or in combination with (1) or (2)]; and for all other wastes, (4) incineration. Any technology may be used, however, if it will achieve the concentration levels specified.			40 CFR 268.30 RCRA Sections 3004(d)(3), (e)(3) 42 U.S.C. 6924(d)(3), (e)(3)
Operation and Maintenance (O&M)	30-year post-closure care to ensure that site is maintained and monitored.	Land disposal closure.		40 CFR 264.310

TABLE T-3 (continued)

SELECTED FEDERAL ACTION-SPECIFIC POTENTIAL APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS a/

LEXINGTON-BLUEGRASS ARMY DEPOT

FOOTNOTES:

a/ Currently only RCRA, CWA, and SDWA requirements are included. Additional action-specific requirements will be added as additional statutes are analyzed.

b/ Requirements have been proposed but not promulgated for air stripping, hybrid closure, gas collection and miscellaneous treatment unit. When these regulations are promulgated, they will be included in the matrix.

c/ Some action-specific requirements listed may be relevant and appropriate even if RCRA definitions of storage, disposal, or hazardous waste are not met, or if the waste at the site is similar to but not identifiable as a RCRA hazardous waste.

d/ In many cases, there are no defined "units" at a CERCLA site. Instead, there are areas of contamination with differing concentration levels (including hot spots) of hazardous substances, pollutants, or contaminants. When RCRA hazardous wastes are moved into or out of an area of contamination, RCRA disposal requirements are applicable to the waste being managed and certain treatment, storage, or disposal requirements (such as for closure) are applicable to the area where the waste is received.

e/ Regional administrator may revise length of post-closure care period (40 CFR 264.117).

f/ Landfill units meeting the requirements of 40 CFR 264.301(f) are not subject to RCRA minimum technology requirements.

g/ "Waters of the U.S." is defined broadly in 40 CFR 122.2 and includes essentially any water body and wetland.

h/ Section 121 of SARA exempts on-site CERCLA activities from obtaining permits. However, the substantive requirements of a law or regulation must be met. In particular, on-site discharges to surface waters are exempt from procedural NPDES permit regulations. Off-site discharges would be required to apply for an obtain an NPDES permit.

i/ Federal Water Quality Criteria may be relevant and appropriate depending on the designated or potential use of the water, the media affected, the purposes of the criteria, and current information. (CERCLA ‖ 121(d)(2)(B)(i)) Federal Water Quality Criteria for the protection of aquatic life will be relevant and appropriate when environmental factors (e.g., protection of aquatic organisms) are being considered. (50 FR 30784 [July 29, 1985]).

j/ Discharge to POTWs is considered an off-site activity; therefore, requirements related to discharge to a POTW are not ARARs, but are included in this table for reference. Off-site actions must comply with all legally applicable requirements, both substantive and administrative. The concept of "relevant and appropriate" is not available for off-site actions.

TABLE T-4
SELECTED STATE ACTION SPECIFIC POTENTIAL
APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS
LEXINGTON-BLUEGRASS ARMY DEPOT

Actions	Requirement	Prerequisites for Applicability	Citation
Site-wide activities	Endangered Species Critical Habitat Protection	Identified in the Endangered Species Act of 1973 and 50 CFR, Part 424	401 KAR 47:030 Section 3
	Hazardous Waste Generator	Hazardous Waste Determination Generator Identification No.	401 KAR 32:010
	Record Keeping and Reporting		40 KAR 32:040
	Closure and Post-Closure	Closure of Waste Piles	401 KAR 34:070
		Performance Standards, Closure Plan, Decontamination of Equipment	401 KAR 35:070
Waste Piles	Storage Criteria, Design, Monitoring, Repairing, Closure, Special Requirements		401 KAR 34:210
	Containment, Closure, Protection from Wind		401 KAR 35:210
Container Storage	Use and Management	Known Integrity of Structure	401 KAR 34:180
Land Disposal	Restrictions		401 KAR 37:010
Transport of Hazardous Wastes	Manifests, Requirement for Off-Site Transport		401 KAR 32:020
	Pre-Transport	Packing, Labeling, Marking, Placard	401 KAR 32:030
Transporters of Hazardous Wastes	Requirements		401 KAR 33:010 401 KAR 33:020 401 KAR 33:030
Permits	General Permitting		401 KAR 38:010
	Permit Conditions		401 KAR 38:010

TABLE T-4 (Continued)
SELECTED STATE ACTION SPECIFIC POTENTIAL
APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS
LEXINGTON-BLUEGRASS ARMY DEPOT

Actions	Requirement	Prerequisites for Applicability	Citation
Surface Water Permits	KPDES	Scope, Limits	401 KAR 5:055
		Conditions	401 KAR 5:065
		Discharge	401 KAR 5:080
	Biochemically Degradable Wastes	Secondary Treatment	401 KAR 5:045
	Water Quality Criteria		401 KAR 5:031
	Discharge to Surface Water	Secondary Treatment	401 KAR 5:035
Air Permit	General Requirements	"Sources"	401 KAR 50:035
	Monitoring		401 KAR 50:050
	Ambient Air Quality Standards		401 KAR 53:010
	New Sources for Toxic Air Pollutants	Limits for Organic Metals	401 KAR 63:022

APPENDIX U

ESTIMATED VOC EMISSION RATES FOR TREATMENT OF LBAD GROUNDWATER VIA AIR STRIPPING

APPENDIX U
ESTIMATED VOC EMISSION RATES FOR TREATMENT OF IMPACTED LBAD GROUNDWATER VIA AIR STRIPPING

Treatment Conditions	
Groundwater Flow Rate (gpm, total flow for all systems):	50
Stripper Efficiency (%):	95
Daily Average Operation Rate for Treatment System (hours):	24

VOC	Maximum VOC Concentration In Groundwater (ug/l)	Calculated Maximum Emission Rate (lb/hour)	State of Kentucky Maximum Allowable Emission Rate (a) (lb/hour)
1,1–Dichloroethane	22	0.00523	0.8268
1,2–Dichloroethenes	34	0.00809	0.8064
1,3–Dimethylbenzene	16	0.00381	NA (b)
Acetone	240	0.05708	1.8168
Benzene	33	0.00785	0.030616
Carbon tetrachloride	1.1	0.00026	0.030616
Chloromethane	4.4	0.00105	0.10716
Ethylbenzene	7.7	0.00183	0.444
Methyl isobutyl ketone	5.3	0.00126	0.20924
Tetrachloroethene	1.1	0.00026	NA (b)
Toluene	69	0.01641	0.38272
Trichloroethene	6.6	0.00157	0.27556
Vinyl chloride	150	0.03568	NA (b)
Xylenes	110	0.02616	0.444

Example Discharge Rate Calculation:
(22 ug/l)(50 gal/min)*(60 min/hr)*(1 l/0.26417 gal)*(1 lb/45,359,300 ug)*(95%) = 0.00523 lb/hour 1,1–Dichloroethane*

(a) Values Were Calculated in Accordance With State of Kentucky Department For Environmental Protection Division For Air Quality Regulations for "New or Modified Sources Emitting Toxic Air Pollutants", 401 KAR 63:022. A Stack Height of 7 Feet Was Used For This Calculation.

(b) Maximum Allowable Emission Rate Could Not Be Calculated Because a Significant Emission Level (M) Value For This Compound is Not Provided in Appendix B to 401 KAR 63:022