LONGHORN ARMY AMMUNITION PLANT KARNACK, TEXAS

ADMINISTRATIVE RECORD

Volume 7 of 19

2010

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Prepared for

Department of the Army
Longhorn Army Ammunition Plant

1976 – 2010

LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS
ADMINISTRATIVE RECORD – CHRONOLOGICAL INDEX

VOLUME 7 of 19

2010

Note: Volume 7, Year 2010, Letter A is listed Out of Date in Sequence and should have been inserted in Year 2001.

A. Title: Report – Final Baseline Risk Assessment: Human Health Evaluation, Volume 2: Appendices for the Site 16 Landfill Remedial Investigation and Feasibility Study LHAAP-16, Longhorn Army Ammunition Plant, Karnack, Texas
Author(s): Jacobs Engineering, Inc., St. Louis, Missouri
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Final
BASELINE RISK ASSESSMENT: HUMAN HEALTH EVALUATION
Volume 2: Appendices

A supplement to the
REMEDIAL INVESTIGATION REPORT

for the

SITE 16 LANDFILL REMEDIAL INVESTIGATION and FEASIBILITY STUDY

At the

Longhorn Army Ammunition Plant (LHAAP)
Karnack, Texas

Submitted to

U.S. Army Corps of Engineers
Tulsa District

Contract No. DACA56-97-D-0001

June 2001

Jacobs Engineering Group, Inc
St. Louis, Missouri

ATTACHMENT 1

RISK CALCULATION SPREADSHEETS

INCIDENTAL INGESTION OF ON-SITE SURFICIAL SOILS (0 - 0.5 FEET)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	IR (mg/day)	FI (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Oral Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Oral Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																	
Aluminum	18100	1.0E-06	100	0.5	52	10	43	25,550	3,650	4.3E-04	3.0E-03	--	--	--	--	--	--
Antimony	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	0.0004	--	--	--	--
Arsenic	6.7	1.0E-06	100	0.5	52	10	43	25,550	3,650	1.6E-07	1.1E-06	1.5	0.0003	2.4E-07	1.0E+02	3.7E-03	5.3E+01
Barium	295	1.0E-06	100	0.5	52	10	43	25,550	3,650	7.0E-06	4.9E-05	--	0.07	--	--	7.0E-04	1.0E+01
Beryllium	1.4	1.0E-06	100	0.5	52	10	43	25,550	3,650	3.3E-08	2.3E-07	--	0.002	--	--	1.2E-04	1.7E+00
Cadmium	0.86	1.0E-06	100	0.5	52	10	43	25,550	3,650	2.0E-08	1.4E-07	--	0.001	--	--	1.4E-04	2.0E+00
Calcium	15100	1.0E-06	100	0.5	52	10	43	25,550	3,650	3.6E-04	2.5E-03	--	--	--	--	--	--
Chromium (III)	31.5	1.0E-06	100	0.5	52	10	43	25,550	3,650	7.5E-07	5.2E-06	--	1.5	--	--	3.5E-06	5.0E-02
Chromium (VI)	31.5	1.0E-06	100	0.5	52	10	43	25,550	3,650	7.5E-07	5.2E-06	--	0.003	--	--	1.7E-03	2.4E+01
Cobalt	19.8	1.0E-06	100	0.5	52	10	43	25,550	3,650	4.7E-07	3.3E-06	--	--	--	--	--	--
Copper	10.5	1.0E-06	100	0.5	52	10	43	25,550	3,650	2.5E-07	1.7E-06	--	--	--	--	--	--
Iron	21500	1.0E-06	100	0.5	52	10	43	25,550	3,650	5.1E-04	3.6E-03	--	--	--	--	--	--
Lead	29	1.0E-06	100	0.5	52	10	43	25,550	3,650	6.9E-07	4.8E-06	--	--	--	--	--	--
Magnesium	1030	1.0E-06	100	0.5	52	10	43	25,550	3,650	2.4E-05	1.7E-04	--	--	--	--	--	--
Manganese	1270	1.0E-06	100	0.5	52	10	43	25,550	3,650	3.0E-05	2.1E-04	--	0.14	--	--	1.5E-03	2.1E+01
Mercury	0.09	1.0E-06	100	0.5	52	10	43	25,550	3,650	2.1E-09	1.5E-08	--	--	--	--	--	--
Nickel	16.23	1.0E-06	100	0.5	52	10	43	25,550	3,650	3.8E-07	2.7E-06	--	0.02	--	--	1.4E-04	2.0E+00
Potassium	1070	1.0E-06	100	0.5	52	10	43	25,550	3,650	2.5E-05	1.8E-04	--	--	--	--	--	--
Selenium	1.4	1.0E-06	100	0.5	52	10	43	25,550	3,650	3.3E-08	2.3E-07	--	0.005	--	--	4.6E-05	6.6E-01
Silver	0.81	1.0E-06	100	0.5	52	10	43	25,550	3,650	1.9E-08	1.3E-07	--	0.005	--	--	2.6E-05	3.7E-01
Sodium	68.5	1.0E-06	100	0.5	52	10	43	25,550	3,650	1.6E-06	1.1E-05	--	--	--	--	--	--
Strontium	62.7	1.0E-06	100	0.5	52	10	43	25,550	3,650	1.5E-06	1.0E-05	--	0.6	--	--	1.7E-05	2.4E-01
Thallium	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	33.5	1.0E-06	100	0.5	52	10	43	25,550	3,650	7.9E-07	5.5E-06	--	0.007	--	--	7.9E-04	1.1E+01
Zinc	55.6	1.0E-06	100	0.5	52	10	43	25,550	3,650	1.3E-06	9.2E-06	--	0.3	--	--	3.1E-05	4.4E-01
Organics																	
Acetone	0.068	1.0E-06	100	0.5	52	10	43	25,550	3,650	1.6E-09	1.1E-08	--	0.1	--	--	1.1E-07	1.6E-03
Butyl Benzyl Phthalate	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Di-n-Butyl Phthalate	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	0.1	--	--	--	--
Methylene Chloride	0.009	1.0E-06	100	0.5	52	10	43	25,550	3,650	2.1E-10	1.5E-09	0.0075	0.06	1.6E-12	6.7E-04	2.5E-08	3.6E-04
Styrene	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Trichloroethene	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	0.011	0.006	--	--	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x CF x IR x FI x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 2.4E-07 Hazard Index: 0.009 [d]

0.007 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

INCIDENTAL INGESTION OF ON-SITE SURFICIAL SOILS (0 - 0.5 FEET)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	IR (mg/day)	FI (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Oral Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Oral Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																	
Aluminum	18100	1.0E-06	100	1.0	60	10	43	25,550	3,650	9.9E-04	6.9E-03	--	--	--	--	--	--
Antimony	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	0.0004	--	--	--	--
Arsenic	6.7	1.0E-06	100	1.0	60	10	43	25,550	3,650	3.7E-07	2.6E-06	1.5	0.0003	5.6E-07	1.0E+02	8.7E-03	5.1E+01
Barium	295	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.6E-05	1.1E-04	--	0.07	--	--	1.6E-03	9.4E+00
Beryllium	1.4	1.0E-06	100	1.0	60	10	43	25,550	3,650	7.6E-08	5.4E-07	--	0.002	--	--	2.7E-04	1.6E+00
Cadmium	0.86	1.0E-06	100	1.0	60	10	43	25,550	3,650	4.7E-08	3.3E-07	--	0.001	--	--	3.3E-04	1.9E+00
Calcium	15100	1.0E-06	100	1.0	60	10	43	25,550	3,650	8.2E-04	5.8E-03	--	--	--	--	--	--
Chromium (III)	31.5	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.7E-06	1.2E-05	--	1.5	--	--	8.0E-06	4.7E-02
Chromium (VI)	31.5	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.7E-06	1.2E-05	--	0.003	--	--	4.0E-03	2.4E+01
Cobalt	19.8	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.1E-06	7.6E-06	--	--	--	--	--	--
Copper	10.5	1.0E-06	100	1.0	60	10	43	25,550	3,650	5.7E-07	4.0E-06	--	--	--	--	--	--
Iron	21500	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.2E-03	8.2E-03	--	--	--	--	--	--
Lead	29	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.6E-06	1.1E-05	--	--	--	--	--	--
Magnesium	1030	1.0E-06	100	1.0	60	10	43	25,550	3,650	5.6E-05	3.9E-04	--	--	--	--	--	--
Manganese	1270	1.0E-06	100	1.0	60	10	43	25,550	3,650	6.9E-05	4.9E-04	--	0.14	--	--	3.5E-03	2.1E+01
Mercury	0.09	1.0E-06	100	1.0	60	10	43	25,550	3,650	4.9E-09	3.4E-08	--	--	--	--	--	--
Nickel	16.23	1.0E-06	100	1.0	60	10	43	25,550	3,650	8.9E-07	6.2E-06	--	0.02	--	--	3.1E-04	1.8E+00
Potassium	1070	1.0E-06	100	1.0	60	10	43	25,550	3,650	5.8E-05	4.1E-04	--	--	--	--	--	--
Selenium	1.4	1.0E-06	100	1.0	60	10	43	25,550	3,650	7.6E-08	5.4E-07	--	0.005	--	--	1.1E-04	6.5E-01
Silver	0.81	1.0E-06	100	1.0	60	10	43	25,550	3,650	4.4E-08	3.1E-07	--	0.005	--	--	6.2E-05	3.6E-01
Sodium	68.5	1.0E-06	100	1.0	60	10	43	25,550	3,650	3.7E-06	2.6E-05	--	--	--	--	--	--
Strontium	62.7	1.0E-06	100	1.0	60	10	43	25,550	3,650	3.4E-06	2.4E-05	--	0.6	--	--	4.0E-05	2.4E-01
Thallium	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	33.5	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.8E-06	1.3E-05	--	0.007	--	--	1.9E-03	1.1E+01
Zinc	55.6	1.0E-06	100	1.0	60	10	43	25,550	3,650	3.0E-06	2.1E-05	--	0.3	--	--	7.0E-05	4.1E-01
Organics																	
Acetone	0.068	1.0E-06	100	1.0	60	10	43	25,550	3,650	3.7E-09	2.6E-08	--	0.1	--	--	2.6E-07	1.5E-03
Butyl Benzyl Phthalate	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Di-n-Butyl Phthalate	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	0.1	--	--	--	--
Methylene Chloride	0.009	1.0E-06	100	1.0	60	10	43	25,550	3,650	4.9E-10	3.4E-09	0.0075	0.06	3.7E-12	6.6E-04	5.7E-08	3.4E-04
Styrene	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Trichloroethene	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	0.011	0.006	--	--	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x CF x IR x FI x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 5.6E-07 Hazard Index: 0.021 [d]

0.017 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

DERMAL CONTACT WITH ON-SITE SURFICIAL SOILS (0 - 0.5 FEET)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	SA (cm^2/day)	AF (mg/cm^2)	ABS (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Absorbed) (mg/kg-day)	NC Chronic Daily Intake (Absorbed) (mg/kg-day)	Dermal Adjusted Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Dermal Adjusted Reference Dose (RfD) (mg/kg-day)	Dermal Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Dermal Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																		
Aluminum	18100	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	5.8E-05 [a]	4.1E-04 [a]	--	--	--	--	--	--
Antimony	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	0.00012	--	--	--	--
Arsenic	6.7	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	2.2E-08	1.5E-07	5	0.00009	1.1E-07	100.0	0.002	51.3
Barium	295	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	9.5E-07	6.6E-06	--	0.021	--	--	3.1E-04	7.9
Beryllium	1.4	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	4.5E-09	3.2E-08	--	0.0006	--	--	5.3E-05	1.36
Cadmium	0.86	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	2.8E-09 [a]	1.9E-08 [a]	--	0.0003	--	--	6.3E-05	1.6E+00
Calcium	15100	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	4.9E-05	3.4E-04	--	--	--	--	--	--
Chromium (III)	31.5	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	1.0E-07	7.1E-07	--	0.45	--	--	1.6E-06	0.041
Chromium (VI)	31.5	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	1.0E-07	7.1E-07	--	0.0009	--	--	0.001	20.4
Cobalt	19.8	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	6.4E-08	4.5E-07	--	--	--	--	--	--
Copper	10.5	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	3.4E-08	2.4E-07	--	--	--	--	--	--
Iron	21500	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	6.9E-05	4.8E-04	--	--	--	--	--	--
Lead	29	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	9.3E-08	6.5E-07	--	--	--	--	--	--
Magnesium	1030	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	3.3E-06	2.3E-05	--	--	--	--	--	--
Manganese	1270	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	4.1E-06	2.9E-05	--	0.042	--	--	0.001	25.6
Mercury	0.09	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	2.9E-10	2.0E-09	--	--	--	--	--	--
Nickel	16.23	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	5.2E-08	3.7E-07	--	0.006	--	--	6.2E-05	1.6E+00
Potassium	1070	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	3.4E-06	2.4E-05	--	--	--	--	--	--
Selenium	1.4	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	4.5E-09	3.2E-08	--	0.0015	--	--	2.1E-05	0.54
Silver	0.81	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	2.6E-09	1.8E-08	--	0.0015	--	--	1.2E-05	0.31
Sodium	68.5	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	2.2E-07	1.5E-06	--	--	--	--	--	--
Strontium	62.7	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	2.0E-07	1.4E-06	--	0.18	--	--	7.8E-06	0.20
Thallium	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	33.5	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	1.1E-07	7.5E-07	--	0.0021	--	--	3.6E-04	9.23
Zinc	55.6	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	1.8E-07 [a]	1.3E-06 [a]	--	0.09	--	--	1.4E-05	0.36
Organics																		
Acetone	0.068	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	2.2E-10	1.5E-09	--	0.1	--	--	1.5E-08	0.00
Butyl Benzyl Phthalate	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Di-n-Butyl Phthalate	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	0.1	--	--	--	--
Methylene Choride	0.009	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	2.9E-11	2.0E-10	0.0075	0.06	2.2E-13	2.0E-04	3.3E-09	0.00
Styrene	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Trichloroethene	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	0.011	0.006	--	--	--	--

Chronic Daily Intake (CDI) (Absorbed; mg/kg-day) = (CS x CF x SA x AF x ABS x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 1.1E-07 Hazard Index: 0.0049 [d]

0.0039 [c]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

00087957

DERMAL CONTACT WITH ON-SITE SURFICIAL SOILS (0 - 0.5 FEET)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	SA (cm²/day)	AF (mg/cm²)	ABS (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Absorbed) (mg/kg-day)	NC Chronic Daily Intake (Absorbed) (mg/kg-day)	Dermal Adjusted Slope Factor (SF) (mg/kg-day)$^{-1}$	Dermal Adjusted Reference Dose (RfD) (mg/kg-day)	Dermal Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Dermal Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																		
Aluminum	18100	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	4.1E-04 [a]	2.8E-03 [a]	--	--	--	--	--	--
Antimony	--	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	--	--	--	0.00012	--	--	--	--
Arsenic	6.7	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	1.5E-07	1.1E-06	5	0.00009	7.5E-07	100.0	0.012	60.0
Barium	295	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	6.6E-06	4.6E-05	--	0.021	--	--	2.2E-03	11.0
Beryllium	1.4	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	3.1E-08	2.2E-07	--	0.0006	--	--	3.7E-04	1.85
Cadmium	0.86	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	1.9E-08 [a]	1.3E-07 [a]	--	0.0003	--	--	4.3E-04	2.15
Calcium	15100	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	3.4E-04	2.4E-03	--	--	--	--	--	--
Chromium (III)	31.5	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	7.1E-07	4.9E-06	--	0.45	--	--	1.1E-05	0.055
Chromium (VI)	31.5	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	7.1E-07	4.9E-06	--	0.0009	--	--	0.005	16.7
Cobalt	19.8	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	4.4E-07	3.1E-06	--	--	--	--	--	--
Copper	10.5	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	2.4E-07	1.6E-06	--	--	--	--	--	--
Iron	21500	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	4.8E-04	3.4E-03	--	--	--	--	--	--
Lead	29	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	6.5E-07	4.5E-06	--	--	--	--	--	--
Magnesium	1030	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	2.3E-05	1.6E-04	--	--	--	--	--	--
Manganese	1270	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	2.8E-05	2.0E-04	--	0.042	--	--	0.005	16.7
Mercury	0.09	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	2.0E-09	1.4E-08	--	--	--	--	--	--
Nickel	16.23	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	3.6E-07	2.5E-06	--	0.006	--	--	0.000	0.0
Potassium	1070	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	2.4E-05	1.7E-04	--	--	--	--	--	--
Selenium	1.4	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	3.1E-08	2.2E-07	--	0.0015	--	--	0.000	0.0
Silver	0.81	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	1.8E-08	1.3E-07	--	0.0015	--	--	0.000	0.0
Sodium	68.5	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	1.5E-06	1.1E-05	--	--	--	--	--	--
Strontium	62.7	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	1.4E-06	9.8E-06	--	0.18	--	--	0.000	0.0
Thallium	--	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	33.5	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	7.5E-07	5.3E-06	--	0.0021	--	--	0.003	10.0
Zinc	55.6	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	1.2E-06 [a]	8.7E-06 [a]	--	0.09	--	--	0.000	0.0
Organics																		
Acetone	0.068	1.0E-06	4,100	1.0	0.1	60	10	43	25,550	3,650	1.5E-08	1.1E-07	--	0.1	--	--	0.000	0.0
Butyl Benzyl Phthalate	--	1.0E-06	4,100	1.0	0.1	60	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Di-n-Butyl Phthalate	--	1.0E-06	4,100	1.0	0.1	60	10	43	25,550	3,650	--	--	--	0.1	--	--	--	--
Methylene Chloride	0.009	1.0E-06	4,100	1.0	0.1	60	10	43	25,550	3,650	2.0E-09	1.4E-08	0.0075	0.06	1.5E-11	2.0E-03	0.000	0.0
Styrene	--	1.0E-06	4,100	1.0	0.1	60	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Trichloroethene	--	1.0E-06	4,100	1.0	0.1	60	10	43	25,550	3,650	--	--	0.011	0.006	--	--	--	--

Chronic Daily Intake (CDI) (Absorbed; mg/kg-day) = (CS x CF x SA x AF x ABS x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR$_i$
Chemical-Specific HQ = CDI / RfD; HI = CDI$_1$/RfD$_1$ + CDI$_2$/RfD$_2$ + ... + CDI$_i$/RfD$_i$

Total Pathway Risk: 7.5E-07 Hazard Index: 0.03 [d]

0.02 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

INHALATION OF PARTICULATES FROM SURFICIAL SOILS (0-0.5 FEET)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m3/day)	ED (years)	EF (days/yr)	BW (kg)	PEF (m^3/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																
Aluminum	18100	10	10	52	43	1.32E+09	25,550	3,650	6.5E-08	4.5E-07	--	--	--	--	--	--
Antimony	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Arsenic	6.7	10	10	52	43	1.32E+09	25,550	3,650	2.4E-11	1.7E-10	15	--	3.6E-10	7.2	--	--
Barium	295	10	10	52	43	1.32E+09	25,550	3,650	1.1E-09	7.4E-09	--	0.00014	--	--	0.0001	5.0E+00
Beryllium	1.4	10	10	52	43	1.32E+09	25,550	3,650	5.0E-12	3.5E-11	8.4	0.0000057	4.2E-11	0.8	6.1E-06	3.1E-01
Cadmium	0.86	10	10	52	43	1.32E+09	25,550	3,650	3.1E-12	2.2E-11	6.3	--	2.0E-11	4.0E-01	--	--
Calcium	15100	10	10	52	43	1.32E+09	25,550	3,650	5.4E-08	3.8E-07	--	--	--	--	--	--
Chromium (III)	31.5	10	10	52	43	1.32E+09	25,550	3,650	1.1E-10	7.9E-10	--	--	--	--	--	--
Chromium (VI)	31.5	10	10	52	43	1.32E+09	25,550	3,650	1.1E-10	7.9E-10	42	0.000029	4.6E-09	9.2E+01	2.72E-05	1.4E+00
Cobalt	19.8	10	10	52	43	1.32E+09	25,550	3,650	7.1E-11	5.0E-10	--	--	--	--	--	--
Copper	10.5	10	10	52	43	1.32E+09	25,550	3,650	3.8E-11	2.6E-10	--	--	--	--	--	--
Iron	21500	10	10	52	43	1.32E+09	25,550	3,650	7.7E-08	5.4E-07	--	--	--	--	--	--
Lead	29	10	10	52	43	1.32E+09	25,550	3,650	1.0E-10	7.3E-10	--	--	--	--	--	--
Magnesium	1030	10	10	52	43	1.32E+09	25,550	3,650	3.7E-09	2.6E-08	--	--	--	--	--	--
Manganese	1270	10	10	52	43	1.32E+09	25,550	3,650	4.6E-09	3.2E-08	--	0.000014	--	--	0.002	1.0E+02
Mercury	0.09	10	10	52	43	1.32E+09	25,550	3,650	3.2E-13	2.3E-12	--	0.000086	--	--	2.7E-08	1.4E-03
Nickel	16.23	10	10	52	43	1.32E+09	25,550	3,650	5.8E-11	4.1E-10	--	--	--	--	--	--
Potassium	1070	10	10	52	43	1.32E+09	25,550	3,650	3.8E-09	2.7E-08	--	--	--	--	--	--
Selenium	1.4	10	10	52	43	1.32E+09	25,550	3,650	5.0E-12	3.5E-11	--	--	--	--	--	--
Silver	0.81	10	10	52	43	1.32E+09	25,550	3,650	2.9E-12	2.0E-11	--	--	--	--	--	--
Sodium	68.5	10	10	52	43	1.32E+09	25,550	3,650	2.5E-10	1.7E-09	--	--	--	--	--	--
Strontium	62.7	10	10	52	43	1.32E+09	25,550	3,650	2.2E-10	1.6E-09	--	--	--	--	--	--
Thallium	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	33.5	10	10	52	43	1.32E+09	25,550	3,650	1.2E-10	8.4E-10	--	--	--	--	--	--
Zinc	55.6	10	10	52	43	1.32E+09	25,550	3,650	2.0E-10	1.4E-09	--	--	--	--	--	--
Organics																
Acetone	0.068	10	10	52	43	1.32E+09	25,550	3,650	2.4E-13	1.7E-12	--	--	--	--	--	--
Butyl Benzyl Phthalate	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Di-n-Butyl Phthalate	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Methylene Chloride	0.009	10	10	52	43	1.32E+09	25,550	3,650	3.2E-14	2.3E-13	0.00165	0.857	5.3E-17	0.00	2.7E-13	1.4E-08
Styrene	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	0.286	--	--	--	--
Trichloroethene	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	0.00595	--	--	--	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x IR x EF x ED) / (PEF*BW*AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 5.0E-09 Hazard Index: 0.002

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

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INHALATION OF PARTICULATES FROM SURFICIAL SOILS (0-0.5 FEET)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m3/day)	ED (years)	EF (days/yr)	BW (kg)	PEF (m^3/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) ($(mg/kg-day)^{-1}$)	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																
Aluminum	18100	10	10	60	43	1.32E+09	25,550	3,650	7.5E-08	5.2E-07	--	--	--	--	--	--
Antimony	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Arsenic	6.7	10	10	60	43	1.32E+09	25,550	3,650	2.8E-11	1.9E-10	15	--	4.2E-10	7.0	--	--
Barium	295	10	10	60	43	1.32E+09	25,550	3,650	1.2E-09	8.5E-09	--	0.00014	--	--	0.0001	3.3E+00
Beryllium	1.4	10	10	60	43	1.32E+09	25,550	3,650	5.8E-12	4.1E-11	8.4	0.0000057	4.9E-11	0.8	7.2E-06	2.4E-01
Cadmium	0.86	10	10	60	43	1.32E+09	25,550	3,650	3.6E-12	2.5E-11	6.3	--	2.3E-11	3.8E-01	--	--
Calcium	15100	10	10	60	43	1.32E+09	25,550	3,650	6.2E-08	4.4E-07	--	--	--	--	--	--
Chromium (III)	31.5	10	10	60	43	1.32E+09	25,550	3,650	1.3E-10	9.1E-10	--	--	--	--	--	--
Chromium (VI)	31.5	10	10	60	43	1.32E+09	25,550	3,650	1.3E-10	9.1E-10	42	0.000029	5.5E-09	9.2E+01	3.14E-05	1.0E+00
Cobalt	19.8	10	10	60	43	1.32E+09	25,550	3,650	8.2E-11	5.7E-10	--	--	--	--	--	--
Copper	10.5	10	10	60	43	1.32E+09	25,550	3,650	4.3E-11	3.0E-10	--	--	--	--	--	--
Iron	21500	10	10	60	43	1.32E+09	25,550	3,650	8.9E-08	6.2E-07	--	--	--	--	--	--
Lead	29	10	10	60	43	1.32E+09	25,550	3,650	1.2E-10	8.4E-10	--	--	--	--	--	--
Magnesium	1030	10	10	60	43	1.32E+09	25,550	3,650	4.3E-09	3.0E-08	--	--	--	--	--	--
Manganese	1270	10	10	60	43	1.32E+09	25,550	3,650	5.3E-09	3.7E-08	--	0.000014	--	--	0.003	1.0E+02
Mercury	0.09	10	10	60	43	1.32E+09	25,550	3,650	3.7E-13	2.6E-12	--	0.000086	--	--	3.0E-08	1.0E-03
Nickel	16.23	10	10	60	43	1.32E+09	25,550	3,650	6.7E-11	4.7E-10	--	--	--	--	--	--
Potassium	1070	10	10	60	43	1.32E+09	25,550	3,650	4.4E-09	3.1E-08	--	--	--	--	--	--
Selenium	1.4	10	10	60	43	1.32E+09	25,550	3,650	5.8E-12	4.1E-11	--	--	--	--	--	--
Silver	0.81	10	10	60	43	1.32E+09	25,550	3,650	3.4E-12	2.3E-11	--	--	--	--	--	--
Sodium	68.5	10	10	60	43	1.32E+09	25,550	3,650	2.8E-10	2.0E-09	--	--	--	--	--	--
Strontium	62.7	10	10	60	43	1.32E+09	25,550	3,650	2.6E-10	1.8E-09	--	--				
Thallium	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	33.5	10	10	60	43	1.32E+09	25,550	3,650	1.4E-10	9.7E-10	--	--	--	--	--	--
Zinc	55.6	10	10	60	43	1.32E+09	25,550	3,650	2.3E-10	1.6E-09	--	--	--	--	--	--
Organics																
Acetone	0.068	10	10	60	43	1.32E+09	25,550	3,650	2.8E-13	2.0E-12	--	--	--	--	--	--
Butyl Benzyl Phthalate	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Di-n-Butyl Phthalate	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Methylene Chloride	0.009	10	10	60	43	1.32E+09	25,550	3,650	3.7E-14	2.6E-13	0.00165	0.857	6.1E-17	0.00	3.0E-13	1.0E-08
Styrene	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	0.286	--	--	--	--
Trichloroethene	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	0.00595	--	--	--	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x IR x EF x ED) / (PEF*BW*AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 6.0E-09 Hazard Index: 0.003

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

INHALATION OF VOLATILES FROM SOILS
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m3/day)	ED (years)	EF (days/yr)	BW (kg)	VF (m^3/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcino-genic Risk
Inorganics																
Aluminum	12,750	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Antimony	1.67	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Arsenic	5.49	10	10	52	43	--	25,550	3,650	--	--	15	--	--	--	--	--
Barium	179	10	10	52	43	--	25,550	3,650	--	--	--	0.00014	--	--	--	--
Beryllium	0.98	10	10	52	43	--	25,550	3,650	--	--	8.4	0.0000057	--	--	--	--
Cadmium	0.57	10	10	52	43	--	25,550	3,650	--	--	6.3	--	--	--	--	--
Calcium	5580	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Chromium (III)	19.4	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Chromium (VI)	19.4	10	10	52	43	--	25,550	3,650	--	--	42	0.000029	--	--	--	--
Cobalt	12.6	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Copper	7.19	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Iron	21400	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Lead	13.04	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Magnesium	1540	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Manganese	1270	10	10	52	43	--	25,550	3,650	--	--	--	0.000014	--	--	--	--
Mercury	0.07	10	10	52	43	--	25,550	3,650	--	--	--	0.000086	--	--	--	--
Nickel	15.8	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Potassium	917	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Selenium	0.68	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Silver	0.65	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Sodium	419	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Strontium	40.1	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Thallium	0.9	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	33.8	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Zinc	44.3	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Organics																
Acetone	0.013	10	10	52	43	5.39E+03	25,550	3,650	1.1E-08	8.0E-08	--	--	--	--	--	--
Butyl Benzyl Phthalate	0.237	10	10	52	43	3.59E+06	25,550	3,650	3.1E-10	2.2E-09	--	--	--	--	--	--
Di-n-Butyl Phthalate	0.373	10	10	52	43	3.44E+06	25,550	3,650	5.1E-10	3.6E-09	--	--	--	--	--	--
Methylene Chloride	0.0049	10	10	52	43	1.07E+03	25,550	3,650	2.2E-08	1.5E-07	0.00165	0.857	3.6E-11	6.55	1.8E-07	6.7E+01
Styrene	0.0044	10	10	52	43	5.76E+03	25,550	3,650	3.6E-09	2.5E-08	--	0.286	--	--	8.7E-08	3.2E+01
Trichloroethene	0.025	10	10	52	43	1.40E+03	25,550	3,650	8.5E-08	5.9E-07	0.00595	--	5.1E-10	9.3E+01	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x IR x EF x ED) / (VF*PEF*BW*AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 5.5E-10 Hazard Index: 0.00000027

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

Constituent	Di (cm2/s)	H (dimensionless)	Dw (cm2/s)	Kd (cm3/g)	Q/C	T (sec)	DA (cm2/s)	VF (m3/kg)
Acetone	0.124	1.59E-03	1.14E-05	0.00345	53.11	3.15E+08	1.066E-04	5.39E+03
Butyl Benzyl Phthalate	0.0174	5.17E-05	4.83E-06	345	53.11	3.15E+08	2.400E-10	3.59E+06
Di-n-Butyl Phthalate	0.0438	3.85E-08	7.86E-06	203.4	53.11	3.15E+08	2.621E-10	3.44E+06
Methylene Chloride	0.101	8.98E-02	1.17E-05	0.0702	53.11	3.15E+08	2.698E-03	1.07E+03
Styrene	0.071	0.113	8.00E-06	4.656	53.11	3.15E+08	9.352E-05	5.76E+03
Trichloroethene	0.079	0.422	9.10E-06	0.996	53.11	3.15E+08	1.579E-03	1.40E+03

VF calculated per USEPA Soil Screening Guidance (USEPA, 1996)

INHALATION OF VOLATILES FROM SOILS
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m3/day)	ED (years)	EF (days/yr)	BW (kg)	VF (m^3/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																
Aluminum	12,750	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Antimony	1.67	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Arsenic	5.49	10	10	60	43	--	25,550	3,650	--	--	15	--	--	--	--	--
Barium	179	10	10	60	43	--	25,550	3,650	--	--	--	0.00014	--	--	--	--
Beryllium	0.98	10	10	60	43	--	25,550	3,650	--	--	8.4	0.0000057	--	--	--	--
Cadmium	0.57	10	10	60	43	--	25,550	3,650	--	--	6.3	--	--	--	--	--
Calcium	5580	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Chromium (III)	19.4	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Chromium (VI)	19.4	10	10	60	43	--	25,550	3,650	--	--	42	0.000029	--	--	--	--
Cobalt	12.6	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Copper	7.19	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Iron	21400	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Lead	13.04	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Magnesium	1540	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Manganese	1270	10	10	60	43	--	25,550	3,650	--	--	--	0.000014	--	--	--	--
Mercury	0.07	10	10	60	43	--	25,550	3,650	--	--	--	0.000086	--	--	--	--
Nickel	15.8	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Potassium	917	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Selenium	0.68	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Silver	0.65	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Sodium	419	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Strontium	40.1	10	10	60	43	--	25,550	3,650	--	--	--	--				
Thallium	0.9	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	33.8	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Zinc	44.3	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Organics																
Acetone	0.013	10	10	60	43	5.39E+03	25,550	3,650	1.3E-08	9.2E-08	--	--	--	--	--	--
Butyl Benzyl Phthalate	0.237	10	10	60	43	3.59E+06	25,550	3,650	3.6E-10	2.5E-09	--	--	--	--	--	--
Di-n-Butyl Phthalate	0.373	10	10	60	43	3.44E+06	25,550	3,650	5.9E-10	4.1E-09	--	--	--	--	--	--
Methylene Chloride	0.0049	10	10	60	43	1.07E+03	25,550	3,650	2.5E-08	1.8E-07	0.00165	0.857	4.1E-11	6.61	2.1E-07	6.8E+01
Styrene	0.0044	10	10	60	43	5.76E+03	25,550	3,650	4.2E-09	2.9E-08	--	0.286	--	--	1.0E-07	3.2E+01
Trichloroethene	0.025	10	10	60	43	1.40E+03	25,550	3,650	9.8E-08	6.8E-07	0.00595	--	5.8E-10	93.55	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x IR x EF x ED) / (VF*PEF*BW*AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i

Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 6.2E-10 Hazard Index: 0.00000031

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

Constituent	Di (cm2/s)	H (dimensionless)	Dw (cm2/s)	Kd (cm3/g)	Q/C	T (sec)	DA (cm2/s)	VF (m3/kg)
Acetone	0.124	1.59E-03	1.14E-05	0.00345	53.11	3.15E+08	1.066E-04	5.39E+03
Butyl Benzyl Phthalate	0.0174	5.17E-05	4.83E-06	345	53.11	3.15E+08	2.400E-10	3.59E+06
Di-n-Butyl Phthalate	0.0438	3.85E-08	7.86E-06	203.4	53.11	3.15E+08	2.621E-10	3.44E+06
Methylene Chloride	0.101	8.98E-02	1.17E-05	0.0702	53.11	3.15E+08	2.698E-03	1.07E+03
Styrene	0.071	0.113	8.00E-06	4.656	53.11	3.15E+08	9.352E-05	5.76E+03
Trichloroethene	0.079	0.422	9.10E-06	0.996	53.11	3.15E+08	1.579E-03	1.40E+03

VF calculated per USEPA Soil Screening Guidance (USEPA, 1996)

INCIDENTAL INGESTION OF ON-SITE SURFICIAL/SUBSURFACE SOILS (0 - 5 FEET)
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	IR (mg/day)	FI (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Oral Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Oral Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																	
Aluminum	21500	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.7E-03	2.1E-02	--	--	--	--	--	--
Antimony	1.64	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.1E-07	1.6E-06	--	0.0004	--	--	4.0E-03	8.3E+00
Arsenic	7.44	1.0E-06	100	1.0	250	9	70	25,550	3,285	9.4E-07	7.3E-06	1.5	0.0003	1.4E-06	1.0E+02	2.4E-02	5.0E+01
Barium	171.8	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.2E-05	1.7E-04	--	0.07	--	--	2.4E-03	5.0E+00
Beryllium	1.4	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.8E-07	1.4E-06	--	0.002	--	--	7.0E-04	1.5E+00
Cadmium	0.57	1.0E-06	100	1.0	250	9	70	25,550	3,285	7.2E-08	5.6E-07	--	0.001	--	--	5.6E-04	1.2E+00
Calcium	15100	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.9E-03	1.5E-02	--	--	--	--	--	--
Chromium (III)	22.7	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.9E-06	2.2E-05	--	1.5	--	--	1.5E-05	3.1E-02
Chromium (VI)	22.7	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.9E-06	2.2E-05	--	0.003	--	--	7.3E-03	1.5E+01
Cobalt	19.8	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.5E-06	1.9E-05	--	--	--	--	--	--
Copper	9.17	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.2E-06	9.0E-06	--	--	--	--	--	--
Iron	26000	1.0E-06	100	1.0	250	9	70	25,550	3,285	3.3E-03	2.5E-02	--	--	--	--	--	--
Lead	18.05	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.3E-06	1.8E-05	--	--	--	--	--	--
Magnesium	1420	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.8E-04	1.4E-03	--	--	--	--	--	--
Manganese	1270	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.6E-04	1.2E-03	--	0.14	--	--	8.6E-03	1.8E+01
Mercury	0.07	1.0E-06	100	1.0	250	9	70	25,550	3,285	8.8E-09	6.8E-08	--	--	--	--	--	--
Nickel	11.81	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.5E-06	1.2E-05	--	0.02	--	--	6.0E-04	1.3E+00
Potassium	1560	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.0E-04	1.5E-03	--	--	--	--	--	--
Selenium	0.74	1.0E-06	100	1.0	250	9	70	25,550	3,285	9.3E-08	7.2E-07	--	0.005	--	--	1.4E-04	2.9E-01
Silver	0.69	1.0E-06	100	1.0	250	9	70	25,550	3,285	8.7E-08	6.8E-07	--	0.005	--	--	1.4E-04	2.9E-01
Sodium	144	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.8E-05	1.4E-04	--	--	--	--	--	--
Strontium	62.7	1.0E-06	100	1.0	250	9	70	25,550	3,285	7.9E-06	6.1E-05	--	0.6	--	--	1.0E-04	2.1E-01
Thallium	1.18	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.5E-07	1.2E-06	--	--	--	--	--	--
Vanadium	43.3	1.0E-06	100	1.0	250	9	70	25,550	3,285	5.4E-06	4.2E-05	--	0.007	--	--	6.0E-03	1.3E+01
Zinc	79.2	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.0E-05	7.7E-05	--	0.3	--	--	2.6E-04	5.4E-01
Organics																	
Acetone	0.016	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.0E-09	1.6E-08	--	0.1	--	--	1.6E-07	3.3E-04
Butyl Benzyl Phthalate	0.332	1.0E-06	100	1.0	250	9	70	25,550	3,285	4.2E-08	3.2E-07	--	0.2	--	--	1.6E-06	3.3E-03
Di-n-Butyl Phthalate	0.675	1.0E-06	100	1.0	250	9	70	25,550	3,285	8.5E-08	6.6E-07	--	0.1	--	--	6.6E-06	1.4E-02
Methylene Chloride	0.0064	1.0E-06	100	1.0	250	9	70	25,550	3,285	8.1E-10	6.3E-09	0.0075	0.06	6.1E-12	4.4E-04	1.1E-07	2.3E-04
Styrene	0.0081	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.0E-09	7.9E-09	--	0.2	--	--	4.0E-08	8.3E-05
Trichloroethene	0.011	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.4E-09	1.1E-08	0.011	0.006	1.5E-11	1.1E-03	1.8E-06	3.8E-03

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x CF x IR x FI x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 1.4E-06 Hazard Index: 0.055 [d]

0.048 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

00087965

INCIDENTAL INGESTION OF ON-SITE SURFICIAL/SUBSURFACE SOILS (0 - 5 FEET)
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	IR (mg/day)	FI (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Oral Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Oral Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																	
Aluminum	21500	1.0E-06	100	1.0	250	25	70	25,550	9,125	7.5E-03	2.1E-02	--	--	--	--	--	--
Antimony	1.64	1.0E-06	100	1.0	250	25	70	25,550	9,125	5.7E-07	1.6E-06	--	0.0004	--	--	4.0E-03	8.3E+00
Arsenic	7.44	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.6E-06	7.3E-06	1.5	0.0003	3.9E-06	1.0E+02	2.4E-02	5.0E+01
Barium	171.8	1.0E-06	100	1.0	250	25	70	25,550	9,125	6.0E-05	1.7E-04	--	0.07	--	--	2.4E-03	5.0E+00
Beryllium	1.4	1.0E-06	100	1.0	250	25	70	25,550	9,125	4.9E-07	1.4E-06	--	0.002	--	--	7.0E-04	1.5E+00
Cadmium	0.57	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.0E-07	5.6E-07	--	0.001	--	--	5.6E-04	1.2E+00
Calcium	15100	1.0E-06	100	1.0	250	25	70	25,550	9,125	5.3E-03	1.5E-02	--	--	--	--	--	--
Chromium (III)	22.7	1.0E-06	100	1.0	250	25	70	25,550	9,125	7.9E-06	2.2E-05	--	1.5	--	--	1.5E-05	3.1E-02
Chromium (VI)	22.7	1.0E-06	100	1.0	250	25	70	25,550	9,125	7.9E-06	2.2E-05	--	0.003	--	--	7.3E-03	1.5E+01
Cobalt	19.8	1.0E-06	100	1.0	250	25	70	25,550	9,125	6.9E-06	1.9E-05	--	--	--	--	--	--
Copper	9.17	1.0E-06	100	1.0	250	25	70	25,550	9,125	3.2E-06	9.0E-06	--	--	--	--	--	--
Iron	26000	1.0E-06	100	1.0	250	25	70	25,550	9,125	9.1E-03	2.5E-02	--	--	--	--	--	--
Lead	18.05	1.0E-06	100	1.0	250	25	70	25,550	9,125	6.3E-06	1.8E-05	--	--	--	--	--	--
Magnesium	1420	1.0E-06	100	1.0	250	25	70	25,550	9,125	5.0E-04	1.4E-03	--	--	--	--	--	--
Manganese	1270	1.0E-06	100	1.0	250	25	70	25,550	9,125	4.4E-04	1.2E-03	--	0.14	--	--	8.6E-03	1.8E+01
Mercury	0.07	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.4E-08	6.8E-08	--	--	--	--	--	--
Nickel	11.81	1.0E-06	100	1.0	250	25	70	25,550	9,125	4.1E-06	1.2E-05	--	0.02	--	--	6.0E-04	1.3E+00
Potassium	1560	1.0E-06	100	1.0	250	25	70	25,550	9,125	5.5E-04	1.5E-03	--	--	--	--	--	--
Selenium	0.74	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.6E-07	7.2E-07	--	0.005	--	--	1.4E-04	2.9E-01
Silver	0.69	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.4E-07	6.8E-07	--	0.005	--	--	1.4E-04	2.9E-01
Sodium	144	1.0E-06	100	1.0	250	25	70	25,550	9,125	5.0E-05	1.4E-04	--	--	--	--	--	--
Strontium	62.7	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.2E-05	6.1E-05	--	0.6	--	--	1.0E-04	2.1E-01
Thallium	1.18	1.0E-06	100	1.0	250	25	70	25,550	9,125	4.1E-07	1.2E-06	--	--	--	--	--	--
Vanadium	43.3	1.0E-06	100	1.0	250	25	70	25,550	9,125	1.5E-05	4.2E-05	--	0.007	--	--	6.0E-03	1.3E+01
Zinc	79.2	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.8E-05	7.7E-05	--	0.3	--	--	2.6E-04	5.4E-01
Organics																	
Acetone	0.016	1.0E-06	100	1.0	250	25	70	25,550	9,125	5.6E-09	1.6E-08	--	0.1	--	--	1.6E-07	3.3E-04
Butyl Benzyl Phthalate	0.332	1.0E-06	100	1.0	250	25	70	25,550	9,125	1.2E-07	3.2E-07	--	0.2	--	--	1.6E-06	3.3E-03
Di-n-Butyl Phthalate	0.675	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.4E-07	6.6E-07	--	0.1	--	--	6.6E-06	1.4E-02
Methylene Chloride	0.0064	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.2E-09	6.3E-09	0.0075	0.06	1.7E-11	4.4E-04	1.1E-07	2.3E-04
Styrene	0.0081	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.8E-09	7.9E-09	--	0.2	--	--	4.0E-08	8.3E-05
Trichloroethene	0.011	1.0E-06	100	1.0	250	25	70	25,550	9,125	3.8E-09	1.1E-08	0.011	0.006	4.2E-11	1.1E-03	1.8E-06	3.8E-03

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x CF x IR x FI x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + \ldots + CDI_i/RfD_i$

Total Pathway Risk: 3.9E-06 Hazard Index: 0.055 [d]

0.048 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

DERMAL CONTACT WITH ON-SITE SURFICIAL/SUBSURFACE SOILS (0 - 5 FEET)
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	SA (cm^2/day)	AF (mg/cm^2)	ABS (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Absorbed) (mg/kg-day)	NC Chronic Daily Intake (Absorbed) (mg/kg-day)	Dermal Adjusted Slope Factor (SF) (mg/kg-day)$^{-1}$	Dermal Adjusted Reference Dose (RfD) (mg/kg-day)	Dermal Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Dermal Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																		
Aluminum	21500	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	1.1E-04 [a]	8.4E-04 [a]	--	--	--	--	--	--
Antimony	1.64	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	8.3E-09	6.4E-08	--	0.00012	--	--	5.3E-04	8.3E+00
Arsenic	7.44	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	3.7E-08	2.9E-07	5	0.00009	1.9E-07	1.0E+02	3.2E-03	5.0E+01
Barium	171.8	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	8.6E-07	6.7E-06	--	0.021	--	--	3.2E-04	5.0E+00
Beryllium	1.4	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	7.0E-09	5.5E-08	--	0.0006	--	--	9.2E-05	1.4E+00
Cadmium	0.57	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	2.9E-09 [a]	2.2E-08 [a]	--	0.0003	--	--	7.3E-05	1.1E+00
Calcium	15100	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	7.6E-05	5.9E-04	--	--	--	--	--	--
Chromium (III)	22.7	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	1.1E-07	8.9E-07	--	0.45	--	--	2.0E-06	3.1E-02
Chromium (VI)	22.7	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	1.1E-07	8.9E-07	--	0.0009	--	--	9.9E-04	1.5E+01
Cobalt	19.8	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	1.0E-07	7.7E-07	--	--	--	--	--	--
Copper	9.17	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	4.6E-08	3.6E-07	--	--	--	--	--	--
Iron	26000	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	1.3E-04	1.0E-03	--	--	--	--	--	--
Lead	18.05	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	9.1E-08	7.1E-07	--	--	--	--	--	--
Magnesium	1420	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	7.1E-06	5.6E-05	--	--	--	--	--	--
Manganese	1270	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	6.4E-06	5.0E-05	--	0.042	--	--	1.2E-03	1.9E+01
Mercury	0.07	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	3.5E-10	2.7E-09	--	--	--	--	--	--
Nickel	11.81	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	5.9E-08	4.6E-07	--	0.006	--	--	7.7E-05	1.2E+00
Potassium	1560	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	7.9E-06	6.1E-05	--	--	--	--	--	--
Selenium	0.74	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	3.7E-09	2.9E-08	--	0.0015	--	--	1.9E-05	3.0E-01
Silver	0.69	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	3.5E-09	2.7E-08	--	0.0015	--	--	1.8E-05	2.8E-01
Sodium	144	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	7.2E-07	5.6E-06	--	--	--	--	--	--
Strontium	62.7	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	3.2E-07	2.5E-06	--	0.18	--	--	1.4E-05	2.2E-01
Thallium	1.18	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	5.9E-09	4.6E-08	--	--	--	--	--	--
Vanadium	43.3	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	2.2E-07	1.7E-06	--	0.0021	--	--	8.1E-04	1.3E+01
Zinc	79.2	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	4.0E-07 [a]	3.1E-06 [a]	--	0.09	--	--	3.4E-05	5.3E-01
Organics																		
Acetone	0.016	1.0E-06	2,000	0.2	0.1	250	9	70	25,550	3,285	8.1E-10	6.3E-09	--	0.1	--	--	6.3E-08	9.8E-04
Butyl Benzyl Phthalate	0.332	1.0E-06	2,000	0.2	0.1	250	9	70	25,550	3,285	1.7E-08	1.3E-07	--	0.2	--	--	6.5E-07	1.0E-02
Di-n-Butyl Phthalate	0.675	1.0E-06	2,000	0.2	0.1	250	9	70	25,550	3,285	3.4E-08	2.6E-07	--	0.1	--	--	2.6E-06	4.1E-02
Methylene Chloride	0.0064	1.0E-06	2,000	0.2	0.1	250	9	70	25,550	3,285	3.2E-10	2.5E-09	0.0075	0.06	2.4E-12	0.00	4.2E-08	0.001
Styrene	0.0081	1.0E-06	2,000	0.2	0.1	250	9	70	25,550	3,285	4.1E-10	3.2E-09	--	0.2	--	--	1.6E-08	2.5E-04
Trichloroethene	0.011	1.0E-06	2,000	0.2	0.1	250	9	70	25,550	3,285	5.5E-10	4.3E-09	0.011	0.006	6.1E-12	3.2E-03	7.2E-07	1.1E-02

Chronic Daily Intake (CDI) (Absorbed; mg/kg-day) = (CS x CF x SA x AF x ABS x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 1.9E-07 Hazard Index: 0.0074 [d]

0.0064 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

DERMAL CONTACT WITH ON-SITE SURFICIAL/SUBSURFACE SOILS (0 - 5 FEET)
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	SA (cm^2/day)	AF (mg/cm^2)	ABS (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Absorbed) (mg/kg-day)	NC Chronic Daily Intake (Absorbed) (mg/kg-day)	Dermal Adjusted Slope Factor (SF) (mg/kg-day)$^{-1}$	Dermal Adjusted Reference Dose (RfD) (mg/kg-day)	Dermal Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Dermal Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcino-genic Risk
Inorganics																		
Aluminum	21500	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	2.4E-03 [a]	6.7E-03 [a]	--	--	--	--	--	--
Antimony	1.64	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.8E-07	5.1E-07	--	0.00012	--	--	4.3E-03	8.6E+00
Arsenic	7.44	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	8.3E-07	2.3E-06	5	0.00009	4.2E-06	1.0E+02	2.6E-02	5.2E+01
Barium	171.8	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.9E-05	5.4E-05	--	0.021	--	--	2.6E-03	5.2E+00
Beryllium	1.4	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.6E-07	4.4E-07	--	0.0006	--	--	7.3E-04	1.5E+00
Cadmium	0.57	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	6.4E-08 [a]	1.8E-07 [a]	--	0.0003	--	--	6.0E-04	1.2E+00
Calcium	15100	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.7E-03	4.7E-03	--	--	--	--	--	--
Chromium (III)	22.7	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	2.5E-06	7.1E-06	--	0.45	--	--	1.6E-05	3.2E-02
Chromium (VI)	22.7	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	2.5E-06	7.1E-06	--	0.0009	--	--	7.9E-03	1.6E+01
Cobalt	19.8	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	2.2E-06	6.2E-06	--	--	--	--	--	--
Copper	9.17	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.0E-06	2.9E-06	--	--	--	--	--	--
Iron	26000	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	2.9E-03	8.1E-03	--	--	--	--	--	--
Lead	18.05	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	2.0E-06	5.7E-06	--	--	--	--	--	--
Magnesium	1420	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.6E-04	4.4E-04	--	--	--	--	--	--
Manganese	1270	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.4E-04	4.0E-04	--	0.042	--	--	9.5E-03	1.9E+01
Mercury	0.07	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	7.8E-09	2.2E-08	--	--	--	--	--	--
Nickel	11.81	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.3E-06	3.7E-06	--	0.006	--	--	6.2E-04	1.2E+00
Potassium	1560	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.7E-04	4.9E-04	--	--	--	--	--	--
Selenium	0.74	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	8.3E-08	2.3E-07	--	0.0015	--	--	1.5E-04	3.0E-01
Silver	0.69	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	7.7E-08	2.2E-07	--	0.0015	--	--	1.5E-04	3.0E-01
Sodium	144	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.6E-05	4.5E-05	--	--	--	--	--	--
Strontium	62.7	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	7.0E-06	2.0E-05	--	0.18	--	--	1.1E-04	2.2E-01
Thallium	1.18	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.3E-07	3.7E-07	--	--	--	--	--	--
Vanadium	43.3	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	4.8E-06	1.4E-05	--	0.0021	--	--	6.7E-03	1.3E+01
Zinc	79.2	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	8.9E-06 [a]	2.5E-05 [a]	--	0.09	--	--	2.8E-04	5.6E-01
Organics																		
Acetone	0.016	1.0E-06	3,200	1.0	0.1	250	25	70	25,550	9,125	1.8E-08	5.0E-08	--	0.1	--	--	5.0E-07	1.0E-03
Butyl Benzyl Phthalate	0.332	1.0E-06	3,200	1.0	0.1	250	25	70	25,550	9,125	3.7E-07	1.0E-06	--	0.2	--	--	5.0E-06	1.0E-02
Di-n-Butyl Phthalate	0.675	1.0E-06	3,200	1.0	0.1	250	25	70	25,550	9,125	7.5E-07	2.1E-06	--	0.1	--	--	2.1E-05	4.2E-02
Methylene Chloride	0.0064	1.0E-06	3,200	1.0	0.1	250	25	70	25,550	9,125	7.2E-09	2.0E-08	0.0075	0.06	5.4E-11	0.00	3.3E-07	0.001
Styrene	0.0081	1.0E-06	3,200	1.0	0.1	250	25	70	25,550	9,125	9.1E-09	2.5E-08	--	0.2	--	--	1.3E-07	2.6E-04
Trichloroethene	0.011	1.0E-06	3,200	1.0	0.1	250	25	70	25,550	9,125	1.2E-08	3.4E-08	0.011	0.006	1.3E-10	3.1E-03	5.7E-06	1.1E-02

Chronic Daily Intake (CDI) (Absorbed; mg/kg-day) = (CS x CF x SA x AF x ABS x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 4.2E-06 Hazard Index: 0.06 [d]

0.05 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

INHALATION OF PARTICULATES FROM SURFICIAL/SUBSURFACE SOILS (0-5 FEET)
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m3/day)	ED (years)	EF (days/yr)	BW (kg)	PEF (m^3/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																
Aluminum	21,500	20	9	250	70	1.32E+09	25,550	3,285	4.1E-07	3.2E-06	--	--	--	--	--	--
Antimony	1.64	20	9	250	70	1.32E+09	25,550	3,285	3.1E-11	2.4E-10	--	--	--	--	--	--
Arsenic	7.44	20	9	250	70	1.32E+09	25,550	3,285	1.4E-10	1.1E-09	15	--	2.1E-09	10.5	--	--
Barium	172	20	9	250	70	1.32E+09	25,550	3,285	3.3E-09	2.5E-08	--	0.00014	--	--	0.0002	1.4E+00
Beryllium	1.4	20	9	250	70	1.32E+09	25,550	3,285	2.7E-11	2.1E-10	8.4	0.0000057	2.3E-10	1.2	3.7E-05	2.6E-01
Cadmium	0.57	20	9	250	70	1.32E+09	25,550	3,285	1.1E-11	8.5E-11	6.3	--	6.9E-11	3.5E-01	--	--
Calcium	15,100	20	9	250	70	1.32E+09	25,550	3,285	2.9E-07	2.2E-06	--	--	--	--	--	--
Chromium (III)	22.7	20	9	250	70	1.32E+09	25,550	3,285	4.3E-10	3.4E-09	--	--	--	--	--	--
Chromium (VI)	22.7	20	9	250	70	1.32E+09	25,550	3,285	4.3E-10	3.4E-09	42	0.000029	1.8E-08	9.0E+01	1.17E-04	8.4E-01
Cobalt	19.8	20	9	250	70	1.32E+09	25,550	3,285	3.8E-10	2.9E-09	--	--	--	--	--	--
Copper	9.17	20	9	250	70	1.32E+09	25,550	3,285	1.7E-10	1.4E-09	--	--	--	--	--	--
Iron	26,000	20	9	250	70	1.32E+09	25,550	3,285	5.0E-07	3.9E-06	--	--	--	--	--	--
Lead	18.1	20	9	250	70	1.32E+09	25,550	3,285	3.5E-10	2.7E-09	--	--	--	--	--	--
Magnesium	1420	20	9	250	70	1.32E+09	25,550	3,285	2.7E-08	2.1E-07	--	--	--	--	--	--
Manganese	1270	20	9	250	70	1.32E+09	25,550	3,285	2.4E-08	1.9E-07	--	0.000014	--	--	0.014	1.0E+02
Mercury	0.07	20	9	250	70	1.32E+09	25,550	3,285	1.3E-12	1.0E-11	--	0.000086	--	--	1.2E-07	8.6E-04
Nickel	11.8	20	9	250	70	1.32E+09	25,550	3,285	2.2E-10	1.7E-09	--	--	--	--	--	--
Potassium	1560	20	9	250	70	1.32E+09	25,550	3,285	3.0E-08	2.3E-07	--	--	--	--	--	--
Selenium	0.74	20	9	250	70	1.32E+09	25,550	3,285	1.4E-11	1.1E-10	--	--	--	--	--	--
Silver	0.69	20	9	250	70	1.32E+09	25,550	3,285	1.3E-11	1.0E-10	--	--	--	--	--	--
Sodium	144	20	9	250	70	1.32E+09	25,550	3,285	2.7E-09	2.1E-08	--	--	--	--	--	--
Strontium	62.7	20	9	250	70	1.32E+09	25,550	3,285	1.2E-09	9.3E-09	--	--	--	--	--	--
Thallium	1.18	20	9	250	70	1.32E+09	25,550	3,285	2.2E-11	1.7E-10	--	--	--	--	--	--
Vanadium	43.3	20	9	250	70	1.32E+09	25,550	3,285	8.3E-10	6.4E-09	--	--	--	--	--	--
Zinc	79.2	20	9	250	70	1.32E+09	25,550	3,285	1.5E-09	1.2E-08	--	--	--	--	--	--
Organics																
Acetone	0.016	20	9	250	70	1.32E+09	25,550	3,285	3.0E-13	2.4E-12	--	--	--	--	--	--
Butyl Benzyl Phthalate	0.332	20	9	250	70	1.32E+09	25,550	3,285	6.3E-12	4.9E-11	--	--	--	--	--	--
Di-n-Butyl Phthalate	0.675	20	9	250	70	1.32E+09	25,550	3,285	1.3E-11	1.0E-10	--	--	--	--	--	--
Methylene Chloride	0.0064	20	9	250	70	1.32E+09	25,550	3,285	1.2E-13	9.5E-13	0.00165	0.857	2.0E-16	0.00	1.1E-12	7.9E-09
Styrene	0.0081	20	9	250	70	1.32E+09	25,550	3,285	1.5E-13	1.2E-12	--	0.286	--	--	4.2E-12	3.0E-08
Trichloroethene	0.011	20	9	250	70	1.32E+09	25,550	3,285	2.1E-13	1.6E-12	0.00595	--	1.2E-15	6.0E-06	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x IR x EF x ED) / (PEF*BW*AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i

Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 2.0E-08 Hazard Index: 0.014

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

INHALATION OF PARTICULATES FROM SURFICIAL/SUBSURFACE SOILS (0-5 FEET)
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m3/day)	ED (years)	EF (days/yr)	BW (kg)	PEF (m^3/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) (mg/kg-day)$^{-1}$	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																
Aluminum	21,500	20	25	250	70	1.32E+09	25,550	9,125	1.1E-06	3.2E-06	--	--	--	--	--	--
Antimony	1.64	20	25	250	70	1.32E+09	25,550	9,125	8.7E-11	2.4E-10	--	--	--	--	--	--
Arsenic	7.44	20	25	250	70	1.32E+09	25,550	9,125	3.9E-10	1.1E-09	15	--	5.9E-09	10.4	--	--
Barium	172	20	25	250	70	1.32E+09	25,550	9,125	9.1E-09	2.5E-08	--	0.00014	--	--	0.0002	1.4E+00
Beryllium	1.4	20	25	250	70	1.32E+09	25,550	9,125	7.4E-11	2.1E-10	8.4	0.0000057	6.2E-10	1.1	3.7E-05	2.6E-01
Cadmium	0.57	20	25	250	70	1.32E+09	25,550	9,125	3.0E-11	8.5E-11	6.3	--	1.9E-10	3.3E-01	--	--
Calcium	15,100	20	25	250	70	1.32E+09	25,550	9,125	8.0E-07	2.2E-06	--	--	--	--	--	--
Chromium (III)	22.7	20	25	250	70	1.32E+09	25,550	9,125	1.2E-09	3.4E-09	--	--	--	--	--	--
Chromium (VI)	22.7	20	25	250	70	1.32E+09	25,550	9,125	1.2E-09	3.4E-09	42	0.000029	5.0E-08	8.8E+01	1.17E-04	8.4E-01
Cobalt	19.8	20	25	250	70	1.32E+09	25,550	9,125	1.0E-09	2.9E-09	--	--	--	--	--	--
Copper	9.17	20	25	250	70	1.32E+09	25,550	9,125	4.9E-10	1.4E-09	--	--	--	--	--	--
Iron	26,000	20	25	250	70	1.32E+09	25,550	9,125	1.4E-06	3.9E-06	--	--	--	--	--	--
Lead	18.1	20	25	250	70	1.32E+09	25,550	9,125	9.6E-10	2.7E-09	--	--	--	--	--	--
Magnesium	1420	20	25	250	70	1.32E+09	25,550	9,125	7.5E-08	2.1E-07	--	--	--	--	--	--
Manganese	1270	20	25	250	70	1.32E+09	25,550	9,125	6.7E-08	1.9E-07	--	0.000014	--	--	0.014	1.0E+02
Mercury	0.07	20	25	250	70	1.32E+09	25,550	9,125	3.7E-12	1.0E-11	--	0.000086	--	--	1.2E-07	8.6E-04
Nickel	11.8	20	25	250	70	1.32E+09	25,550	9,125	6.2E-10	1.7E-09	--	--	--	--	--	--
Potassium	1560	20	25	250	70	1.32E+09	25,550	9,125	8.3E-08	2.3E-07	--	--	--	--	--	--
Selenium	0.74	20	25	250	70	1.32E+09	25,550	9,125	3.9E-11	1.1E-10	--	--	--	--	--	--
Silver	0.69	20	25	250	70	1.32E+09	25,550	9,125	3.7E-11	1.0E-10	--	--	--	--	--	--
Sodium	144	20	25	250	70	1.32E+09	25,550	9,125	7.6E-09	2.1E-08	--	--	--	--	--	--
Strontium	62.7	20	25	250	70	1.32E+09	25,550	9,125	3.3E-09	9.3E-09	--	--	--	--	--	--
Thallium	1.18	20	25	250	70	1.32E+09	25,550	9,125	6.2E-11	1.7E-10	--	--	--	--	--	--
Vanadium	43.3	20	25	250	70	1.32E+09	25,550	9,125	2.3E-09	6.4E-09	--	--	--	--	--	--
Zinc	79.2	20	25	250	70	1.32E+09	25,550	9,125	4.2E-09	1.2E-08	--	--	--	--	--	--
Organics																
Acetone	0.016	20	25	250	70	1.32E+09	25,550	9,125	8.5E-13	2.4E-12	--	--	--	--	--	--
Butyl Benzyl Phthalate	0.332	20	25	250	70	1.32E+09	25,550	9,125	1.8E-11	4.9E-11	--	--	--	--	--	--
Di-n-Butyl Phthalate	0.675	20	25	250	70	1.32E+09	25,550	9,125	3.6E-11	1.0E-10	--	--	--	--	--	--
Methylene Chloride	0.0064	20	25	250	70	1.32E+09	25,550	9,125	3.4E-13	9.5E-13	0.00165	0.857	5.6E-16	0.00	1.1E-12	7.9E-09
Styrene	0.0081	20	25	250	70	1.32E+09	25,550	9,125	4.3E-13	1.2E-12	--	0.286	--	--	4.2E-12	3.0E-08
Trichloroethene	0.011	20	25	250	70	1.32E+09	25,550	9,125	5.8E-13	1.6E-12	0.00595	--	3.5E-15	6.1E-06	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x IR x EF x ED) / (PEF*BW*AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 5.7E-08 Hazard Index: 0.014

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

INHALATION OF VOLATILES FROM SOILS
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m3/day)	ED (years)	EF (days/yr)	BW (kg)	VF (m^3/kg)	PEF (m^3/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																	
Aluminum	12,750	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Antimony	1.67	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Arsenic	5.49	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	15	--	--	--	--	--
Barium	179	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	0.00014	--	--	--	--
Beryllium	0.98	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	8.4	0.0000057	--	--	--	--
Cadmium	0.57	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	6.3	--	--	--	--	--
Calcium	5580	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Chromium (III)	19.4	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Chromium (VI)	19.4	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	42	0.000029	--	--	--	--
Cobalt	12.6	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Copper	7.19	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Iron	21400	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Lead	13.04	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Magnesium	1540	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Manganese	1270	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	0.000014	--	--	--	--
Mercury	0.07	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	0.000086	--	--	--	--
Nickel	15.8	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Potassium	917	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Selenium	0.68	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Silver	0.65	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Sodium	419	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Strontium	40.1	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Thallium	0.9	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Vanadium	33.8	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Zinc	44.3	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Organics																	
Acetone	0.013	20	9	250	70	5.12E+03	1.32E+09	25,550	3,285	6.4E-08	5.0E-07	--	--	--	--	--	--
Butyl Benzyl Phthalate	0.237	20	9	250	70	3.41E+06	1.32E+09	25,550	3,285	1.7E-09	1.4E-08	--	--	--	--	--	--
Di-n-Butyl Phthalate	0.373	20	9	250	70	3.27E+06	1.32E+09	25,550	3,285	2.9E-09	2.2E-08	--	--	--	--	--	--
Methylene Chloride	0.0049	20	9	250	70	1.02E+03	1.32E+09	25,550	3,285	1.2E-07	9.4E-07	0.00165	0.857	2.0E-10	6.67	1.1E-06	6.5E+01
Styrene	0.0044	20	9	250	70	5.47E+03	1.32E+09	25,550	3,285	2.0E-08	1.6E-07	--	0.286	--	--	5.6E-07	3.3E+01
Trichloroethene	0.025	20	9	250	70	1.33E+03	1.32E+09	25,550	3,285	4.7E-07	3.7E-06	0.00595	--	2.8E-09	9.3E+01	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x IR x EF x ED) / (VF*BW*AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i

Total Pathway Risk: 3.0E-09 Hazard Index: 0.0000017

Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

Constituent	Di (cm2/s)	H (dimensionless)	.Dw (cm2/s)	Kd (cm3/g)	Q/C	T (sec)	DA (cm2/s)	VF (m3/kg)
Acetone	0.124	1.59E-03	1.14E-05	0.00345	53.11	2.84E+08	1.066E-04	5.12E+03
Butyl Benzyl Phthalate	0.0174	5.17E-05	4.83E-06	345	53.11	2.84E+08	2.400E-10	3.41E+06
Di-n-Butyl Phthalate	0.0438	3.85E-08	7.86E-06	203.4	53.11	2.84E+08	2.621E-10	3.27E+06
Methylene Chloride	0.101	8.98E-02	1.17E-05	0.0702	53.11	2.84E+08	2.698E-03	1.02E+03
Styrene	0.071	0.113	8.00E-06	4.656	53.11	2.84E+08	9.352E-05	5.47E+03
Trichloroethene	0.079	0.422	9.10E-06	0.996	53.11	2.84E+08	1.579E-03	1.33E+03

VF calculated per USEPA Soil Screening Guidance (USEPA, 1996)

INHALATION OF VOLATILES FROM SOILS
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m3/day)	ED (years)	EF (days/yr)	BW (kg)	VF (m^3/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																
Aluminum	12,750	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Antimony	1.67	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Arsenic	5.49	20	25	250	70	--	25,550	9,125	--	--	15	--	--	--	--	--
Barium	179	20	25	250	70	--	25,550	9,125	--	--	--	0.00014	--	--	--	--
Beryllium	0.98	20	25	250	70	--	25,550	9,125	--	--	8.4	0.0000057	--	--	--	--
Cadmium	0.57	20	25	250	70	--	25,550	9,125	--	--	6.3	--	--	--	--	--
Calcium	5580	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Chromium (III)	19.4	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Chromium (VI)	19.4	20	25	250	70	--	25,550	9,125	--	--	42	0.000029	--	--	--	--
Cobalt	12.6	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Copper	7.19	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Iron	21400	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Lead	13.04	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Magnesium	1540	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Manganese	1270	20	25	250	70	--	25,550	9,125	--	--	--	0.000014	--	--	--	--
Mercury	0.07	20	25	250	70	--	25,550	9,125	--	--	--	0.000086	--	--	--	--
Nickel	15.8	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Potassium	917	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Selenium	0.68	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Silver	0.65	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Sodium	419	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Strontium	40.1	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Thallium	0.9	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Vanadium	33.8	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Zinc	44.3	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Organics																
Acetone	0.013	20	25	250	70	8.53E+03	25,550	9,125	1.1E-07	3.0E-07	--	--	--	--	--	--
Butyl Benzyl Phthalate	0.237	20	25	250	70	5.68E+06	25,550	9,125	2.9E-09	8.2E-09	--	--	--	--	--	--
Di-n-Butyl Phthalate	0.373	20	25	250	70	5.44E+06	25,550	9,125	4.8E-09	1.3E-08	--	--	--	--	--	--
Methylene Chloride	0.0049	20	25	250	70	1.70E+03	25,550	9,125	2.0E-07	5.6E-07	0.00165	0.857	3.3E-10	6.60	6.5E-07	6.5E+01
Styrene	0.0044	20	25	250	70	9.11E+03	25,550	9,125	3.4E-08	9.5E-08	--	0.286	--	--	3.3E-07	3.3E+01
Trichloroethene	0.025	20	25	250	70	2.22E+03	25,550	9,125	7.9E-07	2.2E-06	0.00595	--	4.7E-09	9.4E+01	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x IR x EF x ED) / (VF*PEF*BW*AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 5.0E-09 Hazard Index: 0.000001

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

Constituent	Di (cm2/s)	H (dimensionless)	Dw (cm2/s)	Kd (cm3/g)	Q/C	T (sec)	DA (cm2/s)	VF (m3/kg)
Acetone	0.124	1.59E-03	1.14E-05	0.0035	53.11	7.88E+08	1.065E-04	8.53E+03
Butyl Benzyl Phthalate	0.0174	5.17E-05	4.83E-06	345	53.11	7.88E+08	2.400E-10	5.68E+06
Di-n-Butyl Phthalate	0.0438	3.85E-08	7.86E-06	203.4	53.11	7.88E+08	2.621E-10	5.44E+06
Methylene Chloride	0.101	8.98E-02	1.17E-05	0.0702	53.11	7.88E+08	2.698E-03	1.70E+03
Styrene	0.071	0.113	8.00E-06	4.656	53.11	7.88E+08	9.352E-05	9.11E+03
Trichloroethene	0.079	0.422	9.10E-06	0.996	53.11	7.88E+08	1.579E-03	2.22E+03

VF calculated per USEPA Soil Screening Guidance (USEPA, 1996)

DERMAL CONTACT WITH SURFACE WATER (HARRISON BAYOU)
ON-SITE TRESPASSER
CURRENT SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CW (mg/L)	ED (years)	CF (L/cm²)	SA (cm²)	PC (cm/hr)	ET (hr/day)	EF (days/yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Absorbed) (mg/kg-day)	NC Chronic Daily Intake (Absorbed) (mg/kg-day)	Dermal Adjusted Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Dermal Adjusted Reference Dose (RfD) (mg/kg-day)	Dermal Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Dermal Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcino-genic Risk
Inorganics																		
Barium	0.146	10	0.001	3,400	0.001	2.6	52	43	25,550	3,650	6.1E-07	4.3E-06	--	0.021	--	--	0.0002	95.24
Copper	0.007	10	0.001	3,400	0.001	2.6	52	43	25,550	3,650	2.9E-08	2.1E-07	--	--	--	--	--	--
Lead	0.007	10	0.001	3,400	0.001	2.6	52	43	25,550	3,650	2.9E-08	2.1E-07	--	--	--	--	--	--
Zinc	0.025	10	0.001	3,400	0.001	2.6	52	43	25,550	3,650	1.0E-07	7.3E-07	--	0.09	--	--	8.1E-06	3.86
Organics																		
Acetone	0.0053	10	0.001	3,400	0.001	2.6	52	43	25,550	3,650	2.2E-08	1.6E-07	--	0.1	--	--	1.6E-06	0.76

Chronic Daily Intake (CDI) (Absorbed; mg/kg-day) = (CW x CF x SA x PC x ET x EF x ED) /(BW x AT)

Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i

Total Pathway Risk: 0.00E+00 Hazard Index: 0.00021

Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no permeability constant was available for this constituent.

[c] Dashes (--) indicate that no toxicity factor was available for this constituent.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

DERMAL CONTACT WITH SURFACE WATER (HARRISON BAYOU)
ON-SITE TRESPASSER
CURRENT SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CW (mg/L)	ED (years)	CF (L/cm²)	SA (cm²)	PC (cm/hr)	ET (hr/day)	EF (days/yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Absorbed) (mg/kg-day)	NC Chronic Daily Intake (Absorbed) (mg/kg-day)	Dermal Adjusted Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Dermal Adjusted Reference Dose (RfD) (mg/kg-day)	Dermal Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Dermal Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcino-genic Risk
Inorganics																		
Barium	0.146	10	0.001	4,100	0.001	2.6	60	43	25,550	3,650	8.5E-07	5.9E-06	--	0.021	--	--	0.0003	96.77
Copper	0.007	10	0.001	4,100	0.001	2.6	60	43	25,550	3,650	4.1E-08	2.9E-07	--	--	--	--	--	--
Lead	0.007	10	0.001	4,100	0.001	2.6	60	43	25,550	3,650	4.1E-08	2.9E-07	--	--	--	--	--	--
Zinc	0.025	10	0.001	4,100	0.001	2.6	60	43	25,550	3,650	1.5E-07	1.0E-06	--	0.09	--	--	1.1E-05	3.55
Organics																		
Acetone	0.0053	10	0.001	4,100	0.001	2.6	60	43	25,550	3,650	3.1E-08	2.2E-07	--	0.1	--	--	2.2E-06	0.71

Chronic Daily Intake (CDI) (Absorbed; mg/kg-day) = (CW x CF x SA x PC x ET x EF x ED) /(BW x AT)

Total Pathway Risk: 0.00E+00 Hazard Index: 0.00031

Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i

Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

[a]Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b]Dashes (--) indicate that no permeability constant was available for this constituent.

[c]Dashes (--) indicate that no toxicity factor was available for this constituent.

[d]Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e]Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

INCIDENTAL INGESTION OF SEDIMENT (HARRISON BAYOU)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	IR (mg/day)	FI (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Oral Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Oral Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcino-genic Risk
Inorganics																	
Arsenic	5.22	1.0E-06	100	0.5	52	10	43	25,550	3,650	1.2E-07	8.6E-07	1.5	0.0003	1.8E-07	1.0E+02	2.9E-03	8.1E+01
Barium	235.16	1.0E-06	100	0.5	52	10	43	25,550	3,650	5.6E-06	3.9E-05	--	0.07	--	--	5.6E-04	1.6E+01
Chromium (III)	18.57	1.0E-06	100	0.5	52	10	43	25,550	3,650	4.4E-07	3.1E-06	--	1.5	--	--	2.1E-06	5.8E-02
Chromium (VI)	18.57	1.0E-06	100	0.5	52	10	43	25,550	3,650	4.4E-07	3.1E-06	--	0.003	--	--	1.0E-03	2.8E+01
Copper	8.23	1.0E-06	100	0.5	52	10	43	25,550	3,650	1.9E-07	1.4E-06	--	--	--	--	--	--
Lead	19.5	1.0E-06	100	0.5	52	10	43	25,550	3,650	4.6E-07	3.2E-06	--	--	--	--	--	--
Nickel	13.79	1.0E-06	100	0.5	52	10	43	25,550	3,650	3.3E-07	2.3E-06	--	0.02	--	--	1.2E-04	3.3E+00
Selenium	1.16	1.0E-06	100	0.5	52	10	43	25,550	3,650	2.7E-08	1.9E-07	--	0.005	--	--	3.8E-05	1.1E+00
Thallium	0.59	1.0E-06	100	0.5	52	10	43	25,550	3,650	1.4E-08	9.8E-08	--	--	--	--	--	--
Zinc	38.49	1.0E-06	100	0.5	52	10	43	25,550	3,650	9.1E-07	6.4E-06	--	0.3	--	--	2.1E-05	5.8E-01
Organics																	
Acetone	0.048	1.0E-06	100	0.5	52	10	43	25,550	3,650	1.1E-09	8.0E-09	--	0.1	--	--	8.0E-08	2.2E-03

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x CF x IR x FI x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 1.8E-07 Hazard Index: 0.0046 [d]

0.0036 [c]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

00087977

INCIDENTAL INGESTION OF SEDIMENT (HARRISON BAYOU)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	IR (mg/day)	FI (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Oral Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Oral Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcino-genic Risk
Inorganics																	
Arsenic	5.22	1.0E-06	100	1.0	60	10	43	25,550	3,650	2.9E-07	2.0E-06	1.5	0.0003	4.4E-07	1.0E+02	6.7E-03	8.0E+01
Barium	235.16	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.3E-05	9.0E-05	--	0.07	--	--	1.3E-03	1.5E+01
Chromium (III)	18.57	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.0E-06	7.1E-06	--	1.5	--	--	4.7E-06	5.6E-02
Chromium (VI)	18.57	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.0E-06	7.1E-06	--	0.003	--	--	2.4E-03	2.9E+01
Copper	8.23	1.0E-06	100	1.0	60	10	43	25,550	3,650	4.5E-07	3.1E-06	--	--	--	--	--	--
Lead	19.5	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.1E-06	7.5E-06	--	--	--	--	--	--
Nickel	13.79	1.0E-06	100	1.0	60	10	43	25,550	3,650	7.5E-07	5.3E-06	--	0.02	--	--	2.7E-04	3.2E+00
Selenium	1.16	1.0E-06	100	1.0	60	10	43	25,550	3,650	6.3E-08	4.4E-07	--	0.005	--	--	8.8E-05	1.0E+00
Thallium	0.59	1.0E-06	100	1.0	60	10	43	25,550	3,650	3.2E-08	2.3E-07	--	--	--	--	--	--
Zinc	38.49	1.0E-06	100	1.0	60	10	43	25,550	3,650	2.1E-06	1.5E-05	--	0.3	--	--	5.0E-05	6.0E-01
Organics																	
Acetone	0.048	1.0E-06	100	1.0	60	10	43	25,550	3,650	2.6E-09	1.8E-08	--	0.1	--	--	1.8E-07	2.1E-03

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x CF x IR x FI x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i

Total Pathway Risk: 4.4E-07 Hazard Index: 0.011 [d]

Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

0.0084 [c]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

DERMAL CONTACT WITH SEDIMENT (HARRISON BAYOU)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	SA (cm^2/day)	AF (mg/cm^2)	ABS (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Absorbed) (mg/kg-day)	NC Chronic Daily Intake (Absorbed) (mg/kg-day)	Dermal Adjusted Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Dermal Adjusted Reference Dose (RfD) (mg/kg-day)	Dermal Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Dermal Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcino-genic Risk
Inorganics																		
Arsenic	5.22	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	1.7E-08	1.2E-07	5	9.0E-05	8.5E-08	100.0	0.001	76.9
Barium	235.16	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	7.6E-07	5.3E-06	--	0.021	--	--	2.5E-04	19.2
Chromium (III)	18.57	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	6.0E-08	4.2E-07	--	0.45	--	--	9.3E-07	0.072
Chromium (VI)	18.57	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	6.0E-08	4.2E-07	--	0.0009	--	--	0.000	0.0
Copper	8.23	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	2.6E-08	1.9E-07	--	--	--	--	--	--
Lead	19.5	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	6.3E-08	4.4E-07	--	--	--	--	--	--
Nickel	13.79	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	4.4E-08	3.1E-07	--	0.006	--	--	0.000	0.0
Selenium	1.16	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	3.7E-09	2.6E-08	--	0.0015	--	--	1.7E-05	1.31
Thallium	0.59	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	1.9E-09	1.3E-08	--	--	--	--	--	--
Zinc	38.49	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	1.2E-07	8.7E-07	--	0.09	--	--	9.7E-06	7.5E-01
Organics																		
Acetone	0.048	1.0E-06	3,400	0.2	0.1	52	10	43	25,550	3,650	1.5E-09	1.1E-08	--	0.1	--	--	1.1E-07	8.5E-03

Chronic Daily Intake (CDI) (Absorbed; mg/kg-day) = (CS x CF x SA x AF x ABS x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i

Total Pathway Risk: 8.5E-08 Hazard Index: 0.0013 [d]

Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

0.0013 [e]

[a] Dashes (–) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

00087979

DERMAL CONTACT WITH SEDIMENT (HARRISON BAYOU)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	SA (cm^2/day)	AF (mg/cm^2)	ABS (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Absorbed) (mg/kg-day)	NC Chronic Daily Intake (Absorbed) (mg/kg-day)	Dermal Adjusted Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Dermal Adjusted Reference Dose (RfD) (mg/kg-day)	Dermal Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Dermal Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcino-genic Risk
Inorganics																		
Arsenic	5.22	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	1.2E-07	8.2E-07	5	9.0E-05	6.0E-07	1.0E+02	9.1E-03	8.3E+01
Barium	235.16	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	5.3E-06	3.7E-05	--	0.021	--	--	1.8E-03	1.6E+01
Chromium (III)	18.57	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	4.2E-07	2.9E-06	--	0.45	--	--	6.4E-06	5.8E-02
Chromium (VI)	18.57	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	4.2E-07	2.9E-06	--	0.0009	--	--	3.2E-03	2.9E+01
Copper	8.23	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	1.8E-07	1.3E-06	--	--	--	--	--	--
Lead	19.5	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	4.4E-07	3.1E-06	--	--	--	--	--	--
Nickel	13.79	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	3.1E-07	2.2E-06	--	0.006	--	--	3.7E-04	3.4E+00
Selenium	1.16	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	2.6E-08	1.8E-07	--	0.0015	--	--	1.2E-04	1.1E+00
Thallium	0.59	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	1.3E-08	9.2E-08	--	--	--	--	--	--
Zinc	38.49	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	8.6E-07 [a]	6.0E-06 [a]	--	0.09	--	--	6.7E-05	6.1E-01
Organics																		
Acetone	0.048	1.0E-06	4,100	1.0	0.1	60	10	43	25,550	3,650	1.1E-08	7.5E-08	--	0.1	--	--	7.5E-07	6.8E-03

Chronic Daily Intake (CDI) (Absorbed; mg/kg-day) = (CS x CF x SA x AF x ABS x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i

Total Pathway Risk: 6.0E-07 Hazard Index: 0.015 [d]

Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

0.011 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

INGESTION OF FISH (FROM HARRISON BAYOU)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/g)	IR (g/day)	FI (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Oral Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Oral Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcino-genic Risk
Inorganics																	
Arsenic	--	1.0E-03	6.5	0.10	365	10	43	25,550	3,650	--	--	1.5	0.0003	--	--	--	--
Barium	--	1.0E-03	6.5	0.10	365	10	43	25,550	3,650	--	--	--	0.07	--	--	--	--
Chromium (III)	--	1.0E-03	6.5	0.10	365	10	43	25,550	3,650	--	--	--	1.5	--	--	--	--
Chromium (VI)	--	1.0E-03	6.5	0.10	365	10	43	25,550	3,650	--	--	--	0.003	--	--	--	--
Copper	1.4	1.0E-03	6.5	0.10	365	10	43	25,550	3,650	3.0E-06	2.1E-05	--	--	--	--	--	--
Lead	0.055	1.0E-03	6.5	0.10	365	10	43	25,550	3,650	1.2E-07	8.3E-07	--	--	--	--	--	--
Nickel	--	1.0E-03	6.5	0.10	365	10	43	25,550	3,650	--	--	--	0.02	--	--	--	--
Selenium	--	1.0E-03	6.5	0.10	365	10	43	25,550	3,650	--	--	--	0.005	--	--	--	--
Thallium	--	1.0E-03	6.5	0.10	365	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Zinc	17	1.0E-03	6.5	0.10	365	10	43	25,550	3,650	3.7E-05	2.6E-04	--	0.3	--	--	8.7E-04	8.7E+01
Organics																	
Acetone	0.002	1.0E-03	6.5	0.10	365	10	43	25,550	3,650	4.3E-09	3.0E-08	--	0.1	--	--	3.0E-07	3.0E-02

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x CF x IR x FI x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i

Total Pathway Risk: 0.0E+00 Hazard Index: 0.001

Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

INGESTION OF FISH (FROM HARRISON BAYOU)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/g)	IR (g/day)	FI (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Oral Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Oral Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																	
Arsenic	--	1.0E-03	54.0	0.25	365	10	43	25,550	3,650	--	--	1.5	0.0003	--	--	--	--
Barium	--	1.0E-03	54.0	0.25	365	10	43	25,550	3,650	--	--	--	0.07	--	--	--	--
Chromium (III)	--	1.0E-03	54.0	0.25	365	10	43	25,550	3,650	--	--	--	1.5	--	--	--	--
Chromium (VI)	--	1.0E-03	54.0	0.25	365	10	43	25,550	3,650	--	--	--	0.003	--	--	--	--
Copper	1.4	1.0E-03	54.0	0.25	365	10	43	25,550	3,650	6.3E-05	4.4E-04	--	--	--	--	--	--
Lead	0.055	1.0E-03	54.0	0.25	365	10	43	25,550	3,650	2.5E-06	1.7E-05	--	--	--	--	--	--
Nickel	--	1.0E-03	54.0	0.25	365	10	43	25,550	3,650	--	--	--	0.02	--	--	--	--
Selenium	--	1.0E-03	54.0	0.25	365	10	43	25,550	3,650	--	--	--	0.005	--	--	--	--
Thallium	--	1.0E-03	54.0	0.25	365	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Zinc	17	1.0E-03	54.0	0.25	365	10	43	25,550	3,650	7.6E-04	5.3E-03	--	0.3	--	--	1.8E-02	1.0E+02
Organics																	
Acetone	0.002	1.0E-03	54.0	0.25	365	10	43	25,550	3,650	9.0E-08	6.3E-07	--	0.1	--	--	6.3E-06	3.5E-02

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x CF x IR x FI x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i

Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 0.0E+00 Hazard Index: 0.018

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

INGESTION OF GROUNDWATER
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CW (mg/L)	IR (L/day)	EF (days/yr)	ED (years)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Oral Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Oral Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics															
Aluminum	67	0.7	250	9	70	25,550	3,285	5.9E-02	4.6E-01	--	--	--	--	--	--
Arsenic	0.034	0.7	250	9	70	25,550	3,285	3.0E-05	2.3E-04	1.5	0.0003	4.5E-05	0.2	0.8	0.1
Barium	9.9	0.7	250	9	70	25,550	3,285	8.7E-03	6.8E-02	--	0.07	--	--	0.97	0.15
Beryllium	0.0074	0.7	250	9	70	25,550	3,285	6.5E-06	5.1E-05	--	0.002	--	--	2.6E-02	0.00
Cadmium	0.008	0.7	250	9	70	25,550	3,285	7.0E-06	5.5E-05	--	0.0005	--	--	0.11	0.02
Calcium	490	0.7	250	9	70	25,550	3,285	4.3E-01	3.4E+00	--	--	--	--	--	--
Chromium (III)	5.22	0.7	250	9	70	25,550	3,285	4.6E-03	3.6E-02	--	1.5	--	--	0.02	0.00
Chromium (VI)	5.22	0.7	250	9	70	25,550	3,285	4.6E-03	3.6E-02	--	0.003	--	--	12.00	1.86
Cobalt	1.1	0.7	250	9	70	25,550	3,285	9.7E-04	7.5E-03	--	--	--	--	--	--
Copper	0.484	0.7	250	9	70	25,550	3,285	4.3E-04	3.3E-03	--	--	--	--	--	--
Iron	180	0.7	250	9	70	25,550	3,285	1.6E-01	1.2E+00	--	--	--	--	--	--
Lead	0.2	0.7	250	9	70	25,550	3,285	1.8E-04	1.4E-03	--	--	--	--	--	--
Magnesium	473	0.7	250	9	70	25,550	3,285	4.2E-01	3.2E+00	--	--	--	--	--	--
Manganese	29.8	0.7	250	9	70	25,550	3,285	2.6E-02	2.0E-01	--	0.14	--	--	1.43	0.22
Mercury	0.0016	0.7	250	9	70	25,550	3,285	1.4E-06	1.1E-05	--	--	--	--	--	--
Nickel	1.63	0.7	250	9	70	25,550	3,285	1.4E-03	1.1E-02	--	0.02	--	--	0.55	0.09
Potassium	15	0.7	250	9	70	25,550	3,285	1.3E-02	1.0E-01	--	--	--	--	--	--
Selenium	0.0156	0.7	250	9	70	25,550	3,285	1.4E-05	1.1E-04	--	0.005	--	--	0.02	0.00
Silver	0.114	0.7	250	9	70	25,550	3,285	1.0E-04	7.8E-04	--	0.005	--	--	0.16	0.02
Sodium	4400	0.7	250	9	70	25,550	3,285	3.9E+00	3.0E+01	--	--	--	--	--	--
Strontium	10.4	0.7	250	9	70	25,550	3,285	9.2E-03	7.1E-02	--	0.6	--	--	0.12	0.02
Thallium	0.012	0.7	250	9	70	25,550	3,285	1.1E-05	8.2E-05	--	--	--	--	--	--
Vanadium	0.146	0.7	250	9	70	25,550	3,285	1.3E-04	1.0E-03	--	0.007	--	--	0.14	0.02
Zinc	37	0.7	250	9	70	25,550	3,285	3.3E-02	2.5E-01	--	0.3	--	--	0.83	0.13
Organics															
Acetone	3.92	0.7	250	9	70	25,550	3,285	3.5E-03	2.7E-02	--	0.1	--	--	2.7E-01	4.2E-02
Aldrin	0.00004	0.7	250	9	70	25,550	3,285	3.5E-08	2.7E-07	17	0.00003	6.0E-07	3.0E-03	9.0E-03	1.4E-03
4-Amino-2,6-Dinitrotoluene	0.001	0.7	250	9	70	25,550	3,285	8.8E-07	6.8E-06	0.01	0.000167	8.8E-09	4.4E-05	4.1E-02	6.3E-03
Benzene	0.005	0.7	250	9	70	25,550	3,285	4.4E-06	3.4E-05	0.029	--	1.3E-07	6.5E-04	--	--
Bis(2-Ethyl Hexyl) Phthalate	0.026	0.7	250	9	70	25,550	3,285	2.3E-05	1.8E-04	0.014	0.02	3.2E-07	1.6E-03	9.0E-03	1.4E-03
Bromodichloromethane	0.0084	0.7	250	9	70	25,550	3,285	7.4E-06	5.8E-05	0.062	0.02	4.6E-07	2.3E-03	2.9E-03	4.5E-04
Butyl Benzyl Phthalate	0.007	0.7	250	9	70	25,550	3,285	6.2E-06	4.8E-05	--	0.2	--	--	2.4E-04	3.7E-05
Chloroform	0.12	0.7	250	9	70	25,550	3,285	1.1E-04	8.2E-04	0.0061	0.01	6.7E-07	3.4E-03	8.2E-02	1.3E-02
1,1-Dichloroethane	0.036	0.7	250	9	70	25,550	3,285	3.2E-05	2.5E-04	--	0.1	--	--	2.5E-03	3.9E-04
1,2-Dichloroethane	0.16	0.7	250	9	70	25,550	3,285	1.4E-04	1.1E-03	0.091	--	1.3E-05	6.5E-02	--	--
1,2-Dichloroethene	275	0.7	250	9	70	25,550	3,285	2.4E-01	1.9E+00	--	0.02	--	--	9.5E+01	1.5E+01
1,1-Dichloroethylene	0.74	0.7	250	9	70	25,550	3,285	6.5E-04	5.1E-03	0.6	0.009	3.9E-04	2.0E+00	5.7E-01	8.8E-02
cis-1,2-Dichloroethylene	520	0.7	250	9	70	25,550	3,285	4.6E-01	3.6E+00	--	0.01	--	--	3.6E+02	5.6E+01
1,3-Dinitrobenzene	0.00156	0.7	250	9	70	25,550	3,285	1.4E-06	1.1E-05	--	0.0001	--	--	1.1E-01	1.7E-02
2,6-Dinitrotoluene	0.00026	0.7	250	9	70	25,550	3,285	2.3E-07	1.8E-06	0.68	0.001	1.6E-07	8.0E-04	1.8E-03	2.8E-04
Ethylbenzene	0.005	0.7	250	9	70	25,550	3,285	4.4E-06	3.4E-05	--	0.1	--	--	3.4E-04	5.3E-05
HMX	0.0029	0.7	250	9	70	25,550	3,285	2.6E-06	2.0E-05	--	0.05	--	--	4.0E-04	6.2E-05
Methylene Chloride	3.5	0.7	250	9	70	25,550	3,285	3.1E-03	2.4E-02	0.0075	0.06	2.3E-05	1.2E-01	4.0E-01	6.2E-02
Methyl Ethyl Ketone	0.034	0.7	250	9	70	25,550	3,285	3.0E-05	2.3E-04	--	0.6	--	--	3.8E-04	5.9E-05
Nitrobenzene	0.02	0.7	250	9	70	25,550	3,285	1.8E-05	1.4E-04	--	0.0005	--	--	2.8E-01	4.3E-02
3-Nitrotoluene	0.011	0.7	250	9	70	25,550	3,285	9.7E-06	7.5E-05	--	0.01	--	--	7.5E-03	1.2E-03
RDX	0.2	0.7	250	9	70	25,550	3,285	1.8E-04	1.4E-03	0.11	0.003	2.0E-05	1.0E-01	4.7E-01	7.3E-02
Tetrachloroethylene	0.0014	0.7	250	9	70	25,550	3,285	1.2E-06	9.6E-06	--	0.01	--	--	9.6E-04	1.5E-04
Tetryl	0.036	0.7	250	9	70	25,550	3,285	3.2E-05	2.5E-04	--	--	--	--	--	--
Toluene	0.029	0.7	250	9	70	25,550	3,285	2.6E-05	2.0E-04	--	0.2	--	--	1.0E-03	1.5E-04
1,1,2-Trichloroethane	0.012	0.7	250	9	70	25,550	3,285	1.1E-05	8.2E-05	0.057	0.004	6.3E-07	3.2E-03	2.1E-02	3.3E-03
Trichloroethene	160	0.7	250	9	70	25,550	3,285	1.4E-01	1.1E+00	0.011	0.006	1.5E-03	7.5E+00	1.8E+02	2.8E+01
Trichlorofluoromethane	0.892	0.7	250	9	70	25,550	3,285	7.9E-04	6.1E-03	--	0.3	--	--	2.0E-02	3.1E-03
1,2,4-Trimethylbenzene	0.024	0.7	250	9	70	25,550	3,285	2.1E-05	1.6E-04	--	0.05	--	--	3.2E-03	5.0E-04
1,3,5-Trimethylbenzene	0.016	0.7	250	9	70	25,550	3,285	1.4E-05	1.1E-04	--	0.05	--	--	2.2E-03	3.4E-04
1,3,5-Trinitrobenzene	0.0022	0.7	250	9	70	25,550	3,285	1.9E-06	1.5E-05	--	0.03	--	--	5.0E-04	7.7E-05
2,4,6-Trinitrotoluene	0.24	0.7	250	9	70	25,550	3,285	2.1E-04	1.6E-03	0.03	0.0005	6.3E-06	3.2E-02	3.2E+00	5.0E-01
Vinyl Chloride	11	0.7	250	9	70	25,550	3,285	9.7E-03	7.5E-02	1.9	--	1.8E-02	9.0E+01	--	--
Xylene	0.012	0.7	250	9	70	25,550	3,285	1.1E-05	8.2E-05	--	2	--	--	4.1E-05	6.3E-06

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CW x IR x EF x ED) / (BW x AT)

Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i

Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 2.0E-02 Hazard Index: 657.7 [d] 645.7 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

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INGESTION OF GROUNDWATER
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CW (mg/L)	IR (L/day)	EF (days/yr)	ED (years)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Oral Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Oral Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics															
Aluminum	67	1.0	250	25	70	25,550	9,125	2.3E-01	6.6E-01	--	--	--	--	--	--
Arsenic	0.034	1.0	250	25	70	25,550	9,125	1.2E-04	3.3E-04	1.5	0.0003	1.8E-04	0.2	1.1	0.1
Barium	9.9	1.0	250	25	70	25,550	9,125	3.5E-02	9.7E-02	--	0.07	--	--	1.39	0.15
Beryllium	0.0074	1.0	250	25	70	25,550	9,125	2.6E-05	7.2E-05	--	0.002	--	--	3.6E-02	0.00
Cadmium	0.008	1.0	250	25	70	25,550	9,125	2.8E-05	7.8E-05	--	0.0005	--	--	0.16	0.02
Calcium	490	1.0	250	25	70	25,550	9,125	1.7E+00	4.8E+00	--	--	--	--	--	--
Chromium (III)	5.22	1.0	250	25	70	25,550	9,125	1.8E-02	5.1E-02	--	1.5	--	--	0.03	0.00
Chromium (VI)	5.22	1.0	250	25	70	25,550	9,125	1.8E-02	5.1E-02	--	0.003	--	--	17.00	1.82
Cobalt	1.1	1.0	250	25	70	25,550	9,125	3.8E-03	1.1E-02	--	--	--	--	--	--
Copper	0.484	1.0	250	25	70	25,550	9,125	1.7E-03	4.7E-03	--	--	--	--	--	--
Iron	180	1.0	250	25	70	25,550	9,125	6.3E-01	1.8E+00	--	--	--	--	--	--
Lead	0.2	1.0	250	25	70	25,550	9,125	7.0E-04	2.0E-03	--	--	--	--	--	--
Magnesium	473	1.0	250	25	70	25,550	9,125	1.7E+00	4.6E+00	--	--	--	--	--	--
Manganese	29.8	1.0	250	25	70	25,550	9,125	1.0E-01	2.9E-01	--	0.14	--	--	2.07	0.22
Mercury	0.0016	1.0	250	25	70	25,550	9,125	5.6E-06	1.6E-05	--	--	--	--	--	--
Nickel	1.63	1.0	250	25	70	25,550	9,125	5.7E-03	1.6E-02	--	0.02	--	--	0.80	0.09
Potassium	15	1.0	250	25	70	25,550	9,125	5.2E-02	1.5E-01	--	--	--	--	--	--
Selenium	0.0156	1.0	250	25	70	25,550	9,125	5.5E-05	1.5E-04	--	0.005	--	--	0.03	0.00
Silver	0.114	1.0	250	25	70	25,550	9,125	4.0E-04	1.1E-03	--	0.005	--	--	0.22	0.02
Sodium	4400	1.0	250	25	70	25,550	9,125	1.5E+01	4.3E+01	--	--	--	--	--	--
Strontium	10.4	1.0	250	25	70	25,550	9,125	3.6E-02	1.0E-01	--	0.6	--	--	0.17	0.02
Thallium	0.012	1.0	250	25	70	25,550	9,125	4.2E-05	1.2E-04	--	--	--	--	--	--
Vanadium	0.146	1.0	250	25	70	25,550	9,125	5.1E-04	1.4E-03	--	0.007	--	--	0.20	0.02
Zinc	37	1.0	250	25	70	25,550	9,125	1.3E-01	3.6E-01	--	0.3	--	--	1.20	0.13
Organics															
Acetone	3.92	1.0	250	25	70	25,550	9,125	1.4E-02	3.8E-02	--	0.1	--	--	3.8E-01	4.1E-02
Aldrin	0.00004	1.0	250	25	70	25,550	9,125	1.4E-07	3.9E-07	17	0.00003	2.4E-06	3.1E-03	1.3E-02	1.4E-03
4-Amino-2,6-Dinitrotoluene	0.001	1.0	250	25	70	25,550	9,125	3.5E-06	9.8E-06	0.01	0.000167	3.5E-08	4.5E-05	5.9E-02	6.3E-03
Benzene	0.005	1.0	250	25	70	25,550	9,125	1.7E-05	4.9E-05	0.029	--	4.9E-07	6.3E-04	--	--
Bis(2-Ethyl Hexyl) Phthalate	0.026	1.0	250	25	70	25,550	9,125	9.1E-05	2.5E-04	0.014	0.02	1.3E-06	1.7E-03	1.3E-02	1.4E-03
Bromodichloromethane	0.0084	1.0	250	25	70	25,550	9,125	2.9E-05	8.2E-05	0.062	0.02	1.8E-06	2.3E-03	4.1E-03	4.4E-04
Butyl Benzyl Phthalate	0.007	1.0	250	25	70	25,550	9,125	2.4E-05	6.8E-05	--	0.2	--	--	3.4E-04	3.6E-05
Chloroform	0.12	1.0	250	25	70	25,550	9,125	4.2E-04	1.2E-03	0.0061	0.01	2.6E-06	3.3E-03	1.2E-01	1.3E-02
1,1-Dichloroethane	0.036	1.0	250	25	70	25,550	9,125	1.3E-04	3.5E-04	--	0.1	--	--	3.5E-03	3.7E-04
1,2-Dichloroethane	0.16	1.0	250	25	70	25,550	9,125	5.6E-04	1.6E-03	0.091	--	5.1E-05	6.5E-02	--	--
1,2-Dichloroethene	275	1.0	250	25	70	25,550	9,125	9.6E-01	2.7E+00	--	0.02	--	--	1.4E+02	1.5E+01
1,1-Dichloroethylene	0.74	1.0	250	25	70	25,550	9,125	2.6E-03	7.2E-03	0.6	0.009	1.6E-03	2.1E+00	8.0E-01	8.6E-02
cis-1,2-Dichloroethylene	520	1.0	250	25	70	25,550	9,125	1.8E+00	5.1E+00	--	0.01	--	--	5.1E+02	5.5E+01
1,3-Dinitrobenzene	0.00156	1.0	250	25	70	25,550	9,125	5.5E-06	1.5E-05	--	0.0001	--	--	1.5E-01	1.6E-02
2,6-Dinitrotoluene	0.00026	1.0	250	25	70	25,550	9,125	9.2E-07	2.6E-06	0.68	0.001	6.3E-07	8.1E-04	2.6E-03	2.8E-04
Ethylbenzene	0.005	1.0	250	25	70	25,550	9,125	1.7E-05	4.9E-05	--	0.1	--	--	4.9E-04	5.2E-05
HMX	0.0029	1.0	250	25	70	25,550	9,125	1.0E-05	2.8E-05	--	0.05	--	--	5.6E-04	6.0E-05
Methylene Chloride	3.5	1.0	250	25	70	25,550	9,125	1.2E-02	3.4E-02	0.0075	0.06	9.0E-05	1.2E-01	5.7E-01	6.1E-02
Methyl Ethyl Ketone	0.034	1.0	250	25	70	25,550	9,125	1.2E-04	3.3E-04	--	0.6	--	--	5.5E-04	5.9E-05
Nitrobenzene	0.02	1.0	250	25	70	25,550	9,125	7.0E-05	2.0E-04	--	0.0005	--	--	4.0E-01	4.3E-02
3-Nitrotoluene	0.011	1.0	250	25	70	25,550	9,125	3.8E-05	1.1E-04	--	0.01	--	--	1.1E-02	1.2E-03
RDX	0.2	1.0	250	25	70	25,550	9,125	7.0E-04	2.0E-03	0.11	0.003	7.7E-05	9.9E-02	6.7E-01	7.2E-02
Tetrachloroethylene	0.0014	1.0	250	25	70	25,550	9,125	4.9E-06	1.4E-05	--	0.01	--	--	1.4E-03	1.5E-04
Tetryl	0.036	1.0	250	25	70	25,550	9,125	1.3E-04	3.5E-04	--	--	--	--	--	--
Toluene	0.029	1.0	250	25	70	25,550	9,125	1.0E-04	2.8E-04	--	0.2	--	--	1.4E-03	1.5E-04
1,1,2-Trichloroethane	0.012	1.0	250	25	70	25,550	9,125	4.2E-05	1.2E-04	0.057	0.004	2.4E-06	3.1E-03	3.0E-02	3.2E-03
Trichloroethene	160	1.0	250	25	70	25,550	9,125	5.6E-01	1.6E+00	0.011	0.006	6.2E-03	7.9E+00	2.7E+02	2.9E+01
Trichlorofluoromethane	0.892	1.0	250	25	70	25,550	9,125	3.1E-03	8.7E-03	--	0.3	--	--	2.9E-02	3.1E-03
1,2,4-Trimethylbenzene	0.024	1.0	250	25	70	25,550	9,125	8.4E-05	2.3E-04	--	0.05	--	--	4.6E-03	4.9E-04
1,3,5-Trimethylbenzene	0.016	1.0	250	25	70	25,550	9,125	5.6E-05	1.6E-04	--	0.05	--	--	3.2E-03	3.4E-04
1,3,5-Trinitrobenzene	0.0022	1.0	250	25	70	25,550	9,125	7.7E-06	2.2E-05	--	0.03	--	--	7.3E-04	7.8E-05
2,4,6-Trinitrotoluene	0.24	1.0	250	25	70	25,550	9,125	8.4E-04	2.3E-03	0.03	0.0005	2.5E-05	3.2E-02	4.6E+00	4.9E-01
Vinyl Chloride	11	1.0	250	25	70	25,550	9,125	3.8E-02	1.1E-01	1.9	--	7.0E-02	9.0E+01	--	--
Xylene	0.012	1.0	250	25	70	25,550	9,125	4.2E-05	1.2E-04	--	2	--	--	6.0E-05	6.4E-06

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CW x IR x EF x ED) / (BW x AT)

Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i

Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 7.8E-02 Hazard Index: 952.2 [d] 935.3 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

DERMAL CONTACT WITH GROUNDWATER DURING SHOWERING
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CW (mg/L)	ED (years)	CF (L/cm³)	SA (cm²)	PC (cm/hr)	ET (hr/day)	EF (days/yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Absorbed) (mg/kg-day)	NC Chronic Daily Intake (Absorbed) (mg/kg-day)	Dermal Adjusted Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Dermal Adjusted Reference Dose (RfD) (mg/kg-day)	Dermal Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Dermal Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																		
Aluminum	-- [a]	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	--	--	--	--	--
Arsenic	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	5	0.00009	--	--	--	--
Barium	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	0.021	--	--	--	--
Beryllium	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	0.0006	--	--	--	--
Cadmium	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	0.00015	--	--	--	--
Calcium	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	--	--	--	--	--
Chromium (III)	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	0.45	--	--	--	--
Chromium (VI)	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	0.0009	--	--	--	--
Cobalt	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	--	--	--	--	--
Copper	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	--	--	--	--	--
Iron	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	--	--	--	--	--
Lead	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	--	--	--	--	--
Magnesium	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	--	--	--	--	--
Manganese	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	0.042	--	--	--	--
Mercury	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	--	--	--	--	--
Nickel	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	0.006	--	--	--	--
Potassium	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	--	--	--	--	--
Selenium	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	0.0015	--	--	--	--
Silver	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	0.0015	--	--	--	--
Sodium	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	--	--	--	--	--
Strontium	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	0.18	--	--	--	--
Thallium	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	--	--	--	--	--
Vanadium	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	0.0021	--	--	--	--
Zinc	--	9	0.001	20,000	0.001	0.12	250	70	25,550	3,285	--	--	--	0.09	--	--	--	--
Organics																		
Acetone	--	9	0.001	20,000	0.00058	0.12	250	70	25,550	3,285	--	--	--	0.1	--	--	--	--
Aldrin	--	9	0.001	20,000	0.0016	0.12	250	70	25,550	3,285	--	--	17	0.00003	--	--	--	--
4-Amino-2,6-Dinitrotoluene	0.001	9	0.001	20,000	--	0.12	250	70	25,550	3,285	--	--	0.01	0.000167	--	--	--	--
Benzene	0.005	9	0.001	20,000	0.1	0.12	250	70	25,550	3,285	1.5E-06	1.2E-05	0.029	--	4.4E-08	4.0E-03	--	--
Bis(2-Ethyl Hexyl) Phthalate	--	9	0.001	20,000	0.0076	0.12	250	70	25,550	3,285	--	--	0.014	0.02	--	--	--	--
Bromodichloromethane	--	9	0.001	20,000	0.0058	0.12	250	70	25,550	3,285	--	--	0.062	0.02	--	--	--	--
Butyl Benzyl Phthalate	0.007	9	0.001	20,000	0.018	0.12	250	70	25,550	3,285	3.8E-07	3.0E-06	--	0.2	--	--	0.00002	0.00
Chloroform	0.12	9	0.001	20,000	0.1	0.12	250	70	25,550	3,285	3.6E-05	2.8E-04	0.0061	0.01	2.2E-07	2.0E-02	0.0280	0.02
1,1-Dichloroethane	--	9	0.001	20,000	0.0089	0.12	250	70	25,550	3,285	--	--	--	0.1	--	--	--	--
1,2-Dichloroethane	--	9	0.001	20,000	0.0053	0.12	250	70	25,550	3,285	--	--	0.091	--	--	--	--	--
1,2-Dichloroethene	--	9	0.001	20,000	0.01	0.12	250	70	25,550	3,285	--	--	--	0.020	--	--	--	--
1,1-Dichloroethylene	0.74	9	0.001	20,000	0.016	0.12	250	70	25,550	3,285	3.6E-05	2.8E-04	0.6	0.009	2.2E-05	2.0E+00	0.0311	0.02
cis-1,2-Dichloroethylene	--	9	0.001	20,000	0.01	0.12	250	70	25,550	3,285	--	--	--	0.01	--	--	--	--
1,3-Dinitrobenzene	--	9	0.001	20,000	0.002	0.12	250	70	25,550	3,285	--	--	--	0.0001	--	--	--	--
2,6-Dinitrotoluene	--	9	0.001	20,000	0.0025	0.12	250	70	25,550	3,285	--	--	0.68	0.001	--	--	--	--
Ethylbenzene	0.005	9	0.001	20,000	1	0.12	250	70	25,550	3,285	1.5E-05	1.2E-04	--	0.1	--	--	0.0012	0.00
HMX	0.0029	9	0.001	20,000	--	0.12	250	70	25,550	3,285	--	--	--	0.05	--	--	--	--
Methylene Chloride	--	9	0.001	20,000	0.0045	0.12	250	70	25,550	3,285	--	--	0.0075	0.06	--	--	--	--
Methyl Ethyl Ketone	--	9	0.001	20,000	0.005	0.12	250	70	25,550	3,285	--	--	--	0.6	--	--	--	--
Nitrobenzene	--	9	0.001	20,000	0.006	0.12	250	70	25,550	3,285	--	--	--	0.0005	--	--	--	--
3-Nitrotoluene	0.011	9	0.001	20,000	0.015	0.12	250	70	25,550	3,285	5.0E-07	3.9E-06	--	0.01	--	--	0.0004	0.00
RDX	--	9	0.001	20,000	0.00035	0.12	250	70	25,550	3,285	--	--	0.11	0.003	--	--	--	--
Tetrachloroethylene	0.0014	9	0.001	20,000	0.4	0.12	250	70	25,550	3,285	1.7E-06	1.3E-05	--	0.01	--	--	0.0013	0.00
Tetryl	--	9	0.001	20,000	0.0009	0.12	250	70	25,550	3,285	--	--	--	--	--	--	--	--
Toluene	0.029	9	0.001	20,000	1	0.12	250	70	25,550	3,285	8.8E-05	6.8E-04	--	0.2	--	--	0.0034	0.00
1,1,2-Trichloroethane	--	9	0.001	20,000	0.0084	0.12	250	70	25,550	3,285	--	--	0.057	0.004	--	--	--	--
Trichloroethene	160	9	0.001	20,000	0.2	0.12	250	70	25,550	3,285	9.7E-02	7.5E-01	0.011	0.006	1.1E-03	1.0E+02	125.0000	99.94
Trichlorofluoromethane	0.892	9	0.001	20,000	0.017	0.12	250	70	25,550	3,285	4.6E-05	3.6E-04	--	0.3	--	--	0.0012	0.00
1,2,4-Trimethylbenzene	0.024	9	0.001	20,000	0.138	0.12	250	70	25,550	3,285	1.0E-05	7.8E-05	--	0.05	--	--	0.0016	0.00
1,3,5-Trimethylbenzene	0.016	9	0.001	20,000	0.093	0.12	250	70	25,550	3,285	4.5E-06	3.5E-05	--	0.05	--	--	0.0007	0.00
1,3,5-Trinitrobenzene	0.0022	9	0.001	20,000	--	0.12	250	70	25,550	3,285	--	--	--	0.03	--	--	--	--
2,4,6-Trinitrotoluene	--	9	0.001	20,000	0.003	0.12	250	70	25,550	3,285	--	--	0.03	0.0005	--	--	--	--
Vinyl Chloride	--	9	0.001	20,000	0.0073	0.12	250	70	25,550	3,285	--	--	1.9	--	--	--	--	--
Xylene	0.012	9	0.001	20,000	0.08	0.12	250	70	25,550	3,285	2.9E-06	2.3E-05	--	2	--	--	0.00001	0.00

Chronic Daily Intake (CDI) (Absorbed; mg/kg-day) = (CW x CF x SA x PC x ET x EF x ED) /(BW x AT)

Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i

Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 1.1E-03 Hazard Index: 125.069

[a] Dashes (--) indicate that this constituent was not retained for the dermal calculation since the PC value was not greater than 0.01.

[b] Dashes (--) indicate that no permeability constant was available for this constituent.

[c] Dashes (--) indicate that no toxicity factor was available for this constituent.

DERMAL CONTACT WITH GROUNDWATER DURING SHOWERING
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CW (mg/L)	ED (years)	CF (L/cm³)	SA (cm²)	PC (cm/hr)	ET (hr/day)	EF (days/yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Absorbed) (mg/kg-day)	NC Chronic Daily Intake (Absorbed) (mg/kg-day)	Dermal Adjusted Slope Factor (SF) (mg/kg-day)⁻¹	Dermal Adjusted Reference Dose (RfD) (mg/kg-day)	Dermal Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Dermal Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																		
Aluminum	-- [a]	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	--	--	--	--	--
Arsenic	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	5	0.00009	--	--	--	--
Barium	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	0.021	--	--	--	--
Beryllium	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	0.0006	--	--	--	--
Cadmium	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	0.00015	--	--	--	--
Calcium	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	--	--	--	--	--
Chromium (III)	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	0.45	--	--	--	--
Chromium (VI)	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	0.0009	--	--	--	--
Cobalt	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	--	--	--	--	--
Copper	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	--	--	--	--	--
Iron	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	--	--	--	--	--
Lead	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	--	--	--	--	--
Magnesium	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	--	--	--	--	--
Manganese	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	0.042	--	--	--	--
Mercury	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	--	--	--	--	--
Nickel	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	0.006	--	--	--	--
Potassium	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	--	--	--	--	--
Selenium	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	0.0015	--	--	--	--
Silver	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	0.0015	--	--	--	--
Sodium	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	--	--	--	--	--
Strontium	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	0.18	--	--	--	--
Thallium	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	--	--	--	--	--
Vanadium	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	0.0021	--	--	--	--
Zinc	--	25	0.001	23,000	0.001	0.2	250	70	25,550	9,125	--	--	--	0.09	--	--	--	--
Organics																		
Acetone	--	25	0.001	23,000	0.00058	0.2	250	70	25,550	9,125	--	--	--	0.1	--	--	--	--
Aldrin	--	25	0.001	23,000	0.0016	0.2	250	70	25,550	9,125	--	--	17	0.00003	--	--	--	--
4-Amino-2,6-Dinitrotoluene	0.001	25	0.001	23,000	--	0.2	250	70	25,550	9,125	--	--	0.01	0.000167	--	--	--	--
Benzene	0.005	25	0.001	23,000	0.1	0.2	250	70	25,550	9,125	8.0E-06	2.3E-05	0.029	--	2.3E-07	4.0E-03	--	--
Bis(2-Ethyl Hexyl) Phthalate	--	25	0.001	23,000	0.0076	0.2	250	70	25,550	9,125	--	--	0.014	0.02	--	--	--	--
Bromodichloromethane	--	25	0.001	23,000	0.0058	0.2	250	70	25,550	9,125	--	--	0.062	0.02	--	--	--	--
Butyl Benzyl Phthalate	0.007	25	0.001	23,000	0.018	0.2	250	70	25,550	9,125	2.0E-06	5.7E-06	--	0.2	--	--	2.9E-05	0.00
Chloroform	0.12	25	0.001	23,000	0.1	0.2	250	70	25,550	9,125	1.9E-04	5.4E-04	0.0061	0.01	1.2E-06	2.1E-02	5.4E-02	0.02
1,1-Dichloroethane	--	25	0.001	23,000	0.0089	0.2	250	70	25,550	9,125	--	--	--	0.1	--	--	--	--
1,2-Dichloroethane	--	25	0.001	23,000	0.0053	0.2	250	70	25,550	9,125	--	--	0.091	--	--	--	--	--
1,2-Dichloroethene	--	25	0.001	23,000	0.01	0.2	250	70	25,550	9,125	--	--	--	0.02	--	--	--	--
1,1-Dichloroethylene	0.74	25	0.001	23,000	0.016	0.2	250	70	25,550	9,125	1.9E-04	5.3E-04	0.6	0.009	1.1E-04	1.9E+00	5.9E-02	0.03
cis-1,2-Dichloroethylene	--	25	0.001	23,000	0.01	0.2	250	70	25,550	9,125	--	--	--	0.01	--	--	--	--
1,3-Dinitrobenzene	--	25	0.001	23,000	0.002	0.2	250	70	25,550	9,125	--	--	--	0.0001	--	--	--	--
2,6-Dinitrotoluene	--	25	0.001	23,000	0.0025	0.2	250	70	25,550	9,125	--	--	0.68	0.001	--	--	--	--
Ethylbenzene	0.005	25	0.001	23,000	1	0.2	250	70	25,550	9,125	8.0E-05	2.3E-04	--	0.1	--	--	2.3E-03	0.00
HMX	0.0029	25	0.001	23,000	--	0.2	250	70	25,550	9,125	--	--	--	0.05	--	--	--	--
Methylene Chloride	--	25	0.001	23,000	0.0045	0.2	250	70	25,550	9,125	--	--	0.0075	0.06	--	--	--	--
Methyl Ethyl Ketone	--	25	0.001	23,000	0.005	0.2	250	70	25,550	9,125	--	--	--	0.6	--	--	--	--
Nitrobenzene	--	25	0.001	23,000	0.006	0.2	250	70	25,550	9,125	--	--	--	0.0005	--	--	--	--
3-Nitrotoluene	0.011	25	0.001	23,000	0.015	0.2	250	70	25,550	9,125	2.7E-06	7.4E-06	--	0.01	--	--	7.4E-04	0.00
RDX	--	25	0.001	23,000	0.00035	0.2	250	70	25,550	9,125	--	--	0.11	0.003	--	--	--	--
Tetrachloroethylene	0.0014	25	0.001	23,000	0.4	0.2	250	70	25,550	9,125	9.0E-06	2.5E-05	--	0.01	--	--	2.5E-03	0.00
Tetryl	--	25	0.001	23,000	0.0009	0.2	250	70	25,550	9,125	--	--	--	--	--	--	--	--
Toluene	0.029	25	0.001	23,000	1	0.2	250	70	25,550	9,125	4.7E-04	1.3E-03	--	0.2	--	--	6.5E-03	0.00
1,1,2-Trichloroethane	--	25	0.001	23,000	0.0084	0.2	250	70	25,550	9,125	--	--	0.057	0.004	--	--	--	--
Trichloroethene	160	25	0.001	23,000	0.2	0.2	250	70	25,550	9,125	5.1E-01	1.4E+00	0.011	0.006	5.6E-03	9.8E+01	2.3E+02	99.94
Trichlorofluoromethane	0.892	25	0.001	23,000	0.017	0.2	250	70	25,550	9,125	2.4E-04	6.8E-04	--	0.3	--	--	2.3E-03	0.00
1,2,4-Trimethylbenzene	0.024	25	0.001	23,000	0.138	0.2	250	70	25,550	9,125	5.3E-05	1.5E-04	--	0.05	--	--	3.0E-03	0.00
1,3,5-Trimethylbenzene	0.016	25	0.001	23,000	0.093	0.2	250	70	25,550	9,125	2.4E-05	6.7E-05	--	0.05	--	--	1.3E-03	0.00
1,3,5-Trinitrobenzene	0.0022	25	0.001	23,000	--	0.2	250	70	25,550	9,125	--	--	--	0.03	--	--	--	--
2,4,6-Trinitrotoluene	--	25	0.001	23,000	0.003	0.2	250	70	25,550	9,125	--	--	0.03	0.0005	--	--	--	--
Vinyl Chloride	--	25	0.001	23,000	0.0073	0.2	250	70	25,550	9,125	--	--	1.9	--	--	--	--	--
Xylene	0.012	25	0.001	23,000	0.08	0.2	250	70	25,550	9,125	1.5E-05	4.3E-05	--	2	--	--	2.2E-05	0.00

Chronic Daily Intake (CDI) (Absorbed; mg/kg-day) = (CW x CF x SA x PC x ET x EF x ED)/(BW x AT)

Chemical-Specific CR = CDI x SF; Total CR = Σ CR$_i$

Chemical-Specific HQ = CDI / RfD; HI = CDI$_1$/RfD$_1$ + CDI$_2$/RfD$_2$ + ... + CDI$_i$/RfD$_i$

Total Pathway Risk: 5.7E-03 Hazard Index: 230.132

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no permeability constant was available for this constituent.

[c] Dashes (--) indicate that no toxicity factor was available for this constituent.

INHALATION OF VAPORS FROM GROUNDWATER DURING SHOWERING
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CW (mg/L)	IR (m^3/day)	K (unitless)	ED (years)	EF (days/yr)	CF (L/m3)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Inhalation Reference Dose (RfD) (mg/kg-day)	Inhalation Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Inhalation Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																	
Aluminum	67	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	-- [c]	-- [c]	--	--	--	--
Arsenic	0.034	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	15	-- [c]	--	--	--	--
Barium	9.9	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	-- [c]	1.4E-04	--	--	--	--
Beryllium	0.0074	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	8.4	0.0000057	--	--	--	--
Cadmium	0.008	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	6.3	-- [c]	--	--	--	--
Calcium	490	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	--	--	--	--	--	--
Chromium (III)	5.22	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	-- [c]	-- [c]	--	--	--	--
Chromium (VI)	5.22	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	42	0.000029	--	--	--	--
Cobalt	1.1	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	--	--	--	--	--	--
Copper	0.484	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	--	--	--	--	--	--
Iron	180	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	--	--	--	--	--	--
Lead	0.2	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	-- [c]	-- [c]	--	--	--	--
Magnesium	473	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	--	--	--	--	--	--
Manganese	29.8	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	-- [c]	1.4E-05	--	--	--	--
Mercury	0.0016	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	-- [c]	8.6E-05	--	--	--	--
Nickel	1.63	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	--	--	--	--	--	--
Potassium	15	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	--	--	--	--	--	--
Selenium	0.0156	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	-- [c]	-- [c]	--	--	--	--
Silver	0.114	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	-- [c]	-- [c]	--	--	--	--
Sodium	4400	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	--	--	--	--	--	--
Strontium	10.4	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	--	--	--	--	--	--
Thallium	0.012	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	--	--	--	--	--	--
Vanadium	0.146	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	--	--	--	--	--	--
Zinc	37	7.5	-- [b]	9	250	1000	70	25,550	3,285	--	--	-- [c]	-- [c]	--	--	--	--
Organics																	
Acetone	3.92	7.5	0.0005	9	250	1000	70	25,550	3,285	1.8E-02	1.4E-01	--	--	--	--	--	--
Aldrin	0.00004	7.5	--	9	250	1000	70	25,550	3,285	--	--	17.2	--	--	--	--	--
4-Amino-2,6-Dinitrotoluene	0.001	7.5	0.0005	9	250	1000	70	25,550	3,285	4.7E-06	3.7E-05	--	0.000029	--	--	1.2759	2.6
Benzene	0.005	7.5	0.0005	9	250	1000	70	25,550	3,285	2.4E-05	1.8E-04	0.027	--	6.5E-07	3.3E-03	--	--
Bis(2-Ethyl Hexyl) Phthalate	0.026	7.5	--	9	250	1000	70	25,550	3,285	--	--	--	--	--	--	--	--
Bromodichloromethane	0.0084	7.5	0.0005	9	250	1000	70	25,550	3,285	4.0E-05	3.1E-04	--	--	--	--	--	--
Butyl Benzyl Phthalate	0.007	7.5	--	9	250	1000	70	25,550	3,285	--	--	--	--	--	--	--	--
Chloroform	0.12	7.5	0.0005	9	250	1000	70	25,550	3,285	5.7E-04	4.4E-03	0.081	--	4.6E-05	2.3E-01	--	--
1,1-Dichloroethane	0.036	7.5	0.0005	9	250	1000	70	25,550	3,285	1.7E-04	1.3E-03	--	0.143	--	--	0.0091	0.0
1,2-Dichloroethane	0.16	7.5	0.0005	9	250	1000	70	25,550	3,285	7.5E-04	5.9E-03	0.091	--	6.8E-05	3.4E-01	--	--
1,2-Dichloroethene	275	7.5	0.0005	9	250	1000	70	25,550	3,285	1.3E+00	1.0E+01	--	0.22	--	--	45.4545	93.9
1,1-Dichloroethylene	0.74	7.5	0.0005	9	250	1000	70	25,550	3,285	3.5E-03	2.7E-02	0.18	--	6.3E-04	3.2E+00	--	--
cis-1,2-Dichloroethylene	520	7.5	0.0005	9	250	1000	70	25,550	3,285	2.5E+00	1.9E+01	--	--	--	--	--	--
1,3-Dinitrobenzene	0.00156	7.5	--	9	250	1000	70	25,550	3,285	--	--	-- [c]	-- [c]	--	--	--	--
2,6-Dinitrotoluene	0.00026	7.5	--	9	250	1000	70	25,550	3,285	--	--	-- [c]	-- [c]	--	--	--	--
Ethylbenzene	0.005	7.5	0.0005	9	250	1000	70	25,550	3,285	2.4E-05	1.8E-04	--	0.286	--	--	0.0006	0.0
HMX	0.0029	7.5	0.0005	9	250	1000	70	25,550	3,285	1.4E-05	1.1E-04	-- [c]	-- [c]	--	--	--	--
Methylene Chloride	3.5	7.5	0.0005	9	250	1000	70	25,550	3,285	1.7E-02	1.3E-01	0.0016	0.857	2.7E-05	1.4E-01	0.1517	0.3
Methyl Ethyl Ketone	0.034	7.5	0.0005	9	250	1000	70	25,550	3,285	1.6E-04	1.2E-03	--	0.286	--	--	0.0042	0.0
Nitrobenzene	0.02	7.5	0.0005	9	250	1000	70	25,550	3,285	9.4E-05	7.3E-04	-- [c]	5.7E-04	--	--	1.2807	2.6
3-Nitrotoluene	0.011	7.5	0.0005	9	250	1000	70	25,550	3,285	5.2E-05	4.0E-04	-- [c]	-- [c]	--	--	--	--
RDX	0.2	7.5	0.0005	9	250	1000	70	25,550	3,285	9.4E-04	7.3E-03	-- [c]	-- [c]	--	--	--	--
Tetrachloroethylene	0.0014	7.5	0.0005	9	250	1000	70	25,550	3,285	6.6E-06	5.1E-05	--	--	--	--	--	--
Tetryl	0.036	7.5	--	9	250	1000	70	25,550	3,285	--	--	-- [c]	-- [c]	--	--	--	--
Toluene	0.029	7.5	0.0005	9	250	1000	70	25,550	3,285	1.4E-04	1.1E-03	--	0.114	--	--	0.0096	0.0
1,1,2-Trichloroethane	0.012	7.5	0.0005	9	250	1000	70	25,550	3,285	5.7E-05	4.4E-04	0.056	--	3.2E-06	1.6E-02	--	--
Trichloroethene	160	7.5	0.0005	9	250	1000	70	25,550	3,285	7.5E-01	5.9E+00	0.00595	--	4.5E-03	2.3E+01	--	--
Trichlorofluoromethane	0.892	7.5	0.0005	9	250	1000	70	25,550	3,285	4.2E-03	3.3E-02	--	0.2	--	--	0.1650	0.3
1,2,4-Trimethylbenzene	0.024	7.5	0.0005	9	250	1000	70	25,550	3,285	1.1E-04	8.8E-04	--	0.035	--	--	0.0251	0.1
1,3,5-Trimethylbenzene	0.016	7.5	0.0005	9	250	1000	70	25,550	3,285	7.5E-05	5.9E-04	--	0.035	--	--	0.0169	0.0
1,3,5-Trinitrobenzene	0.0022	7.5	0.0005	9	250	1000	70	25,550	3,285	1.0E-05	8.1E-05	-- [c]	-- [c]	--	--	--	--
2,4,6-Trinitrotoluene	0.24	7.5	--	9	250	1000	70	25,550	3,285	--	--	--	--	--	--	--	--
Vinyl Chloride	11	7.5	0.0005	9	250	1000	70	25,550	3,285	5.2E-02	4.0E-01	0.3	--	1.5E-02	7.5E+01	--	--
Xylene	0.012	7.5	0.0005	9	250	1000	70	25,550	3,285	5.7E-05	4.4E-04	--	--	--	--	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CW x K x CF x IR x ED x EF) /(BW x AT)

Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i

Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 2.0E-02 Hazard Index: 48.393

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] No air concentration could be calculated for this constituent since it does not volatilize.

[c] Dashes (--) indicate that no toxicity factor was available for this constituent.

INHALATION OF VAPORS FROM GROUNDWATER DURING SHOWERING
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CW (mg/L)	IR (m^3/day)	K (unitless)	ED (years)	EF (days/yr)	CF (L/m^3)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) $(mg/kg\text{-}day)^{-1}$	Inhalation Reference Dose (RfD) (mg/kg-day)	Inhalation Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Inhalation Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																	
Aluminum	67	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	-- [c]	-- [c]	--	--	--	--
Arsenic	0.034	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	15	-- [c]	--	--	--	--
Barium	9.9	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	-- [c]	1.4E-04	--	--	--	--
Beryllium	0.0074	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	8.4	0.0000057	--	--	--	--
Cadmium	0.008	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	6.3	-- [c]	--	--	--	--
Calcium	490	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	--	--	--	--	--	--
Chromium (III)	5.22	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	-- [c]	-- [c]	--	--	--	--
Chromium (VI)	5.22	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	42	0.000029	--	--	--	--
Cobalt	1.1	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	--	--	--	--	--	--
Copper	0.484	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	--	--	--	--	--	--
Iron	180	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	--	--	--	--	--	--
Lead	0.2	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	-- [c]	-- [c]	--	--	--	--
Magnesium	473	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	--	--	--	--	--	--
Manganese	29.8	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	-- [c]	1.4E-05	--	--	--	--
Mercury	0.0016	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	-- [c]	8.6E-05	--	--	--	--
Nickel	1.63	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	--	--	--	--	--	--
Potassium	15	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	--	--	--	--	--	--
Selenium	0.0156	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	-- [c]	-- [c]	--	--	--	--
Silver	0.114	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	-- [c]	-- [c]	--	--	--	--
Sodium	4400	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	--	--	--	--	--	--
Strontium	10.4	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	--	--	--	--	--	--
Thallium	0.012	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	--	--	--	--	--	--
Vanadium	0.146	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	--	--	--	--	--	--
Zinc	37	7.5	-- [b]	25	250	1000	70	25,550	9,125	--	--	-- [c]	-- [c]	--	--	--	--
Organics																	
Acetone	3.92	7.5	0.0005	25	250	1000	70	25,550	9,125	5.1E-02	1.4E-01	--	--	--	--	--	--
Aldrin	0.00004	7.5	--	25	250	1000	70	25,550	9,125	--	--	17.2	--	--	--	--	--
4-Amino-2,6-Dinitrotoluene	0.001	7.5	0.0005	25	250	1000	70	25,550	9,125	1.3E-05	3.7E-05	--	0.000029	--	--	1.2759	2.6
Benzene	0.005	7.5	0.0005	25	250	1000	70	25,550	9,125	6.6E-05	1.8E-04	0.027	--	1.8E-06	3.3E-03	--	--
Bis(2-Ethyl Hexyl) Phthalate	0.026	7.5	--	25	250	1000	70	25,550	9,125	--	--	--	--	--	--	--	--
Bromodichloromethane	0.0084	7.5	0.0005	25	250	1000	70	25,550	9,125	1.1E-04	3.1E-04	--	--	--	--	--	--
Butyl Benzyl Phthalate	0.007	7.5	--	25	250	1000	70	25,550	9,125	--	--	--	--	--	--	--	--
Chloroform	0.12	7.5	0.0005	25	250	1000	70	25,550	9,125	1.6E-03	4.4E-03	0.081	--	1.3E-04	2.4E-01	--	--
1,1-Dichloroethane	0.036	7.5	0.0005	25	250	1000	70	25,550	9,125	4.7E-04	1.3E-03	--	0.143	--	--	0.0091	0.0
1,2-Dichloroethane	0.16	7.5	0.0005	25	250	1000	70	25,550	9,125	2.1E-03	5.9E-03	0.091	--	1.9E-04	3.5E-01	--	--
1,2-Dichloroethene	275	7.5	0.0005	25	250	1000	70	25,550	9,125	3.6E+00	1.0E+01	--	0.22	--	--	45.4545	93.9
1,1-Dichloroethylene	0.74	7.5	0.0005	25	250	1000	70	25,550	9,125	9.7E-03	2.7E-02	0.18	--	1.7E-03	3.1E+00	--	--
cis-1,2-Dichloroethylene	520	7.5	0.0005	25	250	1000	70	25,550	9,125	6.8E+00	1.9E+01	--	--	--	--	--	--
1,3-Dinitrobenzene	0.00156	7.5	--	25	250	1000	70	25,550	9,125	--	--	-- [c]	-- [c]	--	--	--	--
2,6-Dinitrotoluene	0.00026	7.5	--	25	250	1000	70	25,550	9,125	--	--	-- [c]	-- [c]	--	--	--	--
Ethylbenzene	0.005	7.5	0.0005	25	250	1000	70	25,550	9,125	6.6E-05	1.8E-04	--	0.286	--	--	0.0006	0.0
HMX	0.0029	7.5	0.0005	25	250	1000	70	25,550	9,125	3.8E-05	1.1E-04	-- [c]	-- [c]	--	--	--	--
Methylene Chloride	3.5	7.5	0.0005	25	250	1000	70	25,550	9,125	4.6E-02	1.3E-01	0.0016	0.857	7.4E-05	1.3E-01	0.1517	0.3
Methyl Ethyl Ketone	0.034	7.5	0.0005	25	250	1000	70	25,550	9,125	4.5E-04	1.2E-03	--	0.286	--	--	0.0042	0.0
Nitrobenzene	0.02	7.5	0.0005	25	250	1000	70	25,550	9,125	2.6E-04	7.3E-04	-- [c]	5.7E-04	--	--	1.2807	2.6
3-Nitrotoluene	0.011	7.5	0.0005	25	250	1000	70	25,550	9,125	1.4E-04	4.0E-04	-- [c]	-- [c]	--	--	--	--
RDX	0.2	7.5	0.0005	25	250	1000	70	25,550	9,125	2.6E-03	7.3E-03	-- [c]	-- [c]	--	--	--	--
Tetrachloroethylene	0.0014	7.5	0.0005	25	250	1000	70	25,550	9,125	1.8E-05	5.1E-05	--	--	--	--	--	--
Tetryl	0.036	7.5	--	25	250	1000	70	25,550	9,125	--	--	-- [c]	-- [c]	--	--	--	--
Toluene	0.029	7.5	0.0005	25	250	1000	70	25,550	9,125	3.8E-04	1.1E-03	--	0.114	--	--	0.0096	0.0
1,1,2-Trichloroethane	0.012	7.5	0.0005	25	250	1000	70	25,550	9,125	1.6E-04	4.4E-04	0.056	--	9.0E-06	1.6E-02	--	--
Trichloroethene	160	7.5	0.0005	25	250	1000	70	25,550	9,125	2.1E+00	5.9E+00	0.00595	--	1.2E-02	2.2E+01	--	--
Trichlorofluoromethane	0.892	7.5	0.0005	25	250	1000	70	25,550	9,125	1.2E-02	3.3E-02	--	0.2	--	--	0.1650	0.3
1,2,4-Trimethylbenzene	0.024	7.5	0.0005	25	250	1000	70	25,550	9,125	3.1E-04	8.8E-04	--	0.035	--	--	0.0251	0.1
1,3,5-Trimethylbenzene	0.016	7.5	0.0005	25	250	1000	70	25,550	9,125	2.1E-04	5.9E-04	--	0.035	--	--	0.0169	0.0
1,3,5-Trinitrobenzene	0.0022	7.5	0.0005	25	250	1000	70	25,550	9,125	2.9E-05	8.1E-05	-- [c]	-- [c]	--	--	--	--
2,4,6-Trinitrotoluene	0.24	7.5	--	25	250	1000	70	25,550	9,125	--	--	--	--	--	--	--	--
Vinyl Chloride	11	7.5	0.0005	25	250	1000	70	25,550	9,125	1.4E-01	4.0E-01	0.3	--	4.1E-02	7.5E+01	--	--
Xylene	0.012	7.5	0.0005	25	250	1000	70	25,550	9,125	1.6E-04	4.4E-04	--	--	--	--	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CW x K x CF x IR x ED x EF) /(BW x AT)

Total Pathway Risk: 5.5E-02 Hazard Index: 48.393

Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i

Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + _ + CDI_i/RfD_i$

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] No air concentration could be calculated for this constituent since it does not volatilize.

[c] Dashes (--) indicate that no toxicity factor was available for this constituent.

ATTACHMENT 2

DATA UTILIZED IN RISK ASSESSMENT

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	Aluminum (mg/kg)	Antimony (mg/kg)		Arsenic (mg/kg)		Barium (mg/kg)		Beryllium (mg/kg)
16SD01	5/2/1993	0:00	0	Primary		0.86	X+	0.86	X+	295	J	
16SD01	2/21/1995	14:20	0	Primary		0.92	X+	2.21		193.4		
16SD01-A				Avg.		0.89		1.535		244.2		
16WW27	5/17/1997	9:15	0	Primary	7030	3.85	U+JX+	3.2		168		0.68
16WW31	5/29/1997	8:27	0	Primary	4520	3.65	U+JX+	6.7		97.2		1.4
16WW35	6/25/1997	9:50	0	Primary	18100	3.15	U+JX+	4.9		80.2		0.8
16WW35	6/25/1997	9:50	0	Duplicate	117000	3.55	U+JX+	6.1		88.7		0.85
16WW35-A				Avg.	17550	3.35		5.5		84.45		0.825
16WW37	5/31/1997	15:55	0	Primary	5640	3.75	U+JX+	4.4		256		0.88

NATURAL LOGS

16SD01-A		-0.117	0.429	5.498	
16WW27	8.858	1.348	1.163	5.124	-0.386
16WW31	8.416	1.295	1.902	4.577	0.336
16WW35-A	9.773	1.209	1.705	4.436	-0.192
16WW37	8.638	1.322	1.482	5.545	-0.128

STATISTICS

Total No.	4	5	5	5	4
Min.	4520	0.86	0.86	80.2	0.68
Max.	18100	3.85	6.7	295	1.4
Avg.(log transformed)	8.921	1.011	1.336	5.036	-0.093
St. Dev.(log transformed)	0.596	0.633	0.577	0.513	0.306
H	4.187		3.209	2.991	2.801
95th UCL of Mean	37773.30	3.36	11.34	377.96	1.57

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	Cadmium (mg/kg)		Calcium (mg/kg)	Chromium (mg/kg)		Cobalt (mg/kg)	Copper (mg/kg)	
16SD01	5/2/1993	0:00	0	Primary	0.86	X+		28.8				
16SD01	2/21/1995	14:20	0	Primary	0.46	X+		13.8			10.5	
16SD01-A				Avg.	0.66			21.3				
16WW27	5/17/1997	9:15	0	Primary	0.32	X+	1540	7.8		10.3	5.5	
16WW31	5/29/1997	8:27	0	Primary	0.77		143	31.5	J	13.3	1.5	X+
16WW35	6/25/1997	9:50	0	Primary	0.265	X+	370	16.7		3.7	4.6	
16WW35	6/25/1997	9:50	0	Duplicate	0.295	X+	483	18.9		4.0	4.4	
16WW35-A				Avg.	0.28		426.5	17.8		3.85	4.5	
16WW37	5/31/1997	15:55	0	Primary	0.31	X+	15100	12.8		19.8	6.7	

NATURAL LOGS

16SD01-A	-0.416		3.059		
16WW27	-1.139	7.34	2.054	2.332	1.705
16WW31	-0.261	4.963	3.45	2.588	0.405
16WW35-A	-1.273	6.056	2.879	1.348	1.504
16WW37	-1.171	9.622	2.549	2.986	1.902

STATISTICS

Total No.	5	4	5	4	4
Min.	0.265	143	7.8	3.7	1.5
Max.	0.86	15100	31.5	19.8	10.5
Avg.(log transformed)	-0.852	6.995	2.798	2.314	1.379
St. Dev.(log transformed)	0.475	2.003	0.528	0.698	0.669
H	2.873	13.129	3.042	4.789	4.617
95th UCL of Mean	0.94	3.18E+10	42.12	88.90	29.55

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Iron (mg/kg)	Lead (mg/kg)	Magnesium (mg/kg)		Manganese (mg/kg)	Mercury (mg/kg)	
16SD01	5/2/93	0:00	0	Primary		29				0.085	X+
16SD01	2/21/95	14:20	0	Primary		18.4				0.09	X+
16SD01-A				Avg.		23.7				0.0875	
16WW27	5/17/97	9:15	0	Primary	10200	12.1	1030		1270	0.051	
16WW31	5/29/97	8:27	0	Primary	19400	14.3	302.5	X+	893	0.023	
16WW35	6/25/97	9:50	0	Primary	17700	7.9	1020		40.9	0.02	
16WW35	6/25/97	9:50	0	Duplicate	21500	9.2	992		53.4	0.03	
16WW35-A				Avg.	19600	8.55	1006		47.15	0.025	
16WW37	5/31/97	15:55	0	Primary	10100	21.8	688		1060	0.062	

NATURAL LOGS

16SD01-A		3.165			-2.436
16WW27	9.23	2.493	6.937	7.147	-2.976
16WW31	9.873	2.66	5.712	6.795	-3.772
16WW35-A	9.883	2.146	6.914	3.853	-3.689
16WW37	9.22	3.082	6.534	6.966	-2.781

STATISTICS

Total No.	4	5	4	4	5
Min.	10100	7.9	302.5	40.9	0.02
Max.	21500	29	1030	1270	0.09
Avg.(log transformed)	9.552	2.709	6.524	6.19	-3.131
St. Dev.(log transformed)	0.377	0.422	0.572	1.565	0.581
H	3.038	2.716	4.055	10.294	3.222
95th UCL of Mean	29270.01	29.11	3061.72	1.82E+07	0.13

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Nickel (mg/kg)		Potassium (mg/kg)		Selenium (mg/kg)		Silver (mg/kg)		Sodium (mg/kg)	
16SD01	5/2/93	0:00	0	Primary	16.8	J			0.86	X+U+J	0.86	X+		
16SD01	2/21/95	14:20	0	Primary	16.6				0.92	X+	0.92	X+		
16SD01-A				Avg.	16.7				0.89		0.89			
16WW27	5/17/97	9:15	0	Primary	9		570		0.32	X+	0.65	X+	68.5	
16WW31	5/29/97	8:27	0	Primary	6.1		302.5	X+	0.21	X+	0.6	X+	21.1	
16WW35	6/25/97	9:50	0	Primary	10.4		1070		1		0.55	U+JX+	63.6	
16WW35	6/25/97	9:50	0	Duplicate	9.7		974		1.4		0.6	U+JX+	24.2	U+
16WW35-A				Avg.	10.05		1022		1.2		0.575		43.875	
16WW37	5/31/97	15:55	0	Primary	8.2		846		0.31	X+	0.55		42.8	

NATURAL LOGS

16SD01-A	2.815		-0.117	-0.117	
16WW27	2.197	6.346	-1.139	-0.431	4.227
16WW31	1.808	5.712	-1.561	-0.511	3.049
16WW35-A	2.308	6.93	0.182	-0.553	3.781
16WW37	2.104	6.741	-1.171	-0.598	3.757

STATISTICS

Total No.	5	4	5	5	4
Min.	6.1	302.5	0.21	0.55	21.1
Max.	16.8	1070	1.4	0.92	68.5
Avg.(log transformed)	2.246	6.432	-0.761	-0.442	3.704
St. Dev.(log transformed)	0.368	0.538	0.751	0.192	0.487
H	2.571	3.867	3.866	2.185	3.595
95th UCL of Mean	16.23	2387.18	2.65	0.81	125.64

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Strontium (mg/kg)	Thallium (mg/kg)		Vanadium (mg/kg)	Zinc (mg/kg)
16SD01	5/2/93	0:00	0	Primary		0.86	X+		
16SD01	2/21/95	14:20	0	Primary		0.46	X+		55.6
16SD01-A				Avg.		0.66			
16WW27	5/17/97	9:15	0	Primary	34.1	0.65	X+	16	27.3
16WW31	5/29/97	8:27	0	Primary	2.4	0.6	X+	33.5	13.3
16WW35	6/25/97	9:50	0	Primary	11.2	0.55	X+	30.6	22.2
16WW35	6/25/97	9:50	0	Duplicate	12.7	0.6	X+	33	20.9
16WW35-A				Avg.	11.95	0.575		31.8	21.55
16WW37	5/31/97	15:55	0	Primary	62.7	0.6	X+	22.5	38.9

NATURAL LOGS

16SD01-A		-0.416		
16WW27	3.529	-0.431	2.773	3.307
16WW31	0.875	-0.511	3.512	2.588
16WW35-A	2.481	-0.553	3.459	3.07
16WW37	4.138	-0.511	3.114	3.661

STATISTICS

Total No.	4	5	4	4
Min.	2.4	0.46	16	13.3
Max.	62.7	0.86	33.5	55.6
Avg.(log transformed)	2.756	-0.484	3.215	3.157
St. Dev.(log transformed)	1.428	0.058	0.343	0.45
H	9.411		2.948	3.416
95th UCL of Mean	102185.92	0.62	47.35	63.17

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,1,1,2-Tetra chloroethane (ug/kg)		1,1,1-trichloro ethane (ug/kg)		1,1,2,2-Tetra chloroethane (ug/kg)		1,1,2-Trichloro ethane (ug/kg)		1,1-Dichloro ethane (ug/kg)		1,1-Dichloro ethene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary			4.3	X+	4.3	X+	4.3	X+	4.3	X+	4.3	X+
16SD01	2/21/95	14:20	0	Primary			4.5	X+	4.5	X+	4.5	X+	4.5	X+	4.5	X+
16SD01-A				Avg.			4.4		4.4		4.4		4.4		4.4	
16WW27	5/17/97	9:15	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35-A				Avg.	2.5		2.5		2.5		2.5		2.5		2.5	
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+

NATURAL LOGS

16SD01-A	1.482	1.482	1.482	1.482	1.482
16WW27	1.099	1.099	1.099	1.099	1.099	1.099
16WW31	1.099	1.099	1.099	1.099	1.099	1.099
16WW35-A	0.916	0.916	0.916	0.916	0.916	0.916
16WW37	1.099	1.099	1.099	1.099	1.099	1.099

STATISTICS

Total No.	4	5	5	5	5	5
Min.	2.5	2.5	2.5	2.5	2.5	2.5
Max.	3	4.5	4.5	4.5	4.5	4.5
Avg.(log transformed)	1.053	1.139	1.139	1.139	1.139	1.139
St. Dev.(log transformed)	0.091	0.207	0.207	0.207	0.207	0.207
H						
95th UCL of Mean	2.88	3.19	3.19	3.19	3.19	3.19

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,1-Dichloro propene (ug/kg)		1,2,3-Trichloro benzene (ug/kg)		1,2,3-Trichloro propane (ug/kg)		1,2,4-Trichloro benzene (ug/kg)		1,2-Dichloro ethane (ug/kg)		DCE12TOT (ug/kg)	
16SD01	5/2/93	0:00	0	Primary					4.3	X+			4.3	X+		
16SD01	2/21/95	14:20	0	Primary					4.5	X+			4.5	X+	4.5	X+
16SD01-A				Avg.					4.4				4.4			
16WW27	5/17/97	9:15	0	Primary	3	X+	3	X+	9.5	X+	3	X+	3	X+		
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	9	X+	3	X+	3	X+		
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	8	X+	2.5	X+	2.5	X+		
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	2.5	X+	7.5	X+	2.5	X+	2.5	X+		
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	2.5	X+	7.5	X+	2.5	X+	2.5	X+		
16WW35-A				Avg.	2.5		2.5		7.666667		2.5		2.5			
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	9.5	X+	3	X+	3	X+		

NATURAL LOGS

16SD01-A				1.482		1.482	
16WW27	1.099	1.099	2.251	1.099	1.099		
16WW31	1.099	1.099	2.197	1.099	1.099	1.504	
16WW35-A	0.916	0.916	2.037	0.916	0.916		
16WW37	1.099	1.099	2.251	1.099	1.099		

STATISTICS

Total No.	4	4	5	4	5	1
Min.	2.5	2.5	4.3	2.5	2.5	4.5
Max.	3	3	9.5	3	4.5	4.5
Avg.(log transformed)	1.053	1.053	2.044	1.053	1.139	1.504
St. Dev.(log transformed)	0.091	0.091	0.326	0.091	0.207	#DIV/0!
H						
95th UCL of Mean	2.88	2.88	8.14	2.88	3.19	#DIV/0!

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,2-Dichloropropane (ug/kg)		1,3-Dichloropropane (ug/kg)		1,4-Dichloro-2-butene (ug/kg)		1,4-Dioxane (ug/kg)		2,2-Dichloropropane (ug/kg)		2-Hexanone (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	4.3	X+			8.5	X+					8.5	X+
16SD01	2/21/95	14:20	0	Primary	4.5	X+			9	X+					9	X+
16SD01-A				Avg.	4.4				8.75						8.75	
16WW27	5/17/97	9:15	0	Primary	3	X+	3	X+	3	X+	650	X+	9.5	X+	13	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	3	X+	600	X+	93.5	X+	12	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	2.5	X+	550	X+	8	X+	10.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	500	X+	7.5	X+	10	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	500	X+	7.5	X+	10	X+
16WW35-A				Avg.	2.5		2.5		2.5		516.6667		7.666667		10.16667	
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	3	X+	600	X+	9.5	X+	12.5	X+

NATURAL LOGS					
16SD01-A	1.482		2.169		2.169
16WW27	1.099	1.099	1.099	6.477	2.251	2.565
16WW31	1.099	1.099	1.099	6.397	4.538	2.485
16WW35-A	0.916	0.916	0.916	6.247	2.037	2.319
16WW37	1.099	1.099	1.099	6.397	2.251	2.526

STATISTICS						
Total No.	5	4	5	4	4	5
Min.	2.5	2.5	2.5	500	7.5	8.5
Max.	4.5	3	9	650	93.5	13
Avg.(log transformed)	1.139	1.053	1.276	6.38	2.769	2.413
St. Dev.(log transformed)	0.207	0.091	0.505	0.096	1.183	0.165
H						
95th UCL of Mean	3.19	2.88	4.07	592.65	32.10	11.32

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Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Acetone (ug/kg)		Acrolein (ug/kg)		Acrylonitrile (ug/kg)		Benzene (ug/kg)		1,2,4-Trimethyl benzene (ug/kg)		1,3,5-Trimethyl benzene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	8.5	X+	8.5	X+	8.5	X+	4.3	X+				
16SD01	2/21/95	14:20	0	Primary	103		46	X+	46	X+	4.5	X+				
16SD01-A				Avg.	55.75		27.25		27.25		4.4					
16WW27	5/17/97	9:15	0	Primary	13	X+	65	X+	65	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	8:27	0	Primary	12	X+	60	X+	60	X+	3	X+	3	X+	3	X+
16WW35	6/25/97	9:50	0	Primary	10.5	X+	55	X+	55	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	20		50	X+	50	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	10	X+	50	X+	50	X+	2.5	X+	2.5	X+	2.5	X+
16WW35-A				Avg.	13.5		51.66667		51.66667		2.5		2.5		2.5	
16WW37	5/31/97	15:55	0	Primary	12.5	X+	60	X+	60	X+	3	X+	3	X+	3	X+

NATURAL LOGS

16SD01-A	4.021	3.305	3.305	1.482	
16WW27	2.565	4.174	4.174	1.099	1.099
16WW31	2.485	4.094	4.094	1.099	1.099
16WW35-A	2.603	3.945	3.945	0.916	0.916
16WW37	2.526	4.094	4.094	1.099	1.099

STATISTICS

Total No.	5	5	5	5	4	4
Min.	8.5	8.5	8.5	2.5	2.5	2.5
Max.	103	65	65	4.5	3	3
Avg.(log transformed)	2.84	3.922	3.922	1.139	1.053	1.053
St. Dev.(log transformed)	0.662	0.355	0.355	0.207	0.091	0.091
H	3.52					
95th UCL of Mean	68.32	53.79	53.79	3.19	2.88	2.88

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Isopropyl benzene (ug/kg)		Bromobenzene (ug/kg)		Bromodichloro methane (ug/kg)		Bromoform (ug/kg)		Carbon disulfide (ug/kg)		Carbon tetrachloride (ug/kg)	
16SD01	5/2/93	0:00	0	Primary					4.3	X+			4.3	X+	4.3	X+
16SD01	2/21/95	14:20	0	Primary					4.5	X+	4.5	X+	4.5	X+	4.5	X+
16SD01-A				Avg.					4.4				4.4		4.4	
16WW27	5/17/97	9:15	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	6.5	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	6	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	5.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	5	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	5	X+
16WW35-A				Avg.	2.5		2.5		2.5		2.5		2.5		5.166667	
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	6	X+

NATURAL LOGS

16SD01-A			1.482		1.482	1.482
16WW27	1.099	1.099	1.099	1.099	1.099	1.872
16WW31	1.099	1.099	1.099	1.099	1.099	1.792
16WW35-A	0.916	0.916	0.916	0.916	0.916	1.642
16WW37	1.099	1.099	1.099	1.099	1.099	1.792

STATISTICS

Total No.	4	4	5	4	5	5
Min.	2.5	2.5	2.5	2.5	2.5	4.3
Max.	3	3	4.5	4.5	4.5	6.5
Avg.(log transformed)	1.053	1.053	1.139	1.053	1.139	1.716
St. Dev.(log transformed)	0.091	0.091	0.207	0.091	0.207	0.155
H						
95th UCL of Mean	2.88	2.88	3.19	2.88	3.19	5.63

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Chlorobenzene (ug/kg)		Bromochloro methane (ug/kg)		Chloroethane (ug/kg)		Chloroform (ug/kg)		cis-1,2-Dichloroethene (ug/kg)		cis-1,3-Dichloropropene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	4.3	X+			8.5	X+	4.3	X+	4.3	X+		
16SD01	2/21/95	14:20	0	Primary	4.5	X+			9	X+	4.5	X+			4.5	X+
16SD01-A				Avg.	4.4				8.75		4.4		4.3			
16WW27	5/17/97	9:15	0	Primary	3	X+	3	X+	6.5	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	6	X+	3	X+	3	X+	3	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	5.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	2.5	X+	5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	2.5	X+	5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35-A				Avg.	2.5		2.5		5.166667		2.5		2.5		2.5	
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	6	X+	3	X+	3	X+	3	X+

NATURAL LOGS

16SD01-A	1.482		2.169	1.482	1.459	
16WW27	1.099	1.099	1.872	1.099	1.099	1.099
16WW31	1.099	1.099	1.792	1.099	1.099	1.099
16WW35-A	0.916	0.916	1.642	0.916	0.916	0.916
16WW37	1.099	1.099	1.792	1.099	1.099	1.099

STATISTICS

Total No.	5	4	5	5	5	4
Min.	2.5	2.5	5	2.5	2.5	2.5
Max.	4.5	3	9	4.5	4.3	4.5
Avg.(log transformed)	1.139	1.053	1.853	1.139	1.134	1.053
St. Dev.(log transformed)	0.207	0.091	0.195	0.207	0.198	0.091
H						
95th UCL of Mean	3.19	2.88	6.50	3.19	3.17	2.88

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,2-Dibromo-3-chloropropane (ug/kg)		Dibromochloro methane (ug/kg)		Dichloro difluoromethane (ug/kg)		1,2-Dibromoetha (ug/kg)		CEVETH (ug/kg)		trans-1,2-Dichloroethene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary			4.3	X+	4.3	X+					4.3	X+
16SD01	2/21/95	14:20	0	Primary			4.5	X+	4.5	X+			9	X+		
16SD01-A				Avg.			4.4		4.4						4.3	
16WW27	5/17/97	9:15	0	Primary	6.5	X+	3	X+	9.5	X+	3	X+			3	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	9	X+	3	X+			3	X+
16WW35	6/25/97	9:50	0	Primary	5.5	X+	2.5	X+	8	X+	2.5	X+			2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	5	X+	2.5	X+	7.5	X+	2.5	X+			2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	5	X+	2.5	X+	7.5	X+	2.5	X+			2.5	X+
16WW35-A				Avg.	5.166667		2.5		7.666667		2.5				2.5	
16WW37	5/31/97	15:55	0	Primary	6	X+	3	X+	9.5	X+	3	X+			3	X+

NATURAL LOGS

16SD01-A		1.482	1.482		1.459
16WW27	1.872	1.099	2.251	1.099	2.197	1.099
16WW31	1.099	1.099	2.197	1.099		1.099
16WW35-A	1.642	0.916	2.037	0.916		0.916
16WW37	1.792	1.099	2.251	1.099		1.099

STATISTICS

Total No.	4	5	5	4	1	5
Min.	3	2.5	4.3	2.5	9	2.5
Max.	6.5	4.5	9.5	3	9	4.3
Avg.(log transformed)	1.601	1.139	2.044	1.053	2.197	1.134
St. Dev.(log transformed)	0.348	0.207	0.326	0.091	#DIV/0!	0.198
H						
95th UCL of Mean	5.27	3.19	8.14	2.88	#DIV/0!	3.17

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Propionitrile (ug/kg)		Ethyl methacrylate (ug/kg)		Ethylbenzene (ug/kg)		Hexachloro butadiene (ug/kg)		Isobutyl alcohol (ug/kg)		1,3-Dichloro benzene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary			4.3	X+	4.3	X+						
16SD01	2/21/95	14:20	0	Primary			4.5	X+	4.5	X+						
16SD01-A				Avg.			4.4		4.4							
16WW27	5/17/97	9:15	0	Primary	65	X+	3	X+	3	X+	3	X+	650	X+	3	X+
16WW31	5/29/97	8:27	0	Primary	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+
16WW35	6/25/97	9:50	0	Primary	55	X+	2.5	X+	2.5	X+	2.5	X+	550	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	50	X+	2.5	X+	2.5	X+	2.5	X+	500	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	50	X+	2.5	X+	2.5	X+	2.5	X+	500	X+	2.5	X+
16WW35-A				Avg.	51.66667		2.5		2.5		2.5		516.6667		2.5	
16WW37	5/31/97	15:55	0	Primary	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+

NATURAL LOGS

16SD01-A		1.482	1.482		
16WW27	4.174	1.099	1.099	1.099	6.477	1.099
16WW31	4.094	1.099	1.099	1.099	6.397	1.099
16WW35-A	3.945	0.916	0.916	0.916	6.247	0.916
16WW37	4.094	1.099	1.099	1.099	6.397	1.099

STATISTICS

Total No.	4	5	5	4	4	4
Min.	50	2.5	2.5	2.5	500	2.5
Max.	65	4.5	4.5	3	650	3
Avg.(log transformed)	4.077	1.139	1.139	1.053	6.38	1.053
St. Dev.(log transformed)	0.096	0.207	0.207	0.091	0.096	0.091
H						
95th UCL of Mean	59.24	3.19	3.19	2.88	592.65	2.88

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Methacrylonitrile (ug/kg)		Bromomethane (ug/kg)		Chloromethane (ug/kg)		2-Butanone (ug/kg)		Iodomethane (ug/kg)		4-Methyl-2-pentanone (ug/kg)	
16SD01	5/2/93	0:00	0	Primary			8.5	X+	8.5	X+	8.5	X+	8.5	X+	8.5	X+
16SD01	2/21/95	14:20	0	Primary			9	X+	9	X+	9	X+	9	X+	9	X+
16SD01-A				Avg.			8.75		8.75		8.75		8.75		8.75	
16WW27	5/17/97	9:15	0	Primary	65	X+	6.5	X+	6.5	X+	13	X+	3	X+	13	X+
16WW31	5/29/97	8:27	0	Primary	60	X+	6	X+	6	X+	12	X+	3	X+	12	X+
16WW35	6/25/97	9:50	0	Primary	55	X+	5.5	X+	5.5	X+	10.5	X+	2.5	X+	10.5	X+
16WW35	6/25/97	9:50	0	Duplicate	50	X+	5	X+	5	X+	10	X+	2.5	X+	10	X+
16WW35	6/25/97	0:00	0	Duplicate	50	X+	5	X+	5	X+	10	X+	2.5	X+	10	X+
16WW35-A				Avg.	51.66667		5.166667		5.166667		10.16667		2.5		10.16667	
16WW37	5/31/97	15:55	0	Primary	60	X+	6	X+	6	X+	12.5	X+	3	X+	12.5	X+
NATURAL LOGS																
16SD01-A							2.169		2.169		2.169		2.169		2.169	
16WW27					4.174		1.872		1.872		2.565		1.099		2.565	
16WW31					4.094		1.792		1.792		2.485		1.099		2.485	
16WW35-A					3.945		1.642		1.642		2.319		0.916		2.319	
16WW37					4.094		1.792		1.792		2.526		1.099		2.526	
STATISTICS																
Total No.					4		5		5		5		5		5	
Min.					50		5		5		8.5		2.5		8.5	
Max.					65		9		9		13		9		13	
Avg.(log transformed)					4.077		1.853		1.853		2.413		1.276		2.413	
St. Dev.(log transformed)					0.096		0.195		0.195		0.165		0.505		0.165	
H																
95th UCL of Mean					59.24		6.50		6.50		11.32		4.07		11.32	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Methyl methacrylate (ug/kg)		Dibromomethane (ug/kg)		Methylene chloride (ug/kg)		Naphthalene (ug/kg)		n-Butylbenzene (ug/kg)		1-Propanamine, N-nitroso-n-pro (ug/kg)	
16SD01	5/2/93	0:00	0	Primary			4.3	X+	4.3	X+						
16SD01	2/21/95	14:20	0	Primary			4.5	X+	9	X+						
16SD01-A				Avg.			4.4		6.65							
16WW27	5/17/97	9:15	0	Primary	32	X+	6.5	X+	2	U+	3	X+	3	X+	3	X+
16WW31	5/29/97	8:27	0	Primary	30	X+	6	X+	5		3	X+	3	X+	3	X+
16WW35	6/25/97	9:50	0	Primary	26.5	X+	5.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	25	X+	5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	25	X+	5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35-A				Avg.	25.5		5.166667		2.5		2.5		2.5		2.5	
16WW37	5/31/97	15:55	0	Primary	31	X+	6	X+	3	X+	3	X+	3	X+	3	X+
NATURAL LOGS																
16SD01-A							1.482		1.895							
16WW27					3.466		1.872		0.405		1.099		1.099		1.099	
16WW31					3.401		1.792		1.609		1.099		1.099		1.099	
16WW35-A					3.239		1.642		0.916		0.916		0.916		0.916	
16WW37					3.434		1.792		1.099		1.099		1.099		1.099	
STATISTICS																
Total No.					4		5		5		4		4		4	
Min.					25		4.3		1.5		2.5		2.5		2.5	
Max.					32		6.5		9		3		3		3	
Avg.(log transformed)					3.385		1.716		1.185		1.053		1.053		1.053	
St. Dev.(log transformed)					0.101		0.155		0.586		0.091		0.091		0.091	
H									3.239							
95th UCL of Mean					29.67		5.63		10.03		2.88		2.88		2.88	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	n-Propyl benzene (ug/kg)		2-Chlortoluene (ug/kg)		1,2-Dichloro benzene (ug/kg)		o-Xylene (ug/kg)		4-Chlorotoluene (ug/kg)		p-Isopropyl toluene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary												
16SD01	2/21/95	14:20	0	Primary												
16SD01-A				Avg.												
16WW27	5/17/97	9:15	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	6	X+	3	X+	3	X+	3	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35-A				Avg.	2.5		2.5		2.5		2.5		2.5		2.5	
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
NATURAL LOGS																
16SD01-A																
16WW27					1.099		1.099		1.099		1.099		1.099		1.099	
16WW31					1.099		1.099		1.792		1.099		1.099		1.099	
16WW35-A					0.916		0.916		0.916		0.916		0.916		0.916	
16WW37					1.099		1.099		1.099		1.099		1.099		1.099	
STATISTICS																
Total No.					4		4		4		4		4		4	
Min.					2.5		2.5		2.5		2.5		2.5		2.5	
Max.					3		3		6		3		3		3	
Avg.(log transformed)					1.053		1.053		1.227		1.053		1.053		1.053	
St. Dev.(log transformed)					0.091		0.091		0.387		0.091		0.091		0.091	
H																
95th UCL of Mean					2.88		2.88		3.68		2.88		2.88		2.88	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,4-Dichloro benzene (ug/kg)		Pentachloro ethane (ug/kg)		sec-Butyl benzene (ug/kg)		Styrene (ug/kg)		tert-Butyl benzene (ug/kg)		Tetrachloro ethene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary							4.3	X+			4.3	X+
16SD01	2/21/95	14:20	0	Primary							4.5	X+			4.5	X+
16SD01-A				Avg.							4.4				4.4	
16WW27	5/17/97	9:15	0	Primary	3	X+	6.5	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	6	X+	3	X+	3	X+	3	X+	3	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	5.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35-A				Avg.	2.5		5.166667		2.5		2.5		2.5		2.5	
16WW37	5/31/97	15:55	0	Primary	3	X+	6	X+	3	X+	3	X+	3	X+	3	X+

NATURAL LOGS

16SD01-A					
16WW27	1.099	1.872	1.099	1.482	1.482
16WW31	1.099	1.792	1.099	1.099	1.099
16WW35-A	0.916	1.642	0.916	1.099	1.099
16WW37	1.099	1.792	1.099	0.916	0.916
				1.099	1.099

STATISTICS

Total No.	4	4	4	5	4	5
Min.	2.5	5	2.5	2.5	2.5	2.5
Max.	3	6.5	3	4.5	3	4.5
Avg.(log transformed)	1.053	1.775	1.053	1.139	1.053	1.139
St. Dev.(log transformed)	0.091	0.096	0.091	0.207	0.091	0.207
H						
95th UCL of Mean	2.88	5.93	2.88	3.19	2.88	3.19

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Toluene (ug/kg)		Trans-1,3-Dichloropropene (ug/kg)		Trichloroethene (ug/kg)		Trichloro fluoromethane (ug/kg)		Vinyl Acetate (ug/kg)		Vinyl chloride (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	4.3	X+	4.3	X+	4.3	X+	4.3	X+	4.3	X+	8.5	X+
16SD01	2/21/95	14:20	0	Primary	4.5	X+	4.5	X+	4.5	X+	4.5	X+	9	X+	9	X+
16SD01-A				Avg.	4.4		4.4		4.4		4.4		6.65		8.75	
16WW27	5/17/97	9:15	0	Primary	3	X+	3	X+	6.5	X+	6.5	X+	13	X+	6.5	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	6	X+	6	X+	12	X+	6	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	5.5	X+	5.5	X+	10.5	X+	5.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	2.5	X+	5	X+	5	X+	10	X+	5	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	2.5	X+	5	X+	5	X+	10	X+	5	X+
16WW35-A				Avg.	2.5		2.5		5.166667		5.166667		10.16667		5.166667	
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	6	X+	6	X+	12.5	X+	6	X+

NATURAL LOGS

16SD01-A	1.482	1.482	1.482	1.482	1.895	2.169
16WW27	1.099	1.099	1.872	1.872	2.565	1.872
16WW31	1.099	1.099	1.792	1.792	2.485	1.792
16WW35-A	0.916	0.916	1.642	1.642	2.319	1.642
16WW37	1.099	1.099	1.792	1.792	2.526	1.792

STATISTICS

Total No.	5	5	5	5	5	5
Min.	2.5	2.5	4.3	4.3	4.3	5
Max.	4.5	4.5	6.5	6.5	13	9
Avg.(log transformed)	1.139	1.139	1.716	1.716	2.358	1.853
St. Dev.(log transformed)	0.207	0.207	0.155	0.155	0.275	0.195
H						
95th UCL of Mean	3.19	3.19	5.63	5.63	10.98	6.50

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-0.5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Xylene (total) (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	4.3	X+
16SD01	2/21/95	14:20	0	Primary	4.5	X+
16SD01-A				Avg.	4.4	
16WW27	5/17/97	9:15	0	Primary	3	X+
16WW31	5/29/97	8:27	0	Primary	3	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+
16WW35-A				Avg.	2.5	
16WW37	5/31/97	15:55	0	Primary	3	X+

NATURAL LOGS

16SD01-A	1.482
16WW27	1.099
16WW31	1.099
16WW35-A	0.916
16WW37	1.099

STATISTICS

Total No.	5
Min.	2.5
Max.	4.5
Avg.(log transformed)	1.139
St. Dev.(log transformed)	0.207
H	
95th UCL of Mean	3.19

Data Qualifier Notations

X+ = Non-Detect Result, one-half of the detection limit value presented.
U+ = Considered Non-Detect based on blank comparison, one-half of the detection limit value is presented.
J = Estimated laboratory result.

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(0-5 feet)

SITE	DATE	TIME	DEPTH (	RESULT TYPE	Aluminum (mg/kg)	Antimony (mg/kg)		Arsenic (mg/kg)		Barium (mg/kg)		Beryllium (mg/kg)	Cadmium (mg/kg)		Calcium (mg/kg)	Chromium (mg/kg)		Cobalt (mg/kg)		Copper (mg/kg)	
16SD01	5/2/93	0:00	0	Primary		0.86	X+	0.86	X+	295	J		0.86	X+		28.8					
16SD01	2/21/95	14:20	0	Primary		0.92	X+	2.21		193.4			0.46	X+		13.8				10.5	
16SD01-A				Avg.		0.89		1.535		244.2			0.66			21.3					
16SD02	4/14/93	0:00	0	Primary		0.635	X+	7.17		52.5			0.635	X+		12.7					
16SD02	2/28/95	10:00	0	Primary		0.69	X+	2.49		53.8			0.345	X+		8.74				6.1	
16SD02-A				Avg.		0.6625		4.83		53.15			0.49			10.72					
16SD03	4/14/93	0:00	0	Primary		0.65	X+	3.85		56.4			0.65	X+		40.9					
16SD03	2/20/95	16:05	0	Primary		0.78	X+	2.02		58.8			0.39	X+		8.16				4	
16SD03-A				Avg.		0.715		2.935		57.6			0.52			24.53					
16SD06	2/20/95	16:35	0	Primary		0.73	X+	2.63		64.5			0.365	X+		13.5				8.5	
16SD07	2/21/95	14:05	0	Primary		0.795	X+	4.13		94.9			0.395	X+		10.2				6.8	
16SD08	2/21/95	9:30	0	Primary		0.655	X+	3.01		383.5			0.325	X+		11.1				6	
16SD09	2/21/95	10:55	0	Primary		0.665	X+	4.8		209.3			0.335	X+		15.7				10.3	
16SD10	2/21/95	17:10	0	Primary		0.65	X+	5.59		72.3			0.325	X+		18.9				3.4	
16SD11	2/28/95	11:15	0	Primary		0.65	X+	3.63		86.1			0.325	X+		22.6				6.1	
16WW01	5/4/93	0:00	0	Primary		0.595	X+	3.34		73.7			0.595	X+		9.57					
16WW01	5/4/93	0:00	0	Duplicate 1		0.58	U+J	3.22	J	84			0.58	X+		15					
16WW01-A				Avg.		0.5875		3.28		78.85			0.5875			12.285					
16WW02	5/12/93	0:00	0	Primary		0.58	U+J	1.43	J	46.7			0.58	X+		17.1					
16WW02	5/12/93	0:00	0	Duplicate 1		0.59	X+	1.72		55			0.59	X+		11.7					
16WW02-A				Avg.		0.585		1.575		50.85			0.585			14.4					
16WW03	5/4/93	0:00	0	Primary		0.625	U+J	14.2	J	94.7			0.625	X+		32.9					
16WW04	5/12/93	0:00	0	Primary		0.6	U+J	6.97	J	111			0.6	X+		16.8					
16WW04	5/12/93	0:00	0	Duplicate 1		0.635	X+	14.3		104			0.635	X+		20.8					
16WW04-A				Avg.		0.6175		10.635		107.5			0.6175			18.8					
16WW05	5/4/93	0:00	0	Primary		0.675	U+J	14.4	J	146			0.675	X+		36.7					
16WW06	5/5/93	0:00	0	Primary		0.65	X+	5.5		216			0.65	X+		23.7					
16WW27	5/17/97	9:15	0	Primary	7030	3.85	U+J	3.2		168		0.68	0.32	X+	1540	7.8		10.3		5.5	
16WW27	5/17/97	10:17	1	Primary	7070	3.6	U+J	2.5		58.8		0.41	0.51		118	8.1		3	X+	4.3	
16WW27-A				Avg.	7050	3.725		2.85		113.4		0.545	0.415		829	7.95		6.65		4.9	
16WW31	5/29/97	8:27	0	Primary	4520	3.65	U+J	6.7		97.2		1.4	0.77		143	31.5	J	13.3		1.5	X+
16WW31	5/29/97	8:55	1	Primary	9740	0.48		6.6		62.3		0.8	0.56		255	22.6	J	3.05	X+	1.55	X+
16WW31-A				Avg.	7130	2.065		6.65		79.75		1.1	0.665		199	27.05		8.175		1.525	
16WW35	6/25/97	9:50	0	Primary	18100	3.15	U+J	4.9		80.2		0.8	0.265	X+	370	16.7		3.7		4.6	
16WW35	6/25/97	9:50	0	Duplicate 1	17000	3.55	U+J	6.1		88.7		0.85	0.295	X+	483	18.9		4.0		4.4	
16WW35	6/25/97	10:05	1	Primary	10600	3.7	U+J	3.4		47.4		0.38	0.31	X+	295	10.4		2.8		4.9	
16WW35-A				Avg.	15233.3	3.46667		4.8		72.1		0.67667	0.29		382.667	15.3333		3.5		4.63333	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(0-5 feet)

SITE	DATE	TIME	DEPTH (	RESULT TYPE	Aluminum (mg/kg)	Antimony (mg/kg)		Arsenic (mg/kg)	Barium (mg/kg)	Beryllium (mg/kg)	Cadmium (mg/kg)		Calcium (mg/kg)	Chromium (mg/kg)	Cobalt (mg/kg)	Copper (mg/kg)
16WW37	5/31/97	15:55	0	Primary	5640	3.75	U+J	4.4	256	0.88	0.31	X+	15100	12.8	19.8	6.7
16WW37	5/31/97	16:10	1	Primary	21500	3.8	U+J	7.5	208	1.2	0.86		3090	23.9	6.6	6.7
16WW37-A				Avg.	13570	3.775		5.95	232	1.04	0.585		9095	18.35	13.2	6.7
NATUAL LOGS																
16SD01-A						-0.117		0.429	5.498		-0.416			3.059		
16SD02-A						-0.412		1.575	3.973		-0.713			2.372		
16SD03-A						-0.335		1.077	4.054		-0.654			3.2		
16SD06						-0.315		0.967	4.167		-1.008			2.603		2.14
16SD07						-0.229		1.418	4.553		-0.929			2.322		1.917
16SD08						-0.423		1.102	5.949		-1.124			2.407		1.792
16SD09						-0.408		1.569	5.344		-1.094			2.754		2.332
16SD10						-0.431		1.721	4.281		-1.124			2.939		1.224
16SD11						-0.431		1.289	4.456		-1.124			3.118		1.808
16WW01-A						-0.532		1.188	4.368		-0.532			2.508		
16WW02-A						-0.536		0.454	3.929		-0.536			2.667		
16WW03						-0.47		2.653	4.551		-0.47			3.493		
16WW04-A						-0.482		2.364	4.677		-0.482			2.934		
16WW05						-0.393		2.667	4.984		-0.393			3.603		
16WW06						-0.431		1.705	5.375		-0.431			3.165		
16WW27-A					8.861	1.315		1.047	4.731	-0.607	-0.879		6.72	2.073	1.895	1.589
16WW31-A					8.872	0.725		1.895	4.379	0.095	-0.408		5.293	3.298	2.101	0.422
16WW35-A					9.631	1.243		1.569	4.278	-0.391	-1.238		5.947	2.73	1.253	1.533
16WW37-A					9.516	1.328		1.783	5.447	0.039	-0.536		9.115	2.91	2.58	1.902
STATISTICS																
Total No.					4	19		19	19	4	19		4	19	4	10
Min.					4520	0.48		0.86	46.7	0.38	0.265		118	7.8	2.8	1.5
Max.					21500	3.85		14.4	383.5	1.4	0.86		15100	40.9	19.8	10.5
Avg.(log transformed)					9.22	-0.07		1.499	4.684	-0.216	-0.742		6.769	2.85	1.957	1.666
St. Dev. (log transformed)					0.411	0.667		0.623	0.587	0.339	0.303		1.669	0.414	0.55	0.536
H					3.228	2.184		2.136	2.097	2.932	1.847		10.966	1.933	3.934	2.273
95th UCL of Mean					23634	1.64		7.44	171.82	1.51	0.57		1.4E+08	22.74	28.72	9.17

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(0-5 feet)

SITE	DATE	TIME	DEPTH (	RESULT TYPE	Iron (mg/kg)	Lead (mg/kg)		Magnesium (mg/kg)		Manganese (mg/kg)	Mercury (mg/kg)		Nickel (mg/kg)		Potassium (mg/kg)		Selenium (mg/kg)		Silver (mg/kg)		Sodium (mg/kg)	
16SD01	5/2/93	0:00	0	Primary		29					0.085	X+	16.8	J			0.86	X+	0.86	X+		
16SD01	2/21/95	14:20	0	Primary		18.4					0.09	X+	16.6				0.92	X+	0.92	X+		
16SD01-A				Avg.		23.7					0.0875		16.7				0.89		0.89			
16SD02	4/14/93	0:00	0	Primary		17.3					0.065	X+	10.3				0.635	X+	0.635	X+		
16SD02	2/28/95	10:00	0	Primary		7.87					0.07	X+	7.75				0.69	X+	0.69	X+		
16SD02-A				Avg.		12.585					0.0675		9.025				0.6625		0.6625			
16SD03	4/14/93	0:00	0	Primary		14.5					0.065	X+	6.08				0.65	X+	0.65	X+		
16SD03	2/20/95	16:05	0	Primary		7					0.08	X+	4.1				0.78	X+	0.78	X+		
16SD03-A				Avg.		10.75					0.0725		5.09				0.715		0.715			
16SD06	2/20/95	16:35	0	Primary		10.5					0.075	X+	8.26				0.73	X+	0.73	X+		
16SD07	2/21/95	14:05	0	Primary		7.62					0.08	X+	6.05				0.795	X+	0.795	X+		
16SD08	2/21/95	9:30	0	Primary		4.58					0.065	X+	14.4				0.655	X+	0.655	X+		
16SD09	2/21/95	10:55	0	Primary		49.3					0.065	X+	17.3				0.8		0.665	X+		
16SD10	2/21/95	17:10	0	Primary		23.4					0.065	X+	5.85				0.325	X+	0.65	X+		
16SD11	2/28/95	11:15	0	Primary		9.99					0.065	X+	8.2				0.325	X+	0.65	X+		
16WW01	5/4/93	0:00	0	Primary		7.08					0.06	X+	5.96				0.595	X+	0.595	X+		
16WW01	5/4/93	0:00	0	Duplicate 1		6.94					0.06	X+	7.45				0.58	U+J	0.58	X+		
16WW01-A				Avg.		7.01					0.06		6.705				0.5875		0.5875			
16WW02	5/12/93	0:00	0	Primary		3.02	J				0.06	X+	5.09				0.58	U+J	0.58	X+		
16WW02	5/12/93	0:00	0	Duplicate 1		3.84					0.06	X+	4.54				0.59	X+	0.59	X+		
16WW02-A				Avg.		3.43					0.06		4.815				0.585		0.585			
16WW03	5/4/93	0:00	0	Primary		11.3					0.06	X+	13.7				0.625	U+J	0.625	X+		
16WW04	5/12/93	0:00	0	Primary		10	J				0.06	X+	9.96				0.6	U+J	0.6	X+		
16WW04	5/12/93	0:00	0	Duplicate 1		9.72					0.065	X+	8.15				0.635	X+	0.635	X+		
16WW04-A				Avg.		9.86					0.0625		9.055				0.6175		0.6175			
16WW05	5/4/93	0:00	0	Primary		15.4					0.07	X+	16.5				0.675	U+J	0.675	X+		
16WW06	5/5/93	0:00	0	Primary		11.5					0.065	X+	11.1				0.65	U+J	0.65	X+		
16WW27	5/17/97	9:15	0	Primary	10200	12.1		1030		1270	0.051		9		570		0.32	X+	0.65	X+	68.5	
16WW27	5/17/97	10:17	1	Primary	9550	7.6		743		204	0.02	X+	2.4	U+	481		0.3	X+	0.6	X+	144	
16WW27-A				Avg.	9875	9.85		886.5		737	0.0355		5.7		525.5		0.31		0.625		106.25	
16WW31	5/29/97	8:27	0	Primary	19400	14.3		302.5	X+	893	0.023		6.1		302.5	X+	0.21	X+	0.6	X+	21.1	
16WW31	5/29/97	8:55	1	Primary	23500	8		306.5	X+	45.4	0.02	X+	5.9		306.5	X+	0.61		0.6	X+	61.0	
16WW31-A				Avg.	21450	11.15		304.5		469.2	0.0215		6		304.5		0.41		0.6		41.05	
16WW35	6/25/97	9:50	0	Primary	17700	7.9		1020		40.9	0.02		10.4		1070		1		0.55	U+J	63.6	
16WW35	6/25/97	9:50	0	Duplicate 1	21500	9.2		992		53.4	0.03		9.7		974		1.4		0.6	U+J	24.2	U+
16WW35	6/25/97	10:05	1	Primary	14600	6		909		29.2	0.04		7		308.5	X+	1		0.6	U+J	97.3	
16WW35-A				Avg.	17933.3	7.7		973.667		41.1667	0.03		9.03333		784.167		1.13333		0.58333		61.6833	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(0-5 feet)

SITE	DATE	TIME	DEPTH (	RESULT TYPE	Iron (mg/kg)	Lead (mg/kg)	Magnesium (mg/kg)	Manganese (mg/kg)	Mercury (mg/kg)	Nickel (mg/kg)	Potassium (mg/kg)	Selenium (mg/kg)		Silver (mg/kg)		Sodium (mg/kg)
16WW37	5/31/97	15:55	0	Primary	10100	21.8	688	1060	0.062	8.2	846	0.31	X+	0.55		42.8
16WW37	5/31/97	16:10	1	Primary	26000	13.1	1420	75.7	0.051	13.7	1560	0.74		0.65	X+	44.1
16WW37-A				Avg.	18050	17.45	1054	567.85	0.0565	10.95	1203	0.525		0.6		43.45
NATUAL LOGS																
16SD01-A						3.165			-2.436	2.815		-0.117		-0.117		
16SD02-A						2.533			-2.696	2.2		-0.412		-0.412		
16SD03-A						2.375			-2.624	1.627		-0.335		-0.335		
16SD06						2.351			-2.59	2.111		-0.315		-0.315		
16SD07						2.031			-2.526	1.8		-0.229		-0.229		
16SD08						1.522			-2.733	2.667		-0.423		-0.423		
16SD09						3.898			-2.733	2.851		-0.223		-0.408		
16SD10						3.153			-2.733	1.766		-1.124		-0.431		
16SD11						2.302			-2.733	2.104		-1.124		-0.431		
16WW01-A						1.947			-2.813	1.903		-0.532		-0.532		
16WW02-A						1.233			-2.813	1.572		-0.536		-0.536		
16WW03						2.425			-2.813	2.617		-0.47		-0.47		
16WW04-A						2.288			-2.773	2.203		-0.482		-0.482		
16WW05						2.734			-2.659	2.803		-0.393		-0.393		
16WW06						2.442			-2.733	2.407		-0.431		-0.431		
16WW27-A					9.198	2.287	6.787	6.603	-3.338	1.74	6.264	-1.171		-0.47		4.666
16WW31-A					9.973	2.411	5.719	6.151	-3.84	1.792	5.719	-0.892		-0.511		3.715
16WW35-A					9.794	2.041	6.881	3.718	-3.507	2.201	6.665	0.125		-0.539		4.122
16WW37-A					9.801	2.859	6.96	6.342	-2.874	2.393	7.093	-0.644		-0.511		3.772
STATISTICS																
Total No.					4	19	4	4	19	19	4	19		19		4
Min.					9550	3.02	302.5	29.2	0.02	2.4	302.5	0.21		0.55		21.1
Max.					26000	49.3	1420	1270	0.09	17.3	1560	1.4		0.92		144
Avg.(log transformed)					9.692	2.421	6.587	5.704	-2.84	2.188	6.435	-0.512		-0.42		4.069
St. Dev. (log transformed)					0.339	0.595	0.583	1.337	0.348	0.421	0.585	0.348		0.109		0.437
H					2.932	2.105	4.115	8.825	1.881	1.939	4.126	1.881		1.734		3.354
95th UCL of Mean					30434	18.05	3435.90	6.7E+05	0.07	11.81	2979.97	0.74		0.69		150.01

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(0-5 feet)

SITE	DATE	TIME	DEPTH (	RESULT TYPE	Strontium (mg/kg)	Thallium (mg/kg)		Vanadium (mg/kg)	Zinc (mg/kg)
16SD01	5/2/93	0:00	0	Primary		0.86	X+		
16SD01	2/21/95	14:20	0	Primary		0.46	X+		55.6
16SD01-A				Avg.		0.66			
16SD02	4/14/93	0:00	0	Primary		0.635	X+		
16SD02	2/28/95	10:00	0	Primary		0.345	X+		28.9
16SD02-A				Avg.		0.49			
16SD03	4/14/93	0:00	0	Primary		0.65	X+		
16SD03	2/20/95	16:05	0	Primary		0.78	X+		24
16SD03-A				Avg.		0.715			
16SD06	2/20/95	16:35	0	Primary		0.73	X+		40.4
16SD07	2/21/95	14:05	0	Primary		0.395	X+		168
16SD08	2/21/95	9:30	0	Primary		0.325	X+		21.6
16SD09	2/21/95	10:55	0	Primary		0.335	X+		57
16SD10	2/21/95	17:10	0	Primary		0.325	X+		13
16SD11	2/28/95	11:15	0	Primary		0.325	X+		29.4
16WW01	5/4/93	0:00	0	Primary		0.595	X+		
16WW01	5/4/93	0:00	0	Duplicate 1		0.58	X+		
16WW01-A				Avg.		0.5875			
16WW02	5/12/93	0:00	0	Primary		5.96			
16WW02	5/12/93	0:00	0	Duplicate 1		0.59	X+		
16WW02-A				Avg.		3.275			
16WW03	5/4/93	0:00	0	Primary		1.89			
16WW04	5/12/93	0:00	0	Primary		1.64			
16WW04	5/12/93	0:00	0	Duplicate 1		1.35			
16WW04-A				Avg.		1.495			
16WW05	5/4/93	0:00	0	Primary		0.675	X+		
16WW06	5/5/93	0:00	0	Primary		2			
16WW27	5/17/97	9:15	0	Primary	34.1	0.65	X+	16	27.3
16WW27	5/17/97	10:17	1	Primary	6.7	0.6	X+	14.3	17.3
16WW27-A				Avg.	20.4	0.625		15.15	22.3
16WW31	5/29/97	8:27	0	Primary	2.4	0.6	X+	33.5	13.3
16WW31	5/29/97	8:55	1	Primary	6.0	0.25		38.9	11.9
16WW31-A				Avg.	4.2	0.425		36.2	12.6
16WW35	6/25/97	9:50	0	Primary	11.2	0.55	X+	30.6	22.2
16WW35	6/25/97	9:50	0	Duplicate 1	12.7	0.6	X+	33	20.9
16WW35	6/25/97	10:05	1	Primary	10.3	0.6	X+	20	18.3
16WW35-A				Avg.	11.4	0.58333		27.8667	20.4667

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(0-5 feet)

SITE	DATE	TIME	DEPTH (	RESULT TYPE	Strontium (mg/kg)	Thallium (mg/kg)		Vanadium (mg/kg)	Zinc (mg/kg)
16WW37	5/31/97	15:55	0	Primary	62.7	0.6	X+	22.5	38.9
16WW37	5/31/97	16:10	1	Primary	26.7	0.18		43.3	27.3
16WW37-A				Avg.	44.7	0.39		32.9	33.1
NATUAL LOGS									
16SD01-A						-0.416			
16SD02-A						-0.713			
16SD03-A						-0.335			
16SD06						-0.315			3.699
16SD07						-0.929			5.124
16SD08						-1.124			3.073
16SD09						-1.094			4.043
16SD10						-1.124			2.565
16SD11						-1.124			3.381
16WW01-A						-0.532			
16WW02-A						1.186			
16WW03						0.637			
16WW04-A						0.402			
16WW05						-0.393			
16WW06						0.693			
16WW27-A					3.016	-0.47		2.718	3.105
16WW31-A					1.435	-0.856		3.589	2.534
16WW35-A					2.434	-0.539		3.327	3.019
16WW37-A					3.8	-0.942		3.493	3.5
STATISTICS									
Total No.					4	19		4	10
Min.					2.4	0.18		14.3	11.9
Max.					62.7	5.96		43.3	168
Avg.(log transformed)					2.671	-0.42		3.282	3.404
St. Dev. (log transformed)					0.996	0.683		0.391	0.765
H					6.644	2.202		3.139	2.648
95th UCL of Mean					1083.10	1.18		58.38	79.19

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,1,1,2-Tetra chloroethane (ug/kg)		1,1,1-trichloro ethane (ug/kg)		1,1,2,2-Tetra chloroethane (ug/kg)		1,1,2-Trichloro ethane (ug/kg)		1,1-Dichloro ethane (ug/kg)		1,1-Dichloro ethene (ug/kg)		1,1-Dichloro propene (ug/kg)		1,2,3-Trichloro benzene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary			4.3	X+	4.3	X+	4.3	X+	4.3	X+	4.3	X+				
16SD01	2/21/95	14:20	0	Primary			4.5	X+	4.5	X+	4.5	X+	4.5	X+	4.5	X+				
16SD01-A				Avg.			4.4		4.4		4.4		4.4		4.4					
16SD02	4/14/93	0:00	0	Primary			3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+				
16SD02	2/28/95	10:00	0	Primary			3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+				
16SD02-A				Avg.			3.325		3.325		3.325		3.325		3.325					
16SD03	4/14/93	0:00	0	Primary			3.25	X+	3.25	X+	3.25	X+	3.25	X+	3.25	X+				
16SD03	2/20/95	16:05	0	Primary			4	X+	4	X+	4	X+	4	X+	4	X+				
16SD03-A				Avg.			3.625		3.625		3.625		3.625		3.625					
16SD06	2/20/95	16:35	0	Primary			3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+				
16SD07	2/21/95	14:05	0	Primary			4	X+	4	X+	4	X+	4	X+	4	X+				
16SD08	2/21/95	9:30	0	Primary			3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+				
16SD09	2/21/95	10:55	0	Primary			3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+				
16SD10	2/21/95	17:10	0	Primary			3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+				
16SD11	2/28/95	11:15	0	Primary			3	X+	3	X+	3	X+	3	X+	3	X+				
16WW01	5/4/93	0:00	0	Primary			3	X+	3	X+	3	X+	3	X+	3	X+				
16WW01	5/4/93	0:00	0	Duplicate 1			2.9	X+	2.9	X+	2.9	X+	2.9	X+	2.9	X+				
16WW01-A				Avg.			2.95		2.95		2.95		2.95		2.95					
16WW02	5/12/93	0:00	0	Primary			2.9	X+	2.9	X+	2.9	X+	2.9	X+	2.9	X+				
16WW02	5/12/93	0:00	0	Duplicate 1			2.95	X+	2.95	X+	2.95	X+	2.95	X+	2.95	X+				
16WW02-A				Avg.			2.925		2.925		2.925		2.925		2.925					
16WW03	5/4/93	0:00	0	Primary			3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+				
16WW04	5/12/93	0:00	0	Primary			3	X+	3	X+	3	X+	3	X+	3	X+				
16WW04	5/12/93	0:00	0	Duplicate 1			3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+				
16WW04-A				Avg.			3.075		3.075		3.075		3.075		3.075					
16WW05	5/4/93	0:00	0	Primary			3.4	X+	3.4	X+	3.4	X+	3.4	X+	3.4	X+				
16WW06	5/5/93	0:00	0	Primary			3.25	X+	3.25	X+	3.25	X+	3.25	X+	3.25	X+				
16WW27	5/17/97	9:15	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW27	5/17/97	10:17	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW27-A				Avg.	3		3		3		3		3		3		3		3	
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	8:55	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31-A				Avg.	3		3		3		3		3		3		3		3	
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate 1	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate 2	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	10:05	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,1,1,2-Tetra chloroethane (ug/kg)		1,1,1-trichloro ethane (ug/kg)		1,1,2,2-Tetra chloroethane (ug/kg)		1,1,2-Trichloro ethane (ug/kg)		1,1-Dichloro ethane (ug/kg)		1,1-Dichloro ethene (ug/kg)		1,1-Dichloro propene (ug/kg)		1,2,3-Trichloro benzene (ug/kg)	
16WW35-A				Avg.	2.625		2.625		2.625		2.625		2.625		2.625		2.625		2.625	
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37	5/31/97	16:10	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37-A				Avg.	3		3		3		3		3		3		3		3	
NATURAL LOGS																				
16SD01-A							1.482		1.482		1.482		1.482		1.482					
16SD02-A							1.201		1.201		1.201		1.201		1.201					
16SD03-A							1.288		1.288		1.288		1.288		1.288					
16SD06							1.253		1.253		1.253		1.253		1.253					
16SD07							1.386		1.386		1.386		1.386		1.386					
16SD08							1.253		1.253		1.253		1.253		1.253					
16SD09							1.253		1.253		1.253		1.253		1.253					
16SD10							1.253		1.253		1.253		1.253		1.253					
16SD11							1.099		1.099		1.099		1.099		1.099					
16WW01-A							1.082		1.082		1.082		1.082		1.082					
16WW02-A							1.073		1.073		1.073		1.073		1.073					
16WW03							1.147		1.147		1.147		1.147		1.147					
16WW04-A							1.123		1.123		1.123		1.123		1.123					
16WW05							1.224		1.224		1.224		1.224		1.224					
16WW06							1.179		1.179		1.179		1.179		1.179					
16WW27-A					1.099		1.099		1.099		1.099		1.099		1.099		1.099		1.099	
16WW31-A					1.099		1.099		1.099		1.099		1.099		1.099		1.099		1.099	
16WW35-A					0.965		0.965		0.965		0.965		0.965		0.965		0.965		0.965	
16WW37-A					1.099		1.099		1.099		1.099		1.099		1.099		1.099		1.099	
STATISTICS																				
Total No.					4		19		19		19		19		19		4		4	
Min.					2.5		2.5		2.5		2.5		2.5		2.5		2.5		2.5	
Max.					3		4.5		4.5		4.5		4.5		4.5		3		3	
Avg.(log transformed)					1.066		1.187		1.187		1.187		1.187		1.187		1.066		1.066	
St. Dev.(log transformed)					0.067		0.122		0.122		0.122		0.122		0.122		0.067		0.067	
H																				
95th UCL of Mean					2.91		3.30		3.30		3.30		3.30		3.30		2.91		2.91	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,2,3-Trichloro propane (ug/kg)		1,2,4-Trichloro benzene (ug/kg)		1,2-Dichloro ethane (ug/kg)		DCE12TOT (ug/kg)		1,2-Dichloro propane (ug/kg)		1,3-Dichloro propane (ug/kg)		1,4-Dichloro-2-butene (ug/kg)		1,4-Dioxane (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	4.3	X+			4.3	X+			4.3	X+			8.5	X+		
16SD01	2/21/95	14:20	0	Primary	4.5	X+			4.5	X+	4.5	X+	4.5	X+			9	X+		
16SD01-A				Avg.	4.4				4.4				4.4				8.75			
16SD02	4/14/93	0:00	0	Primary	3.15	X+			3.15	X+			3.15	X+			6.5	X+		
16SD02	2/28/95	10:00	0	Primary	3.5	X+			3.5	X+	3.5	X+	3.5	X+			7	X+		
16SD02-A				Avg.	3.325				3.325				3.325				6.75			
16SD03	4/14/93	0:00	0	Primary	3.25	X+			3.25	X+			3.25	X+			6.5	X+		
16SD03	2/20/95	16:05	0	Primary	4	X+			4	X+	4	X+	4	X+			8	X+		
16SD03-A				Avg.	3.625				3.625				3.625				7.25			
16SD06	2/20/95	16:35	0	Primary	3.5	X+			3.5	X+	3.5	X+	3.5	X+			7.5	X+		
16SD07	2/21/95	14:05	0	Primary	4	X+			4	X+	4	X+	4	X+			8	X+		
16SD08	2/21/95	9:30	0	Primary	3.5	X+			3.5	X+	3.5	X+	3.5	X+			6.5	X+		
16SD09	2/21/95	10:55	0	Primary	3.5	X+			3.5	X+	3.5	X+	3.5	X+			6.5	X+		
16SD10	2/21/95	17:10	0	Primary	3.5	X+			3.5	X+	3.5	X+	3.5	X+			6.5	X+		
16SD11	2/28/95	11:15	0	Primary	3	X+			3	X+	3	X+	3	X+			6.5	X+		
16WW01	5/4/93	0:00	0	Primary	3	X+			3	X+			3	X+			6	X+		
16WW01	5/4/93	0:00	0	Duplicate 1	2.9	X+			2.9	X+			2.9	X+			6	X+		
16WW01-A				Avg.	2.95				2.95				2.95				6			
16WW02	5/12/93	0:00	0	Primary	2.9	X+			2.9	X+			2.9	X+			6	X+		
16WW02	5/12/93	0:00	0	Duplicate 1	2.95	X+			2.95	X+			2.95	X+			6	X+		
16WW02-A				Avg.	2.925				2.925				2.925				6			
16WW03	5/4/93	0:00	0	Primary	3.15	X+			3.15	X+			3.15	X+			6.5	X+		
16WW04	5/12/93	0:00	0	Primary	3	X+			3	X+			3	X+			6	X+		
16WW04	5/12/93	0:00	0	Duplicate 1	3.15	X+			3.15	X+			3.15	X+			6.5	X+		
16WW04-A				Avg.	3.075				3.075				3.075				6.25			
16WW05	5/4/93	0:00	0	Primary	3.4	X+			3.4	X+			3.4	X+			7	X+		
16WW06	5/5/93	0:00	0	Primary	3.25	X+			3.25	X+			3.25	X+			6.5	X+		
16WW27	5/17/97	9:15	0	Primary	9.5	X+	3	X+	3	X+			3	X+	3	X+	3	X+	650	X+
16WW27	5/17/97	10:17	1	Primary	9	X+	3	X+	3	X+			3	X+	3	X+	3	X+	600	X+
16WW27-A				Avg.	9.25		3		3				3		3		3		625	
16WW31	5/29/97	8:27	0	Primary	9	X+	3	X+	3	X+			3	X+	3	X+	3	X+	600	X+
16WW31	5/29/97	8:55	1	Primary	9	X+	3	X+	3	X+			3	X+	3	X+	3	X+	600	X+
16WW31-A				Avg.	9		3		3				3		3		3		600	
16WW35	6/25/97	9:50	0	Primary	8	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+	2.5	X+	550	X+
16WW35	6/25/97	9:50	0	Duplicate 1	7.5	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+	2.5	X+	500	X+
16WW35	6/25/97	0:00	0	Duplicate 2	7.5	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+	2.5	X+	500	X+
16WW35	6/25/97	10:05	1	Primary	9.5	X+	3	X+	3	X+			3	X+	3	X+	3	X+	600	X+

Longhorn Army Ammuntion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,2,3-Trichloropropane (ug/kg)		1,2,4-Trichlorobenzene (ug/kg)		1,2-Dichloroethane (ug/kg)		DCE12TOT (ug/kg)	1,2-Dichloropropane (ug/kg)		1,3-Dichloropropane (ug/kg)		1,4-Dichloro-2-butene (ug/kg)		1,4-Dioxane (ug/kg)	
16WW35-A				Avg.	8.125		2.625		2.625			2.625		2.625		2.625		537.5	
16WW37	5/31/97	15:55	0	Primary	9.5	X+	3	X+	3	X+		3	X+	3	X+	3	X+	600	X+
16WW37	5/31/97	16:10	1	Primary	9.5	X+	3	X+	3	X+		3	X+	3	X+	3	X+	650	X+
16WW37-A				Avg.	9.5		3		3			3		3		3		625	
NATURAL LOGS																			
16SD01-A					1.482				1.482			1.482				2.169			
16SD02-A					1.201				1.201			1.201				1.91			
16SD03-A					1.288				1.288			1.288				1.981			
16SD06					1.253				1.253		1.253	1.253				2.015			
16SD07					1.386				1.386		1.386	1.386				2.079			
16SD08					1.253				1.253		1.253	1.253				1.872			
16SD09					1.253				1.253		1.253	1.253				1.872			
16SD10					1.253				1.253		1.253	1.253				1.872			
16SD11					1.099				1.099		1.099	1.099				1.872			
16WW01-A					1.082				1.082			1.082				1.792			
16WW02-A					1.073				1.073			1.073				1.792			
16WW03					1.147				1.147			1.147				1.872			
16WW04-A					1.123				1.123			1.123				1.833			
16WW05					1.224				1.224			1.224				1.946			
16WW06					1.179				1.179			1.179				1.872			
16WW27-A					2.225		1.099		1.099			1.099		1.099		1.099		6.438	
16WW31-A					2.197		1.099		1.099			1.099		1.099		1.099		6.397	
16WW35-A					2.095		0.965		0.965			0.965		0.965		0.965		6.287	
16WW37-A					2.251		1.099		1.099			1.099		1.099		1.099		6.438	
STATISTICS																			
Total No.					19		4		19		6	19		4		19		4	
Min.					2.9		2.5		2.5		3	2.5		2.5		2.5		500	
Max.					9.5		3		4.5		4.5	4.5		3		9		650	
Avg.(log transformed)					1.424		1.066		1.187		1.25	1.187		1.066		1.737		6.39	
St. Dev.(log transformed)					0.42		0.067		0.122		0.091	0.122		0.067		0.369		0.071	
H																			
95th UCL of Mean					4.54		2.91		3.30		3.50	3.30		2.91		6.08		597.36	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	2,2-Dichloro propane (ug/kg)		2-Hexanone (ug/kg)		Acetone (ug/kg)		Acrolein (ug/kg)		Acrylonitrile (ug/kg)		Benzene (ug/kg)		1,2,4-Trimethyl benzene (ug/kg)		1,3,5-Trimethyl benzene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary			8.5	X+	8.5	X+	8.5	X+	8.5	X+	4.3	X+				
16SD01	2/21/95	14:20	0	Primary			9	X+	103		46	X+	46	X+	4.5	X+				
16SD01-A				Avg.			8.75		55.75		27.25		27.25		4.4					
16SD02	4/14/93	0:00	0	Primary			6.5	X+	6.5	X+	6.5	X+	6.5	X+	3.15	X+				
16SD02	2/28/95	10:00	0	Primary			7	X+	7	X+	34.5	X+	34.5	X+	3.5	X+				
16SD02-A				Avg.			6.75		6.75		20.5		20.5		3.325					
16SD03	4/14/93	0:00	0	Primary			6.5	X+	6.5	X+	6.5	X+	6.5	X+	3.25	X+				
16SD03	2/20/95	16:05	0	Primary			8	X+	8	X+	39	X+	39	X+	4	X+				
16SD03-A				Avg.			7.25		7.25		22.75		22.75		3.625					
16SD06	2/20/95	16:35	0	Primary			7.5	X+	26		36.5	X+	36.5	X+	3.5	X+				
16SD07	2/21/95	14:05	0	Primary			8	X+	8	X+	39.5	X+	39.5	X+	4	X+				
16SD08	2/21/95	9:30	0	Primary			6.5	X+	6.5	X+	32.5	X+	32.5	X+	3.5	X+				
16SD09	2/21/95	10:55	0	Primary			6.5	X+	6.5	X+	33.5	X+	33.5	X+	3.5	X+				
16SD10	2/21/95	17:10	0	Primary			6.5	X+	6.5	X+	32.5	X+	32.5	X+	3.5	X+				
16SD11	2/28/95	11:15	0	Primary			6.5	X+	22		32.5	X+	32.5	X+	3	X+				
16WW01	5/4/93	0:00	0	Primary			6	X+	6	X+	6	X+	6	X+	3	X+				
16WW01	5/4/93	0:00	0	Duplicate 1			6	X+	6	X+	6	X+	6	X+	2.9	X+				
16WW01-A				Avg.			6		6		6		6		2.95					
16WW02	5/12/93	0:00	0	Primary			6	X+	6	X+	6	X+	6	X+	2.9	X+				
16WW02	5/12/93	0:00	0	Duplicate 1			6	X+	6	X+	6	X+	6	X+	2.95	X+				
16WW02-A				Avg.			6		6		6		6		2.925					
16WW03	5/4/93	0:00	0	Primary			6.5	X+	6.5	X+	6.5	X+	6.5	X+	3.15	X+				
16WW04	5/12/93	0:00	0	Primary			6	X+	6	X+	6	X+	6	X+	3	X+				
16WW04	5/12/93	0:00	0	Duplicate 1			6.5	X+	6.5	X+	6.5	X+	6.5	X+	3.15	X+				
16WW04-A				Avg.			6.25		6.25		6.25		6.25		3.075					
16WW05	5/4/93	0:00	0	Primary			7	X+	7	X+	7	X+	7	X+	3.4	X+				
16WW06	5/5/93	0:00	0	Primary			6.5	X+	6.5	X+	6.5	X+	6.5	X+	3.25	X+				
16WW27	5/17/97	9:15	0	Primary	9.5	X+	13	X+	13	X+	65	X+	65	X+	3	X+	3	X+	3	X+
16WW27	5/17/97	10:17	1	Primary	9	X+	12	X+	12	X+	60	X+	60	X+	3	X+	3	X+	3	X+
16WW27-A				Avg.	9.25		12.5		12.5		62.5		62.5		3		3		3	
16WW31	5/29/97	8:27	0	Primary	93.5	X+	12	X+	12	X+	60	X+	60	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	8:55	1	Primary	9	X+	12	X+	12	X+	60	X+	60	X+	3	X+	3	X+	3	X+
16WW31-A				Avg.	51.25		12		12		60		60		3		3		3	
16WW35	6/25/97	9:50	0	Primary	8	X+	10.5	X+	10.5	X+	55	X+	55	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate 1	7.5	X+	10	X+	20		50	X+	50	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate 2	7.5	X+	10	X+	10	X+	50	X+	50	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	10:05	1	Primary	9.5	X+	12.5	X+	13		60	X+	60	X+	3	X+	3	X+	3	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	2,2-Dichloropropane (ug/kg)		2-Hexanone (ug/kg)		Acetone (ug/kg)		Acrolein (ug/kg)		Acrylonitrile (ug/kg)		Benzene (ug/kg)		1,2,4-Trimethylbenzene (ug/kg)		1,3,5-Trimethylbenzene (ug/kg)	
16WW35-A				Avg.	8.125		10.75		13.375		53.75		53.75		2.625		2.625		2.625	
16WW37	5/31/97	15:55	0	Primary	9.5	X+	12.5	X+	12.5	X+	60	X+	60	X+	3	X+	3	X+	3	X+
16WW37	5/31/97	16:10	1	Primary	9.5	X+	12.5	X+	12.5	X+	65	X+	65	X+	3	X+	3	X+	3	X+
16WW37-A				Avg.	9.5		12.5		12.5		62.5		62.5		3		3		3	
NATURAL LOGS																				
16SD01-A							2.169		4.021		3.305		3.305		1.482					
16SD02-A							1.91		1.91		3.02		3.02		1.201					
16SD03-A							1.981		1.981		3.125		3.125		1.288					
16SD06							2.015		3.258		3.597		3.597		1.253					
16SD07							2.079		2.079		3.676		3.676		1.386					
16SD08							1.872		1.872		3.481		3.481		1.253					
16SD09							1.872		1.872		3.512		3.512		1.253					
16SD10							1.872		1.872		3.481		3.481		1.253					
16SD11							1.872		3.091		3.481		3.481		1.099					
16WW01-A							1.792		1.792		1.792		1.792		1.082					
16WW02-A							1.792		1.792		1.792		1.792		1.073					
16WW03							1.872		1.872		1.872		1.872		1.147					
16WW04-A							1.833		1.833		1.833		1.833		1.123					
16WW05							1.946		1.946		1.946		1.946		1.224					
16WW06							1.872		1.872		1.872		1.872		1.179					
16WW27-A					2.225		2.526		2.526		4.135		4.135		1.099		1.099		1.099	
16WW31-A					3.937		2.485		2.485		4.094		4.094		1.099		1.099		1.099	
16WW35-A					2.095		2.375		2.593		3.984		3.984		0.965		0.965		0.965	
16WW37-A					2.251		2.526		2.526		4.135		4.135		1.099		1.099		1.099	
STATISTICS																				
Total No.					4		19		19		19		19		19		4		4	
Min.					7.5		6		6		6		6		2.5		2.5		2.5	
Max.					93.5		13		103		65		65		4.5		3		3	
Avg.(log transformed)					2.627		2.035		2.273		3.06		3.06		1.187		1.066		1.066	
St. Dev.(log transformed)					0.876		0.254		0.614		0.897		0.897		0.122		0.067		0.067	
H									2.126											
95th UCL of Mean					20.30		7.90		15.95		31.89		31.89		3.30		2.91		2.91	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Isopropyl benzene (ug/kg)		Bromobenzene (ug/kg)		Bromodichloro methane (ug/kg)		Bromoform (ug/kg)		Carbon disulfide (ug/kg)		Carbon tetrachloride (ug/kg)		Chlorobenzene (ug/kg)		Bromochloro methane (ug/kg)	
16SD01	5/2/93	0:00	0	Primary					4.3	X+			4.3	X+	4.3	X+	4.3	X+		
16SD01	2/21/95	14:20	0	Primary					4.5	X+	4.5	X+	4.5	X+	4.5	X+	4.5	X+		
16SD01-A				Avg.					4.4				4.4		4.4		4.4			
16SD02	4/14/93	0:00	0	Primary					3.15	X+			3.15	X+	3.15	X+	3.15	X+		
16SD02	2/28/95	10:00	0	Primary					3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD02-A				Avg.					3.325				3.325		3.325		3.325			
16SD03	4/14/93	0:00	0	Primary					3.25	X+			3.25	X+	3.25	X+	3.25	X+		
16SD03	2/20/95	16:05	0	Primary					4	X+	4	X+	4	X+	4	X+	4	X+		
16SD03-A				Avg.					3.625				3.625		3.625		3.625			
16SD06	2/20/95	16:35	0	Primary					3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD07	2/21/95	14:05	0	Primary					4	X+	4	X+	4	X+	4	X+	4	X+		
16SD08	2/21/95	9:30	0	Primary					3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD09	2/21/95	10:55	0	Primary					3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD10	2/21/95	17:10	0	Primary					3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD11	2/28/95	11:15	0	Primary					3	X+	3	X+	3	X+	3	X+	3	X+		
16WW01	5/4/93	0:00	0	Primary					3	X+			3	X+	3	X+	3	X+		
16WW01	5/4/93	0:00	0	Duplicate 1					2.9	X+			2.9	X+	2.9	X+	2.9	X+		
16WW01-A				Avg.					2.95				2.95		2.95		2.95			
16WW02	5/12/93	0:00	0	Primary					2.9	X+			2.9	X+	2.9	X+	2.9	X+		
16WW02	5/12/93	0:00	0	Duplicate 1					2.95	X+			2.95	X+	2.95	X+	2.95	X+		
16WW02-A				Avg.					2.925				2.925		2.925		2.925			
16WW03	5/4/93	0:00	0	Primary					3.15	X+			3.15	X+	3.15	X+	3.15	X+		
16WW04	5/12/93	0:00	0	Primary					3	X+			3	X+	3	X+	3	X+		
16WW04	5/12/93	0:00	0	Duplicate 1					3.15	X+			3.15	X+	3.15	X+	3.15	X+		
16WW04-A				Avg.					3.075				3.075		3.075		3.075			
16WW05	5/4/93	0:00	0	Primary					3.4	X+			3.4	X+	3.4	X+	3.4	X+		
16WW06	5/5/93	0:00	0	Primary					3.25	X+			3.25	X+	3.25	X+	3.25	X+		
16WW27	5/17/97	9:15	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	6.5	X+	3	X+	3	X+
16WW27	5/17/97	10:17	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW27-A				Avg.	3		3		3		3		3		6.25		3		3	
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW31	5/29/97	8:55	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW31-A				Avg.	3		3		3		3		3		6		3		3	
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	5.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate 1	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate 2	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	10:05	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Isopropyl benzene (ug/kg)		Bromobenzene (ug/kg)		Bromodichloro methane (ug/kg)		Bromoform (ug/kg)		Carbon disulfide (ug/kg)		Carbon tetrachloride (ug/kg)		Chlorobenzene (ug/kg)		Bromochloro methane (ug/kg)	
16WW35-A				Avg.	2.625		2.625		2.625		2.625		2.625		5.375		2.625		2.625	
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW37	5/31/97	16:10	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	6.5	X+	3	X+	3	X+
16WW37-A				Avg.	3		3		3		3		3		6.25		3		3	
NATURAL LOGS																				
16SD01-A									1.482				1.482		1.482		1.482			
16SD02-A									1.201				1.201		1.201		1.201			
16SD03-A									1.288				1.288		1.288		1.288			
16SD06									1.253		1.253		1.253		1.253		1.253			
16SD07									1.386		1.386		1.386		1.386		1.386			
16SD08									1.253		1.253		1.253		1.253		1.253			
16SD09									1.253		1.253		1.253		1.253		1.253			
16SD10									1.253		1.253		1.253		1.253		1.253			
16SD11									1.099		1.099		1.099		1.099		1.099			
16WW01-A									1.082				1.082		1.082		1.082			
16WW02-A									1.073				1.073		1.073		1.073			
16WW03									1.147				1.147		1.147		1.147			
16WW04-A									1.123				1.123		1.123		1.123			
16WW05									1.224				1.224		1.224		1.224			
16WW06									1.179				1.179		1.179		1.179			
16WW27-A					1.099		1.099		1.099		1.099		1.099		1.833		1.099		1.099	
16WW31-A					1.099		1.099		1.099		1.099		1.099		1.792		1.099		1.099	
16WW35-A					0.965		0.965		0.965		0.965		0.965		1.682		0.965		0.965	
16WW37-A					1.099		1.099		1.099		1.099		1.099		1.833		1.099		1.099	
STATISTICS																				
Total No.					4		4		19		10		19		19		19		4	
Min.					2.5		2.5		2.5		2.5		2.5		2.9		2.5		2.5	
Max.					3		3		4.5		4.5		4.5		6.5		4.5		3	
Avg.(log transformed)					1.066		1.066		1.187		1.176		1.187		1.339		1.187		1.066	
St. Dev.(log transformed)					0.067		0.067		0.122		0.123		0.122		0.258		0.122		0.067	
H																				
95th UCL of Mean					2.91		2.91		3.30		3.27		3.30		3.94		3.30		2.91	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Chloroethane (ug/kg)		Chloroform (ug/kg)		cis-1,2-Dichloroethene (ug/kg)		cis-1,3-Dichloropropene (ug/kg)		1,2-Dibromo-3-chloropropane (ug/kg)		Dibromochloromethane (ug/kg)		Dichlorodifluoromethane (ug/kg)		1,2-Dibromoeth (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	8.5	X+	4.3	X+	4.3	X+					4.3	X+	4.3	X+		
16SD01	2/21/95	14:20	0	Primary	9	X+	4.5	X+			4.5	X+			4.5	X+	4.5	X+		
16SD01-A				Avg.	8.75		4.4		4.3						4.4		4.4			
16SD02	4/14/93	0:00	0	Primary	6.5	X+	3.15	X+	3.15	X+					3.15	X+	3.15	X+		
16SD02	2/28/95	10:00	0	Primary	7	X+	3.5	X+			3.5	X+			3.5	X+	3.5	X+		
16SD02-A				Avg.	6.75		3.325		3.15						3.325		3.325			
16SD03	4/14/93	0:00	0	Primary	6.5	X+	3.25	X+	3.25	X+					3.25	X+	3.25	X+		
16SD03	2/20/95	16:05	0	Primary	8	X+	4	X+			4	X+			4	X+	4	X+		
16SD03-A				Avg.	7.25		3.625		3.25						3.625		3.625			
16SD06	2/20/95	16:35	0	Primary	7.5	X+	3.5	X+			3.5	X+			3.5	X+	3.5	X+		
16SD07	2/21/95	14:05	0	Primary	8	X+	4	X+			4	X+			4	X+	4	X+		
16SD08	2/21/95	9:30	0	Primary	6.5	X+	3.5	X+			3.5	X+			3.5	X+	3.5	X+		
16SD09	2/21/95	10:55	0	Primary	6.5	X+	3.5	X+			3.5	X+			3.5	X+	3.5	X+		
16SD10	2/21/95	17:10	0	Primary	6.5	X+	3.5	X+			3.5	X+			3.5	X+	3.5	X+		
16SD11	2/28/95	11:15	0	Primary	6.5	X+	3	X+			3	X+			3	X+	3	X+		
16WW01	5/4/93	0:00	0	Primary	6	X+	3	X+	3	X+					3	X+	3	X+		
16WW01	5/4/93	0:00	0	Duplicate 1	6	X+	2.9	X+	2.9	X+					2.9	X+	2.9	X+		
16WW01-A				Avg.	6		2.95		2.95						2.95		2.95			
16WW02	5/12/93	0:00	0	Primary	6	X+	2.9	X+	2.9	X+					2.9	X+	2.9	X+		
16WW02	5/12/93	0:00	0	Duplicate 1	6	X+	2.95	X+	2.95	X+					2.95	X+	2.95	X+		
16WW02-A				Avg.	6		2.925		2.925						2.925		2.925			
16WW03	5/4/93	0:00	0	Primary	6.5	X+	3.15	X+	3.15	X+					3.15	X+	3.15	X+		
16WW04	5/12/93	0:00	0	Primary	6	X+	3	X+	3	X+					3	X+	3	X+		
16WW04	5/12/93	0:00	0	Duplicate 1	6.5	X+	3.15	X+	3.15	X+					3.15	X+	3.15	X+		
16WW04-A				Avg.	6.25		3.075		3.075						3.075		3.075			
16WW05	5/4/93	0:00	0	Primary	7	X+	3.4	X+	3.4	X+					3.4	X+	3.4	X+		
16WW06	5/5/93	0:00	0	Primary	6.5	X+	3.25	X+	3.25	X+					3.25	X+	3.25	X+		
16WW27	5/17/97	9:15	0	Primary	6.5	X+	3	X+	3	X+	3	X+	6.5	X+	3	X+	9.5	X+	3	X+
16WW27	5/17/97	10:17	1	Primary	6	X+	3	X+	3	X+	3	X+	6	X+	3	X+	9	X+	3	X+
16WW27-A				Avg.	6.25		3		3		3		6.25		3		9.25		3	
16WW31	5/29/97	8:27	0	Primary	6	X+	3	X+	3	X+	3	X+	3	X+	3	X+	9	X+	3	X+
16WW31	5/29/97	8:55	1	Primary	6	X+	3	X+	3	X+	3	X+	6	X+	3	X+	9	X+	3	X+
16WW31-A				Avg.	6		3		3		3		4.5		3		9		3	
16WW35	6/25/97	9:50	0	Primary	5.5	X+	2.5	X+	2.5	X+	2.5	X+	5.5	X+	2.5	X+	8	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate 1	5	X+	2.5	X+	2.5	X+	2.5	X+	5	X+	2.5	X+	7.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate 2	5	X+	2.5	X+	2.5	X+	2.5	X+	5	X+	2.5	X+	7.5	X+	2.5	X+
16WW35	6/25/97	10:05	1	Primary	6	X+	3	X+	3	X+	3	X+	6	X+	3	X+	9.5	X+	3	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Chloroethane (ug/kg)		Chloroform (ug/kg)		cis-1,2-Dichloroethene (ug/kg)		cis-1,3-Dichloropropene (ug/kg)		1,2-Dibromo-3-chloropropane (ug/kg)		Dibromochloromethane (ug/kg)		Dichlorodifluoromethane (ug/kg)		1,2-Dibromoeth (ug/kg)	
16WW35-A				Avg.	5.375		2.625		2.625		2.625		5.375		2.625		8.125		2.625	
16WW37	5/31/97	15:55	0	Primary	6	X+	3	X+	3	X+	3	X+	6	X+	3	X+	9.5	X+	3	X+
16WW37	5/31/97	16:10	1	Primary	6.5	X+	3	X+	3	X+	3	X+	6.5	X+	3	X+	9.5	X+	3	X+
16WW37-A				Avg.	6.25		3		3		3		6.25		3		9.5		3	
NATURAL LOGS																				
16SD01-A					2.169		1.482		1.459						1.482		1.482			
16SD02-A					1.91		1.201		1.147						1.201		1.201			
16SD03-A					1.981		1.288		1.179						1.288		1.288			
16SD06					2.015		1.253				1.253				1.253		1.253			
16SD07					2.079		1.386				1.386				1.386		1.386			
16SD08					1.872		1.253				1.253				1.253		1.253			
16SD09					1.872		1.253				1.253				1.253		1.253			
16SD10					1.872		1.253				1.253				1.253		1.253			
16SD11					1.872		1.099				1.099				1.099		1.099			
16WW01-A					1.792		1.082		1.082						1.082		1.082			
16WW02-A					1.792		1.073		1.073						1.073		1.073			
16WW03					1.872		1.147		1.147						1.147		1.147			
16WW04-A					1.833		1.123		1.123						1.123		1.123			
16WW05					1.946		1.224		1.224						1.224		1.224			
16WW06					1.872		1.179		1.179						1.179		1.179			
16WW27-A					1.833		1.099		1.099		1.099		1.833		1.099		2.225		1.099	
16WW31-A					1.792		1.099		1.099		1.099		1.504		1.099		2.197		1.099	
16WW35-A					1.682		0.965		0.965		0.965		1.682		0.965		2.095		0.965	
16WW37-A					1.833		1.099		1.099		1.099		1.833		1.099		2.251		1.099	
STATISTICS																				
Total No.					19		19		13		10		4		19		19		4	
Min.					5		2.5		2.5		2.5		3		2.5		2.9		2.5	
Max.					9		4.5		4.3		4.5		6.5		4.5		9.5		3	
Avg.(log transformed)					1.889		1.187		1.144		1.176		1.713		1.187		1.424		1.066	
St. Dev.(log transformed)					0.112		0.122		0.114		0.123		0.156		0.122		0.42		0.067	
H																				
95th UCL of Mean					6.65		3.30		3.16		3.27		5.61		3.30		4.54		2.91	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	CEVETH (ug/kg)		trans-1,2-Dichloroethene (ug/kg)		Propionitrile (ug/kg)		Ethyl methacrylate (ug/kg)		Ethylbenzene (ug/kg)		Hexachloro butadiene (ug/kg)		Isobutyl alcohol (ug/kg)		1,3-Dichloro benzene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary			4.3	X+			4.3	X+	4.3	X+						
16SD01	2/21/95	14:20	0	Primary	9	X+					4.5	X+	4.5	X+						
16SD01-A				Avg.			4.3				4.4		4.4							
16SD02	4/14/93	0:00	0	Primary			3.15	X+			3.15	X+	3.15	X+						
16SD02	2/28/95	10:00	0	Primary	7	X+					3.5	X+	3.5	X+						
16SD02-A				Avg.			3.15				3.325		3.325							
16SD03	4/14/93	0:00	0	Primary			3.25	X+			3.25	X+	3.25	X+						
16SD03	2/20/95	16:05	0	Primary	8	X+					4	X+	4	X+						
16SD03-A				Avg.			3.25				3.625		3.625							
16SD06	2/20/95	16:35	0	Primary	7.5	X+					3.5	X+	3.5	X+						
16SD07	2/21/95	14:05	0	Primary	8	X+					4	X+	4	X+						
16SD08	2/21/95	9:30	0	Primary	6.5	X+					3.5	X+	3.5	X+						
16SD09	2/21/95	10:55	0	Primary	6.5	X+					3.5	X+	3.5	X+						
16SD10	2/21/95	17:10	0	Primary	6.5	X+					3.5	X+	3.5	X+						
16SD11	2/28/95	11:15	0	Primary	6.5	X+					3	X+	3	X+						
16WW01	5/4/93	0:00	0	Primary			3	X+			3	X+	3	X+						
16WW01	5/4/93	0:00	0	Duplicate 1			2.9	X+			2.9	X+	2.9	X+						
16WW01-A				Avg.			2.95				2.95		2.95							
16WW02	5/12/93	0:00	0	Primary			2.9	X+			2.9	X+	2.9	X+						
16WW02	5/12/93	0:00	0	Duplicate 1			2.95	X+			2.95	X+	2.95	X+						
16WW02-A				Avg.			2.925				2.925		2.925							
16WW03	5/4/93	0:00	0	Primary			3.15	X+			3.15	X+	3.15	X+						
16WW04	5/12/93	0:00	0	Primary			3	X+			3	X+	3	X+						
16WW04	5/12/93	0:00	0	Duplicate 1			3.15	X+			3.15	X+	3.15	X+						
16WW04-A				Avg.			3.075				3.075		3.075							
16WW05	5/4/93	0:00	0	Primary			3.4	X+			3.4	X+	3.4	X+						
16WW06	5/5/93	0:00	0	Primary			3.25	X+			3.25	X+	3.25	X+						
16WW27	5/17/97	9:15	0	Primary			3	X+	65	X+	3	X+	3	X+	3	X+	650	X+	3	X+
16WW27	5/17/97	10:17	1	Primary			3	X+	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+
16WW27-A				Avg.			3		62.5		3		3		3		625		3	
16WW31	5/29/97	8:27	0	Primary			3	X+	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+
16WW31	5/29/97	8:55	1	Primary			3	X+	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+
16WW31-A				Avg.			3		60		3		3		3		600		3	
16WW35	6/25/97	9:50	0	Primary			2.5	X+	55	X+	2.5	X+	2.5	X+	2.5	X+	550	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate 1			2.5	X+	50	X+	2.5	X+	2.5	X+	2.5	X+	500	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate 2			2.5	X+	50	X+	2.5	X+	2.5	X+	2.5	X+	500	X+	2.5	X+
16WW35	6/25/97	10:05	1	Primary			3	X+	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	CEVETH (ug/kg)	trans-1,2-Dichloroethene (ug/kg)		Propionitrile (ug/kg)		Ethyl methacrylate (ug/kg)		Ethylbenzene (ug/kg)		Hexachloro butadiene (ug/kg)		Isobutyl alcohol (ug/kg)		1,3-Dichloro benzene (ug/kg)	
16WW35-A				Avg.		2.625		53.75		2.625		2.625		2.625		537.5		2.625	
16WW37	5/31/97	15:55	0	Primary		3	X+	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+
16WW37	5/31/97	16:10	1	Primary		3	X+	65	X+	3	X+	3	X+	3	X+	650	X+	3	X+
16WW37-A				Avg.		3		62.5		3		3		3		625		3	
NATURAL LOGS																			
16SD01-A						1.459				1.482		1.482							
16SD02-A						1.147				1.201		1.201							
16SD03-A						1.179				1.288		1.288							
16SD06					2.015					1.253		1.253							
16SD07					2.079					1.386		1.386							
16SD08					1.872					1.253		1.253							
16SD09					1.872					1.253		1.253							
16SD10					1.872					1.253		1.253							
16SD11					1.872					1.099		1.099							
16WW01-A						1.082				1.082		1.082							
16WW02-A						1.073				1.073		1.073							
16WW03						1.147				1.147		1.147							
16WW04-A						1.123				1.123		1.123							
16WW05						1.224				1.224		1.224							
16WW06						1.179				1.179		1.179							
16WW27-A						1.099		4.135		1.099		1.099		1.099		6.438		1.099	
16WW31-A						1.099		4.094		1.099		1.099		1.099		6.397		1.099	
16WW35-A						0.965		3.984		0.965		0.965		0.965		6.287		0.965	
16WW37-A						1.099		4.135		1.099		1.099		1.099		6.438		1.099	
STATISTICS																			
Total No.					6	13		4		19		19		4		4		4	
Min.					6.5	2.5		50		2.5		2.5		2.5		500		2.5	
Max.					9	4.3		65		4.5		4.5		3		650		3	
Avg.(log transformed)					1.93	1.144		4.087		1.187		1.187		1.066		6.39		1.066	
St. Dev.(log transformed)					0.093	0.114		0.071		0.122		0.122		0.067		0.071		0.067	
H																			
95th UCL of Mean					6.92	3.16		59.71		3.30		3.30		2.91		597.36		2.91	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Methacrylo nitrile (ug/kg)		Bromomethane (ug/kg)		Chloromethane (ug/kg)		2-Butanone (ug/kg)		Iodomethane (ug/kg)		4-Methyl-2-pentanone (ug/kg)		Methyl methacrylate (ug/kg)		Dibromomethan (ug/kg)	
16SD01	5/2/93	0:00	0	Primary			8.5	X+	8.5	X+	8.5	X+	8.5	X+	8.5	X+			4.3	X+
16SD01	2/21/95	14:20	0	Primary			9	X+	9	X+	9	X+	9	X+	9	X+			4.5	X+
16SD01-A				Avg.			8.75		8.75		8.75		8.75		8.75				4.4	
16SD02	4/14/93	0:00	0	Primary			6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.15	X+
16SD02	2/28/95	10:00	0	Primary			7	X+	7	X+	7	X+	7	X+	7	X+			3.5	X+
16SD02-A				Avg.			6.75		6.75		6.75		6.75		6.75				3.325	
16SD03	4/14/93	0:00	0	Primary			6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.25	X+
16SD03	2/20/95	16:05	0	Primary			8	X+	8	X+	8	X+	8	X+	8	X+			4	X+
16SD03-A				Avg.			7.25		7.25		7.25		7.25		7.25				3.625	
16SD06	2/20/95	16:35	0	Primary			7.5	X+	7.5	X+	7.5	X+	7.5	X+	7.5	X+			3.5	X+
16SD07	2/21/95	14:05	0	Primary			8	X+	8	X+	8	X+	8	X+	8	X+			4	X+
16SD08	2/21/95	9:30	0	Primary			6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.5	X+
16SD09	2/21/95	10:55	0	Primary			6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.5	X+
16SD10	2/21/95	17:10	0	Primary			6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.5	X+
16SD11	2/28/95	11:15	0	Primary			6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3	X+
16WW01	5/4/93	0:00	0	Primary			6	X+	6	X+	6	X+	6	X+	6	X+			3	X+
16WW01	5/4/93	0:00	0	Duplicate 1			6	X+	6	X+	6	X+	6	X+	6	X+			2.9	X+
16WW01-A				Avg.			6		6		6		6		6				2.95	
16WW02	5/12/93	0:00	0	Primary			6	X+	6	X+	6	X+	6	X+	6	X+			2.9	X+
16WW02	5/12/93	0:00	0	Duplicate 1			6	X+	6	X+	6	X+	6	X+	6	X+			2.95	X+
16WW02-A				Avg.			6		6		6		6		6				2.925	
16WW03	5/4/93	0:00	0	Primary			6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.15	X+
16WW04	5/12/93	0:00	0	Primary			6	X+	6	X+	6	X+	6	X+	6	X+			3	X+
16WW04	5/12/93	0:00	0	Duplicate 1			6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.15	X+
16WW04-A				Avg.			6.25		6.25		6.25		6.25		6.25				3.075	
16WW05	5/4/93	0:00	0	Primary			7	X+	7	X+	7	X+	7	X+	7	X+			3.4	X+
16WW06	5/5/93	0:00	0	Primary			6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.25	X+
16WW27	5/17/97	9:15	0	Primary	65	X+	6.5	X+	6.5	X+	13	X+	3	X+	13	X+	32	X+	6.5	X+
16WW27	5/17/97	10:17	1	Primary	60	X+	6	X+	6	X+	12	X+	3	X+	12	X+	30	X+	6	X+
16WW27-A				Avg.	62.5		6.25		6.25		12.5		3		12.5		31		6.25	
16WW31	5/29/97	8:27	0	Primary	60	X+	6	X+	6	X+	12	X+	3	X+	12	X+	30	X+	6	X+
16WW31	5/29/97	8:55	1	Primary	60	X+	6	X+	6	X+	12	X+	3	X+	12	X+	30.5	X+	6	X+
16WW31-A				Avg.	60		6		6		12		3		12		30.25		6	
16WW35	6/25/97	9:50	0	Primary	55	X+	5.5	X+	5.5	X+	10.5	X+	2.5	X+	10.5	X+	26.5	X+	5.5	X+
16WW35	6/25/97	9:50	0	Duplicate 1	50	X+	5	X+	5	X+	10	X+	2.5	X+	10	X+	25	X+	5	X+
16WW35	6/25/97	0:00	0	Duplicate 2	50	X+	5	X+	5	X+	10	X+	2.5	X+	10	X+	25	X+	5	X+
16WW35	6/25/97	10:05	1	Primary	60	X+	6	X+	6	X+	12.5	X+	3	X+	12.5	X+	31	X+	6	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Methacrylonitrile (ug/kg)		Bromomethane (ug/kg)		Chloromethane (ug/kg)		2-Butanone (ug/kg)		Iodomethane (ug/kg)		4-Methyl-2-pentanone (ug/kg)		Methyl methacrylate (ug/kg)		Dibromomethan (ug/kg)	
16WW35-A				Avg.	53.75		5.375		5.375		10.75		2.625		10.75		26.875		5.375	
16WW37	5/31/97	15:55	0	Primary	60	X+	6	X+	6	X+	12.5	X+	3	X+	12.5	X+	31	X+	6	X+
16WW37	5/31/97	16:10	1	Primary	65	X+	6.5	X+	6.5	X+	12.5	X+	3	X+	12.5	X+	31	X+	6.5	X+
16WW37-A				Avg.	62.5		6.25		6.25		12.5		3		12.5		31		6.25	
NATURAL LOGS																				
16SD01-A							2.169		2.169		2.169		2.169		2.169				1.482	
16SD02-A							1.91		1.91		1.91		1.91		1.91				1.201	
16SD03-A							1.981		1.981		1.981		1.981		1.981				1.288	
16SD06							2.015		2.015		2.015		2.015		2.015				1.253	
16SD07							2.079		2.079		2.079		2.079		2.079				1.386	
16SD08							1.872		1.872		1.872		1.872		1.872				1.253	
16SD09							1.872		1.872		1.872		1.872		1.872				1.253	
16SD10							1.872		1.872		1.872		1.872		1.872				1.253	
16SD11							1.872		1.872		1.872		1.872		1.872				1.099	
16WW01-A							1.792		1.792		1.792		1.792		1.792				1.082	
16WW02-A							1.792		1.792		1.792		1.792		1.792				1.073	
16WW03							1.872		1.872		1.872		1.872		1.872				1.147	
16WW04-A							1.833		1.833		1.833		1.833		1.833				1.123	
16WW05							1.946		1.946		1.946		1.946		1.946				1.224	
16WW06							1.872		1.872		1.872		1.872		1.872				1.179	
16WW27-A					4.135		1.833		1.833		2.526		1.099		2.526		3.434		1.833	
16WW31-A					4.094		1.792		1.792		2.485		1.099		2.485		3.409		1.792	
16WW35-A					3.984		1.682		1.682		2.375		0.965		2.375		3.291		1.682	
16WW37-A					4.135		1.833		1.833		2.526		1.099		2.526		3.434		1.833	
STATISTICS																				
Total No.					4		19		19		19		19		19		4		19	
Min.					50		5		5		6		2.5		6		25		2.9	
Max.					65		9		9		13		9		13		32		6.5	
Avg.(log transformed)					4.087		1.889		1.889		2.035		1.737		2.035		3.392		1.339	
St. Dev.(log transformed)					0.071		0.112		0.112		0.254		0.369		0.254		0.068		0.258	
H																				
95th UCL of Mean					59.71		6.65		6.65		7.90		6.08		7.90		29.79		3.94	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Methylene chloride (ug/kg)		Naphthalene (ug/kg)		n-Butylbenzene (ug/kg)		1-Propanamine, N-nitroso-n-pro (ug/kg)		n-Propyl benzene (ug/kg)		2-Chlortoluene (ug/kg)		1,2-Dichloro benzene (ug/kg)		o-Xylene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	4.3	X+														
16SD01	2/21/95	14:20	0	Primary	9	X+														
16SD01-A				Avg.	6.65															
16SD02	4/14/93	0:00	0	Primary	3.15	X+														
16SD02	2/28/95	10:00	0	Primary	7	X+														
16SD02-A				Avg.	5.075															
16SD03	4/14/93	0:00	0	Primary	3.25	X+														
16SD03	2/20/95	16:05	0	Primary	8	X+														
16SD03-A				Avg.	5.625															
16SD06	2/20/95	16:35	0	Primary	7.5	X+														
16SD07	2/21/95	14:05	0	Primary	8	X+														
16SD08	2/21/95	9:30	0	Primary	6.5	X+														
16SD09	2/21/95	10:55	0	Primary	6.5	X+														
16SD10	2/21/95	17:10	0	Primary	6.5	X+														
16SD11	2/28/95	11:15	0	Primary	6.5	X+														
16WW01	5/4/93	0:00	0	Primary	3	X+														
16WW01	5/4/93	0:00	0	Duplicate 1	2.9	X+														
16WW01-A				Avg.	2.95															
16WW02	5/12/93	0:00	0	Primary	6.5															
16WW02	5/12/93	0:00	0	Duplicate 1	2.95	X+														
16WW02-A				Avg.	4.725															
16WW03	5/4/93	0:00	0	Primary	3.15	X+														
16WW04	5/12/93	0:00	0	Primary	3	X+														
16WW04	5/12/93	0:00	0	Duplicate 1	3.15	X+														
16WW04-A				Avg.	3.075															
16WW05	5/4/93	0:00	0	Primary	3.4	X+														
16WW06	5/5/93	0:00	0	Primary	3.25	X+														
16WW27	5/17/97	9:15	0	Primary	2	U+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW27	5/17/97	10:17	1	Primary	1	U+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW27-A				Avg.	1.25		3		3		3		3		3		3		3	
16WW31	5/29/97	8:27	0	Primary	5		3	X+	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+
16WW31	5/29/97	8:55	1	Primary	10		3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31-A				Avg.	7.5		3		3		3		3		3		4.5		3	
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate 1	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate 2	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	10:05	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Methylene chloride (ug/kg)		Naphthalene (ug/kg)		n-Butylbenzene (ug/kg)		1-Propanamine, N-nitroso-n-pro (ug/kg)		n-Propyl benzene (ug/kg)		2-Chlortoluene (ug/kg)		1,2-Dichloro benzene (ug/kg)		o-Xylene (ug/kg)	
16WW35-A				Avg.	2.625		2.625		2.625		2.625		2.625		2.625		2.625		2.625	
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37	5/31/97	16:10	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37-A				Avg.	3		3		3		3		3		3		3		3	
NATURAL LOGS																				
16SD01-A					1.895															
16SD02-A					1.624															
16SD03-A					1.727															
16SD06					2.015															
16SD07					2.079															
16SD08					1.872															
16SD09					1.872															
16SD10					1.872															
16SD11					1.872															
16WW01-A					1.082															
16WW02-A					1.553															
16WW03					1.147															
16WW04-A					1.123															
16WW05					1.224															
16WW06					1.179															
16WW27-A					0.223		1.099		1.099		1.099		1.099		1.099		1.099		1.099	
16WW31-A					2.015		1.099		1.099		1.099		1.099		1.099		1.504		1.099	
16WW35-A					0.965		0.965		0.965		0.965		0.965		0.965		0.965		0.965	
16WW37-A					1.099		1.099		1.099		1.099		1.099		1.099		1.099		1.099	
STATISTICS																				
Total No.					19		4		4		4		4		4		4		4	
Min.					1		2.5		2.5		2.5		2.5		2.5		2.5		2.5	
Max.					10		3		3		3		3		3		6		3	
Avg.(log transformed)					1.497		1.066		1.066		1.066		1.066		1.066		1.167		1.066	
St. Dev.(log transformed)					0.491		0.067		0.067		0.067		0.067		0.067		0.234		0.067	
H					2.001															
95th UCL of Mean					6.35		2.91		2.91		2.91		2.91		2.91		3.30		2.91	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	4-Chlorotoluene (ug/kg)		p-Isopropyl toluene (ug/kg)		1,4-Dichloro benzene (ug/kg)		Pentachloro ethane (ug/kg)		sec-Butyl benzene (ug/kg)		Styrene (ug/kg)		tert-Butyl benzene (ug/kg)		Tetrachloro ethene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary											4.3	X+			4.3	X+
16SD01	2/21/95	14:20	0	Primary											4.5	X+			4.5	X+
16SD01-A				Avg.											4.4				4.4	
16SD02	4/14/93	0:00	0	Primary											3.15	X+			3.15	X+
16SD02	2/28/95	10:00	0	Primary											3.5	X+			3.5	X+
16SD02-A				Avg.											3.325				3.325	
16SD03	4/14/93	0:00	0	Primary											3.25	X+			3.25	X+
16SD03	2/20/95	16:05	0	Primary											4	X+			4	X+
16SD03-A				Avg.											3.625				3.625	
16SD06	2/20/95	16:35	0	Primary											3.5	X+			3.5	X+
16SD07	2/21/95	14:05	0	Primary											4	X+			4	X+
16SD08	2/21/95	9:30	0	Primary											93				3.5	X+
16SD09	2/21/95	10:55	0	Primary											3.5	X+			3.5	X+
16SD10	2/21/95	17:10	0	Primary											3.5	X+			3.5	X+
16SD11	2/28/95	11:15	0	Primary											3	X+			3	X+
16WW01	5/4/93	0:00	0	Primary											3	X+			3	X+
16WW01	5/4/93	0:00	0	Duplicate 1											2.9	X+			2.9	X+
16WW01-A				Avg.											2.95				2.95	
16WW02	5/12/93	0:00	0	Primary											2.9	X+			2.9	X+
16WW02	5/12/93	0:00	0	Duplicate 1											2.95	X+			2.95	X+
16WW02-A				Avg.											2.925				2.925	
16WW03	5/4/93	0:00	0	Primary											3.15	X+			3.15	X+
16WW04	5/12/93	0:00	0	Primary											3	X+			3	X+
16WW04	5/12/93	0:00	0	Duplicate 1											3.15	X+			3.15	X+
16WW04-A				Avg.											3.075				3.075	
16WW05	5/4/93	0:00	0	Primary											3.4	X+			3.4	X+
16WW06	5/5/93	0:00	0	Primary											3.25	X+			3.25	X+
16WW27	5/17/97	9:15	0	Primary	3	X+	3	X+	3	X+	6.5	X+	3	X+	3	X+	3	X+	3	X+
16WW27	5/17/97	10:17	1	Primary	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+	3	X+	3	X+
16WW27-A				Avg.	3		3		3		6.25		3		3		3		3	
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	8:55	1	Primary	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+	3	X+	3	X+
16WW31-A				Avg.	3		3		3		6		3		3		3		3	
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	2.5	X+	5.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate 1	2.5	X+	2.5	X+	2.5	X+	5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate 2	2.5	X+	2.5	X+	2.5	X+	5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	10:05	1	Primary	3	X+	3	X+	3	X+	6	X+	3	X+	2		3	X+	3	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	4-Chlorotoluene (ug/kg)		p-Isopropyl toluene (ug/kg)		1,4-Dichloro benzene (ug/kg)		Pentachloro ethane (ug/kg)		sec-Butyl benzene (ug/kg)		Styrene (ug/kg)		tert-Butyl benzene (ug/kg)		Tetrachloro ethene (ug/kg)	
16WW35-A				Avg.	2.625		2.625		2.625		5.375		2.625		2.375		2.625		2.625	
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+	3	X+	3	X+
16WW37	5/31/97	16:10	1	Primary	3	X+	3	X+	3	X+	6.5	X+	3	X+	3	X+	3	X+	3	X+
16WW37-A				Avg.	3		3		3		6.25		3		3		3		3	
NATURAL LOGS																				
16SD01-A															1.482				1.482	
16SD02-A															1.201				1.201	
16SD03-A															1.288				1.288	
16SD06															1.253				1.253	
16SD07															1.386				1.386	
16SD08															4.533				1.253	
16SD09															1.253				1.253	
16SD10															1.253				1.253	
16SD11															1.099				1.099	
16WW01-A															1.082				1.082	
16WW02-A															1.073				1.073	
16WW03															1.147				1.147	
16WW04-A															1.123				1.123	
16WW05															1.224				1.224	
16WW06															1.179				1.179	
16WW27-A					1.099		1.099		1.099		1.833		1.099		1.099		1.099		1.099	
16WW31-A					1.099		1.099		1.099		1.792		1.099		1.099		1.099		1.099	
16WW35-A					0.965		0.965		0.965		1.682		0.965		0.865		0.965		0.965	
16WW37-A					1.099		1.099		1.099		1.833		1.099		1.099		1.099		1.099	
STATISTICS																				
Total No.					4		4		4		4		4		19		4		19	
Min.					2.5		2.5		2.5		5		2.5		2		2.5		2.5	
Max.					3		3		3		6.5		3		93		3		4.5	
Avg.(log transformed)					1.066		1.066		1.066		1.785		1.066		1.355		1.066		1.187	
St. Dev.(log transformed)					0.067		0.067		0.067		0.071		0.067		0.781		0.067		0.122	
H															2.319					
95th UCL of Mean					2.91		2.91		2.91		5.97		2.91		8.06		2.91		3.30	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Toluene (ug/kg)		Trans-1,3-Dichloropropene (ug/kg)		Trichloroethene (ug/kg)		Trichloro fluoromethane (ug/kg)		Vinyl Acetate (ug/kg)		Vinyl chloride (ug/kg)		Xylene (total) (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	4.3	X+	4.3	X+	4.3	X+	4.3	X+	4.3	X+	8.5	X+	4.3	X+
16SD01	2/21/95	14:20	0	Primary	4.5	X+	4.5	X+	4.5	X+	4.5	X+	9	X+	9	X+	4.5	X+
16SD01-A				Avg.	4.4		4.4		4.4		4.4		6.65		8.75		4.4	
16SD02	4/14/93	0:00	0	Primary	3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+	6.5	X+	3.15	X+
16SD02	2/28/95	10:00	0	Primary	3.5	X+	3.5	X+	3.5	X+	3.5	X+	7	X+	7	X+	3.5	X+
16SD02-A				Avg.	3.325		3.325		3.325		3.325		5.075		6.75		3.325	
16SD03	4/14/93	0:00	0	Primary	3.25	X+	3.25	X+	3.25	X+	3.25	X+	3.25	X+	6.5	X+	3.25	X+
16SD03	2/20/95	16:05	0	Primary	4	X+	4	X+	4	X+	4	X+	8	X+	8	X+	4	X+
16SD03-A				Avg.	3.625		3.625		3.625		3.625		5.625		7.25		3.625	
16SD06	2/20/95	16:35	0	Primary	3.5	X+	3.5	X+	3.5	X+	3.5	X+	7.5	X+	7.5	X+	3.5	X+
16SD07	2/21/95	14:05	0	Primary	4	X+	4	X+	4	X+	4	X+	8	X+	8	X+	4	X+
16SD08	2/21/95	9:30	0	Primary	3.5	X+	3.5	X+	3.5	X+	3.5	X+	6.5	X+	6.5	X+	3.5	X+
16SD09	2/21/95	10:55	0	Primary	3.5	X+	3.5	X+	3.5	X+	3.5	X+	6.5	X+	6.5	X+	3.5	X+
16SD10	2/21/95	17:10	0	Primary	3.5	X+	3.5	X+	3.5	X+	3.5	X+	6.5	X+	6.5	X+	3.5	X+
16SD11	2/28/95	11:15	0	Primary	3	X+	3	X+	3	X+	3	X+	6.5	X+	6.5	X+	3	X+
16WW01	5/4/93	0:00	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+
16WW01	5/4/93	0:00	0	Duplicate 1	2.9	X+	2.9	X+	2.9	X+	2.9	X+	2.9	X+	6	X+	2.9	X+
16WW01-A				Avg.	2.95		2.95		2.95		2.95		2.95		6		2.95	
16WW02	5/12/93	0:00	0	Primary	2.9	X+	2.9	X+	2.9	X+	2.9	X+	2.9	X+	6	X+	2.9	X+
16WW02	5/12/93	0:00	0	Duplicate 1	2.95	X+	2.95	X+	2.95	X+	2.95	X+	2.95	X+	6	X+	2.95	X+
16WW02-A				Avg.	2.925		2.925		2.925		2.925		2.925		6		2.925	
16WW03	5/4/93	0:00	0	Primary	3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+	6.5	X+	3.15	X+
16WW04	5/12/93	0:00	0	Primary	3	X+	3	X+	65		3	X+	3	X+	6	X+	3	X+
16WW04	5/12/93	0:00	0	Duplicate 1	3.15	X+	3.15	X+	220		3.15	X+	3.15	X+	6.5	X+	3.15	X+
16WW04-A				Avg.	3.075		3.075		142.5		3.075		3.075		6.25		3.075	
16WW05	5/4/93	0:00	0	Primary	3.4	X+	3.4	X+	3.4	X+	3.4	X+	3.4	X+	7	X+	3.4	X+
16WW06	5/5/93	0:00	0	Primary	3.25	X+	3.25	X+	3.25	X+	3.25	X+	3.25	X+	6.5	X+	3.25	X+
16WW27	5/17/97	9:15	0	Primary	3	X+	3	X+	6.5	X+	6.5	X+	13	X+	6.5	X+	3	X+
16WW27	5/17/97	10:17	1	Primary	3	X+	3	X+	6	X+	6	X+	12	X+	6	X+	3	X+
16WW27-A				Avg.	3		3		6.25		6.25		12.5		6.25		3	
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	6	X+	6	X+	12	X+	6	X+	3	X+
16WW31	5/29/97	8:55	1	Primary	3	X+	3	X+	6	X+	6	X+	12	X+	6	X+	3	X+
16WW31-A				Avg.	3		3		6		6		12		6		3	
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	5.5	X+	5.5	X+	10.5	X+	5.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate 1	2.5	X+	2.5	X+	5	X+	5	X+	10	X+	5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate 2	2.5	X+	2.5	X+	5	X+	5	X+	10	X+	5	X+	2.5	X+
16WW35	6/25/97	10:05	1	Primary	3	X+	3	X+	6	X+	6	X+	12.5	X+	6	X+	3	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Toluene (ug/kg)		Trans-1,3-Dichloropropene (ug/kg)		Trichloroethene (ug/kg)		Trichloro fluoromethane (ug/kg)		Vinyl Acetate (ug/kg)		Vinyl chloride (ug/kg)		Xylene (total) (ug/kg)	
16WW35-A				Avg.	2.625		2.625		5.375		5.375		10.75		5.375		2.625	
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	6	X+	6	X+	12.5	X+	6	X+	3	X+
16WW37	5/31/97	16:10	1	Primary	3	X+	3	X+	6.5	X+	6.5	X+	12.5	X+	6.5	X+	3	X+
16WW37-A				Avg.	3		3		6.25		6.25		12.5		6.25		3	
NATURAL LOGS																		
16SD01-A					1.482		1.482		1.482		1.482		1.895		2.169		1.482	
16SD02-A					1.201		1.201		1.201		1.201		1.624		1.91		1.201	
16SD03-A					1.288		1.288		1.288		1.288		1.727		1.981		1.288	
16SD06					1.253		1.253		1.253		1.253		2.015		2.015		1.253	
16SD07					1.386		1.386		1.386		1.386		2.079		2.079		1.386	
16SD08					1.253		1.253		1.253		1.253		1.872		1.872		1.253	
16SD09					1.253		1.253		1.253		1.253		1.872		1.872		1.253	
16SD10					1.253		1.253		1.253		1.253		1.872		1.872		1.253	
16SD11					1.099		1.099		1.099		1.099		1.872		1.872		1.099	
16WW01-A					1.082		1.082		1.082		1.082		1.082		1.792		1.082	
16WW02-A					1.073		1.073		1.073		1.073		1.073		1.792		1.073	
16WW03					1.147		1.147		1.147		1.147		1.147		1.872		1.147	
16WW04-A					1.123		1.123		4.959		1.123		1.123		1.833		1.123	
16WW05					1.224		1.224		1.224		1.224		1.224		1.946		1.224	
16WW06					1.179		1.179		1.179		1.179		1.179		1.872		1.179	
16WW27-A					1.099		1.099		1.833		1.833		2.526		1.833		1.099	
16WW31-A					1.099		1.099		1.792		1.792		2.485		1.792		1.099	
16WW35-A					0.965		0.965		1.682		1.682		2.375		1.682		0.965	
16WW37-A					1.099		1.099		1.833		1.833		2.526		1.833		1.099	
STATISTICS																		
Total No.					19		19		19		19		19		19		19	
Min.					2.5		2.5		2.9		2.9		2.9		5		2.5	
Max.					4.5		4.5		220		6.5		13		9		4.5	
Avg.(log transformed)					1.187		1.187		1.541		1.339		1.767		1.889		1.187	
St. Dev.(log transformed)					0.122		0.122		0.866		0.258		0.509		0.112		0.122	
H									2.428									
95th UCL of Mean					3.30		3.30		11.15		3.94		6.66		6.65		3.30	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	1,2,4-Trichloro benzene (U+g/kg)		1-Chloro naphthalene (U+g/kg)		2,4,5-Trichloro phenol (U+g/kg)		2,4,6-Trichloro phenol (U+g/kg)		2,4-Dichloro phenol (U+g/kg)		2,4-Dimethyl phenol (U+g/kg)		2,4-Dinitro phenol (U+g/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	569	X+	1379.5	X+	284.5	X+	284.5	X+	284.5	X+	1379.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	417.5	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	428.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+	1039	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	393	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+	952.5	X+
16WW01	5/4/93	0:00	0	Duplicate 1	192	X+	383.5	X+	930	X+	192	X+	192	X+	192	X+	930	X+
16WW01-A				Avg.	194.25		388.25		941.25		194.25		194.25		194.25		941.25	
16WW02	5/12/93	0:00	0	Primary	192	X+	383.5	X+	930	X+	192	X+	192	X+	192	X+	930	X+
16WW02	5/12/93	0:00	0	Duplicate 1	194	X+	388	X+	941	X+	194	X+	194	X+	194	X+	941	X+
16WW02-A				Avg.	193		385.75		935.5		193		193		193		935.5	
16WW03	5/4/93	0:00	0	Primary	206.5	X+	412.5	X+	1000	X+	206.5	X+	206.5	X+	206.5	X+	1000	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	397.5	X+	964	X+	199	X+	199	X+	199	X+	964	X+
16WW04	5/12/93	0:00	0	Duplicate 1	209	X+	417.5	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+
16WW04-A				Avg.	204		407.5		988.25		204		204		204		988.25	
16WW05	5/4/93	0:00	0	Primary	223	X+	446	X+	1081	X+	223	X+	223	X+	223	X+	1081	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	428.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+	1039	X+
NATURAL LOG																		
16SD01					5.651		6.344		7.229		5.651		5.651		5.651		7.229	
16SD02					5.342		6.034		6.92		5.342		5.342		5.342		6.92	
16SD03					5.368		6.06		6.946		5.368		5.368		5.368		6.946	
16WW01-A					5.269		5.962		6.847		5.269		5.269		5.269		6.847	
16WW02-A					5.263		5.955		6.841		5.263		5.263		5.263		6.841	
16WW03					5.33		6.022		6.908		5.33		5.33		5.33		6.908	
16WW04-A					5.318		6.01		6.896		5.318		5.318		5.318		6.896	
16WW05					5.407		6.1		6.986		5.407		5.407		5.407		6.986	
16WW06					5.368		6.06		6.946		5.368		5.368		5.368		6.946	
STATISTICS																		
Total No.					9		9		9		9		9		9		9	
Min.					192		383.5		930		192		192		192		930	
Max.					284.5		569		1379.5		284.5		284.5		284.5		1379.5	
Avg. (log transformed)					5.368		6.061		6.947		5.368		5.368		5.368		6.947	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	1,2,4-Trichloro benzene (U+g/kg)	1-Chloro naphthalene (U+g/kg)	2,4,5-Trichloro phenol (U+g/kg)	2,4,6-Trichloro phenol (U+g/kg)	2,4-Dichloro phenol (U+g/kg)	2,4-Dimethyl phenol (U+g/kg)	2,4-Dinitro phenol (U+g/kg)
St. Dev.(log transformed)					0.116	0.116	0.116	0.116	0.116	0.116	0.116
H											
95th UCL of Mean					215.88	431.70	1047.05	215.88	215.88	215.88	1047.05

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	2,4-Dinitro tolU+ene (U+g/kg)		2,6-Dinitro tolU+ene (U+g/kg)		2-Chloro napthalene (U+g/kg)		2-Chlorophenol (U+g/kg)		2-Methyl napthalene (U+g/kg)		3,3'-Dichloro benzidine (U+g/kg)		4,6-Dinitro-2-methylphenol (U+g/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	284.5	X+	284.5	X+	284.5	X+	284.5	X+	569	X+	1379.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	209	X+	209	X+	209	X+	209	X+	417.5	X+	1012.5	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	428.5	X+	1039	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	393	X+	952.5	X+
16WW01	5/4/93	0:00	0	Duplicate 1	192	X+	192	X+	192	X+	192	X+	192	X+	383.5	X+	930	X+
16WW01-A				Avg.	194.25		194.25		194.25		194.25		194.25		388.25		941.25	
16WW02	5/12/93	0:00	0	Primary	192	X+	192	X+	192	X+	192	X+	192	X+	383.5	X+	930	X+
16WW02	5/12/93	0:00	0	Duplicate 1	194	X+	194	X+	194	X+	194	X+	194	X+	388	X+	941	X+
16WW02-A				Avg.	193		193		193		193		193		385.75		935.5	
16WW03	5/4/93	0:00	0	Primary	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+	412.5	X+	1000	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	199	X+	199	X+	199	X+	199	X+	397.5	X+	964	X+
16WW04	5/12/93	0:00	0	Duplicate 1	209	X+	209	X+	209	X+	209	X+	209	X+	417.5	X+	1012.5	X+
16WW04-A				Avg.	204		204		204		204		204		407.5		988.25	
16WW05	5/4/93	0:00	0	Primary	223	X+	223	X+	223	X+	223	X+	223	X+	446	X+	1081	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	428.5	X+	1039	X+
NATURAL LOG																		
16SD01					5.651		5.651		5.651		5.651		5.651		6.344		7.229	
16SD02					5.342		5.342		5.342		5.342		5.342		6.034		6.92	
16SD03					5.368		5.368		5.368		5.368		5.368		6.06		6.946	
16WW01-A					5.269		5.269		5.269		5.269		5.269		5.962		6.847	
16WW02-A					5.263		5.263		5.263		5.263		5.263		5.955		6.841	
16WW03					5.33		5.33		5.33		5.33		5.33		6.022		6.908	
16WW04-A					5.318		5.318		5.318		5.318		5.318		6.01		6.896	
16WW05					5.407		5.407		5.407		5.407		5.407		6.1		6.986	
16WW06					5.368		5.368		5.368		5.368		5.368		6.06		6.946	
STATISTICS																		
Total No.					9		9		9		9		9		9		9	
Min.					192		192		192		192		192		383.5		930	
Max.					284.5		284.5		284.5		284.5		284.5		569		1379.5	
Avg. (log transformed)					5.368		5.368		5.368		5.368		5.368		6.061		6.947	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	2,4-Dinitro tolU+ene (U+g/kg)	2,6-Dinitro tolU+ene (U+g/kg)	2-Chloro napthalene (U+g/kg)	2-Chlorophenol (U+g/kg)	2-Methyl napthalene (U+g/kg)	3,3'-Dichloro benzidine (U+g/kg)	4,6-Dinitro-2-methylphenol (U+g/kg)
St. Dev.(log transformed)					0.116	0.116	0.116	0.116	0.116	0.116	0.116
H											
95th UCL of Mean					215.88	215.88	215.88	215.88	215.88	431.70	1047.05

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	4-Bromophenyl phenyl ether (U+g/kg)		4-Chlorophenyl phenyl ether (U+g/kg)		Acenaphthene (U+g/kg)		Acenaphthylene (U+g/kg)		Anthracene (U+g/kg)		Benzo(a) anthracene (U+g/kg)		Benzo(a)pyrene (U+g/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	284.5	X+	284.5	X+	284.5	X+	284.5	X+	284.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate 1	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+
16WW01-A				Avg.	194.25		194.25		194.25		194.25		194.25		194.25		194.25	
16WW02	5/12/93	0:00	0	Primary	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate 1	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+
16WW02-A				Avg.	193		193		193		193		193		193		193	
16WW03	5/4/93	0:00	0	Primary	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	199	X+	199	X+	199	X+	199	X+	199	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate 1	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+
16WW04-A				Avg.	204		204		204		204		204		204		204	
16WW05	5/4/93	0:00	0	Primary	223	X+	223	X+	223	X+	223	X+	223	X+	223	X+	223	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+
NATURAL LOG																		
16SD01					5.651		5.651		5.651		5.651		5.651		5.651		5.651	
16SD02					5.342		5.342		5.342		5.342		5.342		5.342		5.342	
16SD03					5.368		5.368		5.368		5.368		5.368		5.368		5.368	
16WW01-A					5.269		5.269		5.269		5.269		5.269		5.269		5.269	
16WW02-A					5.263		5.263		5.263		5.263		5.263		5.263		5.263	
16WW03					5.33		5.33		5.33		5.33		5.33		5.33		5.33	
16WW04-A					5.318		5.318		5.318		5.318		5.318		5.318		5.318	
16WW05					5.407		5.407		5.407		5.407		5.407		5.407		5.407	
16WW06					5.368		5.368		5.368		5.368		5.368		5.368		5.368	
STATISTICS																		
Total No.					9		9		9		9		9		9		9	
Min.					192		192		192		192		192		192		192	
Max.					284.5		284.5		284.5		284.5		284.5		284.5		284.5	
Avg. (log transformed)					5.368		5.368		5.368		5.368		5.368		5.368		5.368	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	4-Bromophenyl phenyl ether (U+g/kg)	4-Chlorophenyl phenyl ether (U+g/kg)	Acenaphthene (U+g/kg)	Acenaphthylene (U+g/kg)	Anthracene (U+g/kg)	Benzo(a) anthracene (U+g/kg)	Benzo(a)pyrene (U+g/kg)
St. Dev.(log transformed)					0.116	0.116	0.116	0.116	0.116	0.116	0.116
H											
95th UCL of Mean					215.88	215.88	215.88	215.88	215.88	215.88	215.88

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	Benzo(b) flU+oranthene (U+g/kg)		Benzo(g,h,i) perylene (U+g/kg)		Benzo(k)flU+or anthene (U+g/kg)		Benzoic acid (U+g/kg)		Benzyl alcohol (U+g/kg)		Bis(2-chloro-1-methylethyl) ether (U+g/kg)		Bis(2-chloro ethoX+y) metha (U+g/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	284.5	X+	284.5	X+	1379.5	X+	284.5	X+	284.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	209	X+	209	X+	1012.5	X+	209	X+	209	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate 1	192	X+	192	X+	192	X+	930	X+	192	X+	192	X+	192	X+
16WW01-A				Avg.	194.25		194.25		194.25		941.25		194.25		194.25		194.25	
16WW02	5/12/93	0:00	0	Primary	192	X+	192	X+	192	X+	930	X+	192	X+	192	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate 1	194	X+	194	X+	194	X+	941	X+	194	X+	194	X+	194	X+
16WW02-A				Avg.	193		193		193		935.5		193		193		193	
16WW03	5/4/93	0:00	0	Primary	206.5	X+	206.5	X+	206.5	X+	1000	X+	206.5	X+	206.5	X+	206.5	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	199	X+	199	X+	964	X+	199	X+	199	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate 1	209	X+	209	X+	209	X+	1012.5	X+	209	X+	209	X+	209	X+
16WW04-A				Avg.	204		204		204		988.25		204		204		204	
16WW05	5/4/93	0:00	0	Primary	223	X+	223	X+	223	X+	1081	X+	223	X+	223	X+	223	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+
NATURAL LOG																		
16SD01					5.651		5.651		5.651		7.229		5.651		5.651		5.651	
16SD02					5.342		5.342		5.342		6.92		5.342		5.342		5.342	
16SD03					5.368		5.368		5.368		6.946		5.368		5.368		5.368	
16WW01-A					5.269		5.269		5.269		6.847		5.269		5.269		5.269	
16WW02-A					5.263		5.263		5.263		6.841		5.263		5.263		5.263	
16WW03					5.33		5.33		5.33		6.908		5.33		5.33		5.33	
16WW04-A					5.318		5.318		5.318		6.896		5.318		5.318		5.318	
16WW05					5.407		5.407		5.407		6.986		5.407		5.407		5.407	
16WW06					5.368		5.368		5.368		6.946		5.368		5.368		5.368	
STATISTICS																		
Total No.					9		9		9		9		9		9		9	
Min.					192		192		192		930		192		192		192	
Max.					284.5		284.5		284.5		1379.5		284.5		284.5		284.5	
Avg. (log transformed)					5.368		5.368		5.368		6.947		5.368		5.368		5.368	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	Benzo(b) flU+oranthene (U+g/kg)	Benzo(g,h,i) perylene (U+g/kg)	Benzo(k)flU+or anthene (U+g/kg)	Benzoic acid (U+g/kg)	Benzyl alcohol (U+g/kg)	Bis(2-chloro-1-methylethyl) ether (U+g/kg)	Bis(2-chloro ethoX+y) metha (U+g/kg)
St. Dev.(log transformed)					0.116	0.116	0.116	0.116	0.116	0.116	0.116
H											
95th UCL of Mean					215.88	215.88	215.88	1047.05	215.88	215.88	215.88

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	bis(2-Chloro ethyl) ether (U+g/kg)		Bis(2-ethyl heX+yl)phthalat (U+g/kg)		BU+tyl benzyl phthalate (U+g/kg)		Chrysene (U+g/kg)		Dibenz(a,h) anthracene (U+g/kg)		DibenzofU+ran (U+g/kg)		Diethyl phthalate (U+g/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	284.5	X+	284.5	X+	284.5	X+	284.5	X+	284.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate 1	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+
16WW01-A				Avg.	194.25		194.25		194.25		194.25		194.25		194.25		194.25	
16WW02	5/12/93	0:00	0	Primary	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate 1	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+
16WW02-A				Avg.	193		193		193		193		193		193		193	
16WW03	5/4/93	0:00	0	Primary	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	199	X+	960		199	X+	199	X+	199	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate 1	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+
16WW04-A				Avg.	204		204		584.5		204		204		204		204	
16WW05	5/4/93	0:00	0	Primary	223	X+	223	X+	223	X+	223	X+	223	X+	223	X+	223	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+
NATURAL LOG																		
16SD01					5.651		5.651		5.651		5.651		5.651		5.651		5.651	
16SD02					5.342		5.342		5.342		5.342		5.342		5.342		5.342	
16SD03					5.368		5.368		5.368		5.368		5.368		5.368		5.368	
16WW01-A					5.269		5.269		5.269		5.269		5.269		5.269		5.269	
16WW02-A					5.263		5.263		5.263		5.263		5.263		5.263		5.263	
16WW03					5.33		5.33		5.33		5.33		5.33		5.33		5.33	
16WW04-A					5.318		5.318		6.371		5.318		5.318		5.318		5.318	
16WW05					5.407		5.407		5.407		5.407		5.407		5.407		5.407	
16WW06					5.368		5.368		5.368		5.368		5.368		5.368		5.368	
STATISTICS																		
Total No.					9		9		9		9		9		9		9	
Min.					192		192		192		192		192		192		192	
Max.					284.5		284.5		960		284.5		284.5		284.5		284.5	
Avg. (log transformed)					5.368		5.368		5.485		5.368		5.368		5.368		5.368	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	bis(2-Chloro ethyl) ether (U+g/kg)	Bis(2-ethyl heX+yl)phthalat (U+g/kg)	BU+tyl benzyl phthalate (U+g/kg)	Chrysene (U+g/kg)	Dibenz(a,h) anthracene (U+g/kg)	DibenzofU+ran (U+g/kg)	Diethyl phthalate (U+g/kg)
St. Dev.(log transformed)					0.116	0.116	0.351	0.116	0.116	0.116	0.116
H							2.074				
95th UCL of Mean					215.88	215.88	331.62	215.88	215.88	215.88	215.88

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	Dimethyl phthalate (U+g/kg)		Di-n-bU+tyl phthalate (U+g/kg)		Di-n-octyl phthalate (U+g/kg)		Diphenylamine (U+g/kg)		FIU+oranthene (U+g/kg)		FIU+orene (U+g/kg)		HeX+achloro benzene (U+g/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	284.5	X+	284.5	X+	862	X+	284.5	X+	284.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	209	X+	209	X+	633	X+	209	X+	209	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	649.5	X+	214.5	X+	214.5	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	196.5	X+	196.5	X+	595	X+	196.5	X+	196.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate 1	192	X+	192	X+	192	X+	581.5	X+	192	X+	192	X+	192	X+
16WW01-A				Avg.	194.25		194.25		194.25		588.25		194.25		194.25		194.25	
16WW02	5/12/93	0:00	0	Primary	192	X+	192	X+	192	X+	581.5	X+	192	X+	192	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate 1	194	X+	194	X+	194	X+	588	X+	194	X+	194	X+	194	X+
16WW02-A				Avg.	193		193		193		584.75		193		193		193	
16WW03	5/4/93	0:00	0	Primary	206.5	X+	206.5	X+	206.5	X+	625	X+	206.5	X+	206.5	X+	206.5	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	1600		199	X+	602.5	X+	199	X+	199	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate 1	209	X+	1900		209	X+	633	X+	209	X+	209	X+	209	X+
16WW04-A				Avg.	204		1750		204		617.75		204		204		204	
16WW05	5/4/93	0:00	0	Primary	223	X+	223	X+	223	X+	675.5	X+	223	X+	223	X+	223	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	649.5	X+	214.5	X+	214.5	X+	214.5	X+
NATURAL LOG																		
16SD01					5.651		5.651		5.651		6.759		5.651		5.651		5.651	
16SD02					5.342		5.342		5.342		6.45		5.342		5.342		5.342	
16SD03					5.368		5.368		5.368		6.476		5.368		5.368		5.368	
16WW01-A					5.269		5.269		5.269		6.377		5.269		5.269		5.269	
16WW02-A					5.263		5.263		5.263		6.371		5.263		5.263		5.263	
16WW03					5.33		5.33		5.33		6.438		5.33		5.33		5.33	
16WW04-A					5.318		7.467		5.318		6.426		5.318		5.318		5.318	
16WW05					5.407		5.407		5.407		6.515		5.407		5.407		5.407	
16WW06					5.368		5.368		5.368		6.476		5.368		5.368		5.368	
STATISTICS																		
Total No.					9		9		9		9		9		9		9	
Min.					192		192		192		581.5		192		192		192	
Max.					284.5		1900		284.5		862		284.5		284.5		284.5	
Avg. (log transformed)					5.368		5.607		5.368		6.476		5.368		5.368		5.368	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	Dimethyl phthalate (U+g/kg)	Di-n-bU+tyl phthalate (U+g/kg)	Di-n-octyl phthalate (U+g/kg)	Diphenylamine (U+g/kg)	FIU+oranthene (U+g/kg)	FIU+orene (U+g/kg)	HeX+achloro benzene (U+g/kg)
St. Dev.(log transformed)					0.116	0.707	0.116	0.116	0.116	0.116	0.116
H						2.632					
95th UCL of Mean					215.88	675.08	215.88	653.75	215.88	215.88	215.88

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	HeX+achloro-1,3-bU+tadiene (U+g/kg)		HeX+achloro cyclopentadiene (U+g/kg)		HeX+achloro ethane (U+g/kg)		1,2-Diphenyl hydrazine (U+g/kg)		Indeno (1,2,3-cd) pyrene (U+g/kg)		Isophorone (U+g/kg)		3-Methylphenol (U+g/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	284.5	X+	284.5	X+	1379.5	X+	284.5	X+	284.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	209	X+	209	X+	1012.5	X+	209	X+	209	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate 1	192	X+	192	X+	192	X+	930	X+	192	X+	192	X+	192	X+
16WW01-A				Avg.	194.25		194.25		194.25		941.25		194.25		194.25		194.25	
16WW02	5/12/93	0:00	0	Primary	192	X+	192	X+	192	X+	930	X+	192	X+	192	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate 1	194	X+	194	X+	194	X+	941	X+	194	X+	194	X+	194	X+
16WW02-A				Avg.	193		193		193		935.5		193		193		193	
16WW03	5/4/93	0:00	0	Primary	206.5	X+	206.5	X+	206.5	X+	1000	X+	206.5	X+	206.5	X+	206.5	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	199	X+	199	X+	964	X+	199	X+	199	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate 1	209	X+	209	X+	209	X+	1012.5	X+	209	X+	209	X+	209	X+
16WW04-A				Avg.	204		204		204		988.25		204		204		204	
16WW05	5/4/93	0:00	0	Primary	223	X+	223	X+	223	X+	1081	X+	223	X+	223	X+	223	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+
NATURAL LOG																		
16SD01					5.651		5.651		5.651		7.229		5.651		5.651		5.651	
16SD02					5.342		5.342		5.342		6.92		5.342		5.342		5.342	
16SD03					5.368		5.368		5.368		6.946		5.368		5.368		5.368	
16WW01-A					5.269		5.269		5.269		6.847		5.269		5.269		5.269	
16WW02-A					5.263		5.263		5.263		6.841		5.263		5.263		5.263	
16WW03					5.33		5.33		5.33		6.908		5.33		5.33		5.33	
16WW04-A					5.318		5.318		5.318		6.896		5.318		5.318		5.318	
16WW05					5.407		5.407		5.407		6.986		5.407		5.407		5.407	
16WW06					5.368		5.368		5.368		6.946		5.368		5.368		5.368	
STATISTICS																		
Total No.					9		9		9		9		9		9		9	
Min.					192		192		192		930		192		192		192	
Max.					284.5		284.5		284.5		1379.5		284.5		284.5		284.5	
Avg. (log transformed)					5.368		5.368		5.368		6.947		5.368		5.368		5.368	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	HeX+achloro-1,3-bU+tadiene (U+g/kg)	HeX+achloro cyclopentadiene (U+g/kg)	HeX+achloro ethane (U+g/kg)	1,2-Diphenyl hydrazine (U+g/kg)	Indeno (1,2,3-cd) pyrene (U+g/kg)	Isophorone (U+g/kg)	3-Methylphenol (U+g/kg)
St. Dev.(log transformed)					0.116	0.116	0.116	0.116	0.116	0.116	0.116
H											
95th UCL of Mean					215.88	215.88	215.88	1047.05	215.88	215.88	215.88

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	1,3-Dichloro benzene (U+g/kg)		3-Nitroaniline (U+g/kg)		Naphthalene (U+g/kg)		Nitrobenzene (U+g/kg)		N-Nitroso diphenylamine (U+g/kg)		N-Nitrosodi-n-propylamine (U+g/kg)		2-Methylphenol (U+g/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	1379.5	X+	284.5	X+	284.5	X+	284.5	X+	1379.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate 1	192	X+	930	X+	192	X+	192	X+	192	X+	930	X+	192	X+
16WW01-A				Avg.	194.25		941.25		194.25		194.25		194.25		941.25		194.25	
16WW02	5/12/93	0:00	0	Primary	192	X+	930	X+	192	X+	192	X+	192	X+	930	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate 1	194	X+	941	X+	194	X+	194	X+	194	X+	941	X+	194	X+
16WW02-A				Avg.	193		935.5		193		193		193		935.5		193	
16WW03	5/4/93	0:00	0	Primary	206.5	X+	1000	X+	206.5	X+	206.5	X+	206.5	X+	1000	X+	206.5	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	964	X+	199	X+	199	X+	199	X+	964	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate 1	209	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+	209	X+
16WW04-A				Avg.	204		988.25		204		204		204		988.25		204	
16WW05	5/4/93	0:00	0	Primary	223	X+	1081	X+	223	X+	223	X+	223	X+	1081	X+	223	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+
NATURAL LOG																		
16SD01					5.651		7.229		5.651		5.651		5.651		7.229		5.651	
16SD02					5.342		6.92		5.342		5.342		5.342		6.92		5.342	
16SD03					5.368		6.946		5.368		5.368		5.368		6.946		5.368	
16WW01-A					5.269		6.847		5.269		5.269		5.269		6.847		5.269	
16WW02-A					5.263		6.841		5.263		5.263		5.263		6.841		5.263	
16WW03					5.33		6.908		5.33		5.33		5.33		6.908		5.33	
16WW04-A					5.318		6.896		5.318		5.318		5.318		6.896		5.318	
16WW05					5.407		6.986		5.407		5.407		5.407		6.986		5.407	
16WW06					5.368		6.946		5.368		5.368		5.368		6.946		5.368	
STATISTICS																		
Total No.					9		9		9		9		9		9		9	
Min.					192		930		192		192		192		930		192	
Max.					284.5		1379.5		284.5		284.5		284.5		1379.5		284.5	
Avg. (log transformed)					5.368		6.947		5.368		5.368		5.368		6.947		5.368	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	1,3-Dichloro benzene (U+g/kg)	3-Nitroaniline (U+g/kg)	Naphthalene (U+g/kg)	Nitrobenzene (U+g/kg)	N-Nitroso diphenylamine (U+g/kg)	N-Nitrosodi-n-propylamine (U+g/kg)	2-Methylphenol (U+g/kg)
St. Dev.(log transformed)					0.116	0.116	0.116	0.116	0.116	0.116	0.116
H											
95th UCL of Mean					215.88	1047.05	215.88	215.88	215.88	1047.05	215.88

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	1,2-Dichloro benzene (U+g/kg)		2-Nitroaniline (U+g/kg)		2-Nitrophenol (U+g/kg)		4-Chloroaniline (U+g/kg)		4-Chloro-3-methylphenol (U+g/kg)		Pentachloro phenol (U+g/kg)		4-Methylphenol (U+g/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	1379.5	X+	284.5	X+	284.5	X+	284.5	X+	1379.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate 1	192	X+	930	X+	192	X+	192	X+	192	X+	930	X+	192	X+
16WW01-A				Avg.	194.25		941.25		194.25		194.25		194.25		941.25		194.25	
16WW02	5/12/93	0:00	0	Primary	192	X+	930	X+	192	X+	192	X+	192	X+	930	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate 1	194	X+	941	X+	194	X+	194	X+	194	X+	941	X+	194	X+
16WW02-A				Avg.	193		935.5		193		193		193		935.5		193	
16WW03	5/4/93	0:00	0	Primary	206.5	X+	1000	X+	206.5	X+	206.5	X+	206.5	X+	1000	X+	206.5	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	964	X+	199	X+	199	X+	199	X+	964	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate 1	209	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+	209	X+
16WW04-A				Avg.	204		988.25		204		204		204		988.25		204	
16WW05	5/4/93	0:00	0	Primary	223	X+	1081	X+	223	X+	223	X+	223	X+	1081	X+	223	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+
NATURAL LOG																		
16SD01					5.651		7.229		5.651		5.651		5.651		7.229		5.651	
16SD02					5.342		6.92		5.342		5.342		5.342		6.92		5.342	
16SD03					5.368		6.946		5.368		5.368		5.368		6.946		5.368	
16WW01-A					5.269		6.847		5.269		5.269		5.269		6.847		5.269	
16WW02-A					5.263		6.841		5.263		5.263		5.263		6.841		5.263	
16WW03					5.33		6.908		5.33		5.33		5.33		6.908		5.33	
16WW04-A					5.318		6.896		5.318		5.318		5.318		6.896		5.318	
16WW05					5.407		6.986		5.407		5.407		5.407		6.986		5.407	
16WW06					5.368		6.946		5.368		5.368		5.368		6.946		5.368	
STATISTICS																		
Total No.					9		9		9		9		9		9		9	
Min.					192		930		192		192		192		930		192	
Max.					284.5		1379.5		284.5		284.5		284.5		1379.5		284.5	
Avg. (log transformed)					5.368		6.947		5.368		5.368		5.368		6.947		5.368	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	1,2-Dichloro benzene (U+g/kg)	2-Nitroaniline (U+g/kg)	2-Nitrophenol (U+g/kg)	4-Chloroaniline (U+g/kg)	4-Chloro-3-methylphenol (U+g/kg)	Pentachloro phenol (U+g/kg)	4-Methylphenol (U+g/kg)
St. Dev.(log transformed)					0.116	0.116	0.116	0.116	0.116	0.116	0.116
H											
95th UCL of Mean					215.88	1047.05	215.88	215.88	215.88	1047.05	215.88

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	1,4-Dichloro benzene (U+g/kg)		Phenanthrene (U+g/kg)		Phenol (U+g/kg)		4-Nitroaniline (U+g/kg)		4-Nitrophenol (U+g/kg)		Pyrene (U+g/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	284.5	X+	284.5	X+	1379.5	X+	1379.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	209	X+	209	X+	1012.5	X+	1012.5	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	1039	X+	1039	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	196.5	X+	196.5	X+	952.5	X+	952.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate 1	192	X+	192	X+	192	X+	930	X+	930	X+	192	X+
16WW01-A				Avg.	194.25		194.25		194.25		941.25		941.25		194.25	
16WW02	5/12/93	0:00	0	Primary	192	X+	192	X+	192	X+	930	X+	930	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate 1	194	X+	194	X+	194	X+	941	X+	941	X+	194	X+
16WW02-A				Avg.	193		193		193		935.5		935.5		193	
16WW03	5/4/93	0:00	0	Primary	206.5	X+	206.5	X+	206.5	X+	1000	X+	1000	X+	206.5	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	199	X+	199	X+	964	X+	964	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate 1	209	X+	209	X+	209	X+	1012.5	X+	1012.5	X+	209	X+
16WW04-A				Avg.	204		204		204		988.25		988.25		204	
16WW05	5/4/93	0:00	0	Primary	223	X+	223	X+	223	X+	1081	X+	1081	X+	223	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	1039	X+	1039	X+	214.5	X+
NATURAL LOG																
16SD01					5.651		5.651		5.651		7.229		7.229		5.651	
16SD02					5.342		5.342		5.342		6.92		6.92		5.342	
16SD03					5.368		5.368		5.368		6.946		6.946		5.368	
16WW01-A					5.269		5.269		5.269		6.847		6.847		5.269	
16WW02-A					5.263		5.263		5.263		6.841		6.841		5.263	
16WW03					5.33		5.33		5.33		6.908		6.908		5.33	
16WW04-A					5.318		5.318		5.318		6.896		6.896		5.318	
16WW05					5.407		5.407		5.407		6.986		6.986		5.407	
16WW06					5.368		5.368		5.368		6.946		6.946		5.368	
STATISTICS																
Total No.					9		9		9		9		9		9	
Min.					192		192		192		930		930		192	
Max.					284.5		284.5		284.5		1379.5		1379.5		284.5	
Avg. (log transformed)					5.368		5.368		5.368		6.947		6.947		5.368	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(0-5 feet)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	1,4-Dichloro benzene (U+g/kg)	Phenanthrene (U+g/kg)	Phenol (U+g/kg)	4-Nitroaniline (U+g/kg)	4-Nitrophenol (U+g/kg)	Pyrene (U+g/kg)
St. Dev.(log transformed)					0.116	0.116	0.116	0.116	0.116	0.116
H										
95th UCL of Mean					215.88	215.88	215.88	1047.05	1047.05	215.88

Data Qualifier Notations

X+ = Non-Detect Result, one-half of the detection limit value presented.
U+ = Considered Non-Detect based on blank comparison, one-half of the detection limit value is presented.
J = Estimated laboratory result.

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Aluminum (mg/kg)	Antimony (mg/kg)		Arsenic (mg/kg)		Barium (mg/kg)		Beryllium (mg/kg)	Cadmium (mg/kg)		Calcium (mg/kg)
16SD01	5/2/93	0:00	0	Primary		0.86	X+	0.86	X+	295	J		0.86	X+	
16SD01	2/21/95	14:20	0	Primary		0.92	X+R	2.21		193.4			0.46	X+	
16SD02	4/14/93	0:00	0	Primary		0.635	X+U+J	7.17		52.5			0.635	X+	
16SD02	2/28/95	10:00	0	Primary		0.69	X+R	2.49		53.8			0.345	X+	
16SD03	4/14/93	0:00	0	Primary		0.65	X+U+J	3.85		56.4			0.65	X+	
16SD03	2/20/95	16:05	0	Primary		0.78	X+	2.02		58.8			0.39	X+	
16SD06	2/20/95	16:35	0	Primary		0.73	X+	2.63		64.5			0.365	X+	
16SD07	2/21/95	14:05	0	Primary		0.795	X+R	4.13		94.9			0.395	X+	
16SD08	2/21/95	9:30	0	Primary		0.655	X+R	3.01		383.5			0.325	X+	
16SD09	2/21/95	10:55	0	Primary		0.665	X+R	4.8		209.3			0.335	X+	
16SD10	2/21/95	17:10	0	Primary		0.65	X+R	5.59		72.3			0.325	X+	
16SD11	2/28/95	11:15	0	Primary		0.65	X+R	3.63		86.1			0.325	X+	
16WW01	5/4/93	0:00	0	Primary		0.595	X+	3.34		73.7			0.595	X+	
16WW01	5/4/93	0:00	0	Duplicate 1		0.58	U+JX+	3.22	J	84			0.58	X+	
16WW01-A				Avg.		0.5875		3.28		78.85			0.5875		
16WW01	5/4/93	0:00	5	Primary		0.615	U+JX+	2.28	J	44.2			0.615	X+	
16WW01	5/4/93	0:00	10	Primary		0.625	U+JX+	4.08	J	71.1			0.625	X+	
16WW02	5/12/93	0:00	0	Primary		0.58	U+JX+	1.43	J	46.7			0.58	X+	
16WW02	5/12/93	0:00	0	Duplicate 1		0.59	X+	1.72		55			0.59	X+	
16WW02-A				Avg.		0.585		1.575		50.85			0.585		
16WW02	5/12/93	0:00	5	Primary		0.615	U+JX+	4.34	J	41.1			0.615	X+	
16WW03	5/4/93	0:00	0	Primary		0.625	U+JX+	14.2	J	94.7			0.625	X+	
16WW03	5/4/93	0:00	5	Primary		0.595	U+JX+	2.98	J	28.8			0.595	X+	
16WW03	5/4/93	0:00	10	Primary		0.61	U+JX+	4.5	J	111			0.61	X+	
16WW03	5/4/93	0:00	15	Primary		0.635	U+JX+	3.88	J	438			0.635	X+	
16WW04	5/12/93	0:00	0	Primary		0.6	U+JX+	6.97	J	111			0.6	X+	
16WW04	5/12/93	0:00	0	Duplicate 1		0.635	X+	14.3		104			0.635	X+	
16WW04-A				Avg.		0.6175		10.635		107.5			0.6175		
16WW04	5/12/93	0:00	5	Primary		0.59	U+JX+	3.05	J	28.8			0.59	X+	
16WW04	5/12/93	0:00	10	Primary		0.6	X+	1.97		70.6			0.6	X+	
16WW04	5/12/93	0:00	10	Duplicate 1		0.61	X+	4.21		73.6			0.61	X+	
16WW04-A				Avg.		0.605		3.09		72.1			0.605		
16WW05	5/4/93	0:00	0	Primary		0.675	U+JX+	14.4	J	146			0.675	X+	
16WW05	5/4/93	0:00	5	Primary		0.57	U+JX+	7.66	J	301			0.57	X+	
16WW05	5/4/93	0:00	10	Primary		0.58	U+JX+	2.94	J	166			0.58	X+	
16WW05	5/4/93	0:00	15	Primary		0.595	U+JX+	3.79	J	172			0.595	X+	
16WW05	5/4/93	0:00	20	Primary		0.64	U+JX+	5.09	J	268			0.64	X+	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Aluminum (mg/kg)	Antimony (mg/kg)		Arsenic (mg/kg)		Barium (mg/kg)	Beryllium (mg/kg)	Cadmium (mg/kg)		Calcium (mg/kg)
16WW05	5/4/93	0:00	25	Primary		0.64	U+JX+	3.47	J	139		0.64	X+	
16WW06	5/5/93	0:00	0	Primary		0.65	X+	5.5		216		0.65	X+	
16WW06	5/5/93	0:00	5	Primary		0.635	X+	12		463		0.635	X+	
16WW06	5/5/93	0:00	10	Primary		0.595	X+	2.73		205		0.595	X+	
16WW06	5/5/93	0:00	15	Primary		0.59	X+	7.28		127		0.59	X+	
16WW06	5/5/93	0:00	20	Primary		0.635	X+	3.98		353		0.635	X+	
16WW27	5/17/97	9:15	0	Primary	7030	3.85	U+JX+	3.2		168	0.68	0.32	X+	1540
16WW27	5/17/97	10:17	1	Primary	7070	3.6	U+JX+	2.5		58.8	0.41	0.51		118
16WW31	5/29/97	8:27	0	Primary	4520	3.65	U+JX+	6.7		97.2	1.4	0.77		143
16WW31	5/29/97	8:55	1	Primary	9740	0.48		6.6		62.3	0.8	0.56		255
16WW31	5/29/97	10:09	5	Primary	8350	3.65	U+JX+	1.6		165	0.63	0.305	X+	289
16WW31	5/29/97	11:10	14	Primary	4330	3.25	U+JX+	1.4		60.5	0.43	0.27	X+	523
16WW35	6/25/97	9:50	0	Primary	18100	3.15	U+JX+	4.9		80.2	0.8	0.265	X+	370
16WW35	6/25/97	9:50	0	Duplicate	17000	3.55	U+JX+	6.1		88.7	0.85	0.295	X+	483
16WW35-A				Avg.		3.35		5.5		84.45	0.825	0.28		426.5
16WW35	6/25/97	10:05	1	Primary	10600	3.7	U+JX+	3.4		47.4	0.38	0.31	X+	295
16WW35	6/25/97	11:15	5	Primary	18300	3.75	U+JX+	6.1		120	0.89	0.31	X+	739
16WW37	5/31/97	15:55	0	Primary	5640	3.75	U+JX+	4.4		256	0.88	0.31	X+	15100
16WW37	5/31/97	16:10	1	Primary	21500	3.8	U+JX+	7.5		208	1.2	0.86		3090
16WW37	5/31/97	16:35	5	Primary	6550	3.6	U+JX+	2		48.5	0.31	0.3	X+	358
16WW37	6/1/97	10:01	14	Primary	7850	3.55	U+JX+	3.1		147	0.76	0.295	X+	1070

NATURAL LOGS

16SD01	-0.151	-0.151	5.687	-0.151	
16SD01	-0.083	0.793	5.265	-0.777	
16SD02	-0.454	1.97	3.961	-0.454	
16SD02	-0.371	0.912	3.985	-1.064	
16SD03	-0.431	1.348	4.032	-0.431	
16SD03	-0.248	0.703	4.074	-0.942	
16SD06	-0.315	0.967	4.167	-1.008	
16SD07	-0.229	1.418	4.553	-0.929	
16SD08	-0.423	1.102	5.949	-1.124	
16SD09	-0.408	1.569	5.344	-1.094	
16SD10	-0.431	1.721	4.281	-1.124	
16SD11	-0.431	1.289	4.456	-1.124	
16WW01-A	-0.532	1.188	4.368	-0.532	
16WW01	-0.486	0.824	3.789	-0.486	
16WW01	-0.47	1.406	4.264	-0.47	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Aluminum (mg/kg)	Antimony (mg/kg)	Arsenic (mg/kg)	Barium (mg/kg)	Beryllium (mg/kg)	Cadmium (mg/kg)	Calcium (mg/kg)
16WW02-A						-0.536	0.454	3.929		-0.536	
16WW02						-0.486	1.468	3.716		-0.486	
16WW03						-0.47	2.653	4.551		-0.47	
16WW03						-0.519	1.092	3.36		-0.519	
16WW03						-0.494	1.504	4.71		-0.494	
16WW03						-0.454	1.356	6.082		-0.454	
16WW04-A						-0.482	2.364	4.677		-0.482	
16WW04						-0.528	1.115	3.36		-0.528	
16WW04-A						-0.503	1.128	4.278		-0.503	
16WW05						-0.393	2.667	4.984		-0.393	
16WW05						-0.562	2.036	5.707		-0.562	
16WW05						-0.545	1.078	5.112		-0.545	
16WW05						-0.519	1.332	5.147		-0.519	
16WW05						-0.446	1.627	5.591		-0.446	
16WW05						-0.446	1.244	4.934		-0.446	
16WW06						-0.431	1.705	5.375		-0.431	
16WW06						-0.454	2.485	6.138		-0.454	
16WW06						-0.519	1.004	5.323		-0.519	
16WW06						-0.528	1.985	4.844		-0.528	
16WW06						-0.454	1.381	5.866		-0.454	
16WW27					8.858	1.348	1.163	5.124	-0.386	-1.139	7.34
16WW27					8.864	1.281	0.916	4.074	-0.892	-0.673	4.771
16WW31					8.416	1.295	1.902	4.577	0.336	-0.261	4.963
16WW31					9.184	-0.734	1.887	4.132	-0.223	-0.58	5.541
16WW31					9.03	1.295	0.47	5.106	-0.462	-1.187	5.666
16WW31					8.373	1.179	0.336	4.103	-0.844	-1.309	6.26
16WW35-A						1.209	1.705	4.436	-0.192	-1.273	6.056
16WW35					9.269	1.308	1.224	3.859	-0.968	-1.171	5.687
16WW35					9.815	1.322	1.808	4.787	-0.117	-1.171	6.605
16WW37					8.638	1.322	1.482	5.545	-0.128	-1.171	9.622
16WW37					9.976	1.335	2.015	5.338	0.182	-0.151	8.036
16WW37					8.787	1.281	0.693	3.882	-1.171	-1.204	5.881
16WW37					8.968	1.267	1.131	4.99	-0.274	-1.221	6.975
STATISTICS											
Total No.					12	48	48	48	13	48	13
Min.					4330	0.48	0.86	28.8	0.31	0.265	118
Max.					21500	3.85	14.4	463	1.4	0.86	15100

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	RESULT DEPTH (ft TYPE	Aluminum (mg/kg)	Antimony (mg/kg)	Arsenic (mg/kg)	Barium (mg/kg)	Beryllium (mg/kg)	Cadmium (mg/kg)	Calcium (mg/kg)
Avg.(log transformed)				9.015	-0.011	1.364	4.704	-0.395	-0.708	6.416
St. Dev.(log transformed)				0.493	0.765	0.587	0.726	0.455	0.338	1.33
H				2.133	2.09	1.943	2.056	2.063	1.784	3.455
95th UCL of the Mean				12753.80	1.67	5.49	178.62	0.98	0.57	5580.06

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Chromium (mg/kg)	Cobalt (mg/kg)	Copper (mg/kg)	Iron (mg/kg)	Lead (mg/kg)		Magnesium (mg/kg)
16SD01	5/2/93	0:00	0	Primary	28.8				29		
16SD01	2/21/95	14:20	0	Primary	13.8		10.5		18.4		
16SD02	4/14/93	0:00	0	Primary	12.7				17.3		
16SD02	2/28/95	10:00	0	Primary	8.74		6.1		7.87		
16SD03	4/14/93	0:00	0	Primary	40.9				14.5		
16SD03	2/20/95	16:05	0	Primary	8.16		4		7		
16SD06	2/20/95	16:35	0	Primary	13.5		8.5		10.5		
16SD07	2/21/95	14:05	0	Primary	10.2		6.8		7.62		
16SD08	2/21/95	9:30	0	Primary	11.1		6		4.58		
16SD09	2/21/95	10:55	0	Primary	15.7		10.3		49.3		
16SD10	2/21/95	17:10	0	Primary	18.9		3.4		23.4		
16SD11	2/28/95	11:15	0	Primary	22.6		6.1		9.99		
16WW01	5/4/93	0:00	0	Primary	9.57				7.08		
16WW01	5/4/93	0:00	0	Duplicate	15				6.94		
16WW01-A				Avg.	12.285				7.01		
16WW01	5/4/93	0:00	5	Primary	18				6.89		
16WW01	5/4/93	0:00	10	Primary	12.1				6.12		
16WW02	5/12/93	0:00	0	Primary	17.1				3.02	J	
16WW02	5/12/93	0:00	0	Duplicate	11.7				3.84		
16WW02-A				Avg.	14.4				3.43		
16WW02	5/12/93	0:00	5	Primary	14.6				5.26	J	
16WW03	5/4/93	0:00	0	Primary	32.9				11.3		
16WW03	5/4/93	0:00	5	Primary	11.6				5.03		
16WW03	5/4/93	0:00	10	Primary	16.4				7.84		
16WW03	5/4/93	0:00	15	Primary	17				8.73		
16WW04	5/12/93	0:00	0	Primary	16.8				10	J	
16WW04	5/12/93	0:00	0	Duplicate	20.8				9.72		
16WW04-A				Avg.	18.8				9.86		
16WW04	5/12/93	0:00	5	Primary	11				5.77	J	
16WW04	5/12/93	0:00	10	Primary	11.8				3.28		
16WW04	5/12/93	0:00	10	Duplicate	11.4				5.82		
16WW04-A				Avg.	11.6				4.55		
16WW05	5/4/93	0:00	0	Primary	36.7				15.4		
16WW05	5/4/93	0:00	5	Primary	19.3				10.9		
16WW05	5/4/93	0:00	10	Primary	15.7				7.89		
16WW05	5/4/93	0:00	15	Primary	22.3				10.8		
16WW05	5/4/93	0:00	20	Primary	23.1				13.7		

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Chromium (mg/kg)		Cobalt (mg/kg)		Copper (mg/kg)		Iron (mg/kg)	Lead (mg/kg)	Magnesium (mg/kg)	
16WW05	5/4/93	0:00	25	Primary	19.4							8.24		
16WW06	5/5/93	0:00	0	Primary	23.7							11.5		
16WW06	5/5/93	0:00	5	Primary	25.5							22.3		
16WW06	5/5/93	0:00	10	Primary	20							9.17		
16WW06	5/5/93	0:00	15	Primary	14.9							15.4		
16WW06	5/5/93	0:00	20	Primary	18.2							8.33		
16WW27	5/17/97	9:15	0	Primary	7.8		10.3		5.5		10200	12.1	1030	
16WW27	5/17/97	10:17	1	Primary	8.1		3	X+	4.3		9550	7.6	743	
16WW31	5/29/97	8:27	0	Primary	31.5	J	13.3		1.5	X+	19400	14.3	302.5	X+
16WW31	5/29/97	8:55	1	Primary	22.6	J	3.05	X+	1.55	X+	23500	8	306.5	X+
16WW31	5/29/97	10:09	5	Primary	8	J	8.2		5		6890	7.8	1420	
16WW31	5/29/97	11:10	14	Primary	8.3	J	2.7	X+	4.1		6430	3.4	894	
16WW35	6/25/97	9:50	0	Primary	16.7		3.7		4.6		17700	7.9	1020	
16WW35	6/25/97	9:50	0	Duplicate	18.9		4.0		4.4		21500	9.2	992	
16WW35-A				Avg.	17.8		3.85		4.5		19600	8.55	1006	
16WW35	6/25/97	10:05	1	Primary	10.4		2.8		4.9		14600	6	909	
16WW35	6/25/97	11:15	5	Primary	23.2		8.1		8.8		29900	11.7	2280	
16WW37	5/31/97	15:55	0	Primary	12.8		19.8		6.7		10100	21.8	688	
16WW37	5/31/97	16:10	1	Primary	23.9		6.6		6.7		26000	13.1	1420	
16WW37	5/31/97	16:35	5	Primary	5.9		2.4		3.7		7350	5	616	
16WW37	6/1/97	10:01	14	Primary	10.5		12.6		5.7		14500	10	1660	

NATURAL LOGS

16SD01				3.36				3.367	
16SD01				2.625		2.351		2.912	
16SD02				2.542				2.851	
16SD02				2.168		1.808		2.063	
16SD03				3.711				2.674	
16SD03				2.099		1.386		1.946	
16SD06				2.603		2.14		2.351	
16SD07				2.322		1.917		2.031	
16SD08				2.407		1.792		1.522	
16SD09				2.754		2.332		3.898	
16SD10				2.939		1.224		3.153	
16SD11				3.118		1.808		2.302	
16WW01-A				2.508				1.947	
16WW01				2.89				1.93	
16WW01				2.493				1.812	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	RESULT DEPTH (ft TYPE	Chromium (mg/kg)	Cobalt (mg/kg)	Copper (mg/kg)	Iron (mg/kg)	Lead (mg/kg)	Magnesium (mg/kg)
16WW02-A				2.667				1.233	
16WW02				2.681				1.66	
16WW03				3.493				2.425	
16WW03				2.451				1.615	
16WW03				2.797				2.059	
16WW03				2.833				2.167	
16WW04-A				2.934				2.288	
16WW04				2.398				1.753	
16WW04-A				2.451				1.515	
16WW05				3.603				2.734	
16WW05				2.96				2.389	
16WW05				2.754				2.066	
16WW05				3.105				2.38	
16WW05				3.14				2.617	
16WW05				2.965				2.109	
16WW06				3.165				2.442	
16WW06				3.239				3.105	
16WW06				2.996				2.216	
16WW06				2.701				2.734	
16WW06				2.901				2.12	
16WW27				2.054	2.332	1.705	9.23	2.493	6.937
16WW27				2.092	1.099	1.459	9.164	2.028	6.611
16WW31				3.45	2.588	0.405	9.873	2.66	5.712
16WW31				3.118	1.115	0.438	10.065	2.079	5.725
16WW31				2.079	2.104	1.609	8.838	2.054	7.258
16WW31				2.116	0.993	1.411	8.769	1.224	6.796
16WW35-A				2.879	1.348	1.504	9.883	2.146	6.914
16WW35				2.342	1.03	1.589	9.589	1.792	6.812
16WW35				3.144	2.092	2.175	10.306	2.46	7.732
16WW37				2.549	2.986	1.902	9.22	3.082	6.534
16WW37				3.174	1.887	1.902	10.166	2.573	7.258
16WW37				1.775	0.875	1.308	8.902	1.609	6.423
16WW37				2.351	2.534	1.74	9.582	2.303	7.415
STATISTICS									
Total No.				48	13	22	13	48	13
Min.				5.9	2.4	1.5	6430	3.02	302.5
Max.				40.9	19.8	10.5	29900	49.3	2280

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	RESULT DEPTH (ft TYPE	Chromium (mg/kg)	Cobalt (mg/kg)	Copper (mg/kg)	Iron (mg/kg)	Lead (mg/kg)	Magnesium (mg/kg)
Avg.(log transformed)				2.748	1.768	1.632	9.507	2.269	6.779
St. Dev.(log transformed)				0.445	0.724	0.5	0.525	0.543	0.597
H				1.846	2.407	1.981	2.143	1.912	2.231
95th UCL of the Mean				19.43	12.59	7.19	21366.80	13.04	1543.32

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Manganese (mg/kg)	Mercury (mg/kg)		Nickel (mg/kg)		Potassium (mg/kg)	Selenium (mg/kg)		Silver (mg/kg)		Sodium (mg/kg)
16SD01	5/2/93	0:00	0	Primary		0.085	X+	16.8	J		0.86	X+U+J	0.86	X+	
16SD01	2/21/95	14:20	0	Primary		0.09	X+	16.6			0.92	X+	0.92	X+	
16SD02	4/14/93	0:00	0	Primary		0.065	X+	10.3			0.635	X+U+J	0.635	X+	
16SD02	2/28/95	10:00	0	Primary		0.07	X+	7.75			0.69	X+	0.69	X+	
16SD03	4/14/93	0:00	0	Primary		0.065	X+	6.08			0.65	X+U+J	0.65	X+	
16SD03	2/20/95	16:05	0	Primary		0.08	X+	4.1			0.78	X+	0.78	X+	
16SD06	2/20/95	16:35	0	Primary		0.075	X+	8.26			0.73	X+	0.73	X+	
16SD07	2/21/95	14:05	0	Primary		0.08	X+	6.05			0.795	X+	0.795	X+	
16SD08	2/21/95	9:30	0	Primary		0.065	X+	14.4			0.655	X+	0.655	X+	
16SD09	2/21/95	10:55	0	Primary		0.065	X+	17.3			0.8		0.665	X+	
16SD10	2/21/95	17:10	0	Primary		0.065	X+	5.85			0.325	X+	0.65	X+	
16SD11	2/28/95	11:15	0	Primary		0.065	X+	8.2			0.325	X+	0.65	X+	
16WW01	5/4/93	0:00	0	Primary		0.06	X+	5.96			0.595	X+	0.595	X+	
16WW01	5/4/93	0:00	0	Duplicate		0.06	X+	7.45			0.58	U+JX+	0.58	X+	
16WW01-A				Avg.		0.06		6.705			0.5875		0.5875		
16WW01	5/4/93	0:00	5	Primary		0.06	X+	6.26			0.615	U+JX+	0.615	X+	
16WW01	5/4/93	0:00	10	Primary		0.06	X+	10.3			0.625	U+JX+	0.625	X+	
16WW02	5/12/93	0:00	0	Primary		0.06	X+	5.09			0.58	U+JX+	0.58	X+	
16WW02	5/12/93	0:00	0	Duplicate		0.06	X+	4.54			0.59	X+	0.59	X+	
16WW02-A				Avg.		0.06		4.815			0.585		0.585		
16WW02	5/12/93	0:00	5	Primary		0.06	X+	10.7			0.615	U+JX+	0.615	X+	
16WW03	5/4/93	0:00	0	Primary		0.06	X+	13.7			0.625	U+JX+	0.625	X+	
16WW03	5/4/93	0:00	5	Primary		0.06	X+	8.17			0.595	U+JX+	0.595	X+	
16WW03	5/4/93	0:00	10	Primary		0.06	X+	20.5			0.61	U+JX+	0.61	X+	
16WW03	5/4/93	0:00	15	Primary		0.065	X+	18.8			0.635	U+JX+	0.635	X+	
16WW04	5/12/93	0:00	0	Primary		0.06	X+	9.96			0.6	U+JX+	0.6	X+	
16WW04	5/12/93	0:00	0	Duplicate		0.065	X+	8.15			0.635	X+	0.635	X+	
16WW04-A				Avg.		0.0625		9.055			0.6175		0.6175		
16WW04	5/12/93	0:00	5	Primary		0.06	X+	7.7			0.59	U+JX+	0.59	X+	
16WW04	5/12/93	0:00	10	Primary		0.06	X+	14			0.6	X+	0.6	X+	
16WW04	5/12/93	0:00	10	Duplicate		0.06	X+	13.9			0.61	X+	0.61	X+	
16WW04-A				Avg.		0.06		13.95			0.605		0.605		
16WW05	5/4/93	0:00	0	Primary		0.07	X+	16.5			0.675	U+JX+	0.675	X+	
16WW05	5/4/93	0:00	5	Primary		0.055	X+	47.2			0.57	U+JX+	0.57	X+	
16WW05	5/4/93	0:00	10	Primary		0.06	X+	16.3			0.58	U+JX+	0.58	X+	
16WW05	5/4/93	0:00	15	Primary		0.06	X+	23.5			0.595	U+JX+	0.595	X+	
16WW05	5/4/93	0:00	20	Primary		0.065	X+	23.7			0.64	U+JX+	0.64	X+	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Manganese (mg/kg)	Mercury (mg/kg)		Nickel (mg/kg)		Potassium (mg/kg)		Selenium (mg/kg)		Silver (mg/kg)		Sodium (mg/kg)
16WW05	5/4/93	0:00	25	Primary		0.065	X+	18.3				0.64	U+JX+	0.64	X+	
16WW06	5/5/93	0:00	0	Primary		0.065	X+	11.1				0.65	U+JX+	0.65	X+	
16WW06	5/5/93	0:00	5	Primary		0.065	X+	44.6				0.635	U+JX+	0.635	X+	
16WW06	5/5/93	0:00	10	Primary		0.06	X+	25.4				0.595	U+JX+	0.595	X+	
16WW06	5/5/93	0:00	15	Primary		0.06	X+	13.4				0.59	U+JX+	0.59	X+	
16WW06	5/5/93	0:00	20	Primary		0.065	X+	26.4				0.635	U+JX+	0.635	X+	
16WW27	5/17/97	9:15	0	Primary	1270	0.051		9		570		0.32	X+	0.65	X+	68.5
16WW27	5/17/97	10:17	1	Primary	204	0.02	X+	2.4	U+	481		0.3	X+	0.6	X+	144
16WW31	5/29/97	8:27	0	Primary	893	0.023		6.1		302.5	X+	0.21	X+	0.6	X+	21.1
16WW31	5/29/97	8:55	1	Primary	45.4	0.02	X+	5.9		306.5	X+	0.61		0.6	X+	61.0
16WW31	5/29/97	10:09	5	Primary	19	0.02	X+	10.7		559		0.305	X+	0.6	X+	377
16WW31	5/29/97	11:10	14	Primary	37.5	0.018	X+	9.4		514		0.27	X+	0.55	X+	415
16WW35	6/25/97	9:50	0	Primary	40.9	0.02		10.4		1070		1		0.55	U+JX+	63.6
16WW35	6/25/97	9:50	0	Duplicate	53.4	0.03		9.7		974		1.4		0.6	U+JX+	24.2
16WW35-A				Avg.	47.15	0.025		10.05		1022		1.2		0.575		43.875
16WW35	6/25/97	10:05	1	Primary	29.2	0.04		7		308.5	X+	1		0.6	U+JX+	97.3
16WW35	6/25/97	11:15	5	Primary	189	0.006	X+	15.4		1100		0.98		0.6	U+JX+	458
16WW37	5/31/97	15:55	0	Primary	1060	0.062		8.2		846		0.31	X+	0.55		42.8
16WW37	5/31/97	16:10	1	Primary	75.7	0.051		13.7		1560		0.74		0.65	X+	44.1
16WW37	5/31/97	16:35	5	Primary	21.6	0.02	X+	5.2		504		0.3	X+	0.6	X+	61.4
16WW37	6/1/97	10:01	14	Primary	472	0.0195	X+	18.4		587		0.295	X+	0.6	X+	334
NATURAL LOGS																
16SD01						-2.465		2.821				-0.151		-0.151		
16SD01						-2.408		2.809				-0.083		-0.083		
16SD02						-2.733		2.332				-0.454		-0.454		
16SD02						-2.659		2.048				-0.371		-0.371		
16SD03						-2.733		1.805				-0.431		-0.431		
16SD03						-2.526		1.411				-0.248		-0.248		
16SD06						-2.59		2.111				-0.315		-0.315		
16SD07						-2.526		1.8				-0.229		-0.229		
16SD08						-2.733		2.667				-0.423		-0.423		
16SD09						-2.733		2.851				-0.223		-0.408		
16SD10						-2.733		1.766				-1.124		-0.431		
16SD11						-2.733		2.104				-1.124		-0.431		
16WW01-A						-2.813		1.903				-0.532		-0.532		
16WW01						-2.813		1.834				-0.486		-0.486		
16WW01						-2.813		2.332				-0.47		-0.47		

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Manganese (mg/kg)	Mercury (mg/kg)	Nickel (mg/kg)	Potassium (mg/kg)	Selenium (mg/kg)	Silver (mg/kg)	Sodium (mg/kg)
16WW02-A						-2.813	1.572		-0.536	-0.536	
16WW02						-2.813	2.37		-0.486	-0.486	
16WW03						-2.813	2.617		-0.47	-0.47	
16WW03						-2.813	2.1		-0.519	-0.519	
16WW03						-2.813	3.02		-0.494	-0.494	
16WW03						-2.733	2.934		-0.454	-0.454	
16WW04-A						-2.773	2.203		-0.482	-0.482	
16WW04						-2.813	2.041		-0.528	-0.528	
16WW04-A						-2.813	2.635		-0.503	-0.503	
16WW05						-2.659	2.803		-0.393	-0.393	
16WW05						-2.9	3.854		-0.562	-0.562	
16WW05						-2.813	2.791		-0.545	-0.545	
16WW05						-2.813	3.157		-0.519	-0.519	
16WW05						-2.733	3.165		-0.446	-0.446	
16WW05						-2.733	2.907		-0.446	-0.446	
16WW06						-2.733	2.407		-0.431	-0.431	
16WW06						-2.733	3.798		-0.454	-0.454	
16WW06						-2.813	3.235		-0.519	-0.519	
16WW06						-2.813	2.595		-0.528	-0.528	
16WW06						-2.733	3.273		-0.454	-0.454	
16WW27					7.147	-2.976	2.197	6.346	-1.139	-0.431	4.227
16WW27					5.318	-3.912	0.875	6.176	-1.204	-0.511	4.97
16WW31					6.795	-3.772	1.808	5.712	-1.561	-0.511	3.049
16WW31					3.816	-3.912	1.775	5.725	-0.494	-0.511	4.111
16WW31					2.944	-3.912	2.37	6.326	-1.187	-0.511	5.932
16WW31					3.624	-4.017	2.241	6.242	-1.309	-0.598	6.028
16WW35-A					3.853	-3.689	2.308	6.93	0.182	-0.553	3.781
16WW35					3.374	-3.219	1.946	5.732	0	-0.511	4.578
16WW35					5.242	-5.116	2.734	7.003	-0.02	-0.511	6.127
16WW37					6.966	-2.781	2.104	6.741	-1.171	-0.598	3.757
16WW37					4.327	-2.976	2.617	7.352	-0.301	-0.431	3.786
16WW37					3.073	-3.912	1.649	6.223	-1.204	-0.511	4.117
16WW37					6.157	-3.937	2.912	6.375	-1.221	-0.511	5.811
STATISTICS											
Total No.					13	48	48	13	48	48	13
Min.					19	0.006	2.4	302.5	0.21	0.55	21.1
Max.					1270	0.09	47.2	1560	1.4	0.92	458

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Manganese (mg/kg)	Mercury (mg/kg)	Nickel (mg/kg)	Potassium (mg/kg)	Selenium (mg/kg)	Silver (mg/kg)	Sodium (mg/kg)
Avg.(log transformed)					4.818	-2.996	2.408	6.376	-0.564	-0.457	4.636
St. Dev.(log transformed)					1.539	0.541	0.602	0.512	0.381	0.103	1.033
H					3.863	1.91	1.954	2.127	1.808	1.687	2.908
95th UCL of the Mean					2249.54	0.07	15.81	917.31	0.68	0.65	418.52

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Strontium (mg/kg)	Thallium (mg/kg)		Vanadium (mg/kg)	Zinc (mg/kg)
16SD01	5/2/93	0:00	0	Primary		0.86	X+		
16SD01	2/21/95	14:20	0	Primary		0.46	X+		55.6
16SD02	4/14/93	0:00	0	Primary		0.635	X+		
16SD02	2/28/95	10:00	0	Primary		0.345	X+		28.9
16SD03	4/14/93	0:00	0	Primary		0.65	X+		
16SD03	2/20/95	16:05	0	Primary		0.78	X+		24
16SD06	2/20/95	16:35	0	Primary		0.73	X+		40.4
16SD07	2/21/95	14:05	0	Primary		0.395	X+		168
16SD08	2/21/95	9:30	0	Primary		0.325	X+		21.6
16SD09	2/21/95	10:55	0	Primary		0.335	X+		57
16SD10	2/21/95	17:10	0	Primary		0.325	X+		13
16SD11	2/28/95	11:15	0	Primary		0.325	X+		29.4
16WW01	5/4/93	0:00	0	Primary		0.595	X+		
16WW01	5/4/93	0:00	0	Duplicate		0.58	X+		
16WW01-A				Avg.		0.5875			
16WW01	5/4/93	0:00	5	Primary		0.615	X+		
16WW01	5/4/93	0:00	10	Primary		0.625	X+		
16WW02	5/12/93	0:00	0	Primary		5.96			
16WW02	5/12/93	0:00	0	Duplicate		0.59	X+		
16WW02-A				Avg.		3.275			
16WW02	5/12/93	0:00	5	Primary		0.615	X+		
16WW03	5/4/93	0:00	0	Primary		1.89			
16WW03	5/4/93	0:00	5	Primary		0.595	X+		
16WW03	5/4/93	0:00	10	Primary		1.32			
16WW03	5/4/93	0:00	15	Primary		0.635	X+		
16WW04	5/12/93	0:00	0	Primary		1.64			
16WW04	5/12/93	0:00	0	Duplicate		1.35			
16WW04-A				Avg.		1.495			
16WW04	5/12/93	0:00	5	Primary		0.59	X+		
16WW04	5/12/93	0:00	10	Primary		0.6	X+		
16WW04	5/12/93	0:00	10	Duplicate		0.61	X+		
16WW04-A				Avg.		0.605			
16WW05	5/4/93	0:00	0	Primary		0.675	X+		
16WW05	5/4/93	0:00	5	Primary		0.57	X+		
16WW05	5/4/93	0:00	10	Primary		0.58	X+		
16WW05	5/4/93	0:00	15	Primary		1.19			
16WW05	5/4/93	0:00	20	Primary		0.64	X+		

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Strontium (mg/kg)	Thallium (mg/kg)		Vanadium (mg/kg)	Zinc (mg/kg)
16WW05	5/4/93	0:00	25	Primary		0.64	X+		
16WW06	5/5/93	0:00	0	Primary		2			
16WW06	5/5/93	0:00	5	Primary		2.06			
16WW06	5/5/93	0:00	10	Primary		0.595	X+		
16WW06	5/5/93	0:00	15	Primary		1.35			
16WW06	5/5/93	0:00	20	Primary		1.53			
16WW27	5/17/97	9:15	0	Primary	34.1	0.65	X+	16	27.3
16WW27	5/17/97	10:17	1	Primary	6.7	0.6	X+	14.3	17.3
16WW31	5/29/97	8:27	0	Primary	2.4	0.6	X+	33.5	13.3
16WW31	5/29/97	8:55	1	Primary	6.0	0.25		38.9	11.9
16WW31	5/29/97	10:09	5	Primary	20.2	0.6	X+	8.5	31
16WW31	5/29/97	11:10	14	Primary	13.4	0.55	X+	8.7	22.1
16WW35	6/25/97	9:50	0	Primary	11.2	0.55	X+	30.6	22.2
16WW35	6/25/97	9:50	0	Duplicate U+	12.7	0.6	X+	33	20.9
16WW35-A				Avg.	11.95	0.575		31.8	21.55
16WW35	6/25/97	10:05	1	Primary	10.3	0.6	X+	20	18.3
16WW35	6/25/97	11:15	5	Primary	26.6	0.35		33.1	45.3
16WW37	5/31/97	15:55	0	Primary	62.7	0.6	X+	22.5	38.9
16WW37	5/31/97	16:10	1	Primary	26.7	0.18		43.3	27.3
16WW37	5/31/97	16:35	5	Primary	7.6	0.6	X+	9.37	16.3
16WW37	6/1/97	10:01	14	Primary	23.5	0.6	X+	17.8	38.8

NATURAL LOGS

16SD01						-0.151			
16SD01						-0.777			4.018
16SD02						-0.454			
16SD02						-1.064			3.364
16SD03						-0.431			
16SD03						-0.248			3.178
16SD06						-0.315			3.699
16SD07						-0.929			5.124
16SD08						-1.124			3.073
16SD09						-1.094			4.043
16SD10						-1.124			2.565
16SD11						-1.124			3.381
16WW01-A						-0.532			
16WW01						-0.486			
16WW01						-0.47			

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	RESULT DEPTH (ft	TYPE	Strontium (mg/kg)	Thallium (mg/kg)	Vanadium (mg/kg)	Zinc (mg/kg)
16WW02-A						1.186		
16WW02						-0.486		
16WW03						0.637		
16WW03						-0.519		
16WW03						0.278		
16WW03						-0.454		
16WW04-A						0.402		
16WW04						-0.528		
16WW04-A						-0.503		
16WW05						-0.393		
16WW05						-0.562		
16WW05						-0.545		
16WW05						0.174		
16WW05						-0.446		
16WW05						-0.446		
16WW06						0.693		
16WW06						0.723		
16WW06						-0.519		
16WW06						0.3		
16WW06						0.425		
16WW27					3.529	-0.431	2.773	3.307
16WW27					1.902	-0.511	2.66	2.851
16WW31					0.875	-0.511	3.512	2.588
16WW31					1.792	-1.386	3.661	2.477
16WW31					3.006	-0.511	2.14	3.434
16WW31					2.595	-0.598	2.163	3.096
16WW35-A					2.481	-0.553	3.459	3.07
16WW35					2.332	-0.511	2.996	2.907
16WW35					3.281	-1.05	3.5	3.813
16WW37					4.138	-0.511	3.114	3.661
16WW37					3.285	-1.715	3.768	3.307
16WW37					2.028	-0.511	2.238	2.791
16WW37					3.157	-0.511	2.879	3.658
STATISTICS								
Total No.					13	48	13	22
Min.					2.4	0.18	8.5	11.9
Max.					62.7	5.96	43.3	168

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Metals
(All Soil Depths)

SITE	DATE	TIME	RESULT DEPTH (ft TYPE	Strontium (mg/kg)	Thallium (mg/kg)	Vanadium (mg/kg)	Zinc (mg/kg)
Avg.(log transformed)				2.646	-0.421	2.989	3.337
St. Dev.(log transformed)				0.873	0.566	0.575	0.601
H				2.636	1.928	2.204	2.076
95th UCL of the Mean				40.10	0.90	33.79	44.25

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,1,1,2-Tetra chloroethane (ug/kg)		1,1,1-trichloro ethane (ug/kg)		1,1,2,2-Tetra chloroethane (ug/kg)		1,1,2-Trichloro ethane (ug/kg)		1,1-Dichloro ethane (ug/kg)		1,1-Dichloro ethene (ug/kg)		1,1-Dichloro propene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary			4.3	X+	4.3	X+	4.3	X+	4.3	X+	4.3	X+		
16SD01	2/21/95	14:20	0	Primary			4.5	X+	4.5	X+	4.5	X+	4.5	X+	4.5	X+		
16SD02	4/14/93	0:00	0	Primary			3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+		
16SD02	2/28/95	10:00	0	Primary			3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD03	4/14/93	0:00	0	Primary			3.25	X+	3.25	X+	3.25	X+	3.25	X+	3.25	X+		
16SD03	2/20/95	16:05	0	Primary			4	X+	4	X+	4	X+	4	X+	4	X+		
16SD06	2/20/95	16:35	0	Primary			3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD07	2/21/95	14:05	0	Primary			4	X+	4	X+	4	X+	4	X+	4	X+		
16SD08	2/21/95	9:30	0	Primary			3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD09	2/21/95	10:55	0	Primary			3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD10	2/21/95	17:10	0	Primary			3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD11	2/28/95	11:15	0	Primary			3	X+	3	X+	3	X+	3	X+	3	X+		
16WW01	5/4/93	0:00	0	Primary			3	X+	3	X+	3	X+	3	X+	3	X+		
16WW01	5/4/93	0:00	0	Duplicate 1			2.9	X+	2.9	X+	2.9	X+	2.9	X+	2.9	X+		
16WW01-A				Avg.			2.95		2.95		2.95		2.95		2.95			
16WW01	5/4/93	0:00	5	Primary			3.1	X+	3.1	X+	3.1	X+	3.1	X+	3.1	X+		
16WW01	5/4/93	0:00	10	Primary			3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+		
16WW02	5/12/93	0:00	0	Primary			2.9	X+	2.9	X+	2.9	X+	2.9	X+	2.9	X+		
16WW02	5/12/93	0:00	0	Duplicate 1			2.95	X+	2.95	X+	2.95	X+	2.95	X+	2.95	X+		
16WW02-A				Avg.			2.925		2.925		2.925		2.925		2.925			
16WW02	5/12/93	0:00	5	Primary			3.1	X+	3.1	X+	3.1	X+	3.1	X+	3.1	X+		
16WW03	5/4/93	0:00	0	Primary			3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+		
16WW03	5/4/93	0:00	5	Primary			3	X+	3	X+	3	X+	3	X+	3	X+		
16WW03	5/4/93	0:00	10	Primary			3.05	X+	3.05	X+	3.05	X+	3.05	X+	3.05	X+		
16WW03	5/4/93	0:00	15	Primary			3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+		
16WW04	5/12/93	0:00	0	Primary			3	X+	3	X+	3	X+	3	X+	3	X+		
16WW04	5/12/93	0:00	0	Duplicate 1			3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+		
16WW04-A				Avg.			3.075		3.075		3.075		3.075		3.075			
16WW04	5/12/93	0:00	5	Primary			2.95	X+	2.95	X+	2.95	X+	2.95	X+	2.95	X+		
16WW04	5/12/93	0:00	10	Primary			3.05	X+	3.05	X+	3.05	X+	3.05	X+	3.05	X+		
16WW05	5/4/93	0:00	0	Primary			3.4	X+	3.4	X+	3.4	X+	3.4	X+	3.4	X+		
16WW05	5/4/93	0:00	5	Primary			2.85	X+	2.85	X+	2.85	X+	2.85	X+	2.85	X+		
16WW05	5/4/93	0:00	10	Primary			2.9	X+	2.9	X+	2.9	X+	2.9	X+	2.9	X+		
16WW05	5/4/93	0:00	15	Primary			3	X+	3	X+	3	X+	3	X+	3	X+		
16WW05	5/4/93	0:00	20	Primary			3.2	X+	3.2	X+	3.2	X+	3.2	X+	3.2	X+		
16WW05	5/4/93	0:00	25	Primary			3.2	X+	3.2	X+	3.2	X+	3.2	X+	3.2	X+		
16WW06	5/5/93	0:00	0	Primary			3.25	X+	3.25	X+	3.25	X+	3.25	X+	3.25	X+		
16WW06	5/5/93	0:00	5	Primary			3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+		
16WW06	5/5/93	0:00	10	Primary			3	X+	3	X+	3	X+	3	X+	3	X+		
16WW06	5/5/93	0:00	15	Primary			2.95	X+	2.95	X+	2.95	X+	2.95	X+	2.95	X+		
16WW06	5/5/93	0:00	20	Primary			3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+		
16WW27	5/17/97	9:15	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+

Longhorn Army Ammuntion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,1,1,2-Tetra chloroethane (ug/kg)		1,1,1-trichloro ethane (ug/kg)		1,1,2,2-Tetra chloroethane (ug/kg)		1,1,2-Trichloro ethane (ug/kg)		1,1-Dichloro ethane (ug/kg)		1,1-Dichloro ethene (ug/kg)		1,1-Dichloro propene (ug/kg)	
16WW27	5/17/97	10:17	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	8:55	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	10:09	5	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	11:10	14	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35-A				Avg.	2.5		2.5		2.5		2.5		2.5		2.5		2.5	
16WW35	6/25/97	10:05	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW35	6/25/97	11:15	5	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37	5/31/97	16:10	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37	5/31/97	16:35	5	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37	6/1/97	10:01	14	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+

NATURAL LOGS

16SD01	1.459	1.459	1.459	1.459	1.459
16SD01	1.504	1.504	1.504	1.504	1.504
16SD02	1.147	1.147	1.147	1.147	1.147
16SD02	1.253	1.253	1.253	1.253	1.253
16SD03	1.179	1.179	1.179	1.179	1.179
16SD03	1.386	1.386	1.386	1.386	1.386
16SD06	1.253	1.253	1.253	1.253	1.253
16SD07	1.386	1.386	1.386	1.386	1.386
16SD08	1.253	1.253	1.253	1.253	1.253
16SD09	1.253	1.253	1.253	1.253	1.253
16SD10	1.253	1.253	1.253	1.253	1.253
16SD11	1.099	1.099	1.099	1.099	1.099
16WW01-A	1.082	1.082	1.082	1.082	1.082
16WW01	1.131	1.131	1.131	1.131	1.131
16WW01	1.147	1.147	1.147	1.147	1.147
16WW02-A	1.073	1.073	1.073	1.073	1.073
16WW02	1.131	1.131	1.131	1.131	1.131
16WW03	1.147	1.147	1.147	1.147	1.147
16WW03	1.099	1.099	1.099	1.099	1.099
16WW03	1.115	1.115	1.115	1.115	1.115
16WW03	1.147	1.147	1.147	1.147	1.147
16WW04-A	1.123	1.123	1.123	1.123	1.123
16WW04	1.082	1.082	1.082	1.082	1.082
16WW04	1.115	1.115	1.115	1.115	1.115
16WW05	1.224	1.224	1.224	1.224	1.224
16WW05	1.047	1.047	1.047	1.047	1.047

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,1,1,2-Tetra chloroethane (ug/kg)	1,1,1-trichloro ethane (ug/kg)	1,1,2,2-Tetra chloroethane (ug/kg)	1,1,2-Trichloro ethane (ug/kg)	1,1-Dichloro ethane (ug/kg)	1,1-Dichloro ethene (ug/kg)	1,1-Dichloro propene (ug/kg)
16WW05						1.065	1.065	1.065	1.065	1.065	
16WW05						1.099	1.099	1.099	1.099	1.099	
16WW05						1.163	1.163	1.163	1.163	1.163	
16WW05						1.163	1.163	1.163	1.163	1.163	
16WW06						1.179	1.179	1.179	1.179	1.179	
16WW06						1.147	1.147	1.147	1.147	1.147	
16WW06						1.099	1.099	1.099	1.099	1.099	
16WW06						1.082	1.082	1.082	1.082	1.082	
16WW06						1.147	1.147	1.147	1.147	1.147	
16WW27					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW27					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW31					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW31					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW31					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW31					0.916	0.916	0.916	0.916	0.916	0.916	0.916
16WW35-A					0.916	0.916	0.916	0.916	0.916	0.916	0.916
16WW35					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW35					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW37					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW37					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW37					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW37					1.099	1.099	1.099	1.099	1.099	1.099	1.099
STATISTICS											
Total No.					13	48	48	48	48	48	13
Min.					2.5	2.5	2.5	2.5	2.5	2.5	2.5
Max.					3	4.5	4.5	4.5	4.5	4.5	3
Avg.(log transformed)					1.071	1.149	1.149	1.149	1.149	1.149	1.071
St. Dev.(log transformed)					0.069	0.111	0.111	0.111	0.111	0.111	0.069
H											
95th UCL of Mean					2.93	3.17	3.17	3.17	3.17	3.17	2.93

Longhorn Army Ammuntion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,2,3-Trichloro benzene (ug/kg)		1,2,3-Trichloro propane (ug/kg)		1,2,4-Trichloro benzene (ug/kg)		1,2-Dichloro ethane (ug/kg)		DCE12TOT (ug/kg)		1,2-Dichloro propane (ug/kg)		1,3-Dichloro propane (ug/kg)	
16SD01	5/2/93	0:00	0	Primary			4.3	X+			4.3	X+			4.3	X+		
16SD01	2/21/95	14:20	0	Primary			4.5	X+			4.5	X+	4.5	X+	4.5	X+		
16SD02	4/14/93	0:00	0	Primary			3.15	X+			3.15	X+			3.15	X+		
16SD02	2/28/95	10:00	0	Primary			3.5	X+			3.5	X+	3.5	X+	3.5	X+		
16SD03	4/14/93	0:00	0	Primary			3.25	X+			3.25	X+			3.25	X+		
16SD03	2/20/95	16:05	0	Primary			4	X+			4	X+	4	X+	4	X+		
16SD06	2/20/95	16:35	0	Primary			3.5	X+			3.5	X+	3.5	X+	3.5	X+		
16SD07	2/21/95	14:05	0	Primary			4	X+			4	X+	4	X+	4	X+		
16SD08	2/21/95	9:30	0	Primary			3.5	X+			3.5	X+	3.5	X+	3.5	X+		
16SD09	2/21/95	10:55	0	Primary			3.5	X+			3.5	X+	3.5	X+	3.5	X+		
16SD10	2/21/95	17:10	0	Primary			3.5	X+			3.5	X+	3.5	X+	3.5	X+		
16SD11	2/28/95	11:15	0	Primary			3	X+			3	X+	3	X+	3	X+		
16WW01	5/4/93	0:00	0	Primary			3	X+			3	X+			3	X+		
16WW01	5/4/93	0:00	0	Duplicate			2.9	X+			2.9	X+			2.9	X+		
16WW01-A				Avg.			2.95				2.95				2.95			
16WW01	5/4/93	0:00	5	Primary			3.1	X+			3.1	X+			3.1	X+		
16WW01	5/4/93	0:00	10	Primary			3.15	X+			3.15	X+			3.15	X+		
16WW02	5/12/93	0:00	0	Primary			2.9	X+			2.9	X+			2.9	X+		
16WW02	5/12/93	0:00	0	Duplicate			2.95	X+			2.95	X+			2.95	X+		
16WW02-A				Avg.			2.925				2.925				2.925			
16WW02	5/12/93	0:00	5	Primary			3.1	X+			3.1	X+			3.1	X+		
16WW03	5/4/93	0:00	0	Primary			3.15	X+			3.15	X+			3.15	X+		
16WW03	5/4/93	0:00	5	Primary			3	X+			3	X+			3	X+		
16WW03	5/4/93	0:00	10	Primary			3.05	X+			3.05	X+			3.05	X+		
16WW03	5/4/93	0:00	15	Primary			3.15	X+			3.15	X+			3.15	X+		
16WW04	5/12/93	0:00	0	Primary			3	X+			3	X+			3	X+		
16WW04	5/12/93	0:00	0	Duplicate			3.15	X+			3.15	X+			3.15	X+		
16WW04-A				Avg.			3.075				3.075				3.075			
16WW04	5/12/93	0:00	5	Primary			2.95	X+			2.95	X+			2.95	X+		
16WW04	5/12/93	0:00	10	Primary			3.05	X+			3.05	X+			3.05	X+		
16WW05	5/4/93	0:00	0	Primary			3.4	X+			3.4	X+			3.4	X+		
16WW05	5/4/93	0:00	5	Primary			2.85	X+			2.85	X+			2.85	X+		
16WW05	5/4/93	0:00	10	Primary			2.9	X+			2.9	X+			2.9	X+		
16WW05	5/4/93	0:00	15	Primary			3	X+			3	X+			3	X+		
16WW05	5/4/93	0:00	20	Primary			3.2	X+			3.2	X+			3.2	X+		
16WW05	5/4/93	0:00	25	Primary			3.2	X+			3.2	X+			3.2	X+		
16WW06	5/5/93	0:00	0	Primary			3.25	X+			3.25	X+			3.25	X+		
16WW06	5/5/93	0:00	5	Primary			3.15	X+			3.15	X+			3.15	X+		
16WW06	5/5/93	0:00	10	Primary			3	X+			3	X+			3	X+		
16WW06	5/5/93	0:00	15	Primary			2.95	X+			2.95	X+			2.95	X+		
16WW06	5/5/93	0:00	20	Primary			3.15	X+			3.15	X+			3.15	X+		
16WW27	5/17/97	9:15	0	Primary	3	X+	9.5	X+	3	X+	3	X+			3	X+	3	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,2,3-Trichloro benzene (ug/kg)		1,2,3-Trichloro propane (ug/kg)		1,2,4-Trichloro benzene (ug/kg)		1,2-Dichloro ethane (ug/kg)		DCE12TOT (ug/kg)	1,2-Dichloro propane (ug/kg)		1,3-Dichloro propane (ug/kg)	
16WW27	5/17/97	10:17	1	Primary	3	X+	9	X+	3	X+	3	X+		3	X+	3	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	9	X+	3	X+	3	X+		3	X+	3	X+
16WW31	5/29/97	8:55	1	Primary	3	X+	9	X+	3	X+	3	X+		3	X+	3	X+
16WW31	5/29/97	10:09	5	Primary	3	X+	9	X+	3	X+	3	X+		3	X+	3	X+
16WW31	5/29/97	11:10	14	Primary	2.5	X+	8	X+	2.5	X+	2.5	X+		2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	8	X+	2.5	X+	2.5	X+		2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	7.5	X+	2.5	X+	2.5	X+		2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	7.5	X+	2.5	X+	2.5	X+		2.5	X+	2.5	X+
16WW35-A				Avg.	2.5		7.666667		2.5		2.5			2.5		2.5	
16WW35	6/25/97	10:05	1	Primary	3	X+	9.5	X+	3	X+	3	X+		3	X+	3	X+
16WW35	6/25/97	11:15	5	Primary	3	X+	9.5	X+	3	X+	3	X+		3	X+	3	X+
16WW37	5/31/97	15:55	0	Primary	3	X+	9.5	X+	3	X+	3	X+		3	X+	3	X+
16WW37	5/31/97	16:10	1	Primary	3	X+	9.5	X+	3	X+	3	X+		3	X+	3	X+
16WW37	5/31/97	16:35	5	Primary	3	X+	9	X+	3	X+	3	X+		3	X+	3	X+
16WW37	6/1/97	10:01	14	Primary	3	X+	9	X+	3	X+	3	X+		3	X+	3	X+

NATURAL LOGS

16SD01				1.459		1.459			1.459				
16SD01				1.504		1.504		1.504	1.504				
16SD02				1.147		1.147			1.147				
16SD02				1.253		1.253		1.253	1.253				
16SD03				1.179		1.179			1.179				
16SD03				1.386		1.386		1.386	1.386				
16SD06				1.253		1.253		1.253	1.253				
16SD07				1.386		1.386		1.386	1.386				
16SD08				1.253		1.253		1.253	1.253				
16SD09				1.253		1.253		1.253	1.253				
16SD10				1.253		1.253		1.253	1.253				
16SD11				1.099		1.099		1.099	1.099				
16WW01-A				1.082		1.082			1.082				
16WW01				1.131		1.131			1.131				
16WW01				1.147		1.147			1.147				
16WW02-A				1.073		1.073			1.073				
16WW02				1.131		1.131			1.131				
16WW03				1.147		1.147			1.147				
16WW03				1.099		1.099			1.099				
16WW03				1.115		1.115			1.115				
16WW03				1.147		1.147			1.147				
16WW04-A				1.123		1.123			1.123				
16WW04				1.082		1.082			1.082				
16WW04				1.115		1.115			1.115				
16WW05				1.224		1.224			1.224				
16WW05				1.047		1.047			1.047				

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	TYPE	RESULT	1,2,3-Trichloro benzene (ug/kg)	1,2,3-Trichloro propane (ug/kg)	1,2,4-Trichloro benzene (ug/kg)	1,2-Dichloro ethane (ug/kg)	DCE12TOT (ug/kg)	1,2-Dichloro propane (ug/kg)	1,3-Dichloro propane (ug/kg)
16WW05					1.065		1.065		1.065		1.065	
16WW05					1.099		1.099		1.099		1.099	
16WW05					1.163		1.163		1.163		1.163	
16WW05					1.163		1.163		1.163		1.163	
16WW06					1.179		1.179		1.179		1.179	
16WW06					1.147		1.147		1.147		1.147	
16WW06					1.099		1.099		1.099		1.099	
16WW06					1.082		1.082		1.082		1.082	
16WW06					1.147		1.147		1.147		1.147	
16WW27						1.099	2.251	1.099	1.099		1.099	1.099
16WW27						1.099	2.197	1.099	1.099		1.099	1.099
16WW31						1.099	2.197	1.099	1.099		1.099	1.099
16WW31						1.099	2.197	1.099	1.099		1.099	1.099
16WW31						1.099	2.197	1.099	1.099		1.099	1.099
16WW31						0.916	2.079	0.916	0.916		0.916	0.916
16WW35-A						0.916	2.037	0.916	0.916		0.916	0.916
16WW35						1.099	2.251	1.099	1.099		1.099	1.099
16WW35						1.099	2.251	1.099	1.099		1.099	1.099
16WW37						1.099	2.251	1.099	1.099		1.099	1.099
16WW37						1.099	2.251	1.099	1.099		1.099	1.099
16WW37						1.099	2.197	1.099	1.099		1.099	1.099
16WW37						1.099	2.197	1.099	1.099		1.099	1.099
STATISTICS												
Total No.						13	48	13	48	9	48	13
Min.						2.5	2.85	2.5	2.5	3	2.5	2.5
Max.						3	9.5	3	4.5	4.5	4.5	3
Avg.(log transformed)						1.071	1.454	1.071	1.149	1.293	1.149	1.071
St. Dev.(log transformed)						0.069	0.468	0.069	0.111	0.116	0.111	0.069
H												
95th UCL of Mean						2.93	4.78	2.93	3.17	3.67	3.17	2.93

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,4-Dichloro-2-butene (ug/kg)		1,4-Dioxane (ug/kg)		2,2-Dichloro propane (ug/kg)		2-Hexanone (ug/kg)		Acetone (ug/kg)		Acrolein (ug/kg)		Acrylonitrile (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	8.5	X+					8.5	X+	8.5	X+	8.5	X+	8.5	X+
16SD01	2/21/95	14:20	0	Primary	9	X+					9	X+	103		46	X+	46	X+
16SD02	4/14/93	0:00	0	Primary	6.5	X+					6.5	X+	6.5	X+	6.5	X+	6.5	X+
16SD02	2/28/95	10:00	0	Primary	7	X+					7	X+	7	X+	34.5	X+	34.5	X+
16SD03	4/14/93	0:00	0	Primary	6.5	X+					6.5	X+	6.5	X+	6.5	X+	6.5	X+
16SD03	2/20/95	16:05	0	Primary	8	X+					8	X+	8	X+	39	X+	39	X+
16SD06	2/20/95	16:35	0	Primary	7.5	X+					7.5	X+	26		36.5	X+	36.5	X+
16SD07	2/21/95	14:05	0	Primary	8	X+					8	X+	8	X+	39.5	X+	39.5	X+
16SD08	2/21/95	9:30	0	Primary	6.5	X+					6.5	X+	6.5	X+	32.5	X+	32.5	X+
16SD09	2/21/95	10:55	0	Primary	6.5	X+					6.5	X+	6.5	X+	33.5	X+	33.5	X+
16SD10	2/21/95	17:10	0	Primary	6.5	X+					6.5	X+	6.5	X+	32.5	X+	32.5	X+
16SD11	2/28/95	11:15	0	Primary	6.5	X+					6.5	X+	22		32.5	X+	32.5	X+
16WW01	5/4/93	0:00	0	Primary	6	X+					6	X+	6	X+	6	X+	6	X+
16WW01	5/4/93	0:00	0	Duplicate	6	X+					6	X+	6	X+	6	X+	6	X+
16WW01-A				Avg.	6						6		6		6		6	
16WW01	5/4/93	0:00	5	Primary	6	X+					6	X+	6	X+	6	X+	6	X+
16WW01	5/4/93	0:00	10	Primary	6.5	X+					6.5	X+	6.5	X+	6.5	X+	6.5	X+
16WW02	5/12/93	0:00	0	Primary	6	X+					6	X+	6	X+	6	X+	6	X+
16WW02	5/12/93	0:00	0	Duplicate	6	X+					6	X+	6	X+	6	X+	6	X+
16WW02-A				Avg.	6						6		6		6		6	
16WW02	5/12/93	0:00	5	Primary	6	X+					6	X+	6	X+	6	X+	6	X+
16WW03	5/4/93	0:00	0	Primary	6.5	X+					6.5	X+	6.5	X+	6.5	X+	6.5	X+
16WW03	5/4/93	0:00	5	Primary	6	X+					6	X+	6	X+	6	X+	6	X+
16WW03	5/4/93	0:00	10	Primary	6	X+					6	X+	6	X+	6	X+	6	X+
16WW03	5/4/93	0:00	15	Primary	6.5	X+					6.5	X+	6.5	X+	6.5	X+	6.5	X+
16WW04	5/12/93	0:00	0	Primary	6	X+					6	X+	6	X+	6	X+	6	X+
16WW04	5/12/93	0:00	0	Duplicate	6.5	X+					6.5	X+	6.5	X+	6.5	X+	6.5	X+
16WW04-A				Avg.	6.25						6.25		6.25		6.25		6.25	
16WW04	5/12/93	0:00	5	Primary	6	X+					6	X+	57		6	X+	6	X+
16WW04	5/12/93	0:00	10	Primary	6	X+					6	X+	6	X+	6	X+	6	X+
16WW05	5/4/93	0:00	0	Primary	7	X+					7	X+	7	X+	7	X+	7	X+
16WW05	5/4/93	0:00	5	Primary	5.5	X+					5.5	X+	5.5	X+	5.5	X+	5.5	X+
16WW05	5/4/93	0:00	10	Primary	6	X+					6	X+	6	X+	6	X+	6	X+
16WW05	5/4/93	0:00	15	Primary	6	X+					6	X+	6	X+	6	X+	6	X+
16WW05	5/4/93	0:00	20	Primary	6.5	X+					6.5	X+	6.5	X+	6.5	X+	6.5	X+
16WW05	5/4/93	0:00	25	Primary	6.5	X+					6.5	X+	6.5	X+	6.5	X+	6.5	X+
16WW06	5/5/93	0:00	0	Primary	6.5	X+					6.5	X+	6.5	X+	6.5	X+	6.5	X+
16WW06	5/5/93	0:00	5	Primary	6.5	X+					6.5	X+	6.5	X+	6.5	X+	6.5	X+
16WW06	5/5/93	0:00	10	Primary	6	X+					6	X+	6	X+	6	X+	6	X+
16WW06	5/5/93	0:00	15	Primary	6	X+					6	X+	6	X+	6	X+	6	X+
16WW06	5/5/93	0:00	20	Primary	6.5	X+					6.5	X+	6.5	X+	6.5	X+	6.5	X+
16WW27	5/17/97	9:15	0	Primary	3	X+	650	X+	9.5	X+	13	X+	13	X+	65	X+	65	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,4-Dichloro-2-butene (ug/kg)		1,4-Dioxane (ug/kg)		2,2-Dichloro propane (ug/kg)		2-Hexanone (ug/kg)		Acetone (ug/kg)		Acrolein (ug/kg)		Acrylonitrile (ug/kg)	
16WW27	5/17/97	10:17	1	Primary	3	X+	600	X+	9	X+	12	X+	12	X+	60	X+	60	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	600	X+	93.5	X+	12	X+	12	X+	60	X+	60	X+
16WW31	5/29/97	8:55	1	Primary	3	X+	600	X+	9	X+	12	X+	12	X+	60	X+	60	X+
16WW31	5/29/97	10:09	5	Primary	3	X+	600	X+	9	X+	12	X+	14		60	X+	60	X+
16WW31	5/29/97	11:10	14	Primary	2.5	X+	550	X+	8	X+	11	X+	4	U+	55	X+	55	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	550	X+	8	X+	10.5	X+	10.5	X+	55	X+	55	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	500	X+	7.5	X+	10	X+	20		50	X+	50	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	500	X+	7.5	X+	10	X+	10	X+	50	X+	50	X+
16WW35-A				Avg.	2.5		516.6667		7.666667		10.16667		13.5		51.66667		51.66667	
16WW35	6/25/97	10:05	1	Primary	3	X+	600	X+	9.5	X+	12.5	X+	13		60	X+	60	X+
16WW35	6/25/97	11:15	5	Primary	3	X+	600	X+	9.5	X+	12.5	X+	25		60	X+	60	X+
16WW37	5/31/97	15:55	0	Primary	3	X+	600	X+	9.5	X+	12.5	X+	12.5	X+	60	X+	60	X+
16WW37	5/31/97	16:10	1	Primary	3	X+	650	X+	9.5	X+	12.5	X+	12.5	X+	65	X+	65	X+
16WW37	5/31/97	16:35	5	Primary	3	X+	600	X+	9	X+	12	X+	12	X+	60	X+	60	X+
16WW37	6/1/97	10:01	14	Primary	3	X+	600	X+	9	X+	12	X+	12	X+	60	X+	60	X+

NATURAL LOGS

16SD01	2.14	2.14	2.14	2.14	2.14	2.14
16SD01	2.197	2.197	4.635	3.829	3.829	
16SD02	1.872	1.872	1.872	1.872	1.872	
16SD02	1.946	1.946	1.946	3.541	3.541	
16SD03	1.872	1.872	1.872	1.872	1.872	
16SD03	2.079	2.079	2.079	3.664	3.664	
16SD06	2.015	2.015	3.258	3.597	3.597	
16SD07	2.079	2.079	2.079	3.676	3.676	
16SD08	1.872	1.872	1.872	3.481	3.481	
16SD09	1.872	1.872	1.872	3.512	3.512	
16SD10	1.872	1.872	1.872	3.481	3.481	
16SD11	1.872	1.872	3.091	3.481	3.481	
16WW01-A	1.792	1.792	1.792	1.792	1.792	
16WW01	1.792	1.792	1.792	1.792	1.792	
16WW01	1.872	1.872	1.872	1.872	1.872	
16WW02-A	1.792	1.792	1.792	1.792	1.792	
16WW02	1.792	1.792	1.792	1.792	1.792	
16WW03	1.872	1.872	1.872	1.872	1.872	
16WW03	1.792	1.792	1.792	1.792	1.792	
16WW03	1.792	1.792	1.792	1.792	1.792	
16WW03	1.872	1.872	1.872	1.872	1.872	
16WW04-A	1.833	1.833	1.833	1.833	1.833	
16WW04	1.792	1.792	4.043	1.792	1.792	
16WW04	1.792	1.792	1.792	1.792	1.792	
16WW05	1.946	1.946	1.946	1.946	1.946	
16WW05	1.705	1.705	1.705	1.705	1.705	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,4-Dichloro-2-butene (ug/kg)	1,4-Dioxane (ug/kg)	2,2-Dichloro propane (ug/kg)	2-Hexanone (ug/kg)	Acetone (ug/kg)	Acrolein (ug/kg)	Acrylonitrile (ug/kg)
16WW05					1.792			1.792	1.792	1.792	1.792
16WW05					1.792			1.792	1.792	1.792	1.792
16WW05					1.872			1.872	1.872	1.872	1.872
16WW05					1.872			1.872	1.872	1.872	1.872
16WW06					1.872			1.872	1.872	1.872	1.872
16WW06					1.872			1.872	1.872	1.872	1.872
16WW06					1.792			1.792	1.792	1.792	1.792
16WW06					1.792			1.792	1.792	1.792	1.792
16WW06					1.872			1.872	1.872	1.872	1.872
16WW27					1.099	6.477	2.251	2.565	2.565	4.174	4.174
16WW27					1.099	6.397	2.197	2.485	2.485	4.094	4.094
16WW31					1.099	6.397	4.538	2.485	2.485	4.094	4.094
16WW31					1.099	6.397	2.197	2.485	2.485	4.094	4.094
16WW31					1.099	6.397	2.197	2.485	2.639	4.094	4.094
16WW31					0.916	6.31	2.079	2.398	1.386	4.007	4.007
16WW35-A					0.916	6.247	2.037	2.319	2.603	3.945	3.945
16WW35					1.099	6.397	2.251	2.526	2.565	4.094	4.094
16WW35					1.099	6.397	2.251	2.526	3.219	4.094	4.094
16WW37					1.099	6.397	2.251	2.526	2.526	4.094	4.094
16WW37					1.099	6.477	2.251	2.526	2.526	4.174	4.174
16WW37					1.099	6.397	2.197	2.485	2.485	4.094	4.094
16WW37					1.099	6.397	2.197	2.485	2.485	4.094	4.094
STATISTICS											
Total No.					48	13	13	48	48	48	48
Min.					2.5	500	7.5	5.5	4	5.5	5.5
Max.					9	650	93.5	13	103	65	65
Avg.(log transformed)					1.658	6.391	2.376	2.041	2.193	2.776	2.776
St. Dev.(log transformed)					0.375	0.059	0.653	0.29	0.618	1.045	1.045
H									1.967		
95th UCL of Mean					5.63	597.49	13.32	8.03	12.95	27.72	27.72

Longhorn Army Ammuntion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Benzene (ug/kg)		1,2,4-Trimethyl benzene (ug/kg)		1,3,5-Trimethyl benzene (ug/kg)		Isopropyl benzene (ug/kg)		Bromobenzene (ug/kg)		Bromodichloro methane (ug/kg)		Bromoform (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	4.3	X+									4.3	X+		
16SD01	2/21/95	14:20	0	Primary	4.5	X+									4.5	X+	4.5	X+
16SD02	4/14/93	0:00	0	Primary	3.15	X+									3.15	X+		
16SD02	2/28/95	10:00	0	Primary	3.5	X+									3.5	X+	3.5	X+
16SD03	4/14/93	0:00	0	Primary	3.25	X+									3.25	X+		
16SD03	2/20/95	16:05	0	Primary	4	X+									4	X+	4	X+
16SD06	2/20/95	16:35	0	Primary	3.5	X+									3.5	X+	3.5	X+
16SD07	2/21/95	14:05	0	Primary	4	X+									4	X+	4	X+
16SD08	2/21/95	9:30	0	Primary	3.5	X+									3.5	X+	3.5	X+
16SD09	2/21/95	10:55	0	Primary	3.5	X+									3.5	X+	3.5	X+
16SD10	2/21/95	17:10	0	Primary	3.5	X+									3.5	X+	3.5	X+
16SD11	2/28/95	11:15	0	Primary	3	X+									3	X+	3	X+
16WW01	5/4/93	0:00	0	Primary	3	X+									3	X+		
16WW01	5/4/93	0:00	0	Duplicate	2.9	X+									2.9	X+		
16WW01-A				Avg.	2.95										2.95			
16WW01	5/4/93	0:00	5	Primary	3.1	X+									3.1	X+		
16WW01	5/4/93	0:00	10	Primary	3.15	X+									3.15	X+		
16WW02	5/12/93	0:00	0	Primary	2.9	X+									2.9	X+		
16WW02	5/12/93	0:00	0	Duplicate	2.95	X+									2.95	X+		
16WW02-A				Avg.	2.925										2.925			
16WW02	5/12/93	0:00	5	Primary	3.1	X+									3.1	X+		
16WW03	5/4/93	0:00	0	Primary	3.15	X+									3.15	X+		
16WW03	5/4/93	0:00	5	Primary	3	X+									3	X+		
16WW03	5/4/93	0:00	10	Primary	3.05	X+									3.05	X+		
16WW03	5/4/93	0:00	15	Primary	3.15	X+									3.15	X+		
16WW04	5/12/93	0:00	0	Primary	3	X+									3	X+		
16WW04	5/12/93	0:00	0	Duplicate	3.15	X+									3.15	X+		
16WW04-A				Avg.	3.075										3.075			
16WW04	5/12/93	0:00	5	Primary	2.95	X+									2.95	X+		
16WW04	5/12/93	0:00	10	Primary	3.05	X+									3.05	X+		
16WW05	5/4/93	0:00	0	Primary	3.4	X+									3.4	X+		
16WW05	5/4/93	0:00	5	Primary	2.85	X+									2.85	X+		
16WW05	5/4/93	0:00	10	Primary	2.9	X+									2.9	X+		
16WW05	5/4/93	0:00	15	Primary	3	X+									3	X+		
16WW05	5/4/93	0:00	20	Primary	3.2	X+									3.2	X+		
16WW05	5/4/93	0:00	25	Primary	3.2	X+									3.2	X+		
16WW06	5/5/93	0:00	0	Primary	3.25	X+									3.25	X+		
16WW06	5/5/93	0:00	5	Primary	3.15	X+									3.15	X+		
16WW06	5/5/93	0:00	10	Primary	3	X+									3	X+		
16WW06	5/5/93	0:00	15	Primary	2.95	X+									2.95	X+		
16WW06	5/5/93	0:00	20	Primary	3.15	X+									3.15	X+		
16WW27	5/17/97	9:15	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+

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Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Benzene (ug/kg)		1,2,4-Trimethyl benzene (ug/kg)		1,3,5-Trimethyl benzene (ug/kg)		Isopropyl benzene (ug/kg)		Bromobenzene (ug/kg)		Bromodichloro methane (ug/kg)		Bromoform (ug/kg)	
16WW27	5/17/97	10:17	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	8:55	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	10:09	5	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	11:10	14	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35-A				Avg.	2.5		2.5		2.5		2.5		2.5		2.5		2.5	
16WW35	6/25/97	10:05	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW35	6/25/97	11:15	5	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37	5/31/97	16:10	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37	5/31/97	16:35	5	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37	6/1/97	10:01	14	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+

NATURAL LOGS

16SD01	1.459				1.459	
16SD01	1.504				1.504	1.504
16SD02	1.147				1.147	
16SD02	1.253				1.253	1.253
16SD03	1.179				1.179	
16SD03	1.386				1.386	1.386
16SD06	1.253				1.253	1.253
16SD07	1.386				1.386	1.386
16SD08	1.253				1.253	1.253
16SD09	1.253				1.253	1.253
16SD10	1.253				1.253	1.253
16SD11	1.099				1.099	1.099
16WW01-A	1.082				1.082	
16WW01	1.131				1.131	
16WW01	1.147				1.147	
16WW02-A	1.073				1.073	
16WW02	1.131				1.131	
16WW03	1.147				1.147	
16WW03	1.099				1.099	
16WW03	1.115				1.115	
16WW03	1.147				1.147	
16WW04-A	1.123				1.123	
16WW04	1.082				1.082	
16WW04	1.115				1.115	
16WW05	1.224				1.224	
16WW05	1.047				1.047	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Benzene (ug/kg)	1,2,4-Trimethyl benzene (ug/kg)	1,3,5-Trimethyl benzene (ug/kg)	Isopropyl benzene (ug/kg)	Bromobenzene (ug/kg)	Bromodichloro methane (ug/kg)	Bromoform (ug/kg)
16WW05					1.065					1.065	
16WW05					1.099					1.099	
16WW05					1.163					1.163	
16WW05					1.163					1.163	
16WW06					1.179					1.179	
16WW06					1.147					1.147	
16WW06					1.099					1.099	
16WW06					1.082					1.082	
16WW06					1.147					1.147	
16WW27					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW27					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW31					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW31					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW31					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW31					0.916	0.916	0.916	0.916	0.916	0.916	0.916
16WW35-A					0.916	0.916	0.916	0.916	0.916	0.916	0.916
16WW35					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW35					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW37					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW37					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW37					1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW37					1.099	1.099	1.099	1.099	1.099	1.099	1.099
STATISTICS											
Total No.					48	13	13	13	13	48	22
Min.					2.5	2.5	2.5	2.5	2.5	2.5	2.5
Max.					4.5	3	3	3	3	4.5	4.5
Avg.(log transformed)					1.149	1.071	1.071	1.071	1.071	1.149	1.162
St. Dev.(log transformed)					0.111	0.069	0.069	0.069	0.069	0.111	0.143
H											
95th UCL of Mean					3.17	2.93	2.93	2.93	2.93	3.17	3.23

Longhorn Army Ammuntion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Carbon disulfide (ug/kg)		Carbon tetrachloride (ug/kg)		Chlorobenzene (ug/kg)		Bromochloro methane (ug/kg)		Chloroethane (ug/kg)		Chloroform (ug/kg)		cis-1,2-Dichloroethene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	4.3	X+	4.3	X+	4.3	X+			8.5	X+	4.3	X+	4.3	X+
16SD01	2/21/95	14:20	0	Primary	4.5	X+	4.5	X+	4.5	X+			9	X+	4.5	X+		
16SD02	4/14/93	0:00	0	Primary	3.15	X+	3.15	X+	3.15	X+			6.5	X+	3.15	X+	3.15	X+
16SD02	2/28/95	10:00	0	Primary	3.5	X+	3.5	X+	3.5	X+			7	X+	3.5	X+		
16SD03	4/14/93	0:00	0	Primary	3.25	X+	3.25	X+	3.25	X+			6.5	X+	3.25	X+	3.25	X+
16SD03	2/20/95	16:05	0	Primary	4	X+	4	X+	4	X+			8	X+	4	X+		
16SD06	2/20/95	16:35	0	Primary	3.5	X+	3.5	X+	3.5	X+			7.5	X+	3.5	X+		
16SD07	2/21/95	14:05	0	Primary	4	X+	4	X+	4	X+			8	X+	4	X+		
16SD08	2/21/95	9:30	0	Primary	3.5	X+	3.5	X+	3.5	X+			6.5	X+	3.5	X+		
16SD09	2/21/95	10:55	0	Primary	3.5	X+	3.5	X+	3.5	X+			6.5	X+	3.5	X+		
16SD10	2/21/95	17:10	0	Primary	3.5	X+	3.5	X+	3.5	X+			6.5	X+	3.5	X+		
16SD11	2/28/95	11:15	0	Primary	3	X+	3	X+	3	X+			6.5	X+	3	X+		
16WW01	5/4/93	0:00	0	Primary	3	X+	3	X+	3	X+			6	X+	3	X+	3	X+
16WW01	5/4/93	0:00	0	Duplicate	2.9	X+	2.9	X+	2.9	X+			6	X+	2.9	X+	2.9	X+
16WW01-A				Avg.	2.95		2.95		2.95				6		2.95		2.95	
16WW01	5/4/93	0:00	5	Primary	3.1	X+	3.1	X+	3.1	X+			6	X+	3.1	X+	3.1	X+
16WW01	5/4/93	0:00	10	Primary	3.15	X+	3.15	X+	3.15	X+			6.5	X+	3.15	X+	3.15	X+
16WW02	5/12/93	0:00	0	Primary	2.9	X+	2.9	X+	2.9	X+			6	X+	2.9	X+	2.9	X+
16WW02	5/12/93	0:00	0	Duplicate	2.95	X+	2.95	X+	2.95	X+			6	X+	2.95	X+	2.95	X+
16WW02-A				Avg.	2.925		2.925		2.925				6		2.925		2.925	
16WW02	5/12/93	0:00	5	Primary	3.1	X+	3.1	X+	3.1	X+			6	X+	3.1	X+	3.1	X+
16WW03	5/4/93	0:00	0	Primary	3.15	X+	3.15	X+	3.15	X+			6.5	X+	3.15	X+	3.15	X+
16WW03	5/4/93	0:00	5	Primary	3	X+	3	X+	3	X+			6	X+	3	X+	3	X+
16WW03	5/4/93	0:00	10	Primary	3.05	X+	3.05	X+	3.05	X+			6	X+	3.05	X+	3.05	X+
16WW03	5/4/93	0:00	15	Primary	3.15	X+	3.15	X+	3.15	X+			6.5	X+	3.15	X+	3.15	X+
16WW04	5/12/93	0:00	0	Primary	3	X+	3	X+	3	X+			6	X+	3	X+	3	X+
16WW04	5/12/93	0:00	0	Duplicate	3.15	X+	3.15	X+	3.15	X+			6.5	X+	3.15	X+	3.15	X+
16WW04-A				Avg.	3.075		3.075		3.075				6.25		3.075		3.075	
16WW04	5/12/93	0:00	5	Primary	2.95	X+	2.95	X+	2.95	X+			6	X+	2.95	X+	2.95	X+
16WW04	5/12/93	0:00	10	Primary	3.05	X+	3.05	X+	3.05	X+			6	X+	3.05	X+	3.05	X+
16WW05	5/4/93	0:00	0	Primary	3.4	X+	3.4	X+	3.4	X+			7	X+	3.4	X+	3.4	X+
16WW05	5/4/93	0:00	5	Primary	2.85	X+	2.85	X+	2.85	X+			5.5	X+	2.85	X+	2.85	X+
16WW05	5/4/93	0:00	10	Primary	2.9	X+	2.9	X+	2.9	X+			6	X+	2.9	X+	2.9	X+
16WW05	5/4/93	0:00	15	Primary	3	X+	3	X+	3	X+			6	X+	3	X+	3	X+
16WW05	5/4/93	0:00	20	Primary	3.2	X+	3.2	X+	3.2	X+			6.5	X+	3.2	X+	3.2	X+
16WW05	5/4/93	0:00	25	Primary	3.2	X+	3.2	X+	3.2	X+			6.5	X+	3.2	X+	3.2	X+
16WW06	5/5/93	0:00	0	Primary	3.25	X+	3.25	X+	3.25	X+			6.5	X+	3.25	X+	3.25	X+
16WW06	5/5/93	0:00	5	Primary	3.15	X+	3.15	X+	3.15	X+			6.5	X+	3.15	X+	3.15	X+
16WW06	5/5/93	0:00	10	Primary	3	X+	3	X+	3	X+			6	X+	3	X+	3	X+
16WW06	5/5/93	0:00	15	Primary	2.95	X+	2.95	X+	2.95	X+			6	X+	2.95	X+	2.95	X+
16WW06	5/5/93	0:00	20	Primary	3.15	X+	3.15	X+	3.15	X+			6.5	X+	3.15	X+	3.15	X+
16WW27	5/17/97	9:15	0	Primary	3	X+	6.5	X+	3	X+	3	X+	6.5	X+	3	X+	3	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Carbon disulfide (ug/kg)		Carbon tetrachloride (ug/kg)		Chlorobenzene (ug/kg)		Bromochloro methane (ug/kg)		Chloroethane (ug/kg)		Chloroform (ug/kg)		cis-1,2-Dichloroethene (ug/kg)	
16WW27	5/17/97	10:17	1	Primary	3	X+	6	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	6	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW31	5/29/97	8:55	1	Primary	3	X+	6	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW31	5/29/97	10:09	5	Primary	3	X+	6	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW31	5/29/97	11:10	14	Primary	2.5	X+	5.5	X+	2.5	X+	2.5	X+	5.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	5.5	X+	2.5	X+	2.5	X+	5.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	5	X+	2.5	X+	2.5	X+	5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	5	X+	2.5	X+	2.5	X+	5	X+	2.5	X+	2.5	X+
16WW35-A				Avg.	2.5		5.166667		2.5		2.5		5.166667		2.5		2.5	
16WW35	6/25/97	10:05	1	Primary	3	X+	6	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW35	6/25/97	11:15	5	Primary	3	X+	6	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW37	5/31/97	15:55	0	Primary	3	X+	6	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW37	5/31/97	16:10	1	Primary	3	X+	6.5	X+	3	X+	3	X+	6.5	X+	3	X+	3	X+
16WW37	5/31/97	16:35	5	Primary	3	X+	6	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW37	6/1/97	10:01	14	Primary	3	X+	6	X+	3	X+	3	X+	6	X+	3	X+	3	X+
NATURAL LOGS																		
16SD01					1.459		1.459		1.459				2.14		1.459		1.459	
16SD01					1.504		1.504		1.504				2.197		1.504			
16SD02					1.147		1.147		1.147				1.872		1.147		1.147	
16SD02					1.253		1.253		1.253				1.946		1.253			
16SD03					1.179		1.179		1.179				1.872		1.179		1.179	
16SD03					1.386		1.386		1.386				2.079		1.386			
16SD06					1.253		1.253		1.253				2.015		1.253			
16SD07					1.386		1.386		1.386				2.079		1.386			
16SD08					1.253		1.253		1.253				1.872		1.253			
16SD09					1.253		1.253		1.253				1.872		1.253			
16SD10					1.253		1.253		1.253				1.872		1.253			
16SD11					1.099		1.099		1.099				1.872		1.099			
16WW01-A					1.082		1.082		1.082				1.792		1.082		1.082	
16WW01					1.131		1.131		1.131				1.792		1.131		1.131	
16WW01					1.147		1.147		1.147				1.872		1.147		1.147	
16WW02-A					1.073		1.073		1.073				1.792		1.073		1.073	
16WW02					1.131		1.131		1.131				1.792		1.131		1.131	
16WW03					1.147		1.147		1.147				1.872		1.147		1.147	
16WW03					1.099		1.099		1.099				1.792		1.099		1.099	
16WW03					1.115		1.115		1.115				1.792		1.115		1.115	
16WW03					1.147		1.147		1.147				1.872		1.147		1.147	
16WW04-A					1.123		1.123		1.123				1.833		1.123		1.123	
16WW04					1.082		1.082		1.082				1.792		1.082		1.082	
16WW04					1.115		1.115		1.115				1.792		1.115		1.115	
16WW05					1.224		1.224		1.224				1.946		1.224		1.224	
16WW05					1.047		1.047		1.047				1.705		1.047		1.047	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Carbon disulfide (ug/kg)	Carbon tetrachloride (ug/kg)	Chlorobenzene (ug/kg)	Bromochloro methane (ug/kg)	Chloroethane (ug/kg)	Chloroform (ug/kg)	cis-1,2-Dichloroethene (ug/kg)
16WW05					1.065	1.065	1.065		1.792	1.065	1.065
16WW05					1.099	1.099	1.099		1.792	1.099	1.099
16WW05					1.163	1.163	1.163		1.872	1.163	1.163
16WW05					1.163	1.163	1.163		1.872	1.163	1.163
16WW06					1.179	1.179	1.179		1.872	1.179	1.179
16WW06					1.147	1.147	1.147		1.872	1.147	1.147
16WW06					1.099	1.099	1.099		1.792	1.099	1.099
16WW06					1.082	1.082	1.082		1.792	1.082	1.082
16WW06					1.147	1.147	1.147		1.872	1.147	1.147
16WW27					1.099	1.872	1.099	1.099	1.872	1.099	1.099
16WW27					1.099	1.792	1.099	1.099	1.792	1.099	1.099
16WW31					1.099	1.792	1.099	1.099	1.792	1.099	1.099
16WW31					1.099	1.792	1.099	1.099	1.792	1.099	1.099
16WW31					1.099	1.792	1.099	1.099	1.792	1.099	1.099
16WW31					0.916	1.705	0.916	0.916	1.705	0.916	0.916
16WW35-A					0.916	1.642	0.916	0.916	1.642	0.916	0.916
16WW35					1.099	1.792	1.099	1.099	1.792	1.099	1.099
16WW35					1.099	1.792	1.099	1.099	1.792	1.099	1.099
16WW37					1.099	1.792	1.099	1.099	1.792	1.099	1.099
16WW37					1.099	1.872	1.099	1.099	1.872	1.099	1.099
16WW37					1.099	1.792	1.099	1.099	1.792	1.099	1.099
16WW37					1.099	1.792	1.099	1.099	1.792	1.099	1.099
STATISTICS											
Total No.					48	48	48	13	48	48	39
Min.					2.5	2.85	2.5	2.5	5	2.5	2.5
Max.					4.5	6.5	4.5	3	9	4.5	4.3
Avg.(log transformed)					1.149	1.343	1.149	1.071	1.851	1.149	1.116
St. Dev.(log transformed)					0.111	0.29	0.111	0.069	0.105	0.111	0.08
H											
95th UCL of Mean					3.17	4.00	3.17	2.93	6.40	3.17	3.06

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	cis-1,3-Dichloropropene (ug/kg)		1,2-Dibromo-3-chloropropane (ug/kg)		Dibromochloro methane (ug/kg)		Dichloro difluoromethane (ug/kg)		1,2-Dibromoetha (ug/kg)		CEVETH (ug/kg)		trans-1,2-Dichloroethene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary					4.3	X+	4.3	X+					4.3	X+
16SD01	2/21/95	14:20	0	Primary	4.5	X+			4.5	X+	4.5	X+			9	X+		
16SD02	4/14/93	0:00	0	Primary					3.15	X+	3.15	X+					3.15	X+
16SD02	2/28/95	10:00	0	Primary	3.5	X+			3.5	X+	3.5	X+			7	X+		
16SD03	4/14/93	0:00	0	Primary					3.25	X+	3.25	X+					3.25	X+
16SD03	2/20/95	16:05	0	Primary	4	X+			4	X+	4	X+			8	X+		
16SD06	2/20/95	16:35	0	Primary	3.5	X+			3.5	X+	3.5	X+			7.5	X+		
16SD07	2/21/95	14:05	0	Primary	4	X+			4	X+	4	X+			8	X+		
16SD08	2/21/95	9:30	0	Primary	3.5	X+			3.5	X+	3.5	X+			6.5	X+		
16SD09	2/21/95	10:55	0	Primary	3.5	X+			3.5	X+	3.5	X+			6.5	X+		
16SD10	2/21/95	17:10	0	Primary	3.5	X+			3.5	X+	3.5	X+			6.5	X+		
16SD11	2/28/95	11:15	0	Primary	3	X+			3	X+	3	X+			6.5	X+		
16WW01	5/4/93	0:00	0	Primary					3	X+	3	X+					3	X+
16WW01	5/4/93	0:00	0	Duplicate					2.9	X+	2.9	X+					2.9	X+
16WW01-A				Avg.					2.95		2.95						2.95	
16WW01	5/4/93	0:00	5	Primary					3.1	X+	3.1	X+					3.1	X+
16WW01	5/4/93	0:00	10	Primary					3.15	X+	3.15	X+					3.15	X+
16WW02	5/12/93	0:00	0	Primary					2.9	X+	2.9	X+					2.9	X+
16WW02	5/12/93	0:00	0	Duplicate					2.95	X+	2.95	X+					2.95	X+
16WW02-A				Avg.					2.925		2.925						2.925	
16WW02	5/12/93	0:00	5	Primary					3.1	X+	3.1	X+					3.1	X+
16WW03	5/4/93	0:00	0	Primary					3.15	X+	3.15	X+					3.15	X+
16WW03	5/4/93	0:00	5	Primary					3	X+	3	X+					3	X+
16WW03	5/4/93	0:00	10	Primary					3.05	X+	3.05	X+					3.05	X+
16WW03	5/4/93	0:00	15	Primary					3.15	X+	3.15	X+					3.15	X+
16WW04	5/12/93	0:00	0	Primary					3	X+	3	X+					3	X+
16WW04	5/12/93	0:00	0	Duplicate					3.15	X+	3.15	X+					3.15	X+
16WW04-A				Avg.					3.075		3.075						3.075	
16WW04	5/12/93	0:00	5	Primary					2.95	X+	2.95	X+					2.95	X+
16WW04	5/12/93	0:00	10	Primary					3.05	X+	3.05	X+					3.05	X+
16WW05	5/4/93	0:00	0	Primary					3.4	X+	3.4	X+					3.4	X+
16WW05	5/4/93	0:00	5	Primary					2.85	X+	2.85	X+					2.85	X+
16WW05	5/4/93	0:00	10	Primary					2.9	X+	2.9	X+					2.9	X+
16WW05	5/4/93	0:00	15	Primary					3	X+	3	X+					3	X+
16WW05	5/4/93	0:00	20	Primary					3.2	X+	3.2	X+					3.2	X+
16WW05	5/4/93	0:00	25	Primary					3.2	X+	3.2	X+					3.2	X+
16WW06	5/5/93	0:00	0	Primary					3.25	X+	3.25	X+					3.25	X+
16WW06	5/5/93	0:00	5	Primary					3.15	X+	3.15	X+					3.15	X+
16WW06	5/5/93	0:00	10	Primary					3	X+	3	X+					3	X+
16WW06	5/5/93	0:00	15	Primary					2.95	X+	2.95	X+					2.95	X+
16WW06	5/5/93	0:00	20	Primary					3.15	X+	3.15	X+					3.15	X+
16WW27	5/17/97	9:15	0	Primary	3	X+	6.5	X+	3	X+	9.5	X+	3	X+			3	X+

Longhorn Army Ammuntion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	cis-1,3-Dichloropropene (ug/kg)		1,2-Dibromo-3-chloropropane (ug/kg)		Dibromochloromethane (ug/kg)		Dichlorodifluoromethane (ug/kg)		1,2-Dibromoetha (ug/kg)		CEVETH (ug/kg)	trans-1,2-Dichloroethene (ug/kg)	
16WW27	5/17/97	10:17	1	Primary	3	X+	6	X+	3	X+	9	X+	3	X+		3	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	3	X+	9	X+	3	X+		3	X+
16WW31	5/29/97	8:55	1	Primary	3	X+	6	X+	3	X+	9	X+	3	X+		3	X+
16WW31	5/29/97	10:09	5	Primary	3	X+	6	X+	3	X+	9	X+	3	X+		3	X+
16WW31	5/29/97	11:10	14	Primary	2.5	X+	5.5	X+	2.5	X+	8	X+	2.5	X+		2.5	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	5.5	X+	2.5	X+	8	X+	2.5	X+		2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	5	X+	2.5	X+	7.5	X+	2.5	X+		2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	5	X+	2.5	X+	7.5	X+	2.5	X+		2.5	X+
16WW35-A				Avg.	2.5		5.166667		2.5		7.666667		2.5			2.5	
16WW35	6/25/97	10:05	1	Primary	3	X+	6	X+	3	X+	9.5	X+	3	X+		3	X+
16WW35	6/25/97	11:15	5	Primary	3	X+	6	X+	3	X+	9.5	X+	3	X+		3	X+
16WW37	5/31/97	15:55	0	Primary	3	X+	6	X+	3	X+	9.5	X+	3	X+		3	X+
16WW37	5/31/97	16:10	1	Primary	3	X+	6.5	X+	3	X+	9.5	X+	3	X+		3	X+
16WW37	5/31/97	16:35	5	Primary	3	X+	6	X+	3	X+	9	X+	3	X+		3	X+
16WW37	6/1/97	10:01	14	Primary	3	X+	6	X+	3	X+	9	X+	3	X+		3	X+

NATURAL LOGS

16SD01									1.459		1.459					1.459	
16SD01					1.504				1.504		1.504				2.197		
16SD02									1.147		1.147					1.147	
16SD02					1.253				1.253		1.253				1.946		
16SD03									1.179		1.179					1.179	
16SD03					1.386				1.386		1.386				2.079		
16SD06					1.253				1.253		1.253				2.015		
16SD07					1.386				1.386		1.386				2.079		
16SD08					1.253				1.253		1.253				1.872		
16SD09					1.253				1.253		1.253				1.872		
16SD10					1.253				1.253		1.253				1.872		
16SD11					1.099				1.099		1.099				1.872		
16WW01-A									1.082		1.082					1.082	
16WW01									1.131		1.131					1.131	
16WW01									1.147		1.147					1.147	
16WW02-A									1.073		1.073					1.073	
16WW02									1.131		1.131					1.131	
16WW03									1.147		1.147					1.147	
16WW03									1.099		1.099					1.099	
16WW03									1.115		1.115					1.115	
16WW03									1.147		1.147					1.147	
16WW04-A									1.123		1.123					1.123	
16WW04									1.082		1.082					1.082	
16WW04									1.115		1.115					1.115	
16WW05									1.224		1.224					1.224	
16WW05									1.047		1.047					1.047	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	cis-1,3-Dichloropropene (ug/kg)	1,2-Dibromo-3-chloropropane (ug/kg)	Dibromochloro methane (ug/kg)	Dichloro difluoromethane (ug/kg)	1,2-Dibromoetha (ug/kg)	CEVETH (ug/kg)	trans-1,2-Dichloroethene (ug/kg)
16WW05							1.065	1.065			1.065
16WW05							1.099	1.099			1.099
16WW05							1.163	1.163			1.163
16WW05							1.163	1.163			1.163
16WW06							1.179	1.179			1.179
16WW06							1.147	1.147			1.147
16WW06							1.099	1.099			1.099
16WW06							1.082	1.082			1.082
16WW06							1.147	1.147			1.147
16WW27					1.099	1.872	1.099	2.251	1.099		1.099
16WW27					1.099	1.792	1.099	2.197	1.099		1.099
16WW31					1.099	1.099	1.099	2.197	1.099		1.099
16WW31					1.099	1.792	1.099	2.197	1.099		1.099
16WW31					1.099	1.792	1.099	2.197	1.099		1.099
16WW31					0.916	1.705	0.916	2.079	0.916		0.916
16WW35-A					0.916	1.642	0.916	2.037	0.916		0.916
16WW35					1.099	1.792	1.099	2.251	1.099		1.099
16WW35					1.099	1.792	1.099	2.251	1.099		1.099
16WW37					1.099	1.792	1.099	2.251	1.099		1.099
16WW37					1.099	1.872	1.099	2.251	1.099		1.099
16WW37					1.099	1.792	1.099	2.197	1.099		1.099
16WW37					1.099	1.792	1.099	2.197	1.099		1.099
STATISTICS											
Total No.					22	13	48	48	13	9	39
Min.					2.5	3	2.5	2.85	2.5	6.5	2.5
Max.					4.5	6.5	4.5	9.5	3	9	4.3
Avg.(log transformed)					1.162	1.733	1.149	1.454	1.071	1.978	1.116
St. Dev.(log transformed)					0.143	0.199	0.111	0.468	0.069	0.12	0.08
H											
95th UCL of Mean					3.23	5.77	3.17	4.78	2.93	7.28	3.06

Longhorn Army Ammuntion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Propionitrile (ug/kg)		Ethyl methacrylate (ug/kg)		Ethylbenzene (ug/kg)		Hexachloro butadiene (ug/kg)		Isobutyl alcohol (ug/kg)		1,3-Dichloro benzene (ug/kg)		Methacrylo nitrile (ug/kg)	
16SD01	5/2/93	0:00	0	Primary			4.3	X+	4.3	X+								
16SD01	2/21/95	14:20	0	Primary			4.5	X+	4.5	X+								
16SD02	4/14/93	0:00	0	Primary			3.15	X+	3.15	X+								
16SD02	2/28/95	10:00	0	Primary			3.5	X+	3.5	X+								
16SD03	4/14/93	0:00	0	Primary			3.25	X+	3.25	X+								
16SD03	2/20/95	16:05	0	Primary			4	X+	4	X+								
16SD06	2/20/95	16:35	0	Primary			3.5	X+	3.5	X+								
16SD07	2/21/95	14:05	0	Primary			4	X+	4	X+								
16SD08	2/21/95	9:30	0	Primary			3.5	X+	3.5	X+								
16SD09	2/21/95	10:55	0	Primary			3.5	X+	3.5	X+								
16SD10	2/21/95	17:10	0	Primary			3.5	X+	3.5	X+								
16SD11	2/28/95	11:15	0	Primary			3	X+	3	X+								
16WW01	5/4/93	0:00	0	Primary			3	X+	3	X+								
16WW01	5/4/93	0:00	0	Duplicate			2.9	X+	2.9	X+								
16WW01-A				Avg.			2.95		2.95									
16WW01	5/4/93	0:00	5	Primary			3.1	X+	3.1	X+								
16WW01	5/4/93	0:00	10	Primary			3.15	X+	3.15	X+								
16WW02	5/12/93	0:00	0	Primary			2.9	X+	2.9	X+								
16WW02	5/12/93	0:00	0	Duplicate			2.95	X+	2.95	X+								
16WW02-A				Avg.			2.925		2.925									
16WW02	5/12/93	0:00	5	Primary			3.1	X+	3.1	X+								
16WW03	5/4/93	0:00	0	Primary			3.15	X+	3.15	X+								
16WW03	5/4/93	0:00	5	Primary			3	X+	3	X+								
16WW03	5/4/93	0:00	10	Primary			3.05	X+	3.05	X+								
16WW03	5/4/93	0:00	15	Primary			3.15	X+	3.15	X+								
16WW04	5/12/93	0:00	0	Primary			3	X+	3	X+								
16WW04	5/12/93	0:00	0	Duplicate			3.15	X+	3.15	X+								
16WW04-A				Avg.			3.075		3.075									
16WW04	5/12/93	0:00	5	Primary			2.95	X+	2.95	X+								
16WW04	5/12/93	0:00	10	Primary			3.05	X+	3.05	X+								
16WW05	5/4/93	0:00	0	Primary			3.4	X+	3.4	X+								
16WW05	5/4/93	0:00	5	Primary			2.85	X+	2.85	X+								
16WW05	5/4/93	0:00	10	Primary			2.9	X+	2.9	X+								
16WW05	5/4/93	0:00	15	Primary			3	X+	3	X+								
16WW05	5/4/93	0:00	20	Primary			3.2	X+	3.2	X+								
16WW05	5/4/93	0:00	25	Primary			3.2	X+	3.2	X+								
16WW06	5/5/93	0:00	0	Primary			3.25	X+	3.25	X+								
16WW06	5/5/93	0:00	5	Primary			3.15	X+	3.15	X+								
16WW06	5/5/93	0:00	10	Primary			3	X+	3	X+								
16WW06	5/5/93	0:00	15	Primary			2.95	X+	2.95	X+								
16WW06	5/5/93	0:00	20	Primary			3.15	X+	3.15	X+								
16WW27	5/17/97	9:15	0	Primary	65	X+	3	X+	3	X+	3	X+	650	X+	3	X+	65	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Propionitrile (ug/kg)		Ethyl methacrylate (ug/kg)		Ethylbenzene (ug/kg)		Hexachloro butadiene (ug/kg)		Isobutyl alcohol (ug/kg)		1,3-Dichloro benzene (ug/kg)		Methacrylo nitrile (ug/kg)	
16WW27	5/17/97	10:17	1	Primary	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+	60	X+
16WW31	5/29/97	8:27	0	Primary	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+	60	X+
16WW31	5/29/97	8:55	1	Primary	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+	60	X+
16WW31	5/29/97	10:09	5	Primary	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+	60	X+
16WW31	5/29/97	11:10	14	Primary	55	X+	2.5	X+	2.5	X+	2.5	X+	550	X+	2.5	X+	55	X+
16WW35	6/25/97	9:50	0	Primary	55	X+	2.5	X+	2.5	X+	2.5	X+	550	X+	2.5	X+	55	X+
16WW35	6/25/97	9:50	0	Duplicate	50	X+	2.5	X+	2.5	X+	2.5	X+	500	X+	2.5	X+	50	X+
16WW35	6/25/97	0:00	0	Duplicate	50	X+	2.5	X+	2.5	X+	2.5	X+	500	X+	2.5	X+	50	X+
16WW35-A				Avg.	51.66667		2.5		2.5		2.5		516.6667		2.5		51.66667	
16WW35	6/25/97	10:05	1	Primary	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+	60	X+
16WW35	6/25/97	11:15	5	Primary	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+	60	X+
16WW37	5/31/97	15:55	0	Primary	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+	60	X+
16WW37	5/31/97	16:10	1	Primary	65	X+	3	X+	3	X+	3	X+	650	X+	3	X+	65	X+
16WW37	5/31/97	16:35	5	Primary	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+	60	X+
16WW37	6/1/97	10:01	14	Primary	60	X+	3	X+	3	X+	3	X+	600	X+	3	X+	60	X+

NATURAL LOGS

16SD01	1.459	1.459
16SD01	1.504	1.504
16SD02	1.147	1.147
16SD02	1.253	1.253
16SD03	1.179	1.179
16SD03	1.386	1.386
16SD06	1.253	1.253
16SD07	1.386	1.386
16SD08	1.253	1.253
16SD09	1.253	1.253
16SD10	1.253	1.253
16SD11	1.099	1.099
16WW01-A	1.082	1.082
16WW01	1.131	1.131
16WW01	1.147	1.147
16WW02-A	1.073	1.073
16WW02	1.131	1.131
16WW03	1.147	1.147
16WW03	1.099	1.099
16WW03	1.115	1.115
16WW03	1.147	1.147
16WW04-A	1.123	1.123
16WW04	1.082	1.082
16WW04	1.115	1.115
16WW05	1.224	1.224
16WW05	1.047	1.047

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Propionitrile (ug/kg)	Ethyl methacrylate (ug/kg)	Ethylbenzene (ug/kg)	Hexachloro butadiene (ug/kg)	Isobutyl alcohol (ug/kg)	1,3-Dichloro benzene (ug/kg)	Methacrylo nitrile (ug/kg)
16WW05						1.065	1.065				
16WW05						1.099	1.099				
16WW05						1.163	1.163				
16WW05						1.163	1.163				
16WW06						1.179	1.179				
16WW06						1.147	1.147				
16WW06						1.099	1.099				
16WW06						1.082	1.082				
16WW06						1.147	1.147				
16WW27					4.174	1.099	1.099	1.099	6.477	1.099	4.174
16WW27					4.094	1.099	1.099	1.099	6.397	1.099	4.094
16WW31					4.094	1.099	1.099	1.099	6.397	1.099	4.094
16WW31					4.094	1.099	1.099	1.099	6.397	1.099	4.094
16WW31					4.094	1.099	1.099	1.099	6.397	1.099	4.094
16WW31					4.007	0.916	0.916	0.916	6.31	0.916	4.007
16WW35-A					3.945	0.916	0.916	0.916	6.247	0.916	3.945
16WW35					4.094	1.099	1.099	1.099	6.397	1.099	4.094
16WW35					4.094	1.099	1.099	1.099	6.397	1.099	4.094
16WW37					4.094	1.099	1.099	1.099	6.397	1.099	4.094
16WW37					4.174	1.099	1.099	1.099	6.477	1.099	4.174
16WW37					4.094	1.099	1.099	1.099	6.397	1.099	4.094
16WW37					4.094	1.099	1.099	1.099	6.397	1.099	4.094
STATISTICS											
Total No.					13	48	48	13	13	13	13
Min.					50	2.5	2.5	2.5	500	2.5	50
Max.					65	4.5	4.5	3	650	3	65
Avg.(log transformed)					4.088	1.149	1.149	1.071	6.391	1.071	4.088
St. Dev.(log transformed)					0.059	0.111	0.111	0.069	0.059	0.069	0.059
H											
95th UCL of Mean					59.72	3.17	3.17	2.93	597.49	2.93	59.72

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Bromomethane (ug/kg)		Chloromethane (ug/kg)		2-Butanone (ug/kg)		Iodomethane (ug/kg)		4-Methyl-2-pentanone (ug/kg)		Methyl methacrylate (ug/kg)		Dibromomethane (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	8.5	X+	8.5	X+	8.5	X+	8.5	X+	8.5	X+			4.3	X+
16SD01	2/21/95	14:20	0	Primary	9	X+	9	X+	9	X+	9	X+	9	X+			4.5	X+
16SD02	4/14/93	0:00	0	Primary	6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.15	X+
16SD02	2/28/95	10:00	0	Primary	7	X+	7	X+	7	X+	7	X+	7	X+			3.5	X+
16SD03	4/14/93	0:00	0	Primary	6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.25	X+
16SD03	2/20/95	16:05	0	Primary	8	X+	8	X+	8	X+	8	X+	8	X+			4	X+
16SD06	2/20/95	16:35	0	Primary	7.5	X+	7.5	X+	7.5	X+	7.5	X+	7.5	X+			3.5	X+
16SD07	2/21/95	14:05	0	Primary	8	X+	8	X+	8	X+	8	X+	8	X+			4	X+
16SD08	2/21/95	9:30	0	Primary	6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.5	X+
16SD09	2/21/95	10:55	0	Primary	6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.5	X+
16SD10	2/21/95	17:10	0	Primary	6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.5	X+
16SD11	2/28/95	11:15	0	Primary	6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3	X+
16WW01	5/4/93	0:00	0	Primary	6	X+	6	X+	6	X+	6	X+	6	X+			3	X+
16WW01	5/4/93	0:00	0	Duplicate	6	X+	6	X+	6	X+	6	X+	6	X+			2.9	X+
16WW01-A				Avg.	6		6		6		6		6				2.95	
16WW01	5/4/93	0:00	5	Primary	6	X+	6	X+	6	X+	6	X+	6	X+			3.1	X+
16WW01	5/4/93	0:00	10	Primary	6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.15	X+
16WW02	5/12/93	0:00	0	Primary	6	X+	6	X+	6	X+	6	X+	6	X+			2.9	X+
16WW02	5/12/93	0:00	0	Duplicate	6	X+	6	X+	6	X+	6	X+	6	X+			2.95	X+
16WW02-A				Avg.	6		6		6		6		6				2.925	
16WW02	5/12/93	0:00	5	Primary	6	X+	6	X+	6	X+	6	X+	6	X+			3.1	X+
16WW03	5/4/93	0:00	0	Primary	6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.15	X+
16WW03	5/4/93	0:00	5	Primary	6	X+	6	X+	6	X+	6	X+	6	X+			3	X+
16WW03	5/4/93	0:00	10	Primary	6	X+	6	X+	6	X+	6	X+	6	X+			3.05	X+
16WW03	5/4/93	0:00	15	Primary	6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.15	X+
16WW04	5/12/93	0:00	0	Primary	6	X+	6	X+	6	X+	6	X+	6	X+			3	X+
16WW04	5/12/93	0:00	0	Duplicate	6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.15	X+
16WW04-A				Avg.	6.25		6.25		6.25		6.25		6.25				3.075	
16WW04	5/12/93	0:00	5	Primary	6	X+	6	X+	6	X+	6	X+	6	X+			2.95	X+
16WW04	5/12/93	0:00	10	Primary	6	X+	6	X+	6	X+	6	X+	6	X+			3.05	X+
16WW05	5/4/93	0:00	0	Primary	7	X+	7	X+	7	X+	7	X+	7	X+			3.4	X+
16WW05	5/4/93	0:00	5	Primary	5.5	X+	5.5	X+	5.5	X+	5.5	X+	5.5	X+			2.85	X+
16WW05	5/4/93	0:00	10	Primary	6	X+	6	X+	6	X+	6	X+	6	X+			2.9	X+
16WW05	5/4/93	0:00	15	Primary	6	X+	6	X+	6	X+	6	X+	6	X+			3	X+
16WW05	5/4/93	0:00	20	Primary	6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.2	X+
16WW05	5/4/93	0:00	25	Primary	6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.2	X+
16WW06	5/5/93	0:00	0	Primary	6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.25	X+
16WW06	5/5/93	0:00	5	Primary	6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.15	X+
16WW06	5/5/93	0:00	10	Primary	6	X+	6	X+	6	X+	6	X+	6	X+			3	X+
16WW06	5/5/93	0:00	15	Primary	6	X+	6	X+	6	X+	6	X+	6	X+			2.95	X+
16WW06	5/5/93	0:00	20	Primary	6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+			3.15	X+
16WW27	5/17/97	9:15	0	Primary	6.5	X+	6.5	X+	13	X+	3	X+	13	X+	32	X+	6.5	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Bromomethane (ug/kg)		Chloromethane (ug/kg)		2-Butanone (ug/kg)		Iodomethane (ug/kg)		4-Methyl-2-pentanone (ug/kg)		Methyl methacrylate (ug/kg)		Dibromomethane (ug/kg)	
16WW27	5/17/97	10:17	1	Primary	6	X+	6	X+	12	X+	3	X+	12	X+	30	X+	6	X+
16WW31	5/29/97	8:27	0	Primary	6	X+	6	X+	12	X+	3	X+	12	X+	30	X+	6	X+
16WW31	5/29/97	8:55	1	Primary	6	X+	6	X+	12	X+	3	X+	12	X+	30.5	X+	6	X+
16WW31	5/29/97	10:09	5	Primary	6	X+	6	X+	12	X+	3	X+	12	X+	30.5	X+	6	X+
16WW31	5/29/97	11:10	14	Primary	5.5	X+	5.5	X+	11	X+	2.5	X+	11	X+	27	X+	5.5	X+
16WW35	6/25/97	9:50	0	Primary	5.5	X+	5.5	X+	10.5	X+	2.5	X+	10.5	X+	26.5	X+	5.5	X+
16WW35	6/25/97	9:50	0	Duplicate	5	X+	5	X+	10	X+	2.5	X+	10	X+	25	X+	5	X+
16WW35	6/25/97	0:00	0	Duplicate	5	X+	5	X+	10	X+	2.5	X+	10	X+	25	X+	5	X+
16WW35-A				Avg.	5.166667		5.166667		10.16667		2.5		10.16667		25.5		5.166667	
16WW35	6/25/97	10:05	1	Primary	6	X+	6	X+	12.5	X+	3	X+	12.5	X+	31	X+	6	X+
16WW35	6/25/97	11:15	5	Primary	6	X+	6	X+	12.5	X+	3	X+	12.5	X+	31	X+	6	X+
16WW37	5/31/97	15:55	0	Primary	6	X+	6	X+	12.5	X+	3	X+	12.5	X+	31	X+	6	X+
16WW37	5/31/97	16:10	1	Primary	6.5	X+	6.5	X+	12.5	X+	3	X+	12.5	X+	31	X+	6.5	X+
16WW37	5/31/97	16:35	5	Primary	6	X+	6	X+	12	X+	3	X+	12	X+	30	X+	6	X+
16WW37	6/1/97	10:01	14	Primary	6	X+	6	X+	12	X+	3	X+	12	X+	30	X+	6	X+

NATURAL LOGS

16SD01	2.14	2.14	2.14	2.14	2.14	1.459
16SD01	2.197	2.197	2.197	2.197	2.197	1.504
16SD02	1.872	1.872	1.872	1.872	1.872	1.147
16SD02	1.946	1.946	1.946	1.946	1.946	1.253
16SD03	1.872	1.872	1.872	1.872	1.872	1.179
16SD03	2.079	2.079	2.079	2.079	2.079	1.386
16SD06	2.015	2.015	2.015	2.015	2.015	1.253
16SD07	2.079	2.079	2.079	2.079	2.079	1.386
16SD08	1.872	1.872	1.872	1.872	1.872	1.253
16SD09	1.872	1.872	1.872	1.872	1.872	1.253
16SD10	1.872	1.872	1.872	1.872	1.872	1.253
16SD11	1.872	1.872	1.872	1.872	1.872	1.099
16WW01-A	1.792	1.792	1.792	1.792	1.792	1.082
16WW01	1.792	1.792	1.792	1.792	1.792	1.131
16WW01	1.872	1.872	1.872	1.872	1.872	1.147
16WW02-A	1.792	1.792	1.792	1.792	1.792	1.073
16WW02	1.792	1.792	1.792	1.792	1.792	1.131
16WW03	1.872	1.872	1.872	1.872	1.872	1.147
16WW03	1.792	1.792	1.792	1.792	1.792	1.099
16WW03	1.792	1.792	1.792	1.792	1.792	1.115
16WW03	1.872	1.872	1.872	1.872	1.872	1.147
16WW04-A	1.833	1.833	1.833	1.833	1.833	1.123
16WW04	1.792	1.792	1.792	1.792	1.792	1.082
16WW04	1.792	1.792	1.792	1.792	1.792	1.115
16WW05	1.946	1.946	1.946	1.946	1.946	1.224
16WW05	1.705	1.705	1.705	1.705	1.705	1.047

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	RESULT DEPTH (ft TYPE	Bromomethane (ug/kg)	Chloromethane (ug/kg)	2-Butanone (ug/kg)	Iodomethane (ug/kg)	4-Methyl-2-pentanone (ug/kg)	Methyl methacrylate (ug/kg)	Dibromomethane (ug/kg)
16WW05				1.792	1.792	1.792	1.792	1.792		1.065
16WW05				1.792	1.792	1.792	1.792	1.792		1.099
16WW05				1.872	1.872	1.872	1.872	1.872		1.163
16WW05				1.872	1.872	1.872	1.872	1.872		1.163
16WW06				1.872	1.872	1.872	1.872	1.872		1.179
16WW06				1.872	1.872	1.872	1.872	1.872		1.147
16WW06				1.792	1.792	1.792	1.792	1.792		1.099
16WW06				1.792	1.792	1.792	1.792	1.792		1.082
16WW06				1.872	1.872	1.872	1.872	1.872		1.147
16WW27				1.872	1.872	2.565	1.099	2.565	3.466	1.872
16WW27				1.792	1.792	2.485	1.099	2.485	3.401	1.792
16WW31				1.792	1.792	2.485	1.099	2.485	3.401	1.792
16WW31				1.792	1.792	2.485	1.099	2.485	3.418	1.792
16WW31				1.792	1.792	2.485	1.099	2.485	3.418	1.792
16WW31				1.705	1.705	2.398	0.916	2.398	3.296	1.705
16WW35-A				1.642	1.642	2.319	0.916	2.319	3.239	1.642
16WW35				1.792	1.792	2.526	1.099	2.526	3.434	1.792
16WW35				1.792	1.792	2.526	1.099	2.526	3.434	1.792
16WW37				1.792	1.792	2.526	1.099	2.526	3.434	1.792
16WW37				1.872	1.872	2.526	1.099	2.526	3.434	1.872
16WW37				1.792	1.792	2.485	1.099	2.485	3.401	1.792
16WW37				1.792	1.792	2.485	1.099	2.485	3.401	1.792
STATISTICS										
Total No.				48	48	48	48	48	13	48
Min.				5	5	5.5	2.5	5.5	25	2.85
Max.				9	9	13	9	13	32	6.5
Avg.(log transformed)				1.851	1.851	2.041	1.658	2.041	3.398	1.343
St. Dev.(log transformed)				0.105	0.105	0.29	0.375	0.29	0.062	0.29
H										
95th UCL of Mean				6.40	6.40	8.03	5.63	8.03	29.96	4.00

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	TYPE	RESULT	Methylene chloride (ug/kg)		Naphthalene (ug/kg)		n-Butylbenzene (ug/kg)		1-Propanamine, N-nitroso-n-pro (ug/kg)		n-Propyl benzene (ug/kg)		2-Chlortoluene (ug/kg)		1,2-Dichloro benzene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	4.3	X+													
16SD01	2/21/95	14:20	0	Primary	9	X+													
16SD02	4/14/93	0:00	0	Primary	3.15	X+													
16SD02	2/28/95	10:00	0	Primary	7	X+													
16SD03	4/14/93	0:00	0	Primary	3.25	X+													
16SD03	2/20/95	16:05	0	Primary	8	X+													
16SD06	2/20/95	16:35	0	Primary	7.5	X+													
16SD07	2/21/95	14:05	0	Primary	8	X+													
16SD08	2/21/95	9:30	0	Primary	6.5	X+													
16SD09	2/21/95	10:55	0	Primary	6.5	X+													
16SD10	2/21/95	17:10	0	Primary	6.5	X+													
16SD11	2/28/95	11:15	0	Primary	6.5	X+													
16WW01	5/4/93	0:00	0	Primary	3	X+													
16WW01	5/4/93	0:00	0	Duplicate	2.9	X+													
16WW01-A				Avg.	2.95														
16WW01	5/4/93	0:00	5	Primary	3.1	X+													
16WW01	5/4/93	0:00	10	Primary	3.15	X+													
16WW02	5/12/93	0:00	0	Primary	6.5														
16WW02	5/12/93	0:00	0	Duplicate	2.95	X+													
16WW02-A				Avg.	4.725														
16WW02	5/12/93	0:00	5	Primary	3.1	X+													
16WW03	5/4/93	0:00	0	Primary	3.15	X+													
16WW03	5/4/93	0:00	5	Primary	3	X+													
16WW03	5/4/93	0:00	10	Primary	3.05	X+													
16WW03	5/4/93	0:00	15	Primary	3.15	X+													
16WW04	5/12/93	0:00	0	Primary	3	X+													
16WW04	5/12/93	0:00	0	Duplicate	3.15	X+													
16WW04-A				Avg.	3.075														
16WW04	5/12/93	0:00	5	Primary	7														
16WW04	5/12/93	0:00	10	Primary	3.05	X+													
16WW05	5/4/93	0:00	0	Primary	3.4	X+													
16WW05	5/4/93	0:00	5	Primary	2.85	X+													
16WW05	5/4/93	0:00	10	Primary	2.9	X+													
16WW05	5/4/93	0:00	15	Primary	3	X+													
16WW05	5/4/93	0:00	20	Primary	3.2	X+													
16WW05	5/4/93	0:00	25	Primary	3.2	X+													
16WW06	5/5/93	0:00	0	Primary	3.25	X+													
16WW06	5/5/93	0:00	5	Primary	3.15	X+													
16WW06	5/5/93	0:00	10	Primary	3	X+													
16WW06	5/5/93	0:00	15	Primary	2.95	X+													
16WW06	5/5/93	0:00	20	Primary	3.15	X+													
16WW27	5/17/97	9:15	0	Primary	2	U+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Methylene chloride (ug/kg)		Naphthalene (ug/kg)		n-Butylbenzene (ug/kg)		1-Propanamine, N-nitroso-n-pro (ug/kg)		n-Propyl benzene (ug/kg)		2-Chlortoluene (ug/kg)		1,2-Dichloro benzene (ug/kg)	
16WW27	5/17/97	10:17	1	Primary	1	U+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	8:27	0	Primary	5		3	X+	3	X+	3	X+	3	X+	3	X+	6	X+
16WW31	5/29/97	8:55	1	Primary	10		3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	10:09	5	Primary	15		3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW31	5/29/97	11:10	14	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+
16WW35-A				Avg.	2.5		2.5		2.5		2.5		2.5		2.5		2.5	
16WW35	6/25/97	10:05	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW35	6/25/97	11:15	5	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37	5/31/97	16:10	1	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37	5/31/97	16:35	5	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW37	6/1/97	10:01	14	Primary	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+	3	X+

NATURAL LOGS

16SD01	1.459
16SD01	2.197
16SD02	1.147
16SD02	1.946
16SD03	1.179
16SD03	2.079
16SD06	2.015
16SD07	2.079
16SD08	1.872
16SD09	1.872
16SD10	1.872
16SD11	1.872
16WW01-A	1.082
16WW01	1.131
16WW01	1.147
16WW02-A	1.553
16WW02	1.131
16WW03	1.147
16WW03	1.099
16WW03	1.115
16WW03	1.147
16WW04-A	1.123
16WW04	1.946
16WW04	1.115
16WW05	1.224
16WW05	1.047

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	TYPE	RESULT	Methylene chloride (ug/kg)	Naphthalene (ug/kg)	n-Butylbenzene (ug/kg)	1-Propanamine, N-nitroso-n-pro (ug/kg)	n-Propyl benzene (ug/kg)	2-Chlortoluene (ug/kg)	1,2-Dichloro benzene (ug/kg)
16WW05						1.065						
16WW05						1.099						
16WW05						1.163						
16WW05						1.163						
16WW06						1.179						
16WW06						1.147						
16WW06						1.099						
16WW06						1.082						
16WW06						1.147						
16WW27						0.405	1.099	1.099	1.099	1.099	1.099	1.099
16WW27						0	1.099	1.099	1.099	1.099	1.099	1.099
16WW31						1.609	1.099	1.099	1.099	1.099	1.099	1.792
16WW31						2.303	1.099	1.099	1.099	1.099	1.099	1.099
16WW31						2.708	1.099	1.099	1.099	1.099	1.099	1.099
16WW31						0.916	0.916	0.916	0.916	0.916	0.916	0.916
16WW35-A						0.916	0.916	0.916	0.916	0.916	0.916	0.916
16WW35						1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW35						1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW37						1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW37						1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW37						1.099	1.099	1.099	1.099	1.099	1.099	1.099
16WW37						1.099	1.099	1.099	1.099	1.099	1.099	1.099
STATISTICS												
Total No.						48	13	13	13	13	13	13
Min.						1	2.5	2.5	2.5	2.5	2.5	2.5
Max.						15	3	3	3	3	3	6
Avg.(log transformed)						1.337	1.071	1.071	1.071	1.071	1.071	1.124
St. Dev.(log transformed)						0.495	0.069	0.069	0.069	0.069	0.069	0.212
H						1.878						
95th UCL of Mean						4.93	2.93	2.93	2.93	2.93	2.93	3.15

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	o-Xylene (ug/kg)		4-Chlorotoluene (ug/kg)		p-Isopropyl toluene (ug/kg)		1,4-Dichloro benzene (ug/kg)		Pentachloro ethane (ug/kg)		sec-Butyl benzene (ug/kg)		Styrene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary													4.3	X+
16SD01	2/21/95	14:20	0	Primary													4.5	X+
16SD02	4/14/93	0:00	0	Primary													3.15	X+
16SD02	2/28/95	10:00	0	Primary													3.5	X+
16SD03	4/14/93	0:00	0	Primary													3.25	X+
16SD03	2/20/95	16:05	0	Primary													4	X+
16SD06	2/20/95	16:35	0	Primary													3.5	X+
16SD07	2/21/95	14:05	0	Primary													4	X+
16SD08	2/21/95	9:30	0	Primary													93	
16SD09	2/21/95	10:55	0	Primary													3.5	X+
16SD10	2/21/95	17:10	0	Primary													3.5	X+
16SD11	2/28/95	11:15	0	Primary													3	X+
16WW01	5/4/93	0:00	0	Primary													3	X+
16WW01	5/4/93	0:00	0	Duplicate													2.9	X+
16WW01-A				Avg.													2.95	
16WW01	5/4/93	0:00	5	Primary													3.1	X+
16WW01	5/4/93	0:00	10	Primary													3.15	X+
16WW02	5/12/93	0:00	0	Primary													2.9	X+
16WW02	5/12/93	0:00	0	Duplicate													2.95	X+
16WW02-A				Avg.													2.925	
16WW02	5/12/93	0:00	5	Primary													3.1	X+
16WW03	5/4/93	0:00	0	Primary													3.15	X+
16WW03	5/4/93	0:00	5	Primary													3	X+
16WW03	5/4/93	0:00	10	Primary													3.05	X+
16WW03	5/4/93	0:00	15	Primary													3.15	X+
16WW04	5/12/93	0:00	0	Primary													3	X+
16WW04	5/12/93	0:00	0	Duplicate													3.15	X+
16WW04-A				Avg.													3.075	
16WW04	5/12/93	0:00	5	Primary													2.95	X+
16WW04	5/12/93	0:00	10	Primary													3.05	X+
16WW05	5/4/93	0:00	0	Primary													3.4	X+
16WW05	5/4/93	0:00	5	Primary													2.85	X+
16WW05	5/4/93	0:00	10	Primary													2.9	X+
16WW05	5/4/93	0:00	15	Primary													3	X+
16WW05	5/4/93	0:00	20	Primary													3.2	X+
16WW05	5/4/93	0:00	25	Primary													3.2	X+
16WW06	5/5/93	0:00	0	Primary													3.25	X+
16WW06	5/5/93	0:00	5	Primary													3.15	X+
16WW06	5/5/93	0:00	10	Primary													3	X+
16WW06	5/5/93	0:00	15	Primary													2.95	X+
16WW06	5/5/93	0:00	20	Primary													3.15	X+
16WW27	5/17/97	9:15	0	Primary	3	X+	3	X+	3	X+	3	X+	6.5	X+	3	X+	3	X+

Longhorn Army Ammuntion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	o-Xylene (ug/kg)		4-Chlorotoluene (ug/kg)		p-Isopropyl toluene (ug/kg)		1,4-Dichloro benzene (ug/kg)		Pentachloro ethane (ug/kg)		sec-Butyl benzene (ug/kg)		Styrene (ug/kg)	
16WW27	5/17/97	10:17	1	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW31	5/29/97	8:55	1	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW31	5/29/97	10:09	5	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW31	5/29/97	11:10	14	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	5.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	5.5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	5	X+	2.5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	5	X+	2.5	X+	2.5	X+
16WW35-A				Avg.	2.5		2.5		2.5		2.5		5.166667		2.5		2.5	
16WW35	6/25/97	10:05	1	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+	2	
16WW35	6/25/97	11:15	5	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW37	5/31/97	16:10	1	Primary	3	X+	3	X+	3	X+	3	X+	6.5	X+	3	X+	3	X+
16WW37	5/31/97	16:35	5	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+
16WW37	6/1/97	10:01	14	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	3	X+	3	X+

NATURAL LOGS

16SD01	1.459
16SD01	1.504
16SD02	1.147
16SD02	1.253
16SD03	1.179
16SD03	1.386
16SD06	1.253
16SD07	1.386
16SD08	4.533
16SD09	1.253
16SD10	1.253
16SD11	1.099
16WW01-A	1.082
16WW01	1.131
16WW01	1.147
16WW02-A	1.073
16WW02	1.131
16WW03	1.147
16WW03	1.099
16WW03	1.115
16WW03	1.147
16WW04-A	1.123
16WW04	1.082
16WW04	1.115
16WW05	1.224
16WW05	1.047

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	o-Xylene (ug/kg)	4-Chlorotoluene (ug/kg)	p-Isopropyl toluene (ug/kg)	1,4-Dichloro benzene (ug/kg)	Pentachloro ethane (ug/kg)	sec-Butyl benzene (ug/kg)	Styrene (ug/kg)
16WW05											1.065
16WW05											1.099
16WW05											1.163
16WW05											1.163
16WW06											1.179
16WW06											1.147
16WW06											1.099
16WW06											1.082
16WW06											1.147
16WW27					1.099	1.099	1.099	1.099	1.872	1.099	1.099
16WW27					1.099	1.099	1.099	1.099	1.792	1.099	1.099
16WW31					1.099	1.099	1.099	1.099	1.792	1.099	1.099
16WW31					1.099	1.099	1.099	1.099	1.792	1.099	1.099
16WW31					1.099	1.099	1.099	1.099	1.792	1.099	1.099
16WW31					0.916	0.916	0.916	0.916	1.705	0.916	0.916
16WW35-A					0.916	0.916	0.916	0.916	1.642	0.916	0.916
16WW35					1.099	1.099	1.099	1.099	1.792	1.099	0.693
16WW35					1.099	1.099	1.099	1.099	1.792	1.099	1.099
16WW37					1.099	1.099	1.099	1.099	1.792	1.099	1.099
16WW37					1.099	1.099	1.099	1.099	1.872	1.099	1.099
16WW37					1.099	1.099	1.099	1.099	1.792	1.099	1.099
16WW37					1.099	1.099	1.099	1.099	1.792	1.099	1.099
STATISTICS											
Total No.					13	13	13	13	13	13	48
Min.					2.5	2.5	2.5	2.5	5	2.5	2
Max.					3	3	3	3	6.5	3	93
Avg.(log transformed)					1.071	1.071	1.071	1.071	1.786	1.071	1.209
St. Dev.(log transformed)					0.069	0.069	0.069	0.069	0.059	0.069	0.506
H											1.885
95th UCL of Mean					2.93	2.93	2.93	2.93	5.98	2.93	4.38

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	TYPE	RESULT	tert-Butyl benzene (ug/kg)		Tetrachloro ethene (ug/kg)		Toluene (ug/kg)		Trans-1,3-Dichloropropene (ug/kg)		Trichloroethene (ug/kg)		Trichloro fluoromethane (ug/kg)		Vinyl Acetate (ug/kg)	
16SD01	5/2/93	0:00	0	Primary				4.3	X+	4.3	X+	4.3	X+	4.3	X+	4.3	X+	4.3	X+
16SD01	2/21/95	14:20	0	Primary				4.5	X+	4.5	X+	4.5	X+	4.5	X+	4.5	X+	9	X+
16SD02	4/14/93	0:00	0	Primary				3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+
16SD02	2/28/95	10:00	0	Primary				3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+	7	X+
16SD03	4/14/93	0:00	0	Primary				3.25	X+	3.25	X+	3.25	X+	3.25	X+	3.25	X+	3.25	X+
16SD03	2/20/95	16:05	0	Primary				4	X+	4	X+	4	X+	4	X+	4	X+	8	X+
16SD06	2/20/95	16:35	0	Primary				3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+	7.5	X+
16SD07	2/21/95	14:05	0	Primary				4	X+	4	X+	4	X+	4	X+	4	X+	8	X+
16SD08	2/21/95	9:30	0	Primary				3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+	6.5	X+
16SD09	2/21/95	10:55	0	Primary				3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+	6.5	X+
16SD10	2/21/95	17:10	0	Primary				3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+	6.5	X+
16SD11	2/28/95	11:15	0	Primary				3	X+	3	X+	3	X+	3	X+	3	X+	6.5	X+
16WW01	5/4/93	0:00	0	Primary				3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW01	5/4/93	0:00	0	Duplicate				2.9	X+	2.9	X+	2.9	X+	2.9	X+	2.9	X+	2.9	X+
16WW01-A				Avg.				2.95		2.95		2.95		2.95		2.95		2.95	
16WW01	5/4/93	0:00	5	Primary				3.1	X+	3.1	X+	3.1	X+	3.1	X+	3.1	X+	3.1	X+
16WW01	5/4/93	0:00	10	Primary				3.15	X+	3.15	X+	3.15	X+	9.8		3.15	X+	3.15	X+
16WW02	5/12/93	0:00	0	Primary				2.9	X+	2.9	X+	2.9	X+	2.9	X+	2.9	X+	2.9	X+
16WW02	5/12/93	0:00	0	Duplicate				2.95	X+	2.95	X+	2.95	X+	2.95	X+	2.95	X+	2.95	X+
16WW02-A				Avg.				2.925		2.925		2.925		2.925		2.925		2.925	
16WW02	5/12/93	0:00	5	Primary				3.1	X+	3.1	X+	3.1	X+	3.1	X+	3.1	X+	3.1	X+
16WW03	5/4/93	0:00	0	Primary				3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+
16WW03	5/4/93	0:00	5	Primary				3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW03	5/4/93	0:00	10	Primary				3.05	X+	3.05	X+	3.05	X+	220		3.05	X+	3.05	X+
16WW03	5/4/93	0:00	15	Primary				3.15	X+	3.15	X+	3.15	X+	64		3.15	X+	3.15	X+
16WW04	5/12/93	0:00	0	Primary				3	X+	3	X+	3	X+	65		3	X+	3	X+
16WW04	5/12/93	0:00	0	Duplicate				3.15	X+	3.15	X+	3.15	X+	220		3.15	X+	3.15	X+
16WW04-A				Avg.				3.075		3.075		3.075		142.5		3.075		3.075	
16WW04	5/12/93	0:00	5	Primary				2.95	X+	2.95	X+	2.95	X+	140		2.95	X+	2.95	X+
16WW04	5/12/93	0:00	10	Primary				3.05	X+	3.05	X+	3.05	X+	1500	J	3.05	X+	3.05	X+
16WW05	5/4/93	0:00	0	Primary				3.4	X+	3.4	X+	3.4	X+	3.4	X+	3.4	X+	3.4	X+
16WW05	5/4/93	0:00	5	Primary				2.85	X+	2.85	X+	2.85	X+	2.85	X+	2.85	X+	2.85	X+
16WW05	5/4/93	0:00	10	Primary				2.9	X+	2.9	X+	2.9	X+	2.9	X+	2.9	X+	2.9	X+
16WW05	5/4/93	0:00	15	Primary				3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW05	5/4/93	0:00	20	Primary				3.2	X+	3.2	X+	3.2	X+	3.2	X+	3.2	X+	3.2	X+
16WW05	5/4/93	0:00	25	Primary				3.2	X+	3.2	X+	3.2	X+	3.2	X+	3.2	X+	3.2	X+
16WW06	5/5/93	0:00	0	Primary				3.25	X+	3.25	X+	3.25	X+	3.25	X+	3.25	X+	3.25	X+
16WW06	5/5/93	0:00	5	Primary				3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+
16WW06	5/5/93	0:00	10	Primary				3	X+	3	X+	3	X+	3	X+	3	X+	3	X+
16WW06	5/5/93	0:00	15	Primary				2.95	X+	2.95	X+	2.95	X+	2.95	X+	2.95	X+	2.95	X+
16WW06	5/5/93	0:00	20	Primary				3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+	3.15	X+
16WW27	5/17/97	9:15	0	Primary	3		X+	3	X+	3	X+	3	X+	6.5	X+	6.5	X+	13	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	tert-Butyl benzene (ug/kg)		Tetrachloro ethene (ug/kg)		Toluene (ug/kg)		Trans-1,3-Dichloropropene (ug/kg)		Trichloroethene (ug/kg)		Trichloro fluoromethane (ug/kg)		Vinyl Acetate (ug/kg)	
16WW27	5/17/97	10:17	1	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	6	X+	12	X+
16WW31	5/29/97	8:27	0	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	6	X+	12	X+
16WW31	5/29/97	8:55	1	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	6	X+	12	X+
16WW31	5/29/97	10:09	5	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	6	X+	12	X+
16WW31	5/29/97	11:10	14	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	5.5	X+	5.5	X+	11	X+
16WW35	6/25/97	9:50	0	Primary	2.5	X+	2.5	X+	2.5	X+	2.5	X+	5.5	X+	5.5	X+	10.5	X+
16WW35	6/25/97	9:50	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	5	X+	5	X+	10	X+
16WW35	6/25/97	0:00	0	Duplicate	2.5	X+	2.5	X+	2.5	X+	2.5	X+	5	X+	5	X+	10	X+
16WW35-A				Avg.	2.5		2.5		2.5		2.5		5.166667		5.166667		10.16667	
16WW35	6/25/97	10:05	1	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	6	X+	12.5	X+
16WW35	6/25/97	11:15	5	Primary	3	X+	3	X+	3	X+	3	X+	18		6	X+	12.5	X+
16WW37	5/31/97	15:55	0	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	6	X+	12.5	X+
16WW37	5/31/97	16:10	1	Primary	3	X+	3	X+	3	X+	3	X+	6.5	X+	6.5	X+	12.5	X+
16WW37	5/31/97	16:35	5	Primary	3	X+	3	X+	1	U+	3	X+	6	X+	6	X+	12	X+
16WW37	6/1/97	10:01	14	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	6	X+	12	X+

NATURAL LOGS

16SD01							1.459		1.459		1.459		1.459		1.459		1.459	
16SD01							1.504		1.504		1.504		1.504		1.504		2.197	
16SD02							1.147		1.147		1.147		1.147		1.147		1.147	
16SD02							1.253		1.253		1.253		1.253		1.253		1.946	
16SD03							1.179		1.179		1.179		1.179		1.179		1.179	
16SD03							1.386		1.386		1.386		1.386		1.386		2.079	
16SD06							1.253		1.253		1.253		1.253		1.253		2.015	
16SD07							1.386		1.386		1.386		1.386		1.386		2.079	
16SD08							1.253		1.253		1.253		1.253		1.253		1.872	
16SD09							1.253		1.253		1.253		1.253		1.253		1.872	
16SD10							1.253		1.253		1.253		1.253		1.253		1.872	
16SD11							1.099		1.099		1.099		1.099		1.099		1.872	
16WW01-A							1.082		1.082		1.082		1.082		1.082		1.082	
16WW01							1.131		1.131		1.131		1.131		1.131		1.131	
16WW01							1.147		1.147		1.147		2.282		1.147		1.147	
16WW02-A							1.073		1.073		1.073		1.073		1.073		1.073	
16WW02							1.131		1.131		1.131		1.131		1.131		1.131	
16WW03							1.147		1.147		1.147		1.147		1.147		1.147	
16WW03							1.099		1.099		1.099		1.099		1.099		1.099	
16WW03							1.115		1.115		1.115		5.394		1.115		1.115	
16WW03							1.147		1.147		1.147		4.159		1.147		1.147	
16WW04-A							1.123		1.123		1.123		4.959		1.123		1.123	
16WW04							1.082		1.082		1.082		4.942		1.082		1.082	
16WW04							1.115		1.115		1.115		7.313		1.115		1.115	
16WW05							1.224		1.224		1.224		1.224		1.224		1.224	
16WW05							1.047		1.047		1.047		1.047		1.047		1.047	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	TYPE	RESULT	tert-Butyl benzene (ug/kg)	Tetrachloro ethene (ug/kg)	Toluene (ug/kg)	Trans-1,3-Dichloropropene (ug/kg)	Trichloroethene (ug/kg)	Trichloro fluoromethane (ug/kg)	Vinyl Acetate (ug/kg)
16WW05							1.065	1.065	1.065	1.065	1.065	1.065
16WW05							1.099	1.099	1.099	1.099	1.099	1.099
16WW05							1.163	1.163	1.163	1.163	1.163	1.163
16WW05							1.163	1.163	1.163	1.163	1.163	1.163
16WW06							1.179	1.179	1.179	1.179	1.179	1.179
16WW06							1.147	1.147	1.147	1.147	1.147	1.147
16WW06							1.099	1.099	1.099	1.099	1.099	1.099
16WW06							1.082	1.082	1.082	1.082	1.082	1.082
16WW06							1.147	1.147	1.147	1.147	1.147	1.147
16WW27						1.099	1.099	1.099	1.099	1.872	1.872	2.565
16WW27						1.099	1.099	1.099	1.099	1.792	1.792	2.485
16WW31						1.099	1.099	1.099	1.099	1.792	1.792	2.485
16WW31						1.099	1.099	1.099	1.099	1.792	1.792	2.485
16WW31						1.099	1.099	1.099	1.099	1.792	1.792	2.485
16WW31						0.916	0.916	0.916	0.916	1.705	1.705	2.398
16WW35-A						0.916	0.916	0.916	0.916	1.642	1.642	2.319
16WW35						1.099	1.099	1.099	1.099	1.792	1.792	2.526
16WW35						1.099	1.099	1.099	1.099	2.89	1.792	2.526
16WW37						1.099	1.099	1.099	1.099	1.792	1.792	2.526
16WW37						1.099	1.099	1.099	1.099	1.872	1.872	2.526
16WW37						1.099	1.099	0	1.099	1.792	1.792	2.485
16WW37						1.099	1.099	1.099	1.099	1.792	1.792	2.485
STATISTICS												
Total No.						13	48	48	48	48	48	48
Min.						2.5	2.5	1	2.5	2.85	2.85	2.85
Max.						3	4.5	4.5	4.5	1500	6.5	13
Avg.(log transformed)						1.071	1.149	1.126	1.149	1.831	1.343	1.66
St. Dev.(log transformed)						0.069	0.111	0.2	0.111	1.316	0.29	0.604
H										2.677		
95th UCL of Mean						2.93	3.17	3.15	3.17	24.80	4.00	6.31

Longhorn Army Ammuntion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Vinyl chloride (ug/kg)		Xylene (total) (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	8.5	X+	4.3	X+
16SD01	2/21/95	14:20	0	Primary	9	X+	4.5	X+
16SD02	4/14/93	0:00	0	Primary	6.5	X+	3.15	X+
16SD02	2/28/95	10:00	0	Primary	7	X+	3.5	X+
16SD03	4/14/93	0:00	0	Primary	6.5	X+	3.25	X+
16SD03	2/20/95	16:05	0	Primary	8	X+	4	X+
16SD06	2/20/95	16:35	0	Primary	7.5	X+	3.5	X+
16SD07	2/21/95	14:05	0	Primary	8	X+	4	X+
16SD08	2/21/95	9:30	0	Primary	6.5	X+	3.5	X+
16SD09	2/21/95	10:55	0	Primary	6.5	X+	3.5	X+
16SD10	2/21/95	17:10	0	Primary	6.5	X+	3.5	X+
16SD11	2/28/95	11:15	0	Primary	6.5	X+	3	X+
16WW01	5/4/93	0:00	0	Primary	6	X+	3	X+
16WW01	5/4/93	0:00	0	Duplicate	6	X+	2.9	X+
16WW01-A				Avg.	6		2.95	
16WW01	5/4/93	0:00	5	Primary	6	X+	3.1	X+
16WW01	5/4/93	0:00	10	Primary	6.5	X+	3.15	X+
16WW02	5/12/93	0:00	0	Primary	6	X+	2.9	X+
16WW02	5/12/93	0:00	0	Duplicate	6	X+	2.95	X+
16WW02-A				Avg.	6		2.925	
16WW02	5/12/93	0:00	5	Primary	6	X+	3.1	X+
16WW03	5/4/93	0:00	0	Primary	6.5	X+	3.15	X+
16WW03	5/4/93	0:00	5	Primary	6	X+	3	X+
16WW03	5/4/93	0:00	10	Primary	6	X+	3.05	X+
16WW03	5/4/93	0:00	15	Primary	6.5	X+	3.15	X+
16WW04	5/12/93	0:00	0	Primary	6	X+	3	X+
16WW04	5/12/93	0:00	0	Duplicate	6.5	X+	3.15	X+
16WW04-A				Avg.	6.25		3.075	
16WW04	5/12/93	0:00	5	Primary	6	X+	2.95	X+
16WW04	5/12/93	0:00	10	Primary	6	X+	3.05	X+
16WW05	5/4/93	0:00	0	Primary	7	X+	3.4	X+
16WW05	5/4/93	0:00	5	Primary	5.5	X+	2.85	X+
16WW05	5/4/93	0:00	10	Primary	6	X+	2.9	X+
16WW05	5/4/93	0:00	15	Primary	6	X+	3	X+
16WW05	5/4/93	0:00	20	Primary	6.5	X+	3.2	X+
16WW05	5/4/93	0:00	25	Primary	6.5	X+	3.2	X+
16WW06	5/5/93	0:00	0	Primary	6.5	X+	3.25	X+
16WW06	5/5/93	0:00	5	Primary	6.5	X+	3.15	X+
16WW06	5/5/93	0:00	10	Primary	6	X+	3	X+
16WW06	5/5/93	0:00	15	Primary	6	X+	2.95	X+
16WW06	5/5/93	0:00	20	Primary	6.5	X+	3.15	X+
16WW27	5/17/97	9:15	0	Primary	6.5	X+	3	X+

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Vinyl chloride (ug/kg)		Xylene (total) (ug/kg)	
16WW27	5/17/97	10:17	1	Primary	6	X+	3	X+
16WW31	5/29/97	8:27	0	Primary	6	X+	3	X+
16WW31	5/29/97	8:55	1	Primary	6	X+	3	X+
16WW31	5/29/97	10:09	5	Primary	6	X+	3	X+
16WW31	5/29/97	11:10	14	Primary	5.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Primary	5.5	X+	2.5	X+
16WW35	6/25/97	9:50	0	Duplicate	5	X+	2.5	X+
16WW35	6/25/97	0:00	0	Duplicate	5	X+	2.5	X+
16WW35-A				Avg.	5.166667		2.5	
16WW35	6/25/97	10:05	1	Primary	6	X+	3	X+
16WW35	6/25/97	11:15	5	Primary	6	X+	3	X+
16WW37	5/31/97	15:55	0	Primary	6	X+	3	X+
16WW37	5/31/97	16:10	1	Primary	6.5	X+	3	X+
16WW37	5/31/97	16:35	5	Primary	6	X+	3	X+
16WW37	6/1/97	10:01	14	Primary	6	X+	3	X+

NATURAL LOGS

16SD01	2.14	1.459
16SD01	2.197	1.504
16SD02	1.872	1.147
16SD02	1.946	1.253
16SD03	1.872	1.179
16SD03	2.079	1.386
16SD06	2.015	1.253
16SD07	2.079	1.386
16SD08	1.872	1.253
16SD09	1.872	1.253
16SD10	1.872	1.253
16SD11	1.872	1.099
16WW01-A	1.792	1.082
16WW01	1.792	1.131
16WW01	1.872	1.147
16WW02-A	1.792	1.073
16WW02	1.792	1.131
16WW03	1.872	1.147
16WW03	1.792	1.099
16WW03	1.792	1.115
16WW03	1.872	1.147
16WW04-A	1.833	1.123
16WW04	1.792	1.082
16WW04	1.792	1.115
16WW05	1.946	1.224
16WW05	1.705	1.047

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Volatile Organic Compounds (VOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Vinyl chloride (ug/kg)	Xylene (total) (ug/kg)
16WW05					1.792	1.065
16WW05					1.792	1.099
16WW05					1.872	1.163
16WW05					1.872	1.163
16WW06					1.872	1.179
16WW06					1.872	1.147
16WW06					1.792	1.099
16WW06					1.792	1.082
16WW06					1.872	1.147
16WW27					1.872	1.099
16WW27					1.792	1.099
16WW31					1.792	1.099
16WW31					1.792	1.099
16WW31					1.792	1.099
16WW31					1.705	0.916
16WW35-A					1.642	0.916
16WW35					1.792	1.099
16WW35					1.792	1.099
16WW37					1.792	1.099
16WW37					1.872	1.099
16WW37					1.792	1.099
16WW37					1.792	1.099
STATISTICS						
Total No.					48	48
Min.					5	2.5
Max.					9	4.5
Avg.(log transformed)					1.851	1.149
St. Dev.(log transformed)					0.105	0.111
H						
95th UCL of Mean					6.40	3.17

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,2,4-Trichloro benzene (ug/kg)		1-Chloro naphthalene (ug/kg)		2,4,5-Trichloro phenol (ug/kg)		2,4,6-Trichloro phenol (ug/kg)		2,4-Dichloro phenol (ug/kg)		2,4-Dimethyl phenol (ug/kg)		2,4-Dinitro phenol (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	569	X+	1379.5	X+	284.5	X+	284.5	X+	284.5	X+	1379.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	417.5	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	428.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+	1039	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	393	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+	952.5	X+
16WW01	5/4/93	0:00	0	Duplicate	192	X+	383.5	X+	930	X+	192	X+	192	X+	192	X+	930	X+
16WW01-A				Avg.	194.25		388.25		941.25		194.25		194.25		194.25		941.25	
16WW01	5/4/93	0:00	5	Primary	203.5	X+	407.5	X+	987.5	X+	203.5	X+	203.5	X+	203.5	X+	987.5	X+
16WW01	5/4/93	0:00	10	Primary	206.5	X+	412.5	X+	1000	X+	206.5	X+	206.5	X+	206.5	X+	1000	X+
16WW02	5/12/93	0:00	0	Primary	192	X+	383.5	X+	930	X+	192	X+	192	X+	192	X+	930	X+
16WW02	5/12/93	0:00	0	Duplicate	194	X+	388	X+	941	X+	194	X+	194	X+	194	X+	941	X+
16WW02-A				Avg.	193		385.75		935.5		193		193		193		935.5	
16WW02	5/12/93	0:00	5	Primary	203.5	X+	407.5	X+	987.5	X+	203.5	X+	203.5	X+	203.5	X+	987.5	X+
16WW03	5/4/93	0:00	0	Primary	206.5	X+	412.5	X+	1000	X+	206.5	X+	206.5	X+	206.5	X+	1000	X+
16WW03	5/4/93	0:00	5	Primary	196.5	X+	393	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+	952.5	X+
16WW03	5/4/93	0:00	10	Primary	201	X+	402.5	X+	975.5	X+	201	X+	201	X+	201	X+	975.5	X+
16WW03	5/4/93	0:00	15	Primary	209	X+	417.5	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	397.5	X+	964	X+	199	X+	199	X+	199	X+	964	X+
16WW04	5/12/93	0:00	0	Duplicate	209	X+	417.5	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+
16WW04-A				Avg.	204		407.5		988.25		204		204		204		988.25	
16WW04	5/12/93	0:00	5	Primary	194	X+	388	X+	941	X+	194	X+	194	X+	194	X+	941	X+
16WW04	5/12/93	0:00	10	Primary	199	X+	397.5	X+	964	X+	199	X+	199	X+	199	X+	964	X+
16WW04	5/12/93	0:00	10	Duplicate	201	X+	402.5	X+	975.5	X+	201	X+	201	X+	201	X+	975.5	X+
16WW04-A				Avg.	200		400		969.75		200		200		200		969.75	
16WW05	5/4/93	0:00	0	Primary	223	X+	446	X+	1081	X+	223	X+	223	X+	223	X+	1081	X+
16WW05	5/4/93	0:00	5	Primary	187.5	X+	375	X+	909	X+	187.5	X+	187.5	X+	187.5	X+	909	X+
16WW05	5/4/93	0:00	10	Primary	192	X+	383.5	X+	930	X+	192	X+	192	X+	192	X+	930	X+
16WW05	5/4/93	0:00	15	Primary	196.5	X+	393	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+	952.5	X+
16WW05	5/4/93	0:00	20	Primary	211.5	X+	423	X+	1025.5	X+	211.5	X+	211.5	X+	211.5	X+	1025.5	X+
16WW05	5/4/93	0:00	25	Primary	211.5	X+	423	X+	1025.5	X+	211.5	X+	211.5	X+	211.5	X+	1025.5	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	428.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+	1039	X+
16WW06	5/5/93	0:00	5	Primary	209	X+	417.5	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+
16WW06	5/5/93	0:00	10	Primary	196.5	X+	393	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+	952.5	X+
16WW06	5/5/93	0:00	15	Primary	194	X+	388	X+	941	X+	194	X+	194	X+	194	X+	941	X+
16WW06	5/5/93	0:00	20	Primary	209	X+	417.5	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+
NATURAL LOGS																		
16SD01					5.651		6.344		7.229		5.651		5.651		5.651		7.229	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	RESULT DEPTH (ft TYPE	1,2,4-Trichloro benzene (ug/kg)	1-Chloro naphthalene (ug/kg)	2,4,5-Trichloro phenol (ug/kg)	2,4,6-Trichloro phenol (ug/kg)	2,4-Dichloro phenol (ug/kg)	2,4-Dimethyl phenol (ug/kg)	2,4-Dinitro phenol (ug/kg)
16SD02				5.342	6.034	6.92	5.342	5.342	5.342	6.92
16SD03				5.368	6.06	6.946	5.368	5.368	5.368	6.946
16WW01-A				5.269	5.962	6.847	5.269	5.269	5.269	6.847
16WW01				5.316	6.01	6.895	5.316	5.316	5.316	6.895
16WW01				5.33	6.022	6.908	5.33	5.33	5.33	6.908
16WW02-A				5.263	5.955	6.841	5.263	5.263	5.263	6.841
16WW02				5.316	6.01	6.895	5.316	5.316	5.316	6.895
16WW03				5.33	6.022	6.908	5.33	5.33	5.33	6.908
16WW03				5.281	5.974	6.859	5.281	5.281	5.281	6.859
16WW03				5.303	5.998	6.883	5.303	5.303	5.303	6.883
16WW03				5.342	6.034	6.92	5.342	5.342	5.342	6.92
16WW04-A				5.318	6.01	6.896	5.318	5.318	5.318	6.896
16WW04				5.268	5.961	6.847	5.268	5.268	5.268	6.847
16WW04-A				5.298	5.991	6.877	5.298	5.298	5.298	6.877
16WW05				5.407	6.1	6.986	5.407	5.407	5.407	6.986
16WW05				5.234	5.927	6.812	5.234	5.234	5.234	6.812
16WW05				5.257	5.949	6.835	5.257	5.257	5.257	6.835
16WW05				5.281	5.974	6.859	5.281	5.281	5.281	6.859
16WW05				5.354	6.047	6.933	5.354	5.354	5.354	6.933
16WW05				5.354	6.047	6.933	5.354	5.354	5.354	6.933
16WW06				5.368	6.06	6.946	5.368	5.368	5.368	6.946
16WW06				5.342	6.034	6.92	5.342	5.342	5.342	6.92
16WW06				5.281	5.974	6.859	5.281	5.281	5.281	6.859
16WW06				5.268	5.961	6.847	5.268	5.268	5.268	6.847
16WW06				5.342	6.034	6.92	5.342	5.342	5.342	6.92
STATISTICS										
Total No.				26	26	26	26	26	26	26
Min.				187.5	375	909	187.5	187.5	187.5	909
Max.				284.5	569	1379.5	284.5	284.5	284.5	1379.5
Avg.(log transformed)				5.326	6.019	6.905	5.326	5.326	5.326	6.905
St. Dev.(log transformed)				0.078	0.078	0.078	0.078	0.078	0.078	0.078
H										
95th UCL of Mean				206.24	412.42	1000.29	206.24	206.24	206.24	1000.29

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	2,4-Dinitro toluene (ug/kg)		2,6-Dinitro toluene (ug/kg)		2-Chloro napthalene (ug/kg)		2-Chlorophenol (ug/kg)		2-Methyl napthalene (ug/kg)		3,3'-Dichloro benzidine (ug/kg)		4,6-Dinitro-2-methylphenol (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	284.5	X+	284.5	X+	284.5	X+	284.5	X+	569	X+	1379.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	209	X+	209	X+	209	X+	209	X+	417.5	X+	1012.5	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	428.5	X+	1039	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	393	X+	952.5	X+
16WW01	5/4/93	0:00	0	Duplicate	192	X+	192	X+	192	X+	192	X+	192	X+	383.5	X+	930	X+
16WW01-A				Avg.	194.25		194.25		194.25		194.25		194.25		388.25		941.25	
16WW01	5/4/93	0:00	5	Primary	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+	407.5	X+	987.5	X+
16WW01	5/4/93	0:00	10	Primary	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+	412.5	X+	1000	X+
16WW02	5/12/93	0:00	0	Primary	192	X+	192	X+	192	X+	192	X+	192	X+	383.5	X+	930	X+
16WW02	5/12/93	0:00	0	Duplicate	194	X+	194	X+	194	X+	194	X+	194	X+	388	X+	941	X+
16WW02-A				Avg.	193		193		193		193		193		385.75		935.5	
16WW02	5/12/93	0:00	5	Primary	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+	407.5	X+	987.5	X+
16WW03	5/4/93	0:00	0	Primary	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+	412.5	X+	1000	X+
16WW03	5/4/93	0:00	5	Primary	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	393	X+	952.5	X+
16WW03	5/4/93	0:00	10	Primary	201	X+	201	X+	201	X+	201	X+	201	X+	402.5	X+	975.5	X+
16WW03	5/4/93	0:00	15	Primary	209	X+	209	X+	209	X+	209	X+	209	X+	417.5	X+	1012.5	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	199	X+	199	X+	199	X+	199	X+	397.5	X+	964	X+
16WW04	5/12/93	0:00	0	Duplicate	209	X+	209	X+	209	X+	209	X+	209	X+	417.5	X+	1012.5	X+
16WW04-A				Avg.	204		204		204		204		204		407.5		988.25	
16WW04	5/12/93	0:00	5	Primary	194	X+	194	X+	194	X+	194	X+	194	X+	388	X+	941	X+
16WW04	5/12/93	0:00	10	Primary	199	X+	199	X+	199	X+	199	X+	199	X+	397.5	X+	964	X+
16WW04	5/12/93	0:00	10	Duplicate	201	X+	201	X+	201	X+	201	X+	201	X+	402.5	X+	975.5	X+
16WW04-A				Avg.	200		200		200		200		200		400		969.75	
16WW05	5/4/93	0:00	0	Primary	223	X+	223	X+	223	X+	223	X+	223	X+	446	X+	1081	X+
16WW05	5/4/93	0:00	5	Primary	187.5	X+	187.5	X+	187.5	X+	187.5	X+	187.5	X+	375	X+	909	X+
16WW05	5/4/93	0:00	10	Primary	192	X+	192	X+	192	X+	192	X+	192	X+	383.5	X+	930	X+
16WW05	5/4/93	0:00	15	Primary	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	393	X+	952.5	X+
16WW05	5/4/93	0:00	20	Primary	211.5	X+	211.5	X+	211.5	X+	211.5	X+	211.5	X+	423	X+	1025.5	X+
16WW05	5/4/93	0:00	25	Primary	211.5	X+	211.5	X+	211.5	X+	211.5	X+	211.5	X+	423	X+	1025.5	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	428.5	X+	1039	X+
16WW06	5/5/93	0:00	5	Primary	209	X+	209	X+	209	X+	209	X+	209	X+	417.5	X+	1012.5	X+
16WW06	5/5/93	0:00	10	Primary	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	393	X+	952.5	X+
16WW06	5/5/93	0:00	15	Primary	194	X+	194	X+	194	X+	194	X+	194	X+	388	X+	941	X+
16WW06	5/5/93	0:00	20	Primary	209	X+	209	X+	209	X+	209	X+	209	X+	417.5	X+	1012.5	X+
NATURAL LOGS																		
16SD01					5.651		5.651		5.651		5.651		5.651		6.344		7.229	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	2,4-Dinitro toluene (ug/kg)	2,6-Dinitro toluene (ug/kg)	2-Chloro napthalene (ug/kg)	2-Chlorophenol (ug/kg)	2-Methyl napthalene (ug/kg)	3,3'-Dichloro benzidine (ug/kg)	4,6-Dinitro-2-methylphenol (ug/kg)
16SD02					5.342	5.342	5.342	5.342	5.342	6.034	6.92
16SD03					5.368	5.368	5.368	5.368	5.368	6.06	6.946
16WW01-A					5.269	5.269	5.269	5.269	5.269	5.962	6.847
16WW01					5.316	5.316	5.316	5.316	5.316	6.01	6.895
16WW01					5.33	5.33	5.33	5.33	5.33	6.022	6.908
16WW02-A					5.263	5.263	5.263	5.263	5.263	5.955	6.841
16WW02					5.316	5.316	5.316	5.316	5.316	6.01	6.895
16WW03					5.33	5.33	5.33	5.33	5.33	6.022	6.908
16WW03					5.281	5.281	5.281	5.281	5.281	5.974	6.859
16WW03					5.303	5.303	5.303	5.303	5.303	5.998	6.883
16WW03					5.342	5.342	5.342	5.342	5.342	6.034	6.92
16WW04-A					5.318	5.318	5.318	5.318	5.318	6.01	6.896
16WW04					5.268	5.268	5.268	5.268	5.268	5.961	6.847
16WW04-A					5.298	5.298	5.298	5.298	5.298	5.991	6.877
16WW05					5.407	5.407	5.407	5.407	5.407	6.1	6.986
16WW05					5.234	5.234	5.234	5.234	5.234	5.927	6.812
16WW05					5.257	5.257	5.257	5.257	5.257	5.949	6.835
16WW05					5.281	5.281	5.281	5.281	5.281	5.974	6.859
16WW05					5.354	5.354	5.354	5.354	5.354	6.047	6.933
16WW05					5.354	5.354	5.354	5.354	5.354	6.047	6.933
16WW06					5.368	5.368	5.368	5.368	5.368	6.06	6.946
16WW06					5.342	5.342	5.342	5.342	5.342	6.034	6.92
16WW06					5.281	5.281	5.281	5.281	5.281	5.974	6.859
16WW06					5.268	5.268	5.268	5.268	5.268	5.961	6.847
16WW06					5.342	5.342	5.342	5.342	5.342	6.034	6.92
STATISTICS											
Total No.					26	26	26	26	26	26	26
Min.					187.5	187.5	187.5	187.5	187.5	375	909
Max.					284.5	284.5	284.5	284.5	284.5	569	1379.5
Avg.(log transformed)					5.326	5.326	5.326	5.326	5.326	6.019	6.905
St. Dev.(log transformed)					0.078	0.078	0.078	0.078	0.078	0.078	0.078
H											
95th UCL of Mean					206.24	206.24	206.24	206.24	206.24	412.42	1000.29

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	4-Bromophenyl phenyl ether (ug/kg)		4-Chlorophenyl phenyl ether (ug/kg)		Acenaphthene (ug/kg)		Acenaphthylene (ug/kg)		Anthracene (ug/kg)		Benzo(a) anthracene (ug/kg)		Benzo(a)pyrene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	284.5	X+	284.5	X+	284.5	X+	284.5	X+	284.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+
16WW01-A				Avg.	194.25		194.25		194.25		194.25		194.25		194.25		194.25	
16WW01	5/4/93	0:00	5	Primary	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+
16WW01	5/4/93	0:00	10	Primary	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+
16WW02	5/12/93	0:00	0	Primary	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+
16WW02-A				Avg.	193		193		193		193		193		193		193	
16WW02	5/12/93	0:00	5	Primary	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+
16WW03	5/4/93	0:00	0	Primary	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+
16WW03	5/4/93	0:00	5	Primary	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW03	5/4/93	0:00	10	Primary	201	X+	201	X+	201	X+	201	X+	201	X+	201	X+	201	X+
16WW03	5/4/93	0:00	15	Primary	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	199	X+	199	X+	199	X+	199	X+	199	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+
16WW04-A				Avg.	204		204		204		204		204		204		204	
16WW04	5/12/93	0:00	5	Primary	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+
16WW04	5/12/93	0:00	10	Primary	199	X+	199	X+	199	X+	199	X+	199	X+	199	X+	199	X+
16WW04	5/12/93	0:00	10	Duplicate	201	X+	201	X+	201	X+	201	X+	201	X+	201	X+	201	X+
16WW04-A				Avg.	200		200		200		200		200		200		200	
16WW05	5/4/93	0:00	0	Primary	223	X+	223	X+	223	X+	223	X+	223	X+	223	X+	223	X+
16WW05	5/4/93	0:00	5	Primary	187.5	X+	187.5	X+	187.5	X+	187.5	X+	187.5	X+	187.5	X+	187.5	X+
16WW05	5/4/93	0:00	10	Primary	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+
16WW05	5/4/93	0:00	15	Primary	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW05	5/4/93	0:00	20	Primary	211.5	X+	211.5	X+	211.5	X+	211.5	X+	211.5	X+	211.5	X+	211.5	X+
16WW05	5/4/93	0:00	25	Primary	211.5	X+	211.5	X+	211.5	X+	211.5	X+	211.5	X+	211.5	X+	211.5	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+
16WW06	5/5/93	0:00	5	Primary	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+
16WW06	5/5/93	0:00	10	Primary	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW06	5/5/93	0:00	15	Primary	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+
16WW06	5/5/93	0:00	20	Primary	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+
NATURAL LOGS																		
16SD01					5.651		5.651		5.651		5.651		5.651		5.651		5.651	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	4-Bromophenyl phenyl ether (ug/kg)	4-Chlorophenyl phenyl ether (ug/kg)	Acenaphthene (ug/kg)	Acenaphthylene (ug/kg)	Anthracene (ug/kg)	Benzo(a) anthracene (ug/kg)	Benzo(a)pyrene (ug/kg)
16SD02					5.342	5.342	5.342	5.342	5.342	5.342	5.342
16SD03					5.368	5.368	5.368	5.368	5.368	5.368	5.368
16WW01-A					5.269	5.269	5.269	5.269	5.269	5.269	5.269
16WW01					5.316	5.316	5.316	5.316	5.316	5.316	5.316
16WW01					5.33	5.33	5.33	5.33	5.33	5.33	5.33
16WW02-A					5.263	5.263	5.263	5.263	5.263	5.263	5.263
16WW02					5.316	5.316	5.316	5.316	5.316	5.316	5.316
16WW03					5.33	5.33	5.33	5.33	5.33	5.33	5.33
16WW03					5.281	5.281	5.281	5.281	5.281	5.281	5.281
16WW03					5.303	5.303	5.303	5.303	5.303	5.303	5.303
16WW03					5.342	5.342	5.342	5.342	5.342	5.342	5.342
16WW04-A					5.318	5.318	5.318	5.318	5.318	5.318	5.318
16WW04					5.268	5.268	5.268	5.268	5.268	5.268	5.268
16WW04-A					5.298	5.298	5.298	5.298	5.298	5.298	5.298
16WW05					5.407	5.407	5.407	5.407	5.407	5.407	5.407
16WW05					5.234	5.234	5.234	5.234	5.234	5.234	5.234
16WW05					5.257	5.257	5.257	5.257	5.257	5.257	5.257
16WW05					5.281	5.281	5.281	5.281	5.281	5.281	5.281
16WW05					5.354	5.354	5.354	5.354	5.354	5.354	5.354
16WW05					5.354	5.354	5.354	5.354	5.354	5.354	5.354
16WW06					5.368	5.368	5.368	5.368	5.368	5.368	5.368
16WW06					5.342	5.342	5.342	5.342	5.342	5.342	5.342
16WW06					5.281	5.281	5.281	5.281	5.281	5.281	5.281
16WW06					5.268	5.268	5.268	5.268	5.268	5.268	5.268
16WW06					5.342	5.342	5.342	5.342	5.342	5.342	5.342
STATISTICS											
Total No.					26	26	26	26	26	26	26
Min.					187.5	187.5	187.5	187.5	187.5	187.5	187.5
Max.					284.5	284.5	284.5	284.5	284.5	284.5	284.5
Avg.(log transformed)					5.326	5.326	5.326	5.326	5.326	5.326	5.326
St. Dev.(log transformed)					0.078	0.078	0.078	0.078	0.078	0.078	0.078
H											
95th UCL of Mean					206.24	206.24	206.24	206.24	206.24	206.24	206.24

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Benzo(b) fluoranthene (ug/kg)		Benzo(g,h,i) perylene (ug/kg)		Benzo(k)fluor anthene (ug/kg)		Benzoic acid (ug/kg)		Benzyl alcohol (ug/kg)		Bis(2-chloro-1-methylethyl) ether (ug/kg)		Bis(2-chloro ethoxy) methane (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	284.5	X+	284.5	X+	1379.5	X+	284.5	X+	284.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	209	X+	209	X+	1012.5	X+	209	X+	209	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate	192	X+	192	X+	192	X+	930	X+	192	X+	192	X+	192	X+
16WW01-A				Avg.	194.25		194.25		194.25		941.25		194.25		194.25		194.25	
16WW01	5/4/93	0:00	5	Primary	203.5	X+	203.5	X+	203.5	X+	987.5	X+	203.5	X+	203.5	X+	203.5	X+
16WW01	5/4/93	0:00	10	Primary	206.5	X+	206.5	X+	206.5	X+	1000	X+	206.5	X+	206.5	X+	206.5	X+
16WW02	5/12/93	0:00	0	Primary	192	X+	192	X+	192	X+	930	X+	192	X+	192	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate	194	X+	194	X+	194	X+	941	X+	194	X+	194	X+	194	X+
16WW02-A				Avg.	193		193		193		935.5		193		193		193	
16WW02	5/12/93	0:00	5	Primary	203.5	X+	203.5	X+	203.5	X+	987.5	X+	203.5	X+	203.5	X+	203.5	X+
16WW03	5/4/93	0:00	0	Primary	206.5	X+	206.5	X+	206.5	X+	1000	X+	206.5	X+	206.5	X+	206.5	X+
16WW03	5/4/93	0:00	5	Primary	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW03	5/4/93	0:00	10	Primary	201	X+	201	X+	201	X+	975.5	X+	201	X+	201	X+	201	X+
16WW03	5/4/93	0:00	15	Primary	209	X+	209	X+	209	X+	1012.5	X+	209	X+	209	X+	209	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	199	X+	199	X+	964	X+	199	X+	199	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate	209	X+	209	X+	209	X+	1012.5	X+	209	X+	209	X+	209	X+
16WW04-A				Avg.	204		204		204		988.25		204		204		204	
16WW04	5/12/93	0:00	5	Primary	194	X+	194	X+	194	X+	941	X+	194	X+	194	X+	194	X+
16WW04	5/12/93	0:00	10	Primary	199	X+	199	X+	199	X+	964	X+	199	X+	199	X+	199	X+
16WW04	5/12/93	0:00	10	Duplicate	201	X+	201	X+	201	X+	975.5	X+	201	X+	201	X+	201	X+
16WW04-A				Avg.	200		200		200		969.75		200		200		200	
16WW05	5/4/93	0:00	0	Primary	223	X+	223	X+	223	X+	1081	X+	223	X+	223	X+	223	X+
16WW05	5/4/93	0:00	5	Primary	187.5	X+	187.5	X+	187.5	X+	909	X+	187.5	X+	187.5	X+	187.5	X+
16WW05	5/4/93	0:00	10	Primary	192	X+	192	X+	192	X+	930	X+	192	X+	192	X+	192	X+
16WW05	5/4/93	0:00	15	Primary	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW05	5/4/93	0:00	20	Primary	211.5	X+	211.5	X+	211.5	X+	1025.5	X+	211.5	X+	211.5	X+	211.5	X+
16WW05	5/4/93	0:00	25	Primary	211.5	X+	211.5	X+	211.5	X+	1025.5	X+	211.5	X+	211.5	X+	211.5	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+
16WW06	5/5/93	0:00	5	Primary	209	X+	209	X+	209	X+	1012.5	X+	209	X+	209	X+	209	X+
16WW06	5/5/93	0:00	10	Primary	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW06	5/5/93	0:00	15	Primary	194	X+	194	X+	194	X+	941	X+	194	X+	194	X+	194	X+
16WW06	5/5/93	0:00	20	Primary	209	X+	209	X+	209	X+	1012.5	X+	209	X+	209	X+	209	X+
NATURAL LOGS																		
16SD01					5.651		5.651		5.651		7.229		5.651		5.651		5.651	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Benzo(b) fluoranthene (ug/kg)	Benzo(g,h,i) perylene (ug/kg)	Benzo(k)fluor anthene (ug/kg)	Benzoic acid (ug/kg)	Benzyl alcohol (ug/kg)	Bis(2-chloro-1-methylethyl) ether (ug/kg)	Bis(2-chloro ethoxy) methane (ug/kg)
16SD02					5.342	5.342	5.342	6.92	5.342	5.342	5.342
16SD03					5.368	5.368	5.368	6.946	5.368	5.368	5.368
16WW01-A					5.269	5.269	5.269	6.847	5.269	5.269	5.269
16WW01					5.316	5.316	5.316	6.895	5.316	5.316	5.316
16WW01					5.33	5.33	5.33	6.908	5.33	5.33	5.33
16WW02-A					5.263	5.263	5.263	6.841	5.263	5.263	5.263
16WW02					5.316	5.316	5.316	6.895	5.316	5.316	5.316
16WW03					5.33	5.33	5.33	6.908	5.33	5.33	5.33
16WW03					5.281	5.281	5.281	6.859	5.281	5.281	5.281
16WW03					5.303	5.303	5.303	6.883	5.303	5.303	5.303
16WW03					5.342	5.342	5.342	6.92	5.342	5.342	5.342
16WW04-A					5.318	5.318	5.318	6.896	5.318	5.318	5.318
16WW04					5.268	5.268	5.268	6.847	5.268	5.268	5.268
16WW04-A					5.298	5.298	5.298	6.877	5.298	5.298	5.298
16WW05					5.407	5.407	5.407	6.986	5.407	5.407	5.407
16WW05					5.234	5.234	5.234	6.812	5.234	5.234	5.234
16WW05					5.257	5.257	5.257	6.835	5.257	5.257	5.257
16WW05					5.281	5.281	5.281	6.859	5.281	5.281	5.281
16WW05					5.354	5.354	5.354	6.933	5.354	5.354	5.354
16WW05					5.354	5.354	5.354	6.933	5.354	5.354	5.354
16WW06					5.368	5.368	5.368	6.946	5.368	5.368	5.368
16WW06					5.342	5.342	5.342	6.92	5.342	5.342	5.342
16WW06					5.281	5.281	5.281	6.859	5.281	5.281	5.281
16WW06					5.268	5.268	5.268	6.847	5.268	5.268	5.268
16WW06					5.342	5.342	5.342	6.92	5.342	5.342	5.342
STATISTICS											
Total No.					26	26	26	26	26	26	26
Min.					187.5	187.5	187.5	909	187.5	187.5	187.5
Max.					284.5	284.5	284.5	1379.5	284.5	284.5	284.5
Avg.(log transformed)					5.326	5.326	5.326	6.905	5.326	5.326	5.326
St. Dev.(log transformed)					0.078	0.078	0.078	0.078	0.078	0.078	0.078
H											
95th UCL of Mean					206.24	206.24	206.24	1000.29	206.24	206.24	206.24

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	bis(2-Chloro ethyl) ether (ug/kg)		Bis(2-ethyl hexyl)phthalate (ug/kg)		Butyl benzyl phthalate (ug/kg)		Chrysene (ug/kg)		Dibenz(a,h) anthracene (ug/kg)		Dibenzofuran (ug/kg)		Diethyl phthalate (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	284.5	X+	284.5	X+	284.5	X+	284.5	X+	284.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+
16WW01-A				Avg.	194.25		194.25		194.25		194.25		194.25		194.25		194.25	
16WW01	5/4/93	0:00	5	Primary	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+
16WW01	5/4/93	0:00	10	Primary	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+
16WW02	5/12/93	0:00	0	Primary	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+
16WW02-A				Avg.	193		193		193		193		193		193		193	
16WW02	5/12/93	0:00	5	Primary	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+
16WW03	5/4/93	0:00	0	Primary	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+	206.5	X+
16WW03	5/4/93	0:00	5	Primary	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW03	5/4/93	0:00	10	Primary	201	X+	201	X+	201	X+	201	X+	201	X+	201	X+	201	X+
16WW03	5/4/93	0:00	15	Primary	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	199	X+	960		199	X+	199	X+	199	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+
16WW04-A				Avg.	204		204		584.5		204		204		204		204	
16WW04	5/12/93	0:00	5	Primary	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+
16WW04	5/12/93	0:00	10	Primary	199	X+	199	X+	199	X+	199	X+	199	X+	199	X+	199	X+
16WW04	5/12/93	0:00	10	Duplicate	201	X+	201	X+	201	X+	201	X+	201	X+	201	X+	201	X+
16WW04-A				Avg.	200		200		200		200		200		200		200	
16WW05	5/4/93	0:00	0	Primary	223	X+	223	X+	223	X+	223	X+	223	X+	223	X+	223	X+
16WW05	5/4/93	0:00	5	Primary	187.5	X+	187.5	X+	187.5	X+	187.5	X+	187.5	X+	187.5	X+	187.5	X+
16WW05	5/4/93	0:00	10	Primary	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+	192	X+
16WW05	5/4/93	0:00	15	Primary	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW05	5/4/93	0:00	20	Primary	211.5	X+	211.5	X+	211.5	X+	211.5	X+	211.5	X+	211.5	X+	211.5	X+
16WW05	5/4/93	0:00	25	Primary	211.5	X+	211.5	X+	211.5	X+	211.5	X+	211.5	X+	211.5	X+	211.5	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+	214.5	X+
16WW06	5/5/93	0:00	5	Primary	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+
16WW06	5/5/93	0:00	10	Primary	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW06	5/5/93	0:00	15	Primary	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+	194	X+
16WW06	5/5/93	0:00	20	Primary	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+	209	X+
NATURAL LOGS																		
16SD01					5.651		5.651		5.651		5.651		5.651		5.651		5.651	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	bis(2-Chloro ethyl) ether (ug/kg)	Bis(2-ethyl hexyl)phthalate (ug/kg)	Butyl benzyl phthalate (ug/kg)	Chrysene (ug/kg)	Dibenz(a,h) anthracene (ug/kg)	Dibenzofuran (ug/kg)	Diethyl phthalate (ug/kg)
16SD02					5.342	5.342	5.342	5.342	5.342	5.342	5.342
16SD03					5.368	5.368	5.368	5.368	5.368	5.368	5.368
16WW01-A					5.269	5.269	5.269	5.269	5.269	5.269	5.269
16WW01					5.316	5.316	5.316	5.316	5.316	5.316	5.316
16WW01					5.33	5.33	5.33	5.33	5.33	5.33	5.33
16WW02-A					5.263	5.263	5.263	5.263	5.263	5.263	5.263
16WW02					5.316	5.316	5.316	5.316	5.316	5.316	5.316
16WW03					5.33	5.33	5.33	5.33	5.33	5.33	5.33
16WW03					5.281	5.281	5.281	5.281	5.281	5.281	5.281
16WW03					5.303	5.303	5.303	5.303	5.303	5.303	5.303
16WW03					5.342	5.342	5.342	5.342	5.342	5.342	5.342
16WW04-A					5.318	5.318	6.371	5.318	5.318	5.318	5.318
16WW04					5.268	5.268	5.268	5.268	5.268	5.268	5.268
16WW04-A					5.298	5.298	5.298	5.298	5.298	5.298	5.298
16WW05					5.407	5.407	5.407	5.407	5.407	5.407	5.407
16WW05					5.234	5.234	5.234	5.234	5.234	5.234	5.234
16WW05					5.257	5.257	5.257	5.257	5.257	5.257	5.257
16WW05					5.281	5.281	5.281	5.281	5.281	5.281	5.281
16WW05					5.354	5.354	5.354	5.354	5.354	5.354	5.354
16WW05					5.354	5.354	5.354	5.354	5.354	5.354	5.354
16WW06					5.368	5.368	5.368	5.368	5.368	5.368	5.368
16WW06					5.342	5.342	5.342	5.342	5.342	5.342	5.342
16WW06					5.281	5.281	5.281	5.281	5.281	5.281	5.281
16WW06					5.268	5.268	5.268	5.268	5.268	5.268	5.268
16WW06					5.342	5.342	5.342	5.342	5.342	5.342	5.342
STATISTICS											
Total No.					26	26	26	26	26	26	26
Min.					187.5	187.5	187.5	187.5	187.5	187.5	187.5
Max.					284.5	284.5	960	284.5	284.5	284.5	284.5
Avg.(log transformed)					5.326	5.326	5.367	5.326	5.326	5.326	5.326
St. Dev.(log transformed)					0.078	0.078	0.219	0.078	0.078	0.078	0.078
H							1.764				
95th UCL of Mean					206.24	206.24	237.04	206.24	206.24	206.24	206.24

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Dimethyl phthalate (ug/kg)		Di-n-butyl phthalate (ug/kg)		Di-n-octyl phthalate (ug/kg)		Diphenylamine (ug/kg)		Fluoranthene (ug/kg)		Fluorene (ug/kg)		Hexachloro benzene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	284.5	X+	284.5	X+	862	X+	284.5	X+	284.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	209	X+	209	X+	633	X+	209	X+	209	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	649.5	X+	214.5	X+	214.5	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	196.5	X+	196.5	X+	595	X+	196.5	X+	196.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate	192	X+	192	X+	192	X+	581.5	X+	192	X+	192	X+	192	X+
16WW01-A				Avg.	194.25		194.25		194.25		588.25		194.25		194.25		194.25	
16WW01	5/4/93	0:00	5	Primary	203.5	X+	203.5	X+	203.5	X+	617.5	X+	203.5	X+	203.5	X+	203.5	X+
16WW01	5/4/93	0:00	10	Primary	206.5	X+	206.5	X+	206.5	X+	625	X+	206.5	X+	206.5	X+	206.5	X+
16WW02	5/12/93	0:00	0	Primary	192	X+	192	X+	192	X+	581.5	X+	192	X+	192	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate	194	X+	194	X+	194	X+	588	X+	194	X+	194	X+	194	X+
16WW02-A				Avg.	193		193		193		584.75		193		193		193	
16WW02	5/12/93	0:00	5	Primary	203.5	X+	203.5	X+	203.5	X+	617.5	X+	203.5	X+	203.5	X+	203.5	X+
16WW03	5/4/93	0:00	0	Primary	206.5	X+	206.5	X+	206.5	X+	625	X+	206.5	X+	206.5	X+	206.5	X+
16WW03	5/4/93	0:00	5	Primary	196.5	X+	196.5	X+	196.5	X+	595	X+	196.5	X+	196.5	X+	196.5	X+
16WW03	5/4/93	0:00	10	Primary	201	X+	201	X+	201	X+	610	X+	201	X+	201	X+	201	X+
16WW03	5/4/93	0:00	15	Primary	209	X+	209	X+	209	X+	633	X+	209	X+	209	X+	209	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	1600		199	X+	602.5	X+	199	X+	199	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate	209	X+	1900		209	X+	633	X+	209	X+	209	X+	209	X+
16WW04-A				Avg.	204		1750		204		617.75		204		204		204	
16WW04	5/12/93	0:00	5	Primary	194	X+	194	X+	194	X+	588	X+	194	X+	194	X+	194	X+
16WW04	5/12/93	0:00	10	Primary	199	X+	199	X+	199	X+	602.5	X+	199	X+	199	X+	199	X+
16WW04	5/12/93	0:00	10	Duplicate	201	X+	201	X+	201	X+	610	X+	201	X+	201	X+	201	X+
16WW04-A				Avg.	200		200		200		606.25		200		200		200	
16WW05	5/4/93	0:00	0	Primary	223	X+	223	X+	223	X+	675.5	X+	223	X+	223	X+	223	X+
16WW05	5/4/93	0:00	5	Primary	187.5	X+	900		187.5	X+	568	X+	187.5	X+	187.5	X+	187.5	X+
16WW05	5/4/93	0:00	10	Primary	192	X+	930		192	X+	581.5	X+	192	X+	192	X+	192	X+
16WW05	5/4/93	0:00	15	Primary	196.5	X+	196.5	X+	196.5	X+	595	X+	196.5	X+	196.5	X+	196.5	X+
16WW05	5/4/93	0:00	20	Primary	211.5	X+	211.5	X+	211.5	X+	641	X+	211.5	X+	211.5	X+	211.5	X+
16WW05	5/4/93	0:00	25	Primary	211.5	X+	211.5	X+	211.5	X+	641	X+	211.5	X+	211.5	X+	211.5	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	649.5	X+	214.5	X+	214.5	X+	214.5	X+
16WW06	5/5/93	0:00	5	Primary	209	X+	209	X+	209	X+	633	X+	209	X+	209	X+	209	X+
16WW06	5/5/93	0:00	10	Primary	196.5	X+	196.5	X+	196.5	X+	595	X+	196.5	X+	196.5	X+	196.5	X+
16WW06	5/5/93	0:00	15	Primary	194	X+	194	X+	194	X+	588	X+	194	X+	194	X+	194	X+
16WW06	5/5/93	0:00	20	Primary	209	X+	209	X+	209	X+	633	X+	209	X+	209	X+	209	X+
NATURAL LOGS																		
16SD01					5.651		5.651		5.651		6.759		5.651		5.651		5.651	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	RESULT DEPTH (ft TYPE	Dimethyl phthalate (ug/kg)	Di-n-butyl phthalate (ug/kg)	Di-n-octyl phthalate (ug/kg)	Diphenylamine (ug/kg)	Fluoranthene (ug/kg)	Fluorene (ug/kg)	Hexachloro benzene (ug/kg)
16SD02				5.342	5.342	5.342	6.45	5.342	5.342	5.342
16SD03				5.368	5.368	5.368	6.476	5.368	5.368	5.368
16WW01-A				5.269	5.269	5.269	6.377	5.269	5.269	5.269
16WW01				5.316	5.316	5.316	6.426	5.316	5.316	5.316
16WW01				5.33	5.33	5.33	6.438	5.33	5.33	5.33
16WW02-A				5.263	5.263	5.263	6.371	5.263	5.263	5.263
16WW02				5.316	5.316	5.316	6.426	5.316	5.316	5.316
16WW03				5.33	5.33	5.33	6.438	5.33	5.33	5.33
16WW03				5.281	5.281	5.281	6.389	5.281	5.281	5.281
16WW03				5.303	5.303	5.303	6.413	5.303	5.303	5.303
16WW03				5.342	5.342	5.342	6.45	5.342	5.342	5.342
16WW04-A				5.318	7.467	5.318	6.426	5.318	5.318	5.318
16WW04				5.268	5.268	5.268	6.377	5.268	5.268	5.268
16WW04-A				5.298	5.298	5.298	6.407	5.298	5.298	5.298
16WW05				5.407	5.407	5.407	6.515	5.407	5.407	5.407
16WW05				5.234	6.802	5.234	6.342	5.234	5.234	5.234
16WW05				5.257	6.835	5.257	6.366	5.257	5.257	5.257
16WW05				5.281	5.281	5.281	6.389	5.281	5.281	5.281
16WW05				5.354	5.354	5.354	6.463	5.354	5.354	5.354
16WW05				5.354	5.354	5.354	6.463	5.354	5.354	5.354
16WW06				5.368	5.368	5.368	6.476	5.368	5.368	5.368
16WW06				5.342	5.342	5.342	6.45	5.342	5.342	5.342
16WW06				5.281	5.281	5.281	6.389	5.281	5.281	5.281
16WW06				5.268	5.268	5.268	6.377	5.268	5.268	5.268
16WW06				5.342	5.342	5.342	6.45	5.342	5.342	5.342
STATISTICS										
Total No.				26	26	26	26	26	26	26
Min.				187.5	192	187.5	568	187.5	187.5	187.5
Max.				284.5	1900	284.5	862	284.5	284.5	284.5
Avg.(log transformed)				5.326	5.53	5.326	6.435	5.326	5.326	5.326
St. Dev.(log transformed)				0.078	0.569	0.078	0.078	0.078	0.078	0.078
H					2.014					
95th UCL of Mean				206.24	372.81	206.24	625.18	206.24	206.24	206.24

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Hexachloro-1,3-butadiene (ug/kg)		Hexachloro cyclopentadiene (ug/kg)		Hexachloro ethane (ug/kg)		1,2-Diphenyl hydrazine (ug/kg)		Indeno (1,2,3-cd) pyrene (ug/kg)		Isophorone (ug/kg)		3-Methylphenol (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	284.5	X+	284.5	X+	1379.5	X+	284.5	X+	284.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	209	X+	209	X+	1012.5	X+	209	X+	209	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate	192	X+	192	X+	192	X+	930	X+	192	X+	192	X+	192	X+
16WW01-A				Avg.	194.25		194.25		194.25		941.25		194.25		194.25		194.25	
16WW01	5/4/93	0:00	5	Primary	203.5	X+	203.5	X+	203.5	X+	987.5	X+	203.5	X+	203.5	X+	203.5	X+
16WW01	5/4/93	0:00	10	Primary	206.5	X+	206.5	X+	206.5	X+	1000	X+	206.5	X+	206.5	X+	206.5	X+
16WW02	5/12/93	0:00	0	Primary	192	X+	192	X+	192	X+	930	X+	192	X+	192	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate	194	X+	194	X+	194	X+	941	X+	194	X+	194	X+	194	X+
16WW02-A				Avg.	193		193		193		935.5		193		193		193	
16WW02	5/12/93	0:00	5	Primary	203.5	X+	203.5	X+	203.5	X+	987.5	X+	203.5	X+	203.5	X+	203.5	X+
16WW03	5/4/93	0:00	0	Primary	206.5	X+	206.5	X+	206.5	X+	1000	X+	206.5	X+	206.5	X+	206.5	X+
16WW03	5/4/93	0:00	5	Primary	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW03	5/4/93	0:00	10	Primary	201	X+	201	X+	201	X+	975.5	X+	201	X+	201	X+	201	X+
16WW03	5/4/93	0:00	15	Primary	209	X+	209	X+	209	X+	1012.5	X+	209	X+	209	X+	209	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	199	X+	199	X+	964	X+	199	X+	199	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate	209	X+	209	X+	209	X+	1012.5	X+	209	X+	209	X+	209	X+
16WW04-A				Avg.	204		204		204		988.25		204		204		204	
16WW04	5/12/93	0:00	5	Primary	194	X+	194	X+	194	X+	941	X+	194	X+	194	X+	194	X+
16WW04	5/12/93	0:00	10	Primary	199	X+	199	X+	199	X+	964	X+	199	X+	199	X+	199	X+
16WW04	5/12/93	0:00	10	Duplicate	201	X+	201	X+	201	X+	975.5	X+	201	X+	201	X+	201	X+
16WW04-A				Avg.	200		200		200		969.75		200		200		200	
16WW05	5/4/93	0:00	0	Primary	223	X+	223	X+	223	X+	1081	X+	223	X+	223	X+	223	X+
16WW05	5/4/93	0:00	5	Primary	187.5	X+	187.5	X+	187.5	X+	909	X+	187.5	X+	187.5	X+	187.5	X+
16WW05	5/4/93	0:00	10	Primary	192	X+	192	X+	192	X+	930	X+	192	X+	192	X+	192	X+
16WW05	5/4/93	0:00	15	Primary	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW05	5/4/93	0:00	20	Primary	211.5	X+	211.5	X+	211.5	X+	1025.5	X+	211.5	X+	211.5	X+	211.5	X+
16WW05	5/4/93	0:00	25	Primary	211.5	X+	211.5	X+	211.5	X+	1025.5	X+	211.5	X+	211.5	X+	211.5	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+
16WW06	5/5/93	0:00	5	Primary	209	X+	209	X+	209	X+	1012.5	X+	209	X+	209	X+	209	X+
16WW06	5/5/93	0:00	10	Primary	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+
16WW06	5/5/93	0:00	15	Primary	194	X+	194	X+	194	X+	941	X+	194	X+	194	X+	194	X+
16WW06	5/5/93	0:00	20	Primary	209	X+	209	X+	209	X+	1012.5	X+	209	X+	209	X+	209	X+
NATURAL LOGS																		
16SD01					5.651		5.651		5.651		7.229		5.651		5.651		5.651	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	RESULT DEPTH (ft TYPE	Hexachloro-1,3-butadiene (ug/kg)	Hexachloro cyclopentadiene (ug/kg)	Hexachloro ethane (ug/kg)	1,2-Diphenyl hydrazine (ug/kg)	Indeno (1,2,3-cd) pyrene (ug/kg)	Isophorone (ug/kg)	3-Methylphenol (ug/kg)
16SD02				5.342	5.342	5.342	6.92	5.342	5.342	5.342
16SD03				5.368	5.368	5.368	6.946	5.368	5.368	5.368
16WW01-A				5.269	5.269	5.269	6.847	5.269	5.269	5.269
16WW01				5.316	5.316	5.316	6.895	5.316	5.316	5.316
16WW01				5.33	5.33	5.33	6.908	5.33	5.33	5.33
16WW02-A				5.263	5.263	5.263	6.841	5.263	5.263	5.263
16WW02				5.316	5.316	5.316	6.895	5.316	5.316	5.316
16WW03				5.33	5.33	5.33	6.908	5.33	5.33	5.33
16WW03				5.281	5.281	5.281	6.859	5.281	5.281	5.281
16WW03				5.303	5.303	5.303	6.883	5.303	5.303	5.303
16WW03				5.342	5.342	5.342	6.92	5.342	5.342	5.342
16WW04-A				5.318	5.318	5.318	6.896	5.318	5.318	5.318
16WW04				5.268	5.268	5.268	6.847	5.268	5.268	5.268
16WW04-A				5.298	5.298	5.298	6.877	5.298	5.298	5.298
16WW05				5.407	5.407	5.407	6.986	5.407	5.407	5.407
16WW05				5.234	5.234	5.234	6.812	5.234	5.234	5.234
16WW05				5.257	5.257	5.257	6.835	5.257	5.257	5.257
16WW05				5.281	5.281	5.281	6.859	5.281	5.281	5.281
16WW05				5.354	5.354	5.354	6.933	5.354	5.354	5.354
16WW05				5.354	5.354	5.354	6.933	5.354	5.354	5.354
16WW06				5.368	5.368	5.368	6.946	5.368	5.368	5.368
16WW06				5.342	5.342	5.342	6.92	5.342	5.342	5.342
16WW06				5.281	5.281	5.281	6.859	5.281	5.281	5.281
16WW06				5.268	5.268	5.268	6.847	5.268	5.268	5.268
16WW06				5.342	5.342	5.342	6.92	5.342	5.342	5.342
STATISTICS										
Total No.				26	26	26	26	26	26	26
Min.				187.5	187.5	187.5	909	187.5	187.5	187.5
Max.				284.5	284.5	284.5	1379.5	284.5	284.5	284.5
Avg.(log transformed)				5.326	5.326	5.326	6.905	5.326	5.326	5.326
St. Dev.(log transformed)				0.078	0.078	0.078	0.078	0.078	0.078	0.078
H										
95th UCL of Mean				206.24	206.24	206.24	1000.29	206.24	206.24	206.24

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,3-Dichloro benzene (ug/kg)		3-Nitroaniline (ug/kg)		Naphthalene (ug/kg)		Nitrobenzene (ug/kg)		N-Nitroso diphenylamine (ug/kg)		N-Nitrosodi-n-propylamine (ug/kg)		2-Methylphenol (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	1379.5	X+	284.5	X+	284.5	X+	284.5	X+	1379.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate	192	X+	930	X+	192	X+	192	X+	192	X+	930	X+	192	X+
16WW01-A				Avg.	194.25		941.25		194.25		194.25		194.25		941.25		194.25	
16WW01	5/4/93	0:00	5	Primary	203.5	X+	987.5	X+	203.5	X+	203.5	X+	203.5	X+	987.5	X+	203.5	X+
16WW01	5/4/93	0:00	10	Primary	206.5	X+	1000	X+	206.5	X+	206.5	X+	206.5	X+	1000	X+	206.5	X+
16WW02	5/12/93	0:00	0	Primary	192	X+	930	X+	192	X+	192	X+	192	X+	930	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate	194	X+	941	X+	194	X+	194	X+	194	X+	941	X+	194	X+
16WW02-A				Avg.	193		935.5		193		193		193		935.5		193	
16WW02	5/12/93	0:00	5	Primary	203.5	X+	987.5	X+	203.5	X+	203.5	X+	203.5	X+	987.5	X+	203.5	X+
16WW03	5/4/93	0:00	0	Primary	206.5	X+	1000	X+	206.5	X+	206.5	X+	206.5	X+	1000	X+	206.5	X+
16WW03	5/4/93	0:00	5	Primary	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+
16WW03	5/4/93	0:00	10	Primary	201	X+	975.5	X+	201	X+	201	X+	201	X+	975.5	X+	201	X+
16WW03	5/4/93	0:00	15	Primary	209	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+	209	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	964	X+	199	X+	199	X+	199	X+	964	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate	209	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+	209	X+
16WW04-A				Avg.	204		988.25		204		204		204		988.25		204	
16WW04	5/12/93	0:00	5	Primary	194	X+	941	X+	194	X+	194	X+	194	X+	941	X+	194	X+
16WW04	5/12/93	0:00	10	Primary	199	X+	964	X+	199	X+	199	X+	199	X+	964	X+	199	X+
16WW04	5/12/93	0:00	10	Duplicate	201	X+	975.5	X+	201	X+	201	X+	201	X+	975.5	X+	201	X+
16WW04-A				Avg.	200		969.75		200		200		200		969.75		200	
16WW05	5/4/93	0:00	0	Primary	223	X+	1081	X+	223	X+	223	X+	223	X+	1081	X+	223	X+
16WW05	5/4/93	0:00	5	Primary	187.5	X+	909	X+	187.5	X+	187.5	X+	187.5	X+	909	X+	187.5	X+
16WW05	5/4/93	0:00	10	Primary	192	X+	930	X+	192	X+	192	X+	192	X+	930	X+	192	X+
16WW05	5/4/93	0:00	15	Primary	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+
16WW05	5/4/93	0:00	20	Primary	211.5	X+	1025.5	X+	211.5	X+	211.5	X+	211.5	X+	1025.5	X+	211.5	X+
16WW05	5/4/93	0:00	25	Primary	211.5	X+	1025.5	X+	211.5	X+	211.5	X+	211.5	X+	1025.5	X+	211.5	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+
16WW06	5/5/93	0:00	5	Primary	209	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+	209	X+
16WW06	5/5/93	0:00	10	Primary	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+
16WW06	5/5/93	0:00	15	Primary	194	X+	941	X+	194	X+	194	X+	194	X+	941	X+	194	X+
16WW06	5/5/93	0:00	20	Primary	209	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+	209	X+
NATURAL LOGS																		
16SD01					5.651		7.229		5.651		5.651		5.651		7.229		5.651	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,3-Dichloro benzene (ug/kg)	3-Nitroaniline (ug/kg)	Naphthalene (ug/kg)	Nitrobenzene (ug/kg)	N-Nitroso diphenylamine (ug/kg)	N-Nitrosodi-n-propylamine (ug/kg)	2-Methylphenol (ug/kg)
16SD02					5.342	6.92	5.342	5.342	5.342	6.92	5.342
16SD03					5.368	6.946	5.368	5.368	5.368	6.946	5.368
16WW01-A					5.269	6.847	5.269	5.269	5.269	6.847	5.269
16WW01					5.316	6.895	5.316	5.316	5.316	6.895	5.316
16WW01					5.33	6.908	5.33	5.33	5.33	6.908	5.33
16WW02-A					5.263	6.841	5.263	5.263	5.263	6.841	5.263
16WW02					5.316	6.895	5.316	5.316	5.316	6.895	5.316
16WW03					5.33	6.908	5.33	5.33	5.33	6.908	5.33
16WW03					5.281	6.859	5.281	5.281	5.281	6.859	5.281
16WW03					5.303	6.883	5.303	5.303	5.303	6.883	5.303
16WW03					5.342	6.92	5.342	5.342	5.342	6.92	5.342
16WW04-A					5.318	6.896	5.318	5.318	5.318	6.896	5.318
16WW04					5.268	6.847	5.268	5.268	5.268	6.847	5.268
16WW04-A					5.298	6.877	5.298	5.298	5.298	6.877	5.298
16WW05					5.407	6.986	5.407	5.407	5.407	6.986	5.407
16WW05					5.234	6.812	5.234	5.234	5.234	6.812	5.234
16WW05					5.257	6.835	5.257	5.257	5.257	6.835	5.257
16WW05					5.281	6.859	5.281	5.281	5.281	6.859	5.281
16WW05					5.354	6.933	5.354	5.354	5.354	6.933	5.354
16WW05					5.354	6.933	5.354	5.354	5.354	6.933	5.354
16WW06					5.368	6.946	5.368	5.368	5.368	6.946	5.368
16WW06					5.342	6.92	5.342	5.342	5.342	6.92	5.342
16WW06					5.281	6.859	5.281	5.281	5.281	6.859	5.281
16WW06					5.268	6.847	5.268	5.268	5.268	6.847	5.268
16WW06					5.342	6.92	5.342	5.342	5.342	6.92	5.342
STATISTICS											
Total No.					26	26	26	26	26	26	26
Min.					187.5	909	187.5	187.5	187.5	909	187.5
Max.					284.5	1379.5	284.5	284.5	284.5	1379.5	284.5
Avg.(log transformed)					5.326	6.905	5.326	5.326	5.326	6.905	5.326
St. Dev.(log transformed)					0.078	0.078	0.078	0.078	0.078	0.078	0.078
H											
95th UCL of Mean					206.24	1000.29	206.24	206.24	206.24	1000.29	206.24

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,2-Dichlorobenzene (ug/kg)		2-Nitroaniline (ug/kg)		2-Nitrophenol (ug/kg)		4-Chloroaniline (ug/kg)		4-Chloro-3-methylphenol (ug/kg)		Pentachlorophenol (ug/kg)		4-Methylphenol (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	1379.5	X+	284.5	X+	284.5	X+	284.5	X+	1379.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate	192	X+	930	X+	192	X+	192	X+	192	X+	930	X+	192	X+
16WW01-A				Avg.	194.25		941.25		194.25		194.25		194.25		941.25		194.25	
16WW01	5/4/93	0:00	5	Primary	203.5	X+	987.5	X+	203.5	X+	203.5	X+	203.5	X+	987.5	X+	203.5	X+
16WW01	5/4/93	0:00	10	Primary	206.5	X+	1000	X+	206.5	X+	206.5	X+	206.5	X+	1000	X+	206.5	X+
16WW02	5/12/93	0:00	0	Primary	192	X+	930	X+	192	X+	192	X+	192	X+	930	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate	194	X+	941	X+	194	X+	194	X+	194	X+	941	X+	194	X+
16WW02-A				Avg.	193		935.5		193		193		193		935.5		193	
16WW02	5/12/93	0:00	5	Primary	203.5	X+	987.5	X+	203.5	X+	203.5	X+	203.5	X+	987.5	X+	203.5	X+
16WW03	5/4/93	0:00	0	Primary	206.5	X+	1000	X+	206.5	X+	206.5	X+	206.5	X+	1000	X+	206.5	X+
16WW03	5/4/93	0:00	5	Primary	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+
16WW03	5/4/93	0:00	10	Primary	201	X+	975.5	X+	201	X+	201	X+	201	X+	975.5	X+	201	X+
16WW03	5/4/93	0:00	15	Primary	209	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+	209	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	964	X+	199	X+	199	X+	199	X+	964	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate	209	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+	209	X+
16WW04-A				Avg.	204		988.25		204		204		204		988.25		204	
16WW04	5/12/93	0:00	5	Primary	194	X+	941	X+	194	X+	194	X+	194	X+	941	X+	194	X+
16WW04	5/12/93	0:00	10	Primary	199	X+	964	X+	199	X+	199	X+	199	X+	964	X+	199	X+
16WW04	5/12/93	0:00	10	Duplicate	201	X+	975.5	X+	201	X+	201	X+	201	X+	975.5	X+	201	X+
16WW04-A				Avg.	200		969.75		200		200		200		969.75		200	
16WW05	5/4/93	0:00	0	Primary	223	X+	1081	X+	223	X+	223	X+	223	X+	1081	X+	223	X+
16WW05	5/4/93	0:00	5	Primary	187.5	X+	909	X+	187.5	X+	187.5	X+	187.5	X+	909	X+	187.5	X+
16WW05	5/4/93	0:00	10	Primary	192	X+	930	X+	192	X+	192	X+	192	X+	930	X+	192	X+
16WW05	5/4/93	0:00	15	Primary	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+
16WW05	5/4/93	0:00	20	Primary	211.5	X+	1025.5	X+	211.5	X+	211.5	X+	211.5	X+	1025.5	X+	211.5	X+
16WW05	5/4/93	0:00	25	Primary	211.5	X+	1025.5	X+	211.5	X+	211.5	X+	211.5	X+	1025.5	X+	211.5	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	1039	X+	214.5	X+	214.5	X+	214.5	X+	1039	X+	214.5	X+
16WW06	5/5/93	0:00	5	Primary	209	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+	209	X+
16WW06	5/5/93	0:00	10	Primary	196.5	X+	952.5	X+	196.5	X+	196.5	X+	196.5	X+	952.5	X+	196.5	X+
16WW06	5/5/93	0:00	15	Primary	194	X+	941	X+	194	X+	194	X+	194	X+	941	X+	194	X+
16WW06	5/5/93	0:00	20	Primary	209	X+	1012.5	X+	209	X+	209	X+	209	X+	1012.5	X+	209	X+
NATURAL LOGS																		
16SD01					5.651		7.229		5.651		5.651		5.651		7.229		5.651	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,2-Dichloro benzene (ug/kg)	2-Nitroaniline (ug/kg)	2-Nitrophenol (ug/kg)	4-Chloroaniline (ug/kg)	4-Chloro-3-methylphenol (ug/kg)	Pentachloro phenol (ug/kg)	4-Methylphenol (ug/kg)
16SD02					5.342	6.92	5.342	5.342	5.342	6.92	5.342
16SD03					5.368	6.946	5.368	5.368	5.368	6.946	5.368
16WW01-A					5.269	6.847	5.269	5.269	5.269	6.847	5.269
16WW01					5.316	6.895	5.316	5.316	5.316	6.895	5.316
16WW01					5.33	6.908	5.33	5.33	5.33	6.908	5.33
16WW02-A					5.263	6.841	5.263	5.263	5.263	6.841	5.263
16WW02					5.316	6.895	5.316	5.316	5.316	6.895	5.316
16WW03					5.33	6.908	5.33	5.33	5.33	6.908	5.33
16WW03					5.281	6.859	5.281	5.281	5.281	6.859	5.281
16WW03					5.303	6.883	5.303	5.303	5.303	6.883	5.303
16WW03					5.342	6.92	5.342	5.342	5.342	6.92	5.342
16WW04-A					5.318	6.896	5.318	5.318	5.318	6.896	5.318
16WW04					5.268	6.847	5.268	5.268	5.268	6.847	5.268
16WW04-A					5.298	6.877	5.298	5.298	5.298	6.877	5.298
16WW05					5.407	6.986	5.407	5.407	5.407	6.986	5.407
16WW05					5.234	6.812	5.234	5.234	5.234	6.812	5.234
16WW05					5.257	6.835	5.257	5.257	5.257	6.835	5.257
16WW05					5.281	6.859	5.281	5.281	5.281	6.859	5.281
16WW05					5.354	6.933	5.354	5.354	5.354	6.933	5.354
16WW05					5.354	6.933	5.354	5.354	5.354	6.933	5.354
16WW06					5.368	6.946	5.368	5.368	5.368	6.946	5.368
16WW06					5.342	6.92	5.342	5.342	5.342	6.92	5.342
16WW06					5.281	6.859	5.281	5.281	5.281	6.859	5.281
16WW06					5.268	6.847	5.268	5.268	5.268	6.847	5.268
16WW06					5.342	6.92	5.342	5.342	5.342	6.92	5.342
STATISTICS											
Total No.					26	26	26	26	26	26	26
Min.					187.5	909	187.5	187.5	187.5	909	187.5
Max.					284.5	1379.5	284.5	284.5	284.5	1379.5	284.5
Avg.(log transformed)					5.326	6.905	5.326	5.326	5.326	6.905	5.326
St. Dev.(log transformed)					0.078	0.078	0.078	0.078	0.078	0.078	0.078
H											
95th UCL of Mean					206.24	1000.29	206.24	206.24	206.24	1000.29	206.24

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,4-Dichloro benzene (ug/kg)		Phenanthrene (ug/kg)		Phenol (ug/kg)		4-Nitroaniline (ug/kg)		4-Nitrophenol (ug/kg)		Pyrene (ug/kg)	
16SD01	5/2/93	0:00	0	Primary	284.5	X+	284.5	X+	284.5	X+	1379.5	X+	1379.5	X+	284.5	X+
16SD02	4/14/93	0:00	0	Primary	209	X+	209	X+	209	X+	1012.5	X+	1012.5	X+	209	X+
16SD03	4/14/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	1039	X+	1039	X+	214.5	X+
16WW01	5/4/93	0:00	0	Primary	196.5	X+	196.5	X+	196.5	X+	952.5	X+	952.5	X+	196.5	X+
16WW01	5/4/93	0:00	0	Duplicate	192	X+	192	X+	192	X+	930	X+	930	X+	192	X+
16WW01-A				Avg.	194.25		194.25		194.25		941.25		941.25		194.25	
16WW01	5/4/93	0:00	5	Primary	203.5	X+	203.5	X+	203.5	X+	987.5	X+	987.5	X+	203.5	X+
16WW01	5/4/93	0:00	10	Primary	206.5	X+	206.5	X+	206.5	X+	1000	X+	1000	X+	206.5	X+
16WW02	5/12/93	0:00	0	Primary	192	X+	192	X+	192	X+	930	X+	930	X+	192	X+
16WW02	5/12/93	0:00	0	Duplicate	194	X+	194	X+	194	X+	941	X+	941	X+	194	X+
16WW02-A				Avg.	193		193		193		935.5		935.5		193	
16WW02	5/12/93	0:00	5	Primary	203.5	X+	203.5	X+	203.5	X+	987.5	X+	987.5	X+	203.5	X+
16WW03	5/4/93	0:00	0	Primary	206.5	X+	206.5	X+	206.5	X+	1000	X+	1000	X+	206.5	X+
16WW03	5/4/93	0:00	5	Primary	196.5	X+	196.5	X+	196.5	X+	952.5	X+	952.5	X+	196.5	X+
16WW03	5/4/93	0:00	10	Primary	201	X+	201	X+	201	X+	975.5	X+	975.5	X+	201	X+
16WW03	5/4/93	0:00	15	Primary	209	X+	209	X+	209	X+	1012.5	X+	1012.5	X+	209	X+
16WW04	5/12/93	0:00	0	Primary	199	X+	199	X+	199	X+	964	X+	964	X+	199	X+
16WW04	5/12/93	0:00	0	Duplicate	209	X+	209	X+	209	X+	1012.5	X+	1012.5	X+	209	X+
16WW04-A				Avg.	204		204		204		988.25		988.25		204	
16WW04	5/12/93	0:00	5	Primary	194	X+	194	X+	194	X+	941	X+	941	X+	194	X+
16WW04	5/12/93	0:00	10	Primary	199	X+	199	X+	199	X+	964	X+	964	X+	199	X+
16WW04	5/12/93	0:00	10	Duplicate	201	X+	201	X+	201	X+	975.5	X+	975.5	X+	201	X+
16WW04-A				Avg.	200		200		200		969.75		969.75		200	
16WW05	5/4/93	0:00	0	Primary	223	X+	223	X+	223	X+	1081	X+	1081	X+	223	X+
16WW05	5/4/93	0:00	5	Primary	187.5	X+	187.5	X+	187.5	X+	909	X+	909	X+	187.5	X+
16WW05	5/4/93	0:00	10	Primary	192	X+	192	X+	192	X+	930	X+	930	X+	192	X+
16WW05	5/4/93	0:00	15	Primary	196.5	X+	196.5	X+	196.5	X+	952.5	X+	952.5	X+	196.5	X+
16WW05	5/4/93	0:00	20	Primary	211.5	X+	211.5	X+	211.5	X+	1025.5	X+	1025.5	X+	211.5	X+
16WW05	5/4/93	0:00	25	Primary	211.5	X+	211.5	X+	211.5	X+	1025.5	X+	1025.5	X+	211.5	X+
16WW06	5/5/93	0:00	0	Primary	214.5	X+	214.5	X+	214.5	X+	1039	X+	1039	X+	214.5	X+
16WW06	5/5/93	0:00	5	Primary	209	X+	209	X+	209	X+	1012.5	X+	1012.5	X+	209	X+
16WW06	5/5/93	0:00	10	Primary	196.5	X+	196.5	X+	196.5	X+	952.5	X+	952.5	X+	196.5	X+
16WW06	5/5/93	0:00	15	Primary	194	X+	194	X+	194	X+	941	X+	941	X+	194	X+
16WW06	5/5/93	0:00	20	Primary	209	X+	209	X+	209	X+	1012.5	X+	1012.5	X+	209	X+
NATURAL LOGS																
16SD01					5.651		5.651		5.651		7.229		7.229		5.651	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Soil Results for
Semi-Volatile Organic Compounds (SVOCs)
(all soil depths)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,4-Dichloro benzene (ug/kg)	Phenanthrene (ug/kg)	Phenol (ug/kg)	4-Nitroaniline (ug/kg)	4-Nitrophenol (ug/kg)	Pyrene (ug/kg)
16SD02					5.342	5.342	5.342	6.92	6.92	5.342
16SD03					5.368	5.368	5.368	6.946	6.946	5.368
16WW01-A					5.269	5.269	5.269	6.847	6.847	5.269
16WW01					5.316	5.316	5.316	6.895	6.895	5.316
16WW01					5.33	5.33	5.33	6.908	6.908	5.33
16WW02-A					5.263	5.263	5.263	6.841	6.841	5.263
16WW02					5.316	5.316	5.316	6.895	6.895	5.316
16WW03					5.33	5.33	5.33	6.908	6.908	5.33
16WW03					5.281	5.281	5.281	6.859	6.859	5.281
16WW03					5.303	5.303	5.303	6.883	6.883	5.303
16WW03					5.342	5.342	5.342	6.92	6.92	5.342
16WW04-A					5.318	5.318	5.318	6.896	6.896	5.318
16WW04					5.268	5.268	5.268	6.847	6.847	5.268
16WW04-A					5.298	5.298	5.298	6.877	6.877	5.298
16WW05					5.407	5.407	5.407	6.986	6.986	5.407
16WW05					5.234	5.234	5.234	6.812	6.812	5.234
16WW05					5.257	5.257	5.257	6.835	6.835	5.257
16WW05					5.281	5.281	5.281	6.859	6.859	5.281
16WW05					5.354	5.354	5.354	6.933	6.933	5.354
16WW05					5.354	5.354	5.354	6.933	6.933	5.354
16WW06					5.368	5.368	5.368	6.946	6.946	5.368
16WW06					5.342	5.342	5.342	6.92	6.92	5.342
16WW06					5.281	5.281	5.281	6.859	6.859	5.281
16WW06					5.268	5.268	5.268	6.847	6.847	5.268
16WW06					5.342	5.342	5.342	6.92	6.92	5.342
STATISTICS										
Total No.					26	26	26	26	26	26
Min.					187.5	187.5	187.5	909	909	187.5
Max.					284.5	284.5	284.5	1379.5	1379.5	284.5
Avg.(log transformed)					5.326	5.326	5.326	6.905	6.905	5.326
St. Dev.(log transformed)					0.078	0.078	0.078	0.078	0.078	0.078
H										
95th UCL of Mean					206.24	206.24	206.24	1000.29	1000.29	206.24

Data Qualifier Notations

X+ = Non-Detect Result, one-half of the detection limit value presented.
U+ = Considered Non-Detect based on blank comparison, one-half of the detection limit value is presented.
J = Estimated laboratory result.

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Metals

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Aluminum (mg/kg)	Antimony (mg/kg)		Arsenic (mg/kg)		Barium (mg/kg)		Beryllium (mg/kg)	Cadmium (mg/kg)		Calcium (mg/kg)	Chromium (mg/kg)
16SD04	4/14/93	0:00	0	Primary		0.615	X+U	4.88		224			0.615	X+		20.2
16SD04	2/21/95	8:55	0	Primary		0.625	X+R	8.53		57.5			0.315	X+		21.8
16SD05	5/2/93	0:00	0	Primary		0.835	X+	0.835	X+	91.3	J		0.835	X+		15.2
16SD05	5/2/93	0:00	0	Duplicate 1		0.845	X+	0.845	X+	220			0.845	X+		28.3
16SD05-A				Avg.		0.84		0.84		155.65			0.84			21.75
16SD12	2/21/95	9:50	0	Primary		0.62	X+R	2.23		58.8			0.31	X+		6.33
16SD13	2/21/95	10:05	0	Primary		0.68	X+R	3.26		182.1			0.34	X+		8.42
16SD14	2/21/95	14:50	0	Primary		0.755	X+R	2.72		139			0.38	X+		8.76
16SD14	2/21/95	14:50	0	Duplicate 1		0.75	X+	2.84		103.9			0.375	X+		8.68
16SD14-A				Avg.		0.7525		2.78		121.45			0.3775			8.72
16SD15	3/16/95	15:15	0	Primary		0.72	X+R	3.75	J	236.7			0.36	X+U+J		20.8
16SD16	3/16/95	16:45	0	Primary		0.685	X+R	1.91	J	150.3			0.34	X+U+J		13.2
16SD17	3/6/95	14:45	0	Primary		0.71	X+R	1.57	J	117.6			0.355	X+		6.88
16SD17	3/6/95	14:45	0	Duplicate 1		0.58	X+	1.28		82.9			0.29	X+		4.25
16SD17-A				Avg.		0.645		1.425		100.25			0.3225			5.565
16SD18	3/6/95	11:00	0	Primary		0.67	X+R	2.28	J	145.3			0.335	X+		9.13
16SD19	3/6/95	10:00	0	Primary		0.82	X+R	2.3	J	266.1			0.41	X+		17.8
16SD20	3/6/95	12:45	0	Primary		0.735	X+R	3.24	J	278			0.37	X+		12.5
16SD21	5/10/95	14:55	0	Primary		0.65	X+	6.9		194			0.335	X+		12
NATURAL LOGS																
16SD04						-0.486		1.585		5.412			-0.486			3.006
16SD04						-0.47		2.144		4.052			-1.155			3.082
16SD05-A						-0.174		-0.174		5.048			-0.174			3.08
16SD12						-0.478		0.802		4.074			-1.171			1.845
16SD13						-0.386		1.182		5.205			-1.079			2.131
16SD14-A						-0.284		1.022		4.8			-0.974			2.166
16SD15						-0.329		1.322		5.467			-1.022			3.035
16SD16						-0.378		0.647		5.013			-1.079			2.58
16SD17-A						-0.439		0.354		4.608			-1.132			1.716
16SD18						-0.4		0.824		4.979			-1.094			2.212
16SD19						-0.198		0.833		5.584			-0.892			2.879
16SD20						-0.308		1.176		5.628			-0.994			2.526
16SD21						-0.431		1.932		5.268			-1.094			2.485
STATISTICS																
Total No.						13		13		13		0	13		0	13

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Metals

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Aluminum (mg/kg)	Antimony (mg/kg)	Arsenic (mg/kg)	Barium (mg/kg)	Beryllium (mg/kg)	Cadmium (mg/kg)	Calcium (mg/kg)	Chromium (mg/kg)
Min.						0.58	0.835	57.5	0	0.29	0	4.25
Max.						0.845	8.53	278	0	0.845	0	28.3
Avg.(log transformed)						-0.366	1.05	5.011	#DIV/0!	-0.95	#DIV/0!	2.519
St. Dev.(log transformed)						0.102	0.624	0.515	#DIV/0!	0.293	#DIV/0!	0.478
H							2.268	2.13				2.088
95th UCL of Mean						0.69712	5.223827	235.162	#DIV/0!	0.403703	#DIV/0!	18.56649

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Metals

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Cobalt (mg/kg)	Copper (mg/kg)	Iron (mg/kg)	Lead (mg/kg)		Magnesium (mg/kg)	Manganese (mg/kg)	Mercury (mg/kg)		Nickel (mg/kg)	
16SD04	4/14/93	0:00	0	Primary				17.7				0.06	X+	14.4	
16SD04	2/21/95	8:55	0	Primary		5.8		12.5				0.065	X+	9.54	
16SD05	5/2/93	0:00	0	Primary				31.2				0.085	X+	10.7	J
16SD05	5/2/93	0:00	0	Duplicate 1				26.1				0.085	X+	24	
16SD05-A				Avg.				28.65				0.085		17.35	
16SD12	2/21/95	9:50	0	Primary		4.7		6.3				0.06	X+	4.7	
16SD13	2/21/95	10:05	0	Primary		5.8		10.9				0.07	X+	10.1	
16SD14	2/21/95	14:50	0	Primary		4.8		11.6				0.075	X+	10.1	
16SD14	2/21/95	14:50	0	Duplicate 1		4.5		10.02994				0.075	X+	7.19	
16SD14-A				Avg.		4.65		10.81497				0.075		8.645	
16SD15	3/16/95	15:15	0	Primary		8.1		26.7	J			0.07	X+	17.3	
16SD16	3/16/95	16:45	0	Primary		11.9		14.3	J			0.07	X+	11.5	
16SD17	3/6/95	14:45	0	Primary		3.8		7.83	J			0.07	X+	6.95	
16SD17	3/6/95	14:45	0	Duplicate 1		4.4		6.04				0.06	X+	5.52	
16SD17-A				Avg.		4.1		6.935				0.065		6.235	
16SD18	3/6/95	11:00	0	Primary		3.2		8.98				0.065	X+	4.97	
16SD19	3/6/95	10:00	0	Primary		8.2		14.8				0.08	X+	12.7	
16SD20	3/6/95	12:45	0	Primary		8.6		13.3				0.075	X+	11.1	
16SD21	5/10/95	14:55	0	Primary		5.3		19.3				0.065	X+	9.8	
NATURAL LOGS															
16SD04								2.874				-2.813		2.667	
16SD04						1.758		2.526				-2.733		2.255	
16SD05-A								3.355				-2.465		2.854	
16SD12						1.548		1.841				-2.813		1.548	
16SD13						1.758		2.389				-2.659		2.313	
16SD14-A						1.537		2.381				-2.59		2.157	
16SD15						2.092		3.285				-2.659		2.851	
16SD16						2.477		2.66				-2.659		2.442	
16SD17-A						1.411		1.937				-2.733		1.83	
16SD18						1.163		2.195				-2.733		1.603	
16SD19						2.104		2.695				-2.526		2.542	
16SD20						2.152		2.588				-2.59		2.407	
16SD21						1.668		2.96				-2.733		2.282	
STATISTICS															
Total No.					0	11	0	13.		0	0	13		13	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Metals

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Cobalt (mg/kg)	Copper (mg/kg)	Iron (mg/kg)	Lead (mg/kg)	Magnesium (mg/kg)	Manganese (mg/kg)	Mercury (mg/kg)	Nickel (mg/kg)
Min.					0	3.2	0	6.04	0	0	0.06	4.7
Max.					0	11.9	0	31.2	0	0	0.085	24
Avg.(log transformed)					#DIV/0!	1.788	#DIV/0!	2.591	#DIV/0!	#DIV/0!	-2.67	2.289
St. Dev.(log transformed)					#DIV/0!	0.383	#DIV/0!	0.459	#DIV/0!	#DIV/0!	0.105	0.421
H						2.036		2.067				2.026
95th UCL of Mean					#DIV/0!	8.231227	#DIV/0!	19.49625	#DIV/0!	#DIV/0!	0.069635	13.78864

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Metals

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Potassium (mg/kg)	Selenium (mg/kg)		Silver (mg/kg)		Sodium (mg/kg)	Strontium (mg/kg)	Thallium (mg/kg)		Vanadium (mg/kg)	Zinc (mg/kg)
16SD04	4/14/93	0:00	0	Primary		1.95	J	0.615	X+			0.615	X+		
16SD04	2/21/95	8:55	0	Primary		0.625	X+	0.625	X+			0.315	X+		39
16SD05	5/2/93	0:00	0	Primary		0.835	X+U	0.835	X+			1.83			
16SD05	5/2/93	0:00	0	Duplicate 1		0.845	X+	0.845	X+			0.845	X+		
16SD05-A				Avg.		0.84		0.84				1.3375			
16SD12	2/21/95	9:50	0	Primary		0.31	X+	0.62	X+			0.31	X+		16
16SD13	2/21/95	10:05	0	Primary		0.34	X+	0.68	X+			0.34	X+		24
16SD14	2/21/95	14:50	0	Primary		0.755	X+	0.755	X+			0.38	X+		25.1
16SD14	2/21/95	14:50	0	Duplicate 1		0.375	X+	0.75	X+			0.375	X+		21.4
16SD14-A				Avg.		0.565		0.7525				0.3775			23.25
16SD15	3/16/95	15:15	0	Primary		0.87		0.72	X+			0.36	X+		51.1
16SD16	3/16/95	16:45	0	Primary		0.82		0.685	X+			0.34	X+		33.9
16SD17	3/6/95	14:45	0	Primary		0.355	X+U	0.71	X+			0.355	X+		19.7
16SD17	3/6/95	14:45	0	Duplicate 1		0.7		0.58	X+			0.29	X+		15
16SD17-A				Avg.		0.5275		0.645				0.3225			17.35
16SD18	3/6/95	11:00	0	Primary		0.8	J	0.67	X+			0.335	X+		18.2
16SD19	3/6/95	10:00	0	Primary		1.15	J	0.82	X+			0.41	X+		46
16SD20	3/6/95	12:45	0	Primary		1.33	J	0.735	X+			0.37	X+		32
16SD21	5/10/95	14:55	0	Primary		0.335	X+	0.65	X+			0.65	X+		24.2
NATURAL LOGS															
16SD04						0.668		-0.486				-0.486			
16SD04						-0.47		-0.47				-1.155			3.664
16SD05-A						-0.174		-0.174				0.291			
16SD12						-1.171		-0.478				-1.171			2.773
16SD13						-1.079		-0.386				-1.079			3.178
16SD14-A						-0.571		-0.284				-0.974			3.146
16SD15						-0.139		-0.329				-1.022			3.934
16SD16						-0.198		-0.378				-1.079			3.523
16SD17-A						-0.64		-0.439				-1.132			2.854
16SD18						-0.223		-0.4				-1.094			2.901
16SD19						0.14		-0.198				-0.892			3.829
16SD20						0.285		-0.308				-0.994			3.466
16SD21						-1.094		-0.431				-0.431			3.186
STATISTICS															
Total No.					0	13		13		0	0	13		0	11

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Metals

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Potassium (mg/kg)	Selenium (mg/kg)	Silver (mg/kg)	Sodium (mg/kg)	Strontium (mg/kg)	Thallium (mg/kg)	Vanadium (mg/kg)	Zinc (mg/kg)
Min.					0	0.31	0.58	0	0	0.29	0	15
Max.					0	1.95	0.845	0	0	1.83	0	51.1
Avg.(log transformed)					#DIV/0!	-0.359	-0.366	#DIV/0!	#DIV/0!	-0.863	#DIV/0!	3.314
St. Dev.(log transformed)					#DIV/0!	0.555	0.102	#DIV/0!	#DIV/0!	0.42	#DIV/0!	0.397
H						2.18				2.025		2.051
95th UCL of Mean					#DIV/0!	1.155208	0.69712	#DIV/0!	#DIV/0!	0.589028	#DIV/0!	38.4859

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Explosives

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	2,4-Dinitro toluene (mg/kg)		2,6-Dinitro toluene (mg/kg)		2-Amino-4,6-dinitrotoluene (mg/kg)		4-Amino-2,6-dinitrotoluene (mg/kg)		HMX (mg/kg)		1,3-Dinitro benzene (mg/kg)		3-Nitrotoluene (mg/kg)		Nitrobenzene (mg/kg)	
16SD04	2/21/95	8:55	0	Primary	0.25	X+	0.25	X+	0.45	X+	0.55	X+	0.45	X+	0.25	X+	0.45	X+	0.3	X+
16SD05	5/2/93	0:00	0	Primary	0.215	X+	0.22	X+	0.215	X+	0.215	X+	1.85	X+	0.215	X+	0.215	X+	0.22	X+
16SD05	5/2/93	0:00	0	Duplicate 1	0.22	X+	0.23	X+	0.22	X+	0.22	X+	1.95	X+	0.22	X+	0.22	X+	0.23	X+
16SD05-A				Avg.	0.2175		0.225		0.2175		0.2175		1.9		0.2175		0.2175		0.225	
16SD12	2/21/95	9:50	0	Primary	0.25	X+	0.25	X+	0.45	X+	0.55	X+	0.45	X+	0.25	X+	0.45	X+	0.3	X+
16SD13	2/21/95	10:05	0	Primary	0.25	X+	0.25	X+	0.45	X+	0.55	X+	0.45	X+	0.25	X+	0.45	X+	0.3	X+
16SD14	2/21/95	14:50	0	Primary	0.25	X+	0.25	X+	0.45	X+	0.55	X+	0.45	X+	0.25	X+	0.45	X+	0.3	X+
16SD14	2/21/95	14:50	0	Duplicate 1	0.25	X+	0.25	X+	0.45	X+	0.55	X+	0.45	X+	0.25	X+	0.45	X+	0.3	X+
16SD14-A				Avg.	0.25		0.25		0.45		0.55		0.45		0.25		0.45		0.3	
16SD15	3/16/95	15:15	0	Primary	0.25	X+	0.25	X+	0.45	X+	0.55	X+	0.45	X+	0.25	X+	0.45	X+	0.3	X+
16SD16	3/16/95	16:45	0	Primary	0.25	X+	0.25	X+	0.45	X+	0.55	X+	0.45	X+	0.25	X+	0.45	X+	0.3	X+
16SD17	3/6/95	14:45	0	Primary	0.25	X+	0.25	X+	0.45	X+	0.55	X+	0.45	X+	0.25	X+	0.45	X+	0.3	X+
16SD17	3/6/95	14:45	0	Duplicate 1	0.25	X+	0.25	X+	0.45	X+	0.55	X+	0.45	X+	0.25	X+	0.45	X+	0.3	X+
16SD17-A				Avg.	0.25		0.25		0.45		0.55		0.45		0.25		0.45		0.3	
16SD18	3/6/95	11:00	0	Primary	0.25	X+	0.25	X+	0.45	X+	0.55	X+	0.45	X+	0.25	X+	0.45	X+	0.3	X+
16SD19	3/6/95	10:00	0	Primary	0.25	X+	0.25	X+	0.45	X+	0.55	X+	0.45	X+	0.25	X+	0.45	X+	0.3	X+
16SD20	3/6/95	12:45	0	Primary	0.25	X+	0.25	X+	0.45	X+	0.55	X+	0.45	X+	0.25	X+	0.45	X+	0.3	X+
16SD21	5/10/95	14:55	0	Primary	0.25	X+	0.25	X+	0.45	X+	0.55	X+	0.45	X+	0.25	X+	0.45	X+	0.3	X+
NATURAL LOGS																				
16SD04					-1.386		-1.386		-0.799		-0.598		-0.799		-1.386		-0.799		-1.204	
16SD05-A					-1.526		-1.492		-1.526		-1.526		0.642		-1.526		-1.526		-1.492	
16SD12					-1.386		-1.386		-0.799		-0.598		-0.799		-1.386		-0.799		-1.204	
16SD13					-1.386		-1.386		-0.799		-0.598		-0.799		-1.386		-0.799		-1.204	
16SD14-A					-1.386		-1.386		-0.799		-0.598		-0.799		-1.386		-0.799		-1.204	
16SD15					-1.386		-1.386		-0.799		-0.598		-0.799		-1.386		-0.799		-1.204	
16SD16					-1.386		-1.386		-0.799		-0.598		-0.799		-1.386		-0.799		-1.204	
16SD17-A					-1.386		-1.386		-0.799		-0.598		-0.799		-1.386		-0.799		-1.204	
16SD18					-1.386		-1.386		-0.799		-0.598		-0.799		-1.386		-0.799		-1.204	
16SD19					-1.386		-1.386		-0.799		-0.598		-0.799		-1.386		-0.799		-1.204	
16SD20					-1.386		-1.386		-0.799		-0.598		-0.799		-1.386		-0.799		-1.204	
16SD21					-1.386		-1.386		-0.799		-0.598		-0.799		-1.386		-0.799		-1.204	
STATISTICS																				
Total No.					12		12		12		12		12		12		12		12	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Explosives

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	2,4-Dinitro toluene (mg/kg)	2,6-Dinitro toluene (mg/kg)	2-Amino-4,6-dinitrotoluene (mg/kg)	4-Amino-2,6-dinitrotoluene (mg/kg)	HMX (mg/kg)	1,3-Dinitro benzene (mg/kg)	3-Nitrotoluene (mg/kg)	Nitrobenzene (mg/kg)
Min.					0.215	0.22	0.215	0.215	0.45	0.215	0.215	0.22
Max.					0.25	0.25	0.45	0.55	1.95	0.25	0.45	0.3
Avg.(log transformed)					-1.398	-1.395	-0.86	-0.675	-0.679	-1.398	-0.86	-1.228
St. Dev.(log transformed)					0.04	0.031	0.21	0.268	0.416	0.04	0.21	0.083
H												
95th UCL of Mean					0.247288	0.247952	0.432596	0.527774	0.552959	0.247288	0.432596	0.293888

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Explosives

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	2-Nitro toluene (mg/kg)		4-Nitro toluene (mg/kg)		RDX (mg/kg)		1,3,5-Trinitro benzene (mg/kg)		Tetryl (mg/kg)		Trinitrotoluene 2,4,6- (TNT) (mg/kg)	
16SD04	2/21/95	8:55	0	Primary	0.45	X+	0.55	X+	0.25	X+	0.35	X+	0.95	X+	0.25	X+
16SD05	5/2/93	0:00	0	Primary	0.215	X+	0.215	X+	0.85	X+	0.215	X+	0.55	X+	0.215	X+U+J
16SD05	5/2/93	0:00	0	Duplicate 1	0.22	X+	0.22	X+	0.9	X+	0.22	X+	0.55	X+	0.22	X+
16SD05-A				Avg.	0.2175		0.2175		0.875		0.2175		0.55		0.2175	
16SD12	2/21/95	9:50	0	Primary	0.45	X+	0.55	X+	0.25	X+	0.35	X+	0.95	X+	0.25	X+
16SD13	2/21/95	10:05	0	Primary	0.45	X+	0.55	X+	0.25	X+	0.35	X+	0.95	X+	0.25	X+
16SD14	2/21/95	14:50	0	Primary	0.45	X+	0.55	X+	0.25	X+	0.35	X+	0.95	X+	0.25	X+
16SD14	2/21/95	14:50	0	Duplicate 1	0.45	X+	0.55	X+	0.25	X+	0.35	X+	0.95	X+	0.25	X+
16SD14-A				Avg.	0.45		0.55		0.25		0.35		0.95		0.25	
16SD15	3/16/95	15:15	0	Primary	0.45	X+	0.55	X+	0.25	X+	0.35	X+	0.95	X+	0.25	X+
16SD16	3/16/95	16:45	0	Primary	0.45	X+	0.55	X+	0.25	X+	0.35	X+	0.95	X+	0.25	X+
16SD17	3/6/95	14:45	0	Primary	0.45	X+	0.55	X+	0.25	X+	0.35	X+	0.95	X+	0.25	X+
16SD17	3/6/95	14:45	0	Duplicate 1	0.45	X+	0.55	X+	0.25	X+	0.35	X+	0.95	X+	0.25	X+
16SD17-A				Avg.	0.45		0.55		0.25		0.35		0.95		0.25	
16SD18	3/6/95	11:00	0	Primary	0.45	X+	0.55	X+	0.25	X+	0.35	X+	0.95	X+	0.25	X+
16SD19	3/6/95	10:00	0	Primary	0.45	X+	0.55	X+	0.25	X+	0.35	X+	0.95	X+	0.25	X+
16SD20	3/6/95	12:45	0	Primary	0.45	X+	0.55	X+	0.25	X+	0.35	X+	0.95	X+	0.25	X+
16SD21	5/10/95	14:55	0	Primary	0.45	X+	0.55	X+	0.25	X+	0.35	X+	0.95	X+	0.25	X+

NATURAL LOGS

16SD04					-0.799		-0.598		-1.386		-1.05		-0.051		-1.386	
16SD05-A					-1.526		-1.526		-0.134		-1.526		-0.598		-1.526	
16SD12					-0.799		-0.598		-1.386		-1.05		-0.051		-1.386	
16SD13					-0.799		-0.598		-1.386		-1.05		-0.051		-1.386	
16SD14-A					-0.799		-0.598		-1.386		-1.05		-0.051		-1.386	
16SD15					-0.799		-0.598		-1.386		-1.05		-0.051		-1.386	
16SD16					-0.799		-0.598		-1.386		-1.05		-0.051		-1.386	
16SD17-A					-0.799		-0.598		-1.386		-1.05		-0.051		-1.386	
16SD18					-0.799		-0.598		-1.386		-1.05		-0.051		-1.386	
16SD19					-0.799		-0.598		-1.386		-1.05		-0.051		-1.386	
16SD20					-0.799		-0.598		-1.386		-1.05		-0.051		-1.386	
16SD21					-0.799		-0.598		-1.386		-1.05		-0.051		-1.386	

STATISTICS

Total No.					**12**		12		12		12		12		12	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Explosives

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	2-Nitro toluene (mg/kg)	4-Nitro toluene (mg/kg)	RDX (mg/kg)	1,3,5-Trinitro benzene (mg/kg)	Tetryl (mg/kg)	Trinitrotoluene 2,4,6- (TNT) (mg/kg)
Min.					0.215	0.215	0.25	0.215	0.55	0.215
Max.					0.45	0.55	0.9	0.35	0.95	0.25
Avg.(log transformed)					-0.86	-0.675	-1.282	-1.09	-0.097	-1.398
St. Dev.(log transformed)					0.21	0.268	0.361	0.137	0.158	0.04
H										
95th UCL of Mean					0.432596	0.527774	0.296165	0.339387	0.918955	0.247288

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	1,2,4-Trichloro benzene (ug/kg)		1-Chloro naphthalene (ug/kg)		2,4,5-Trichloro phenol (ug/kg)		2,4,6-Trichloro phenol (ug/kg)		2,4-Dichloro phenol (ug/kg)		2,4-Dimethyl phenol (ug/kg)		2,4-Dinitro phenol (ug/kg)	
16SD04	4/14/93	0:00	0	Primary	203.5	X+	407.5	X+	987.5	X+	203.5	X+	203.5	X+	203.5	X+	987.5	X+
16SD05	5/2/93	0:00	0	Primary	275	X+	550	X+	1333.5	X+	275	X+	275	X+	275	X+	1333.5	X+
16SD05	5/2/93	0:00	0	Duplicate 1	279.5	X+	559.5	X+	1356	X+	279.5	X+	279.5	X+	279.5	X+	1356	X+

Note: all results are ND

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	2,4-Dinitro toluene (ug/kg)		2,6-Dinitro toluene (ug/kg)		2-Chloro napthalene (ug/kg)		2-Chlorophenol (ug/kg)		2-Methyl napthalene (ug/kg)		3,3'-Dichloro benzidine (ug/kg)		4,6-Dinitro-2-methylphenol (ug/kg)	
16SD04	4/14/93	0:00	0	Primary	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+	407.5	X+	987.5	X+
16SD05	5/2/93	0:00	0	Primary	275	X+	275	X+	275	X+	275	X+	275	X+	550	X+	1333.5	X+
16SD05	5/2/93	0:00	0	Duplicate 1	279.5	X+	279.5	X+	279.5	X+	279.5	X+	279.5	X+	559.5	X+	1356	X+

Note: all results are ND

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	4-Bromophenyl phenyl ether (ug/kg)		4-Chlorophenyl phenyl ether (ug/kg)		Acenaphthene (ug/kg)		Acenaphthylene (ug/kg)		Anthracene (ug/kg)		Benzo(a) anthracene (ug/kg)		Benzo(a)pyrene (ug/kg)	
16SD04	4/14/93	0:00	0	Primary	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+
16SD05	5/2/93	0:00	0	Primary	275	X+	275	X+	275	X+	275	X+	275	X+	275	X+	275	X+
16SD05	5/2/93	0:00	0	Duplicate 1	279.5	X+	279.5	X+	279.5	X+	279.5	X+	279.5	X+	279.5	X+	279.5	X+

Note: all results are ND

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	Benzo(b) fluoranthene (ug/kg)		Benzo(g,h,i) perylene (ug/kg)		Benzo(k)fluor anthene (ug/kg)		Benzoic acid (ug/kg)		Benzyl alcohol (ug/kg)		Bis(2-chloro-1-methylethyl) ether (ug/kg)		Bis(2-chloro ethoxy) methan (ug/kg)	
16SD04	4/14/93	0:00	0	Primary	203.5	X+	203.5	X+	203.5	X+	987.5	X+	203.5	X+	203.5	X+	203.5	X+
16SD05	5/2/93	0:00	0	Primary	275	X+	275	X+	275	X+	1333.5	X+	275	X+	275	X+	275	X+
16SD05	5/2/93	0:00	0	Duplicate 1	279.5	X+	279.5	X+	279.5	X+	1356	X+	279.5	X+	279.5	X+	279.5	X+

Note: all results are ND

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	bis(2-Chloro ethyl) ether (ug/kg)		Bis(2-ethyl hexyl)phthalate (ug/kg)		Butyl benzyl phthalate (ug/kg)		Chrysene (ug/kg)		Dibenz(a,h) anthracene (ug/kg)		Dibenzofuran (ug/kg)		Diethyl phthalate (ug/kg)	
16SD04	4/14/93	0:00	0	Primary	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+	203.5	X+
16SD05	5/2/93	0:00	0	Primary	275	X+	275	X+	275	X+	275	X+	275	X+	275	X+	275	X+
16SD05	5/2/93	0:00	0	Duplicate 1	279.5	X+	279.5	X+	279.5	X+	279.5	X+	279.5	X+	279.5	X+	279.5	X+

Note: all results are ND

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	Dimethyl phthalate (ug/kg)		Di-n-butyl phthalate (ug/kg)		Di-n-octyl phthalate (ug/kg)		Diphenylamine (ug/kg)		Fluoranthene (ug/kg)		Fluorene (ug/kg)		Hexachloro benzene (ug/kg)	
16SD04	4/14/93	0:00	0	Primary	203.5	X+	203.5	X+	203.5	X+	617.5	X+	203.5	X+	203.5	X+	203.5	X+
16SD05	5/2/93	0:00	0	Primary	275	X+	275	X+	275	X+	833.5	X+	275	X+	275	X+	275	X+
16SD05	5/2/93	0:00	0	Duplicate 1	279.5	X+	279.5	X+	279.5	X+	847.5	X+	279.5	X+	279.5	X+	279.5	X+

Note: all results are ND

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	Hexachloro-1,3-butadiene (ug/kg)		Hexachloro cyclopentadiene (ug/kg)		Hexachloro ethane (ug/kg)		1,2-Diphenyl hydrazine (ug/kg)		Indeno (1,2,3-cd) pyrene (ug/kg)		Isophorone (ug/kg)		3-Methylphenol (ug/kg)	
16SD04	4/14/93	0:00	0	Primary	203.5	X+	203.5	X+	203.5	X+	987.5	X+	203.5	X+	203.5	X+	203.5	X+
16SD05	5/2/93	0:00	0	Primary	275	X+	275	X+	275	X+	1333.5	X+	275	X+	275	X+	275	X+
16SD05	5/2/93	0:00	0	Duplicate 1	279.5	X+	279.5	X+	279.5	X+	1356	X+	279.5	X+	279.5	X+	279.5	X+

Note: all results are ND

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	1,3-Dichloro benzene (ug/kg)		3-Nitroaniline (ug/kg)		Naphthalene (ug/kg)		Nitrobenzene (ug/kg)		N-Nitroso diphenylamine (ug/kg)		N-Nitrosodi-n-propylamine (ug/kg)		2-Methylphenol (ug/kg)	
16SD04	4/14/93	0:00	0	Primary	203.5	X+	987.5	X+	203.5	X+	203.5	X+	203.5	X+	987.5	X+	203.5	X+
16SD05	5/2/93	0:00	0	Primary	275	X+	1333.5	X+	275	X+	275	X+	275	X+	1333.5	X+	275	X+
16SD05	5/2/93	0:00	0	Duplicate 1	279.5	X+	1356	X+	279.5	X+	279.5	X+	279.5	X+	1356	X+	279.5	X+

Note: all results are ND

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	1,2-Dichloro benzene (ug/kg)		2-Nitroaniline (ug/kg)		2-Nitrophenol (ug/kg)		4-Chloroaniline (ug/kg)		4-Chloro-3-methylphenol (ug/kg)		Pentachloro phenol (ug/kg)		4-Methylphenol (ug/kg)	
16SD04	4/14/93	0:00	0	Primary	203.5	X+	987.5	X+	203.5	X+	203.5	X+	203.5	X+	987.5	X+	203.5	X+
16SD05	5/2/93	0:00	0	Primary	275	X+	1333.5	X+	275	X+	275	X+	275	X+	1333.5	X+	275	X+
16SD05	5/2/93	0:00	0	Duplicate 1	279.5	X+	1356	X+	279.5	X+	279.5	X+	279.5	X+	1356	X+	279.5	X+

Note: all results are ND

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	DEPTH (ft)	RESULT TYPE	1,4-Dichloro benzene (ug/kg)		Phenanthrene (ug/kg)		Phenol (ug/kg)		4-Nitroaniline (ug/kg)		4-Nitrophenol (ug/kg)		Pyrene (ug/kg)	
16SD04	4/14/93	0:00	0	Primary	203.5	X+	203.5	X+	203.5	X+	987.5	X+	987.5	X+	203.5	X+
16SD05	5/2/93	0:00	0	Primary	275	X+	275	X+	275	X+	1333.5	X+	1333.5	X+	275	X+
16SD05	5/2/93	0:00	0	Duplicate 1	279.5	X+	279.5	X+	279.5	X+	1356	X+	1356	X+	279.5	X+

Note: all results are ND

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,1,1,2-Tetra chloroethane (ug/kg)	1,1,1-trichloro ethane (ug/kg)		1,1,2,2-Tetra chloroethane (ug/kg)		1,1,2-Trichloro ethane (ug/kg)		1,1-Dichloro ethane (ug/kg)		1,1-Dichloro ethene (ug/kg)		1,1-Dichloro propene (ug/kg)	1,2,3-Trichloro benzene (ug/kg)
16SD04	4/14/93	0:00	0	Primary		3.35	X+	3.35	X+	3.35	X+	3.35	X+	3.35	X+		
16SD04	2/21/95	8:55	0	Primary		3	X+	3	X+	3	X+	3	X+	3	X+		
16SD05	5/2/93	0:00	0	Primary		4.15	X+	4.15	X+	4.15	X+	4.15	X+	4.15	X+		
16SD05	5/2/93	0:00	0	Duplicate 1		4.25	X+	4.25	X+	4.25	X+	4.25	X+	4.25	X+		
16SD05-A				Avg.		4.2		4.2		4.2		4.2		4.2			
16SD12	2/21/95	9:50	0	Primary		3	X+	3	X+	3	X+	3	X+	3	X+		
16SD13	2/21/95	10:05	0	Primary		3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD14	2/21/95	14:50	0	Primary		4	X+	4	X+	4	X+	4	X+	4	X+		
16SD14	2/21/95	14:50	0	Duplicate 1		3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD14-A				Avg.		3.75		3.75		3.75		3.75		3.75			
16SD15	3/16/95	0:00	0	Primary		3.5	X+U	3.5	X+U	3.5	X+U	3.5	X+U	3.5	X+U+J		
16SD16	3/16/95	16:45	0	Primary		3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD17	3/6/95	14:45	0	Primary		3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD17	3/6/95	14:45	0	Duplicate 1		3	X+	3	X+	3	X+	3	X+	3	X+		
16SD17-A				Avg.		3.25		3.25		3.25		3.25		3.25			
16SD18	3/6/95	11:00	0	Primary		3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD19	3/6/95	10:00	0	Primary		4	X+	4	X+	4	X+	4	X+	4	X+		
16SD20	3/6/95	12:45	0	Primary		3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD21	5/10/95	14:55	0	Primary		3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
NATURAL LOGS																	
16SD04						1.209		1.209		1.209		1.209		1.209			
16SD04						1.099		1.099		1.099		1.099		1.099			
16SD05-A						1.435		1.435		1.435		1.435		1.435			
16SD12						1.099		1.099		1.099		1.099		1.099			
16SD13						1.253		1.253		1.253		1.253		1.253			
16SD14-A						1.322		1.322		1.322		1.322		1.322			
16SD15						1.253		1.253		1.253		1.253		1.253			
16SD16						1.253		1.253		1.253		1.253		1.253			
16SD17-A						1.179		1.179		1.179		1.179		1.179			
16SD18						1.253		1.253		1.253		1.253		1.253			
16SD19						1.386		1.386		1.386		1.386		1.386			
16SD20						1.253		1.253		1.253		1.253		1.253			
16SD21						1.253		1.253		1.253		1.253		1.253			
STATISTICS																	
Total No.						13		13		13		13		13		0	0

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,1,1,2-Tetra chloroethane (ug/kg)	1,1,1-trichloro ethane (ug/kg)	1,1,2,2-Tetra chloroethane (ug/kg)	1,1,2-Trichloro ethane (ug/kg)	1,1-Dichloro ethane (ug/kg)	1,1-Dichloro ethene (ug/kg)	1,1-Dichloro propene (ug/kg)	1,2,3-Trichloro benzene (ug/kg)
Min.						3	3	3	3	3	0	0
Max.						4.25	4.25	4.25	4.25	4.25	0	0
Avg.(log transformed)						1.25	1.25	1.25	1.25	1.25	#DIV/0!	#DIV/0!
St. Dev (log transformed)						0.096	0.096	0.096	0.096	0.096	#DIV/0!	#DIV/0!
H												
95th UCL of Mean						3.51	3.51	3.51	3.51	3.51	#DIV/0!	#DIV/0!

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,2,3-Trichloro propane (ug/kg)		1,2,4-Trichloro benzene (ug/kg)	1,2-Dichloro ethane (ug/kg)		DCE12TOT (ug/kg)		1,2-Dichloro propane (ug/kg)		1,3-Dichloro propane (ug/kg)	1,4-Dichloro-2-butene (ug/kg)		1,4-Dioxane (ug/kg)
16SD04	4/14/93	0:00	0	Primary	3.35	X+		3.35	X+			3.35	X+		6.5	X+	
16SD04	2/21/95	8:55	0	Primary	3	X+		3	X+	3	X+	3	X+		6.5	X+	
16SD05	5/2/93	0:00	0	Primary	4.15	X+		4.15	X+			4.15	X+		8.5	X+	
16SD05	5/2/93	0:00	0	Duplicate 1	4.25	X+		4.25	X+			4.25	X+		8.5	X+	
16SD05-A				Avg.	4.2			4.2				4.2			8.5		
16SD12	2/21/95	9:50	0	Primary	3	X+		3	X+	3	X+	3	X+		6	X+	
16SD13	2/21/95	10:05	0	Primary	3.5	X+		3.5	X+	3.5	X+	3.5	X+		7	X+	
16SD14	2/21/95	14:50	0	Primary	4	X+		4	X+	4	X+	4	X+		7.5	X+	
16SD14	2/21/95	14:50	0	Duplicate 1	3.5	X+		3.5	X+	3.5	X+	3.5	X+		7.5	X+	
16SD14-A				Avg.	3.75			3.75		3.75		3.75			7.5		
16SD15	3/16/95	0:00	0	Primary	3.5	X+U+J		3.5	X+U	3.5	X+U	3.5	X+U+J		7	X+U+J	
16SD16	3/16/95	16:45	0	Primary	3.5	X+		3.5	X+	3.5	X+	3.5	X+		7	X+	
16SD17	3/6/95	14:45	0	Primary	3.5	X+		3.5	X+	3.5	X+	3.5	X+		7	X+	
16SD17	3/6/95	14:45	0	Duplicate 1	3	X+		3	X+	3	X+	3	X+		6	X+	
16SD17-A				Avg.	3.25			3.25		3.25		3.25			6.5		
16SD18	3/6/95	11:00	0	Primary	3.5	X+		3.5	X+	3.5	X+	3.5	X+		6.5	X+	
16SD19	3/6/95	10:00	0	Primary	4	X+		4	X+	4	X+	4	X+		8	X+	
16SD20	3/6/95	12:45	0	Primary	3.5	X+		3.5	X+	3.5	X+	3.5	X+		7.5	X+	
16SD21	5/10/95	14:55	0	Primary	3.5	X+		3.5	X+	3.5	X+	3.5	X+		6.5	X+	
NATURAL LOGS																	
16SD04					1.209			1.209				1.209			1.872		
16SD04					1.099			1.099		1.099		1.099			1.872		
16SD05-A					1.435			1.435				1.435			2.14		
16SD12					1.099			1.099		1.099		1.099			1.792		
16SD13					1.253			1.253		1.253		1.253			1.946		
16SD14-A					1.322			1.322		1.322		1.322			2.015		
16SD15					1.253			1.253		1.253		1.253			1.946		
16SD16					1.253			1.253		1.253		1.253			1.946		
16SD17-A					1.179			1.179		1.179		1.179			1.872		
16SD18					1.253			1.253		1.253		1.253			1.872		
16SD19					1.386			1.386		1.386		1.386			2.079		
16SD20					1.253			1.253		1.253		1.253			2.015		
16SD21					1.253			1.253		1.253		1.253			1.872		
STATISTICS																	
Total No.					13		0	13		11		13		0	13		0

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	2,2-Dichloro propane (ug/kg)	2-Hexanone (ug/kg)		Acetone (ug/kg)		Acrolein (ug/kg)		Acrylonitrile (ug/kg)		Benzene (ug/kg)		1,2,4-Trimethyl benzene (ug/kg)	1,3,5-Trimethyl benzene (ug/kg)
16SD04	4/14/93	0:00	0	Primary		6.5	X+	6.5	X+	6.5	X+	6.5	X+	3.35	X+		
16SD04	2/21/95	8:55	0	Primary		6.5	X+	6.5	X+	31.5	X+	31.5	X+	3	X+		
16SD05	5/2/93	0:00	0	Primary		8.5	X+	8.5	X+	8.5	X+	8.5	X+	4.15	X+		
16SD05	5/2/93	0:00	0	Duplicate 1		8.5	X+	170		8.5	X+	8.5	X+	4.25	X+		
16SD05-A				Avg.		8.5		89.25		8.5		8.5		4.2			
16SD12	2/21/95	9:50	0	Primary		6	X+	6	X+	31	X+	31	X+	3	X+		
16SD13	2/21/95	10:05	0	Primary		7	X+	7	X+	34	X+	34	X+	3.5	X+		
16SD14	2/21/95	14:50	0	Primary		7.5	X+	7.5	X+	38	X+	38	X+	4	X+		
16SD14	2/21/95	14:50	0	Duplicate 1		7.5	X+	7.5	X+	37.5	X+	37.5	X+	3.5	X+		
16SD14-A				Avg.		7.5		7.5		37.75		37.75		3.75			
16SD15	3/16/95	0:00	0	Primary		7	X+U	12	J	35.5	X+U	35.5	X+U	3.5	X+U+J		
16SD16	3/16/95	16:45	0	Primary		7	X+	7	X+	34	X+	34	X+	3.5	X+		
16SD17	3/6/95	14:45	0	Primary		7	X+	7	X+	35.5	X+	35.5	X+	3.5	X+		
16SD17	3/6/95	14:45	0	Duplicate 1		6	X+	6	X+	29	X+	29	X+	3	X+		
16SD17-A				Avg.		6.5		6.5		32.25		32.25		3.25			
16SD18	3/6/95	11:00	0	Primary		6.5	X+	17.5	U+	33.5	X+	33.5	X+	3.5	X+		
16SD19	3/6/95	10:00	0	Primary		8	X+	79		41	X+	41	X+	4	X+		
16SD20	3/6/95	12:45	0	Primary		7.5	X+	41	U+	37	X+	37	X+	3.5	X+		
16SD21	5/10/95	14:55	0	Primary		6.5	X+	13		33.5	X+	33.5	X+	3.5	X+		
NATURAL LOGS																	
16SD04						1.872		1.872		1.872		1.872		1.209			
16SD04						1.872		1.872		3.45		3.45		1.099			
16SD05-A						2.14		4.491		2.14		2.14		1.435			
16SD12						1.792		1.792		3.434		3.434		1.099			
16SD13						1.946		1.946		3.526		3.526		1.253			
16SD14-A						2.015		2.015		3.631		3.631		1.322			
16SD15						1.946		2.485		3.57		3.57		1.253			
16SD16						1.946		1.946		3.526		3.526		1.253			
16SD17-A						1.872		1.872		3.474		3.474		1.179			
16SD18						1.872		2.862		3.512		3.512		1.253			
16SD19						2.079		4.369		3.714		3.714		1.386			
16SD20						2.015		3.714		3.611		3.611		1.253			
16SD21						1.872		2.565		3.512		3.512		1.253			
STATISTICS																	
Total No.					0	13		13		13		13		13		0	0

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	2,2-Dichloro propane (ug/kg)	2-Hexanone (ug/kg)	Acetone (ug/kg)	Acrolein (ug/kg)	Acrylonitrile (ug/kg)	Benzene (ug/kg)	1,2,4-Trimethyl benzene (ug/kg)	1,3,5-Trimethyl benzene (ug/kg)
Min.					0	6	6	6.5	6.5	3	0	0
Max.					0	8.5	170	41	41	4.25	0	0
Avg.(log transformed)					#DIV/0!	1.941	2.6	3.306	3.306	1.25	#DIV/0!	#DIV/0!
St. Dev (log transformed)					#DIV/0!	0.098	0.978	0.584	0.584	0.096	#DIV/0!	#DIV/0!
H							2.811					
95th UCL of Mean					#DIV/0!	7.00	48.03	32.35	32.35	3.51	#DIV/0!	#DIV/0!

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Isopropyl benzene (ug/kg)		Bromobenzene (ug/kg)		Bromodichloro methane (ug/kg)		Bromoform (ug/kg)		Carbon disulfide (ug/kg)		Carbon tetrachloride (ug/kg)		Chlorobenzene (ug/kg)		Bromochloro methane (ug/kg)	
16SD04	4/14/93	0:00	0	Primary					3.35	X+			3.35	X+	3.35	X+	3.35	X+		
16SD04	2/21/95	8:55	0	Primary					3	X+	3	X+	3	X+	3	X+	3	X+		
16SD05	5/2/93	0:00	0	Primary					4.15	X+			4.15	X+	4.15	X+	4.15	X+		
16SD05	5/2/93	0:00	0	Duplicate 1					4.25	X+			4.25	X+	4.25	X+	4.25	X+		
16SD05-A				Avg.					4.2				4.2		4.2		4.2			
16SD12	2/21/95	9:50	0	Primary					3	X+	3	X+	3	X+	3	X+	3	X+		
16SD13	2/21/95	10:05	0	Primary					3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD14	2/21/95	14:50	0	Primary					4	X+	4	X+	4	X+	4	X+	4	X+		
16SD14	2/21/95	14:50	0	Duplicate 1					3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD14-A				Avg.					3.75		3.75		3.75		3.75		3.75			
16SD15	3/16/95	0:00	0	Primary					3.5	X+U	3.5	X+U	3.5	X+U	3.5	X+U	3.5	X+U+J		
16SD16	3/16/95	16:45	0	Primary					3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD17	3/6/95	14:45	0	Primary					3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD17	3/6/95	14:45	0	Duplicate 1					3	X+	3	X+	3	X+	3	X+	3	X+		
16SD17-A				Avg.					3.25		3.25		3.25		3.25		3.25			
16SD18	3/6/95	11:00	0	Primary					3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD19	3/6/95	10:00	0	Primary					4	X+	4	X+	4	X+	4	X+	4	X+		
16SD20	3/6/95	12:45	0	Primary					3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
16SD21	5/10/95	14:55	0	Primary					3.5	X+	3.5	X+	3.5	X+	3.5	X+	3.5	X+		
NATURAL LOGS																				
16SD04									1.209				1.209		1.209		1.209			
16SD04									1.099		1.099		1.099		1.099		1.099			
16SD05-A									1.435				1.435		1.435		1.435			
16SD12									1.099		1.099		1.099		1.099		1.099			
16SD13									1.253		1.253		1.253		1.253		1.253			
16SD14-A									1.322		1.322		1.322		1.322		1.322			
16SD15									1.253		1.253		1.253		1.253		1.253			
16SD16									1.253		1.253		1.253		1.253		1.253			
16SD17-A									1.179		1.179		1.179		1.179		1.179			
16SD18									1.253		1.253		1.253		1.253		1.253			
16SD19									1.386		1.386		1.386		1.386		1.386			
16SD20									1.253		1.253		1.253		1.253		1.253			
16SD21									1.253		1.253		1.253		1.253		1.253			
STATISTICS																				
Total No.						0		0	13		11		13		13		13		0	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Isopropyl benzene (ug/kg)	Bromobenzene (ug/kg)	Bromodichloro methane (ug/kg)	Bromoform (ug/kg)	Carbon disulfide (ug/kg)	Carbon tetrachloride (ug/kg)	Chlorobenzene (ug/kg)	Bromochloro methane (ug/kg)
Min.					0	0	3	3	3	3	3	0
Max.					0	0	4.25	4	4.25	4.25	4.25	0
Avg.(log transformed)					#DIV/0!	#DIV/0!	1.25	1.237	1.25	1.25	1.25	#DIV/0!
St. Dev (log transformed)					#DIV/0!	#DIV/0!	0.096	0.085	0.096	0.096	0.096	#DIV/0!
H												
95th UCL of Mean					#DIV/0!	#DIV/0!	3.51	3.46	3.51	3.51	3.51	#DIV/0!

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Chloroethane (ug/kg)		Chloroform (ug/kg)		cis-1,2-Dichloroethene (ug/kg)		cis-1,3-Dichloropropene (ug/kg)		1,2-Dibromo-3-chloropropane (ug/kg)		Dibromochloro methane (ug/kg)		Dichloro difluoromethane (ug/kg)		1,2-Dibromoeth (ug/kg)	
16SD04	4/14/93	0:00	0	Primary	6.5	X+	3.35	X+	3.35	X+					3.35	X+	3.35	X+		
16SD04	2/21/95	8:55	0	Primary	6.5	X+	3	X+			3	X+			3	X+	3	X+		
16SD05	5/2/93	0:00	0	Primary	8.5	X+	4.15	X+	4.15	X+					4.15	X+	4.15	X+		
16SD05	5/2/93	0:00	0	Duplicate 1	8.5	X+	4.25	X+	4.25	X+					4.25	X+	4.25	X+		
16SD05-A				Avg.	8.5		4.2		4.2						4.2		4.2			
16SD12	2/21/95	9:50	0	Primary	6	X+	3	X+			3	X+			3	X+	3	X+		
16SD13	2/21/95	10:05	0	Primary	7	X+	3.5	X+			3.5	X+			3.5	X+	3.5	X+		
16SD14	2/21/95	14:50	0	Primary	7.5	X+	4	X+			4	X+			4	X+	4	X+		
16SD14	2/21/95	14:50	0	Duplicate 1	7.5	X+	3.5	X+			3.5	X+			3.5	X+	3.5	X+		
16SD14-A				Avg.	7.5		3.75				3.75				3.75		3.75			
16SD15	3/16/95	0:00	0	Primary	7	X+U	3.5	X+U+J			3.5	X+U+J			3.5	X+U	3.5	X+U+J		
16SD16	3/16/95	16:45	0	Primary	7	X+	3.5	X+			3.5	X+			3.5	X+	3.5	X+		
16SD17	3/6/95	14:45	0	Primary	7	X+	3.5	X+			3.5	X+			3.5	X+	3.5	X+		
16SD17	3/6/95	14:45	0	Duplicate 1	6	X+	3	X+			3	X+			3	X+	3	X+		
16SD17-A				Avg.	6.5		3.25				3.25				3.25		3.25			
16SD18	3/6/95	11:00	0	Primary	6.5	X+	3.5	X+			3.5	X+			3.5	X+	3.5	X+		
16SD19	3/6/95	10:00	0	Primary	8	X+	4	X+			4	X+			4	X+	4	X+		
16SD20	3/6/95	12:45	0	Primary	7.5	X+	3.5	X+			3.5	X+			3.5	X+	3.5	X+		
16SD21	5/10/95	14:55	0	Primary	6.5	X+	3.5	X+			3.5	X+			3.5	X+	3.5	X+		
NATURAL LOGS																				
16SD04					1.872		1.209		1.209						1.209		1.209			
16SD04					1.872		1.099				1.099				1.099		1.099			
16SD05-A					2.14		1.435		1.435						1.435		1.435			
16SD12					1.792		1.099				1.099				1.099		1.099			
16SD13					1.946		1.253				1.253				1.253		1.253			
16SD14-A					2.015		1.322				1.322				1.322		1.322			
16SD15					1.946		1.253				1.253				1.253		1.253			
16SD16					1.946		1.253				1.253				1.253		1.253			
16SD17-A					1.872		1.179				1.179				1.179		1.179			
16SD18					1.872		1.253				1.253				1.253		1.253			
16SD19					2.079		1.386				1.386				1.386		1.386			
16SD20					2.015		1.253				1.253				1.253		1.253			
16SD21					1.872		1.253				1.253				1.253		1.253			
STATISTICS																				
Total No.					13		13		2		11		0		13		13		0	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Chloroethane (ug/kg)	Chloroform (ug/kg)	cis-1,2-Dichloroethene (ug/kg)	cis-1,3-Dichloropropene (ug/kg)	1,2-Dibromo-3-chloropropane (ug/kg)	Dibromochloromethane (ug/kg)	Dichlorodifluoromethane (ug/kg)	1,2-Dibromoeth (ug/kg)
Min.					6	3	3.35	3	0	3	3	0
Max.					8.5	4.25	4.25	4	0	4.25	4.25	0
Avg.(log transformed)					1.941	1.25	1.322	1.237	#DIV/0!	1.25	1.25	#DIV/0!
St. Dev (log transformed)					0.098	0.096	0.16	0.085	#DIV/0!	0.096	0.096	#DIV/0!
H												
95th UCL of Mean					7.00	3.51	3.80	3.46	#DIV/0!	3.51	3.51	#DIV/0!

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	CEVETH (ug/kg)		trans-1,2-Dichloroethene (ug/kg)		Propionitrile (ug/kg)	Ethyl methacrylate (ug/kg)		Ethylbenzene (ug/kg)		Hexachloro butadiene (ug/kg)	Isobutyl alcohol (ug/kg)	1,3-Dichloro benzene (ug/kg)
16SD04	4/14/93	0:00	0	Primary			3.35	X+		3.35	X+	3.35	X+			
16SD04	2/21/95	8:55	0	Primary	6.5	X+				3	X+	3	X+			
16SD05	5/2/93	0:00	0	Primary			4.15	X+		4.15	X+	4.15	X+			
16SD05	5/2/93	0:00	0	Duplicate 1			4.25	X+		4.25	X+	4.25	X+			
16SD05-A				Avg.			4.2			4.2		4.2				
16SD12	2/21/95	9:50	0	Primary	6	X+				3	X+	3	X+			
16SD13	2/21/95	10:05	0	Primary	7	X+				3.5	X+	3.5	X+			
16SD14	2/21/95	14:50	0	Primary	7.5	X+				4	X+	4	X+			
16SD14	2/21/95	14:50	0	Duplicate 1	7.5	X+				3.5	X+	3.5	X+			
16SD14-A				Avg.	7.5					3.75		3.75				
16SD15	3/16/95	0:00	0	Primary	7	X+U+J				3.5	X+U	3.5	X+U+J			
16SD16	3/16/95	16:45	0	Primary	7	X+				3.5	X+	3.5	X+			
16SD17	3/6/95	14:45	0	Primary	7	X+				3.5	X+	3.5	X+			
16SD17	3/6/95	14:45	0	Duplicate 1	6	X+				3	X+	3	X+			
16SD17-A				Avg.	6.5					3.25		3.25				
16SD18	3/6/95	11:00	0	Primary	6.5	X+				3.5	X+	3.5	X+			
16SD19	3/6/95	10:00	0	Primary	8	X+				4	X+	4	X+			
16SD20	3/6/95	12:45	0	Primary	7.5	X+				3.5	X+	3.5	X+			
16SD21	5/10/95	14:55	0	Primary	6.5	X+				3.5	X+	3.5	X+			
NATURAL LOGS																
16SD04							1.209			1.209		1.209				
16SD04					1.872					1.099		1.099				
16SD05-A							1.435			1.435		1.435				
16SD12					1.792					1.099		1.099				
16SD13					1.946					1.253		1.253				
16SD14-A					2.015					1.322		1.322				
16SD15					1.946					1.253		1.253				
16SD16					1.946					1.253		1.253				
16SD17-A					1.872					1.179		1.179				
16SD18					1.872					1.253		1.253				
16SD19					2.079					1.386		1.386				
16SD20					2.015					1.253		1.253				
16SD21					1.872					1.253		1.253				
STATISTICS																
Total No.					11		2		0	13		13		0	0	0

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	CEVETH (ug/kg)	trans-1,2-Dichloroethene (ug/kg)	Propionitrile (ug/kg)	Ethyl methacrylate (ug/kg)	Ethylbenzene (ug/kg)	Hexachloro butadiene (ug/kg)	Isobutyl alcohol (ug/kg)	1,3-Dichloro benzene (ug/kg)
Min.					6	3.35	0	3	3	0	0	0
Max.					8	4.25	0	4.25	4.25	0	0	0
Avg.(log transformed)					1.93	1.322	#DIV/0!	1.25	1.25	#DIV/0!	#DIV/0!	#DIV/0!
St. Dev (log transformed)					0.084	0.16	#DIV/0!	0.096	0.096	#DIV/0!	#DIV/0!	#DIV/0!
H												
95th UCL of Mean					6.91	3.80	#DIV/0!	3.51	3.51	#DIV/0!	#DIV/0!	#DIV/0!

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Methacrylonitrile (ug/kg)	Bromomethane (ug/kg)		Chloromethane (ug/kg)		2-Butanone (ug/kg)		Iodomethane (ug/kg)		4-Methyl-2-pentanone (ug/kg)		Methyl methacrylate (ug/kg)	Dibromomethan (ug/kg)	
16SD04	4/14/93	0:00	0	Primary		6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+		3.35	X+
16SD04	2/21/95	8:55	0	Primary		6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+		3	X+
16SD05	5/2/93	0:00	0	Primary		8.5	X+	8.5	X+	8.5	X+	8.5	X+	8.5	X+		4.15	X+
16SD05	5/2/93	0:00	0	Duplicate 1		8.5	X+	8.5	X+	8.5	X+	8.5	X+	8.5	X+		4.25	X+
16SD05-A				Avg.		8.5		8.5		8.5		8.5		8.5			4.2	
16SD12	2/21/95	9:50	0	Primary		6	X+	6	X+	6	X+	6	X+	6	X+		3	X+
16SD13	2/21/95	10:05	0	Primary		7	X+	7	X+	7	X+	7	X+	7	X+		3.5	X+
16SD14	2/21/95	14:50	0	Primary		7.5	X+	7.5	X+	7.5	X+	7.5	X+	7.5	X+		4	X+
16SD14	2/21/95	14:50	0	Duplicate 1		7.5	X+	7.5	X+	7.5	X+	7.5	X+	7.5	X+		3.5	X+
16SD14-A				Avg.		7.5		7.5		7.5		7.5		7.5			3.75	
16SD15	3/16/95	0:00	0	Primary		7	X+U	7	X+U	7	X+U	7	X+U	7	X+U+J		3.5	X+U
16SD16	3/16/95	16:45	0	Primary		7	X+	7	X+	7	X+	7	X+	7	X+		3.5	X+
16SD17	3/6/95	14:45	0	Primary		7	X+	7	X+	7	X+	7	X+	7	X+		3.5	X+
16SD17	3/6/95	14:45	0	Duplicate 1		6	X+	6	X+	6	X+	6	X+	6	X+		3	X+
16SD17-A				Avg.		6.5		6.5		6.5		6.5		6.5			3.25	
16SD18	3/6/95	11:00	0	Primary		6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+		3.5	X+
16SD19	3/6/95	10:00	0	Primary		8	X+	8	X+	8	X+	8	X+	8	X+		4	X+
16SD20	3/6/95	12:45	0	Primary		7.5	X+	7.5	X+	7.5	X+	7.5	X+	7.5	X+		3.5	X+
16SD21	5/10/95	14:55	0	Primary		6.5	X+	6.5	X+	6.5	X+	6.5	X+	6.5	X+		3.5	X+
NATURAL LOGS																		
16SD04						1.872		1.872		1.872		1.872		1.872			1.209	
16SD04						1.872		1.872		1.872		1.872		1.872			1.099	
16SD05-A						2.14		2.14		2.14		2.14		2.14			1.435	
16SD12						1.792		1.792		1.792		1.792		1.792			1.099	
16SD13						1.946		1.946		1.946		1.946		1.946			1.253	
16SD14-A						2.015		2.015		2.015		2.015		2.015			1.322	
16SD15						1.946		1.946		1.946		1.946		1.946			1.253	
16SD16						1.946		1.946		1.946		1.946		1.946			1.253	
16SD17-A						1.872		1.872		1.872		1.872		1.872			1.179	
16SD18						1.872		1.872		1.872		1.872		1.872			1.253	
16SD19						2.079		2.079		2.079		2.079		2.079			1.386	
16SD20						2.015		2.015		2.015		2.015		2.015			1.253	
16SD21						1.872		1.872		1.872		1.872		1.872			1.253	
STATISTICS																		
Total No.					0	13		13		13		13		13		0	13	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Methacrylonitrile (ug/kg)	Bromomethane (ug/kg)	Chloromethane (ug/kg)	2-Butanone (ug/kg)	Iodomethane (ug/kg)	4-Methyl-2-pentanone (ug/kg)	Methyl methacrylate (ug/kg)	Dibromomethan (ug/kg)
Min.					0	6	6	6	6	6	0	3
Max.					0	8.5	8.5	8.5	8.5	8.5	0	4.25
Avg.(log transformed)					#DIV/0!	1.941	1.941	1.941	1.941	1.941	#DIV/0!	1.25
St. Dev (log transformed)					#DIV/0!	0.098	0.098	0.098	0.098	0.098	#DIV/0!	0.096
H												
95th UCL of Mean					#DIV/0!	7.00	7.00	7.00	7.00	7.00	#DIV/0!	3.51

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Methylene chloride (ug/kg)		Naphthalene (ug/kg)	n-Butylbenzene (ug/kg)	1-Propanamine, N-nitroso-n-pro (ug/kg)	n-Propyl benzene (ug/kg)	2-Chlortoluene (ug/kg)	1,2-Dichloro benzene (ug/kg)	o-Xylene (ug/kg)
16SD04	4/14/93	0:00	0	Primary	3.35	X+							
16SD04	2/21/95	8:55	0	Primary	6.5	X+							
16SD05	5/2/93	0:00	0	Primary	4.15	X+							
16SD05	5/2/93	0:00	0	Duplicate 1	4.25	X+							
16SD05-A				Avg.	4.2								
16SD12	2/21/95	9:50	0	Primary	6	X+							
16SD13	2/21/95	10:05	0	Primary	7	X+							
16SD14	2/21/95	14:50	0	Primary	7.5	X+							
16SD14	2/21/95	14:50	0	Duplicate 1	7.5	X+							
16SD14-A				Avg.	7.5								
16SD15	3/16/95	0:00	0	Primary	19.5	U+							
16SD16	3/16/95	16:45	0	Primary	7	X+							
16SD17	3/6/95	14:45	0	Primary	7	X+							
16SD17	3/6/95	14:45	0	Duplicate 1	6	X+							
16SD17-A				Avg.	6.5								
16SD18	3/6/95	11:00	0	Primary	6.5	X+							
16SD19	3/6/95	10:00	0	Primary	8	X+							
16SD20	3/6/95	12:45	0	Primary	7.5	X+							
16SD21	5/10/95	14:55	0	Primary	6.5	X+							
NATURAL LOGS													
16SD04					1.209								
16SD04					1.872								
16SD05-A					1.435								
16SD12					1.792								
16SD13					1.946								
16SD14-A					2.015								
16SD15					2.97								
16SD16					1.946								
16SD17-A					1.872								
16SD18					1.872								
16SD19					2.079								
16SD20					2.015								
16SD21					1.872								
STATISTICS													
Total No.					13		0	0	0	0	0	0	0

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Methylene chloride (ug/kg)	Naphthalene (ug/kg)	n-Butylbenzene (ug/kg)	1-Propanamine, N-nitroso-n-pro (ug/kg)	n-Propyl benzene (ug/kg)	2-Chlortoluene (ug/kg)	1,2-Dichloro benzene (ug/kg)	o-Xylene (ug/kg)
Min.					3.35	0	0	0	0	0	0	0
Max.					19.5	0	0	0	0	0	0	0
Avg.(log transformed)					1.915	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!
St. Dev (log transformed)					0.399	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!
H												
95th UCL of Mean					7.35	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	4-Chlorotoluene (ug/kg)	p-Isopropyl toluene (ug/kg)	1,4-Dichloro benzene (ug/kg)	Pentachloro ethane (ug/kg)	sec-Butyl benzene (ug/kg)	Styrene (ug/kg)		tert-Butyl benzene (ug/kg)	Tetrachloro ethene (ug/kg)	
16SD04	4/14/93	0:00	0	Primary						3.35	X+		3.35	X+
16SD04	2/21/95	8:55	0	Primary						3	X+		3	X+
16SD05	5/2/93	0:00	0	Primary						4.15	X+		4.15	X+
16SD05	5/2/93	0:00	0	Duplicate 1						4.25	X+		4.25	X+
16SD05-A				Avg.						4.2			4.2	
16SD12	2/21/95	9:50	0	Primary						3	X+		3	X+
16SD13	2/21/95	10:05	0	Primary						3.5	X+		3.5	X+
16SD14	2/21/95	14:50	0	Primary						4	X+		4	X+
16SD14	2/21/95	14:50	0	Duplicate 1						3.5	X+		3.5	X+
16SD14-A				Avg.						3.75			3.75	
16SD15	3/16/95	0:00	0	Primary						3.5	X+U+J		3.5	X+U
16SD16	3/16/95	16:45	0	Primary						3.5	X+		3.5	X+
16SD17	3/6/95	14:45	0	Primary						3.5	X+		3.5	X+
16SD17	3/6/95	14:45	0	Duplicate 1						3	X+		3	X+
16SD17-A				Avg.						3.25			3.25	
16SD18	3/6/95	11:00	0	Primary						3.5	X+		3.5	X+
16SD19	3/6/95	10:00	0	Primary						4	X+		4	X+
16SD20	3/6/95	12:45	0	Primary						3.5	X+		3.5	X+
16SD21	5/10/95	14:55	0	Primary						3.5	X+		3.5	X+
NATURAL LOGS														
16SD04										1.209			1.209	
16SD04										1.099			1.099	
16SD05-A										1.435			1.435	
16SD12										1.099			1.099	
16SD13										1.253			1.253	
16SD14-A										1.322			1.322	
16SD15										1.253			1.253	
16SD16										1.253			1.253	
16SD17-A										1.179			1.179	
16SD18										1.253			1.253	
16SD19										1.386			1.386	
16SD20										1.253			1.253	
16SD21										1.253			1.253	
STATISTICS														
Total No.					0	0	0	0	0	13		0	13	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	4-Chlorotoluene (ug/kg)	p-Isopropyl toluene (ug/kg)	1,4-Dichloro benzene (ug/kg)	Pentachloro ethane (ug/kg)	sec-Butyl benzene (ug/kg)	Styrene (ug/kg)	tert-Butyl benzene (ug/kg)	Tetrachloro ethene (ug/kg)
Min.					0	0	0	0	0	3	0	3
Max.					0	0	0	0	0	4.25	0	4.25
Avg.(log transformed)					#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	1.25	#DIV/0!	1.25
St. Dev (log transformed)					#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	0.096	#DIV/0!	0.096
H												
95th UCL of Mean					#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	3.51	#DIV/0!	3.51

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Toluene (ug/kg)		Trans-1,3-Dichloropropene (ug/kg)		Trichloroethene (ug/kg)		Trichloro fluoromethane (ug/kg)		Vinyl Acetate (ug/kg)		Vinyl chloride (ug/kg)		Xylene (total) (ug/kg)	
16SD04	4/14/93	0:00	0	Primary	3.35	X+	3.35	X+	3.35	X+	3.35	X+	3.35	X+	6.5	X+	3.35	X+
16SD04	2/21/95	8:55	0	Primary	3	X+	3	X+	3	X+	3	X+	6.5	X+	6.5	X+	3	X+
16SD05	5/2/93	0:00	0	Primary	4.15	X+	4.15	X+	4.15	X+	4.15	X+	4.15	X+	8.5	X+	4.15	X+
16SD05	5/2/93	0:00	0	Duplicate 1	4.25	X+	4.25	X+	4.25	X+	4.25	X+	4.25	X+	8.5	X+	4.25	X+
16SD05-A				Avg.	4.2		4.2		4.2		4.2		4.2		8.5		4.2	
16SD12	2/21/95	9:50	0	Primary	3	X+	3	X+	3	X+	3	X+	6	X+	6	X+	3	X+
16SD13	2/21/95	10:05	0	Primary	3.5	X+	3.5	X+	3.5	X+	3.5	X+	7	X+	7	X+	3.5	X+
16SD14	2/21/95	14:50	0	Primary	4	X+	4	X+	4	X+	4	X+	7.5	X+	7.5	X+	4	X+
16SD14	2/21/95	14:50	0	Duplicate 1	3.5	X+	3.5	X+	3.5	X+	3.5	X+	7.5	X+	7.5	X+	3.5	X+
16SD14-A				Avg.	3.75		3.75		3.75		3.75		7.5		7.5		3.75	
16SD15	3/16/95	0:00	0	Primary	3.5	X+U	3.5	X+U	3.5	X+U	3.5	X+U	7	X+U	7	X+U	3.5	X+U+J
16SD16	3/16/95	16:45	0	Primary	3.5	X+	3.5	X+	3.5	X+	3.5	X+	7	X+	7	X+	3.5	X+
16SD17	3/6/95	14:45	0	Primary	3.5	X+	3.5	X+	3.5	X+	3.5	X+	7	X+	7	X+	3.5	X+
16SD17	3/6/95	14:45	0	Duplicate 1	3	X+	3	X+	3	X+	3	X+	6	X+	6	X+	3	X+
16SD17-A				Avg.	3.25		3.25		3.25		3.25		6.5		6.5		3.25	
16SD18	3/6/95	11:00	0	Primary	3.5	X+	3.5	X+	3.5	X+	3.5	X+	6.5	X+	6.5	X+	3.5	X+
16SD19	3/6/95	10:00	0	Primary	4	X+	4	X+	4	X+	4	X+	8	X+	8	X+	4	X+
16SD20	3/6/95	12:45	0	Primary	3.5	X+	3.5	X+	3.5	X+	3.5	X+	7.5	X+	7.5	X+	3.5	X+
16SD21	5/10/95	14:55	0	Primary	3.5	X+	3.5	X+	3.5	X+	3.5	X+	6.5	X+	6.5	X+	3.5	X+
NATURAL LOGS																		
16SD04					1.209		1.209		1.209		1.209		1.209		1.872		1.209	
16SD04					1.099		1.099		1.099		1.099		1.872		1.872		1.099	
16SD05-A					1.435		1.435		1.435		1.435		1.435		2.14		1.435	
16SD12					1.099		1.099		1.099		1.099		1.792		1.792		1.099	
16SD13					1.253		1.253		1.253		1.253		1.946		1.946		1.253	
16SD14-A					1.322		1.322		1.322		1.322		2.015		2.015		1.322	
16SD15					1.253		1.253		1.253		1.253		1.946		1.946		1.253	
16SD16					1.253		1.253		1.253		1.253		1.946		1.946		1.253	
16SD17-A					1.179		1.179		1.179		1.179		1.872		1.872		1.179	
16SD18					1.253		1.253		1.253		1.253		1.872		1.872		1.253	
16SD19					1.386		1.386		1.386		1.386		2.079		2.079		1.386	
16SD20					1.253		1.253		1.253		1.253		2.015		2.015		1.253	
16SD21					1.253		1.253		1.253		1.253		1.872		1.872		1.253	
STATISTICS																		
Total No.					13		13		13		13		13		13		13	

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	Toluene (ug/kg)	Trans-1,3-Dichloropropene (ug/kg)	Trichloroethene (ug/kg)	Trichloro fluoromethane (ug/kg)	Vinyl Acetate (ug/kg)	Vinyl chloride (ug/kg)	Xylene (total) (ug/kg)
Min.					3	3	3	3	3.35	6	3
Max.					4.25	4.25	4.25	4.25	8	8.5	4.25
Avg.(log transformed)					1.25	1.25	1.25	1.25	1.836	1.941	1.25
St. Dev (log transformed)					0.096	0.096	0.096	0.096	0.245	0.098	0.096
H											
95th UCL of Mean					3.51	3.51	3.51	3.51	6.46	7.00	3.51

Data Qualifier Notations

X+ = Non-Detect Result, one-half of the detection limit value presented.
U+ = Considered Non-Detect based on blank comparison, one-half of the detection limit value is presented.
J = Estimated laboratory result.

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Aluminum (mg/l)	Antimony (ug/l)		Arsenic (ug/l)		Barium (ug/l)	Beryllium (mg/l)	Cadmium (ug/l)		Calcium (mg/l)	Chromium (ug/l)	
16SW04	4/14/93	0:00	Primary		15	X+	5	X+U	140		2.5	X+		5	X+
16SW04	2/21/95	9:00	Primary		5	X+	2.5	X+	177		2.5	X+		5	X+
16SW05	5/13/93	0:00	Primary		15	X+	5	X+	155		2.5	X+		5	X+
16SW12	2/21/95	9:45	Primary		5	X+	2.5	X+	183		2.5	X+		5	X+
16SW13	2/21/95	10:00	Primary		5	X+	2.5	X+	96		2.5	X+		5	X+
16SW14	2/21/95	14:50	Primary		5	X+U	2.5	X+	90		2.5	X+		5	X+
16SW14	2/21/95	14:50	Duplicate 1		5	X+U	2.5	X+	88		2.5	X+		5	X+
16SW14-A			Avg.		5		2.5		89		2.5			5	
16SW15	3/16/95	15:15	Primary		5	X+R	2.5	X+U	84		2.5	X+		5	X+
16SW16	3/16/95	16:50	Primary		5	X+R	2.5	X+U	124		2.5	X+		5	X+
16SW17	3/6/95	14:45	Primary		5	X+	2.5	X+	88		2.5	X+		5	X+
16SW17	3/6/95	14:45	Duplicate 1		5	X+	2.5	X+	89		2.5	X+		5	X+
16SW17-A			Avg.		5		2.5		88.5		2.5			5	
16SW18	3/6/95	11:00	Primary		5	X+	2.5	X+	171		2.5	X+		5	X+
16SW19	3/6/95	10:00	Primary		5	X+	2.5	X+	72		2.5	X+		5	X+
16SW20	3/6/95	12:45	Primary		5	X+	2.5	X+	62		2.5	X+		5	X+
16SW21	5/10/95	14:55	Primary		5	X+	2.5	X+	101		2.5	X+		5	X+
NATURAL LOGS															
16SW04					2.708		1.609		4.942		0.916			1.609	
16SW04					1.609		0.916		5.176		0.916			1.609	
16SW05					2.708		1.609		5.043		0.916			1.609	
16SW12					1.609		0.916		5.209		0.916			1.609	
16SW13					1.609		0.916		4.564		0.916			1.609	
16SW14-A					1.609		0.916		4.489		0.916			1.609	
16SW15					1.609		0.916		4.431		0.916			1.609	
16SW16					1.609		0.916		4.82		0.916			1.609	
16SW17-A					1.609		0.916		4.483		0.916			1.609	
16SW18					1.609		0.916		5.142		0.916			1.609	
16SW19					1.609		0.916		4.277		0.916			1.609	
16SW20					1.609		0.916		4.127		0.916			1.609	
16SW21					1.609		0.916		4.615		0.916			1.609	

STATISTICS

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Aluminum (mg/l)	Antimony (ug/l)	Arsenic (ug/l)	Barium (ug/l)	Beryllium (mg/l)	Cadmium (ug/l)	Calcium (mg/l)	Chromium (ug/l)
Total No.					13	13	13	0	13	0	13
Min.					5	2.5	62	0	2.5	0	5
Max.					15	5	183	0	2.5	0	5
Avg.(log transformed)					1.778	1.023	4.717	#DIV/0!	0.916	#DIV/0!	1.609
St. Dev.(log transformed)					0.413	0.26	0.361	#DIV/0!	0	#DIV/0!	0
H							1.966				
95th UCL of the Mean					6.44487	2.877149	146.5022	#DIV/0!	2.499273	#DIV/0!	4.997811

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Cobalt (mg/l)	Copper (mg/l)		Iron (mg/l)	Lead (ug/l)		Magnesium (mg/l)	Manganese (mg/l)	Mercury (ug/l)		Nickel (ug/l)	
16SW04	4/14/93	0:00	Primary					1	X+			1	X+	25	X+
16SW04	2/21/95	9:00	Primary		0.0025	X+		2.5	X+			0.1	X+	20	X+
16SW05	5/13/93	0:00	Primary					1	X+			1	X+	25	X+
16SW12	2/21/95	9:45	Primary		0.005			2.5	X+			0.1	X+	20	X+
16SW13	2/21/95	10:00	Primary		0.005			2.5	X+			0.1	X+	20	X+
16SW14	2/21/95	14:50	Primary		0.0025	X+		10	X+			0.1	X+	20	X+
16SW14	2/21/95	14:50	Duplicate 1		0.0025	X+		10	X+			0.1	X+	20	X+
16SW14-A			Avg.		0.0025			10				0.1		20	
16SW15	3/16/95	15:15	Primary		0.0025	X+		2	U+			0.1	X+	20	X+
16SW16	3/16/95	16:50	Primary		0.0025	X+		9	J			0.1	X+	20	X+
16SW17	3/6/95	14:45	Primary		0.009			1	X+U+J			0.1	X+	20	X+
16SW17	3/6/95	14:45	Duplicate 1		0.009			1	X+			0.1	X+	20	X+
16SW17-A			Avg.		0.009			1				0.1		20	
16SW18	3/6/95	11:00	Primary		0.006			10	X+			0.1	X+	20	X+
16SW19	3/6/95	10:00	Primary		0.007			1	X+			0.1	X+	20	X+
16SW20	3/6/95	12:45	Primary		0.009			1	X+			0.1	X+	20	X+
16SW21	5/10/95	14:55	Primary		0.0025	X+		2.5	X+			0.1	X+	20	X+
NATURAL LOGS															
16SW04								0				0		3.219	
16SW04					-5.991			0.916				-2.303		2.996	
16SW05								0				0		3.219	
16SW12					-5.298			0.916				-2.303		2.996	
16SW13					-5.298			0.916				-2.303		2.996	
16SW14-A					-5.991			2.303				-2.303		2.996	
16SW15					-5.991			0.693				-2.303		2.996	
16SW16					-5.991			2.197				-2.303		2.996	
16SW17-A					-4.711			0				-2.303		2.996	
16SW18					-5.116			2.303				-2.303		2.996	
16SW19					-4.962			0				-2.303		2.996	
16SW20					-4.711			0				-2.303		2.996	
16SW21					-5.991			0.916				-2.303		2.996	

STATISTICS

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Cobalt (mg/l)	Copper (mg/l)	Iron (mg/l)	Lead (ug/l)	Magnesium (mg/l)	Manganese (mg/l)	Mercury (ug/l)	Nickel (ug/l)
Total No.				0	11	0	13	0	0	13	13
Min.				0	0.0025	0	1	0	0	0.1	20
Max.				0	0.009	0	10	0	0	1	25
Avg.(log transformed)				#DIV/0!	-5.459	#DIV/0!	0.858	#DIV/0!	#DIV/0!	-1.949	3.03
St. Dev.(log transformed)				#DIV/0!	0.543	#DIV/0!	0.899	#DIV/0!	#DIV/0!	0.865	0.084
H					2.235		2.677				
95th UCL of the Mean				#DIV/0!	0.007242	#DIV/0!	7.076877	#DIV/0!	#DIV/0!	0.207031	20.77038

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Potassium (mg/l)	Selenium (ug/l)		Silver (ug/l)		Sodium (mg/l)	Strontium (mg/l)	Thallium (ug/l)		Vanadium (mg/l)	Zinc (mg/l)
16SW04	4/14/93	0:00	Primary		5	X+	35	X+			5	X+		
16SW04	2/21/95	9:00	Primary		2.5	X+	5	X+			2.5	X+		0.019
16SW05	5/13/93	0:00	Primary		5	X+	35	X+			5	X+		
16SW12	2/21/95	9:45	Primary		5	X+	5	X+			2.5	X+		0.013
16SW13	2/21/95	10:00	Primary		5	X+	5	X+			2.5	X+		0.009
16SW14	2/21/95	14:50	Primary		5	X+	5	X+			25	X+		0.054
16SW14	2/21/95	14:50	Duplicate 1		2.5	X+	5	X+			25	X+		0.034
16SW14-A			Avg.		3.75		5				25			0.044
16SW15	3/16/95	15:15	Primary		2.5	X+	5	X+			2.5	X+		0.014
16SW16	3/16/95	16:50	Primary		2.5	X+	5	X+			2.5	X+		0.02
16SW17	3/6/95	14:45	Primary		5	X+U	5	X+			2.5	X+		0.01
16SW17	3/6/95	14:45	Duplicate 1		5	X+	5	X+			2.5	X+		0.01
16SW17-A			Avg.		5		5				2.5			0.01
16SW18	3/6/95	11:00	Primary		5	X+U	5	X+			2.5	X+		0.032
16SW19	3/6/95	10:00	Primary		5	X+U	5	X+			2.5	X+		0.014
16SW20	3/6/95	12:45	Primary		2.5	X+U	5	X+			2.5	X+		0.009
16SW21	5/10/95	14:55	Primary		5	X+	5	X+			50	X+		0.017
NATURAL LOGS														
16SW04					1.609		3.555				1.609			
16SW04					0.916		1.609				0.916			-3.963
16SW05					1.609		3.555				1.609			
16SW12					1.609		1.609				0.916			-4.343
16SW13					1.609		1.609				0.916			-4.711
16SW14-A					1.322		1.609				3.219			-3.124
16SW15					0.916		1.609				0.916			-4.269
16SW16					0.916		1.609				0.916			-3.912
16SW17-A					1.609		1.609				0.916			-4.605
16SW18					1.609		1.609				0.916			-3.442
16SW19					1.609		1.609				0.916			-4.269
16SW20					0.916		1.609				0.916			-4.711
16SW21					1.609		1.609				3.912			-4.075

STATISTICS

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Potassium (mg/l)	Selenium (ug/l)	Silver (ug/l)	Sodium (mg/l)	Strontium (mg/l)	Thallium (ug/l)	Vanadium (mg/l)	Zinc (mg/l)
Total No.				0	13	13	0	0	13	0	11
Min.				0	2.5	5	0	0	2.5	0	0.009
Max.				0	5	35	0	0	50	0	0.054
Avg.(log transformed)				#DIV/0!	1.374	1.908	#DIV/0!	#DIV/0!	1.43	#DIV/0!	-4.129
St. Dev.(log transformed)				#DIV/0!	0.327	0.731	#DIV/0!	#DIV/0!	0.992	#DIV/0!	0.505
H											2.183
95th UCL of the Mean				#DIV/0!	4.168118	8.803774	#DIV/0!	#DIV/0!	6.834833	#DIV/0!	0.025917

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	1,1,1,2-Tetra chloroethane (ug/l)	1,1,1-trichloro ethane (ug/l)		1,1,2,2-Tetra chloroethane (ug/l)		1,1,2-Trichloro ethane (ug/l)		1,1-Dichloro ethane (ug/l)		1,1-Dichloro ethylene (ug/l)		1,1-Dichloro propene (ug/l)	1,2,3-Trichloro benzene (ug/l)	1,2,3-Trichloro propane (ug/l)		1,2,4-Trich benzene (ug/l)
16SW04	4/14/93	0:00	Primary		2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+			2.5	X+	
16SW04	2/21/95	9:00	Primary		2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+			2.5	X+	
16SW05	5/13/93	0:00	Primary		2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+			2.5	X+	
16SW12	2/21/95	9:45	Primary		2.5	U+J	2.5	U+J	2.5	U+J	2.5	U+J	2.5	U+JX+			2.5	U+JX+	
16SW13	2/21/95	10:00	Primary		2.5	U+J	2.5	U+J	2.5	U+J	2.5	U+J	2.5	U+JX+			2.5	U+JX+	
16SW14	2/21/95	14:50	Primary		2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+			2.5	X+	
16SW14	2/21/95	14:50	Duplicate 1		2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+			2.5	X+	
16SW14-A			Avg.		2.5		2.5		2.5		2.5		2.5				2.5		
16SW15	3/16/95	15:15	Primary		2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+			2.5	X+	
16SW16	3/16/95	16:50	Primary		2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+			2.5	X+	
16SW17	3/6/95	14:45	Primary		2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+			2.5	X+	
16SW17	3/6/95	14:45	Duplicate 1		2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+			2.5	X+	
16SW17-A			Avg.		2.5		2.5		2.5		2.5		2.5				2.5		
16SW18	3/6/95	11:00	Primary		2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+			2.5	X+	
16SW19	3/6/95	10:00	Primary		2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+			2.5	X+	
16SW20	3/6/95	12:45	Primary		2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+			2.5	X+	
16SW21	5/10/95	14:55	Primary		2.5	X+	2.5	X+	2.5	X+	2.5	X+	2.5	X+			2.5	X+	
NATURAL LOGS																			
16SW04					0.916		0.916		0.916		0.916		0.916				0.916		
16SW04					0.916		0.916		0.916		0.916		0.916				0.916		
16SW05					0.916		0.916		0.916		0.916		0.916				0.916		
16SW12					0.916		0.916		0.916		0.916		0.916				0.916		
16SW13					0.916		0.916		0.916		0.916		0.916				0.916		
16SW14-A					0.916		0.916		0.916		0.916		0.916				0.916		
16SW15					0.916		0.916		0.916		0.916		0.916				0.916		
16SW16					0.916		0.916		0.916		0.916		0.916				0.916		
16SW17-A					0.916		0.916		0.916		0.916		0.916				0.916		
16SW18					0.916		0.916		0.916		0.916		0.916				0.916		
16SW19					0.916		0.916		0.916		0.916		0.916				0.916		
16SW20					0.916		0.916		0.916		0.916		0.916				0.916		
16SW21					0.916		0.916		0.916		0.916		0.916				0.916		
STATISTICS																			
Total No.					13		13		13		13		13		0	0	13		0
Min.					2.5		2.5		2.5		2.5		2.5		0	0	2.5		0
Max.					2.5		2.5		2.5		2.5		2.5		0	0	2.5		0

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	1,1,1,2-Tetrachloroethane (ug/l)	1,1,1-trichloroethane (ug/l)	1,1,2,2-Tetrachloroethane (ug/l)	1,1,2-Trichloroethane (ug/l)	1,1-Dichloroethane (ug/l)	1,1-Dichloroethylene (ug/l)	1,1-Dichloropropene (ug/l)	1,2,3-Trichlorobenzene (ug/l)	1,2,3-Trichloropropane (ug/l)	1,2,4-Trichbenzene (ug/l)
Avg.(log transformed)					0.916	0.916	0.916	0.916	0.916	#DIV/0!	#DIV/0!	0.916	#DIV/0!
St. Dev.(log transformed)					0	0	0	0	0	#DIV/0!	#DIV/0!	0	#DIV/0!
H													
95th UCL of the Mean					2.499273	2.499273	2.499273	2.499273	2.499273	#DIV/0!	#DIV/0!	2.499273	#DIV/0!

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	loro	1,2-Dichloro ethane (ug/l)		1,2-Dichloro ethene (ug/l)		1,2-Dichloro propane (ug/l)		1,3-Dichloro propane (ug/l)	1,4-Dichloro-2-butene (ug/l)		1,4-Dioxane (ug/l)	2,2-Dichloro propane (ug/l)	2-Hexanone (ug/l)		Acetone (ug/l)	
16SW04	4/14/93	0:00	Primary		2.5	X+			2.5	X+		5	X+			5	X+	5	X+
16SW04	2/21/95	9:00	Primary		2.5	X+	2.5	X+	2.5	X+		5	X+			5	X+	5	X+
16SW05	5/13/93	0:00	Primary		2.5	X+			2.5	X+		5	X+			5	X+	5	X+
16SW12	2/21/95	9:45	Primary		2.5	U+J	2.5	U+J	2.5	U+JX+		5	U+JX+			5	U+J	5	U+J
16SW13	2/21/95	10:00	Primary		2.5	U+J	2.5	U+J	2.5	U+JX+		5	U+JX+			5	U+J	5	U+J
16SW14	2/21/95	14:50	Primary		2.5	X+	2.5	X+	2.5	X+		5	X+			5	X+	8	J
16SW14	2/21/95	14:50	Duplicate 1		2.5	X+	2.5	X+	2.5	X+		5	X+			5	X+	5	X+
16SW14-A			Avg.		2.5		2.5		2.5			5				5		6.5	
16SW15	3/16/95	15:15	Primary		2.5	X+	2.5	X+	2.5	X+		5	X+			5	X+	5	X+
16SW16	3/16/95	16:50	Primary		2.5	X+	2.5	X+	2.5	X+		5	X+			5	X+	5	X+
16SW17	3/6/95	14:45	Primary		2.5	X+	2.5	X+	2.5	X+		5	X+			5	X+	5	X+
16SW17	3/6/95	14:45	Duplicate 1		2.5	X+	2.5	X+	2.5	X+		5	X+			5	X+	5	X+
16SW17-A			Avg.		2.5		2.5		2.5			5				5		5	
16SW18	3/6/95	11:00	Primary		2.5	X+	2.5	X+	2.5	X+		5	X+			5	X+	5	X+
16SW19	3/6/95	10:00	Primary		2.5	X+	2.5	X+	2.5	X+		5	X+			5	X+	5	X+
16SW20	3/6/95	12:45	Primary		2.5	X+	2.5	X+	2.5	X+		5	X+			5	X+	5	X+
16SW21	5/10/95	14:55	Primary		2.5	X+	2.5	X+	2.5	X+		5	X+			5	X+	5	X+
NATURAL LOGS																			
16SW04					0.916				0.916			1.609				1.609		1.609	
16SW04					0.916		0.916		0.916			1.609				1.609		1.609	
16SW05					0.916				0.916			1.609				1.609		1.609	
16SW12					0.916		0.916		0.916			1.609				1.609		1.609	
16SW13					0.916		0.916		0.916			1.609				1.609		1.609	
16SW14-A					0.916		0.916		0.916			1.609				1.609		1.872	
16SW15					0.916		0.916		0.916			1.609				1.609		1.609	
16SW16					0.916		0.916		0.916			1.609				1.609		1.609	
16SW17-A					0.916		0.916		0.916			1.609				1.609		1.609	
16SW18					0.916		0.916		0.916			1.609				1.609		1.609	
16SW19					0.916		0.916		0.916			1.609				1.609		1.609	
16SW20					0.916		0.916		0.916			1.609				1.609		1.609	
16SW21					0.916		0.916		0.916			1.609				1.609		1.609	
STATISTICS																			
Total No.					13		11		13		0	13		0		13		13	
Min.					2.5		2.5		2.5		0	5		0		5		5	
Max.					2.5		2.5		2.5		0	5		0		5		8	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	loro	1,2-Dichloro ethane (ug/l)	1,2-Dichloro ethene (ug/l)	1,2-Dichloro propane (ug/l)	1,3-Dichloro propane (ug/l)	1,4-Dichloro-2-butene (ug/l)	1,4-Dioxane (ug/l)	2,2-Dichloro propane (ug/l)	2-Hexanone (ug/l)	Acetone (ug/l)
Avg.(log transformed)					0.916	0.916	0.916	#DIV/0!	1.609	#DIV/0!	#DIV/0!	1.609	1.629
St. Dev.(log transformed)					0	0	0	#DIV/0!	0	#DIV/0!	#DIV/0!	0	0.073
H													1.749
95th UCL of the Mean					2.499273	2.499273	2.499273	#DIV/0!	4.997811	#DIV/0!	#DIV/0!	4.997811	5.30432

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Acrolein (ug/l)		Acrylonitrile (ug/l)		Benzene (ug/l)		Benzene, 1,2,4-trimethyl (ug/l)	Benzene, 1,3,5-trimethyl- (ug/l)	Benzene, 1-methylethyl- (ug/l)	Bromobenzene (ug/l)	Bromodichloro methane (ug/l)		Bromoform (ug/l)		Carbon disulfide (ug/l)
16SW04	4/14/93	0:00	Primary	5	X+	5	X+	2.5	X+					2.5	X+			2.5
16SW04	2/21/95	9:00	Primary	25	X+	25	X+	2.5	X+					2.5	X+	2.5	X+	2.5
16SW05	5/13/93	0:00	Primary	5	X+	5	X+	2.5	X+					2.5	X+			2.5
16SW12	2/21/95	9:45	Primary	25	U+J	25	U+J	2.5	U+JX+					2.5	U+J	2.5	U+J	2.5
16SW13	2/21/95	10:00	Primary	25	U+J	25	U+J	2.5	U+JX+					2.5	U+J	2.5	U+J	2.5
16SW14	2/21/95	14:50	Primary	25	X+	25	X+	2.5	X+					2.5	X+	2.5	X+	2.5
16SW14	2/21/95	14:50	Duplicate 1	25	X+	25	X+	2.5	X+					2.5	X+	2.5	X+	2.5
16SW14-A			Avg.	25		25		2.5						2.5		2.5		2.5
16SW15	3/16/95	15:15	Primary	25	X+	25	X+	2.5	X+					2.5	X+	2.5	X+	2.5
16SW16	3/16/95	16:50	Primary	25	X+	25	X+	2.5	X+					2.5	X+	2.5	X+	2.5
16SW17	3/6/95	14:45	Primary	25	X+	25	X+	2.5	X+					2.5	X+	2.5	X+	2.5
16SW17	3/6/95	14:45	Duplicate 1	25	X+	25	X+	2.5	X+					2.5	X+	2.5	X+	2.5
16SW17-A			Avg.	25		25		2.5						2.5		2.5		2.5
16SW18	3/6/95	11:00	Primary	25	X+	25	X+	2.5	X+					2.5	X+	2.5	X+	2.5
16SW19	3/6/95	10:00	Primary	25	X+	25	X+	2.5	X+					2.5	X+	2.5	X+	2.5
16SW20	3/6/95	12:45	Primary	25	X+	25	X+	2.5	X+					2.5	X+	2.5	X+	2.5
16SW21	5/10/95	14:55	Primary	25	X+	25	X+	2.5	X+					2.5	X+	2.5	X+	2.5
NATURAL LOGS																		
16SW04				1.609		1.609		0.916						0.916				0.916
16SW04				3.219		3.219		0.916						0.916		0.916		0.916
16SW05				1.609		1.609		0.916						0.916				0.916
16SW12				3.219		3.219		0.916						0.916		0.916		0.916
16SW13				3.219		3.219		0.916						0.916		0.916		0.916
16SW14-A				3.219		3.219		0.916						0.916		0.916		0.916
16SW15				3.219		3.219		0.916						0.916		0.916		0.916
16SW16				3.219		3.219		0.916						0.916		0.916		0.916
16SW17-A				3.219		3.219		0.916						0.916		0.916		0.916
16SW18				3.219		3.219		0.916						0.916		0.916		0.916
16SW19				3.219		3.219		0.916						0.916		0.916		0.916
16SW20				3.219		3.219		0.916						0.916		0.916		0.916
16SW21				3.219		3.219		0.916						0.916		0.916		0.916
STATISTICS																		
Total No.				13		13		13		0	0	0	0	13		11		13
Min.				5		5		2.5		0	0	0	0	2.5		2.5		2.5
Max.				25		25		2.5		0	0	0	0	2.5		2.5		2.5

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Acrolein (ug/l)	Acrylonitrile (ug/l)	Benzene (ug/l)	Benzene, 1,2,4-trimethyl (ug/l)	Benzene, 1,3,5-trimethyl- (ug/l)	Benzene, 1-methylethyl- (ug/l)	Bromobenzene (ug/l)	Bromodichloromethane (ug/l)	Bromoform (ug/l)	Carbon disulfide (ug/l)
Avg.(log transformed)				2.971	2.971	0.916	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	0.916	0.916	0.916
St. Dev.(log transformed)				0.605	0.605	0	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	0	0	0
H													
95th UCL of the Mean				23.42989	23.42989	2.499273	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	2.499273	2.499273	2.499273

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE		Carbon tetrachloride (ug/l)		Chlorobenzene (ug/l)		Chlorobromo methane (ug/l)	Chloroethane (ug/l)		Chloroform (ug/l)		cis-1,2-Dichloro ethylene (ug/l)		cis-1,3-Dichloropropen (ug/l)	DBCP (ug/l)	Dibromochloro methane (ug/l)	
16SW04	4/14/93	0:00	Primary	X+	2.5	X+	2.5	X+		5	X+	2.5	X+	2.5	X+			2.5	X+
16SW04	2/21/95	9:00	Primary	X+	2.5	X+	2.5	X+		5	X+	2.5	X+			2.5	X+	2.5	X+
16SW05	5/13/93	0:00	Primary	X+	2.5	X+	2.5	X+		5	X+	2.5	X+	2.5	X+			2.5	X+
16SW12	2/21/95	9:45	Primary	U+J	2.5	U+J	2.5	U+JX+		5	U+J	2.5	U+JX+			2.5	U+JX+	2.5	U+J
16SW13	2/21/95	10:00	Primary	U+J	2.5	U+J	2.5	U+JX+		5	U+J	2.5	U+JX+			2.5	U+JX+	2.5	U+J
16SW14	2/21/95	14:50	Primary	X+	2.5	X+	2.5	X+		5	X+	2.5	X+			2.5	X+	2.5	X+
16SW14	2/21/95	14:50	Duplicate 1	X+	2.5	X+	2.5	X+		5	X+	2.5	X+			2.5	X+	2.5	X+
16SW14-A			Avg.		2.5		2.5			5		2.5				2.5		2.5	
16SW15	3/16/95	15:15	Primary	X+	2.5	X+	2.5	X+		5	X+	2.5	X+			2.5	X+	2.5	X+
16SW16	3/16/95	16:50	Primary	X+	2.5	X+	2.5	X+		5	X+	2.5	X+			2.5	X+	2.5	X+
16SW17	3/6/95	14:45	Primary	X+	2.5	X+	2.5	X+		5	X+	2.5	X+			2.5	X+	2.5	X+
16SW17	3/6/95	14:45	Duplicate 1	X+	2.5	X+	2.5	X+		5	X+	2.5	X+			2.5	X+	2.5	X+
16SW17-A			Avg.		2.5		2.5			5		2.5				2.5		2.5	
16SW18	3/6/95	11:00	Primary	X+	2.5	X+	2.5	X+		5	X+	2.5	X+			2.5	X+	2.5	X+
16SW19	3/6/95	10:00	Primary	X+	2.5	X+	2.5	X+		5	X+	2.5	X+			2.5	X+	2.5	X+
16SW20	3/6/95	12:45	Primary	X+	2.5	X+	2.5	X+		5	X+	2.5	X+			2.5	X+	2.5	X+
16SW21	5/10/95	14:55	Primary	X+	2.5	X+	2.5	X+		5	X+	2.5	X+			2.5	X+	2.5	X+
NATURAL LOGS																			
16SW04					0.916		0.916			1.609		0.916		0.916				0.916	
16SW04					0.916		0.916			1.609		0.916				0.916		0.916	
16SW05					0.916		0.916			1.609		0.916		0.916				0.916	
16SW12					0.916		0.916			1.609		0.916				0.916		0.916	
16SW13					0.916		0.916			1.609		0.916				0.916		0.916	
16SW14-A					0.916		0.916			1.609		0.916				0.916		0.916	
16SW15					0.916		0.916			1.609		0.916				0.916		0.916	
16SW16					0.916		0.916			1.609		0.916				0.916		0.916	
16SW17-A					0.916		0.916			1.609		0.916				0.916		0.916	
16SW18					0.916		0.916			1.609		0.916				0.916		0.916	
16SW19					0.916		0.916			1.609		0.916				0.916		0.916	
16SW20					0.916		0.916			1.609		0.916				0.916		0.916	
16SW21					0.916		0.916			1.609		0.916				0.916		0.916	
STATISTICS																			
Total No.					13		13		0	13		13		2		11	0	13	
Min.					2.5		2.5		0	5		2.5		2.5		2.5	0	2.5	
Max.					2.5		2.5		0	5		2.5		2.5		2.5	0	2.5	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Carbon tetrachloride (ug/l)	Chlorobenzene (ug/l)	Chlorobromo methane (ug/l)	Chloroethane (ug/l)	Chloroform (ug/l)	cis-1,2-Dichloro ethylene (ug/l)	cis-1,3-Dichloropropen (ug/l)	DBCP (ug/l)	Dibromochloro methane (ug/l)
Avg.(log transformed)				0.916	0.916	#DIV/0!	1.609	0.916	0.916	0.916	#DIV/0!	0.916
St. Dev.(log transformed)				0	0	#DIV/0!	0	0	0	0	#DIV/0!	0
H												
95th UCL of the Mean				2.499273	2.499273	#DIV/0!	4.997811	2.499273	2.499273	2.499273	#DIV/0!	2.499273

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Dichloro difluoromethane (ug/l)		EDB (ug/l)	Ethene, (2-chloroethoxy)- (ug/l)		Ethene, 1,2-dichloro-, (E)- (ug/l)		Ethyl cyanide (ug/l)	Ethyl methacrylate (ug/l)		Ethylbenzene (ug/l)		Hexachloro butadiene (ug/l)	Isobutyl alcohol (ug/l)	m-Dichlor benzene (ug/l)
16SW04	4/14/93	0:00	Primary	2.5	X+				2.5	X+		2.5	X+	2.5	X+			
16SW04	2/21/95	9:00	Primary	2.5	X+		5	X+				2.5	X+	2.5	X+			
16SW05	5/13/93	0:00	Primary	2.5	X+				2.5	X+		2.5	X+	2.5	X+			
16SW12	2/21/95	9:45	Primary	2.5	U+JX+		5	U+JX+				2.5	U+J	2.5	U+JX+			
16SW13	2/21/95	10:00	Primary	2.5	U+JX+		5	U+JX+				2.5	U+J	2.5	U+JX+			
16SW14	2/21/95	14:50	Primary	2.5	X+		5	X+				2.5	X+	2.5	X+			
16SW14	2/21/95	14:50	Duplicate 1	2.5	X+		5	X+				2.5	X+	2.5	X+			
16SW14-A			Avg.	2.5			5					2.5		2.5				
16SW15	3/16/95	15:15	Primary	2.5	X+		5	X+				2.5	X+	2.5	X+			
16SW16	3/16/95	16:50	Primary	2.5	X+		5	X+				2.5	X+	2.5	X+			
16SW17	3/6/95	14:45	Primary	2.5	X+		5	X+				2.5	X+	2.5	X+			
16SW17	3/6/95	14:45	Duplicate 1	2.5	X+		5	X+				2.5	X+	2.5	X+			
16SW17-A			Avg.	2.5			5					2.5		2.5				
16SW18	3/6/95	11:00	Primary	2.5	X+		5	X+				2.5	X+	2.5	X+			
16SW19	3/6/95	10:00	Primary	2.5	X+		5	X+				2.5	X+	2.5	X+			
16SW20	3/6/95	12:45	Primary	2.5	X+		5	X+				2.5	X+	2.5	X+			
16SW21	5/10/95	14:55	Primary	2.5	X+		5	X+				2.5	X+	2.5	X+			
NATURAL LOGS																		
16SW04				0.916					0.916			0.916		0.916				
16SW04				0.916			1.609					0.916		0.916				
16SW05				0.916					0.916			0.916		0.916				
16SW12				0.916			1.609					0.916		0.916				
16SW13				0.916			1.609					0.916		0.916				
16SW14-A				0.916			1.609					0.916		0.916				
16SW15				0.916			1.609					0.916		0.916				
16SW16				0.916			1.609					0.916		0.916				
16SW17-A				0.916			1.609					0.916		0.916				
16SW18				0.916			1.609					0.916		0.916				
16SW19				0.916			1.609					0.916		0.916				
16SW20				0.916			1.609					0.916		0.916				
16SW21				0.916			1.609					0.916		0.916				
STATISTICS																		
Total No.				13	0		11		2		0	13		13		0	0	0
Min.				2.5	0		5		2.5		0	2.5		2.5		0	0	0
Max.				2.5	0		5		2.5		0	2.5		2.5		0	0	0

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Dichloro difluoromethane (ug/l)	EDB (ug/l)	Ethene, (2-chloroethoxy)- (ug/l)	Ethene, 1,2-dichloro-, (E)- (ug/l)	Ethyl cyanide (ug/l)	Ethyl methacrylate (ug/l)	Ethylbenzene (ug/l)	Hexachloro butadiene (ug/l)	Isobutyl alcohol (ug/l)	m-Dichlor benzene (ug/l)
Avg.(log transformed)				0.916	#DIV/0!	1.609	0.916	#DIV/0!	0.916	0.916	#DIV/0!	#DIV/0!	#DIV/0!
St. Dev.(log transformed)				0	#DIV/0!	0	0	#DIV/0!	0	0	#DIV/0!	#DIV/0!	#DIV/0!
H													
95th UCL of the Mean				2.499273	#DIV/0!	4.997811	2.499273	#DIV/0!	2.499273	2.499273	#DIV/0!	#DIV/0!	#DIV/0!

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Methacrylo nitrile (ug/l)	Methyl bromide (ug/l)		Methyl chloride (ug/l)		Methyl ethyl ketone (ug/l)		Methyl iodide (ug/l)		Methyl isobutyl ketone (MIBK) (ug/l)		Methyl methacrylate (ug/l)	Methylene bromide (ug/l)		Methylene chloride (ug/l)	
16SW04	4/14/93	0:00	Primary		5	X+	5	X+	5	X+	5	X+	5	X+		2.5	X+	2.5	X+
16SW04	2/21/95	9:00	Primary		5	X+	5	X+	5	X+	5	X+	5	X+		2.5	X+	5	X+
16SW05	5/13/93	0:00	Primary		5	X+	5	X+	5	X+	5	X+	5	X+		2.5	X+	2.5	X+
16SW12	2/21/95	9:45	Primary		5	U+J	5	U+J	5	U+J	5	U+J	5	U+JX+		2.5	U+J	5	U+J
16SW13	2/21/95	10:00	Primary		5	U+J	5	U+J	5	U+J	5	U+J	5	U+JX+		2.5	U+J	5	U+J
16SW14	2/21/95	14:50	Primary		5	X+	5	X+	5	X+	5	X+	5	X+		2.5	X+	5	X+
16SW14	2/21/95	14:50	Duplicate 1		5	X+	5	X+	5	X+	5	X+	5	X+		2.5	X+	5	X+
16SW14-A			Avg.		5		5		5		5		5			2.5		5	
16SW15	3/16/95	15:15	Primary		5	X+	5	X+	5	X+	5	X+	5	X+		2.5	X+	5	X+
16SW16	3/16/95	16:50	Primary		5	X+	5	X+	5	X+	5	X+	5	X+		2.5	X+	5	X+
16SW17	3/6/95	14:45	Primary		5	X+	5	X+	5	X+	5	X+	5	X+		2.5	X+	5	X+
16SW17	3/6/95	14:45	Duplicate 1		5	X+	5	X+	5	X+	5	X+	5	X+		2.5	X+	5	X+
16SW17-A			Avg.		5		5		5		5		5			2.5		5	
16SW18	3/6/95	11:00	Primary		5	X+	5	X+	5	X+	5	X+	5	X+		2.5	X+	5	X+
16SW19	3/6/95	10:00	Primary		5	X+	5	X+	5	X+	5	X+	5	X+		2.5	X+	5	X+
16SW20	3/6/95	12:45	Primary		5	X+	5	X+	5	X+	5	X+	5	X+		2.5	X+	5	X+
16SW21	5/10/95	14:55	Primary		5	X+	5	X+	5	X+	5	X+	5	X+		2.5	X+	5	X+

NATURAL LOGS

16SW04					1.609		1.609		1.609		1.609		1.609			0.916		0.916	
16SW04					1.609		1.609		1.609		1.609		1.609			0.916		1.609	
16SW05					1.609		1.609		1.609		1.609		1.609			0.916		0.916	
16SW12					1.609		1.609		1.609		1.609		1.609			0.916		1.609	
16SW13					1.609		1.609		1.609		1.609		1.609			0.916		1.609	
16SW14-A					1.609		1.609		1.609		1.609		1.609			0.916		1.609	
16SW15					1.609		1.609		1.609		1.609		1.609			0.916		1.609	
16SW16					1.609		1.609		1.609		1.609		1.609			0.916		1.609	
16SW17-A					1.609		1.609		1.609		1.609		1.609			0.916		1.609	
16SW18					1.609		1.609		1.609		1.609		1.609			0.916		1.609	
16SW19					1.609		1.609		1.609		1.609		1.609			0.916		1.609	
16SW20					1.609		1.609		1.609		1.609		1.609			0.916		1.609	
16SW21					1.609		1.609		1.609		1.609		1.609			0.916		1.609	

STATISTICS

Total No.			0		13		13		13		13		13			13		13	
Min.			0		5		5		5		5		5		0	2.5		2.5	
Max.			0		5		5		5		5		5		0	2.5		5	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Methacrylonitrile (ug/l)	Methyl bromide (ug/l)	Methyl chloride (ug/l)	Methyl ethyl ketone (ug/l)	Methyl iodide (ug/l)	Methyl isobutyl ketone (MIBK) (ug/l)	Methyl methacrylate (ug/l)	Methylene bromide (ug/l)	Methylene chloride (ug/l)
Avg.(log transformed)				#DIV/0!	1.609	1.609	1.609	1.609	1.609	#DIV/0!	0.916	1.502
St. Dev.(log transformed)				#DIV/0!	0	0	0	0	0	#DIV/0!	0	0.26
H												
95th UCL of the Mean				#DIV/0!	4.997811	4.997811	4.997811	4.997811	4.997811	#DIV/0!	2.499273	4.64504

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Naphthalene (ug/l)	n-Butylbenzene (ug/l)	n-Propyl benzene (ug/l)	o-Chlorotoluene (ug/l)	o-Dichloro benzene (ug/l)	o-Xylene (ug/l)	p-Chlorotoluene (ug/l)	p-Cymene (ug/l)	p-Dichloro benzene (ug/l)	Pentachlo ethane (ug/l)
16SW04	4/14/93	0:00	Primary										
16SW04	2/21/95	9:00	Primary										
16SW05	5/13/93	0:00	Primary										
16SW12	2/21/95	9:45	Primary										
16SW13	2/21/95	10:00	Primary										
16SW14	2/21/95	14:50	Primary										
16SW14	2/21/95	14:50	Duplicate 1										
16SW14-A			Avg.										
16SW15	3/16/95	15:15	Primary										
16SW16	3/16/95	16:50	Primary										
16SW17	3/6/95	14:45	Primary										
16SW17	3/6/95	14:45	Duplicate 1										
16SW17-A			Avg.										
16SW18	3/6/95	11:00	Primary										
16SW19	3/6/95	10:00	Primary										
16SW20	3/6/95	12:45	Primary										
16SW21	5/10/95	14:55	Primary										
NATURAL LOGS													
16SW04													
16SW04													
16SW05													
16SW12													
16SW13													
16SW14-A													
16SW15													
16SW16													
16SW17-A													
16SW18													
16SW19													
16SW20													
16SW21													
STATISTICS													
Total No.				0	0	0	0	0	0	0	0	0	0
Min.				0	0	0	0	0	0	0	0	0	0
Max.				0	0	0	0	0	0	0	0	0	0

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Naphthalene (ug/l)	n-Butylbenzene (ug/l)	n-Propyl benzene (ug/l)	o-Chlorotoluene (ug/l)	o-Dichloro benzene (ug/l)	o-Xylene (ug/l)	p-Chlorotoluene (ug/l)	p-Cymene (ug/l)	p-Dichloro benzene (ug/l)	Pentachloethane (ug/l)
Avg.(log transformed)				#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!
St. Dev.(log transformed)				#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!
H													
95th UCL of the Mean				#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!	#DIV/0!

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	o sec-Butyl benzene (ug/l)		Styrene (ug/l)		tert-Butyl benzene (ug/l)		Tetrachloro ethylene (ug/l)		Toluene (ug/l)		Trans-1,3-Dichloropropen (ug/l)		trans-1,4-Dichloro-2-butene (ug/l)		Trichloro ethylene (ug/l)		Trichloro fluoromethane (ug/l)	
16SW04	4/14/93	0:00	Primary			2.5	X+			2.5	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+
16SW04	2/21/95	9:00	Primary			2.5	X+			2.5	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+
16SW05	5/13/93	0:00	Primary			2.5	X+			2.5	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+
16SW12	2/21/95	9:45	Primary			2.5	U+JX+			2.5	U+J	2.5	U+J	2.5	U+JX+			2.5	U+J	2.5	U+J
16SW13	2/21/95	10:00	Primary			2.5	U+JX+			2.5	U+J	2.5	U+J	2.5	U+JX+			2.5	U+J	2.5	U+J
16SW14	2/21/95	14:50	Primary			2.5	X+			2.5	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+
16SW14	2/21/95	14:50	Duplicate 1			2.5	X+			2.5	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+
16SW14-A			Avg.			2.5				2.5		2.5		2.5				2.5		2.5	
16SW15	3/16/95	15:15	Primary			2.5	X+			2.5	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+
16SW16	3/16/95	16:50	Primary			2.5	X+			2.5	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+
16SW17	3/6/95	14:45	Primary			2.5	X+			2.5	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+
16SW17	3/6/95	14:45	Duplicate 1			2.5	X+			2.5	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+
16SW17-A			Avg.			2.5				2.5		2.5		2.5				2.5		2.5	
16SW18	3/6/95	11:00	Primary			2.5	X+			2.5	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+
16SW19	3/6/95	10:00	Primary			2.5	X+			2.5	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+
16SW20	3/6/95	12:45	Primary			2.5	X+			2.5	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+
16SW21	5/10/95	14:55	Primary			2.5	X+			2.5	X+	2.5	X+	2.5	X+			2.5	X+	2.5	X+
NATURAL LOGS																					
16SW04						0.916				0.916		0.916		0.916				0.916		0.916	
16SW04						0.916				0.916		0.916		0.916				0.916		0.916	
16SW05						0.916				0.916		0.916		0.916				0.916		0.916	
16SW12						0.916				0.916		0.916		0.916				0.916		0.916	
16SW13						0.916				0.916		0.916		0.916				0.916		0.916	
16SW14-A						0.916				0.916		0.916		0.916				0.916		0.916	
16SW15						0.916				0.916		0.916		0.916				0.916		0.916	
16SW16						0.916				0.916		0.916		0.916				0.916		0.916	
16SW17-A						0.916				0.916		0.916		0.916				0.916		0.916	
16SW18						0.916				0.916		0.916		0.916				0.916		0.916	
16SW19						0.916				0.916		0.916		0.916				0.916		0.916	
16SW20						0.916				0.916		0.916		0.916				0.916		0.916	
16SW21						0.916				0.916		0.916		0.916				0.916		0.916	
STATISTICS																					
Total No.					0	13			0	13		13		13		0		13		13	
Min.					0	2.5			0	2.5		2.5		2.5		0		2.5		2.5	
Max.					0	2.5			0	2.5		2.5		2.5		0		2.5		2.5	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	o	sec-Butyl benzene (ug/l)	Styrene (ug/l)	tert-Butyl benzene (ug/l)	Tetrachloro ethylene (ug/l)	Toluene (ug/l)	Trans-1,3-Dichloropropen (ug/l)	trans-1,4-Dichloro-2-butene (ug/l)	Trichloro ethylene (ug/l)	Trichloro fluoromethane (ug/l)
Avg.(log transformed)					#DIV/0!	0.916	#DIV/0!	0.916	0.916	0.916	#DIV/0!	0.916	0.916
St. Dev.(log transformed)					#DIV/0!	0	#DIV/0!	0	0	0	#DIV/0!	0	0
H													
95th UCL of the Mean					#DIV/0!	2.499273	#DIV/0!	2.499273	2.499273	2.499273	#DIV/0!	2.499273	2.499273

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Vinyl Acetate (ug/l)		Vinyl chloride (ug/l)		Xylene (total) (ug/l)	
16SW04	4/14/93	0:00	Primary	5	X+	5	X+	2.5	X+
16SW04	2/21/95	9:00	Primary	5	X+	5	X+	2.5	X+
16SW05	5/13/93	0:00	Primary	5	X+	5	X+	2.5	X+
16SW12	2/21/95	9:45	Primary	5	U+J	5	U+J	2.5	U+JX+
16SW13	2/21/95	10:00	Primary	5	U+J	5	U+J	2.5	U+JX+
16SW14	2/21/95	14:50	Primary	5	X+	5	X+	2.5	X+
16SW14	2/21/95	14:50	Duplicate 1	5	X+	5	X+	2.5	X+
16SW14-A			Avg.	5		5		2.5	
16SW15	3/16/95	15:15	Primary	5	X+	5	X+	2.5	X+
16SW16	3/16/95	16:50	Primary	5	X+	5	X+	2.5	X+
16SW17	3/6/95	14:45	Primary	5	X+	5	X+	2.5	X+
16SW17	3/6/95	14:45	Duplicate 1	5	X+	5	X+	2.5	X+
16SW17-A			Avg.	5		5		2.5	
16SW18	3/6/95	11:00	Primary	5	X+	5	X+	2.5	X+
16SW19	3/6/95	10:00	Primary	5	X+	5	X+	2.5	X+
16SW20	3/6/95	12:45	Primary	5	X+	5	X+	2.5	X+
16SW21	5/10/95	14:55	Primary	5	X+	5	X+	2.5	X+

NATURAL LOGS

16SW04	1.609	1.609	0.916
16SW04	1.609	1.609	0.916
16SW05	1.609	1.609	0.916
16SW12	1.609	1.609	0.916
16SW13	1.609	1.609	0.916
16SW14-A	1.609	1.609	0.916
16SW15	1.609	1.609	0.916
16SW16	1.609	1.609	0.916
16SW17-A	1.609	1.609	0.916
16SW18	1.609	1.609	0.916
16SW19	1.609	1.609	0.916
16SW20	1.609	1.609	0.916
16SW21	1.609	1.609	0.916

STATISTICS

Total No.	13	13	13
Min.	5	5	2.5
Max.	5	5	2.5

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Surface Water Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Vinyl Acetate (ug/l)	Vinyl chloride (ug/l)	Xylene (total) (ug/l)
Avg.(log transformed)				1.609	1.609	0.916
St. Dev.(log transformed)				0	0	0
H						
95th UCL of the Mean				4.997811	4.997811	2.499273

Data Qualifier Notations

X+ = Non-Detect Result, one-half of the detection limit value presented.
U+ = Considered Non-Detect based on blank comparison, one-half of the detection limit value is presented.
J = Estimated laboratory result.

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Aluminum (mg/l)	Antimony (ug/l)		Arsenic (ug/l)	Barium (ug/l)	Beryllium (mg/l)	Cadmium (ug/l)	Calcium (mg/l)
122	6/2/93	0:00	Primary		< 30		17	1200		5.45	
122	6/14/95	13:42	Primary		< 10		13	945		< 5	
16EW01	2/21/96	10:55	Primary	< 0.1	< 12	UJ	< 5	101		< 5	
16EW01	2/21/96	16:48	Primary	< 0.1	< 12	UJ	< 5	111		< 5	
16EW01	2/21/96	20:35	Primary	0.12	< 12	UJ	< 5	99		< 5	
16EW01	2/24/96	12:35	Primary	< 0.1	< 12	UJ	< 5	54		< 5	
16EW01	2/25/96	23:55	Primary	< 0.1	< 12	UJ	< 5	40		< 5	
16EW01	2/25/96	23:55	Duplicate 1	< 0.1	< 12	UJ	< 5	38		< 5	
16EW01	2/27/96	12:10	Primary	< 0.1	< 12	UJ	< 5	28		< 5	
16EW01	3/16/96	11:30	Primary	0.82	< 12	UJ	< 5	44		< 5	
16EW01	3/17/96	16:10	Primary	< 0.1	< 12	UJ	< 5	23		< 5	
16EW01	3/19/96	11:30	Primary	< 0.1	< 12	UJ	< 5	23		< 5	
16EW01	10/29/97	10:00	Primary	< 1	< 5		< 50	< 1000	< 0.0025	< 4	290
16EW01	10/29/97	10:00	Duplicate 1	< 1	< 5		< 50	< 1000	< 0.0025	< 4	300
16EW02	2/23/96	9:40	Primary	0.23	< 12	UJ	< 5	45		< 5	
16EW02	2/23/96	14:00	Primary	26.8	< 12	UJ	7	192		< 5	
16EW02	2/28/96	12:20	Primary	0.26	< 12	UJ	< 5	50		< 5	
16EW02	2/29/96	11:10	Primary	0.2	< 12	UJ	< 5	36		< 5	
16EW02	2/29/96	21:25	Primary	1.39	< 12	UJ	< 5	36		< 5	
16EW02	3/13/96	11:35	Primary	1.45	< 12	UJ	< 5	50		< 5	
16EW02	3/14/96	12:00	Primary	0.15	< 12	UJ	< 5	31		< 5	
16EW02	3/14/96	12:00	Duplicate 1	0.28	< 12	UJ	< 5	34		< 5	
16EW02	3/16/96	12:00	Primary	0.11	< 12	UJ	< 5	32		< 5	
16EW02	3/19/96	12:00	Primary	< 0.1	< 12	UJ	< 5	28		< 5	
16EW02	10/29/97	10:30	Primary	< 1	< 5		< 50	< 1000	< 0.0025	< 4	370
16EW03	10/29/97	11:19	Primary	1.4	< 5		< 10	< 200	< 0.0005	1.8	150
16EW04	10/29/97	10:59	Primary	< 0.2	< 5		< 10	< 200	< 0.0005	< 0.8	74
16EW05	10/29/97	11:15	Primary	< 4	< 5		< 200	< 4000	< 0.01	< 16	490
16EW06	10/29/97	12:20	Primary	< 1	< 5		< 50	< 1000	< 0.0025	< 4	160
16EW07	10/29/97	11:54	Primary	< 0.2	< 5		< 10	< 200	< 0.0005	< 0.8	200
16EW08	10/29/97	11:30	Primary	< 0.2	< 5		< 10	570	< 0.0005	< 0.8	39
16PB02	3/18/95	16:50	Primary		< 10		< 5	1040		< 5	
16PB02	3/18/95	16:50	Duplicate 1		< 10		< 5	900		< 5	
16PB04	3/18/95	13:45	Primary		< 10		< 5	126.7		< 5	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Aluminum (mg/l)	Antimony (ug/l)	Arsenic (ug/l)	Barium (ug/l)	Beryllium (mg/l)	Cadmium (ug/l)	Calcium (mg/l)
16PB05	3/18/95	14:50	Primary		< 10	< 5	196		< 5	
16PB06	3/18/95	17:00	Primary		< 10	10	2550		< 5	
16PB07	3/19/95	14:20	Primary		< 10	< 5	552		8	
16PB08	3/21/95	9:45	Primary		< 10	12	703		< 5	
16PB09	3/18/95	15:30	Primary		< 10	15	1130		< 5	
16PB10	3/20/95	14:30	Primary		< 10	12	323		< 5	
16PB11	3/20/95	15:45	Primary		< 10	6	2240		< 5	
16PB13	3/21/95	10:55	Primary		< 10	6	352		< 5	
16PB14	3/21/95	10:20	Primary		< 20	30	684		< 5	
16PB16	3/20/95	16:40	Primary		< 10	33	5130		< 5	
16PB17	3/20/95	16:30	Primary		< 10	31	1300		< 5	
16PB19	3/21/95	15:00	Primary		< 10	< 5	383		< 5	
16PB20	3/21/95	12:45	Primary		< 10	17	652		< 5	
16PB21	3/20/95	17:15	Primary		< 10	26	1160		< 5	
16WW01	6/10/93	0:00	Primary		< 30	< 10	30.3		< 5	
16WW01	6/14/95	14:18	Primary		< 20	< 5	17		< 5	
16WW02	6/10/93	0:00	Primary		< 30	< 10	213		< 5	
16WW02	6/13/95	16:30	Primary		< 10	< 10	662		< 5	
16WW03	6/8/93	0:00	Primary		< 30	< 10	582		< 5	
16WW03	6/14/95	16:04	Primary		< 20	< 10	390		< 25	
16WW04	6/8/93	0:00	Primary		< 30	< 10	44		< 5	
16WW04	6/15/95	10:58	Primary		< 200	< 10	< 50		< 25	
16WW04	6/15/95	10:58	Duplicate 1		< 100	< 10	< 50		< 25	
16WW05	6/11/93	0:00	Primary		< 30	< 10	544		< 5	
16WW05	6/11/93	0:00	Duplicate 1		< 30	< 10	539		< 5	
16WW05	6/14/95	16:48	Primary		< 10	< 5	477		< 5	
16WW05	10/22/97	17:10	Primary	0.94	< 5	< 20	480	< 0.001	< 1.6	110
16WW06	6/11/93	0:00	Primary		< 30	< 10	825		< 5	
16WW06	6/14/95	16:42	Primary		< 10	< 5	778		< 5	
16WW06	10/22/97	14:50	Primary	0.23	< 5	< 10	750	< 0.0005	< 0.8	66
16WW07	6/11/93	0:00	Primary		< 30	< 10	285		< 5	
16WW07	6/14/95	12:00	Primary		< 10	< 5	487		< 5	
16WW08	6/11/93	0:00	Primary		< 30	< 10	60.8		< 5	
16WW08	6/14/95	11:40	Primary		< 10	< 5	43		< 5	
16WW08	6/14/95	11:40	Duplicate 1		< 10	< 5	43		< 5	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Aluminum (mg/l)	Antimony (ug/l)	Arsenic (ug/l)	Barium (ug/l)	Beryllium (mg/l)	Cadmium (ug/l)	Calcium (mg/l)
16WW09	6/10/93	0:00	Primary		< 30	< 10	221		< 5	
16WW09	6/15/95	12:15	Primary		< 20	7	9900		< 10	
16WW10	6/9/93	0:00	Primary		< 30	< 10	421		< 5	
16WW10	6/15/95	15:40	Primary		< 10	< 10	302		< 5	
16WW11	6/9/93	0:00	Primary		< 30	< 10	49.1		< 5	
16WW11	6/9/93	0:00	Duplicate 1		< 30	< 10	51.4		< 5	
16WW11	6/16/95	11:42	Primary		< 20	34 J	42 J		< 5	
16WW12	6/13/95	17:45	Primary		< 10	< 5	48		< 5	
16WW12	10/27/97	13:39	Primary	9.9	< 5	< 50	< 1000	< 0.0025	< 4	270
16WW12	10/27/97	13:39	Duplicate 1	12	< 5	< 50	< 1000	< 0.0025	< 4	270
16WW13	6/13/95	16:55	Primary		< 10	< 5	66		< 5	
16WW13	10/27/97	16:05	Primary	4.6	< 5	< 100	< 2000	< 0.005	< 8	470
16WW14	6/13/95	16:45	Primary		< 10	< 5	176		< 5	
16WW14	10/24/97	13:55	Primary	3.3	< 5	< 50	< 1000	< 0.0025	< 4	170
16WW15	6/22/95	10:10	Primary		< 10	20 J	32 J		< 5	
16WW15	10/28/97	15:25	Primary	0.75	< 5	< 10	< 200	< 0.0005	< 0.8	< 5
16WW16	6/15/95	10:40	Primary		< 10	< 5	60		< 5	
16WW16	10/27/97	16:00	Primary	17	< 5	< 50	< 1000	< 0.0025	< 4	180
16WW17	6/16/95	15:07	Primary		< 10	17 J	6500 J		< 5	
16WW17	10/27/97	16:44	Primary	0.21	< 5	< 10	< 200	< 0.0005	< 0.8	< 5
16WW18	6/15/95	14:08	Primary		< 10	< 5	60		< 5	
16WW18	10/28/97	15:09	Primary	0.27	< 5	< 10	< 200	< 0.0005	< 0.8	< 5
16WW19	10/24/97	11:15	Primary	3.1	< 5	< 10	< 200	< 0.0005	< 0.8	13
16WW20	10/24/97	16:15	Primary	40	< 5	23	470	0.0074	2.3	20
16WW21	10/28/97	14:45	Primary	2.1	< 5	< 10	< 200	< 0.0005	< 0.8	16
16WW22	10/24/97	13:44	Primary	8.4	< 5	< 50	< 1000	< 0.0025	< 4	310
16WW23	10/23/97	10:39	Primary	0.57	< 5	< 10	1400	< 0.0005	< 0.8	140
16WW24	10/23/97	12:49	Primary	46	< 5	< 10	2300	0.0023	< 0.8	34
16WW25	10/23/97	10:39	Primary	< 0.2	< 5	< 10	< 200	< 0.0005	< 0.8	170
16WW26	10/23/97	12:49	Primary	9.3	< 5	11	220	0.0006	< 0.8	5.7
16WW27	10/21/97	10:50	Primary	10	< 5	< 10	930	0.0009	< 0.8	48
16WW28	10/21/97	12:55	Primary	1.9	< 5	< 10	470	< 0.0005	< 0.8	21
16WW29	10/21/97	10:55	Primary	< 0.2	< 5	< 10	210	< 0.0005	< 0.8	66
16WW30	10/21/97	12:40	Primary	< 0.2	< 5	< 10	770	< 0.0005	< 0.8	42
16WW31	10/22/97	10:09	Primary	< 0.4	< 5	< 20	< 400	< 0.001	< 1.6	100

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Aluminum (mg/l)	Antimony (ug/l)	Arsenic (ug/l)	Barium (ug/l)	Beryllium (mg/l)	Cadmium (ug/l)	Calcium (mg/l)
16WW31	10/22/97	10:09	Duplicate 1	< 0.4	< 5	< 20	< 400	< 0.001	< 1.6	100
16WW32	10/22/97	12:40	Primary	3.6	< 5	< 50	1300	< 0.0025	< 4	240
16WW33	10/22/97	10:00	Primary	2.7	< 5	< 20	< 400	< 0.001	< 1.6	130
16WW34	10/22/97	12:20	Primary	67	< 5	14	1800	0.0037	1.1	92
16WW35	10/24/97	11:10	Primary	< 5	< 5	< 250	< 5000	< 0.0125	< 20	420
16WW36	10/27/97	13:50	Primary	7.9	< 5	< 100	< 2000	< 0.005	< 8	230
16WW37	10/21/97	16:39	Primary	7.7	< 5	< 100	< 2000	< 0.005	< 8	430
16WW37	10/21/97	16:39	Duplicate 1	6.9	< 5	< 100	< 2000	< 0.005	< 8	440
16WW38	10/22/97	16:09	Primary	11	< 5	< 50	< 1000	< 0.0025	< 4	170

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Chromium (ug/l)	Cobalt (mg/l)	Copper (mg/l)	Iron (mg/l)		Lead (ug/l)		Magnesium (mg/l)	Manganese (mg/l)
122	6/2/93	0:00	Primary	< 10					3.67			
122	6/14/95	13:42	Primary	< 10					< 2	UJ		
16EW01	2/21/96	10:55	Primary	< 10		0.034	0.69		< 60		319	0.747
16EW01	2/21/96	16:48	Primary	< 10		0.026	0.17		< 60		288	0.637
16EW01	2/21/96	20:35	Primary	< 10		0.03	0.26		< 60		318	0.571
16EW01	2/24/96	12:35	Primary	< 10		0.032	0.2		< 60		333	0.672
16EW01	2/25/96	23:55	Primary	< 10		0.03	0.14		< 60		331	0.538
16EW01	2/25/96	23:55	Duplicate 1	< 10		0.028	0.15		< 60		312	0.507
16EW01	2/27/96	12:10	Primary	< 10		0.038	0.13	U	< 60		334	0.537
16EW01	3/16/96	11:30	Primary	< 10		0.045	6.21		< 60		349	1.03
16EW01	3/17/96	16:10	Primary	< 10		0.033	< 0.1		< 60		319	0.558
16EW01	3/19/96	11:30	Primary	< 10		0.029	< 0.1		< 60		293	0.506
16EW01	10/29/97	10:00	Primary	< 50	< 0.25	< 0.125	< 0.5		< 15		220	0.38
16EW01	10/29/97	10:00	Duplicate 1	< 50	< 0.25	< 0.125	< 0.5		< 15		230	0.403
16EW02	2/23/96	9:40	Primary	< 10		0.031	12.8		< 60		443	1.43
16EW02	2/23/96	14:00	Primary	47		0.043	28.1		< 60		374	1.68
16EW02	2/28/96	12:20	Primary	< 10		0.021	< 0.1		< 60		360	1.59
16EW02	2/29/96	11:10	Primary	< 10		0.037	4.94		< 60		473	1.41
16EW02	2/29/96	21:25	Primary	10		0.031	2.59		< 60		356	1.35
16EW02	3/13/96	11:35	Primary	< 10		0.051	2.39		< 60		322	1.39
16EW02	3/14/96	12:00	Primary	< 10		0.366	X 0.71		< 60		320	1.11
16EW02	3/14/96	12:00	Duplicate 1	< 10		0.13	X 0.92		< 60		347	1.2
16EW02	3/16/96	12:00	Primary	< 10		0.484	0.37		< 60		376	1.23
16EW02	3/19/96	12:00	Primary	< 10		0.071	0.2		< 60		359	1.13
16EW02	10/29/97	10:30	Primary	< 50	< 0.25	< 0.125	< 0.5		< 15		300	1.35
16EW03	10/29/97	11:19	Primary	586	< 0.05	0.042	3.9		< 3		120	2.79
16EW04	10/29/97	10:59	Primary	< 10	< 0.05	< 0.025	0.27		< 3		73	0.713
16EW05	10/29/97	11:15	Primary	< 200	< 1	< 0.5	24		< 60		400	5.55
16EW06	10/29/97	12:20	Primary	< 50	< 0.25	< 0.125	40		< 15		120	3.22
16EW07	10/29/97	11:54	Primary	< 10	< 0.05	< 0.025	110		< 3		120	5.53
16EW08	10/29/97	11:30	Primary	12	< 0.05	< 0.025	37		< 3		26	1.4
16PB02	3/18/95	16:50	Primary	64					11			
16PB02	3/18/95	16:50	Duplicate 1	36					8			
16PB04	3/18/95	13:45	Primary	< 10					< 20			

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Chromium (ug/l)	Cobalt (mg/l)	Copper (mg/l)	Iron (mg/l)	Lead (ug/l)		Magnesium (mg/l)	Manganese (mg/l)
16PB05	3/18/95	14:50	Primary	51				40			
16PB06	3/18/95	17:00	Primary	147				100			
16PB07	3/19/95	14:20	Primary	40				6			
16PB08	3/21/95	9:45	Primary	156				30			
16PB09	3/18/95	15:30	Primary	232				140			
16PB10	3/20/95	14:30	Primary	99				140			
16PB11	3/20/95	15:45	Primary	19				8			
16PB13	3/21/95	10:55	Primary	27				< 20			
16PB14	3/21/95	10:20	Primary	259				60			
16PB16	3/20/95	16:40	Primary	646				200			
16PB17	3/20/95	16:30	Primary	121				60			
16PB19	3/21/95	15:00	Primary	57				8			
16PB20	3/21/95	12:45	Primary	82				< 2			
16PB21	3/20/95	17:15	Primary	308				30			
16WW01	6/10/93	0:00	Primary	< 10				< 2			
16WW01	6/14/95	14:18	Primary	< 10				< 4	UJ		
16WW02	6/10/93	0:00	Primary	< 10				< 2			
16WW02	6/13/95	16:30	Primary	11				< 4	UJ		
16WW03	6/8/93	0:00	Primary	15.9				< 2			
16WW03	6/14/95	16:04	Primary	< 50				< 40	UJ		
16WW04	6/8/93	0:00	Primary	< 10				< 2			
16WW04	6/15/95	10:58	Primary	< 50				< 40	UJ		
16WW04	6/15/95	10:58	Duplicate 1	< 50				< 40			
16WW05	6/11/93	0:00	Primary	< 10				6			
16WW05	6/11/93	0:00	Duplicate 1	< 10				6.13			
16WW05	6/14/95	16:48	Primary	< 10				< 4	UJ		
16WW05	10/22/97	17:10	Primary	< 20	< 0.1	< 0.05	1.1	< 6		92	< 0.03
16WW06	6/11/93	0:00	Primary	< 10				< 2			
16WW06	6/14/95	16:42	Primary	< 10				< 4	UJ		
16WW06	10/22/97	14:50	Primary	< 10	< 0.05	< 0.025	8	< 3		48	1
16WW07	6/11/93	0:00	Primary	< 10				11.3			
16WW07	6/14/95	12:00	Primary	< 10				< 20	UJ		
16WW08	6/11/93	0:00	Primary	< 10				< 2			
16WW08	6/14/95	11:40	Primary	< 10				< 4	UJ		
16WW08	6/14/95	11:40	Duplicate 1	< 10				< 4			

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Chromium (ug/l)	Cobalt (mg/l)	Copper (mg/l)	Iron (mg/l)	Lead (ug/l)		Magnesium (mg/l)	Manganese (mg/l)
16WW09	6/10/93	0:00	Primary	< 10				3.24			
16WW09	6/15/95	12:15	Primary	< 20				< 40	UJ		
16WW10	6/9/93	0:00	Primary	< 10				< 2			
16WW10	6/15/95	15:40	Primary	< 10				< 40	UJ		
16WW11	6/9/93	0:00	Primary	< 10				< 2			
16WW11	6/9/93	0:00	Duplicate 1	< 10				< 2			
16WW11	6/16/95	11:42	Primary	< 10				< 4			
16WW12	6/13/95	17:45	Primary	10				< 4	UJ		
16WW12	10/27/97	13:39	Primary	1490	< 0.25	< 0.125	18	< 15		210	0.791
16WW12	10/27/97	13:39	Duplicate 1	1820	< 0.25	< 0.125	23	< 15		210	0.785
16WW13	6/13/95	16:55	Primary	32				< 40	UJ		
16WW13	10/27/97	16:05	Primary	< 100	1.1	< 0.25	29	< 30		460	29.8
16WW14	6/13/95	16:45	Primary	12				< 4	UJ		
16WW14	10/24/97	13:55	Primary	3090	< 0.25	< 0.125	16	< 15		120	0.483
16WW15	6/22/95	10:10	Primary	12				30			
16WW15	10/28/97	15:25	Primary	< 10	< 0.05	< 0.025	1.1	9		< 5	0.018
16WW16	6/15/95	10:40	Primary	29				< 40	UJ		
16WW16	10/27/97	16:00	Primary	324	< 0.25	< 0.125	21	< 15		140	0.831
16WW17	6/16/95	15:07	Primary	27				20			
16WW17	10/27/97	16:44	Primary	< 10	< 0.05	< 0.025	0.39	7		< 5	< 0.015
16WW18	6/15/95	14:08	Primary	51				< 20	UJ		
16WW18	10/28/97	15:09	Primary	< 10	< 0.05	< 0.025	0.41	< 3		< 5	0.015
16WW19	10/24/97	11:15	Primary	60	< 0.05	< 0.025	3.1	6		< 5	0.059
16WW20	10/24/97	16:15	Primary	129	0.059	0.062	41	52		15	0.747
16WW21	10/28/97	14:45	Primary	18	< 0.05	< 0.025	1.9	< 3		8.2	0.25
16WW22	10/24/97	13:44	Primary	69	< 0.25	< 0.125	11	< 15		220	0.167
16WW23	10/23/97	10:39	Primary	37	< 0.05	< 0.025	180	< 3		81	11.9
16WW24	10/23/97	12:49	Primary	5220	0.197	0.051	160	37		32	4.77
16WW25	10/23/97	10:39	Primary	19	< 0.05	< 0.025	110	< 3		99	3.19
16WW26	10/23/97	12:49	Primary	114	< 0.05	< 0.025	14	8		6.9	0.286
16WW27	10/21/97	10:50	Primary	66	< 0.05	< 0.025	80	11		29	2.62
16WW28	10/21/97	12:55	Primary	37	< 0.05	< 0.025	37	3		16	1.16
16WW29	10/21/97	10:55	Primary	< 10	< 0.05	< 0.025	32	< 3		39	1.49
16WW30	10/21/97	12:40	Primary	60	< 0.05	< 0.025	28	< 3		31	1.47
16WW31	10/22/97	10:09	Primary	40	< 0.1	< 0.05	0.7	< 6		69	0.527

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Chromium (ug/l)	Cobalt (mg/l)	Copper (mg/l)	Iron (mg/l)	Lead (ug/l)	Magnesium (mg/l)	Manganese (mg/l)
16WW31	10/22/97	10:09	Duplicate 1	41	< 0.1	< 0.05	0.66	< 6	68	0.499
16WW32	10/22/97	12:40	Primary	369	< 0.25	< 0.125	7	< 15	130	0.196
16WW33	10/22/97	10:00	Primary	82	< 0.1	< 0.05	4.3	< 6	86	5.08
16WW34	10/22/97	12:20	Primary	2850	0.053	0.082	120	57	74	1.03
16WW35	10/24/97	11:10	Primary	< 250	< 1.25	< 0.625	15	< 75	430	7.35
16WW36	10/27/97	13:50	Primary	< 100	< 0.5	< 0.25	11	< 30	200	2.9
16WW37	10/21/97	16:39	Primary	201	< 0.5	< 0.25	9.9	< 30	310	5.6
16WW37	10/21/97	16:39	Duplicate 1	251	< 0.5	< 0.25	10	< 30	320	5.7
16WW38	10/22/97	16:09	Primary	242	< 0.25	< 0.125	17	< 15	130	0.229

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Mercury (ug/l)		Nickel (ug/l)	Potassium (mg/l)	Selenium (ug/l)	Silver (ug/l)	Sodium (mg/l)	Strontium (mg/l)
122	6/2/93	0:00	Primary	< 2		< 50		< 10	< 70		
122	6/14/95	13:42	Primary	< 0.2		< 40		< 10	< 10		
16EW01	2/21/96	10:55	Primary	< 0.2		32		< 50	< 10		5.8
16EW01	2/21/96	16:48	Primary	< 0.2		21		< 50	< 10		5.3
16EW01	2/21/96	20:35	Primary	< 0.2		18		< 50	< 10		5.83
16EW01	2/24/96	12:35	Primary	< 0.2		27		< 50	< 10		6.1
16EW01	2/25/96	23:55	Primary	< 0.2		15		< 50	< 10		6.09
16EW01	2/25/96	23:55	Duplicate 1	< 0.2		16		< 50	< 10		5.76
16EW01	2/27/96	12:10	Primary	< 0.2		18		< 50	< 10		6.12
16EW01	3/16/96	11:30	Primary	0.2		47		< 50	< 10		6.89
16EW01	3/17/96	16:10	Primary	0.3		< 40		< 50	< 10		6.04
16EW01	3/19/96	11:30	Primary	< 0.2		< 40		< 50	< 10		5.7
16EW01	10/29/97	10:00	Primary	< 0.2		< 200	< 25	< 25	< 50	730	5.49
16EW01	10/29/97	10:00	Duplicate 1	< 0.2		< 200	< 25	< 25	< 50	770	5.84
16EW02	2/23/96	9:40	Primary	0.27	J	59		< 50	< 10		7.93
16EW02	2/23/96	14:00	Primary	0.23	J	79		< 50	< 10		6.56
16EW02	2/28/96	12:20	Primary	< 0.2		61		< 50	< 10		6.3
16EW02	2/29/96	11:10	Primary	0.3	J	54		< 50	< 10		8.5
16EW02	2/29/96	21:25	Primary	0.2	J	45		< 50	< 10		6.26
16EW02	3/13/96	11:35	Primary	0.2		52		< 50	< 10		5.8
16EW02	3/14/96	12:00	Primary	0.2		< 40		< 50	< 10		5.86
16EW02	3/14/96	12:00	Duplicate 1	0.2		< 40		< 50	< 10		6.4
16EW02	3/16/96	12:00	Primary	0.2		49		< 50	< 10		7
16EW02	3/19/96	12:00	Primary	0.2		< 40		< 50	< 10		6.59
16EW02	10/29/97	10:30	Primary	< 0.2		< 200	< 25	< 25	< 50	720	6.92
16EW03	10/29/97	11:19	Primary	< 0.2		457	< 5	< 5	14	200	3.3
16EW04	10/29/97	10:59	Primary	< 0.2		< 40	< 5	< 5	< 10	240	1.73
16EW05	10/29/97	11:15	Primary	< 0.2		< 800	< 100	< 100	< 200	2100	9.9
16EW06	10/29/97	12:20	Primary	< 0.2		< 200	< 25	< 25	< 50	530	3.61
16EW07	10/29/97	11:54	Primary	< 0.2		< 40	< 5	10	< 10	200	5.99
16EW08	10/29/97	11:30	Primary	< 0.2		< 40	< 5	8	< 10	100	1.11
16PB02	3/18/95	16:50	Primary	< 0.2		< 40		< 100	< 10		
16PB02	3/18/95	16:50	Duplicate 1	< 0.2		< 40		< 100	< 10		
16PB04	3/18/95	13:45	Primary	< 0.2		< 40		< 5	< 10		

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Mercury (ug/l)	Nickel (ug/l)	Potassium (mg/l)	Selenium (ug/l)	Silver (ug/l)	Sodium (mg/l)	Strontium (mg/l)
16PB05	3/18/95	14:50	Primary	< 0.2	62		< 10	< 10		
16PB06	3/18/95	17:00	Primary	0.3	78		< 100	< 10		
16PB07	3/19/95	14:20	Primary	< 0.2	56		< 10	< 10		
16PB08	3/21/95	9:45	Primary	0.2	140		< 50	< 10		
16PB09	3/18/95	15:30	Primary	1.6	267		< 10	< 10		
16PB10	3/20/95	14:30	Primary	< 0.2	200		< 10	< 10		
16PB11	3/20/95	15:45	Primary	< 0.2	< 40		< 10	< 10		
16PB13	3/21/95	10:55	Primary	< 0.2	< 40		< 100	< 10		
16PB14	3/21/95	10:20	Primary	0.5	270		< 100	< 10		
16PB16	3/20/95	16:40	Primary	1.2	350		< 50	< 10		
16PB17	3/20/95	16:30	Primary	0.3	88		8	< 10		
16PB19	3/21/95	15:00	Primary	0.2	54		< 5	< 10		
16PB20	3/21/95	12:45	Primary	< 0.2	130		< 100	< 10		
16PB21	3/20/95	17:15	Primary	0.4	360		< 100	< 10		
16WW01	6/10/93	0:00	Primary	< 2	< 50		< 10	< 70		
16WW01	6/14/95	14:18	Primary	< 0.2	50		< 10	< 10		
16WW02	6/10/93	0:00	Primary	< 2	< 50		< 10	< 70		
16WW02	6/13/95	16:30	Primary	< 0.2	70		< 100	< 10		
16WW03	6/8/93	0:00	Primary	< 2	142		< 10	< 70		
16WW03	6/14/95	16:04	Primary	0.86	< 200		< 100	< 50		
16WW04	6/8/93	0:00	Primary	< 2	< 50		15.6	< 70		
16WW04	6/15/95	10:58	Primary	0.3	< 200		< 100	< 50		
16WW04	6/15/95	10:58	Duplicate 1	0.3	< 200		< 100	< 50		
16WW05	6/11/93	0:00	Primary	< 2	< 50		< 10	< 70		
16WW05	6/11/93	0:00	Duplicate 1	< 2	< 50		< 10	< 70		
16WW05	6/14/95	16:48	Primary	< 0.2	< 40		< 10	< 10		
16WW05	10/22/97	17:10	Primary	< 0.2	< 80	< 10	10	< 20	390	2.55
16WW06	6/11/93	0:00	Primary	< 2	< 50		< 10	< 70		
16WW06	6/14/95	16:42	Primary	< 0.2	< 40		< 10	< 10		
16WW06	10/22/97	14:50	Primary	< 0.2	< 40	< 5	< 5	< 10	170	2.32
16WW07	6/11/93	0:00	Primary	< 2	< 50		< 10	< 70		
16WW07	6/14/95	12:00	Primary	< 0.2	< 40		< 10	< 10		
16WW08	6/11/93	0:00	Primary	< 2	< 50		< 10	< 70		
16WW08	6/14/95	11:40	Primary	< 0.2	< 40		< 10	< 10		
16WW08	6/14/95	11:40	Duplicate 1	< 0.2	< 40		< 10	< 10		

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Mercury (ug/l)	Nickel (ug/l)	Potassium (mg/l)	Selenium (ug/l)	Silver (ug/l)	Sodium (mg/l)	Strontium (mg/l)
16WW09	6/10/93	0:00	Primary	< 2	< 50		< 10	< 70		
16WW09	6/15/95	12:15	Primary	< 0.2	< 80		< 10	< 20		
16WW10	6/9/93	0:00	Primary	< 2	< 50		< 10	< 70		
16WW10	6/15/95	15:40	Primary	< 0.2	< 40		< 10	< 10		
16WW11	6/9/93	0:00	Primary	< 2	< 50		< 10	< 70		
16WW11	6/9/93	0:00	Duplicate 1	< 2	< 50		< 10	< 70		
16WW11	6/16/95	11:42	Primary	< 0.2	< 40		< 100	< 10		
16WW12	6/13/95	17:45	Primary	< 0.2	< 40		< 100	< 10		
16WW12	10/27/97	13:39	Primary	< 0.2	< 200	< 25	< 25	< 50	540	5.33
16WW12	10/27/97	13:39	Duplicate 1	< 0.2	< 200	< 25	< 25	< 50	530	5.24
16WW13	6/13/95	16:55	Primary	< 0.2	360		< 100	< 10		
16WW13	10/27/97	16:05	Primary	< 0.2	484	< 50	< 50	< 100	860	10.4
16WW14	6/13/95	16:45	Primary	< 0.2	< 40		< 10	< 10		
16WW14	10/24/97	13:55	Primary	< 0.2	1630	< 25	< 25	67	520	3.05
16WW15	6/22/95	10:10	Primary	< 0.2	< 40		< 50	< 10		
16WW15	10/28/97	15:25	Primary	< 0.2	< 40	< 5	< 5	< 10	230	0.058
16WW16	6/15/95	10:40	Primary	< 0.2	42		< 10	< 10		
16WW16	10/27/97	16:00	Primary	< 0.2	< 200	< 25	< 25	< 50	580	2.93
16WW17	6/16/95	15:07	Primary	< 0.2	< 40		< 5	< 10		
16WW17	10/27/97	16:44	Primary	< 0.2	< 40	< 5	< 5	< 10	190	< 0.05
16WW18	6/15/95	14:08	Primary	< 0.2	58		< 10	< 10		
16WW18	10/28/97	15:09	Primary	< 0.2	< 40	< 5	< 5	< 10	220	< 0.05
16WW19	10/24/97	11:15	Primary	< 0.2	57	< 5	< 5	< 10	150	0.411
16WW20	10/24/97	16:15	Primary	< 0.2	155	15	10	< 10	53	0.989
16WW21	10/28/97	14:45	Primary	< 0.2	56	< 5	< 5	< 10	79	0.736
16WW22	10/24/97	13:44	Primary	< 0.2	< 200	< 25	< 25	< 50	810	5.58
16WW23	10/23/97	10:39	Primary	< 0.2	< 40	< 5	8	< 10	200	2.9
16WW24	10/23/97	12:49	Primary	< 0.2	751	10	8	114	220	0.92
16WW25	10/23/97	10:39	Primary	< 0.2	178	5.1	< 5	< 10	170	4.68
16WW26	10/23/97	12:49	Primary	< 0.2	170	< 5	< 5	< 10	16	0.122
16WW27	10/21/97	10:50	Primary	< 0.2	50	< 5	< 5	< 10	110	1.14
16WW28	10/21/97	12:55	Primary	< 0.2	< 40	< 5	< 5	< 10	100	0.492
16WW29	10/21/97	10:55	Primary	< 0.2	< 40	< 5	< 5	< 10	120	2.05
16WW30	10/21/97	12:40	Primary	< 0.2	175	< 5	< 5	< 10	140	1.12
16WW31	10/22/97	10:09	Primary	< 0.2	< 80	< 10	< 10	< 20	270	2.09

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Mercury (ug/l)	Nickel (ug/l)	Potassium (mg/l)	Selenium (ug/l)	Silver (ug/l)	Sodium (mg/l)	Strontium (mg/l)
16WW31	10/22/97	10:09	Duplicate 1	< 0.2	< 80	< 10	< 10	< 20	270	2.07
16WW32	10/22/97	12:40	Primary	< 0.2	< 200	< 25	< 25	< 50	430	4.32
16WW33	10/22/97	10:00	Primary	< 0.2	< 80	< 10	< 10	< 20	360	2.97
16WW34	10/22/97	12:20	Primary	< 0.2	699	15	7	52	250	1.96
16WW35	10/24/97	11:10	Primary	< 0.2	< 1000	< 120	< 125	< 250	4400	8.66
16WW36	10/27/97	13:50	Primary	< 0.2	< 400	< 50	< 50	< 100	1200	3.85
16WW37	10/21/97	16:39	Primary	< 0.2	< 400	< 50	< 50	< 100	1300	8.56
16WW37	10/21/97	16:39	Duplicate 1	< 0.2	< 400	< 50	< 50	< 100	1300	8.65
16WW38	10/22/97	16:09	Primary	< 0.2	< 200	< 25	< 25	< 50	570	3

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE		Thallium (ug/l)		Vanadium (mg/l)		Zinc (mg/l)	
122	6/2/93	0:00	Primary	<	10					
122	6/14/95	13:42	Primary	<	100					
16EW01	2/21/96	10:55	Primary	<	5000				0.08	
16EW01	2/21/96	16:48	Primary	<	5000				0.024	
16EW01	2/21/96	20:35	Primary	<	5000				0.025	
16EW01	2/24/96	12:35	Primary	<	5000				0.032	
16EW01	2/25/96	23:55	Primary	<	5000				0.024	
16EW01	2/25/96	23:55	Duplicate 1	<	5000				0.023	
16EW01	2/27/96	12:10	Primary	<	5000			<	0.02	
16EW01	3/16/96	11:30	Primary	<	5000				12.7	
16EW01	3/17/96	16:10	Primary	<	5000				0.073	
16EW01	3/19/96	11:30	Primary	<	5000				0.083	
16EW01	10/29/97	10:00	Primary	<	1	<	0.25	<	0.1	
16EW01	10/29/97	10:00	Duplicate 1	<	1	<	0.25	<	0.1	
16EW02	2/23/96	9:40	Primary	<	1000				0.256	
16EW02	2/23/96	14:00	Primary	<	1000				0.13	
16EW02	2/28/96	12:20	Primary	<	5000				0.344	
16EW02	2/29/96	11:10	Primary	<	5000				0.074	
16EW02	2/29/96	21:25	Primary	<	5000				0.05	
16EW02	3/13/96	11:35	Primary	<	5000				37	
16EW02	3/14/96	12:00	Primary	<	5000				0.481	X
16EW02	3/14/96	12:00	Duplicate 1	<	5000				0.14	X
16EW02	3/16/96	12:00	Primary	<	5000				0.859	
16EW02	3/19/96	12:00	Primary	<	5000				0.089	
16EW02	10/29/97	10:30	Primary	<	1	<	0.25	<	0.1	
16EW03	10/29/97	11:19	Primary	<	1	<	0.05		0.2	
16EW04	10/29/97	10:59	Primary	<	1	<	0.05		0.021	
16EW05	10/29/97	11:15	Primary	<	1	<	1	<	0.4	
16EW06	10/29/97	12:20	Primary	<	1	<	0.25		0.199	
16EW07	10/29/97	11:54	Primary	<	1	<	0.05	<	0.02	
16EW08	10/29/97	11:30	Primary	<	1	<	0.05	<	0.02	
16PB02	3/18/95	16:50	Primary	<	50					
16PB02	3/18/95	16:50	Duplicate 1	<	50					
16PB04	3/18/95	13:45	Primary	<	100					

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE	Thallium (ug/l)	Vanadium (mg/l)	Zinc (mg/l)
16PB05	3/18/95	14:50	Primary	< 100		
16PB06	3/18/95	17:00	Primary	< 50		
16PB07	3/19/95	14:20	Primary	< 100		
16PB08	3/21/95	9:45	Primary	< 50		
16PB09	3/18/95	15:30	Primary	< 50		
16PB10	3/20/95	14:30	Primary	< 50		
16PB11	3/20/95	15:45	Primary	< 50		
16PB13	3/21/95	10:55	Primary	< 50		
16PB14	3/21/95	10:20	Primary	< 100		
16PB16	3/20/95	16:40	Primary	< 50		
16PB17	3/20/95	16:30	Primary	< 50		
16PB19	3/21/95	15:00	Primary	< 5		
16PB20	3/21/95	12:45	Primary	< 50		
16PB21	3/20/95	17:15	Primary	< 50		
16WW01	6/10/93	0:00	Primary	< 10		
16WW01	6/14/95	14:18	Primary	< 1000		
16WW02	6/10/93	0:00	Primary	< 10		
16WW02	6/13/95	16:30	Primary	< 1000		
16WW03	6/8/93	0:00	Primary	< 10		
16WW03	6/14/95	16:04	Primary	< 5000		
16WW04	6/8/93	0:00	Primary	< 10		
16WW04	6/15/95	10:58	Primary	< 5000		
16WW04	6/15/95	10:58	Duplicate 1	< 5000		
16WW05	6/11/93	0:00	Primary	< 10		
16WW05	6/11/93	0:00	Duplicate 1	< 10		
16WW05	6/14/95	16:48	Primary	< 1000		
16WW05	10/22/97	17:10	Primary	< 1	< 0.1	< 0.04
16WW06	6/11/93	0:00	Primary	< 10		
16WW06	6/14/95	16:42	Primary	< 1000		
16WW06	10/22/97	14:50	Primary	< 1	< 0.05	< 0.02
16WW07	6/11/93	0:00	Primary	< 10		
16WW07	6/14/95	12:00	Primary	< 100		
16WW08	6/11/93	0:00	Primary	< 10		
16WW08	6/14/95	11:40	Primary	< 500		
16WW08	6/14/95	11:40	Duplicate 1	< 1000		

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE		Thallium (ug/l)		Vanadium (mg/l)		Zinc (mg/l)
16WW09	6/10/93	0:00	Primary	<	10				
16WW09	6/15/95	12:15	Primary	<	5000				
16WW10	6/9/93	0:00	Primary	<	10				
16WW10	6/15/95	15:40	Primary	<	1000				
16WW11	6/9/93	0:00	Primary	<	10				
16WW11	6/9/93	0:00	Duplicate 1	<	10				
16WW11	6/16/95	11:42	Primary	<	1000				
16WW12	6/13/95	17:45	Primary	<	1000				
16WW12	10/27/97	13:39	Primary	<	1	<	0.25	<	0.1
16WW12	10/27/97	13:39	Duplicate 1	<	1	<	0.25	<	0.1
16WW13	6/13/95	16:55	Primary	<	5000				
16WW13	10/27/97	16:05	Primary	<	1	<	0.5		0.224
16WW14	6/13/95	16:45	Primary	<	1000				
16WW14	10/24/97	13:55	Primary	<	1	<	0.25	<	0.1
16WW15	6/22/95	10:10	Primary	<	500				
16WW15	10/28/97	15:25	Primary	<	1	<	0.05		0.058
16WW16	6/15/95	10:40	Primary	<	5000				
16WW16	10/27/97	16:00	Primary	<	1	<	0.25	<	0.1
16WW17	6/16/95	15:07	Primary	<	500				
16WW17	10/27/97	16:44	Primary	<	1	<	0.05		0.025
16WW18	6/15/95	14:08	Primary	<	1000				
16WW18	10/28/97	15:09	Primary	<	1	<	0.05	<	0.02
16WW19	10/24/97	11:15	Primary	<	1	<	0.05	<	0.02
16WW20	10/24/97	16:15	Primary	<	1		0.128		0.2
16WW21	10/28/97	14:45	Primary	<	1	<	0.05	<	0.02
16WW22	10/24/97	13:44	Primary	<	1	<	0.25	<	0.1
16WW23	10/23/97	10:39	Primary	<	1	<	0.05	<	0.02
16WW24	10/23/97	12:49	Primary	<	1		0.097		0.12
16WW25	10/23/97	10:39	Primary	<	1	<	0.05	<	0.02
16WW26	10/23/97	12:49	Primary	<	1	<	0.05		0.064
16WW27	10/21/97	10:50	Primary	<	1	<	0.05		0.053
16WW28	10/21/97	12:55	Primary	<	1	<	0.05	<	0.02
16WW29	10/21/97	10:55	Primary	<	1	<	0.05	<	0.02
16WW30	10/21/97	12:40	Primary		12	<	0.05		0.032
16WW31	10/22/97	10:09	Primary	<	1	<	0.1	<	0.04

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Metals

SITE	DATE	TIME	RESULT TYPE		Thallium (ug/l)		Vanadium (mg/l)		Zinc (mg/l)
16WW31	10/22/97	10:09	Duplicate 1	<	1	<	0.1	<	0.04
16WW32	10/22/97	12:40	Primary	<	1	<	0.25	<	0.1
16WW33	10/22/97	10:00	Primary	<	1	<	0.1	<	0.04
16WW34	10/22/97	12:20	Primary	<	1		0.146		0.341
16WW35	10/24/97	11:10	Primary	<	1	<	1.25	<	0.5
16WW36	10/27/97	13:50	Primary	<	1	<	0.5	<	0.2
16WW37	10/21/97	16:39	Primary	<	1	<	0.5	<	0.2
16WW37	10/21/97	16:39	Duplicate 1	<	1	<	0.5	<	0.2
16WW38	10/22/97	16:09	Primary	<	1	<	0.25	<	0.1

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Explosives

SITE	DATE	TIME	RESULT TYPE	1,3-DNB (ug/l)		2,4-Dinitro toluene (ug/l)		2,6-Dinitro toluene (ug/l)		2-Amino-4,6 dinitrotoluene (ug/l)		4-Amino-2,6 dinitrotoluene (ug/l)		HMX (ug/l)		1,3-Dinitro benzene (ug/l)		3-Nitrotoluene (ug/l)	
122	6/2/93	0:00	Primary			< 0.02		< 0.31	UJ	< 0.035		< 0.06		< 0.8	UJ	< 0.11		< 0.25	
122	6/14/95	13:42	Primary			< 0.5		< 0.5		< 0.5		< 0.6		2.9		< 0.2		< 0.4	
16EW01	2/21/96	10:55	Primary			< 0.032	UJ	0.142	J	< 0.041	UJ	0.261		< 0.089		1.2		< 0.088	UJ
16EW01	2/21/96	16:48	Primary			0.041	UJ	0.191	J	< 0.041	UJ	0.597		< 0.089		1		< 0.088	UJ
16EW01	2/21/96	20:35	Primary			0.048	UJ	0.164	J	< 0.041	UJ	0.358		< 0.089		1.11		< 0.088	UJ
16EW01	2/24/96	12:35	Primary			< 0.032	UJ	0.263	J	< 0.041	UJ	< 0.042		< 0.089		1.56		< 0.088	UJ
16EW01	2/25/96	23:55	Primary			0.04	UJ	0.179	J	< 0.041	UJ	< 0.042		0.12		1.22		< 0.088	UJ
16EW01	2/25/96	23:55	Duplicate 1			0.035	UJ	0.18	J	< 0.041	UJ	< 0.042		< 0.089		1.11		< 0.088	UJ
16EW01	2/27/96	12:10	Primary			0.041	UJ	0.179	J	< 0.041	UJ	0.104		< 0.089		0.977		< 0.088	UJ
16EW01	3/16/96	11:30	Primary			< 0.032	UJ	< 0.04		< 0.041	UJ	0.059		< 0.089		0.546	J	< 0.088	UJ
16EW01	3/17/96	16:10	Primary			< 0.032	UJ	< 0.04		< 0.041	UJ	0.074		< 0.089		0.826	J	< 0.088	UJ
16EW01	3/19/96	11:30	Primary			< 0.032	UJ	< 0.04		< 0.041	UJ	0.061		< 0.089		0.904	J	< 0.088	UJ
16EW01	10/29/97	10:00	Primary	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.2	R			< 0.2	R
16EW01	10/29/97	10:00	Duplicate 1	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.2	R			< 0.2	R
16EW01	1/22/98	12:15	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW01	6/11/98	15:10	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW01	6/11/98	15:10	Duplicate 1			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW02	2/23/96	9:40	Primary			< 0.032	UJ	< 0.04	UJ	< 0.041	UJ	0.781		< 0.089		1.29		< 0.088	UJ
16EW02	2/23/96	14:00	Primary			< 0.032	UJ	< 0.04	UJ	< 0.041	UJ	0.875		< 0.089		1.13		< 0.088	UJ
16EW02	2/28/96	12:20	Primary			< 0.032	UJ	0.049	J	< 0.041	UJ	0.29		< 0.089		0.764		< 0.088	UJ
16EW02	2/29/96	11:10	Primary			0.034	UJ	0.062	J	< 0.041	UJ	0.431	J	< 0.089	UJ	1.25	J	< 0.088	UJ
16EW02	2/29/96	21:25	Primary			0.043	UJ	0.117		< 0.041	UJ	0.168		< 0.089		1.22		< 0.088	UJ
16EW02	3/16/96	12:00	Primary			< 0.032	UJ	< 0.04		< 0.041	UJ	0.23		< 0.089		0.725	J	< 0.088	UJ
16EW02	3/19/96	12:00	Primary			< 0.032	UJ	< 0.04		< 0.041	UJ	0.263		< 0.089		0.485	J	< 0.088	UJ
16EW02	10/29/97	10:30	Primary	< 0.1	UJ	< 0.1	UJ	< 0.1		< 0.1	UJ	< 0.1		< 0.2				< 0.2	UJ
16EW02	1/22/98	12:00	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW02	6/11/98	15:15	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW03	10/29/97	11:20	Primary	< 0.1	UJ	< 0.1	UJ	< 0.1		< 0.1	UJ	< 0.1		< 0.2				< 0.2	UJ
16EW03	1/22/98	11:55	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW03	6/11/98	15:20	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW04	10/29/97	11:00	Primary	< 0.1	UJ	< 0.1	UJ	< 0.1		< 0.1	UJ	< 0.1		< 0.2				< 0.2	UJ
16EW04	1/22/98	11:50	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW04	6/11/98	15:25	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW05	10/29/97	11:15	Primary	< 0.1	UJ	< 0.1	UJ	< 0.1		< 0.1	UJ	< 0.1		< 0.2				< 0.2	UJ
16EW05	1/22/98	11:35	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW05	6/11/98	15:30	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW06	10/29/97	12:20	Primary	< 0.1	UJ	< 0.1	UJ	< 0.1		< 0.1	UJ	< 0.1		< 0.2				< 0.2	UJ

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Explosives

SITE	DATE	TIME	RESULT TYPE	1,3-DNB (ug/l)		2,4-Dinitro toluene (ug/l)		2,6-Dinitro toluene (ug/l)		2-Amino-4,6 dinitrotoluene (ug/l)		4-Amino-2,6 dinitrotoluene (ug/l)		HMX (ug/l)		1,3-Dinitro benzene (ug/l)		3-Nitrotoluene (ug/l)	
16EW06	1/22/98	11:25	Primary			< 0.26		< 0.25		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW06	6/11/98	15:35	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW07	10/29/97	11:55	Primary	< 0.1	UJ	< 0.1	UJ	< 0.1		< 0.1	UJ	< 0.1		< 0.2				< 0.2	UJ
16EW07	1/22/98	11:45	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW07	6/11/98	15:40	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW08	10/29/97	11:30	Primary	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.2	R			< 0.2	R
16EW08	1/22/98	10:10	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW08	1/22/98	10:10	Duplicate 1			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16EW08	6/11/98	15:45	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16PB02	3/18/95	0:00	Primary			< 5				< 5				< 2		< 2		< 5	
16PB02	3/18/95	0:00	Duplicate 1			< 5				< 5				< 2		< 2		< 5	
16PB04	3/19/95	0:00	Primary			< 5				< 5				< 2		< 2		< 5	
16PB05	3/18/95	0:00	Primary			< 5				< 5				< 2		< 2		< 5	
16PB06	3/18/95	0:00	Primary			< 5				< 5				< 2		< 2		< 5	
16PB07	3/19/95	0:00	Primary			< 5				< 5				< 2		< 2		< 5	
16PB08	3/21/95	0:00	Primary			< 5				< 5				< 2		< 2		< 5	
16PB09	3/19/95	0:00	Primary			< 5				< 5				< 2		< 2		< 10	
16PB10	3/20/95	0:00	Primary			< 5				< 5				< 2		< 2		6.9	
16PB11	3/20/95	0:00	Primary			< 5				< 5				< 2		< 2		11	
16PB13	3/21/95	0:00	Primary			< 5				< 5				< 2		< 2		< 10	
16PB14	3/21/95	0:00	Primary			< 5				< 5				< 2		< 2		< 10	
16PB16	3/20/95	0:00	Primary			< 5				< 5				< 2		< 2		< 10	
16PB17	3/20/95	0:00	Primary			< 5				< 5				< 2		< 2		< 10	
16PB19	3/21/95	0:00	Primary			< 5				< 5				< 2		< 2		< 5	
16PB20	3/21/95	0:00	Primary			< 5				< 5				< 2		< 2		< 10	
16PB21	3/20/95	0:00	Primary			< 5				< 5				< 2		< 2		< 10	
16PB25	3/28/95	0:00	Primary			< 5				< 5				< 2		< 2		< 10	
16PB26	3/28/95	0:00	Primary			< 5				< 5				< 2		< 2		< 10	
16PB27	3/28/95	0:00	Primary			< 5				< 5				< 2		< 2		< 10	
16PB28	3/28/95	0:00	Primary			< 5				< 5				< 2		< 2		< 10	
16WW01	6/10/93	0:00	Primary			< 0.02		< 0.31	UJ	< 0.035		< 0.06		< 0.8	UJ	< 0.11	UJ	< 0.25	
16WW01	6/19/95	12:20	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW02	6/10/93	0:00	Primary			< 0.02		< 0.31	UJ	< 0.035		< 0.06		< 0.8	UJ	< 0.11	UJ	< 0.25	
16WW02	6/13/95	16:30	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW03	6/8/93	0:00	Primary			< 0.02		< 0.31	UJ	< 0.035		0.66	B	< 0.8	UJ	< 0.11	UJ	< 0.25	
16WW03	6/14/95	16:04	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW04	6/8/93	0:00	Primary			< 0.02		< 0.31	UJ	< 0.035		1	B	< 0.8	UJ	< 0.11	UJ	< 0.25	
16WW04	6/20/95	9:25	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Explosives

SITE	DATE	TIME	RESULT TYPE	1,3-DNB (ug/l)		2,4-Dinitro toluene (ug/l)		2,6-Dinitro toluene (ug/l)		2-Amino-4,6 dinitrotoluene (ug/l)		4-Amino-2,6 dinitrotoluene (ug/l)		HMX (ug/l)		1,3-Dinitro benzene (ug/l)		3-Nitrotoluene (ug/l)	
16WW04	6/20/95	9:25	Duplicate 1			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW04	6/29/95	12:55	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW05	6/11/93	0:00	Primary			< 0.02		< 0.31	UJ	< 0.035		< 0.06		< 0.8	UJ	< 0.11		< 0.25	
16WW05	6/11/93	0:00	Duplicate 1			< 0.02		< 0.31		< 0.035		< 0.06		< 0.8		< 0.11		< 0.25	
16WW05	6/19/95	13:07	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW05	10/22/97	17:10	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW06	6/11/93	0:00	Primary			< 0.020?		< 0.31?	UJ	< 0.035?		< 0.060?		< 0.80?	UJ	< 0.11?		< 0.25?	
16WW06	6/19/95	12:50	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW06	10/22/97	14:50	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW07	6/11/93	0:00	Primary			< 0.02		< 0.31	UJ	< 0.035		< 0.06		< 0.8	UJ	< 0.11		< 0.25	
16WW07	6/14/95	12:00	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW08	6/11/93	0:00	Primary			< 0.02		< 0.31	UJ	< 0.035		< 0.06		< 0.8	UJ	< 0.11		< 0.25	
16WW08	6/14/95	11:40	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW08	6/14/95	11:40	Duplicate 1			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW09	6/10/93	0:00	Primary			< 0.02		< 0.31	UJ	< 0.035		< 0.06		< 0.8	UJ	< 0.11		< 0.25	
16WW09	6/19/95	14:55	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW10	6/9/93	0:00	Primary			< 0.02		< 0.31	UJ	< 0.035		< 0.06		< 0.8	UJ	< 0.11	UJ	< 0.25	
16WW10	6/19/95	16:20	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW11	6/9/93	0:00	Primary			< 0.02		< 0.31	UJ	< 0.035		< 0.06		< 0.8	UJ	< 0.11	UJ	< 0.25	
16WW11	6/9/93	0:00	Duplicate 1			< 0.02		< 0.31		< 0.035		< 0.06		< 0.8		< 0.11		< 0.25	
16WW11	6/16/95	11:42	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW12	6/13/95	17:45	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW12	7/10/97	17:45	Primary			< 0.4		< 0.4		< 0.4		< 0.4		< 1		< 0.4		< 0.4	
16WW12	10/27/97	13:40	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW12	10/27/97	13:40	Duplicate 1	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW12	1/20/98	16:15	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW12	6/11/98	12:05	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW13	6/13/95	16:55	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW13	10/27/97	16:05	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW14	6/13/95	16:45	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW14	10/24/97	13:55	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW14	1/20/98	13:30	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW14	1/20/98	13:30	Duplicate 1			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW14	6/15/98	9:20	Primary			< 0.26	UJ	< 0.26	UJ	< 0.13		< 0.13		< 0.26		< 0.13	UJ	< 0.26	
16WW15	6/22/95	10:10	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW15	10/28/97	15:25	Primary	< 0.1	R	< 0.1		< 0.1		< 0.1	R	< 0.1		< 0.2				< 0.2	R
16WW16	6/20/95	11:00	Primary			< 0.5	UJ	< 0.5	UJ	< 0.5	UJ	< 0.6	UJ	< 0.4	UJ	< 0.2	UJ	< 0.4	UJ
16WW16	10/27/97	16:00	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Explosives

SITE	DATE	TIME	RESULT TYPE	1,3-DNB (ug/l)		2,4-Dinitro toluene (ug/l)		2,6-Dinitro toluene (ug/l)		2-Amino-4,6 dinitrotoluene (ug/l)		4-Amino-2,6 dinitrotoluene (ug/l)		HMX (ug/l)		1,3-Dinitro benzene (ug/l)		3-Nitrotoluene (ug/l)	
16WW16	1/21/98	12:15	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW16	6/15/98	11:15	Primary			< 0.26	UJ	< 0.26	UJ	< 0.13		< 0.13		< 0.26		< 0.13	UJ	< 0.26	
16WW17	6/16/95	15:07	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW17	10/27/97	16:45	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW18	6/20/95	14:02	Primary			< 0.5		< 0.5		< 0.5		< 0.6		< 0.4		< 0.2		< 0.4	
16WW18	10/28/97	15:10	Primary	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.2	R			< 0.2	R
16WW19	10/24/97	11:15	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW20	10/24/97	16:15	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW21	10/28/97	14:45	Primary	< 0.1	R	< 0.1		< 0.1		< 0.1	R	< 0.1		< 0.2				< 0.2	R
16WW22	7/10/97	17:50	Primary			< 0.4		< 0.4		< 0.4		< 0.4		< 1		< 0.4		< 0.4	
16WW22	10/24/97	13:45	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW22	1/20/98	14:50	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW22	6/11/98	10:15	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW23	10/23/97	10:40	Primary	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.2	R			< 0.2	R
16WW24	10/23/97	12:50	Primary	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.2	R			< 0.2	R
16WW25	7/10/97	11:15	Primary			< 0.2	UJ	< 0.2	UJ	< 0.2	UJ	< 0.2	UJ	< 0.5	UJ	< 0.2	UJ	< 0.2	UJ
16WW25	10/23/97	10:40	Primary	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.2	R			< 0.2	R
16WW25	1/21/98	17:35	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW25	6/12/98	11:10	Primary			< 0.26	UJ	< 0.26	UJ	< 0.13		< 0.13		< 0.26		< 0.13	UJ	< 0.26	
16WW26	7/10/97	11:30	Primary			< 0.2	UJ	< 0.2	UJ	< 0.2	UJ	< 0.2	UJ	< 0.5	UJ	< 0.2	UJ	< 0.2	UJ
16WW26	10/23/97	12:50	Primary	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.1	R	< 0.2	R			< 0.2	R
16WW26	1/21/98	17:00	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW26	6/12/98	9:30	Primary			< 0.26	UJ	< 0.26	UJ	< 0.13		< 0.13		< 0.26		< 0.13	UJ	< 0.26	
16WW27	10/21/97	10:50	Primary	< 0.1	R	< 0.1	UJ	< 0.1		< 0.1	R	< 0.1		< 0.2				< 0.2	R
16WW28	10/21/97	12:55	Primary	< 0.1	R	< 0.1		< 0.1		< 0.1	R	< 0.1		< 0.2				< 0.2	R
16WW29	10/21/97	10:55	Primary	< 0.1	R	< 0.1		< 0.1		< 0.1	R	< 0.1		< 0.2				< 0.2	R
16WW29	1/22/98	13:15	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW29	6/11/98	14:30	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW29	6/11/98	14:30	Duplicate 1			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW30	10/21/97	12:40	Primary	< 0.1	R	< 0.1		< 0.1		< 0.1	R	< 0.1		< 0.2				< 0.2	R
16WW30	1/22/98	13:30	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW30	6/15/98	14:00	Primary			< 0.26	UJ	< 0.26	UJ	< 0.13		< 0.13		< 0.26		< 0.13	UJ	< 0.26	
16WW31	7/9/97	11:05	Primary			< 0.2		< 0.2		< 0.2		< 0.2		< 0.5		< 0.2		< 0.2	
16WW31	10/22/97	10:10	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW31	10/22/97	10:10	Duplicate 1	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW32	7/9/97	10:30	Primary			< 0.2		< 0.2		< 0.2		< 0.2		< 0.5		< 0.2		0.72	
16WW32	10/22/97	12:40	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW33	7/9/97	14:02	Primary			< 0.2		< 0.2		< 0.2		< 0.2		< 0.5		< 0.2		0.2	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Explosives

SITE	DATE	TIME	RESULT TYPE	1,3-DNB (ug/l)		2,4-Dinitro toluene (ug/l)		2,6-Dinitro toluene (ug/l)		2-Amino-4,6 dinitrotoluene (ug/l)		4-Amino-2,6 dinitrotoluene (ug/l)		HMX (ug/l)		1,3-Dinitro benzene (ug/l)		3-Nitrotoluene (ug/l)	
16WW33	10/22/97	10:00	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW34	7/9/97	14:19	Primary			< 0.2		< 0.2		< 0.2		< 0.2		< 0.5		< 0.2		1	
16WW34	10/22/97	12:20	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW35	7/10/97	16:15	Primary			< 2		< 2		< 2		< 2		< 5		< 2		< 2	
16WW35	10/24/97	11:10	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW35	1/21/98	11:30	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW35	6/12/98	13:35	Primary			< 0.26	UJ	< 0.26	UJ	< 0.13		< 0.13		< 0.26		< 0.13	UJ	< 0.26	
16WW36	7/10/97	15:26	Primary			< 1		< 1		< 1		< 1		< 2.5		< 1		< 1	
16WW36	10/27/97	13:50	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW36	1/20/98	16:15	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW36	6/12/98	15:30	Primary			< 0.26	UJ	< 0.26	UJ	< 0.13		< 0.13		< 0.26		< 0.13	UJ	< 0.26	
16WW37	10/21/97	16:40	Primary	< 0.1	R	< 0.1	UJ	< 0.1	UJ	< 0.1	R	< 0.1	UJ	< 0.2	UJ			< 0.2	R
16WW37	10/21/97	16:40	Duplicate 1	< 0.1	R	< 0.1	UJ	< 0.1	UJ	< 0.1	R	< 0.1	UJ	< 0.2	UJ			< 0.2	R
16WW37	1/21/98	13:30	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW37	6/14/98	11:00	Primary			< 0.26	UJ	< 0.26	UJ	< 0.13	UJ	< 0.13	UJ	< 0.26	UJ	< 0.13	UJ	< 0.26	UJ
16WW38	10/22/97	16:10	Primary	< 0.1		< 0.1		< 0.1		< 0.1		< 0.1		< 0.2				< 0.2	
16WW38	1/21/98	13:00	Primary			< 0.26		< 0.26		< 0.13		< 0.13		< 0.26		< 0.13		< 0.26	
16WW38	6/14/98	12:45	Primary			< 0.26	UJ	< 0.26	UJ	< 0.13		< 0.13		< 0.26		< 0.13	UJ	< 0.26	
MAXIMUM				0.1		5		2		5		2		5		2		11	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Explosives

SITE	DATE	TIME	RESULT TYPE	Nitrobenzene (ug/l)		2-Nitro toluene (ug/l)		4-Nitro toluene (ug/l)		RDX (ug/l)		1,3,5-Trinitro benzene (ug/l)		Tetryl (ug/l)		2,4,6-Trinitrotoluene (ug/l)	
122	6/2/93	0:00	Primary	< 0.25		< 0.25		< 0.25		< 0.84		< 0.26		< 0.8		< 0.11	
122	6/14/95	13:42	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16EW01	2/21/96	10:55	Primary	0.184	UJ	< 0.077	UJ	< 0.089	UJ	0.958	J	< 0.043	UJ	< 0.049	UJ	< 0.044	UJ
16EW01	2/21/96	16:48	Primary	< 0.045	UJ	< 0.077	UJ	< 0.089	UJ	1.9	J	< 0.043	UJ	< 0.049	UJ	< 0.044	UJ
16EW01	2/21/96	20:35	Primary	0.223	UJ	< 0.077	UJ	< 0.089	UJ	1.13	J	< 0.043	UJ	< 0.049	UJ	< 0.044	UJ
16EW01	2/24/96	12:35	Primary	< 0.045	UJ	< 0.077	UJ	< 0.089	UJ	< 0.086	UJ	< 0.043	UJ	< 0.049	UJ	1.19	UJ
16EW01	2/25/96	23:55	Primary	< 0.045	UJ	< 0.077	UJ	< 0.089	UJ	< 0.086	UJ	< 0.043	UJ	< 0.049	UJ	1.56	UJ
16EW01	2/25/96	23:55	Duplicate 1	0.176	UJ	< 0.077	UJ	< 0.089	UJ	< 0.086	UJ	< 0.043	UJ	< 0.049	UJ	1.03	UJ
16EW01	2/27/96	12:10	Primary	0.141	UJ	< 0.077	UJ	< 0.089	UJ	0.351	J	< 0.043	UJ	< 0.049	UJ	< 0.044	UJ
16EW01	3/16/96	11:30	Primary	< 0.045	UJ	< 0.077	UJ	< 0.089	UJ	< 0.086		< 0.043	UJ	< 0.049	UJ	< 0.044	UJ
16EW01	3/17/96	16:10	Primary	< 0.045	UJ	< 0.077	UJ	< 0.089	UJ	< 0.086		< 0.043	UJ	< 0.049	UJ	< 0.044	UJ
16EW01	3/19/96	11:30	Primary	< 0.045	UJ	< 0.077	UJ	< 0.089	UJ	0.27		< 0.043	UJ	< 0.049	UJ	< 0.044	UJ
16EW01	10/29/97	10:00	Primary	< 0.1	R	< 0.2	R	< 0.2	R	< 0.2	R	< 0.1	R	< 0.2	R	< 0.1	R
16EW01	10/29/97	10:00	Duplicate 1	< 0.1	R	< 0.2	R	< 0.2	R	< 0.2	R	< 0.1	R	< 0.2	R	< 0.1	R
16EW01	1/22/98	12:15	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW01	6/11/98	15:10	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW01	6/11/98	15:10	Duplicate 1	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW02	2/23/96	9:40	Primary	< 0.045	UJ	< 0.077	UJ	< 0.089	UJ	2.56	J	< 0.043	UJ	< 0.049	UJ	< 0.044	UJ
16EW02	2/23/96	14:00	Primary	0.344	UJ	< 0.077	UJ	< 0.089	UJ	4.75	J	< 0.043	UJ	< 0.049	UJ	< 0.044	UJ
16EW02	2/28/96	12:20	Primary	0.158	UJ	< 0.077	UJ	< 0.089	UJ	1.62	J	< 0.043	UJ	< 0.049	UJ	< 0.044	UJ
16EW02	2/29/96	11:10	Primary	0.274	UJ	< 0.077	UJ	< 0.089	UJ	1.95	J	< 0.043	UJ	0.349	UJ	< 0.044	UJ
16EW02	2/29/96	21:25	Primary	0.138	UJ	< 0.077	UJ	< 0.089	UJ	1.36		< 0.043	UJ	< 0.049	UJ	< 0.044	UJ
16EW02	3/16/96	12:00	Primary	< 0.045	UJ	< 0.077	UJ	< 0.089	UJ	1.33		< 0.043	UJ	< 0.049	UJ	< 0.044	UJ
16EW02	3/19/96	12:00	Primary	< 0.045	UJ	< 0.077	UJ	< 0.089	UJ	1.44		< 0.043	UJ	< 0.049	UJ	< 0.044	UJ
16EW02	10/29/97	10:30	Primary	< 0.1	UJ	< 0.2	UJ	< 0.2	UJ	< 0.2	UJ	< 0.1	UJ	< 0.2	UJ	< 0.1	UJ
16EW02	1/22/98	12:00	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW02	6/11/98	15:15	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW03	10/29/97	11:20	Primary	< 0.1	UJ	< 0.2	UJ	< 0.2	UJ	< 0.2	UJ	< 0.1	UJ	< 0.2	UJ	< 0.1	UJ
16EW03	1/22/98	11:55	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW03	6/11/98	15:20	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW04	10/29/97	11:00	Primary	< 0.1	UJ	< 0.2	UJ	< 0.2	UJ	< 0.2	UJ	< 0.1	UJ	< 0.2	UJ	< 0.1	UJ
16EW04	1/22/98	11:50	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW04	6/11/98	15:25	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW05	10/29/97	11:15	Primary	< 0.1	UJ	< 0.2	UJ	< 0.2	UJ	< 0.2	UJ	< 0.1	UJ	< 0.2	UJ	< 0.1	UJ
16EW05	1/22/98	11:35	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW05	6/11/98	15:30	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW06	10/29/97	12:20	Primary	< 0.1	UJ	< 0.2	UJ	< 0.2	UJ	< 0.2	UJ	< 0.1	UJ	< 0.2	UJ	< 0.1	UJ

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Explosives

SITE	DATE	TIME	RESULT TYPE	Nitrobenzene (ug/l)		2-Nitro toluene (ug/l)		4-Nitro toluene (ug/l)		RDX (ug/l)		1,3,5-Trinitro benzene (ug/l)		Tetryl (ug/l)		2,4,6-Trinitrotoluene (ug/l)	
16EW06	1/22/98	11:25	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW06	6/11/98	15:35	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW07	10/29/97	11:55	Primary	< 0.1	UJ	< 0.2	UJ	< 0.2	UJ	< 0.2	UJ	< 0.1	UJ	< 0.2	UJ	< 0.1	UJ
16EW07	1/22/98	11:45	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW07	6/11/98	15:40	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW08	10/29/97	11:30	Primary	< 0.1	R	< 0.2	R	< 0.2	R	< 0.2	R	< 0.1	R	< 0.2	R	< 0.1	R
16EW08	1/22/98	10:10	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW08	1/22/98	10:10	Duplicate 1	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16EW08	6/11/98	15:45	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16PB02	3/18/95	0:00	Primary	< 2		< 5		< 5		< 2		< 2		< 2		< 2	
16PB02	3/18/95	0:00	Duplicate 1	< 2		< 5		< 5		< 2		< 2		< 2		< 2	
16PB04	3/19/95	0:00	Primary	6.3		< 5		< 5		< 2		2.2		< 2		< 2	
16PB05	3/18/95	0:00	Primary	3.3		< 5		< 5		< 2		< 2		< 2		< 2	
16PB06	3/18/95	0:00	Primary	< 2		< 5		< 5		< 2		< 2		< 2		< 2	
16PB07	3/19/95	0:00	Primary	< 2		< 5		< 5		< 2		< 2		< 2		< 2	
16PB08	3/21/95	0:00	Primary	< 2		< 10		< 15		< 2		< 2		< 5		< 2	
16PB09	3/19/95	0:00	Primary	< 2		< 5		< 5		< 2		< 2		< 2		< 2	
16PB10	3/20/95	0:00	Primary	< 2		< 10		< 15		< 2		< 2		< 5		< 2	
16PB11	3/20/95	0:00	Primary	< 2		< 10		< 15		< 2		< 2		< 5		< 2	
16PB13	3/21/95	0:00	Primary	20		< 10		< 15		200		< 2		< 5		< 2	
16PB14	3/21/95	0:00	Primary	< 2		< 10		< 15		< 2		< 2		< 5		< 2	
16PB16	3/20/95	0:00	Primary	< 2		< 10		< 15		< 2		< 2		5.7		< 2	
16PB17	3/20/95	0:00	Primary	< 2		< 5		< 5		< 2		< 2		< 2		< 2	
16PB19	3/21/95	0:00	Primary	< 2		< 10		< 15		< 2		< 2		< 5		240	
16PB20	3/21/95	0:00	Primary	< 2		< 10		< 15		< 2		< 2		< 5		18	
16PB21	3/20/95	0:00	Primary	< 2		< 10		< 15		< 2		< 2		36		< 2	
16PB25	3/28/95	0:00	Primary	< 2		< 10		< 15		< 2		< 2		< 5		12	
16PB26	3/28/95	0:00	Primary	< 2		< 10		< 15		< 2		< 2		< 5		< 2	
16PB27	3/28/95	0:00	Primary	< 2		< 10		< 15		< 2		< 2		< 5		51	
16PB28	3/28/95	0:00	Primary	< 2		< 10		< 15		< 2		< 2		< 5		< 2	
16WW01	6/10/93	0:00	Primary	< 0.25		< 0.25	UJ	< 0.25		< 0.84		< 0.26		< 0.8		< 0.11	UJ
16WW01	6/19/95	12:20	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW02	6/10/93	0:00	Primary	< 0.25		< 0.25	UJ	< 0.25		< 0.84		< 0.26		< 0.8		< 0.11	UJ
16WW02	6/13/95	16:30	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW03	6/8/93	0:00	Primary	< 0.25		< 0.25	UJ	< 0.25		< 0.84		< 0.26		< 0.8		< 0.11	UJ
16WW03	6/14/95	16:04	Primary	< 0.6		< 0.6		< 0.4		0.6		< 0.5		< 0.4		0.9	
16WW04	6/8/93	0:00	Primary	< 0.25		< 0.25	UJ	< 0.25		< 0.84		< 0.26		< 0.8		< 0.11	UJ
16WW04	6/20/95	9:25	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Explosives

SITE	DATE	TIME	RESULT TYPE	Nitrobenzene (ug/l)		2-Nitro toluene (ug/l)		4-Nitro toluene (ug/l)		RDX (ug/l)		1,3,5-Trinitro benzene (ug/l)		Tetryl (ug/l)		2,4,6-Trinitrotoluene (ug/l)	
16WW04	6/20/95	9:25	Duplicate 1	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW04	6/29/95	12:55	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW05	6/11/93	0:00	Primary	1.5		< 0.25		< 0.25		< 0.84		< 0.26		< 0.8		< 0.11	
16WW05	6/11/93	0:00	Duplicate 1	1.9		< 0.25		< 0.25		< 0.84		< 0.26		< 0.8		< 0.11	
16WW05	6/19/95	13:07	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW05	10/22/97	17:10	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW06	6/11/93	0:00	Primary	< 0.25?		< 0.25?		< 0.25?		< 0.84?		< 0.26?		< 0.80?		< 0.11?	
16WW06	6/19/95	12:50	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW06	10/22/97	14:50	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW07	6/11/93	0:00	Primary	< 0.25		< 0.25		< 0.25		< 0.84		< 0.26		< 0.8		< 0.11	
16WW07	6/14/95	12:00	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW08	6/11/93	0:00	Primary	< 0.25		< 0.25		< 0.25		< 0.84		< 0.26		< 0.8		< 0.11	
16WW08	6/14/95	11:40	Primary	< 0.6		< 0.6		< 0.4		< 0.6		0.68		< 0.4		< 0.7	
16WW08	6/14/95	11:40	Duplicate 1	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW09	6/10/93	0:00	Primary	< 0.25		< 0.25	UJ	< 0.25		< 0.84		< 0.26		< 0.8		< 0.11	
16WW09	6/19/95	14:55	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW10	6/9/93	0:00	Primary	< 0.25		< 0.25	UJ	< 0.25		< 0.84		< 0.26		< 0.8		< 0.11	UJ
16WW10	6/19/95	16:20	Primary	< 0.6		< 0.6		< 0.4		< 0.6		0.74		< 0.4		< 0.7	
16WW11	6/9/93	0:00	Primary	< 0.25		< 0.25	UJ	< 0.25		< 0.84		< 0.26		< 0.8		< 0.11	UJ
16WW11	6/9/93	0:00	Duplicate 1	< 0.25		< 0.25		< 0.25		< 0.84		< 0.26		< 0.8		< 0.11	
16WW11	6/16/95	11:42	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW12	6/13/95	17:45	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW12	7/10/97	17:45	Primary	< 0.4		< 0.4		< 0.4		< 1		< 0.4		< 0.4		< 0.4	
16WW12	10/27/97	13:40	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW12	10/27/97	13:40	Duplicate 1	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW12	1/20/98	16:15	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW12	6/11/98	12:05	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW13	6/13/95	16:55	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW13	10/27/97	16:05	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW14	6/13/95	16:45	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW14	10/24/97	13:55	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW14	1/20/98	13:30	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW14	1/20/98	13:30	Duplicate 1	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW14	6/15/98	9:20	Primary	< 0.26	UJ	< 0.26		< 0.26		< 0.26		< 0.39	UJ	< 0.26		< 0.26	UJ
16WW15	6/22/95	10:10	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW15	10/28/97	15:25	Primary	< 0.1	R	< 0.2	R	< 0.2		< 0.2	R	< 0.1		< 0.2		< 0.1	
16WW16	6/20/95	11:00	Primary	< 0.6	UJ	< 0.6	UJ	< 0.4	UJ	< 0.6	UJ	< 0.5	UJ	< 0.4	UJ	< 0.7	UJ
16WW16	10/27/97	16:00	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Explosives

SITE	DATE	TIME	RESULT TYPE	Nitrobenzene (ug/l)		2-Nitro toluene (ug/l)		4-Nitro toluene (ug/l)		RDX (ug/l)		1,3,5-Trinitro benzene (ug/l)		Tetryl (ug/l)		2,4,6-Trinitrotoluene (ug/l)	
16WW16	1/21/98	12:15	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		4.4		< 0.26	
16WW16	6/15/98	11:15	Primary	< 0.26	UJ	< 0.26		< 0.26		< 0.26		< 0.39	UJ	3.3		< 0.26	UJ
16WW17	6/16/95	15:07	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW17	10/27/97	16:45	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW18	6/20/95	14:02	Primary	< 0.6		< 0.6		< 0.4		< 0.6		< 0.5		< 0.4		< 0.7	
16WW18	10/28/97	15:10	Primary	< 0.1	R	< 0.2	R	< 0.2	R	< 0.2	R	< 0.1	R	< 0.2	R	< 0.1	R
16WW19	10/24/97	11:15	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW20	10/24/97	16:15	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW21	10/28/97	14:45	Primary	< 0.1	R	< 0.2	R	< 0.2		< 0.2	R	< 0.1		< 0.2		< 0.1	
16WW22	7/10/97	17:50	Primary	< 0.4		< 0.4		< 0.4		< 1		< 0.4		< 0.4		< 0.4	
16WW22	10/24/97	13:45	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW22	1/20/98	14:50	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW22	6/11/98	10:15	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW23	10/23/97	10:40	Primary	< 0.1	R	< 0.2	R	< 0.2	R	< 0.2	R	< 0.1	R	< 0.2	R	< 0.1	R
16WW24	10/23/97	12:50	Primary	< 0.1	R	< 0.2	R	< 0.2	R	< 0.2	R	< 0.1	R	< 0.2	R	< 0.1	R
16WW25	7/10/97	11:15	Primary	< 0.2	UJ	< 0.2	UJ	< 0.2	UJ	< 0.5	UJ	< 0.2	UJ	< 0.2	UJ	< 0.2	UJ
16WW25	10/23/97	10:40	Primary	< 0.1	R	< 0.2	R	< 0.2	R	< 0.2	R	< 0.1	R	< 0.2	R	< 0.1	R
16WW25	1/21/98	17:35	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW25	6/12/98	11:10	Primary	< 0.26	UJ	< 0.26		< 0.26		< 0.26		< 0.39	UJ	< 0.26		< 0.26	UJ
16WW26	7/10/97	11:30	Primary	< 0.2	UJ	< 0.2	UJ	< 0.2	UJ	< 0.5	UJ	< 0.2	UJ	< 0.2	UJ	< 0.2	UJ
16WW26	10/23/97	12:50	Primary	< 0.1	R	< 0.2	R	< 0.2	R	< 0.2	R	< 0.1	R	< 0.2	R	< 0.1	R
16WW26	1/21/98	17:00	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW26	6/12/98	9:30	Primary	< 0.26	UJ	< 0.26		< 0.26		< 0.26		< 0.39	UJ	< 0.26		< 0.26	UJ
16WW27	10/21/97	10:50	Primary	< 0.1	R	< 0.2	R	< 0.2		< 0.2	R	< 0.1		< 0.2	R	< 0.1	
16WW28	10/21/97	12:55	Primary	< 0.1	R	< 0.2	R	< 0.2		< 0.2	R	< 0.1		< 0.2	R	< 0.1	
16WW29	10/21/97	10:55	Primary	< 0.1	R	< 0.2	R	< 0.2		< 0.2	R	< 0.1		< 0.2	R	< 0.1	
16WW29	1/22/98	13:15	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW29	6/11/98	14:30	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW29	6/11/98	14:30	Duplicate 1	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW30	10/21/97	12:40	Primary	< 0.1	R	< 0.2	R	< 0.2		< 0.2	R	< 0.1		< 0.2	R	< 0.1	
16WW30	1/22/98	13:30	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW30	6/15/98	14:00	Primary	< 0.26	UJ	< 0.26		< 0.26		< 0.26		< 0.39	UJ	< 0.26		< 0.26	UJ
16WW31	7/9/97	11:05	Primary	< 0.2		< 0.2		< 0.2		< 0.5		< 0.2		< 0.2		< 0.2	
16WW31	10/22/97	10:10	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW31	10/22/97	10:10	Duplicate 1	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW32	7/9/97	10:30	Primary	< 0.2		< 0.2		< 0.2		< 0.5		< 0.2		< 0.2		< 0.2	
16WW32	10/22/97	12:40	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW33	7/9/97	14:02	Primary	< 0.2		< 0.2		< 0.2		< 0.5		< 0.2		< 0.2		< 0.2	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Explosives

SITE	DATE	TIME	RESULT TYPE	Nitrobenzene (ug/l)		2-Nitro toluene (ug/l)		4-Nitro toluene (ug/l)		RDX (ug/l)		1,3,5-Trinitro benzene (ug/l)		Tetryl (ug/l)		2,4,6-Trinitrotoluene (ug/l)	
16WW33	10/22/97	10:00	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW34	7/9/97	14:19	Primary	< 0.2		< 0.2		< 0.2		< 0.5		< 0.2		< 0.2		< 0.2	
16WW34	10/22/97	12:20	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW35	7/10/97	16:15	Primary	< 2		< 2		< 2		< 5		< 2		< 2		< 2	
16WW35	10/24/97	11:10	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW35	1/21/98	11:30	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW35	6/12/98	13:35	Primary	< 0.26	UJ	< 0.26		< 0.26		< 0.26		< 0.39	UJ	< 0.26		< 0.26	UJ
16WW36	7/10/97	15:26	Primary	< 1		< 1		< 1		< 2.5		< 1		< 1		< 1	
16WW36	10/27/97	13:50	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW36	1/20/98	16:15	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW36	6/12/98	15:30	Primary	< 0.26	UJ	< 0.26		< 0.26		< 0.26		< 0.39	UJ	< 0.26		< 0.26	UJ
16WW37	10/21/97	16:40	Primary	< 0.1	R	< 0.2	R	< 0.2	UJ	< 0.2	R	< 0.1	UJ	< 0.2	R	< 0.1	UJ
16WW37	10/21/97	16:40	Duplicate 1	< 0.1	R	< 0.2	R	< 0.2	UJ	< 0.2	R	< 0.1	UJ	< 0.2	R	< 0.1	UJ
16WW37	1/21/98	13:30	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW37	6/14/98	11:00	Primary	< 0.26	UJ	< 0.26	UJ	< 0.26	UJ	< 0.26	UJ	< 0.39	UJ	< 0.26	UJ	< 0.26	UJ
16WW38	10/22/97	16:10	Primary	< 0.1		< 0.2		< 0.2		< 0.2		< 0.1		< 0.2		< 0.1	
16WW38	1/21/98	13:00	Primary	< 0.26		< 0.26		< 0.26		< 0.26		< 0.39		< 0.26		< 0.26	
16WW38	6/14/98	12:45	Primary	< 0.26	UJ	< 0.26		< 0.26		< 0.26		< 0.39	UJ	< 0.26		< 0.26	UJ
MAXIMUM				20		10		15		200		2.2		36		240	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
PCB's and Chlorinated Pesticides

SITE	DATE	TIME	RESULT TYPE	4,4'-DDD (ug/l)	DDE (ug/l)	Aldrin (ug/l)		alpha-BHC (ug/l)	Aroclor 1016 (ug/l)	Aroclor 1221 (ug/l)	Aroclor 1232 (ug/l)	Aroclor 1242 (ug/l)
16EW01	10/29/97	10:00	Primary	< 0.05	< 0.05	0.04	J	< 0.02	< 0.5	< 1	< 0.5	< 0.5
16EW01	10/29/97	10:00	Duplicate 1	< 0.05	< 0.05	0.04	J	< 0.02	< 0.5	< 1	< 0.5	< 0.5
16WW16	10/27/97	16:00	Primary	< 0.05	< 0.05	< 0.02		< 0.02	< 0.5	< 1	< 0.5	< 0.5
16WW36	10/27/97	13:50	Primary	< 0.05	< 0.05	< 0.02		< 0.02	< 0.5	< 1	< 0.5	< 0.5
16WW36	10/27/97	13:50	Duplicate 1	< 0.05	< 0.05	< 0.02		< 0.02	< 0.5	< 1	< 0.5	< 0.5
MAXIMUM				0.05	0.05	0.04		0.02	0.5	1	0.5	0.5

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
PCB's and Chlorinated Pesticides

SITE	DATE	TIME	RESULT TYPE	Aroclor 1248 (ug/l)	Aroclor 1254 (ug/l)	Aroclor 1260 (ug/l)	beta-BHC (ug/l)	Chlordane (ug/l)	delta-BHC (ug/l)	Dieldrin (ug/l)	Endosulfan-I (ug/l)
16EW01	10/29/97	10:00	Primary	< 0.5	< 0.5	< 0.5	< 0.02	< 0.5	< 0.02	< 0.05	< 0.02
16EW01	10/29/97	10:00	Duplicate 1	< 0.5	< 0.5	< 0.5	< 0.02	< 0.5	< 0.02	< 0.05	< 0.02
16WW16	10/27/97	16:00	Primary	< 0.5	< 0.5	< 0.5	< 0.02	< 0.5	< 0.02	< 0.05	< 0.02
16WW36	10/27/97	13:50	Primary	< 0.5	< 0.5	< 0.5	< 0.02	< 0.5	< 0.02	< 0.05	< 0.02
16WW36	10/27/97	13:50	Duplicate 1	< 0.5	< 0.5	< 0.5	< 0.02	< 0.5	< 0.02	< 0.05	< 0.02
MAXIMUM				0.5	0.5	0.5	0.02	0.5	0.02	0.05	0.02

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
PCB's and Chlorinated Pesticides

SITE	DATE	TIME	RESULT TYPE		Endosulfan II (ug/l)		Endosulfan sulfate (ug/l)		Endrin (ug/l)		Endrin aldehyde (ug/l)		Heptachlor (ug/l)		Heptachlor epoxide (ug/l)		gamma-BHC (ug/l)		Methoxychlor (ug/l)
16EW01	10/29/97	10:00	Primary	<	0.05	<	0.05	<	0.05	<	0.05	<	0.02	<	0.02	<	0.02	<	0.25
16EW01	10/29/97	10:00	Duplicate 1	<	0.05	<	0.05	<	0.05	<	0.05	<	0.02	<	0.02	<	0.02	<	0.25
16WW16	10/27/97	16:00	Primary	<	0.05	<	0.05	<	0.05	<	0.05	<	0.02	<	0.02	<	0.02	<	0.25
16WW36	10/27/97	13:50	Primary	<	0.05	<	0.05	<	0.05	<	0.05	<	0.02	<	0.02	<	0.02	<	0.25
16WW36	10/27/97	13:50	Duplicate 1	<	0.05	<	0.05	<	0.05	<	0.05	<	0.02	<	0.02	<	0.02	<	0.25
MAXIMUM					0.05		0.05		0.05		0.05		0.02		0.02		0.02		0.25

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
PCB's and Chlorinated Pesticides

SITE	DATE	TIME	RESULT TYPE		Toxaphene (ug/l)
16EW01	10/29/97	10:00	Primary	<	2.5
16EW01	10/29/97	10:00	Duplicate 1	<	2.5
16WW16	10/27/97	16:00	Primary	<	2.5
16WW36	10/27/97	13:50	Primary	<	2.5
16WW36	10/27/97	13:50	Duplicate 1	<	2.5
MAXIMUM					2.5

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	RESULT TYPE	1,2,4-Trichloro benzene (ug/l)	1-Chloro naphthalene (ug/l)	2,4,5-Trichloro phenol (ug/l)	2,4,6-Trichloro phenol (ug/l)	2,4-Dichloro phenol (ug/l)	2,4-Dimethyl phenol (ug/l)	2,4-Dinitro phenol (ug/l)	2,4-Dinitro toluene (ug/l)
122	6/2/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 50	< 10
16EW01	2/21/96	10:55	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW01	2/21/96	16:48	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW01	2/21/96	20:35	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW01	2/24/96	12:35	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW01	2/25/96	23:55	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW01	2/25/96	23:55	Duplicate 1	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW01	2/27/96	12:10	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW01	3/16/96	11:30	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW01	3/17/96	16:10	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW01	3/19/96	11:30	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW02	2/23/96	9:40	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW02	2/23/96	14:00	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW02	2/28/96	12:20	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW02	2/29/96	11:10	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW02	2/29/96	21:25	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW02	3/13/96	11:35	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW02	3/14/96	12:00	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW02	3/14/96	12:00	Duplicate 1	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW02	3/16/96	12:00	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16EW02	3/19/96	12:00	Primary	< 10		< 10	< 10	< 10	< 10	< 25	< 10
16WW01	6/10/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 50	< 10
16WW02	6/10/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 50	< 10
16WW03	6/8/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 50	< 10
16WW04	6/8/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 50	< 10
16WW05	6/11/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 50	< 10
16WW05	6/11/93	0:00	Duplicate 1	< 10	< 10	< 50	< 10	< 10	< 10	< 50	< 10
16WW06	6/11/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 50	< 10
16WW07	6/11/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 50	< 10
16WW08	6/11/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 50	< 10
16WW09	6/10/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 50	< 10
16WW10	6/9/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 50	< 10
16WW11	6/9/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 50	< 10
16WW11	6/9/93	0:00	Duplicate 1	< 10	< 10	< 50	< 10	< 10	< 10	< 50	< 10
MAXIMUM				10	10	50	10	10	10	50	10

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	RESULT TYPE	2,6-Dinitro toluene (ug/l)	2-Chloro napthalene (ug/l)	2-Chlorophenol (ug/l)	2-Methyl napthalene (ug/l)	3,3'-Dichloro benzidine (ug/l)	4,6-Dinitro, 2-methylphenol (ug/l)	4-bromophenyl-phenylether (ug/l)	4-Bromophenyl phenyl ether (ug/l)
122	6/2/93	0:00	Primary	< 10	< 10	< 10	< 10	< 20	< 50		< 10
16EW01	2/21/96	10:55	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW01	2/21/96	16:48	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW01	2/21/96	20:35	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW01	2/24/96	12:35	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW01	2/25/96	23:55	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW01	2/25/96	23:55	Duplicate 1	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW01	2/27/96	12:10	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW01	3/16/96	11:30	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW01	3/17/96	16:10	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW01	3/19/96	11:30	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW02	2/23/96	9:40	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW02	2/23/96	14:00	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW02	2/28/96	12:20	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW02	2/29/96	11:10	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW02	2/29/96	21:25	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW02	3/13/96	11:35	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW02	3/14/96	12:00	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW02	3/14/96	12:00	Duplicate 1	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW02	3/16/96	12:00	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16EW02	3/19/96	12:00	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	
16WW01	6/10/93	0:00	Primary	< 10	< 10	< 10	< 10	< 20	< 50		< 10
16WW02	6/10/93	0:00	Primary	< 10	< 10	< 10	< 10	< 20	< 50		< 10
16WW03	6/8/93	0:00	Primary	< 10	< 10	< 10	< 10	< 20	< 50		< 10
16WW04	6/8/93	0:00	Primary	< 10	< 10	< 10	< 10	< 20	< 50		< 10
16WW05	6/11/93	0:00	Primary	< 10	< 10	< 10	< 10	< 20	< 50		< 10
16WW05	6/11/93	0:00	Duplicate 1	< 10	< 10	< 10	< 10	< 20	< 50		< 10
16WW06	6/11/93	0:00	Primary	< 10	< 10	< 10	< 10	< 20	< 50		< 10
16WW07	6/11/93	0:00	Primary	< 10	< 10	< 10	< 10	< 20	< 50		< 10
16WW08	6/11/93	0:00	Primary	< 10	< 10	< 10	< 10	< 20	< 50		< 10
16WW09	6/10/93	0:00	Primary	< 10	< 10	< 10	< 10	< 20	< 50		< 10
16WW10	6/9/93	0:00	Primary	< 10	< 10	< 10	< 10	< 20	< 50		< 10
16WW11	6/9/93	0:00	Primary	< 10	< 10	< 10	< 10	< 20	< 50		< 10
16WW11	6/9/93	0:00	Duplicate 1	< 10	< 10	< 10	< 10	< 20	< 50		< 10
MAXIMUM				10	10	10	10	20	50	10	10

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	RESULT TYPE	4-Chlorophenyl phenyl ether (ug/l)	Acenaphthene (ug/l)	Acenaphthylene (ug/l)	Anthracene (ug/l)	Benzidine (ug/l)	Benzo(a) anthracene (ug/l)	Benzo(a)pyrene (ug/l)	Benzo(b) fluoranthene (ug/l)
122	6/2/93	0:00	Primary	< 10	< 10	< 10	< 10		< 10	< 10	< 10
16EW01	2/21/96	10:55	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW01	2/21/96	16:48	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW01	2/21/96	20:35	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW01	2/24/96	12:35	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW01	2/25/96	23:55	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW01	2/25/96	23:55	Duplicate 1	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW01	2/27/96	12:10	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW01	3/16/96	11:30	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW01	3/17/96	16:10	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW01	3/19/96	11:30	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW02	2/23/96	9:40	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW02	2/23/96	14:00	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW02	2/28/96	12:20	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW02	2/29/96	11:10	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW02	2/29/96	21:25	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW02	3/13/96	11:35	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW02	3/14/96	12:00	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW02	3/14/96	12:00	Duplicate 1	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW02	3/16/96	12:00	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16EW02	3/19/96	12:00	Primary	< 10	< 10	< 10	< 10	< 25	< 10	< 10	< 10
16WW01	6/10/93	0:00	Primary	< 10	< 10	< 10	< 10		< 10	< 10	< 10
16WW02	6/10/93	0:00	Primary	< 10	< 10	< 10	< 10		< 10	< 10	< 10
16WW03	6/8/93	0:00	Primary	< 10	< 10	< 10	< 10		< 10	< 10	< 10
16WW04	6/8/93	0:00	Primary	< 10	< 10	< 10	< 10		< 10	< 10	< 10
16WW05	6/11/93	0:00	Primary	< 10	< 10	< 10	< 10		< 10	< 10	< 10
16WW05	6/11/93	0:00	Duplicate 1	< 10	< 10	< 10	< 10		< 10	< 10	< 10
16WW06	6/11/93	0:00	Primary	< 10	< 10	< 10	< 10		< 10	< 10	< 10
16WW07	6/11/93	0:00	Primary	< 10	< 10	< 10	< 10		< 10	< 10	< 10
16WW08	6/11/93	0:00	Primary	< 10	< 10	< 10	< 10		< 10	< 10	< 10
16WW09	6/10/93	0:00	Primary	< 10	< 10	< 10	< 10		< 10	< 10	< 10
16WW10	6/9/93	0:00	Primary	< 10	< 10	< 10	< 10		< 10	< 10	< 10
16WW11	6/9/93	0:00	Primary	< 10	< 10	< 10	< 10		< 10	< 10	< 10
16WW11	6/9/93	0:00	Duplicate 1	< 10	< 10	< 10	< 10		< 10	< 10	< 10
MAXIMUM				10	10	10	10	25	10	10	10

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	RESULT TYPE	Benzo(g,h,i) perylene (ug/l)	Benzo(k)fluor anthene (ug/l)	Benzoic acid (ug/l)	Benzyl alcohol (ug/l)	Bis(2-chloro-1-methylethyl) ether (ug/l)	Bis(2-chloro ethoxy) methane (ug/l)	bis(2-Chloro ethyl) ether (ug/l)	bis(2-Ethyl hexyl) pthalate (ug/l)	
122	6/2/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 10	< 10	
16EW01	2/21/96	10:55	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	2/21/96	16:48	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	2/21/96	20:35	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	2/24/96	12:35	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	2/25/96	23:55	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	2/25/96	23:55	Duplicate 1	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	2/27/96	12:10	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	3/16/96	11:30	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	3/17/96	16:10	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	3/19/96	11:30	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW02	2/23/96	9:40	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	6	U
16EW02	2/23/96	14:00	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW02	2/28/96	12:20	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW02	2/29/96	11:10	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW02	2/29/96	21:25	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	11	
16EW02	3/13/96	11:35	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	18	
16EW02	3/14/96	12:00	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	11	
16EW02	3/14/96	12:00	Duplicate 1	< 10	< 10	< 10	< 10	< 10	< 10	< 10	11	
16EW02	3/16/96	12:00	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	26	
16EW02	3/19/96	12:00	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	15	
16WW01	6/10/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 10	22	U
16WW02	6/10/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 10	< 10	
16WW03	6/8/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 10	< 10	
16WW04	6/8/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 10	< 10	
16WW05	6/11/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 10	< 10	
16WW05	6/11/93	0:00	Duplicate 1	< 10	< 10	< 50	< 10	< 10	< 10	< 10	< 10	
16WW06	6/11/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 10	< 10	
16WW07	6/11/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 10	< 10	
16WW08	6/11/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 10	< 10	
16WW09	6/10/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 10	< 10	
16WW10	6/9/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 10	< 10	
16WW11	6/9/93	0:00	Primary	< 10	< 10	< 50	< 10	< 10	< 10	< 10	< 10	
16WW11	6/9/93	0:00	Duplicate 1	< 10	< 10	< 50	< 10	< 10	< 10	< 10	< 10	
MAXIMUM				10	10	50	10	10	10	10	26	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	RESULT TYPE	Butyl benzyl phthalate (ug/l)	Carbazole (ug/l)	Chrysene (ug/l)	Dibenz(a,h) anthracene (ug/l)	Dibenzofuran (ug/l)	Diethyl phthalate (ug/l)	Dimethyl phthalate (ug/l)	Di-n-butyl phthalate (ug/l)	
122	6/2/93	0:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	2/21/96	10:55	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	2/21/96	16:48	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	2/21/96	20:35	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	2/24/96	12:35	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	2/25/96	23:55	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	2/25/96	23:55	Duplicate 1	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	2/27/96	12:10	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW01	3/16/96	11:30	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	56	U
16EW01	3/17/96	16:10	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	55	U
16EW01	3/19/96	11:30	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW02	2/23/96	9:40	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW02	2/23/96	14:00	Primary	7	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW02	2/28/96	12:20	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW02	2/29/96	11:10	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW02	2/29/96	21:25	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16EW02	3/13/96	11:35	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	15	U
16EW02	3/14/96	12:00	Primary	5	< 10	< 10	< 10	< 10	< 10	< 10	32	U
16EW02	3/14/96	12:00	Duplicate 1	7	< 10	< 10	< 10	< 10	< 10	< 10	36	U
16EW02	3/16/96	12:00	Primary	5	< 10	< 10	< 10	< 10	< 10	< 10	48	U
16EW02	3/19/96	12:00	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10	
16WW01	6/10/93	0:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10	
16WW02	6/10/93	0:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10	
16WW03	6/8/93	0:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10	
16WW04	6/8/93	0:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10	
16WW05	6/11/93	0:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10	
16WW05	6/11/93	0:00	Duplicate 1	< 10		< 10	< 10	< 10	< 10	< 10	< 10	
16WW06	6/11/93	0:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10	
16WW07	6/11/93	0:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10	
16WW08	6/11/93	0:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10	
16WW09	6/10/93	0:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10	
16WW10	6/9/93	0:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10	
16WW11	6/9/93	0:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10	
16WW11	6/9/93	0:00	Duplicate 1	< 10		< 10	< 10	< 10	< 10	< 10	< 10	
MAXIMUM				10	10	10	10	10	10	10	56	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	RESULT TYPE	Di-n-octyl phthalate (ug/l)	Diphenylamine (ug/l)	Fluoranthene (ug/l)	Fluorene (ug/l)	Hexachloro benzene (ug/l)	Hexachloro-1,3-butadiene (ug/l)	Hexachloro cyclopentadiene (ug/l)	Hexachloro ethane (ug/l)
122	6/2/93	0:00	Primary	< 10	< 20	< 10	< 10	< 10	< 10	< 10	< 10
16EW01	2/21/96	10:55	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW01	2/21/96	16:48	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW01	2/21/96	20:35	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW01	2/24/96	12:35	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW01	2/25/96	23:55	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW01	2/25/96	23:55	Duplicate 1	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW01	2/27/96	12:10	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW01	3/16/96	11:30	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW01	3/17/96	16:10	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW01	3/19/96	11:30	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW02	2/23/96	9:40	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW02	2/23/96	14:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW02	2/28/96	12:20	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW02	2/29/96	11:10	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW02	2/29/96	21:25	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW02	3/13/96	11:35	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW02	3/14/96	12:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW02	3/14/96	12:00	Duplicate 1	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW02	3/16/96	12:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16EW02	3/19/96	12:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10	< 10
16WW01	6/10/93	0:00	Primary	< 10	< 20	< 10	< 10	< 10	< 10	< 10	< 10
16WW02	6/10/93	0:00	Primary	< 10	< 20	< 10	< 10	< 10	< 10	< 10	< 10
16WW03	6/8/93	0:00	Primary	< 10	< 20	< 10	< 10	< 10	< 10	< 10	< 10
16WW04	6/8/93	0:00	Primary	< 10	< 20	< 10	< 10	< 10	< 10	< 10	< 10
16WW05	6/11/93	0:00	Primary	< 10	< 20	< 10	< 10	< 10	< 10	< 10	< 10
16WW05	6/11/93	0:00	Duplicate 1	< 10	< 20	< 10	< 10	< 10	< 10	< 10	< 10
16WW06	6/11/93	0:00	Primary	< 10	< 20	< 10	< 10	< 10	< 10	< 10	< 10
16WW07	6/11/93	0:00	Primary	< 10	< 20	< 10	< 10	< 10	< 10	< 10	< 10
16WW08	6/11/93	0:00	Primary	< 10	< 20	< 10	< 10	< 10	< 10	< 10	< 10
16WW09	6/10/93	0:00	Primary	< 10	< 20	< 10	< 10	< 10	< 10	< 10	< 10
16WW10	6/9/93	0:00	Primary	< 10	< 20	< 10	< 10	< 10	< 10	< 10	< 10
16WW11	6/9/93	0:00	Primary	< 10	< 20	< 10	< 10	< 10	< 10	< 10	< 10
16WW11	6/9/93	0:00	Duplicate 1	< 10	< 20	< 10	< 10	< 10	< 10	< 10	< 10
MAXIMUM				10	20	10	10	10	10	10	10

Longhorn Army Ammuntiion Plant
Karnac, Texas
Site 16 - Sediment Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	DEPTH (ft	RESULT TYPE	1,2,3-Trichloro propane (ug/kg)	1,2,4-Trichloro benzene (ug/kg)	1,2-Dichloro ethane (ug/kg)	DCE12TOT (ug/kg)	1,2-Dichloro propane (ug/kg)	1,3-Dichloro propane (ug/kg)	1,4-Dichloro-2-butene (ug/kg)	1,4-Dioxane (ug/kg)
Min.					3	0	3	3	3	0	6	0
Max.					4.25	0	4.25	4	4.25	0	8.5	0
Avg.(log transformed)					1.25	#DIV/0!	1.25	1.237	1.25	#DIV/0!	1.941	#DIV/0!
St. Dev (log transformed)					0.096	#DIV/0!	0.096	0.085	0.096	#DIV/0!	0.098	#DIV/0!
H												
95th UCL of Mean					3.51	#DIV/0!	3.51	3.46	3.51	#DIV/0!	7.00	#DIV/0!

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	RESULT TYPE	1,2-Diphenyl hydrazine (ug/l)	Indeno (1,2,3-cd) pyrene (ug/l)	Isophorone (ug/l)	3-Methylphenol (ug/l)	1,3-Dichloro benzene (ug/l)	3-Nitroaniline (ug/l)	Naphthalene (ug/l)	Nitrobenzene (ug/l)
122	6/2/93	0:00	Primary	< 50	< 10	< 10	< 10	< 10	< 50	< 10	< 10
16EW01	2/21/96	10:55	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW01	2/21/96	16:48	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW01	2/21/96	20:35	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW01	2/24/96	12:35	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW01	2/25/96	23:55	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW01	2/25/96	23:55	Duplicate 1		< 10	< 10		< 10	< 25	< 10	< 10
16EW01	2/27/96	12:10	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW01	3/16/96	11:30	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW01	3/17/96	16:10	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW01	3/19/96	11:30	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW02	2/23/96	9:40	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW02	2/23/96	14:00	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW02	2/28/96	12:20	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW02	2/29/96	11:10	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW02	2/29/96	21:25	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW02	3/13/96	11:35	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW02	3/14/96	12:00	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW02	3/14/96	12:00	Duplicate 1		< 10	< 10		< 10	< 25	< 10	< 10
16EW02	3/16/96	12:00	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16EW02	3/19/96	12:00	Primary		< 10	< 10		< 10	< 25	< 10	< 10
16WW01	6/10/93	0:00	Primary	< 50	< 10	< 10	< 10	< 10	< 50	< 10	< 10
16WW02	6/10/93	0:00	Primary	< 50	< 10	< 10	< 10	< 10	< 50	< 10	< 10
16WW03	6/8/93	0:00	Primary	< 50	< 10	< 10	< 10	< 10	< 50	< 10	< 10
16WW04	6/8/93	0:00	Primary	< 50	< 10	< 10	< 10	< 10	< 50	< 10	< 10
16WW05	6/11/93	0:00	Primary	< 50	< 10	< 10	< 10	< 10	< 50	< 10	< 10
16WW05	6/11/93	0:00	Duplicate 1	< 50	< 10	< 10	< 10	< 10	< 50	< 10	< 10
16WW06	6/11/93	0:00	Primary	< 50	< 10	< 10	< 10	< 10	< 50	< 10	< 10
16WW07	6/11/93	0:00	Primary	< 50	< 10	< 10	< 10	< 10	< 50	< 10	< 10
16WW08	6/11/93	0:00	Primary	< 50	< 10	< 10	< 10	< 10	< 50	< 10	< 10
16WW09	6/10/93	0:00	Primary	< 50	< 10	< 10	< 10	< 10	< 50	< 10	< 10
16WW10	6/9/93	0:00	Primary	< 50	< 10	< 10	< 10	< 10	< 50	< 10	< 10
16WW11	6/9/93	0:00	Primary	< 50	< 10	< 10	< 10	< 10	< 50	< 10	< 10
16WW11	6/9/93	0:00	Duplicate 1	< 50	< 10	< 10	< 10	< 10	< 50	< 10	< 10
MAXIMUM				50	10	10	10	10	50	10	10

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	RESULT TYPE	N-Nitroso dimethylamine (ug/l)	N-Nitroso diphenylamine (ug/l)	N-Nitrosodi-n-propylamine (ug/l)	2-Methylphenol (ug/l)	1,2-Dichloro benzene (ug/l)	2-Nitroaniline (ug/l)	2-Nitrophenol (ug/l)	4-Chloroaniline (ug/l)
122	6/2/93	0:00	Primary		< 10	< 50	< 10	< 10	< 50	< 10	< 10
16EW01	2/21/96	10:55	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW01	2/21/96	16:48	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW01	2/21/96	20:35	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW01	2/24/96	12:35	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW01	2/25/96	23:55	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW01	2/25/96	23:55	Duplicate 1	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW01	2/27/96	12:10	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW01	3/16/96	11:30	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW01	3/17/96	16:10	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW01	3/19/96	11:30	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW02	2/23/96	9:40	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW02	2/23/96	14:00	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW02	2/28/96	12:20	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW02	2/29/96	11:10	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW02	2/29/96	21:25	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW02	3/13/96	11:35	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW02	3/14/96	12:00	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW02	3/14/96	12:00	Duplicate 1	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW02	3/16/96	12:00	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16EW02	3/19/96	12:00	Primary	< 10	< 10	< 10	< 10	< 10	< 25	< 10	< 10
16WW01	6/10/93	0:00	Primary		< 10	< 50	< 10	< 10	< 50	< 10	< 10
16WW02	6/10/93	0:00	Primary		< 10	< 50	< 10	< 10	< 50	< 10	< 10
16WW03	6/8/93	0:00	Primary		< 10	< 50	< 10	< 10	< 50	< 10	< 10
16WW04	6/8/93	0:00	Primary		< 10	< 50	< 10	< 10	< 50	< 10	< 10
16WW05	6/11/93	0:00	Primary		< 10	< 50	< 10	< 10	< 50	< 10	< 10
16WW05	6/11/93	0:00	Duplicate 1		< 10	< 50	< 10	< 10	< 50	< 10	< 10
16WW06	6/11/93	0:00	Primary		< 10	< 50	< 10	< 10	< 50	< 10	< 10
16WW07	6/11/93	0:00	Primary		< 10	< 50	< 10	< 10	< 50	< 10	< 10
16WW08	6/11/93	0:00	Primary		< 10	< 50	< 10	< 10	< 50	< 10	< 10
16WW09	6/10/93	0:00	Primary		< 10	< 50	< 10	< 10	< 50	< 10	< 10
16WW10	6/9/93	0:00	Primary		< 10	< 50	< 10	< 10	< 50	< 10	< 10
16WW11	6/9/93	0:00	Primary		< 10	< 50	< 10	< 10	< 50	< 10	< 10
16WW11	6/9/93	0:00	Duplicate 1		< 10	< 50	< 10	< 10	< 50	< 10	< 10
MAXIMUM				10	10	50	10	10	50	10	10

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	RESULT TYPE	4-Chloro-3-methylphenol (ug/l)	Pentachloro phenol (ug/l)	4-Methylphenol (ug/l)	1,4-Dichloro benzene (ug/l)	Phenanthrene (ug/l)	Phenol (ug/l)	Phenol-d5 (ug/l)	4-Nitroaniline (ug/l)
122	6/2/93	0:00	Primary	< 10	< 50	< 10	< 10	< 10	< 10		< 50
16EW01	2/21/96	10:55	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW01	2/21/96	16:48	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW01	2/21/96	20:35	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW01	2/24/96	12:35	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW01	2/25/96	23:55	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW01	2/25/96	23:55	Duplicate 1	< 10	< 25	< 10	< 10	< 10	< 10		< 25
16EW01	2/27/96	12:10	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW01	3/16/96	11:30	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW01	3/17/96	16:10	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW01	3/19/96	11:30	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW02	2/23/96	9:40	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW02	2/23/96	14:00	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW02	2/28/96	12:20	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW02	2/29/96	11:10	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW02	2/29/96	21:25	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW02	3/13/96	11:35	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW02	3/14/96	12:00	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW02	3/14/96	12:00	Duplicate 1	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW02	3/16/96	12:00	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16EW02	3/19/96	12:00	Primary	< 10	< 25	< 10	< 10	< 10	< 10	0	< 25
16WW01	6/10/93	0:00	Primary	< 10	< 50	< 10	< 10	< 10	< 10		< 50
16WW02	6/10/93	0:00	Primary	< 10	< 50	< 10	< 10	< 10	< 10		< 50
16WW03	6/8/93	0:00	Primary	< 10	< 50	< 10	< 10	< 10	< 10		< 50
16WW04	6/8/93	0:00	Primary	< 10	< 50	< 10	< 10	< 10	< 10		< 50
16WW05	6/11/93	0:00	Primary	< 10	< 50	< 10	< 10	< 10	< 10		< 50
16WW05	6/11/93	0:00	Duplicate 1	< 10	< 50	< 10	< 10	< 10	< 10		< 50
16WW06	6/11/93	0:00	Primary	< 10	< 50	< 10	< 10	< 10	< 10		< 50
16WW07	6/11/93	0:00	Primary	< 10	< 50	< 10	< 10	< 10	< 10		< 50
16WW08	6/11/93	0:00	Primary	< 10	< 50	< 10	< 10	< 10	< 10		< 50
16WW09	6/10/93	0:00	Primary	< 10	< 50	< 10	< 10	< 10	< 10		< 50
16WW10	6/9/93	0:00	Primary	< 10	< 50	< 10	< 10	< 10	< 10		< 50
16WW11	6/9/93	0:00	Primary	< 10	< 50	< 10	< 10	< 10	< 10		< 50
16WW11	6/9/93	0:00	Duplicate 1	< 10	< 50	< 10	< 10	< 10	< 10		< 50
MAXIMUM				10	50	10	10	10	10	0	50

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Semi-Volatile Organic Compounds (SVOCs)

SITE	DATE	TIME	RESULT TYPE		4-Nitrophenol (ug/l)		Pyrene (ug/l)
122	6/2/93	0:00	Primary	<	50	<	10
16EW01	2/21/96	10:55	Primary	<	25	<	10
16EW01	2/21/96	16:48	Primary	<	25	<	10
16EW01	2/21/96	20:35	Primary	<	25	<	10
16EW01	2/24/96	12:35	Primary	<	25	<	10
16EW01	2/25/96	23:55	Primary	<	25	<	10
16EW01	2/25/96	23:55	Duplicate 1	<	25	<	10
16EW01	2/27/96	12:10	Primary	<	25	<	10
16EW01	3/16/96	11:30	Primary	<	25	<	10
16EW01	3/17/96	16:10	Primary	<	25	<	10
16EW01	3/19/96	11:30	Primary	<	25	<	10
16EW02	2/23/96	9:40	Primary	<	25	<	10
16EW02	2/23/96	14:00	Primary	<	25	<	10
16EW02	2/28/96	12:20	Primary	<	25	<	10
16EW02	2/29/96	11:10	Primary	<	25	<	10
16EW02	2/29/96	21:25	Primary	<	25	<	10
16EW02	3/13/96	11:35	Primary	<	25	<	10
16EW02	3/14/96	12:00	Primary	<	25	<	10
16EW02	3/14/96	12:00	Duplicate 1	<	25	<	10
16EW02	3/16/96	12:00	Primary	<	25	<	10
16EW02	3/19/96	12:00	Primary	<	25	<	10
16WW01	6/10/93	0:00	Primary	<	50	<	10
16WW02	6/10/93	0:00	Primary	<	50	<	10
16WW03	6/8/93	0:00	Primary	<	50	<	10
16WW04	6/8/93	0:00	Primary	<	50	<	10
16WW05	6/11/93	0:00	Primary	<	50	<	10
16WW05	6/11/93	0:00	Duplicate 1	<	50	<	10
16WW06	6/11/93	0:00	Primary	<	50	<	10
16WW07	6/11/93	0:00	Primary	<	50	<	10
16WW08	6/11/93	0:00	Primary	<	50	<	10
16WW09	6/10/93	0:00	Primary	<	50	<	10
16WW10	6/9/93	0:00	Primary	<	50	<	10
16WW11	6/9/93	0:00	Primary	<	50	<	10
16WW11	6/9/93	0:00	Duplicate 1	<	50	<	10
MAXIMUM					50		10

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	1,1,1,2-Tetra chloroethane (ug/l)	1,1,1-trichloro ethane (ug/l)	1,1,2,2-Tetra chloroethane (ug/l)	1,1,2-Trichloro ethane (ug/l)	1,1-Dichloro ethane (ug/l)	1,1-Dichloro ethylene (ug/l)	1,1-Dichloro propene (ug/l)	1,2,3-Trichloro benzene (ug/l)
122	6/2/93	0:00	Primary		< 5	< 5	< 5	< 5	< 5		
122	6/14/95	13:42	Primary		< 5	< 5	< 5	< 5	< 5		
16EW01	2/21/96	10:55	Primary		< 5	< 5	< 5	< 5	36		
16EW01	2/21/96	16:48	Primary		< 1250	< 1250	< 1250	< 1250	< 1250		
16EW01	2/21/96	20:35	Primary		< 1250	< 1250	< 1250	< 1250	< 1250		
16EW01	2/24/96	12:35	Primary		< 1250	< 1250	< 1250	< 1250	< 1250		
16EW01	2/25/96	23:55	Primary		< 1250	< 1250	< 1250	< 1250	< 1250		
16EW01	2/25/96	23:55	Duplicate 1		< 1250	< 1250	< 1250	< 1250	< 1250		
16EW01	2/27/96	12:10	Primary		< 1250	< 1250	< 1250	< 1250	< 1250		
16EW01	3/16/96	11:30	Primary		< 1250	< 1250	< 1250	< 1250	< 1250		
16EW01	3/17/96	16:10	Primary		< 1250	< 1250	< 1250	< 1250	< 1250		
16EW01	3/19/96	11:30	Primary		< 1250	< 1250	< 1250	< 1250	< 1250		
16EW01	10/29/97	10:00	Primary	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000
16EW01	10/29/97	10:00	Duplicate 1	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000
16EW01	1/22/98	12:15	Primary	< 40	< 40	< 40	< 40	< 40	< 40	< 40	< 40
16EW01	6/11/98	15:10	Primary	< 40	< 40	< 40	< 40	< 40	< 40	< 40	< 40
16EW01	6/11/98	15:10	Duplicate 1	< 40	< 40	< 40	< 40	< 40	45	< 40	< 40
16EW02	2/23/96	9:40	Primary		< 500	< 500	< 500	< 500	< 500		
16EW02	2/23/96	14:00	Primary		< 500	< 500	< 500	< 500	< 500		
16EW02	2/28/96	12:20	Primary		< 500	< 500	< 500	< 500	< 500		
16EW02	2/29/96	11:10	Primary		< 500	< 500	< 500	< 500	< 500		
16EW02	2/29/96	21:25	Primary		< 500	< 500	< 500	< 500	< 500		
16EW02	3/13/96	11:35	Primary		< 500	< 500	< 500	< 500	< 500		
16EW02	3/14/96	12:00	Primary		< 500	< 500	< 500	< 500	< 500		
16EW02	3/14/96	12:00	Duplicate 1		< 500	< 500	< 500	< 500	< 500		
16EW02	3/16/96	12:00	Primary		< 500	< 500	< 500	< 500	< 500		
16EW02	3/19/96	12:00	Primary		< 500	< 500	< 500	< 500	< 500		
16EW02	10/29/97	10:30	Primary	< 400	< 400	< 400	< 400	< 400	< 400	< 400	< 400
16EW02	1/22/98	12:00	Primary	< 250	< 250	< 250	< 250	< 250	< 250	< 250	< 250
16EW02	6/11/98	15:15	Primary	< 200	< 200	< 200	< 200	< 200	< 200	< 200	< 200
16EW03	10/29/97	11:19	Primary	< 40	< 40	< 40	< 40	< 40	< 40	< 40	< 40
16EW03	1/22/98	11:55	Primary	< 40	< 40	< 40	< 40	< 40	< 40	< 40	< 40
16EW03	6/11/98	15:20	Primary	< 40	< 40	< 40	< 40	< 40	< 40	< 40	< 40
16EW04	10/29/97	10:50	Primary	< 25	< 25	< 25	< 25	< 25	< 25	< 25	< 25
16EW04	1/22/98	11:50	Primary	< 20	< 20	< 20	< 20	< 20	< 20	< 20	< 20
16EW04	6/11/98	15:25	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10
16EW05	10/29/97	11:15	Primary	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	1,1,1,2-Tetra chloroethane (ug/l)	1,1,1-trichloro ethane (ug/l)	1,1,2,2-Tetra chloroethane (ug/l)	1,1,2-Trichloro ethane (ug/l)	1,1-Dichloro ethane (ug/l)	1,1-Dichloro ethylene (ug/l)	1,1-Dichloro propene (ug/l)	1,2,3-Trichloro benzene (ug/l)
16EW05	1/22/98	11:35	Primary	< 500	< 500	< 500	< 500	< 500	< 500	< 500	< 500
16EW05	6/11/98	15:30	Primary	< 500	< 500	< 500	< 500	< 500	< 500	< 500	< 500
16EW06	10/29/97	12:20	Primary	< 400	< 400	< 400	< 400	< 400	< 400	< 400	< 400
16EW06	1/22/98	11:25	Primary	< 20	< 20	< 20	< 20	< 20	< 20	< 20	< 20
16EW06	6/11/98	15:35	Primary	< 20	< 20	< 20	< 20	< 20	< 20	< 20	< 20
16EW07	10/29/97	11:54	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16EW07	1/22/98	11:45	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16EW07	6/11/98	15:40	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16EW08	10/29/97	11:30	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16EW08	1/22/98	10:10	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16EW08	1/22/98	10:10	Duplicate 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16EW08	6/11/98	15:45	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16PB02	3/18/95	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1		
16PB02	3/18/95	0:00	Duplicate 1	< 1	< 1	< 1	< 1	< 1	< 1		
16PB04	3/18/95	0:00	Primary	< 100	< 100	< 100	< 100	< 100	< 100		
16PB05	3/18/95	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1		
16PB06	3/18/95	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1		
16PB07	3/18/95	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1		
16PB08	3/21/95	0:00	Primary	< 10000	< 10000	< 10000	< 10000	< 10000	< 10000		
16PB09	3/18/95	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1		
16PB10	3/20/95	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1		
16PB11	3/20/95	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1		
16PB13	3/21/95	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1		
16PB14	3/21/95	0:00	Primary	< 1	< 1	< 1	3.4	1	170		
16PB16	3/20/95	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1		
16PB17	3/20/95	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1		
16PB19	3/21/95	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1		
16PB20	3/21/95	0:00	Primary	< 100	< 100	< 100	< 100	< 100	< 100		
16PB21	3/20/95	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1		
16PB25	3/28/95	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1		
16PB26	3/28/95	0:00	Primary	< 100	< 100	< 100	< 100	< 100	< 100		
16PB27	3/28/95	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1		
16PB28	3/28/95	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1		
16WW01	6/10/93	0:00	Primary		< 25	< 25	< 25	< 25	< 25		
16WW01	6/14/95	14:18	Primary		< 5	< 5	< 5	< 5	6		
16WW01	6/25/97	0:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW02	6/10/93	0:00	Primary		< 5	< 5	< 5	< 5	< 5		
16WW02	6/13/95	16:30	Primary		< 5	< 5	< 5	< 5	< 5		

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	1,1,1,2-Tetra chloroethane (ug/l)	1,1,1-trichloro ethane (ug/l)	1,1,2,2-Tetra chloroethane (ug/l)	1,1,2-Trichloro ethane (ug/l)	1,1-Dichloro ethane (ug/l)	1,1-Dichloro ethylene (ug/l)	1,1-Dichloro propene (ug/l)	1,2,3-Trichloro benzene (ug/l)
16WW03	6/8/93	0:00	Primary		< 5	< 5	< 5	< 5	< 5		
16WW03	6/14/95	16:04	Primary		< 5	< 5	< 5	< 5	27		
16WW04	6/8/93	0:00	Primary		< 5	< 5	< 5	< 5	< 5		
16WW04	6/15/95	10:58	Primary		< 5	< 5	< 5	< 5	< 5		
16WW04	6/15/95	10:58	Duplicate 1		< 5	< 5	< 5	< 5	< 5		
16WW05	6/11/93	0:00	Primary		< 5	< 5	< 5	< 5	< 5		
16WW05	6/11/93	0:00	Duplicate 1		< 5	< 5	< 5	< 5	< 5		
16WW05	6/14/95	16:48	Primary		< 5	< 5	< 5	< 5	< 5		
16WW05	10/22/97	17:10	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW06	6/11/93	0:00	Primary		< 5	< 5	< 5	< 5	< 5		
16WW06	6/14/95	16:42	Primary		< 5	< 5	< 5	< 5	< 5		
16WW06	10/22/97	14:50	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW07	6/11/93	0:00	Primary		< 5	< 5	< 5	< 5	< 5		
16WW07	6/14/95	12:00	Primary		< 5	< 5	< 5	< 5	< 5		
16WW08	6/11/93	0:00	Primary		< 5	< 5	< 5	< 5	< 5		
16WW08	6/14/95	11:40	Primary		< 5	< 5	< 5	< 5	< 5		
16WW08	6/14/95	11:40	Duplicate 1		< 5	< 5	< 5	< 5	< 5		
16WW09	6/10/93	0:00	Primary		< 5	< 5	< 5	< 5	< 5		
16WW09	6/15/95	12:15	Primary		< 5	< 5	< 5	< 5	< 5		
16WW10	6/9/93	0:00	Primary		< 5	< 5	< 5	< 5	< 5		
16WW10	6/15/95	15:40	Primary		< 5	< 5	< 5	< 5	< 5		
16WW11	6/9/93	0:00	Primary		< 5	< 5	< 5	< 5	< 5		
16WW11	6/9/93	0:00	Duplicate 1		< 5	< 5	< 5	< 5	< 5		
16WW11	6/16/95	11:42	Primary		< 5	< 5	< 5	< 5	< 5		
16WW12	6/13/95	17:45	Primary		< 5	< 5	< 5	< 5	< 5		
16WW12	10/27/97	13:39	Primary	< 1	< 1	< 1	< 1	< 1	7.4	< 1	< 1
16WW12	10/27/97	13:39	Duplicate 1	< 1	< 1	< 1	< 1	< 1	8.1	< 1	< 1
16WW12	1/20/98	16:15	Primary	< 80	< 80	< 80	< 80	< 80	< 80	< 80	< 80
16WW12	6/11/98	12:05	Primary	< 80	< 80	< 80	< 80	< 80	< 80	< 80	< 80
16WW13	6/13/95	16:55	Primary		< 5	< 5	< 5	< 5	< 5		
16WW13	10/27/97	16:05	Primary	< 1	< 1	< 1	< 1	< 1	1.7	< 1	< 1
16WW14	6/13/95	16:45	Primary		< 5	< 5	< 5	< 5	< 5		
16WW14	10/24/97	13:55	Primary	< 1	< 1	< 1	< 1	< 1	(0.53)	< 1	< 1
16WW14	1/20/98	13:30	Primary	< 1	< 1	< 1	< 1	< 1	(0.71)	< 1	< 1
16WW14	1/20/98	13:30	Duplicate 1	< 1	< 1	< 1	< 1	< 1	(0.75)	< 1	< 1
16WW14	6/15/98	9:20	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW15	6/22/95	10:10	Primary		< 5	< 5	< 5	< 5	< 5		
16WW15	10/28/97	15:25	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	1,1,1,2-Tetra chloroethane (ug/l)	1,1,1-trichloro ethane (ug/l)	1,1,2,2-Tetra chloroethane (ug/l)	1,1,2-Trichloro ethane (ug/l)	1,1-Dichloro ethane (ug/l)	1,1-Dichloro ethylene (ug/l)	1,1-Dichloro propene (ug/l)	1,2,3-Trichloro benzene (ug/l)
16WW16	6/15/95	10:40	Primary		< 5	< 5	12	34	603		
16WW16	10/27/97	16:00	Primary	< 10	< 10	< 10	< 10	36	740	< 10	< 10
16WW16	1/21/98	12:15	Primary	< 4000	< 4000	< 4000	< 4000	< 4000	< 4000	< 4000	< 4000
16WW16	6/15/98	11:15	Primary	< 4000	< 4000	< 4000	< 4000	< 4000	< 4000	< 4000	< 4000
16WW17	6/16/95	15:07	Primary		< 5	< 5	< 5	< 5	< 5		
16WW17	10/27/97	16:44	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW18	6/15/95	14:08	Primary		< 5	< 5	< 5	< 5	< 5		
16WW18	10/28/97	15:09	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW19	10/24/97	11:15	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW20	10/24/97	16:15	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW21	10/28/97	14:45	Primary	< 1	< 1	< 1	< 1	< 1	(0.99)	< 1	< 1
16WW22	10/24/97	13:44	Primary	< 1	< 1	< 1	< 1	(0.60)	3.8	< 1	< 1
16WW22	1/20/98	14:50	Primary	< 50	< 50	< 50	< 50	< 50	< 50	< 50	< 50
16WW22	6/11/98	10:15	Primary	< 50	< 50	< 50	< 50	< 50	< 50	< 50	< 50
16WW23	10/23/97	10:39	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW24	10/23/97	12:49	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW25	10/23/97	10:39	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW25	1/21/98	17:35	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW25	6/12/98	11:10	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW26	10/23/97	12:49	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW26	1/21/98	17:00	Primary	< 8	< 8	< 8	< 8	< 8	< 8	< 8	< 8
16WW26	6/12/98	9:30	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW27	10/21/97	10:50	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW28	10/21/97	12:55	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW29	10/21/97	10:55	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW29	1/22/98	13:15	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW29	6/11/98	14:30	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW29	6/11/98	14:30	Duplicate 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW30	10/21/97	12:40	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW30	1/22/98	13:30	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW30	6/15/98	14:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW31	10/22/97	10:09	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW31	10/22/97	10:09	Duplicate 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW32	10/22/97	12:40	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW33	10/22/97	10:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW34	10/22/97	12:20	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW35	10/24/97	11:10	Primary	< 1	< 1	< 1	< 1	2.2	24	< 1	< 1
16WW35	1/21/98	11:30	Primary	< 8	< 8	< 8	< 8	< 8	< 8	< 8	< 8

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	1,1,1,2-Tetra chloroethane (ug/l)	1,1,1-trichloro ethane (ug/l)	1,1,2,2-Tetra chloroethane (ug/l)	1,1,2-Trichloro ethane (ug/l)	1,1-Dichloro ethane (ug/l)	1,1-Dichloro ethylene (ug/l)	1,1-Dichloro propene (ug/l)	1,2,3-Trichloro benzene (ug/l)
16WW35	6/12/98	13:35	Primary	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5
16WW36	10/27/97	13:50	Primary	< 10	< 10	< 10	< 10	< 10	16	< 10	< 10
16WW36	1/20/98	16:15	Primary	< 200	< 200	< 200	< 200	< 200	< 200	< 200	< 200
16WW36	6/12/98	15:30	Primary	< 200	< 200	< 200	< 200	< 200	< 200	< 200	< 200
16WW37	10/21/97	16:39	Primary	< 50	< 50	< 50	< 50	< 50	< 50	< 50	< 50
16WW37	10/21/97	16:39	Duplicate 1	< 50	< 50	< 50	< 50	< 50	< 50	< 50	< 50
16WW37	1/21/98	13:30	Primary	< 25	< 25	< 25	< 25	< 25	< 25	< 25	< 25
16WW37	6/14/98	11:00	Primary	< 25	< 25	< 25	< 25	< 25	< 25	< 25	< 25
16WW38	10/22/97	16:09	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW38	1/21/98	13:00	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10
16WW38	6/14/98	12:45	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
MAXIMUM				10000	10000	10000	10000	10000	10000	4000	4000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	1,2,3-Trichloro propane (ug/l)	1,2,4-Trichloro benzene (ug/l)	1,2-Dichloro ethane (ug/l)	1,2-Dichloro ethene (ug/l)	1,2-Dichloro propane (ug/l)	1,3-Dichloro propane (ug/l)	1,4-Dichloro-2-butene (ug/l)	1,4-Dioxane (ug/l)
122	6/2/93	0:00	Primary	< 5		< 5		< 5		< 10	
122	6/14/95	13:42	Primary	< 5		< 5	< 5	< 5		< 10	
16EW01	2/21/96	10:55	Primary			85	1665	< 5			
16EW01	2/21/96	16:48	Primary			< 1250	< 1250	< 1250			
16EW01	2/21/96	20:35	Primary			< 1250	2578	< 1250			
16EW01	2/24/96	12:35	Primary			< 1250	< 1250	< 1250			
16EW01	2/25/96	23:55	Primary			< 1250	2430	< 1250			
16EW01	2/25/96	23:55	Duplicate 1			< 1250	2303	< 1250			
16EW01	2/27/96	12:10	Primary			< 1250	2468	< 1250			
16EW01	3/16/96	11:30	Primary			< 1250	< 1250	< 1250			
16EW01	3/17/96	16:10	Primary			< 1250	< 1250	< 1250			
16EW01	3/19/96	11:30	Primary			< 1250	< 1250	< 1250			
16EW01	10/29/97	10:00	Primary	< 1000	< 1000	< 1000		< 1000	< 1000	< 10000	< 200000
16EW01	10/29/97	10:00	Duplicate 1	< 1000	< 1000	< 1000		< 1000	< 1000	< 10000	< 200000
16EW01	1/22/98	12:15	Primary	< 40	< 40	< 40		< 40	< 40	< 400	< 8000
16EW01	6/11/98	15:10	Primary	< 40	< 40	140		< 40	< 40	< 400	< 8000
16EW01	6/11/98	15:10	Duplicate 1	< 40	< 40	160		< 40	< 40	< 400	< 8000
16EW02	2/23/96	9:40	Primary			< 500	< 500	< 500			
16EW02	2/23/96	14:00	Primary			< 500	< 500	< 500			
16EW02	2/28/96	12:20	Primary			< 500	< 500	< 500			
16EW02	2/29/96	11:10	Primary			< 500	< 500	< 500			
16EW02	2/29/96	21:25	Primary			< 500	< 500	< 500			
16EW02	3/13/96	11:35	Primary			< 500	< 500	< 500			
16EW02	3/14/96	12:00	Primary			< 500	< 500	< 500			
16EW02	3/14/96	12:00	Duplicate 1			< 500	< 500	< 500			
16EW02	3/16/96	12:00	Primary			< 500	< 500	< 500			
16EW02	3/19/96	12:00	Primary			< 500	< 500	< 500			
16EW02	10/29/97	10:30	Primary	< 400	< 400	< 400		< 400	< 400	< 4000	< 80000
16EW02	1/22/98	12:00	Primary	< 250	< 250	< 250		< 250	< 250	< 2500	< 50000
16EW02	6/11/98	15:15	Primary	< 200	< 200	< 200		< 200	< 200	< 2000	< 40000
16EW03	10/29/97	11:19	Primary	< 40	< 40	< 40		< 40	< 40	< 400	< 8000
16EW03	1/22/98	11:55	Primary	< 40	< 40	< 40		< 40	< 40	< 400	< 8000
16EW03	6/11/98	15:20	Primary	< 40	< 40	< 40		< 40	< 40	< 400	< 8000
16EW04	10/29/97	10:50	Primary	< 25	< 25	< 25		< 25	< 25	< 250	< 5000
16EW04	1/22/98	11:50	Primary	< 20	< 20	< 20		< 20	< 20	< 200	< 4000
16EW04	6/11/98	15:25	Primary	< 10	< 10	< 10		< 10	< 10	< 100	< 2000
16EW05	10/29/97	11:15	Primary	< 1000	< 1000	< 1000		< 1000	< 1000	< 10000	< 200000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	1,2,3-Trichloro propane (ug/l)	1,2,4-Trichloro benzene (ug/l)	1,2-Dichloro ethane (ug/l)	1,2-Dichloro ethene (ug/l)	1,2-Dichloro propane (ug/l)	1,3-Dichloro propane (ug/l)	1,4-Dichloro-2-butene (ug/l)	1,4-Dioxane (ug/l)
16EW05	1/22/98	11:35	Primary	< 500	< 500	< 500		< 500	< 500	< 5000	< 100000
16EW05	6/11/98	15:30	Primary	< 500	< 500	< 500		< 500	< 500	< 5000	< 100000
16EW06	10/29/97	12:20	Primary	< 400	< 400	< 400		< 400	< 400	< 4000	< 80000
16EW06	1/22/98	11:25	Primary	< 20	< 20	< 20		< 20	< 20	< 200	< 4000
16EW06	6/11/98	15:35	Primary	< 20	< 20	< 20		< 20	< 20	< 200	< 4000
16EW07	10/29/97	11:54	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16EW07	1/22/98	11:45	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16EW07	6/11/98	15:40	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16EW08	10/29/97	11:30	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16EW08	1/22/98	10:10	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16EW08	1/22/98	10:10	Duplicate 1	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16EW08	6/11/98	15:45	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16PB02	3/18/95	0:00	Primary	< 1		< 1		< 1		< 1	
16PB02	3/18/95	0:00	Duplicate 1	< 1		< 1		< 1		< 1	
16PB04	3/18/95	0:00	Primary	< 100		< 100		< 100		< 100	
16PB05	3/18/95	0:00	Primary	< 1		< 1		< 1		< 1	
16PB06	3/18/95	0:00	Primary	< 1		< 1		< 1		< 1	
16PB07	3/18/95	0:00	Primary	< 1		< 1		< 1		< 1	
16PB08	3/21/95	0:00	Primary	< 10000		< 10000		< 10000		< 10000	
16PB09	3/18/95	0:00	Primary	< 1		< 1		< 1		< 1	
16PB10	3/20/95	0:00	Primary	< 1		< 1		< 1		< 1	
16PB11	3/20/95	0:00	Primary	< 1		< 1		< 1		< 1	
16PB13	3/21/95	0:00	Primary	< 1		< 1		< 1		< 1	
16PB14	3/21/95	0:00	Primary	< 1		11		< 1		< 1	
16PB16	3/20/95	0:00	Primary	< 1		< 1		< 1		< 1	
16PB17	3/20/95	0:00	Primary	< 1		< 1		< 1		< 1	
16PB19	3/21/95	0:00	Primary	< 1		< 1		< 1		< 1	
16PB20	3/21/95	0:00	Primary	< 100		< 100		< 100		< 100	
16PB21	3/20/95	0:00	Primary	< 1		1.5		< 1		< 1	
16PB25	3/28/95	0:00	Primary	< 1		< 1		< 1		< 1	
16PB26	3/28/95	0:00	Primary	< 100		< 100		< 100		< 100	
16PB27	3/28/95	0:00	Primary	< 1		< 1		< 1		< 1	
16PB28	3/28/95	0:00	Primary	< 1		< 1		< 1		< 1	
16WW01	6/10/93	0:00	Primary	< 25		< 25		< 25		< 50	
16WW01	6/14/95	14:18	Primary	< 5		< 5	321	< 5		< 10	
16WW01	6/25/97	0:00	Primary	< 1	< 1	< 1		< 1	< 1		< 200
16WW02	6/10/93	0:00	Primary	< 5		< 5		< 5		< 10	
16WW02	6/13/95	16:30	Primary	< 5		< 5	< 5	< 5		< 10	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	1,2,3-Trichloro propane (ug/l)	1,2,4-Trichloro benzene (ug/l)	1,2-Dichloro ethane (ug/l)	1,2-Dichloro ethene (ug/l)	1,2-Dichloro propane (ug/l)	1,3-Dichloro propane (ug/l)	1,4-Dichloro-2-butene (ug/l)	1,4-Dioxane (ug/l)
16WW03	6/8/93	0:00	Primary	< 5		< 5		< 5		< 10	
16WW03	6/14/95	16:04	Primary	< 5		< 5	66	< 5		< 10	
16WW04	6/8/93	0:00	Primary	< 5		< 5		< 5		< 10	
16WW04	6/15/95	10:58	Primary	< 5		< 5	41	< 5		< 10	
16WW04	6/15/95	10:58	Duplicate 1	< 5		< 5	40	< 5		< 10	
16WW05	6/11/93	0:00	Primary	< 5		< 5		< 5		< 10	
16WW05	6/11/93	0:00	Duplicate 1	< 5		< 5		< 5		< 10	
16WW05	6/14/95	16:48	Primary	< 5		< 5	< 5	< 5		< 10	
16WW05	10/22/97	17:10	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW06	6/11/93	0:00	Primary	< 5		< 5		< 5		< 10	
16WW06	6/14/95	16:42	Primary	< 5		< 5	< 5	< 5		< 10	
16WW06	10/22/97	14:50	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW07	6/11/93	0:00	Primary	< 5		< 5		< 5		< 10	
16WW07	6/14/95	12:00	Primary	< 5		< 5	< 5	< 5		< 10	
16WW08	6/11/93	0:00	Primary	< 5		< 5		< 5		< 10	
16WW08	6/14/95	11:40	Primary	< 5		< 5	< 5	< 5		< 10	
16WW08	6/14/95	11:40	Duplicate 1	< 5		< 5	< 5	< 5		< 10	
16WW09	6/10/93	0:00	Primary	< 5		< 5		< 5		< 10	
16WW09	6/15/95	12:15	Primary	< 5		< 5	16	< 5		< 10	
16WW10	6/9/93	0:00	Primary	< 5		< 5		< 5		< 10	
16WW10	6/15/95	15:40	Primary	< 5		< 5	27	< 5		< 10	
16WW11	6/9/93	0:00	Primary	< 5		< 5		< 5		< 10	
16WW11	6/9/93	0:00	Duplicate 1	< 5		< 5		< 5		< 10	
16WW11	6/16/95	11:42	Primary	< 5		< 5	23	< 5		< 10	
16WW12	6/13/95	17:45	Primary	< 5		< 5	< 5	< 5		< 10	
16WW12	10/27/97	13:39	Primary	< 1	< 1	24		< 1	< 1	< 10	< 200
16WW12	10/27/97	13:39	Duplicate 1	< 1	< 1	24		< 1	< 1	< 10	< 200
16WW12	1/20/98	16:15	Primary	< 80	< 80	< 80		< 80	< 80	< 800	< 16000
16WW12	6/11/98	12:05	Primary	< 80	< 80	< 80		< 80	< 80	< 800	< 16000
16WW13	6/13/95	16:55	Primary	< 5		< 5	< 5	< 5		< 10	
16WW13	10/27/97	16:05	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW14	6/13/95	16:45	Primary	< 5		< 5	< 5	< 5		< 10	
16WW14	10/24/97	13:55	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW14	1/20/98	13:30	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW14	1/20/98	13:30	Duplicate 1	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW14	6/15/98	9:20	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW15	6/22/95	10:10	Primary	< 5		< 5	< 5	< 5		< 10	
16WW15	10/28/97	15:25	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	1,2,3-Trichloro propane (ug/l)	1,2,4-Trichloro benzene (ug/l)	1,2-Dichloro ethane (ug/l)	1,2-Dichloro ethene (ug/l)	1,2-Dichloro propane (ug/l)	1,3-Dichloro propane (ug/l)	1,4-Dichloro-2-butene (ug/l)	1,4-Dioxane (ug/l)
16WW16	6/15/95	10:40	Primary	< 5		103	275000	< 5		< 10	
16WW16	10/27/97	16:00	Primary	< 10	< 10	96		< 10	< 10	< 100	< 2000
16WW16	1/21/98	12:15	Primary	< 4000	< 4000	< 4000		< 4000	< 4000	< 40000	< 800000
16WW16	6/15/98	11:15	Primary	< 4000	< 4000	< 4000		< 4000	< 4000	< 40000	< 800000
16WW17	6/16/95	15:07	Primary	< 5		< 5	< 5	< 5		< 10	
16WW17	10/27/97	16:44	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW18	6/15/95	14:08	Primary	< 5		< 5	< 5	< 5		< 10	
16WW18	10/28/97	15:09	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW19	10/24/97	11:15	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW20	10/24/97	16:15	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW21	10/28/97	14:45	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW22	10/24/97	13:44	Primary	< 1	< 1	22		< 1	< 1	< 10	< 200
16WW22	1/20/98	14:50	Primary	< 50	< 50	< 50		< 50	< 50	< 500	< 10000
16WW22	6/11/98	10:15	Primary	< 50	< 50	< 50		< 50	< 50	< 500	< 10000
16WW23	10/23/97	10:39	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW24	10/23/97	12:49	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW25	10/23/97	10:39	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW25	1/21/98	17:35	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW25	6/12/98	11:10	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW26	10/23/97	12:49	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW26	1/21/98	17:00	Primary	< 8	< 8	< 8		< 8	< 8	< 80	< 1600
16WW26	6/12/98	9:30	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW27	10/21/97	10:50	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW28	10/21/97	12:55	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW29	10/21/97	10:55	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW29	1/22/98	13:15	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW29	6/11/98	14:30	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW29	6/11/98	14:30	Duplicate 1	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW30	10/21/97	12:40	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW30	1/22/98	13:30	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW30	6/15/98	14:00	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW31	10/22/97	10:09	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW31	10/22/97	10:09	Duplicate 1	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW32	10/22/97	12:40	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW33	10/22/97	10:00	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW34	10/22/97	12:20	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW35	10/24/97	11:10	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW35	1/21/98	11:30	Primary	< 8	< 8	< 8		< 8	< 8	< 80	< 1600

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	1,2,3-Trichloro propane (ug/l)	1,2,4-Trichloro benzene (ug/l)	1,2-Dichloro ethane (ug/l)	1,2-Dichloro ethene (ug/l)	1,2-Dichloro propane (ug/l)	1,3-Dichloro propane (ug/l)	1,4-Dichloro-2-butene (ug/l)	1,4-Dioxane (ug/l)
16WW35	6/12/98	13:35	Primary	< 5	< 5	< 5		< 5	< 5	< 50	< 1000
16WW36	10/27/97	13:50	Primary	< 10	< 10	< 10		< 10	< 10	< 100	< 2000
16WW36	1/20/98	16:15	Primary	< 200	< 200	< 200		< 200	< 200	< 2000	< 40000
16WW36	6/12/98	15:30	Primary	< 200	< 200	< 200		< 200	< 200	< 2000	< 40000
16WW37	10/21/97	16:39	Primary	< 50	< 50	< 50		< 50	< 50	< 500	< 10000
16WW37	10/21/97	16:39	Duplicate 1	< 50	< 50	< 50		< 50	< 50	< 500	< 10000
16WW37	1/21/98	13:30	Primary	< 25	< 25	< 25		< 25	< 25	< 250	< 5000
16WW37	6/14/98	11:00	Primary	< 25	< 25	< 25		< 25	< 25	< 250	< 5000
16WW38	10/22/97	16:09	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
16WW38	1/21/98	13:00	Primary	< 10	< 10	< 10		< 10	< 10	< 100	< 2000
16WW38	6/14/98	12:45	Primary	< 1	< 1	< 1		< 1	< 1	< 10	< 200
MAXIMUM				10000	4000	10000	275000	10000	4000	40000	800000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	2,2-Dichloro propane (ug/l)	2-Hexanone (ug/l)	Acetone (ug/l)	Acrolein (ug/l)	Acrylonitrile (ug/l)	Benzene (ug/l)	Benzene, 1,2,4-trimethyl (ug/l)	Benzene, 1,3,5-trimethyl- (ug/l)
122	6/2/93	0:00	Primary		< 10	< 10	< 10	< 10	< 5		
122	6/14/95	13:42	Primary		< 10	10	< 50	< 50	< 5		
16EW01	2/21/96	10:55	Primary		< 10	< 10			< 5		
16EW01	2/21/96	16:48	Primary		< 2500	3920			< 1250		
16EW01	2/21/96	20:35	Primary		< 2500	< 2500			< 1250		
16EW01	2/24/96	12:35	Primary		< 2500	< 2500			< 1250		
16EW01	2/25/96	23:55	Primary		< 2500	< 2500			< 1250		
16EW01	2/25/96	23:55	Duplicate 1		< 2500	< 2500			< 1250		
16EW01	2/27/96	12:10	Primary		< 2500	< 2500			< 1250		
16EW01	3/16/96	11:30	Primary		< 2500	< 2500			< 1250		
16EW01	3/17/96	16:10	Primary		< 2500	< 2500			< 1250		
16EW01	3/19/96	11:30	Primary		< 2500	< 2500			< 1250		
16EW01	10/29/97	10:00	Primary	< 1000	< 5000	< 5000	< 20000	< 20000	< 1000	< 1000	< 1000
16EW01	10/29/97	10:00	Duplicate 1	< 1000	< 5000	< 5000	< 20000	< 20000	< 1000	< 1000	< 1000
16EW01	1/22/98	12:15	Primary	< 40	< 200	< 200	< 800	< 800	< 40	< 40	< 40
16EW01	6/11/98	15:10	Primary	< 40	< 200	< 200	< 800	< 800	< 40	< 40	< 40
16EW01	6/11/98	15:10	Duplicate 1	< 40	< 200	< 200	< 800	< 800	< 40	< 40	< 40
16EW02	2/23/96	9:40	Primary		< 1000	< 1000			< 500		
16EW02	2/23/96	14:00	Primary		< 1000	< 1000			< 500		
16EW02	2/28/96	12:20	Primary		< 1000	< 1000			< 500		
16EW02	2/29/96	11:10	Primary		< 1000	< 1000			< 500		
16EW02	2/29/96	21:25	Primary		< 1000	< 1000			< 500		
16EW02	3/13/96	11:35	Primary		< 1000	< 1000			< 500		
16EW02	3/14/96	12:00	Primary		< 1000	< 1000			< 500		
16EW02	3/14/96	12:00	Duplicate 1		< 1000	< 1000			< 500		
16EW02	3/16/96	12:00	Primary		< 1000	< 1000			< 500		
16EW02	3/19/96	12:00	Primary		< 1000	< 1000			< 500		
16EW02	10/29/97	10:30	Primary	< 400	< 2000	< 2000	< 8000	< 8000	< 400	< 400	< 400
16EW02	1/22/98	12:00	Primary	< 250	< 1200	< 1200	< 5000	< 5000	< 250	< 250	< 250
16EW02	6/11/98	15:15	Primary	< 200	< 1000	< 1000	< 4000	< 4000	< 200	< 200	< 200
16EW03	10/29/97	11:19	Primary	< 40	< 200	< 200	< 800	< 800	< 40	< 40	< 40
16EW03	1/22/98	11:55	Primary	< 40	< 200	< 200	< 800	< 800	< 40	< 40	< 40
16EW03	6/11/98	15:20	Primary	< 40	< 200	< 200	< 800	< 800	< 40	< 40	< 40
16EW04	10/29/97	10:50	Primary	< 25	< 120	< 120	< 500	< 500	< 25	< 25	< 25
16EW04	1/22/98	11:50	Primary	< 20	< 100	< 100	< 400	< 400	< 20	< 20	< 20
16EW04	6/11/98	15:25	Primary	< 10	< 50	< 50	< 200	< 200	< 10	< 10	< 10
16EW05	10/29/97	11:15	Primary	< 1000	< 5000	< 5000	< 20000	< 20000	< 1000	< 1000	< 1000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	2,2-Dichloro propane (ug/l)	2-Hexanone (ug/l)	Acetone (ug/l)	Acrolein (ug/l)	Acrylonitrile (ug/l)	Benzene (ug/l)	Benzene, 1,2,4-trimethyl (ug/l)	Benzene, 1,3,5-trimethyl- (ug/l)
16EW05	1/22/98	11:35	Primary	< 500	< 2500	< 2500	< 10000	< 10000	< 500	< 500	< 500
16EW05	6/11/98	15:30	Primary	< 500	< 2500	< 2500	< 10000	< 10000	< 500	< 500	< 500
16EW06	10/29/97	12:20	Primary	< 400	< 2000	< 2000	< 8000	< 8000	< 400	< 400	< 400
16EW06	1/22/98	11:25	Primary	< 20	< 100	< 100	< 400	< 400	< 20	< 20	< 20
16EW06	6/11/98	15:35	Primary	< 20	< 100	< 100	< 400	< 400	< 20	< 20	< 20
16EW07	10/29/97	11:54	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16EW07	1/22/98	11:45	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16EW07	6/11/98	15:40	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16EW08	10/29/97	11:30	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16EW08	1/22/98	10:10	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16EW08	1/22/98	10:10	Duplicate 1	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16EW08	6/11/98	15:45	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16PB02	3/18/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		
16PB02	3/18/95	0:00	Duplicate 1		< 1	< 1	< 1	< 1	< 1		
16PB04	3/18/95	0:00	Primary		< 100	< 100	< 100	< 100	< 100		
16PB05	3/18/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		
16PB06	3/18/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		
16PB07	3/18/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		
16PB08	3/21/95	0:00	Primary		< 10000	< 10000	< 10000	< 10000	< 10000		
16PB09	3/18/95	0:00	Primary		< 1	< 1	< 1	< 1	1		
16PB10	3/20/95	0:00	Primary		< 1	2.4	< 1	< 1	< 1		
16PB11	3/20/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		
16PB13	3/21/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		
16PB14	3/21/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		
16PB16	3/20/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		
16PB17	3/20/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		
16PB19	3/21/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		
16PB20	3/21/95	0:00	Primary		< 100	< 100	< 100	< 100	< 100		
16PB21	3/20/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		
16PB25	3/28/95	0:00	Primary		< 1	< 1	< 1	< 1	1		
16PB26	3/28/95	0:00	Primary		< 100	< 100	< 100	< 100	< 100		
16PB27	3/28/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		
16PB28	3/28/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		
16WW01	6/10/93	0:00	Primary		< 50	< 50	< 50	< 50	< 25		
16WW01	6/14/95	14:18	Primary		< 10	< 10	< 50	< 50	< 5		
16WW01	6/25/97	0:00	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW02	6/10/93	0:00	Primary		< 10	< 10	< 10	< 10	< 5		
16WW02	6/13/95	16:30	Primary		< 10	< 10	< 50	< 50	< 5		

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	2,2-Dichloro propane (ug/l)	2-Hexanone (ug/l)	Acetone (ug/l)	Acrolein (ug/l)	Acrylonitrile (ug/l)	Benzene (ug/l)	Benzene, 1,2,4-trimethyl (ug/l)	Benzene, 1,3,5-trimethyl- (ug/l)
16WW03	6/8/93	0:00	Primary		< 10	< 10	< 10	< 10	< 5		
16WW03	6/14/95	16:04	Primary		< 10	< 10	< 50	< 50	< 5		
16WW04	6/8/93	0:00	Primary		< 10	< 10	< 10	< 10	< 5		
16WW04	6/15/95	10:58	Primary		< 10	< 10	< 50	< 50	< 5		
16WW04	6/15/95	10:58	Duplicate 1		< 10	< 10	< 50	< 50	< 5		
16WW05	6/11/93	0:00	Primary		< 10	< 10	< 10	< 10	< 5		
16WW05	6/11/93	0:00	Duplicate 1		< 10	23	< 10	< 10	< 5		
16WW05	6/14/95	16:48	Primary		< 10	< 10	< 50	< 50	< 5		
16WW05	10/22/97	17:10	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW06	6/11/93	0:00	Primary		< 10	17	< 10	< 10	< 5		
16WW06	6/14/95	16:42	Primary		< 10	< 10	< 50	< 50	< 5		
16WW06	10/22/97	14:50	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW07	6/11/93	0:00	Primary		< 10	< 10	< 10	< 10	< 5		
16WW07	6/14/95	12:00	Primary		< 10	< 10	< 50	< 50	< 5		
16WW08	6/11/93	0:00	Primary		< 10	< 10	< 10	< 10	< 5		
16WW08	6/14/95	11:40	Primary		< 10	< 10	< 50	< 50	< 5		
16WW08	6/14/95	11:40	Duplicate 1		< 10	< 10	< 50	< 50	< 5		
16WW09	6/10/93	0:00	Primary		< 10	< 10	< 10	< 10	< 5		
16WW09	6/15/95	12:15	Primary		< 10	< 10	< 50	< 50	< 5		
16WW10	6/9/93	0:00	Primary		< 10	< 10	< 10	< 10	< 5		
16WW10	6/15/95	15:40	Primary		< 10	< 10	< 50	< 50	< 5		
16WW11	6/9/93	0:00	Primary		< 10	< 10	< 10	< 10	< 5		
16WW11	6/9/93	0:00	Duplicate 1		< 10	< 10	< 10	< 10	< 5		
16WW11	6/16/95	11:42	Primary		< 10	< 10	< 50	< 50	< 5		
16WW12	6/13/95	17:45	Primary		< 10	< 10	< 50	< 50	< 5		
16WW12	10/27/97	13:39	Primary	< 1	< 5	< 5	< 20	< 20	4.1	< 1	< 1
16WW12	10/27/97	13:39	Duplicate 1	< 1	< 5	< 5	< 20	< 20	4.1	< 1	< 1
16WW12	1/20/98	16:15	Primary	< 80	< 400	< 400	< 1600	< 1600	< 80	< 80	< 80
16WW12	6/11/98	12:05	Primary	< 80	< 400	< 400	< 1600	< 1600	< 80	< 80	< 80
16WW13	6/13/95	16:55	Primary		< 10	< 10	< 50	< 50	< 5		
16WW13	10/27/97	16:05	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW14	6/13/95	16:45	Primary		< 10	< 10	< 50	< 50	< 5		
16WW14	10/24/97	13:55	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW14	1/20/98	13:30	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW14	1/20/98	13:30	Duplicate 1	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW14	6/15/98	9:20	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW15	6/22/95	10:10	Primary		< 10	< 10	< 50	< 50	< 5		
16WW15	10/28/97	15:25	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	2,2-Dichloro propane (ug/l)	2-Hexanone (ug/l)	Acetone (ug/l)	Acrolein (ug/l)	Acrylonitrile (ug/l)	Benzene (ug/l)	Benzene, 1,2,4-trimethyl (ug/l)	Benzene, 1,3,5-trimethyl- (ug/l)
16WW16	6/15/95	10:40	Primary		< 10	< 10	< 50	< 50	5		
16WW16	10/27/97	16:00	Primary	< 10	< 50	< 50	< 200	< 200	< 10	24	16
16WW16	1/21/98	12:15	Primary	< 4000	< 20000	< 20000	< 80000	< 80000	< 4000	< 4000	< 4000
16WW16	6/15/98	11:15	Primary	< 4000	< 20000	< 20000	< 80000	< 80000	< 4000	< 4000	< 4000
16WW17	6/16/95	15:07	Primary		< 10	10	< 50	< 50	< 5		
16WW17	10/27/97	16:44	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW18	6/15/95	14:08	Primary		< 10	< 10	< 50	< 50	< 5		
16WW18	10/28/97	15:09	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW19	10/24/97	11:15	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW20	10/24/97	16:15	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW21	10/28/97	14:45	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW22	10/24/97	13:44	Primary	< 1	< 5	< 5	< 20	< 20	1.8	< 1	< 1
16WW22	1/20/98	14:50	Primary	< 50	< 250	< 250	< 1000	< 1000	< 50	< 50	< 50
16WW22	6/11/98	10:15	Primary	< 50	< 250	< 250	< 1000	< 1000	< 50	< 50	< 50
16WW23	10/23/97	10:39	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW24	10/23/97	12:49	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW25	10/23/97	10:39	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW25	1/21/98	17:35	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW25	6/12/98	11:10	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW26	10/23/97	12:49	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW26	1/21/98	17:00	Primary	< 8	< 40	< 40	< 160	< 160	< 8	< 8	< 8
16WW26	6/12/98	9:30	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW27	10/21/97	10:50	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW28	10/21/97	12:55	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW29	10/21/97	10:55	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW29	1/22/98	13:15	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW29	6/11/98	14:30	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW29	6/11/98	14:30	Duplicate 1	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW30	10/21/97	12:40	Primary	< 1	< 5	< 5	< 20	< 20	(0.83)	< 1	< 1
16WW30	1/22/98	13:30	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW30	6/15/98	14:00	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW31	10/22/97	10:09	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW31	10/22/97	10:09	Duplicate 1	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW32	10/22/97	12:40	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW33	10/22/97	10:00	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW34	10/22/97	12:20	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW35	10/24/97	11:10	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW35	1/21/98	11:30	Primary	< 8	< 40	< 40	< 160	< 160	< 8	< 8	< 8

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	2,2-Dichloro propane (ug/l)	2-Hexanone (ug/l)	Acetone (ug/l)	Acrolein (ug/l)	Acrylonitrile (ug/l)	Benzene (ug/l)	Benzene, 1,2,4-trimethyl (ug/l)	Benzene, 1,3,5-trimethyl- (ug/l)
16WW35	6/12/98	13:35	Primary	< 5	< 25	< 25	< 100	< 100	< 5	< 5	< 5
16WW36	10/27/97	13:50	Primary	< 10	< 50	< 50	< 200	< 200	< 10	< 10	< 10
16WW36	1/20/98	16:15	Primary	< 200	< 1000	< 1000	< 4000	< 4000	< 200	< 200	< 200
16WW36	6/12/98	15:30	Primary	< 200	< 1000	< 1000	< 4000	< 4000	< 200	< 200	< 200
16WW37	10/21/97	16:39	Primary	< 50	< 250	< 250	< 1000	< 1000	< 50	< 50	< 50
16WW37	10/21/97	16:39	Duplicate 1	< 50	< 250	< 250	< 1000	< 1000	< 50	< 50	< 50
16WW37	1/21/98	13:30	Primary	< 25	< 120	< 120	< 500	< 500	< 25	< 25	< 25
16WW37	6/14/98	11:00	Primary	< 25	< 120	< 120	< 500	< 500	< 25	< 25	< 25
16WW38	10/22/97	16:09	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
16WW38	1/21/98	13:00	Primary	< 10	< 50	< 50	< 200	< 200	< 10	< 10	< 10
16WW38	6/14/98	12:45	Primary	< 1	< 5	< 5	< 20	< 20	< 1	< 1	< 1
MAXIMUM				4000	20000	20000	80000	80000	10000	4000	4000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Benzene, 1-methylethyl- (ug/l)	Bromobenzene (ug/l)	Bromodichloro methane (ug/l)	Bromoform (ug/l)	Carbon disulfide (ug/l)	Carbon tetrachloride (ug/l)	Chlorobenzene (ug/l)	Chlorobromo methane (ug/l)
122	6/2/93	0:00	Primary			< 5		< 5	< 5	< 5	
122	6/14/95	13:42	Primary			< 5	< 5	< 5	< 5	< 5	
16EW01	2/21/96	10:55	Primary			< 5	< 5	< 5	< 5	< 5	
16EW01	2/21/96	16:48	Primary			< 1250	< 1250	< 1250	< 1250	< 1250	
16EW01	2/21/96	20:35	Primary			< 1250	< 1250	< 1250	< 1250	< 1250	
16EW01	2/24/96	12:35	Primary			< 1250	< 1250	< 1250	< 1250	< 1250	
16EW01	2/25/96	23:55	Primary			< 1250	< 1250	< 1250	< 1250	< 1250	
16EW01	2/25/96	23:55	Duplicate 1			< 1250	< 1250	< 1250	< 1250	< 1250	
16EW01	2/27/96	12:10	Primary			< 1250	< 1250	< 1250	< 1250	< 1250	
16EW01	3/16/96	11:30	Primary			< 1250	< 1250	< 1250	< 1250	< 1250	
16EW01	3/17/96	16:10	Primary			< 1250	< 1250	< 1250	< 1250	< 1250	
16EW01	3/19/96	11:30	Primary			< 1250	< 1250	< 1250	< 1250	< 1250	
16EW01	10/29/97	10:00	Primary	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000
16EW01	10/29/97	10:00	Duplicate 1	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000
16EW01	1/22/98	12:15	Primary	< 40	< 40	< 40	< 40	< 40	< 40	< 40	< 40
16EW01	6/11/98	15:10	Primary	< 40	< 40	< 40	< 40	< 40	< 40	< 40	< 40
16EW01	6/11/98	15:10	Duplicate 1	< 40	< 40	< 40	< 40	< 40	< 40	< 40	< 40
16EW02	2/23/96	9:40	Primary			< 500	< 500	< 500	< 500	< 500	
16EW02	2/23/96	14:00	Primary			< 500	< 500	< 500	< 500	< 500	
16EW02	2/28/96	12:20	Primary			< 500	< 500	< 500	< 500	< 500	
16EW02	2/29/96	11:10	Primary			< 500	< 500	< 500	< 500	< 500	
16EW02	2/29/96	21:25	Primary			< 500	< 500	< 500	< 500	< 500	
16EW02	3/13/96	11:35	Primary			< 500	< 500	< 500	< 500	< 500	
16EW02	3/14/96	12:00	Primary			< 500	< 500	< 500	< 500	< 500	
16EW02	3/14/96	12:00	Duplicate 1			< 500	< 500	< 500	< 500	< 500	
16EW02	3/16/96	12:00	Primary			< 500	< 500	< 500	< 500	< 500	
16EW02	3/19/96	12:00	Primary			< 500	< 500	< 500	< 500	< 500	
16EW02	10/29/97	10:30	Primary	< 400	< 400	< 400	< 400	< 400	< 400	< 400	< 400
16EW02	1/22/98	12:00	Primary	< 250	< 250	< 250	< 250	< 250	< 250	< 250	< 250
16EW02	6/11/98	15:15	Primary	< 200	< 200	< 200	< 200	< 200	< 200	< 200	< 200
16EW03	10/29/97	11:19	Primary	< 40	< 40	< 40	< 40	< 40	< 40	< 40	< 40
16EW03	1/22/98	11:55	Primary	< 40	< 40	< 40	< 40	< 40	< 40	< 40	< 40
16EW03	6/11/98	15:20	Primary	< 40	< 40	< 40	< 40	< 40	< 40	< 40	< 40
16EW04	10/29/97	10:50	Primary	< 25	< 25	< 25	< 25	< 25	< 25	< 25	< 25
16EW04	1/22/98	11:50	Primary	< 20	< 20	< 20	< 20	< 20	< 20	< 20	< 20
16EW04	6/11/98	15:25	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10
16EW05	10/29/97	11:15	Primary	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000	< 1000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Benzene, 1-methylethyl- (ug/l)	Bromobenzene (ug/l)	Bromodichloro methane (ug/l)	Bromoform (ug/l)	Carbon disulfide (ug/l)	Carbon tetrachloride (ug/l)	Chlorobenzene (ug/l)	Chlorobromo methane (ug/l)
16EW05	1/22/98	11:35	Primary	< 500	< 500	< 500	< 500	< 500	< 500	< 500	< 500
16EW05	6/11/98	15:30	Primary	< 500	< 500	< 500	< 500	< 500	< 500	< 500	< 500
16EW06	10/29/97	12:20	Primary	< 400	< 400	< 400	< 400	< 400	< 400	< 400	< 400
16EW06	1/22/98	11:25	Primary	< 20	< 20	< 20	< 20	< 20	< 20	< 20	< 20
16EW06	6/11/98	15:35	Primary	< 20	< 20	< 20	< 20	< 20	< 20	< 20	< 20
16EW07	10/29/97	11:54	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16EW07	1/22/98	11:45	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16EW07	6/11/98	15:40	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16EW08	10/29/97	11:30	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16EW08	1/22/98	10:10	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16EW08	1/22/98	10:10	Duplicate 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16EW08	6/11/98	15:45	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16PB02	3/18/95	0:00	Primary			< 1		< 1	< 1	< 1	
16PB02	3/18/95	0:00	Duplicate 1			< 1		< 1	< 1	< 1	
16PB04	3/18/95	0:00	Primary			< 100		< 100	< 100	< 100	
16PB05	3/18/95	0:00	Primary			< 1		< 1	< 1	< 1	
16PB06	3/18/95	0:00	Primary			< 1		< 1	< 1	< 1	
16PB07	3/18/95	0:00	Primary			< 1		< 1	< 1	< 1	
16PB08	3/21/95	0:00	Primary			< 10000		< 10000	< 10000	< 10000	
16PB09	3/18/95	0:00	Primary			< 1		< 1	< 1	< 1	
16PB10	3/20/95	0:00	Primary			< 1		< 1	< 1	< 1	
16PB11	3/20/95	0:00	Primary			< 1		< 1	< 1	< 1	
16PB13	3/21/95	0:00	Primary			< 1		< 1	< 1	< 1	
16PB14	3/21/95	0:00	Primary			< 1		< 1	< 1	< 1	
16PB16	3/20/95	0:00	Primary			< 1		< 1	< 1	< 1	
16PB17	3/20/95	0:00	Primary			< 1		< 1	< 1	< 1	
16PB19	3/21/95	0:00	Primary			< 1		< 1	< 1	< 1	
16PB20	3/21/95	0:00	Primary			< 100		< 100	< 100	< 100	
16PB21	3/20/95	0:00	Primary			< 1		< 1	< 1	< 1	
16PB25	3/28/95	0:00	Primary			< 1		< 1	< 1	< 1	
16PB26	3/28/95	0:00	Primary			< 100		< 100	< 100	< 100	
16PB27	3/28/95	0:00	Primary			< 1		< 1	< 1	< 1	
16PB28	3/28/95	0:00	Primary			< 1		< 1	< 1	< 1	
16WW01	6/10/93	0:00	Primary			< 25		< 25	< 25	< 25	
16WW01	6/14/95	14:18	Primary			< 5	< 5	< 5	< 5	< 5	
16WW01	6/25/97	0:00	Primary	< 1	< 1	< 1	< 1			< 1	< 1
16WW02	6/10/93	0:00	Primary			< 5		< 5	< 5	< 5	
16WW02	6/13/95	16:30	Primary			< 5	< 5	< 5	< 5	< 5	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Benzene, 1-methylethyl- (ug/l)	Bromobenzene (ug/l)	Bromodichloro methane (ug/l)	Bromoform (ug/l)	Carbon disulfide (ug/l)	Carbon tetrachloride (ug/l)	Chlorobenzene (ug/l)	Chlorobromo methane (ug/l)
16WW03	6/8/93	0:00	Primary			< 5		< 5	< 5	< 5	
16WW03	6/14/95	16:04	Primary			< 5	< 5	< 5	< 5	< 5	
16WW04	6/8/93	0:00	Primary			< 5		< 5	< 5	< 5	
16WW04	6/15/95	10:58	Primary			< 5	< 5	< 5	< 5	< 5	
16WW04	6/15/95	10:58	Duplicate 1			< 5	< 5	< 5	< 5	< 5	
16WW05	6/11/93	0:00	Primary			< 5		< 5	< 5	< 5	
16WW05	6/11/93	0:00	Duplicate 1			< 5		< 5	< 5	< 5	
16WW05	6/14/95	16:48	Primary			< 5	< 5	< 5	< 5	< 5	
16WW05	10/22/97	17:10	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW06	6/11/93	0:00	Primary			< 5		< 5	< 5	< 5	
16WW06	6/14/95	16:42	Primary			< 5	< 5	< 5	< 5	< 5	
16WW06	10/22/97	14:50	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW07	6/11/93	0:00	Primary			< 5		< 5	< 5	< 5	
16WW07	6/14/95	12:00	Primary			< 5	< 5	< 5	< 5	< 5	
16WW08	6/11/93	0:00	Primary			< 5		< 5	< 5	< 5	
16WW08	6/14/95	11:40	Primary			< 5	< 5	< 5	< 5	< 5	
16WW08	6/14/95	11:40	Duplicate 1			< 5	< 5	< 5	< 5	< 5	
16WW09	6/10/93	0:00	Primary			< 5		< 5	< 5	< 5	
16WW09	6/15/95	12:15	Primary			< 5	< 5	< 5	< 5	< 5	
16WW10	6/9/93	0:00	Primary			< 5		< 5	< 5	< 5	
16WW10	6/15/95	15:40	Primary			< 5	< 5	< 5	< 5	< 5	
16WW11	6/9/93	0:00	Primary			< 5		< 5	< 5	< 5	
16WW11	6/9/93	0:00	Duplicate 1			< 5		< 5	< 5	< 5	
16WW11	6/16/95	11:42	Primary			< 5	< 5	< 5	< 5	< 5	
16WW12	6/13/95	17:45	Primary			< 5	< 5	< 5	< 5	< 5	
16WW12	10/27/97	13:39	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW12	10/27/97	13:39	Duplicate 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW12	1/20/98	16:15	Primary	< 80	< 80	< 80	< 80	< 80	< 80	< 80	< 80
16WW12	6/11/98	12:05	Primary	< 80	< 80	< 80	< 80	< 80	< 80	< 80	< 80
16WW13	6/13/95	16:55	Primary			< 5	< 5	< 5	< 5	< 5	
16WW13	10/27/97	16:05	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW14	6/13/95	16:45	Primary			< 5	< 5	< 5	< 5	< 5	
16WW14	10/24/97	13:55	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW14	1/20/98	13:30	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW14	1/20/98	13:30	Duplicate 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW14	6/15/98	9:20	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW15	6/22/95	10:10	Primary			< 5	< 5	< 5	< 5	< 5	
16WW15	10/28/97	15:25	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Benzene, 1-methylethyl- (ug/l)	Bromobenzene (ug/l)	Bromodichloro methane (ug/l)	Bromoform (ug/l)	Carbon disulfide (ug/l)	Carbon tetrachloride (ug/l)	Chlorobenzene (ug/l)	Chlorobromo methane (ug/l)
16WW16	6/15/95	10:40	Primary			< 5	< 5	< 5	< 5	< 5	
16WW16	10/27/97	16:00	Primary	< 10 •	< 10	< 10	< 10	< 10	< 10	< 10	< 10
16WW16	1/21/98	12:15	Primary	< 4000	< 4000	< 4000	< 4000	< 4000	< 4000	< 4000	< 4000
16WW16	6/15/98	11:15	Primary	< 4000	< 4000	< 4000	< 4000	< 4000	< 4000	< 4000	< 4000
16WW17	6/16/95	15:07	Primary			< 5	< 5	< 5	< 5	< 5	
16WW17	10/27/97	16:44	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW18	6/15/95	14:08	Primary			< 5	< 5	< 5	< 5	< 5	
16WW18	10/28/97	15:09	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW19	10/24/97	11:15	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW20	10/24/97	16:15	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW21	10/28/97	14:45	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW22	10/24/97	13:44	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW22	1/20/98	14:50	Primary	< 50	< 50	< 50	< 50	< 50	< 50	< 50	< 50
16WW22	6/11/98	10:15	Primary	< 50	< 50	< 50	< 50	< 50	< 50	< 50	< 50
16WW23	10/23/97	10:39	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW24	10/23/97	12:49	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW25	10/23/97	10:39	Primary	< 1	< 1	8.4	< 1	< 1	< 1	< 1	< 1
16WW25	1/21/98	17:35	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW25	6/12/98	11:10	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW26	10/23/97	12:49	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW26	1/21/98	17:00	Primary	< 8	< 8	< 8	< 8	< 8	< 8	< 8	< 8
16WW26	6/12/98	9:30	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW27	10/21/97	10:50	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW28	10/21/97	12:55	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW29	10/21/97	10:55	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW29	1/22/98	13:15	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW29	6/11/98	14:30	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW29	6/11/98	14:30	Duplicate 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW30	10/21/97	12:40	Primary	< 1	< 1	2.9	< 1	< 1	< 1	< 1	< 1
16WW30	1/22/98	13:30	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW30	6/15/98	14:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW31	10/22/97	10:09	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW31	10/22/97	10:09	Duplicate 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW32	10/22/97	12:40	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW33	10/22/97	10:00	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW34	10/22/97	12:20	Primary	< 1	< 1	1.1	< 1	< 1	< 1	< 1	< 1
16WW35	10/24/97	11:10	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW35	1/21/98	11:30	Primary	< 8	< 8	< 8	< 8	< 8	< 8	< 8	< 8

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Benzene, 1-methylethyl- (ug/l)	Bromobenzene (ug/l)	Bromodichloro methane (ug/l)	Bromoform (ug/l)	Carbon disulfide (ug/l)	Carbon tetrachloride (ug/l)	Chlorobenzene (ug/l)	Chlorobromo methane (ug/l)
16WW35	6/12/98	13:35	Primary	< 5	< 5	< 5	< 5	< 5	< 5	< 5	< 5
16WW36	10/27/97	13:50	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10
16WW36	1/20/98	16:15	Primary	< 200	< 200	< 200	< 200	< 200	< 200	< 200	< 200
16WW36	6/12/98	15:30	Primary	< 200	< 200	< 200	< 200	< 200	< 200	< 200	< 200
16WW37	10/21/97	16:39	Primary	< 50	< 50	< 50	< 50	< 50	< 50	< 50	< 50
16WW37	10/21/97	16:39	Duplicate 1	< 50	< 50	< 50	< 50	< 50	< 50	< 50	< 50
16WW37	1/21/98	13:30	Primary	< 25	< 25	< 25	< 25	< 25	< 25	< 25	< 25
16WW37	6/14/98	11:00	Primary	< 25	< 25	< 25	< 25	< 25	< 25	< 25	< 25
16WW38	10/22/97	16:09	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
16WW38	1/21/98	13:00	Primary	< 10	< 10	< 10	< 10	< 10	< 10	< 10	< 10
16WW38	6/14/98	12:45	Primary	< 1	< 1	< 1	< 1	< 1	< 1	< 1	< 1
MAXIMUM				4000	4000	10000	4000	10000	10000	10000	4000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Chloroethane (ug/l)	Chloroform (ug/l)	cis-1,2-Dichloro ethylene (ug/l)	cis-1,3-Dichloropropene (ug/l)	DBCP (ug/l)	Dibromochloro methane (ug/l)	Dichloro difluoromethane (ug/l)	EDB (ug/l)
122	6/2/93	0:00	Primary	< 10	< 5	< 5			< 5	< 5	
122	6/14/95	13:42	Primary	< 10	< 5		< 5		< 5	< 5	
16EW01	2/21/96	10:55	Primary	< 10	13		< 5		< 5		
16EW01	2/21/96	16:48	Primary	< 2500	< 1250		< 1250		< 1250		
16EW01	2/21/96	20:35	Primary	< 2500	< 1250		< 1250		< 1250		
16EW01	2/24/96	12:35	Primary	< 2500	< 1250		< 1250		< 1250		
16EW01	2/25/96	23:55	Primary	< 2500	< 1250		< 1250		< 1250		
16EW01	2/25/96	23:55	Duplicate 1	< 2500	< 1250		< 1250		< 1250		
16EW01	2/27/96	12:10	Primary	< 2500	< 1250		< 1250		< 1250		
16EW01	3/16/96	11:30	Primary	< 2500	< 1250		< 1250		< 1250		
16EW01	3/17/96	16:10	Primary	< 2500	< 1250		< 1250		< 1250		
16EW01	3/19/96	11:30	Primary	< 2500	< 1250		< 1250		< 1250		
16EW01	10/29/97	10:00	Primary	< 2000	< 1000	6600	< 5000	< 4000	< 5000	< 1000	< 1000
16EW01	10/29/97	10:00	Duplicate 1	< 2000	< 1000	7400	< 5000	< 4000	< 5000	< 1000	< 1000
16EW01	1/22/98	12:15	Primary	< 80	< 40	590	< 200	< 160	< 200	< 40	< 40
16EW01	6/11/98	15:10	Primary	< 80	< 40	8100	< 200	< 160	< 200	< 40	< 40
16EW01	6/11/98	15:10	Duplicate 1	< 80	< 40	9500	< 200	< 160	< 200	< 40	< 40
16EW02	2/23/96	9:40	Primary	< 1000	< 500		< 500		< 500		
16EW02	2/23/96	14:00	Primary	< 1000	< 500		< 500		< 500		
16EW02	2/28/96	12:20	Primary	< 1000	< 500		< 500		< 500		
16EW02	2/29/96	11:10	Primary	< 1000	< 500		< 500		< 500		
16EW02	2/29/96	21:25	Primary	< 1000	< 500		< 500		< 500		
16EW02	3/13/96	11:35	Primary	< 1000	< 500		< 500		< 500		
16EW02	3/14/96	12:00	Primary	< 1000	< 500		< 500		< 500		
16EW02	3/14/96	12:00	Duplicate 1	< 1000	< 500		< 500		< 500		
16EW02	3/16/96	12:00	Primary	< 1000	< 500		< 500		< 500		
16EW02	3/19/96	12:00	Primary	< 1000	< 500		< 500		< 500		
16EW02	10/29/97	10:30	Primary	< 800	< 400	< 400	< 2000	< 1600	< 2000	< 400	< 400
16EW02	1/22/98	12:00	Primary	< 500	< 250	(130)	< 1200	< 1000	< 1200	< 250	< 250
16EW02	6/11/98	15:15	Primary	< 400	< 200	< 200	< 1000	< 800	< 1000	< 200	< 200
16EW03	10/29/97	11:19	Primary	< 80	< 40	< 40	< 200	< 160	< 200	< 40	< 40
16EW03	1/22/98	11:55	Primary	< 80	< 40	(23)	< 200	< 160	< 200	< 40	< 40
16EW03	6/11/98	15:20	Primary	< 80	< 40	42	< 200	< 160	< 200	< 40	< 40
16EW04	10/29/97	10:50	Primary	< 50	< 25	< 25	< 120	< 100	< 120	< 25	< 25
16EW04	1/22/98	11:50	Primary	< 40	< 20	< 20	< 100	< 80	< 100	< 20	< 20
16EW04	6/11/98	15:25	Primary	< 20	< 10	13	< 50	< 40	< 50	< 10	< 10
16EW05	10/29/97	11:15	Primary	< 2000	< 1000	2200	< 5000	< 4000	< 5000	< 1000	< 1000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Chloroethane (ug/l)	Chloroform (ug/l)	cis-1,2-Dichloro ethylene (ug/l)	cis-1,3-Dichloropropene (ug/l)	DBCP (ug/l)	Dibromochloro methane (ug/l)	Dichloro difluoromethane (ug/l)	EDB (ug/l)
16EW05	1/22/98	11:35	Primary	< 1000	< 500	2700	< 2500	< 2000	< 2500	< 500	< 500
16EW05	6/11/98	15:30	Primary	< 1000	< 500	2000	< 2500	< 2000	< 2500	< 500	< 500
16EW06	10/29/97	12:20	Primary	< 800	< 400	1500	< 2000	< 1600	< 2000	< 400	< 400
16EW06	1/22/98	11:25	Primary	< 40	< 20	(15)	< 100	< 80	< 100	< 20	< 20
16EW06	6/11/98	15:35	Primary	< 40	< 20	1400	< 100	< 80	< 100	< 20	< 20
16EW07	10/29/97	11:54	Primary	< 2	< 1	< 1	< 5	< 4	< 5	< 1	< 1
16EW07	1/22/98	11:45	Primary	< 2	< 1	< 1	< 5	< 4	< 5	< 1	< 1
16EW07	6/11/98	15:40	Primary	< 2	< 1	< 1	< 5	< 4	< 5	< 1	< 1
16EW08	10/29/97	11:30	Primary	< 2	< 1	2.5	< 5	< 4	< 5	< 1	< 1
16EW08	1/22/98	10:10	Primary	< 2	< 1	< 1	< 5	< 4	< 5	< 1	< 1
16EW08	1/22/98	10:10	Duplicate 1	< 2	< 1	< 1	< 5	< 4	< 5	< 1	< 1
16EW08	6/11/98	15:45	Primary	< 2	< 1	< 1	< 5	< 4	< 5	< 1	< 1
16PB02	3/18/95	0:00	Primary	< 1	< 1	< 1	< 1		< 1	< 1	
16PB02	3/18/95	0:00	Duplicate 1	< 1	< 1	< 1	< 1		< 1	< 1	
16PB04	3/18/95	0:00	Primary	< 100	< 100	1100	< 100		< 100	< 100	
16PB05	3/18/95	0:00	Primary	< 1	< 1	< 1	< 1		< 1	< 1	
16PB06	3/18/95	0:00	Primary	< 1	< 1	< 1	< 1		< 1	< 1	
16PB07	3/18/95	0:00	Primary	< 1	< 1	< 1	< 1		< 1	< 1	
16PB08	3/21/95	0:00	Primary	< 10000	< 10000	520000	< 10000		< 10000	< 10000	
16PB09	3/18/95	0:00	Primary	< 1	4.8	1000	< 1		< 1	< 1	
16PB10	3/20/95	0:00	Primary	< 1	< 1	2.6	< 1		< 1	< 1	
16PB11	3/20/95	0:00	Primary	< 1	< 1	< 1	< 1		< 1	< 1	
16PB13	3/21/95	0:00	Primary	< 1	< 1	3.7	< 1		< 1	< 1	
16PB14	3/21/95	0:00	Primary	< 1	11	300	< 1		< 1	< 1	
16PB16	3/20/95	0:00	Primary	< 1	< 1	< 1	< 1		< 1	< 1	
16PB17	3/20/95	0:00	Primary	< 1	< 1	< 1	< 1		< 1	< 1	
16PB19	3/21/95	0:00	Primary	< 1	< 1	< 1	< 1		< 1	< 1	
16PB20	3/21/95	0:00	Primary	< 100	< 100	110	< 100		< 100	< 100	
16PB21	3/20/95	0:00	Primary	< 1	< 1	26	< 1		< 1	< 1	
16PB25	3/28/95	0:00	Primary	< 1	< 1	< 1	< 1		< 1	< 1	
16PB26	3/28/95	0:00	Primary	< 100	< 100	< 100	< 100		< 100	< 100	
16PB27	3/28/95	0:00	Primary	< 1	< 1	< 1	< 1		< 1	< 1	
16PB28	3/28/95	0:00	Primary	< 1	< 1	< 1	< 1		< 1	< 1	
16WW01	6/10/93	0:00	Primary	< 50	< 25	35			< 25	< 25	
16WW01	6/14/95	14:18	Primary	< 10	< 5		< 5		< 5	< 5	
16WW01	6/25/97	0:00	Primary	< 2	1	< 1	< 5	< 4	< 1	< 1	< 1
16WW02	6/10/93	0:00	Primary	< 10	< 5	< 5			< 5	< 5	
16WW02	6/13/95	16:30	Primary	< 10	< 5		< 5		< 5	< 5	

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Chloroethane (ug/l)	Chloroform (ug/l)	cis-1,2-Dichloro ethylene (ug/l)	cis-1,3-Dichloropropene (ug/l)	DBCP (ug/l)	Dibromochloro methane (ug/l)	Dichloro difluoromethane (ug/l)	EDB (ug/l)
16WW03	6/8/93	0:00	Primary	< 10	< 5	52			< 5	< 5	
16WW03	6/14/95	16:04	Primary	< 10	< 5		< 5		< 5	< 5	
16WW04	6/8/93	0:00	Primary	< 10	< 5	21			< 5	< 5	
16WW04	6/15/95	10:58	Primary	< 10	< 5		< 5		< 5	< 5	
16WW04	6/15/95	10:58	Duplicate 1	< 10	< 5		< 5		< 5	< 5	
16WW05	6/11/93	0:00	Primary	< 10	< 5	< 5			< 5	< 5	
16WW05	6/11/93	0:00	Duplicate 1	< 10	< 5	< 5			< 5	< 5	
16WW05	6/14/95	16:48	Primary	< 10	< 5		< 5		< 5	< 5	
16WW05	10/22/97	17:10	Primary	< 2	(0.64)	< 1	< 5	< 4	< 5	< 1	< 1
16WW06	6/11/93	0:00	Primary	< 10	< 5	< 5			< 5	< 5	
16WW06	6/14/95	16:42	Primary	< 10	< 5		< 5		< 5	< 5	
16WW06	10/22/97	14:50	Primary	< 2	(0.96)	< 1	< 5	< 4	< 5	< 1	< 1
16WW07	6/11/93	0:00	Primary	< 10	< 5	< 5			< 5	< 5	
16WW07	6/14/95	12:00	Primary	< 10	< 5		< 5		< 5	< 5	
16WW08	6/11/93	0:00	Primary	< 10	< 5	< 5			< 5	< 5	
16WW08	6/14/95	11:40	Primary	< 10	< 5		< 5		< 5	< 5	
16WW08	6/14/95	11:40	Duplicate 1	< 10	< 5		< 5		< 5	< 5	
16WW09	6/10/93	0:00	Primary	< 10	< 5	< 5			< 5	< 5	
16WW09	6/15/95	12:15	Primary	< 10	< 5		< 5		< 5	< 5	
16WW10	6/9/93	0:00	Primary	< 10	< 5	< 5			< 5	< 5	
16WW10	6/15/95	15:40	Primary	< 10	< 5		< 5		< 5	< 5	
16WW11	6/9/93	0:00	Primary	< 10	< 5	< 5			< 5	< 5	
16WW11	6/9/93	0:00	Duplicate 1	< 10	< 5	< 5			< 5	< 5	
16WW11	6/16/95	11:42	Primary	< 10	< 5		< 5		< 5	< 5	
16WW12	6/13/95	17:45	Primary	< 10	< 5		< 5		< 5	< 5	
16WW12	10/27/97	13:39	Primary	< 2	< 1	85	< 5	< 4	< 5	< 1	< 1
16WW12	10/27/97	13:39	Duplicate 1	< 2	< 1	94	< 5	< 4	< 5	< 1	< 1
16WW12	1/20/98	16:15	Primary	< 160	< 80	(68)	< 400	< 320	< 400	< 80	< 80
16WW12	6/11/98	12:05	Primary	< 160	< 80	100	< 400	< 320	< 400	< 80	< 80
16WW13	6/13/95	16:55	Primary	< 10	< 5		< 5		< 5	< 5	
16WW13	10/27/97	16:05	Primary	< 2	1.9	(160)	< 5	< 4	< 5	< 1	< 1
16WW14	6/13/95	16:45	Primary	< 10	< 5		< 5		< 5	< 5	
16WW14	10/24/97	13:55	Primary	< 2	< 1	(0.96)	< 5	< 4	< 5	< 1	< 1
16WW14	1/20/98	13:30	Primary	< 2	< 1	9.3	< 5	< 4	< 5	< 1	< 1
16WW14	1/20/98	13:30	Duplicate 1	< 2	< 1	10	< 5	< 4	< 5	< 1	< 1
16WW14	6/15/98	9:20	Primary	< 2	< 1	< 1	< 5	< 4	< 5	< 1	< 1
16WW15	6/22/95	10:10	Primary	< 10	< 5		< 5		< 5	< 5	
16WW15	10/28/97	15:25	Primary	< 2	< 1	< 1	< 5	< 4	< 5	< 1	< 1

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Chloroethane (ug/l)	Chloroform (ug/l)	cis-1,2-Dichloro ethylene (ug/l)	cis-1,3-Dichloropropene (ug/l)	DBCP (ug/l)	Dibromochloro methane (ug/l)	Dichloro difluoromethane (ug/l)	EDB (ug/l)
16WW16	6/15/95	10:40	Primary	< 10	(4) J		< 5		< 5	< 5	
16WW16	10/27/97	16:00	Primary	< 20	< 10	260000	< 50	< 40	< 50	< 10	< 10
16WW16	1/21/98	12:15	Primary	< 8000	< 4000	270000	< 20000	< 16000	< 20000	< 4000	< 4000
16WW16	6/15/98	11:15	Primary	< 8000	< 4000	260000	< 20000	< 16000	< 20000	< 4000	< 4000
16WW17	6/16/95	15:07	Primary	< 10	< 5		< 5		< 5	< 5	
16WW17	10/27/97	16:44	Primary	< 2	< 1	(0.60)	< 5	< 4	< 5	< 1	< 1
16WW18	6/15/95	14:08	Primary	< 10	< 5		< 5		< 5	< 5	
16WW18	10/28/97	15:09	Primary	< 2	< 1	< 1	< 5	< 4	< 5	< 1	< 1
16WW19	10/24/97	11:15	Primary	< 2	(0.77)	< 1	< 5	< 4	< 5	< 1	< 1
16WW20	10/24/97	16:15	Primary	< 2	(0.61)	< 1	< 5	< 4	< 5	< 1	< 1
16WW21	10/28/97	14:45	Primary	< 2	(0.79)	360	< 5	< 4	< 5	< 1	< 1
16WW22	10/24/97	13:44	Primary	< 2	(0.52)	66	< 5	< 4	< 5	< 1	< 1
16WW22	1/20/98	14:50	Primary	< 100	< 50	120	< 250	< 200	< 250	< 50	< 50
16WW22	6/11/98	10:15	Primary	< 100	< 50	62	< 250	< 200	< 250	< 50	< 50
16WW23	10/23/97	10:39	Primary	< 2	< 1	< 1	< 5	< 4	< 5	< 1	< 1
16WW24	10/23/97	12:49	Primary	< 2	(0.59)	< 1	< 5	< 4	< 5	< 1	< 1
16WW25	10/23/97	10:39	Primary	< 2	36	< 1	< 5	< 4	< 5	< 1	< 1
16WW25	1/21/98	17:35	Primary	< 2	< 1	7.9	< 5	< 4	< 5	< 1	< 1
16WW25	6/12/98	11:10	Primary	< 2	< 1	5	< 5	< 4	< 5	< 1	< 1
16WW26	10/23/97	12:49	Primary	< 2	1.3	2	< 5	< 4	< 5	< 1	< 1
16WW26	1/21/98	17:00	Primary	< 16	< 8	570	< 40	< 32	< 40	< 8	< 8
16WW26	6/12/98	9:30	Primary	< 2	< 1	< 1	< 5	< 4	< 5	< 1	< 1
16WW27	10/21/97	10:50	Primary	< 2	< 1	< 1	< 5	< 4	< 5	< 1	< 1
16WW28	10/21/97	12:55	Primary	< 2	< 1	< 1	< 5	< 4	< 5	< 1	< 1
16WW29	10/21/97	10:55	Primary	< 2	< 1	(0.57)	< 5	< 4	< 5	< 1	< 1
16WW29	1/22/98	13:15	Primary	< 2	< 1	11	< 5	< 4	< 5	< 1	< 1
16WW29	6/11/98	14:30	Primary	< 2	< 1	3.2	< 5	< 4	< 5	< 1	< 1
16WW29	6/11/98	14:30	Duplicate 1	< 2	< 1	3.1	< 5	< 4	< 5	< 1	< 1
16WW30	10/21/97	12:40	Primary	< 2	14	(0.85)	< 5	< 4	< 5	< 1	< 1
16WW30	1/22/98	13:30	Primary	< 2	< 1	11	< 5	< 4	< 5	< 1	< 1
16WW30	6/15/98	14:00	Primary	< 2	< 1	33	< 5	< 4	< 5	< 1	< 1
16WW31	10/22/97	10:09	Primary	< 2	(0.58)	< 1	< 5	< 4	< 5	< 1	< 1
16WW31	10/22/97	10:09	Duplicate 1	< 2	< 1	< 1	< 5	< 4	< 5	< 1	< 1
16WW32	10/22/97	12:40	Primary	< 2	(0.53)	< 1	< 5	< 4	< 5	< 1	< 1
16WW33	10/22/97	10:00	Primary	< 2	(0.69)	< 1	< 5	< 4	< 5	< 1	< 1
16WW34	10/22/97	12:20	Primary	< 2	7.7	< 1	< 5	< 4	< 5	< 1	< 1
16WW35	10/24/97	11:10	Primary	< 2	1.4	52	< 5	< 4	< 5	< 1	< 1
16WW35	1/21/98	11:30	Primary	< 16	< 8	30	< 40	< 32	< 40	< 8	< 8

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Chloroethane (ug/l)	Chloroform (ug/l)	cis-1,2-Dichloro ethylene (ug/l)	cis-1,3-Dichloropropene (ug/l)	DBCP (ug/l)	Dibromochloro methane (ug/l)	Dichloro difluoromethane (ug/l)	EDB (ug/l)
16WW35	6/12/98	13:35	Primary	< 10	< 5	15	< 25	< 20	< 25	< 5	< 5
16WW36	10/27/97	13:50	Primary	< 20	< 10	76	< 50	< 40	< 50	< 10	< 10
16WW36	1/20/98	16:15	Primary	< 400	(120)	< 200	< 1000	< 800	< 1000	< 200	< 200
16WW36	6/12/98	15:30	Primary	< 400	< 200	< 200	< 1000	< 800	< 1000	< 200	< 200
16WW37	10/21/97	16:39	Primary	< 100	< 50	< 50	< 250	< 200	< 250	< 50	< 50
16WW37	10/21/97	16:39	Duplicate 1	< 100	< 50	< 50	< 250	< 200	< 250	< 50	< 50
16WW37	1/21/98	13:30	Primary	< 50	< 25	220	< 120	< 100	< 120	< 25	< 25
16WW37	6/14/98	11:00	Primary	< 50	< 25	33	< 120	< 100	< 120	< 25	< 25
16WW38	10/22/97	16:09	Primary	< 2	< 1	(0.52)	< 5	< 4	< 5	< 1	< 1
16WW38	1/21/98	13:00	Primary	< 20	< 10	520	< 50	< 40	< 50	< 10	< 10
16WW38	6/14/98	12:45	Primary	< 2	< 1	2.6	< 5	< 4	< 5	< 1	< 1
MAXIMUM				10000	10000	520000	20000	16000	20000	10000	4000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Ethene, (2-chloroethoxy)- (ug/l)	Ethene, 1,2-dichloro-, (E)- (ug/l)	Ethyl cyanide (ug/l)	Ethyl methacrylate (ug/l)	Ethylbenzene (ug/l)	Hexachloro butadiene (ug/l)	Isobutyl alcohol (ug/l)	m-Dichloro benzene (ug/l)
122	6/2/93	0:00	Primary		< 5		< 5	< 5			
122	6/14/95	13:42	Primary	< 10			< 5	< 5			
16EW01	2/21/96	10:55	Primary	< 10				< 5			
16EW01	2/21/96	16:48	Primary	< 2500				< 1250			
16EW01	2/21/96	20:35	Primary	< 2500				< 1250			
16EW01	2/24/96	12:35	Primary	< 2500				< 1250			
16EW01	2/25/96	23:55	Primary	< 2500				< 1250			
16EW01	2/25/96	23:55	Duplicate 1	< 2500				< 1250			
16EW01	2/27/96	12:10	Primary	< 2500				< 1250			
16EW01	3/16/96	11:30	Primary	< 2500				< 1250			
16EW01	3/17/96	16:10	Primary	< 2500				< 1250			
16EW01	3/19/96	11:30	Primary	< 2500				< 1250			
16EW01	10/29/97	10:00	Primary		< 1000	< 20000	< 5000	< 1000	< 1000	< 200000	< 1000
16EW01	10/29/97	10:00	Duplicate 1		< 1000	< 20000	< 5000	< 1000	< 1000	< 200000	< 1000
16EW01	1/22/98	12:15	Primary		< 40	< 800	< 200	< 40	< 40	< 8000	< 40
16EW01	6/11/98	15:10	Primary		< 40	< 800	< 200	< 40	< 40	< 8000	< 40
16EW01	6/11/98	15:10	Duplicate 1		< 40	< 800	< 200	< 40	< 40	< 8000	< 40
16EW02	2/23/96	9:40	Primary	< 1000				< 500			
16EW02	2/23/96	14:00	Primary	< 1000				< 500			
16EW02	2/28/96	12:20	Primary	< 1000				< 500			
16EW02	2/29/96	11:10	Primary	< 1000				< 500			
16EW02	2/29/96	21:25	Primary	< 1000				< 500			
16EW02	3/13/96	11:35	Primary	< 1000				< 500			
16EW02	3/14/96	12:00	Primary	< 1000				< 500			
16EW02	3/14/96	12:00	Duplicate 1	< 1000				< 500			
16EW02	3/16/96	12:00	Primary	< 1000				< 500			
16EW02	3/19/96	12:00	Primary	< 1000				< 500			
16EW02	10/29/97	10:30	Primary		< 400	< 8000	< 2000	< 400	< 400	< 80000	< 400
16EW02	1/22/98	12:00	Primary		< 250	< 5000	< 1200	< 250	< 250	< 50000	< 250
16EW02	6/11/98	15:15	Primary		< 200	< 4000	< 1000	< 200	< 200	< 40000	< 200
16EW03	10/29/97	11:19	Primary		< 40	< 800	< 200	< 40	< 40	< 8000	< 40
16EW03	1/22/98	11:55	Primary		< 40	< 800	< 200	< 40	< 40	< 8000	< 40
16EW03	6/11/98	15:20	Primary		< 40	< 800	< 200	< 40	< 40	< 8000	< 40
16EW04	10/29/97	10:50	Primary		< 25	< 500	< 120	< 25	< 25	< 5000	< 25
16EW04	1/22/98	11:50	Primary		< 20	< 400	< 100	< 20	< 20	< 4000	< 20
16EW04	6/11/98	15:25	Primary		< 10	< 200	< 50	< 10	< 10	< 2000	< 10
16EW05	10/29/97	11:15	Primary		< 1000	< 20000	< 5000	< 1000	< 1000	< 200000	< 1000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Ethene, (2-chloroethoxy)- (ug/l)	Ethene, 1,2-dichloro-, (E)- (ug/l)	Ethyl cyanide (ug/l)	Ethyl methacrylate (ug/l)	Ethylbenzene (ug/l)	Hexachloro butadiene (ug/l)	Isobutyl alcohol (ug/l)	m-Dichloro benzene (ug/l)
16EW05	1/22/98	11:35	Primary		< 500	< 10000	< 2500	< 500	< 500	< 100000	< 500
16EW05	6/11/98	15:30	Primary		< 500	< 10000	< 2500	< 500	< 500	< 100000	< 500
16EW06	10/29/97	12:20	Primary		< 400	< 8000	< 2000	< 400	< 400	< 80000	< 400
16EW06	1/22/98	11:25	Primary		< 20	< 400	< 100	< 20	< 20	< 4000	< 20
16EW06	6/11/98	15:35	Primary		< 20	< 400	< 100	< 20	< 20	< 4000	< 20
16EW07	10/29/97	11:54	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16EW07	1/22/98	11:45	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16EW07	6/11/98	15:40	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16EW08	10/29/97	11:30	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16EW08	1/22/98	10:10	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16EW08	1/22/98	10:10	Duplicate 1		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16EW08	6/11/98	15:45	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16PB02	3/18/95	0:00	Primary		< 1		< 1	< 1			
16PB02	3/18/95	0:00	Duplicate 1		< 1		< 1	< 1			
16PB04	3/18/95	0:00	Primary		< 100		< 100	< 100			
16PB05	3/18/95	0:00	Primary		< 1		< 1	< 1			
16PB06	3/18/95	0:00	Primary		< 1		< 1	< 1			
16PB07	3/18/95	0:00	Primary		< 1		< 1	< 1			
16PB08	3/21/95	0:00	Primary		< 10000		< 10000	< 10000			
16PB09	3/18/95	0:00	Primary		< 1		< 1	< 1			
16PB10	3/20/95	0:00	Primary		< 1		< 1	< 1			
16PB11	3/20/95	0:00	Primary		< 1		< 1	< 1			
16PB13	3/21/95	0:00	Primary		< 1		< 1	< 1			
16PB14	3/21/95	0:00	Primary		< 1		< 1	< 1			
16PB16	3/20/95	0:00	Primary		< 1		< 1	< 1			
16PB17	3/20/95	0:00	Primary		< 1		< 1	< 1			
16PB19	3/21/95	0:00	Primary		< 1		< 1	< 1			
16PB20	3/21/95	0:00	Primary		< 100		< 100	< 100			
16PB21	3/20/95	0:00	Primary		< 1		< 1	< 1			
16PB25	3/28/95	0:00	Primary		< 1		< 1	< 1			
16PB26	3/28/95	0:00	Primary		< 100		< 100	< 100			
16PB27	3/28/95	0:00	Primary		< 1		< 1	< 1			
16PB28	3/28/95	0:00	Primary		< 1		< 1	< 1			
16WW01	6/10/93	0:00	Primary		< 25		< 25	< 25			
16WW01	6/14/95	14:18	Primary	< 10			< 5	< 5			
16WW01	6/25/97	0:00	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW02	6/10/93	0:00	Primary		< 5		< 5	< 5			
16WW02	6/13/95	16:30	Primary	< 10			< 5	< 5			

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Ethene, (2-chloroethoxy)- (ug/l)	Ethene, 1,2-dichloro-, (E)- (ug/l)	Ethyl cyanide (ug/l)	Ethyl methacrylate (ug/l)	Ethylbenzene (ug/l)	Hexachloro butadiene (ug/l)	Isobutyl alcohol (ug/l)	m-Dichloro benzene (ug/l)
16WW03	6/8/93	0:00	Primary		< 5		< 5	< 5			
16WW03	6/14/95	16:04	Primary	< 10			< 5	< 5			
16WW04	6/8/93	0:00	Primary		< 5		< 5	< 5			
16WW04	6/15/95	10:58	Primary	< 10			< 5	< 5			
16WW04	6/15/95	10:58	Duplicate 1	< 10			< 5	< 5			
16WW05	6/11/93	0:00	Primary		< 5		< 5	< 5			
16WW05	6/11/93	0:00	Duplicate 1		< 5		< 5	< 5			
16WW05	6/14/95	16:48	Primary	< 10			< 5	< 5			
16WW05	10/22/97	17:10	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW06	6/11/93	0:00	Primary		< 5		< 5	< 5			
16WW06	6/14/95	16:42	Primary	< 10			< 5	< 5			
16WW06	10/22/97	14:50	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW07	6/11/93	0:00	Primary		< 5		< 5	< 5			
16WW07	6/14/95	12:00	Primary	< 10			< 5	< 5			
16WW08	6/11/93	0:00	Primary		< 5		< 5	< 5			
16WW08	6/14/95	11:40	Primary	< 10			< 5	< 5			
16WW08	6/14/95	11:40	Duplicate 1	< 10			< 5	< 5			
16WW09	6/10/93	0:00	Primary		< 5		< 5	< 5			
16WW09	6/15/95	12:15	Primary	< 10			< 5	< 5			
16WW10	6/9/93	0:00	Primary		< 5		< 5	< 5			
16WW10	6/15/95	15:40	Primary	< 10			< 5	< 5			
16WW11	6/9/93	0:00	Primary		< 5		< 5	< 5			
16WW11	6/9/93	0:00	Duplicate 1		< 5		< 5	< 5			
16WW11	6/16/95	11:42	Primary	< 10			< 5	< 5			
16WW12	6/13/95	17:45	Primary	< 10			< 5	< 5			
16WW12	10/27/97	13:39	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW12	10/27/97	13:39	Duplicate 1		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW12	1/20/98	16:15	Primary		< 80	< 1600	< 400	< 80	< 80	< 16000	< 80
16WW12	6/11/98	12:05	Primary		< 80	< 1600	< 400	< 80	< 80	< 16000	< 80
16WW13	6/13/95	16:55	Primary	< 10			< 5	< 5			
16WW13	10/27/97	16:05	Primary		2.1	< 20	< 5	< 1	< 1	< 200	< 1
16WW14	6/13/95	16:45	Primary	< 10			< 5	< 5			
16WW14	10/24/97	13:55	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW14	1/20/98	13:30	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW14	1/20/98	13:30	Duplicate 1		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW14	6/15/98	9:20	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW15	6/22/95	10:10	Primary	< 10			< 5	< 5			
16WW15	10/28/97	15:25	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Ethene, (2-chloroethoxy)- (ug/l)	Ethene, 1,2-dichloro-, (E)- (ug/l)	Ethyl cyanide (ug/l)	Ethyl methacrylate (ug/l)	Ethylbenzene (ug/l)	Hexachloro butadiene (ug/l)	Isobutyl alcohol (ug/l)	m-Dichloro benzene (ug/l)
16WW16	6/15/95	10:40	Primary	< 10			< 5	5			
16WW16	10/27/97	16:00	Primary		130	< 200	< 50	< 10	< 10	< 2000	< 10
16WW16	1/21/98	12:15	Primary		< 4000	< 80000	< 20000	< 4000	< 4000	< 800000	< 4000
16WW16	6/15/98	11:15	Primary		< 4000	< 80000	< 20000	< 4000	< 4000	< 800000	< 4000
16WW17	6/16/95	15:07	Primary	< 10			< 5	< 5			
16WW17	10/27/97	16:44	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW18	6/15/95	14:08	Primary	< 10			< 5	< 5			
16WW18	10/28/97	15:09	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW19	10/24/97	11:15	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW20	10/24/97	16:15	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW21	10/28/97	14:45	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW22	10/24/97	13:44	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW22	1/20/98	14:50	Primary		< 50	< 1000	< 250	< 50	< 50	< 10000	< 50
16WW22	6/11/98	10:15	Primary		< 50	< 1000	< 250	< 50	< 50	< 10000	< 50
16WW23	10/23/97	10:39	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW24	10/23/97	12:49	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW25	10/23/97	10:39	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW25	1/21/98	17:35	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW25	6/12/98	11:10	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW26	10/23/97	12:49	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW26	1/21/98	17:00	Primary		< 8	< 160	< 40	< 8	< 8	< 1600	< 8
16WW26	6/12/98	9:30	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW27	10/21/97	10:50	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW28	10/21/97	12:55	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW29	10/21/97	10:55	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW29	1/22/98	13:15	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW29	6/11/98	14:30	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW29	6/11/98	14:30	Duplicate 1		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW30	10/21/97	12:40	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW30	1/22/98	13:30	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW30	6/15/98	14:00	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW31	10/22/97	10:09	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW31	10/22/97	10:09	Duplicate 1		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW32	10/22/97	12:40	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW33	10/22/97	10:00	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW34	10/22/97	12:20	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW35	10/24/97	11:10	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW35	1/21/98	11:30	Primary		< 8	< 160	< 40	< 8	< 8	< 1600	< 8

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Ethene, (2-chloroethoxy)- (ug/l)	Ethene, 1,2-dichloro-, (E)- (ug/l)	Ethyl cyanide (ug/l)	Ethyl methacrylate (ug/l)	Ethylbenzene (ug/l)	Hexachloro butadiene (ug/l)	Isobutyl alcohol (ug/l)	m-Dichloro benzene (ug/l)
16WW35	6/12/98	13:35	Primary		< 5	< 100	< 25	< 5	< 5	< 1000	< 5
16WW36	10/27/97	13:50	Primary		< 10	< 200	< 50	< 10	< 10	< 2000	< 10
16WW36	1/20/98	16:15	Primary		< 200	< 4000	< 1000	< 200	< 200	< 40000	< 200
16WW36	6/12/98	15:30	Primary		< 200	< 4000	< 1000	< 200	< 200	< 40000	< 200
16WW37	10/21/97	16:39	Primary		< 50	< 1000	< 250	< 50	< 50	< 10000	< 50
16WW37	10/21/97	16:39	Duplicate 1		< 50	< 1000	< 250	< 50	< 50	< 10000	< 50
16WW37	1/21/98	13:30	Primary		< 25	< 500	< 120	< 25	< 25	< 5000	< 25
16WW37	6/14/98	11:00	Primary		< 25	< 500	< 120	< 25	< 25	< 5000	< 25
16WW38	10/22/97	16:09	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
16WW38	1/21/98	13:00	Primary		< 10	< 200	< 50	< 10	< 10	< 2000	< 10
16WW38	6/14/98	12:45	Primary		< 1	< 20	< 5	< 1	< 1	< 200	< 1
MAXIMUM				2500	10000	80000	20000	10000	4000	800000	4000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Methacrylo nitrile (ug/l)	Methyl bromide (ug/l)	Methyl chloride (ug/l)	Methyl ethyl ketone (ug/l)	Methyl iodide (ug/l)	Methyl isobutyl ketone (MIBK) (ug/l)	Methyl methacrylate (ug/l)	Methylene bromide (ug/l)
122	6/2/93	0:00	Primary		< 10	< 10	< 10	< 10	< 10		< 5
122	6/14/95	13:42	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16EW01	2/21/96	10:55	Primary		< 10	< 10	< 10		< 10		
16EW01	2/21/96	16:48	Primary		< 2500	< 2500	< 2500		< 2500		
16EW01	2/21/96	20:35	Primary		< 2500	< 2500	< 2500		< 2500		
16EW01	2/24/96	12:35	Primary		< 2500	< 2500	< 2500		< 2500		
16EW01	2/25/96	23:55	Primary		< 2500	< 2500	< 2500		< 2500		
16EW01	2/25/96	23:55	Duplicate 1		< 2500	< 2500	< 2500		< 2500		
16EW01	2/27/96	12:10	Primary		< 2500	< 2500	< 2500		< 2500		
16EW01	3/16/96	11:30	Primary		< 2500	< 2500	< 2500		< 2500		
16EW01	3/17/96	16:10	Primary		< 2500	< 2500	< 2500		< 2500		
16EW01	3/19/96	11:30	Primary		< 2500	< 2500	< 2500		< 2500		
16EW01	10/29/97	10:00	Primary	< 20000	< 2000	< 2000	< 5000	< 5000	< 5000	< 5000	< 2000
16EW01	10/29/97	10:00	Duplicate 1	< 20000	< 2000	< 2000	< 5000	< 5000	< 5000	< 5000	< 2000
16EW01	1/22/98	12:15	Primary	< 800	< 80	< 80	< 200	< 200	< 200	< 200	< 80
16EW01	6/11/98	15:10	Primary	< 800	< 80	< 80	< 200	< 200	< 200	< 200	< 80
16EW01	6/11/98	15:10	Duplicate 1	< 800	< 80	< 80	< 200	< 200	< 200	< 200	< 80
16EW02	2/23/96	9:40	Primary		< 1000	< 1000	< 1000		< 1000		
16EW02	2/23/96	14:00	Primary		< 1000	< 1000	< 1000		< 1000		
16EW02	2/28/96	12:20	Primary		< 1000	< 1000	< 1000		< 1000		
16EW02	2/29/96	11:10	Primary		< 1000	< 1000	< 1000		< 1000		
16EW02	2/29/96	21:25	Primary		< 1000	< 1000	< 1000		< 1000		
16EW02	3/13/96	11:35	Primary		< 1000	< 1000	< 1000		< 1000		
16EW02	3/14/96	12:00	Primary		< 1000	< 1000	< 1000		< 1000		
16EW02	3/14/96	12:00	Duplicate 1		< 1000	< 1000	< 1000		< 1000		
16EW02	3/16/96	12:00	Primary		< 1000	< 1000	< 1000		< 1000		
16EW02	3/19/96	12:00	Primary		< 1000	< 1000	< 1000		< 1000		
16EW02	10/29/97	10:30	Primary	< 8000	< 800	< 800	< 2000	< 2000	< 2000	< 2000	< 800
16EW02	1/22/98	12:00	Primary	< 5000	< 500	< 500	< 1200	< 1200	< 1200	< 1200	< 500
16EW02	6/11/98	15:15	Primary	< 4000	< 400	< 400	< 1000	< 1000	< 1000	< 1000	< 400
16EW03	10/29/97	11:19	Primary	< 800	< 80	< 80	< 200	< 200	< 200	< 200	< 80
16EW03	1/22/98	11:55	Primary	< 800	< 80	< 80	< 200	< 200	< 200	< 200	< 80
16EW03	6/11/98	15:20	Primary	< 800	< 80	< 80	< 200	< 200	< 200	< 200	< 80
16EW04	10/29/97	10:50	Primary	< 500	< 50	< 50	< 120	< 120	< 120	< 120	< 50
16EW04	1/22/98	11:50	Primary	< 400	< 40	< 40	< 100	< 100	< 100	< 100	< 40
16EW04	6/11/98	15:25	Primary	< 200	< 20	< 20	< 50	< 50	< 50	< 50	< 20
16EW05	10/29/97	11:15	Primary	< 20000	< 2000	< 2000	< 5000	< 5000	< 5000	< 5000	< 2000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Methacrylo nitrile (ug/l)	Methyl bromide (ug/l)	Methyl chloride (ug/l)	Methyl ethyl ketone (ug/l)	Methyl iodide (ug/l)	Methyl isobutyl ketone (MIBK) (ug/l)	Methyl methacrylate (ug/l)	Methylene bromide (ug/l)
16EW05	1/22/98	11:35	Primary	< 10000	< 1000	< 1000	< 2500	< 2500	< 2500	< 2500	< 1000
16EW05	6/11/98	15:30	Primary	< 10000	< 1000	< 1000	< 2500	< 2500	< 2500	< 2500	< 1000
16EW06	10/29/97	12:20	Primary	< 8000	< 800	< 800	< 2000	< 2000	< 2000	< 2000	< 800
16EW06	1/22/98	11:25	Primary	< 400	< 40	< 40	< 100	< 100	< 100	< 100	< 40
16EW06	6/11/98	15:35	Primary	< 400	< 40	< 40	< 100	< 100	< 100	< 100	< 40
16EW07	10/29/97	11:54	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16EW07	1/22/98	11:45	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16EW07	6/11/98	15:40	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16EW08	10/29/97	11:30	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16EW08	1/22/98	10:10	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16EW08	1/22/98	10:10	Duplicate 1	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16EW08	6/11/98	15:45	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16PB02	3/18/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		< 1
16PB02	3/18/95	0:00	Duplicate 1		< 1	< 1	< 1	< 1	< 1		< 1
16PB04	3/18/95	0:00	Primary		< 100	< 100	< 100	< 100	< 100		< 100
16PB05	3/18/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		< 1
16PB06	3/18/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		< 1
16PB07	3/18/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		< 1
16PB08	3/21/95	0:00	Primary		< 10000	< 10000	< 10000	< 10000	< 10000		< 10000
16PB09	3/18/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		< 1
16PB10	3/20/95	0:00	Primary		< 1	< 1	21	< 1	< 1		< 1
16PB11	3/20/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		< 1
16PB13	3/21/95	0:00	Primary		< 1	< 1	10	< 1	< 1		< 1
16PB14	3/21/95	0:00	Primary		< 1	< 1	3.6	< 1	< 1		< 1
16PB16	3/20/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		< 1
16PB17	3/20/95	0:00	Primary		< 1	< 1	34	< 1	< 1		< 1
16PB19	3/21/95	0:00	Primary		< 1	< 1	2.7	< 1	< 1		< 1
16PB20	3/21/95	0:00	Primary		< 100	< 100	< 100	< 100	< 100		< 100
16PB21	3/20/95	0:00	Primary		< 1	< 1	5.9	< 1	< 1		< 1
16PB25	3/28/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		< 1
16PB26	3/28/95	0:00	Primary		< 100	< 100	< 100	< 100	< 100		< 100
16PB27	3/28/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		< 1
16PB28	3/28/95	0:00	Primary		< 1	< 1	< 1	< 1	< 1		< 1
16WW01	6/10/93	0:00	Primary		< 50	< 50	< 50	< 50	< 50		< 25
16WW01	6/14/95	14:18	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW01	6/25/97	0:00	Primary	< 20	< 2	< 2	< 5	< 1	< 5	< 5	< 2
16WW02	6/10/93	0:00	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW02	6/13/95	16:30	Primary		< 10	< 10	< 10	< 10	< 10		< 5

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Methacrylo nitrile (ug/l)	Methyl bromide (ug/l)	Methyl chloride (ug/l)	Methyl ethyl ketone (ug/l)	Methyl iodide (ug/l)	Methyl isobutyl ketone (MIBK) (ug/l)	Methyl methacrylate (ug/l)	Methylene bromide (ug/l)
16WW03	6/8/93	0:00	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW03	6/14/95	16:04	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW04	6/8/93	0:00	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW04	6/15/95	10:58	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW04	6/15/95	10:58	Duplicate 1		< 10	< 10	< 10	< 10	< 10		< 5
16WW05	6/11/93	0:00	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW05	6/11/93	0:00	Duplicate 1		< 10	< 10	< 10	< 10	< 10		< 5
16WW05	6/14/95	16:48	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW05	10/22/97	17:10	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW06	6/11/93	0:00	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW06	6/14/95	16:42	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW06	10/22/97	14:50	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW07	6/11/93	0:00	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW07	6/14/95	12:00	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW08	6/11/93	0:00	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW08	6/14/95	11:40	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW08	6/14/95	11:40	Duplicate 1		< 10	< 10	< 10	< 10	< 10		< 5
16WW09	6/10/93	0:00	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW09	6/15/95	12:15	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW10	6/9/93	0:00	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW10	6/15/95	15:40	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW11	6/9/93	0:00	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW11	6/9/93	0:00	Duplicate 1		< 10	< 10	< 10	< 10	< 10		< 5
16WW11	6/16/95	11:42	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW12	6/13/95	17:45	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW12	10/27/97	13:39	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW12	10/27/97	13:39	Duplicate 1	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW12	1/20/98	16:15	Primary	< 1600	< 160	< 160	< 400	< 400	< 400	< 400	< 160
16WW12	6/11/98	12:05	Primary	< 1600	< 160	< 160	< 400	< 400	< 400	< 400	< 160
16WW13	6/13/95	16:55	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW13	10/27/97	16:05	Primary	< 20	< 2	< 2	6.5	< 5	< 5	< 5	< 2
16WW14	6/13/95	16:45	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW14	10/24/97	13:55	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW14	1/20/98	13:30	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW14	1/20/98	13:30	Duplicate 1	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW14	6/15/98	9:20	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW15	6/22/95	10:10	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW15	10/28/97	15:25	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Methacrylo nitrile (ug/l)	Methyl bromide (ug/l)	Methyl chloride (ug/l)	Methyl ethyl ketone (ug/l)	Methyl iodide (ug/l)	Methyl isobutyl ketone (MIBK) (ug/l)	Methyl methacrylate (ug/l)	Methylene bromide (ug/l)
16WW16	6/15/95	10:40	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW16	10/27/97	16:00	Primary	< 200	< 20	< 20	< 50	< 50	< 50	< 50	< 20
16WW16	1/21/98	12:15	Primary	< 80000	< 8000	< 8000	< 20000	< 20000	< 20000	< 20000	< 8000
16WW16	6/15/98	11:15	Primary	< 80000	< 8000	< 8000	< 20000	< 20000	< 20000	< 20000	< 8000
16WW17	6/16/95	15:07	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW17	10/27/97	16:44	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW18	6/15/95	14:08	Primary		< 10	< 10	< 10	< 10	< 10		< 5
16WW18	10/28/97	15:09	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW19	10/24/97	11:15	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW20	10/24/97	16:15	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW21	10/28/97	14:45	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW22	10/24/97	13:44	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW22	1/20/98	14:50	Primary	< 1000	< 100	< 100	< 250	< 250	< 250	< 250	< 100
16WW22	6/11/98	10:15	Primary	< 1000	< 100	< 100	< 250	< 250	< 250	< 250	< 100
16WW23	10/23/97	10:39	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW24	10/23/97	12:49	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW25	10/23/97	10:39	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW25	1/21/98	17:35	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW25	6/12/98	11:10	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW26	10/23/97	12:49	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW26	1/21/98	17:00	Primary	< 160	< 16	< 16	< 40	< 40	< 40	< 40	< 16
16WW26	6/12/98	9:30	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW27	10/21/97	10:50	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW28	10/21/97	12:55	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW29	10/21/97	10:55	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW29	1/22/98	13:15	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW29	6/11/98	14:30	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW29	6/11/98	14:30	Duplicate 1	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW30	10/21/97	12:40	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW30	1/22/98	13:30	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW30	6/15/98	14:00	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW31	10/22/97	10:09	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW31	10/22/97	10:09	Duplicate 1	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW32	10/22/97	12:40	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW33	10/22/97	10:00	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW34	10/22/97	12:20	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW35	10/24/97	11:10	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW35	1/21/98	11:30	Primary	< 160	< 16	< 16	< 40	< 40	< 40	< 40	< 16

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Methacrylo nitrile (ug/l)	Methyl bromide (ug/l)	Methyl chloride (ug/l)	Methyl ethyl ketone (ug/l)	Methyl iodide (ug/l)	Methyl isobutyl ketone (MIBK) (ug/l)	Methyl methacrylate (ug/l)	Methylene bromide (ug/l)
16WW35	6/12/98	13:35	Primary	< 100	< 10	< 10	< 25	< 25	< 25	< 25	< 10
16WW36	10/27/97	13:50	Primary	< 200	< 20	< 20	< 50	< 50	< 50	< 50	< 20
16WW36	1/20/98	16:15	Primary	< 4000	< 400	< 400	< 1000	< 1000	< 1000	< 1000	< 400
16WW36	6/12/98	15:30	Primary	< 4000	< 400	< 400	< 1000	< 1000	< 1000	< 1000	< 400
16WW37	10/21/97	16:39	Primary	< 1000	< 100	< 100	< 250	< 250	< 250	< 250	< 100
16WW37	10/21/97	16:39	Duplicate 1	< 1000	< 100	< 100	< 250	< 250	< 250	< 250	< 100
16WW37	1/21/98	13:30	Primary	< 500	< 50	< 50	< 120	< 120	< 120	< 120	< 50
16WW37	6/14/98	11:00	Primary	< 500	< 50	< 50	< 120	< 120	< 120	< 120	< 50
16WW38	10/22/97	16:09	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
16WW38	1/21/98	13:00	Primary	< 200	< 20	< 20	< 50	< 50	< 50	< 50	< 20
16WW38	6/14/98	12:45	Primary	< 20	< 2	< 2	< 5	< 5	< 5	< 5	< 2
MAXIMUM				80000	10000	10000	20000	20000	20000	20000	10000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Methylene chloride (ug/l)		Naphthalene (ug/l)	n-Butylbenzene (ug/l)	n-Propyl benzene (ug/l)	o-Chlorotoluene (ug/l)	o-Dichloro benzene (ug/l)	o-Xylene (ug/l)	p-Chlorotoluene (ug/l)
122	6/2/93	0:00	Primary	< 5								
122	6/14/95	13:42	Primary	< 10								
16EW01	2/21/96	10:55	Primary	256	J							
16EW01	2/21/96	16:48	Primary	< 2500								
16EW01	2/21/96	20:35	Primary	< 2500								
16EW01	2/24/96	12:35	Primary	< 2500								
16EW01	2/25/96	23:55	Primary	< 2500								
16EW01	2/25/96	23:55	Duplicate 1	< 2500								
16EW01	2/27/96	12:10	Primary	< 2500								
16EW01	3/16/96	11:30	Primary	< 2500								
16EW01	3/17/96	16:10	Primary	< 2500								
16EW01	3/19/96	11:30	Primary	< 2500								
16EW01	10/29/97	10:00	Primary	3500		< 1000	< 1000	< 1000	< 1000	< 1000		< 1000
16EW01	10/29/97	10:00	Duplicate 1	3500		< 1000	< 1000	< 1000	< 1000	< 1000		< 1000
16EW01	1/22/98	12:15	Primary	57		< 40	< 40	< 40	< 40	< 40		< 40
16EW01	6/11/98	15:10	Primary	1900		< 40	< 40	< 40	< 40	< 40		< 40
16EW01	6/11/98	15:10	Duplicate 1	2100		< 40	< 40	< 40	< 40	< 40		< 40
16EW02	2/23/96	9:40	Primary	< 1000								
16EW02	2/23/96	14:00	Primary	< 1000								
16EW02	2/28/96	12:20	Primary	< 1000								
16EW02	2/29/96	11:10	Primary	< 1000								
16EW02	2/29/96	21:25	Primary	< 1000								
16EW02	3/13/96	11:35	Primary	< 1000								
16EW02	3/14/96	12:00	Primary	< 1000								
16EW02	3/14/96	12:00	Duplicate 1	< 1000								
16EW02	3/16/96	12:00	Primary	< 1000								
16EW02	3/19/96	12:00	Primary	< 1000								
16EW02	10/29/97	10:30	Primary	< 400		< 400	< 400	< 400	< 400	< 400		< 400
16EW02	1/22/98	12:00	Primary	< 250		< 250	< 250	< 250	< 250	< 250		< 250
16EW02	6/11/98	15:15	Primary	< 200		< 200	< 200	< 200	< 200	< 200		< 200
16EW03	10/29/97	11:19	Primary	< 40		< 40	< 40	< 40	< 40	< 40		< 40
16EW03	1/22/98	11:55	Primary	< 40		< 40	< 40	< 40	< 40	< 40		< 40
16EW03	6/11/98	15:20	Primary	< 40		< 40	< 40	< 40	< 40	< 40		< 40
16EW04	10/29/97	10:50	Primary	< 25		< 25	< 25	< 25	< 25	< 25		< 25
16EW04	1/22/98	11:50	Primary	< 20		< 20	< 20	< 20	< 20	< 20		< 20
16EW04	6/11/98	15:25	Primary	< 10		< 10	< 10	< 10	< 10	< 10		< 10
16EW05	10/29/97	11:15	Primary	< 1000		< 1000	< 1000	< 1000	< 1000	< 1000		< 1000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Methylene chloride (ug/l)		Naphthalene (ug/l)	n-Butylbenzene (ug/l)	n-Propyl benzene (ug/l)	o-Chlorotoluene (ug/l)	o-Dichloro benzene (ug/l)	o-Xylene (ug/l)	p-Chlorotoluene (ug/l)
16EW05	1/22/98	11:35	Primary	< 500		< 500	< 500	< 500	< 500	< 500		< 500
16EW05	6/11/98	15:30	Primary	< 500		< 500	< 500	< 500	< 500	< 500		< 500
16EW06	10/29/97	12:20	Primary	< 400		< 400	< 400	< 400	< 400	< 400		< 400
16EW06	1/22/98	11:25	Primary	< 20		< 20	< 20	< 20	< 20	< 20		< 20
16EW06	6/11/98	15:35	Primary	< 20		< 20	< 20	< 20	< 20	< 20		< 20
16EW07	10/29/97	11:54	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16EW07	1/22/98	11:45	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16EW07	6/11/98	15:40	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16EW08	10/29/97	11:30	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16EW08	1/22/98	10:10	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16EW08	1/22/98	10:10	Duplicate 1	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16EW08	6/11/98	15:45	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16PB02	3/18/95	0:00	Primary	< 1								
16PB02	3/18/95	0:00	Duplicate 1	< 1								
16PB04	3/18/95	0:00	Primary	< 100								
16PB05	3/18/95	0:00	Primary	2								
16PB06	3/18/95	0:00	Primary	< 1								
16PB07	3/18/95	0:00	Primary	< 1								
16PB08	3/21/95	0:00	Primary	< 10000								
16PB09	3/18/95	0:00	Primary	2								
16PB10	3/20/95	0:00	Primary	3.1								
16PB11	3/20/95	0:00	Primary	< 1								
16PB13	3/21/95	0:00	Primary	< 1								
16PB14	3/21/95	0:00	Primary	110								
16PB16	3/20/95	0:00	Primary	< 1								
16PB17	3/20/95	0:00	Primary	< 1								
16PB19	3/21/95	0:00	Primary	< 1								
16PB20	3/21/95	0:00	Primary	< 100								
16PB21	3/20/95	0:00	Primary	< 1								
16PB25	3/28/95	0:00	Primary	< 1								
16PB26	3/28/95	0:00	Primary	< 100								
16PB27	3/28/95	0:00	Primary	< 1								
16PB28	3/28/95	0:00	Primary	< 1								
16WW01	6/10/93	0:00	Primary	< 25								
16WW01	6/14/95	14:18	Primary	(4)	J							
16WW01	6/25/97	0:00	Primary			< 1	< 1		< 1	< 1	< 1	< 1
16WW02	6/10/93	0:00	Primary	< 5								
16WW02	6/13/95	16:30	Primary	< 10								

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Methylene chloride (ug/l)		Naphthalene (ug/l)	n-Butylbenzene (ug/l)	n-Propyl benzene (ug/l)	o-Chlorotoluene (ug/l)	o-Dichloro benzene (ug/l)	o-Xylene (ug/l)	p-Chlorotoluene (ug/l)
16WW03	6/8/93	0:00	Primary	520								
16WW03	6/14/95	16:04	Primary	73								
16WW04	6/8/93	0:00	Primary	< 5								
16WW04	6/15/95	10:58	Primary	(5)	J							
16WW04	6/15/95	10:58	Duplicate 1	16								
16WW05	6/11/93	0:00	Primary	< 5								
16WW05	6/11/93	0:00	Duplicate 1	< 5								
16WW05	6/14/95	16:48	Primary	(4)	J							
16WW05	10/22/97	17:10	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW06	6/11/93	0:00	Primary	< 5								
16WW06	6/14/95	16:42	Primary	< 10								
16WW06	10/22/97	14:50	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW07	6/11/93	0:00	Primary	< 5								
16WW07	6/14/95	12:00	Primary	< 10								
16WW08	6/11/93	0:00	Primary	< 5								
16WW08	6/14/95	11:40	Primary	< 10								
16WW08	6/14/95	11:40	Duplicate 1	< 10								
16WW09	6/10/93	0:00	Primary	< 5								
16WW09	6/15/95	12:15	Primary	(4)	J							
16WW10	6/9/93	0:00	Primary	< 5								
16WW10	6/15/95	15:40	Primary	(3)	J							
16WW11	6/9/93	0:00	Primary	< 5								
16WW11	6/9/93	0:00	Duplicate 1	< 5								
16WW11	6/16/95	11:42	Primary	< 10								
16WW12	6/13/95	17:45	Primary	< 10								
16WW12	10/27/97	13:39	Primary	(0.51)		< 1	< 1	< 1	< 1	< 1		< 1
16WW12	10/27/97	13:39	Duplicate 1	(0.56)		< 1	< 1	< 1	< 1	< 1		< 1
16WW12	1/20/98	16:15	Primary	< 80		< 80	< 80	< 80	< 80	< 80		< 80
16WW12	6/11/98	12:05	Primary	< 80		< 80	< 80	< 80	< 80	< 80		< 80
16WW13	6/13/95	16:55	Primary	(3)	J							
16WW13	10/27/97	16:05	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW14	6/13/95	16:45	Primary	< 10								
16WW14	10/24/97	13:55	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW14	1/20/98	13:30	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW14	1/20/98	13:30	Duplicate 1	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW14	6/15/98	9:20	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW15	6/22/95	10:10	Primary	< 10								
16WW15	10/28/97	15:25	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Methylene chloride (ug/l)		Naphthalene (ug/l)	n-Butylbenzene (ug/l)	n-Propyl benzene (ug/l)	o-Chlorotoluene (ug/l)	o-Dichloro benzene (ug/l)	o-Xylene (ug/l)	p-Chlorotoluene (ug/l)
16WW16	6/15/95	10:40	Primary	(5)	J							
16WW16	10/27/97	16:00	Primary	< 10		< 10	< 10	< 10	< 10	< 10		< 10
16WW16	1/21/98	12:15	Primary	< 4000		< 4000	< 4000	< 4000	< 4000	< 4000		< 4000
16WW16	6/15/98	11:15	Primary	< 4000		< 4000	< 4000	< 4000	< 4000	< 4000		< 4000
16WW17	6/16/95	15:07	Primary	(4)	J							
16WW17	10/27/97	16:44	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW18	6/15/95	14:08	Primary	(3)	J							
16WW18	10/28/97	15:09	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW19	10/24/97	11:15	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW20	10/24/97	16:15	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW21	10/28/97	14:45	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW22	10/24/97	13:44	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW22	1/20/98	14:50	Primary	< 50		< 50	< 50	< 50	< 50	< 50		< 50
16WW22	6/11/98	10:15	Primary	< 50		< 50	< 50	< 50	< 50	< 50		< 50
16WW23	10/23/97	10:39	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW24	10/23/97	12:49	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW25	10/23/97	10:39	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW25	1/21/98	17:35	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW25	6/12/98	11:10	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW26	10/23/97	12:49	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW26	1/21/98	17:00	Primary	< 8		< 8	< 8	< 8	< 8	< 8		< 8
16WW26	6/12/98	9:30	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW27	10/21/97	10:50	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW28	10/21/97	12:55	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW29	10/21/97	10:55	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW29	1/22/98	13:15	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW29	6/11/98	14:30	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW29	6/11/98	14:30	Duplicate 1	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW30	10/21/97	12:40	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW30	1/22/98	13:30	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW30	6/15/98	14:00	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW31	10/22/97	10:09	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW31	10/22/97	10:09	Duplicate 1	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW32	10/22/97	12:40	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW33	10/22/97	10:00	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW34	10/22/97	12:20	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW35	10/24/97	11:10	Primary	< 1		< 1	< 1	< 1	< 1	< 1		< 1
16WW35	1/21/98	11:30	Primary	< 8		< 8	< 8	< 8	< 8	< 8		< 8

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Methylene chloride (ug/l)	Naphthalene (ug/l)	n-Butylbenzene (ug/l)	n-Propyl benzene (ug/l)	o-Chlorotoluene (ug/l)	o-Dichloro benzene (ug/l)	o-Xylene (ug/l)	p-Chlorotoluene (ug/l)
16WW35	6/12/98	13:35	Primary	< 5	< 5	< 5	< 5	< 5	< 5		< 5
16WW36	10/27/97	13:50	Primary	< 10	< 10	< 10	< 10	< 10	< 10		< 10
16WW36	1/20/98	16:15	Primary	< 200	< 200	< 200	< 200	< 200	< 200		< 200
16WW36	6/12/98	15:30	Primary	< 200	< 200	< 200	< 200	< 200	< 200		< 200
16WW37	10/21/97	16:39	Primary	< 50	< 50	< 50	< 50	< 50	< 50		< 50
16WW37	10/21/97	16:39	Duplicate 1	< 50	< 50	< 50	< 50	< 50	< 50		< 50
16WW37	1/21/98	13:30	Primary	< 25	< 25	< 25	< 25	< 25	< 25		< 25
16WW37	6/14/98	11:00	Primary	< 25	< 25	< 25	< 25	< 25	< 25		< 25
16WW38	10/22/97	16:09	Primary	< 1	< 1	< 1	< 1	< 1	< 1		< 1
16WW38	1/21/98	13:00	Primary	< 10	< 10	< 10	< 10	< 10	< 10		< 10
16WW38	6/14/98	12:45	Primary	< 1	< 1	< 1	< 1	< 1	< 1		< 1
MAXIMUM				10000	4000	4000	4000	4000	4000	1	4000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	p-Cymene (ug/l)	p-Dichloro benzene (ug/l)	Pentachloro ethane (ug/l)	sec-Butyl benzene (ug/l)	Styrene (ug/l)	tert-Butyl benzene (ug/l)	Tetrachloro ethylene (ug/l)	Toluene (ug/l)
122	6/2/93	0:00	Primary					< 5		< 5	< 5
122	6/14/95	13:42	Primary					< 5		< 5	< 5
16EW01	2/21/96	10:55	Primary					< 5		< 5	< 5
16EW01	2/21/96	16:48	Primary					< 1250		< 1250	< 1250
16EW01	2/21/96	20:35	Primary					< 1250		< 1250	< 1250
16EW01	2/24/96	12:35	Primary					< 1250		< 1250	< 1250
16EW01	2/25/96	23:55	Primary					< 1250		< 1250	< 1250
16EW01	2/25/96	23:55	Duplicate 1					< 1250		< 1250	< 1250
16EW01	2/27/96	12:10	Primary					< 1250		< 1250	< 1250
16EW01	3/16/96	11:30	Primary					< 1250		< 1250	< 1250
16EW01	3/17/96	16:10	Primary					< 1250		< 1250	< 1250
16EW01	3/19/96	11:30	Primary					< 1250		< 1250	< 1250
16EW01	10/29/97	10:00	Primary	< 1000	< 1000	< 10000	< 1000	< 1000	< 1000	< 1000	< 1000
16EW01	10/29/97	10:00	Duplicate 1	< 1000	< 1000	< 10000	< 1000	< 1000	< 1000	< 1000	< 1000
16EW01	1/22/98	12:15	Primary	< 40	< 40	< 400	< 40	< 40	< 40	< 40	< 40
16EW01	6/11/98	15:10	Primary	< 40	< 40	< 400	< 40	< 40	< 40	< 40	< 40
16EW01	6/11/98	15:10	Duplicate 1	< 40	< 40	< 400	< 40	< 40	< 40	< 40	< 40
16EW02	2/23/96	9:40	Primary					< 500		< 500	< 500
16EW02	2/23/96	14:00	Primary					< 500		< 500	< 500
16EW02	2/28/96	12:20	Primary					< 500		< 500	< 500
16EW02	2/29/96	11:10	Primary					< 500		< 500	< 500
16EW02	2/29/96	21:25	Primary					< 500		< 500	< 500
16EW02	3/13/96	11:35	Primary					< 500		< 500	< 500
16EW02	3/14/96	12:00	Primary					< 500		< 500	< 500
16EW02	3/14/96	12:00	Duplicate 1					< 500		< 500	< 500
16EW02	3/16/96	12:00	Primary					< 500		< 500	< 500
16EW02	3/19/96	12:00	Primary					< 500		< 500	< 500
16EW02	10/29/97	10:30	Primary	< 400	< 400	< 4000	< 400	< 400	< 400	< 400	< 400
16EW02	1/22/98	12:00	Primary	< 250	< 250	< 2500	< 250	< 250	< 250	< 250	< 250
16EW02	6/11/98	15:15	Primary	< 200	< 200	< 2000	< 200	< 200	< 200	< 200	< 200
16EW03	10/29/97	11:19	Primary	< 40	< 40	< 400	< 40	< 40	< 40	< 40	< 40
16EW03	1/22/98	11:55	Primary	< 40	< 40	< 400	< 40	< 40	< 40	< 40	< 40
16EW03	6/11/98	15:20	Primary	< 40	< 40	< 400	< 40	< 40	< 40	< 40	< 40
16EW04	10/29/97	10:50	Primary	< 25	< 25	< 250	< 25	< 25	< 25	< 25	< 25
16EW04	1/22/98	11:50	Primary	< 20	< 20	< 200	< 20	< 20	< 20	< 20	< 20
16EW04	6/11/98	15:25	Primary	< 10	< 10	< 100	< 10	< 10	< 10	< 10	< 10
16EW05	10/29/97	11:15	Primary	< 1000	< 1000	< 10000	< 1000	< 1000	< 1000	< 1000	< 1000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	p-Cymene (ug/l)	p-Dichloro benzene (ug/l)	Pentachloro ethane (ug/l)	sec-Butyl benzene (ug/l)	Styrene (ug/l)	tert-Butyl benzene (ug/l)	Tetrachloro ethylene (ug/l)	Toluene (ug/l)
16EW05	1/22/98	11:35	Primary	< 500	< 500	< 5000	< 500	< 500	< 500	< 500	< 500
16EW05	6/11/98	15:30	Primary	< 500	< 500	< 5000	< 500	< 500	< 500	< 500	< 500
16EW06	10/29/97	12:20	Primary	< 400	< 400	< 4000	< 400	< 400	< 400	< 400	< 400
16EW06	1/22/98	11:25	Primary	< 20	< 20	< 200	< 20	< 20	< 20	< 20	< 20
16EW06	6/11/98	15:35	Primary	< 20	< 20	< 200	< 20	< 20	< 20	< 20	< 20
16EW07	10/29/97	11:54	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16EW07	1/22/98	11:45	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16EW07	6/11/98	15:40	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16EW08	10/29/97	11:30	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16EW08	1/22/98	10:10	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16EW08	1/22/98	10:10	Duplicate 1	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16EW08	6/11/98	15:45	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16PB02	3/18/95	0:00	Primary					< 1		< 1	1.4
16PB02	3/18/95	0:00	Duplicate 1					< 1		< 1	1.5
16PB04	3/18/95	0:00	Primary					< 100		< 100	< 100
16PB05	3/18/95	0:00	Primary					< 1		< 1	3.3
16PB06	3/18/95	0:00	Primary					< 1		< 1	< 1
16PB07	3/18/95	0:00	Primary					< 1		< 1	2.2
16PB08	3/21/95	0:00	Primary					< 10000		< 10000	< 10000
16PB09	3/18/95	0:00	Primary					< 1		< 1	1.7
16PB10	3/20/95	0:00	Primary					< 1		< 1	8.2
16PB11	3/20/95	0:00	Primary					< 1		< 1	< 1
16PB13	3/21/95	0:00	Primary					< 1		< 1	4.2
16PB14	3/21/95	0:00	Primary					< 1		1.4	2.1
16PB16	3/20/95	0:00	Primary					< 1		< 1	< 1
16PB17	3/20/95	0:00	Primary					< 1		< 1	3.4
16PB19	3/21/95	0:00	Primary					< 1		< 1	< 1
16PB20	3/21/95	0:00	Primary					< 100		< 100	< 100
16PB21	3/20/95	0:00	Primary					< 1		< 1	1.2
16PB25	3/28/95	0:00	Primary					< 1		< 1	2.8
16PB26	3/28/95	0:00	Primary					< 100		< 100	< 100
16PB27	3/28/95	0:00	Primary					< 1		< 1	< 1
16PB28	3/28/95	0:00	Primary					< 1		< 1	1.2
16WW01	6/10/93	0:00	Primary					< 25		< 25	
16WW01	6/14/95	14:18	Primary					< 5		< 5	< 5
16WW01	6/25/97	0:00	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW02	6/10/93	0:00	Primary					< 5		< 5	
16WW02	6/13/95	16:30	Primary					< 5		< 5	< 5

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	p-Cymene (ug/l)	p-Dichloro benzene (ug/l)	Pentachloro ethane (ug/l)	sec-Butyl benzene (ug/l)	Styrene (ug/l)	tert-Butyl benzene (ug/l)	Tetrachloro ethylene (ug/l)	Toluene (ug/l)
16WW03	6/8/93	0:00	Primary					< 5		< 5	
16WW03	6/14/95	16:04	Primary					< 5		< 5	< 5
16WW04	6/8/93	0:00	Primary					< 5		< 5	
16WW04	6/15/95	10:58	Primary					< 5		< 5	< 5
16WW04	6/15/95	10:58	Duplicate 1					< 5		< 5	< 5
16WW05	6/11/93	0:00	Primary					< 5		< 5	
16WW05	6/11/93	0:00	Duplicate 1					< 5		< 5	
16WW05	6/14/95	16:48	Primary					< 5		< 5	< 5
16WW05	10/22/97	17:10	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW06	6/11/93	0:00	Primary					< 5		< 5	
16WW06	6/14/95	16:42	Primary					< 5		< 5	< 5
16WW06	10/22/97	14:50	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW07	6/11/93	0:00	Primary					< 5		< 5	
16WW07	6/14/95	12:00	Primary					< 5		< 5	< 5
16WW08	6/11/93	0:00	Primary					< 5		< 5	
16WW08	6/14/95	11:40	Primary					< 5		< 5	< 5
16WW08	6/14/95	11:40	Duplicate 1					< 5		< 5	< 5
16WW09	6/10/93	0:00	Primary					< 5		< 5	
16WW09	6/15/95	12:15	Primary					< 5		< 5	< 5
16WW10	6/9/93	0:00	Primary					< 5		< 5	
16WW10	6/15/95	15:40	Primary					< 5		< 5	< 5
16WW11	6/9/93	0:00	Primary					< 5		< 5	
16WW11	6/9/93	0:00	Duplicate 1					< 5		< 5	
16WW11	6/16/95	11:42	Primary					< 5		< 5	< 5
16WW12	6/13/95	17:45	Primary					< 5		< 5	< 5
16WW12	10/27/97	13:39	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW12	10/27/97	13:39	Duplicate 1	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW12	1/20/98	16:15	Primary	< 80	< 80	< 800	< 80	< 80	< 80	< 80	< 80
16WW12	6/11/98	12:05	Primary	< 80	< 80	< 800	< 80	< 80	< 80	< 80	< 80
16WW13	6/13/95	16:55	Primary					< 5		< 5	< 5
16WW13	10/27/97	16:05	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW14	6/13/95	16:45	Primary					< 5		< 5	< 5
16WW14	10/24/97	13:55	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW14	1/20/98	13:30	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW14	1/20/98	13:30	Duplicate 1	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW14	6/15/98	9:20	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW15	6/22/95	10:10	Primary					< 5		< 5	< 5
16WW15	10/28/97	15:25	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	p-Cymene (ug/l)	p-Dichloro benzene (ug/l)	Pentachloro ethane (ug/l)	sec-Butyl benzene (ug/l)	Styrene (ug/l)	tert-Butyl benzene (ug/l)	Tetrachloro ethylene (ug/l)	Toluene (ug/l)
16WW16	6/15/95	10:40	Primary					< 5		< 5	29
16WW16	10/27/97	16:00	Primary	< 10	< 10	< 100	< 10	< 10	< 10	< 10	< 10
16WW16	1/21/98	12:15	Primary	< 4000	< 4000	< 40000	< 4000	< 4000	< 4000	< 4000	< 4000
16WW16	6/15/98	11:15	Primary	< 4000	< 4000	< 40000	< 4000	< 4000	< 4000	< 4000	< 4000
16WW17	6/16/95	15:07	Primary					< 5		< 5	< 5
16WW17	10/27/97	16:44	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW18	6/15/95	14:08	Primary					< 5		< 5	< 5
16WW18	10/28/97	15:09	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW19	10/24/97	11:15	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW20	10/24/97	16:15	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW21	10/28/97	14:45	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW22	10/24/97	13:44	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW22	1/20/98	14:50	Primary	< 50	< 50	< 500	< 50	< 50	< 50	< 50	< 50
16WW22	6/11/98	10:15	Primary	< 50	< 50	< 500	< 50	< 50	< 50	< 50	< 50
16WW23	10/23/97	10:39	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW24	10/23/97	12:49	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW25	10/23/97	10:39	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW25	1/21/98	17:35	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW25	6/12/98	11:10	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW26	10/23/97	12:49	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW26	1/21/98	17:00	Primary	< 8	< 8	< 80	< 8	< 8	< 8	< 8	< 8
16WW26	6/12/98	9:30	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW27	10/21/97	10:50	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW28	10/21/97	12:55	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW29	10/21/97	10:55	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW29	1/22/98	13:15	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW29	6/11/98	14:30	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW29	6/11/98	14:30	Duplicate 1	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW30	10/21/97	12:40	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW30	1/22/98	13:30	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW30	6/15/98	14:00	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW31	10/22/97	10:09	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW31	10/22/97	10:09	Duplicate 1	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW32	10/22/97	12:40	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW33	10/22/97	10:00	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW34	10/22/97	12:20	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW35	10/24/97	11:10	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW35	1/21/98	11:30	Primary	< 8	< 8	< 80	< 8	< 8	< 8	< 8	< 8

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	p-Cymene (ug/l)	p-Dichloro benzene (ug/l)	Pentachloro ethane (ug/l)	sec-Butyl benzene (ug/l)	Styrene (ug/l)	tert-Butyl benzene (ug/l)	Tetrachloro ethylene (ug/l)	Toluene (ug/l)
16WW35	6/12/98	13:35	Primary	< 5	< 5	< 50	< 5	< 5	< 5	< 5	< 5
16WW36	10/27/97	13:50	Primary	< 10	< 10	< 100	< 10	< 10	< 10	< 10	< 10
16WW36	1/20/98	16:15	Primary	< 200	< 200	< 2000	< 200	< 200	< 200	< 200	< 200
16WW36	6/12/98	15:30	Primary	< 200	< 200	< 2000	< 200	< 200	< 200	< 200	< 200
16WW37	10/21/97	16:39	Primary	< 50	< 50	< 500	< 50	< 50	< 50	< 50	< 50
16WW37	10/21/97	16:39	Duplicate 1	< 50	< 50	< 500	< 50	< 50	< 50	< 50	< 50
16WW37	1/21/98	13:30	Primary	< 25	< 25	< 250	< 25	< 25	< 25	< 25	< 25
16WW37	6/14/98	11:00	Primary	< 25	< 25	< 250	< 25	< 25	< 25	< 25	< 25
16WW38	10/22/97	16:09	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
16WW38	1/21/98	13:00	Primary	< 10	< 10	< 100	< 10	< 10	< 10	< 10	< 10
16WW38	6/14/98	12:45	Primary	< 1	< 1	< 10	< 1	< 1	< 1	< 1	< 1
MAXIMUM				4000	4000	40000	4000	10000	4000	10000	10000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Trans-1,3-Dichloropropene (ug/l)	trans-1,4-Dichloro-2-butene (ug/l)	Trichloro ethylene (ug/l)	Trichloro fluoromethane (ug/l)	Vinyl Acetate (ug/l)	Vinyl chloride (ug/l)	Xylene (total) (ug/l)
122	6/2/93	0:00	Primary	< 5		< 5	< 5	< 10	< 10	< 5
122	6/14/95	13:42	Primary	< 5		< 5	< 5	< 10	< 10	< 5
16EW01	2/21/96	10:55	Primary	< 5		34473		< 10	42	< 5
16EW01	2/21/96	16:48	Primary	< 1250		44095		< 2500	< 2500	< 1250
16EW01	2/21/96	20:35	Primary	< 1250		50370		< 2500	< 2500	< 1250
16EW01	2/24/96	12:35	Primary	< 1250		34560		< 2500	< 2500	< 1250
16EW01	2/25/96	23:55	Primary	< 1250		43365		< 2500	< 2500	< 1250
16EW01	2/25/96	23:55	Duplicate 1	< 1250		41730		< 2500	< 2500	< 1250
16EW01	2/27/96	12:10	Primary	< 1250		39345		< 2500	< 2500	< 1250
16EW01	3/16/96	11:30	Primary	< 1250		42498		< 2500	< 2500	< 1250
16EW01	3/17/96	16:10	Primary	< 1250		52380		< 2500	< 2500	< 1250
16EW01	3/19/96	11:30	Primary	< 1250		52060		< 2500	< 2500	< 1250
16EW01	10/29/97	10:00	Primary	< 1000		57000	< 2000	< 5000	< 1000	< 1000
16EW01	10/29/97	10:00	Duplicate 1	< 1000		58000	< 2000	< 5000	< 1000	< 1000
16EW01	1/22/98	12:15	Primary	< 40		2000	< 80	< 200	< 40	< 40
16EW01	6/11/98	15:10	Primary	< 40		44000	< 80	< 200	82	< 40
16EW01	6/11/98	15:10	Duplicate 1	< 40		46000	< 80	< 200	100	< 40
16EW02	2/23/96	9:40	Primary	< 500		12100		< 1000	< 1000	< 500
16EW02	2/23/96	14:00	Primary	< 500		13231		< 1000	< 1000	< 500
16EW02	2/28/96	12:20	Primary	< 500		15797		< 1000	< 1000	< 500
16EW02	2/29/96	11:10	Primary	< 500		15832		< 1000	< 1000	< 500
16EW02	2/29/96	21:25	Primary	< 500		19742		< 1000	< 1000	< 500
16EW02	3/13/96	11:35	Primary	< 500		18710		< 1000	< 1000	< 500
16EW02	3/14/96	12:00	Primary	< 500		17800		< 1000	< 1000	< 500
16EW02	3/14/96	12:00	Duplicate 1	< 500		18400		< 1000	< 1000	< 500
16EW02	3/16/96	12:00	Primary	< 500		19941		< 1000	< 1000	< 500
16EW02	3/19/96	12:00	Primary	< 500		17578		< 1000	< 1000	< 500
16EW02	10/29/97	10:30	Primary	< 400		21000	< 800	< 2000	< 400	< 400
16EW02	1/22/98	12:00	Primary	< 250		18000	< 500	< 1200	< 250	< 250
16EW02	6/11/98	15:15	Primary	< 200		11000	< 400	< 1000	< 200	< 200
16EW03	10/29/97	11:19	Primary	< 40		2200	< 80	< 200	< 40	< 40
16EW03	1/22/98	11:55	Primary	< 40		2000	< 80	< 200	< 40	< 40
16EW03	6/11/98	15:20	Primary	< 40		2800	< 80	< 200	< 40	< 40
16EW04	10/29/97	10:50	Primary	< 25		1900	< 50	< 120	< 25	< 25
16EW04	1/22/98	11:50	Primary	< 20		790	< 40	< 100	< 20	< 20
16EW04	6/11/98	15:25	Primary	< 10		1100	< 20	< 50	< 10	< 10
16EW05	10/29/97	11:15	Primary	< 1000		42000	< 2000	< 5000	< 1000	< 1000

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Trans-1,3-Dichloropropene (ug/l)	trans-1,4-Dichloro-2-butene (ug/l)	Trichloro ethylene (ug/l)	Trichloro fluoromethane (ug/l)	Vinyl Acetate (ug/l)	Vinyl chloride (ug/l)	Xylene (total) (ug/l)
16EW05	1/22/98	11:35	Primary	< 500		46000	< 1000	< 2500	< 500	< 500
16EW05	6/11/98	15:30	Primary	< 500		27000	< 1000	< 2500	< 500	< 500
16EW06	10/29/97	12:20	Primary	< 400		22000	< 800	< 2000	< 400	< 400
16EW06	1/22/98	11:25	Primary	< 20		1100	< 40	< 100	< 20	< 20
16EW06	6/11/98	15:35	Primary	< 20		20000	< 40	< 100	44	< 20
16EW07	10/29/97	11:54	Primary	< 1		(0.84)	< 2	< 5	< 1	< 1
16EW07	1/22/98	11:45	Primary	< 1		2.6	< 2	< 5	< 1	< 1
16EW07	6/11/98	15:40	Primary	< 1		1.6	< 2	< 5	< 1	< 1
16EW08	10/29/97	11:30	Primary	< 1		37	< 2	< 5	< 1	< 1
16EW08	1/22/98	10:10	Primary	< 1		2.3	< 2	< 5	< 1	< 1
16EW08	1/22/98	10:10	Duplicate 1	< 1		3.8	< 2	< 5	< 1	< 1
16EW08	6/11/98	15:45	Primary	< 1		< 1	< 2	< 5	< 1	< 1
16PB02	3/18/95	0:00	Primary	< 1		< 1	< 1	< 1	< 1	1.4
16PB02	3/18/95	0:00	Duplicate 1	< 1		< 1	< 1	< 1	< 1	1.1
16PB04	3/18/95	0:00	Primary	< 100		2300	< 100	< 100	< 100	< 100
16PB05	3/18/95	0:00	Primary	< 1		< 1	< 1	< 1	< 1	2.2
16PB06	3/18/95	0:00	Primary	< 1		< 1	< 1	< 1	< 1	< 1
16PB07	3/18/95	0:00	Primary	< 1		6	< 1	< 1	< 1	2.2
16PB08	3/21/95	0:00	Primary	< 10000		160000	< 10000	< 10000	< 10000	< 10000
16PB09	3/18/95	0:00	Primary	< 1		17000	< 1	< 1	32	1.1
16PB10	3/20/95	0:00	Primary	< 1		15	< 1	< 1	< 1	< 1
16PB11	3/20/95	0:00	Primary	< 1		< 1	< 1	< 1	< 1	< 1
16PB13	3/21/95	0:00	Primary	< 1		80	< 1	< 1	< 1	3.7
16PB14	3/21/95	0:00	Primary	< 1		39000	< 1	< 1	25	1.2
16PB16	3/20/95	0:00	Primary	< 1		< 1	< 1	< 1	< 1	< 1
16PB17	3/20/95	0:00	Primary	< 1		< 1	< 1	< 1	< 1	< 1
16PB19	3/21/95	0:00	Primary	< 1		< 1	< 1	< 1	< 1	< 1
16PB20	3/21/95	0:00	Primary	< 100		9700	< 100	< 100	< 100	< 100
16PB21	3/20/95	0:00	Primary	< 1		3200	< 1	< 1	< 1	< 1
16PB25	3/28/95	0:00	Primary	< 1		< 1	< 1	< 1	< 1	1.3
16PB26	3/28/95	0:00	Primary	< 100		3500	< 100	< 100	< 100	< 100
16PB27	3/28/95	0:00	Primary	< 1		< 1	< 1	< 1	< 1	< 1
16PB28	3/28/95	0:00	Primary	< 1		< 1	< 1	< 1	< 1	< 1
16WW01	6/10/93	0:00	Primary	< 25		560	< 25	< 50	< 50	
16WW01	6/14/95	14:18	Primary	< 5		3950	< 5	< 10	(9) J	< 5
16WW01	6/25/97	0:00	Primary	< 1	< 1	< 1	< 1	< 5		< 1
16WW02	6/10/93	0:00	Primary	< 5		< 5	< 5	< 10	< 10	
16WW02	6/13/95	16:30	Primary	< 5		10	< 5	< 10	< 10	< 5

Longhorn Army Ammunition Plant
Karnac, Texas
Site 16 - Groundwater Results for
Volatile Organic Compounds (VOCs)

SITE	DATE	TIME	RESULT TYPE	Trans-1,3-Dichloropropene (ug/l)	trans-1,4-Dichloro-2-butene (ug/l)	Trichloro ethylene (ug/l)	Trichloro fluoromethane (ug/l)	Vinyl Acetate (ug/l)	Vinyl chloride (ug/l)	Xylene (total) (ug/l)
16WW03	6/8/93	0:00	Primary	< 5		6400	< 5	< 10	< 10	
16WW03	6/14/95	16:04	Primary	< 5		4880	892	< 10	< 10	< 5
16WW04	6/8/93	0:00	Primary	< 5		210	< 5	< 10	< 10	
16WW04	6/15/95	10:58	Primary	< 5		247	< 5	< 10	(8) J	< 5
16WW04	6/15/95	10:58	Duplicate 1	< 5		229	< 5	< 10	< 10	< 5
16WW05	6/11/93	0:00	Primary	< 5		< 5	< 5	< 10	< 10	
16WW05	6/11/93	0:00	Duplicate 1	< 5		< 5	< 5	< 10	< 10	
16WW05	6/14/95	16:48	Primary	< 5		< 5	< 5	< 10	< 10	< 5
16WW05	10/22/97	17:10	Primary	< 1		4.1	< 2	< 5	< 1	< 1
16WW06	6/11/93	0:00	Primary	< 5		< 5	< 5	< 10	< 10	
16WW06	6/14/95	16:42	Primary	< 5		< 5	< 5	< 10	< 10	< 5
16WW06	10/22/97	14:50	Primary	< 1		1.6	< 2	< 5	< 1	< 1
16WW07	6/11/93	0:00	Primary	< 5		< 5	< 5	< 10	< 10	
16WW07	6/14/95	12:00	Primary	< 5		< 5	< 5	< 10	< 10	< 5
16WW08	6/11/93	0:00	Primary	< 5		< 5	< 5	< 10	< 10	
16WW08	6/14/95	11:40	Primary	< 5		< 5	< 5	< 10	< 10	< 5
16WW08	6/14/95	11:40	Duplicate 1	< 5		< 5	< 5	< 10	< 10	< 5
16WW09	6/10/93	0:00	Primary	< 5		< 5	< 5	< 10	< 10	
16WW09	6/15/95	12:15	Primary	< 5		< 5	< 5	< 10	< 10	< 5
16WW10	6/9/93	0:00	Primary	< 5		< 5	< 5	< 10	< 10	
16WW10	6/15/95	15:40	Primary	< 5		6	< 5	< 10	< 10	< 5
16WW11	6/9/93	0:00	Primary	< 5		32	< 5	< 10	< 10	
16WW11	6/9/93	0:00	Duplicate 1	< 5		34	< 5	< 10	< 10	
16WW11	6/16/95	11:42	Primary	< 5		238	< 5	< 10	13	< 5
16WW12	6/13/95	17:45	Primary	< 5		1390	< 5	< 10	< 10	< 5
16WW12	10/27/97	13:39	Primary	< 1		7500	< 2	< 5	12	< 1
16WW12	10/27/97	13:39	Duplicate 1	< 1		7500	< 2	< 5	14	< 1
16WW12	1/20/98	16:15	Primary	< 80		5100	< 160	< 400	< 80	< 80
16WW12	6/11/98	12:05	Primary	< 80		7100	< 160	< 400	< 80	< 80
16WW13	6/13/95	16:55	Primary	< 5		4820	< 5	< 10	(4) J	< 5
16WW13	10/27/97	16:05	Primary	< 1		12000	< 2	< 5	8.8	< 1
16WW14	6/13/95	16:45	Primary	< 5		38	< 5	< 10	< 10	< 5
16WW14	10/24/97	13:55	Primary	< 1		53	< 2	< 5	< 1	< 1
16WW14	1/20/98	13:30	Primary	< 1		82	< 2	< 5	< 1	< 1
16WW14	1/20/98	13:30	Duplicate 1	< 1		82	< 2	< 5	< 1	< 1
16WW14	6/15/98	9:20	Primary	< 1		45	< 2	< 5	< 1	< 1
16WW15	6/22/95	10:10	Primary	< 5		< 5	< 5	< 10	< 10	< 5
16WW15	10/28/97	15:25	Primary	< 1		< 1	< 2	< 5	< 1	< 1

Data Qualifier Notations

< = Non-Detect Result, value presented is the detection limit.
U = Considered Non-Detect based on blank comparison.
J = Estimated laboratory result.

ATTACHMENT 3

BACKGROUND EVALUATION
RISK CALCULATION SPREADSHEETS

INORGANICS BELOW BACKGROUND CONCENTRATIONS REMOVED
INCIDENTAL INGESTION OF ON-SITE SURFICIAL SOILS (0 - 0.5 FEET)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	IR (mg/day)	FI (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Oral Slope Factor (SF) (mg/kg-day)$^{-1}$	Oral Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcino-genic Risk
Inorganics																	
Aluminum	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Antimony	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	0.0004	--	--	--	--
Arsenic	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	1.5	0.0003	--	--	--	--
Barium	295	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.6E-05	1.1E-04	--	0.07	--	--	1.6E-03	2.7E+01
Beryllium	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	0.002	--	--	--	--
Cadmium	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	0.001	--	--	--	--
Calcium	15100	1.0E-06	100	1.0	60	10	43	25,550	3,650	8.2E-04	5.8E-03	--	--	--	--	--	--
Chromium (III)	31.5	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.7E-06	1.2E-05	--	1.5	--	--	8.0E-06	1.3E-01
Chromium (VI)	31.5	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.7E-06	1.2E-05	--	0.003	--	--	4.0E-03	6.7E+01
Cobalt	19.8	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.1E-06	7.6E-06	--	--	--	--	--	--
Copper	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Iron	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Lead	29	1.0E-06	100	1.0	60	10	43	25,550	3,650	1.6E-06	1.1E-05	--	--	--	--	--	--
Magnesium	1030	1.0E-06	100	1.0	60	10	43	25,550	3,650	5.6E-05	3.9E-04	--	--	--	--	--	--
Manganese	1270	1.0E-06	100	1.0	60	10	43	25,550	3,650	6.9E-05	4.9E-04	--	0.14	--	--	3.5E-03	5.8E+01
Mercury	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Nickel	16.23	1.0E-06	100	1.0	60	10	43	25,550	3,650	8.9E-07	6.2E-06	--	0.02	--	--	3.1E-04	5.2E+00
Potassium	1070	1.0E-06	100	1.0	60	10	43	25,550	3,650	5.8E-05	4.1E-04	--	--	--	--	--	--
Selenium	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	0.005	--	--	--	--
Silver	0.81	1.0E-06	100	1.0	60	10	43	25,550	3,650	4.4E-08	3.1E-07	--	0.005	--	--	8.2E-05	1.0E+00
Sodium	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Strontium	62.7	1.0E-06	100	1.0	60	10	43	25,550	3,650	3.4E-06	2.4E-05	--	0.6	--	--	4.0E-05	6.7E-01
Thallium	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	0.007	--	--	--	--
Zinc	55.6	1.0E-06	100	1.0	60	10	43	25,550	3,650	3.0E-06	2.1E-05	--	0.3	--	--	7.0E-05	1.2E+00
Organics																	
Acetone	0.668	1.0E-06	100	1.0	60	10	43	25,550	3,650	3.7E-09	2.6E-08	--	0.1	--	--	2.6E-07	4.3E-03
Butyl Benzyl Phthalate	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Di-n-Butyl Phthalate	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	0.1	--	--	--	--
Methylene Chloride	0.009	1.0E-06	100	1.0	60	10	43	25,550	3,650	4.9E-10	3.4E-09	0.0075	0.06	3.7E-12	1.0E+02	5.7E-08	9.5E-04
Styrene	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Trichloroethene	--	1.0E-06	100	1.0	60	10	43	25,550	3,650	--	--	0.011	0.006	--	--	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x CF x IR x FI x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = $\sum$ CR$_i$
Chemical-Specific HQ = CDI / RfD; HI = CDI$_1$/RfD$_1$ + CDI$_2$/RfD$_2$ + ... + CDI$_i$/RfD$_i$

Total Pathway Risk: 3.7E-12 Hazard Index: 0.010 [d]
0.006 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

INORGANICS BELOW BACKGROUND CONCENTRATIONS REMOVED
INCIDENTAL INGESTION OF ON-SITE SURFICIAL SOILS (0 - 0.5 FEET)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	IR (mg/day)	FI (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Oral Slope Factor (SF) (mg/kg-day)$^{-1}$	Oral Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																	
Aluminum	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Antimony	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	0.0004	--	--	--	--
Arsenic	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	1.5	0.0003	--	--	--	--
Barium	295	1.0E-06	100	0.5	52	10	43	25,550	3,650	7.0E-06	4.9E-05	--	0.07	--	--	7.0E-04	3.5E+01
Beryllium	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	0.002	--	--	--	--
Cadmium	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	0.001	--	--	--	--
Calcium	15100	1.0E-06	100	0.5	52	10	43	25,550	3,650	3.6E-04	2.5E-03	--	--	--	--	--	--
Chromium (III)	31.5	1.0E-06	100	0.5	52	10	43	25,550	3,650	7.5E-07	5.2E-06	--	1.5	--	--	3.5E-06	1.8E-01
Chromium (VI)	31.5	1.0E-06	100	0.5	52	10	43	25,550	3,650	7.5E-07	5.2E-06	--	0.003	--	--	1.7E-03	8.5E+01
Cobalt	19.8	1.0E-06	100	0.5	52	10	43	25,550	3,650	4.7E-07	3.3E-06	--	--	--	--	--	--
Copper	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Iron	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Lead	29	1.0E-06	100	0.5	52	10	43	25,550	3,650	6.9E-07	4.8E-06	--	--	--	--	--	--
Magnesium	1030	1.0E-06	100	0.5	52	10	43	25,550	3,650	2.4E-05	1.7E-04	--	--	--	--	--	--
Manganese	1270	1.0E-06	100	0.5	52	10	43	25,550	3,650	3.0E-05	2.1E-04	--	0.14	--	--	1.5E-03	7.5E+01
Mercury	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Nickel	16.23	1.0E-06	100	0.5	52	10	43	25,550	3,650	3.8E-07	2.7E-06	--	0.02	--	--	1.4E-04	7.0E+00
Potassium	1070	1.0E-06	100	0.5	52	10	43	25,550	3,650	2.5E-05	1.8E-04	--	--	--	--	--	--
Selenium	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	0.005	--	--	--	--
Silver	0.81	1.0E-06	100	0.5	52	10	43	25,550	3,650	1.9E-08	1.3E-07	--	0.005	--	--	2.6E-05	1.3E+00
Sodium	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Strontium	62.7	1.0E-06	100	0.5	52	10	43	25,550	3,650	1.5E-06	1.0E-05	--	0.6	--	--	1.7E-05	8.5E-01
Thallium	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	0.007	--	--	--	--
Zinc	55.6	1.0E-06	100	0.5	52	10	43	25,550	3,650	1.3E-06	9.2E-06	--	0.3	--	--	3.1E-05	1.6E+00
Organics																	
Acetone	0.068	1.0E-06	100	0.5	52	10	43	25,550	3,650	1.6E-09	1.1E-08	--	0.1	--	--	1.1E-07	5.5E-03
Butyl Benzyl Phthalate	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Di-n-Butyl Phthalate	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	0.1	--	--	--	--
Methylene Chloride	0.009	1.0E-06	100	0.5	52	10	43	25,550	3,650	2.1E-10	1.5E-09	0.0075	0.06	1.6E-12	1.0E+02	2.5E-08	1.3E-03
Styrene	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Trichloroethene	--	1.0E-06	100	0.5	52	10	43	25,550	3,650	--	--	0.011	0.006	--	--	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x CF x IR x FI x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR$_i$
Chemical-Specific HQ = CDI / RfD; HI = CDI$_1$/RfD$_1$ + CDI$_2$/RfD$_2$ + ... + CDI$_i$/RfD$_i$

Total Pathway Risk: 1.6E-12 Hazard Index: 0.004 [d]

0.002 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

INORGANICS BELOW BACKGROUND
DERMAL CONTACT WITH ON-SITE SURFICIAL SOILS (0 - 0.5 FEET)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	SA (cm²/day)	AF (mg/cm²)	ABS (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Absorbed) (mg/kg-day)	NC Chronic Daily Intake (Absorbed) (mg/kg-day)	Dermal Adjusted Slope Factor (SF) (mg/kg-day)⁻¹	Dermal Adjusted Reference Dose (RfD) (mg/kg-day)	Dermal Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Dermal Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcino-genic Risk
Inorganics																		
Aluminum	--	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	--[a]	--[a]	--	--	--	--	--	--
Antimony	--	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	--	--	--	0.00012	--	--	--	--
Arsenic	--	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	--	--	5	0.00009	--	--	--	--
Barium	295	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	6.6E-06	4.6E-05	--	0.021	--	--	2.2E-03	22.0
Beryllium	--	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	--	--	--	0.0006	--	--	--	--
Cadmium	--	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	--[a]	--[a]	--	0.0003	--	--	--	--
Calcium	15100	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	3.4E-04	2.4E-03	--	--	--	--	--	--
Chromium (III)	31.5	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	7.1E-07	4.9E-06	--	0.45	--	--	1.1E-05	0.110
Chromium (VI)	31.5	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	7.1E-07	4.9E-06	--	0.0009	--	--	0.005	50.0
Cobalt	19.8	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	4.4E-07	3.1E-06	--	--	--	--	--	--
Copper	--	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Iron	--	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Lead	29	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	6.5E-07	4.5E-06	--	--	--	--	--	--
Magnesium	1030	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	2.3E-05	1.6E-04	--	--	--	--	--	--
Manganese	1270	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	2.8E-05	2.0E-04	--	0.042	--	--	0.005	50.0
Mercury	--	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Nickel	16.23	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	3.6E-07	2.5E-06	--	0.006	--	--	0.000	0.0
Potassium	1070	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	2.4E-05	1.7E-04	--	--	--	--	--	--
Selenium	--	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	--	--	--	0.0015	--	--	--	--
Silver	0.81	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	1.8E-08	1.3E-07	--	0.0015	--	--	0.000	0.0
Sodium	--	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Strontium	62.7	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	1.4E-06	9.8E-06	--	0.18	--	--	0.000	0.0
Thallium	--	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	--	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	--	--	--	0.0021	--	--	--	--
Zinc	35.6	1.0E-06	4,100	1.0	0.01	60	10	43	25,550	3,650	1.2E-06[b]	8.7E-06[b]	--	0.09	--	--	0.000	0.0
Organics																		
Acetone	0.068	1.0E-06	4,100	1.0	0.1	60	10	43	25,550	3,650	1.5E-08	1.1E-07	--	0.1	--	--	0.000	0.0
Butyl Benzyl Phthalate	--	1.0E-06	4,100	1.0	0.1	60	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Di-n-Butyl Phthalate	--	1.0E-06	4,100	1.0	0.1	60	10	43	25,550	3,650	--	--	--	0.1	--	--	--	--
Methylene Chloride	0.009	1.0E-06	4,100	1.0	0.1	60	10	43	25,550	3,650	2.0E-09	1.4E-08	0.0075	0.06	1.5E-11	1.0E+02	0.000	0.0
Styrene	--	1.0E-06	4,100	1.0	0.1	60	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Trichloroethene	--	1.0E-06	4,100	1.0	0.1	60	10	43	25,550	3,650	--	--	0.011	0.006	--	--	--	--

Chronic Daily Intake (CDI) (Absorbed; mg/kg-day) = (CS x CF x SA x AF x ABS x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 1.5E-11 Hazard Index: 0.01[d]

0.01[c]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

INORGANICS BELOW BACKGROUND CONCENTRATIONS REMOVED
DERMAL CONTACT WITH ON-SITE SURFICIAL SOILS (0 - 0.5 FEET)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	SA (cm²/day)	AF (mg/cm²)	ABS (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Absorbed) (mg/kg-day)	NC Chronic Daily Intake (Absorbed) (mg/kg-day)	Dermal Adjusted Slope Factor (SF) (mg/kg-day)⁻¹	Dermal Adjusted Reference Dose (RfD) (mg/kg-day)	Dermal Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Dermal Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcino-genic Risk
Inorganics																		
Aluminum	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	-- [a]	-- [a]	--	--	--	--	--	--
Antimony	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	0.00012	--	--	--	--
Arsenic	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	5	0.00009	--	--	--	--
Barium	295	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	9.5E-07	6.6E-06	--	0.021	--	--	3.1E-04	22.1
Beryllium	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	0.0006	--	--	--	--
Cadmium	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	-- [b]	-- [b]	--	0.0003	--	--	--	--
Calcium	35100	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	4.9E-05	3.4E-04	--	--	--	--	--	--
Chromium (III)	31.5	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	1.0E-07	7.1E-07	--	0.45	--	--	1.6E-06	0.114
Chromium (VI)	31.5	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	1.0E-07	7.1E-07	--	0.0009	--	--	0.001	41.7
Cobalt	19.8	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	6.4E-08	4.5E-07	--	--	--	--	--	--
Copper	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Iron	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Lead	29	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	9.3E-08	6.5E-07	--	--	--	--	--	--
Magnesium	1030	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	3.3E-06	2.3E-05	--	--	--	--	--	--
Manganese	1270	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	4.1E-06	2.9E-05	--	0.042	--	--	0.001	71.4
Mercury	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Nickel	16.23	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	5.2E-08	3.7E-07	--	0.006	--	--	6.2E-05	4.4E+00
Potassium	1070	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	3.4E-06	2.4E-05	--	--	--	--	--	--
Selenium	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	0.0015	--	--	--	--
Silver	0.81	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	2.6E-09	1.8E-08	--	0.0015	--	--	1.2E-05	0.86
Sodium	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Strontium	62.7	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	2.0E-07	1.4E-06	--	0.18	--	--	7.8E-06	0.56
Thallium	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	0.0021	--	--	--	--
Zinc	55.6	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	1.8E-07 [c]	1.3E-06 [c]	--	0.09	--	--	1.4E-05	1.00
Organics																		
Acetone	0.068	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	2.2E-10	1.5E-09	--	0.1	--	--	1.5E-08	0.00
Butyl Benzyl Phthalate	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Di-n-Butyl Phthalate	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	0.1	--	--	--	--
Methylene Chloride	0.009	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	2.9E-11	2.0E-10	0.0075	0.06	2.2E-13	1.0E+02	3.3E-09	0.00
Styrene	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	--	0.2	--	--	--	--
Trichloroethene	--	1.0E-06	3,400	0.2	0.01	52	10	43	25,550	3,650	--	--	0.011	0.006	--	--	--	--

Chronic Daily Intake (CDI) (Absorbed; mg/kg-day) = (CS x CF x SA x AF x ABS x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR$_i$
Chemical-Specific HQ = CDI / RfD; HI = CDI$_1$/RfD$_1$ + CDI$_2$/RfD$_2$ + ... + CDI$_i$/RfD$_i$

Total Pathway Risk: 2.2E-13 Hazard Index: 0.0024 [d]

0.0014 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

INORGANICS BELOW BACKGROUND CONCENTRATIONS REMOVED
INHALATION OF PARTICULATES FROM SURFICIAL SOILS (0-0.5 FEET)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m³/day)	ED (years)	EF (days/yr)	BW (kg)	PEF (m³/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) (mg/kg-day)⁻¹	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																
Aluminum	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Antimony	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Arsenic	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	15	--	--	--	--	--
Barium	295	10	10	52	43	1.32E+09	25,550	3,650	1.1E-09	7.4E-09	--	0.00014	--	--	0.0001	5.0E+00
Beryllium	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	8.4	0.00000057	--	--	--	--
Cadmium	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	6.3	--	--	--	--	--
Calcium	15100	10	10	52	43	1.32E+09	25,550	3,650	5.4E-08	3.8E-07	--	--	--	--	--	--
Chromium (III)	31.5	10	10	52	43	1.32E+09	25,550	3,650	1.1E-10	7.9E-10	--	--	--	--	--	--
Chromium (VI)	31.5	10	10	52	43	1.32E+09	25,550	3,650	1.1E-10	7.9E-10	42	0.0000029	4.6E-09	1.0E+02	2.72E-05	1.4E+00
Cobalt	19.8	10	10	52	43	1.32E+09	25,550	3,650	7.1E-11	5.0E-10	--	--	--	--	--	--
Copper	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Iron	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Lead	29	10	10	52	43	1.32E+09	25,550	3,650	1.0E-10	7.3E-10	--	--	--	--	--	--
Magnesium	1030	10	10	52	43	1.32E+09	25,550	3,650	3.7E-09	2.6E-08	--	--	--	--	--	--
Manganese	1270	10	10	52	43	1.32E+09	25,550	3,650	4.6E-09	3.2E-08	--	0.000014	--	--	0.002	1.0E+02
Mercury	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	0.000086	--	--	--	--
Nickel	16.23	10	10	52	43	1.32E+09	25,550	3,650	5.8E-11	4.1E-10	--	--	--	--	--	--
Potassium	1070	10	10	52	43	1.32E+09	25,550	3,650	3.8E-09	2.7E-08	--	--	--	--	--	--
Selenium	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Silver	0.81	10	10	52	43	1.32E+09	25,550	3,650	2.9E-12	2.0E-11	--	--	--	--	--	--
Sodium	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Strontium	62.7	10	10	52	43	1.32E+09	25,550	3,650	2.2E-10	1.6E-09	--	--	--	--	--	--
Thallium	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Zinc	55.6	10	10	52	43	1.32E+09	25,550	3,650	2.0E-10	1.4E-09	--	--	--	--	--	--
Organics																
Acetone	0.068	10	10	52	43	1.32E+09	25,550	3,650	2.4E-13	1.7E-12	--	--	--	--	--	--
Butyl Benzyl Phthalate	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Di-n-Butyl Phthalate	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Methylene Chloride	0.009	10	10	52	43	1.32E+09	25,550	3,650	3.2E-14	2.3E-13	0.00165	0.857	5.3E-17	0.00	2.7E-13	1.4E-08
Styrene	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	--	0.286	--	--	--	--
Trichloroethene	--	10	10	52	43	1.32E+09	25,550	3,650	--	--	0.00595	--	--	--	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x IR x EF x ED) / (PEF*BW*AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR$_i$
Chemical-Specific HQ = CDI / RfD; HI = CDI$_1$/RfD$_1$ + CDI$_2$/RfD$_2$ + ... + CDI$_i$/RfD$_i$

Total Pathway Risk: 4.6E-09 Hazard Index: 0.002

[1] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[2] Dashes (--) indicate that no toxicity factor was available for this constituent

INORGANICS BELOW BACKGROUND CONCENTRATIONS REMOVED
INHALATION OF PARTICULATES FROM SURFICIAL SOILS (0-0.5 FEET)
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m3/day)	ED (years)	EF (days/yr)	BW (kg)	PEF (m^3/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) (mg/kg-day)$^{-1}$	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																
Aluminum	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Antimony	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Arsenic	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	15	--	--	--	--	--
Barium	295	10	10	60	43	1.32E+09	25,550	3,650	1.2E-09	8.5E-09	--	0.00014	--	--	0.0001	3.3E+00
Beryllium	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	8.4	0.0000057	--	--	--	--
Cadmium	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	6.3	--	--	--	--	--
Calcium	15100	10	10	60	43	1.32E+09	25,550	3,650	6.2E-08	4.4E-07	--	--	--	--	--	--
Chromium (III)	31.5	10	10	60	43	1.32E+09	25,550	3,650	1.3E-10	9.1E-10	--	--	--	--	--	--
Chromium (VI)	31.5	10	10	60	43	1.32E+09	25,550	3,650	1.3E-10	9.1E-10	42	0.000029	5.5E-09	1.0E+02	3.1E-05	1.0E+00
Cobalt	19.8	10	10	60	43	1.32E+09	25,550	3,650	8.2E-11	5.7E-10	--	--	--	--	--	--
Copper	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Iron	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Lead	29	10	10	60	43	1.32E+09	25,550	3,650	1.2E-10	8.4E-10	--	--	--	--	--	--
Magnesium	1930	10	10	60	43	1.32E+09	25,550	3,650	4.0E-09	3.0E-08	--	--	--	--	--	--
Manganese	1270	10	10	60	43	1.32E+09	25,550	3,650	5.3E-09	3.7E-08	--	0.000014	--	--	0.003	1.0E+02
Mercury	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	0.000086	--	--	--	--
Nickel	16.23	10	10	60	43	1.32E+09	25,550	3,650	6.7E-11	4.7E-10	--	--	--	--	--	--
Potassium	1070	10	10	60	43	1.32E+09	25,550	3,650	4.4E-09	3.1E-08	--	--	--	--	--	--
Selenium	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Silver	0.81	10	10	60	43	1.32E+09	25,550	3,650	3.4E-12	2.3E-11	--	--	--	--	--	--
Sodium	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Strontium	62.7	10	10	60	43	1.32E+09	25,550	3,650	2.6E-10	1.8E-09	--	--				
Thallium	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Zinc	55.6	10	10	60	43	1.32E+09	25,550	3,650	2.3E-10	1.6E-09	--	--	--	--	--	--
Organics																
Acetone	0.068	10	10	60	43	1.32E+09	25,550	3,650	2.8E-13	2.0E-12	--	--	--	--	--	--
Butyl Benzyl Phthalate	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Di-n-Butyl Phthalate	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	--	--	--	--	--
Methylene Chloride	0.009	10	10	60	43	1.32E+09	25,550	3,650	3.7E-14	2.6E-13	0.00165	0.857	6.1E-17	0.00	3.0E-13	1.0E-08
Styrene	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	--	0.286	--	--	--	--
Trichloroethene	--	10	10	60	43	1.32E+09	25,550	3,650	--	--	0.00595	--	--	--	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x IR x EF x ED) / (PEF*BW*AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i

Total Pathway Risk: 5.5E-09 Hazard Index: 0.003

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

INORGANICS BELOW BACKGROUND CONCENTRATIONS REMOVED
INHALATION OF VOLATILES FROM SOILS
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m³/day)	ED (years)	EF (days/yr)	BW (kg)	VF (m³/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) (mg/kg-day)⁻¹	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																
Aluminum	--	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Antimony	--	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Arsenic	--	10	10	52	43	--	25,550	3,650	--	--	15	--	--	--	--	--
Barium	179	10	10	52	43	--	25,550	3,650	--	--	--	0.00014	--	--	--	--
Beryllium	--	10	10	52	43	--	25,550	3,650	--	--	8.4	0.0000057	--	--	--	--
Cadmium	--	10	10	52	43	--	25,550	3,650	--	--	6.3	--	--	--	--	--
Calcium	5580	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Chromium (III)	19.4	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Chromium (VI)	19.4	10	10	52	43	--	25,550	3,650	--	--	42	0.0000029	--	--	--	--
Cobalt	12.6	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Copper	--	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Iron	--	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Lead	13.04	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Magnesium	1540	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Manganese	1270	10	10	52	43	--	25,550	3,650	--	--	--	0.000014	--	--	--	--
Mercury	--	10	10	52	43	--	25,550	3,650	--	--	--	0.0000086	--	--	--	--
Nickel	15.8	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Potassium	917	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Selenium	--	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Silver	0.65	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Sodium	--	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Strontium	10.1	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Thallium	--	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	--	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Zinc	44.3	10	10	52	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Organics																
Acetone	0.013	10	10	52	43	5.39E+03	25,550	3,650	1.1E-08	8.0E-08	--	--	--	--	--	--
Butyl Benzyl Phthalate	0.237	10	10	52	43	3.59E+06	25,550	3,650	3.1E-10	2.2E-09	--	--	--	--	--	--
Di-n-Butyl Phthalate	0.373	10	10	52	43	3.44E+06	25,550	3,650	5.1E-10	3.6E-09	--	--	--	--	--	--
Methylene Chloride	0.0049	10	10	52	43	1.07E+03	25,550	3,650	2.2E-08	1.5E-07	0.00165	0.857	3.6E-11	6.5E	1.8E-07	6.7E+01
Styrene	0.0041	10	10	52	43	5.76E+03	25,550	3,650	3.6E-09	2.5E-08	--	0.286	--	--	8.7E-08	3.2E+01
Trichloroethene	0.025	10	10	52	43	1.40E+03	25,550	3,650	8.5E-08	5.9E-07	0.00595	--	5.1E-10	9.3E+01	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x IR x EF x ED) / (VF*PEF*BW*AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR$_i$
Chemical-Specific HQ = CDI / RfD; HI = CDI$_1$/RfD$_1$ + CDI$_2$/RfD$_2$ + ... + CDI$_i$/RfD$_i$

Total Pathway Risk: 5.5E-10 Hazard Index: 0.00000027

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

INORGANICS BELOW BACKGROUND CONCENTRATIONS REMOVED
INHALATION OF VOLATILES FROM SOILS
ON-SITE TRESPASSER POPULATION
CURRENT SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m³/day)	ED (years)	EF (days/yr)	BW (kg)	VF (m^3/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) (mg/kg-day)$^{-1}$	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																
Aluminum	--	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Antimony	--	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Arsenic	--	10	10	60	43	--	25,550	3,650	--	--	15	--	--	--	--	--
Barium	179	10	10	60	43	--	25,550	3,650	--	--	--	0.00014	--	--	--	--
Beryllium	--	10	10	60	43	--	25,550	3,650	--	--	8.4	0.0000057	--	--	--	--
Cadmium	--	10	10	60	43	--	25,550	3,650	--	--	6.3	--	--	--	--	--
Calcium	5580	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Chromium (III)	19.4	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Chromium (VI)	19.4	10	10	60	43	--	25,550	3,650	--	--	42	0.000029	--	--	--	--
Cobalt	12.6	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Copper	--	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Iron	--	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Lead	13.04	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Magnesium	1540	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Manganese	1270	10	10	60	43	--	25,550	3,650	--	--	--	0.000014	--	--	--	--
Mercury	--	10	10	60	43	--	25,550	3,650	--	--	--	0.000086	--	--	--	--
Nickel	15.8	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Potassium	917	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Selenium	--	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Silver	0.65	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Sodium	--	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Strontium	80.1	10	10	60	43	--	25,550	3,650	--	--	--	--				
Thallium	--	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Vanadium	--	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Zinc	44.3	10	10	60	43	--	25,550	3,650	--	--	--	--	--	--	--	--
Organics																
Acetone	0.013	10	10	60	43	5.39E+03	25,550	3,650	1.3E-08	9.2E-08	--	--	--	--	--	--
Butyl Benzyl Phthalate	0.237	10	10	60	43	3.59E+06	25,550	3,650	3.6E-10	2.5E-09	--	--	--	--	--	--
Di-n-Butyl Phthalate	0.373	10	10	60	43	3.44E+06	25,550	3,650	5.9E-10	4.1E-09	--	--	--	--	--	--
Methylene Chloride	0.0049	10	10	60	43	1.07E+03	25,550	3,650	2.5E-08	1.8E-07	0.00165	0.857	4.1E-11	6.61	2.1E-07	6.8E+01
Styrene	0.0044	10	10	60	43	5.76E+03	25,550	3,650	4.2E-09	2.9E-08	--	0.286	--	--	1.0E-07	3.2E+01
Trichloroethene	0.025	10	10	60	43	1.40E+03	25,550	3,650	9.8E-08	6.8E-07	0.00595	--	5.8E-10	93.55	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x IR x EF x ED) / (VF*PEF*BW*AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR_i
Chemical-Specific HQ = CDI / RfD; HI = $CDI_1/RfD_1 + CDI_2/RfD_2 + ... + CDI_i/RfD_i$

Total Pathway Risk: 6.2E-10 Hazard Index: 0.00000031

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

INORGANICS BELOW BACKGROUND
INCIDENTAL INGESTION OF ON-SITE SURFICIAL/SUBSURFACE SOILS (0 - 5 FEET)
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	IR (mg/day)	FI (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Oral Slope Factor (SF) (mg/kg-day)$^{-1}$	Oral Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																	
Aluminum	--	1.0E-06	100	1.0	250	9	70	25,550	3,285	--	--	--	--	--	--	--	--
Antimony	--	1.0E-06	100	1.0	250	9	70	25,550	3,285	--	--	--	0.0004	--	--	--	--
Arsenic	--	1.0E-06	100	1.0	250	9	70	25,550	3,285	--	--	1.5	0.0003	--	--	--	--
Barium	171.8	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.2E-05	1.7E-04	--	0.07	--	--	2.4E-03	2.0E+01
Beryllium	--	1.0E-06	100	1.0	250	9	70	25,550	3,285	--	--	--	0.002	--	--	--	--
Cadmium	--	1.0E-06	100	1.0	250	9	70	25,550	3,285	--	--	--	0.001	--	--	--	--
Calcium	15300	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.9E-03	1.5E-02	--	--	--	--	--	--
Chromium (III)	22.7	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.9E-06	2.2E-05	--	1.5	--	--	1.5E-05	1.3E-01
Chromium (VI)	22.7	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.9E-06	2.2E-05	--	0.003	--	--	7.3E-03	6.1E+01
Cobalt	19.8	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.5E-06	1.9E-05	--	--	--	--	--	--
Copper	--	1.0E-06	100	1.0	250	9	70	25,550	3,285	--	--	--	--	--	--	--	--
Iron	--	1.0E-06	100	1.0	250	9	70	25,550	3,285	--	--	--	--	--	--	--	--
Lead	18.05	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.3E-06	1.8E-05	--	--	--	--	--	--
Magnesium	1420	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.8E-04	1.4E-03	--	--	--	--	--	--
Manganese	1270	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.6E-04	1.2E-03	--	0.14	--	--	8.6E-03	7.2E+01
Mercury	--	1.0E-06	100	1.0	250	9	70	25,550	3,285	--	--	--	--	--	--	--	--
Nickel	11.81	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.5E-06	1.2E-05	--	0.02	--	--	6.0E-04	5.0E+00
Potassium	1560	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.0E-04	1.5E-03	--	--	--	--	--	--
Selenium	--	1.0E-06	100	1.0	250	9	70	25,550	3,285	--	--	--	0.005	--	--	--	--
Silver	0.69	1.0E-06	100	1.0	250	9	70	25,550	3,285	8.7E-08	6.8E-07	--	0.005	--	--	1.4E-04	1.2E+00
Sodium	--	1.0E-06	100	1.0	250	9	70	25,550	3,285	--	--	--	--	--	--	--	--
Strontium	62.7	1.0E-06	100	1.0	250	9	70	25,550	3,285	7.9E-06	6.1E-05	--	0.6	--	--	1.0E-04	8.3E-01
Thallium	--	1.0E-06	100	1.0	250	9	70	25,550	3,285	--	--	--	--	--	--	--	--
Vanadium	--	1.0E-06	100	1.0	250	9	70	25,550	3,285	--	--	--	0.007	--	--	--	--
Zinc	79.2	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.0E-05	7.7E-05	--	0.3	--	--	2.6E-04	2.2E+00
Organics																	
Acetone	0.016	1.0E-06	100	1.0	250	9	70	25,550	3,285	2.0E-09	1.6E-08	--	0.1	--	--	1.6E-07	1.3E-03
Butyl Benzyl Phthalate	0.332	1.0E-06	100	1.0	250	9	70	25,550	3,285	4.2E-08	3.2E-07	--	0.2	--	--	1.6E-06	1.3E-02
Di-n-Butyl Phthalate	0.675	1.0E-06	100	1.0	250	9	70	25,550	3,285	8.5E-08	6.6E-07	--	0.1	--	--	6.6E-06	5.5E-02
Methylene Chloride	0.0064	1.0E-06	100	1.0	250	9	70	25,550	3,285	8.1E-10	6.3E-09	0.0075	0.06	6.1E-12	2.9E+01	1.1E-07	9.2E-04
Styrene	0.0081	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.0E-09	7.9E-09	--	0.2	--	--	4.0E-08	3.3E-04
Trichloroethene	0.011	1.0E-06	100	1.0	250	9	70	25,550	3,285	1.4E-09	1.1E-08	0.011	0.006	1.5E-11	7.1E+01	1.8E-06	1.5E-02

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x CF x IR x FI x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR$_i$

Chemical-Specific HQ = CDI / RfD; HI = CDI$_1$/RfD$_1$ + CDI$_2$/RfD$_2$ + ... + CDI$_i$/RfD$_i$

Total Pathway Risk: 2.1E-11 Hazard Index: 0.019 [d]

0.012 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that an toxicity factor was available for this constituent

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI)

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III)

INORGANICS BELOW BACKGROUND CONCENTRATIONS REMOVED
INCIDENTAL INGESTION OF ON-SITE SURFICIAL/SUBSURFACE SOILS (0 - 5 FEET)
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	IR (mg/day)	FI (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Oral Slope Factor (SF) (mg/kg-day)$^{-1}$	Oral Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																	
Aluminum	--	1.0E-06	100	1.0	250	25	70	25,550	9,125	--	--	--	--	--	--	--	--
Antimony	--	1.0E-06	100	1.0	250	25	70	25,550	9,125	--	--	--	0.0004	--	--	--	--
Arsenic	--	1.0E-06	100	1.0	250	25	70	25,550	9,125	--	--	1.5	0.0003	--	--	--	--
Barium	171.8	1.0E-06	100	1.0	250	25	70	25,550	9,125	6.0E-05	1.7E-04	--	0.07	--	--	2.4E-03	2.0E+01
Beryllium	--	1.0E-06	100	1.0	250	25	70	25,550	9,125	--	--	--	0.002	--	--	--	--
Cadmium	--	1.0E-06	100	1.0	250	25	70	25,550	9,125	--	--	--	0.001	--	--	--	--
Calcium	15100	1.0E-06	100	1.0	250	25	70	25,550	9,125	5.3E-03	1.5E-02	--	--	--	--	--	--
Chromium (III)	22.7	1.0E-06	100	1.0	250	25	70	25,550	9,125	7.9E-06	2.2E-05	--	1.5	--	--	1.5E-05	1.3E-01
Chromium (VI)	22.7	1.0E-06	100	1.0	250	25	70	25,550	9,125	7.9E-06	2.2E-05	--	0.003	--	--	7.3E-03	6.5E+01
Cobalt	19.8	1.0E-06	100	1.0	250	25	70	25,550	9,125	6.9E-06	1.9E-05	--	--	--	--	--	--
Copper	--	1.0E-06	100	1.0	250	25	70	25,550	9,125	--	--	--	--	--	--	--	--
Iron	--	1.0E-06	100	1.0	250	25	70	25,550	9,125	--	--	--	--	--	--	--	--
Lead	18.05	1.0E-06	100	1.0	250	25	70	25,550	9,125	6.3E-06	1.8E-05	--	--	--	--	--	--
Magnesium	1420	1.0E-06	100	1.0	250	25	70	25,550	9,125	5.0E-04	1.4E-03	--	--	--	--	--	--
Manganese	1270	1.0E-06	100	1.0	250	25	70	25,550	9,125	4.4E-04	1.2E-03	--	0.14	--	--	8.6E-03	7.2E+01
Mercury	--	1.0E-06	100	1.0	250[e]	25	70	25,550	9,125	--	--	--	--	--	--	--	--
Nickel	11.81	1.0E-06	100	1.0	250	25	70	25,550	9,125	4.1E-06	1.2E-05	--	0.02	--	--	6.0E-04	5.0E+00
Potassium	1560	1.0E-06	100	1.0	250	25	70	25,550	9,125	5.5E-04	1.5E-03	--	--	--	--	--	--
Selenium	--	1.0E-06	100	1.0	250	25	70	25,550	9,125	--	--	--	0.005	--	--	--	--
Silver	0.69	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.4E-07	6.8E-07	--	0.005	--	--	1.4E-04	1.2E+00
Sodium	--	1.0E-06	100	1.0	250	25	70	25,550	9,125	--	--	--	--	--	--	--	--
Strontium	62.7	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.2E-05	6.1E-05	--	0.6	--	--	1.0E-04	8.3E-01
Thallium	--	1.0E-06	100	1.0	250	25	70	25,550	9,125	--	--	--	--	--	--	--	--
Vanadium	--	1.0E-06	100	1.0	250	25	70	25,550	9,125	--	--	--	0.007	--	--	--	--
Zinc	79.2	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.8E-05	7.7E-05	--	0.3	--	--	2.6E-04	2.2E+00
Organics																	
Acetone	0.016	1.0E-06	100	1.0	250	25	70	25,550	9,125	5.6E-09	1.6E-08	--	0.1	--	--	1.6E-07	1.3E-03
Butyl Benzyl Phthalate	0.332	1.0E-06	100	1.0	250	25	70	25,550	9,125	1.2E-07	3.2E-07	--	0.2	--	--	1.6E-06	1.3E-02
Di-n-Butyl Phthalate	0.675	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.4E-07	6.6E-07	--	0.1	--	--	6.6E-06	5.5E-02
Methylene Chloride	0.0064	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.2E-09	6.3E-09	0.0075	0.06	1.7E-11	2.9E+01	1.1E-07	9.2E-04
Styrene	0.0081	1.0E-06	100	1.0	250	25	70	25,550	9,125	2.8E-09	7.9E-09	--	0.2	--	--	4.0E-08	3.3E-04
Trichloroethene	0.011	1.0E-06	100	1.0	250	25	70	25,550	9,125	3.8E-09	1.1E-08	0.011	0.006	4.2E-11	7.1E+01	1.8E-06	1.5E-02

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x CF x IR x FI x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR$_i$
Chemical-Specific HQ = CDI / RfD; HI = CDI$_1$/RfD$_1$ + CDI$_2$/RfD$_2$ + ... + CDI$_i$/RfD$_i$

Total Pathway Risk: 5.9E-11 Hazard Index: 0.019 [d]
0.012 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

INORGANICS BELOW BACKGROUND CONCENTRATIONS REMOVED
DERMAL CONTACT WITH ON-SITE SURFICIAL/SUBSURFACE SOILS (0 - 5 FEET)
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	SA (cm²/day)	AF (mg/cm²)	ABS (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Absorbed) (mg/kg-day)	NC Chronic Daily Intake (Absorbed) (mg/kg-day)	Dermal Adjusted Slope Factor (SF) (mg/kg-day)⁻¹	Dermal Adjusted Reference Dose (RfD) (mg/kg-day)	Dermal Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Dermal Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																		
Aluminum	--	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	-- [a]	-- [a]	--	--	--	--	--	--
Antimony	--	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	--	--	--	0.00012	--	--	--	--
Arsenic	--	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	--	--	5	0.00009	--	--	--	--
Barium	171.8	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	8.6E-07	6.7E-06	--	0.021	--	--	3.2E-04	1.9E+01
Beryllium	--	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	--	--	--	0.0006	--	--	--	--
Cadmium	--	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	-- [a]	-- [a]	--	0.0003	--	--	--	--
Calcium	15100	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	7.6E-05	5.9E-04	--	--	--	--	--	--
Chromium (III)	22.7	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	1.1E-07	8.9E-07	--	0.45	--	--	2.0E-06	1.2E-01
Chromium (VI)	22.7	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	1.1E-07	8.9E-07	--	0.0009	--	--	9.9E-04	5.8E+01
Cobalt	19.8	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	1.0E-07	7.7E-07	--	--	--	--	--	--
Copper	--	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	--	--	--	--	--	--	--	--
Iron	--	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	--	--	--	--	--	--	--	--
Lead	18.05	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	9.1E-08	7.1E-07	--	--	--	--	--	--
Magnesium	1420	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	7.1E-06	5.6E-05	--	--	--	--	--	--
Manganese	1270	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	6.4E-06	5.0E-05	--	0.042	--	--	1.2E-03	7.1E+01
Mercury	--	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	--	--	--	--	--	--	--	--
Nickel	11.81	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	5.9E-08	4.6E-07	--	0.006	--	--	7.7E-05	4.5E+00
Potassium	1560	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	7.9E-06	6.1E-05	--	--	--	--	--	--
Selenium	--	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	--	--	--	0.0015	--	--	--	--
Silver	0.69	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	3.5E-09	2.7E-08	--	0.0015	--	--	1.8E-05	1.1E+00
Sodium	--	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	--	--	--	--	--	--	--	--
Strontium	62.7	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	3.1E-07	2.5E-06	--	0.18	--	--	1.4E-05	8.2E-01
Thallium	--	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	--	--	--	--	--	--	--	--
Vanadium	--	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	--	--	--	0.0021	--	--	--	--
Zinc	79.2	1.0E-06	2,000	0.2	0.01	250	9	70	25,550	3,285	4.0E-07 [a]	3.1E-06 [a]	--	0.09	--	--	3.4E-05	2.0E+00
Organics																		
Acetone	0.016	1.0E-06	2,000	0.2	0.1	250	9	70	25,550	3,285	8.1E-10	6.3E-09	--	0.1	--	--	6.3E-08	3.7E-03
Butyl Benzyl Phthalate	0.332	1.0E-06	2,000	0.2	0.1	250	9	70	25,550	3,285	1.7E-08	1.3E-07	--	0.2	--	--	6.5E-07	3.8E-02
Di-n-Butyl Phthalate	0.675	1.0E-06	2,000	0.2	0.1	250	9	70	25,550	3,285	3.4E-08	2.6E-07	--	0.1	--	--	2.6E-06	1.5E-03
Methylene Chloride	0.0064	1.0E-06	2,000	0.2	0.1	250	9	70	25,550	3,285	3.2E-10	2.5E-09	0.0075	0.06	2.4E-12	28.24	4.2E-08	0.002
Styrene	0.0081	1.0E-06	2,000	0.2	0.1	250	9	70	25,550	3,285	4.1E-10	3.2E-09	--	0.2	--	--	1.6E-08	9.4E-04
Trichloroethene	0.011	1.0E-06	2,000	0.2	0.1	250	9	70	25,550	3,285	5.5E-10	4.3E-09	0.011	0.006	6.1E-12	7.2E+01	7.2E-07	4.2E-02

Chronic Daily Intake (CDI) (Absorbed; mg/kg-day) = (CS x CF x SA x AF x ABS x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR$_i$
Chemical-Specific HQ = CDI / RfD; HI = CDI$_1$/RfD$_1$ + CDI$_2$/RfD$_2$ + ... + CDI$_i$/RfD$_i$

Total Pathway Risk: 8.5E-12 Hazard Index: 0.0027 [d]

0.0017 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

INORGANICS BELOW BACKGROUND CONCENTRATIONS REMOVED
DERMAL CONTACT WITH ON-SITE SURFICIAL/SUBSURFACE SOILS (0 - 5 FEET)
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	CF (kg/mg)	SA (cm²/day)	AF (mg/cm²)	ABS (unitless)	EF (days/yr)	ED (yr)	BW (kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Absorbed) (mg/kg-day)	NC Chronic Daily Intake (Absorbed) (mg/kg-day)	Dermal Adjusted Slope Factor (SF) (mg/kg-day)[1]	Dermal Adjusted Reference Dose (RfD) (mg/kg-day)	Dermal Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Dermal Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																		
Aluminum	--	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	-- [a]	-- [a]	--	--	--	--	--	--
Antimony	--	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	--	--	--	0.00012	--	--	--	--
Arsenic	--	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	--	--	5	0.00009	--	--	--	--
Barium	171.8	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.9E-05	5.4E-05	--	0.021	--	--	2.6E-03	2.6E+01
Beryllium	--	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	--	--	--	0.0006	--	--	--	--
Cadmium	--	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	-- [a]	-- [a]	--	0.0003	--	--	--	--
Calcium	15100	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.7E-03	4.7E-03	--	--	--	--	--	--
Chromium (III)	22.7	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	2.5E-06	7.1E-06	--	0.45	--	--	1.6E-05	1.6E-01
Chromium (VI)	22.7	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	2.5E-06	7.1E-06	--	0.0009	--	--	7.9E-03	7.9E+01
Cobalt	19.8	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	2.2E-06	6.2E-06	--	--	--	--	--	--
Copper	--	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	--	--	--	--	--	--	--	--
Iron	--	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	--	--	--	--	--	--	--	--
Lead	18.05	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	2.0E-06	5.7E-06	--	--	--	--	--	--
Magnesium	3420	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.6E-04	4.4E-04	--	--	--	--	--	--
Manganese	1270	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.4E-04	4.0E-04	--	0.042	--	--	9.5E-03	9.5E+01
Mercury	--	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	--	--	--	--	--	--	--	--
Nickel	11.81	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.3E-06	3.7E-06	--	0.006	--	--	6.2E-04	6.2E+00
Potassium	1560	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	1.7E-04	4.9E-04	--	--	--	--	--	--
Selenium	--	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	--	--	--	0.0015	--	--	--	--
Silver	0.69	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	7.7E-08	2.2E-07	--	0.0015	--	--	1.5E-04	1.5E+00
Sodium	--	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	--	--	--	--	--	--	--	--
Strontium	62.7	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	7.0E-06	2.0E-05	--	0.18	--	--	1.1E-04	1.1E+00
Thallium	--	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	--	--	--	--	--	--	--	--
Vanadium	--	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	--	--	--	0.0021	--	--	--	--
Zinc	79.2	1.0E-06	3,200	1.0	0.01	250	25	70	25,550	9,125	8.9E-06 [a]	2.5E-05 [a]	--	0.09	--	--	2.8E-04	2.8E+00
Organics																		
Acetone	0.016	1.0E-06	3,200	1.0	0.1	250	25	70	25,550	9,125	1.8E-08	5.0E-08	--	0.1	--	--	5.0E-07	5.0E-03
Butyl Benzyl Phthalate	0.332	1.0E-06	3,200	1.0	0.1	250	25	70	25,550	9,125	3.7E-07	1.0E-06	--	0.2	--	--	5.0E-06	5.0E-02
Di-n-Butyl Phthalate	0.675	1.0E-06	3,200	1.0	0.1	250	25	70	25,550	9,125	7.5E-07	2.1E-06	--	0.1	--	--	2.1E-05	2.1E-01
Methylene Chloride	0.0004	1.0E-06	3,200	1.0	0.1	250	25	70	25,550	9,125	7.2E-09	2.0E-08	0.0075	0.06	5.4E-11	30.00	3.3E-07	0.003
Styrene	0.0081	1.0E-06	3,200	1.0	0.1	250	25	70	25,550	9,125	9.1E-09	2.5E-08	--	0.2	--	--	1.3E-07	1.3E-03
Trichloroethene	0.011	1.0E-06	3,200	1.0	0.1	250	25	70	25,550	9,125	1.2E-08	3.4E-08	0.011	0.006	1.3E-10	7.2E+01	5.7E-06	5.7E-02

Chronic Daily Intake (CDI) (Absorbed; mg/kg-day) = (CS x CF x SA x AF x ABS x EF x ED) / (BW x AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR$_i$
Chemical-Specific HQ = CDI / RfD; HI = CDI$_1$/RfD$_1$ + CDI$_2$/RfD$_2$ + ... + CDI$_i$/RfD$_i$

Total Pathway Risk: 1.8E-10 Hazard Index: 0.02 [d]

0.01 [e]

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

[c] No air concentration could be calculated for this constituent since it does not volatilize.

[d] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (VI).

[e] Chromium by valence not measured (only total chromium available); hazard index (HI) calculated assuming 100 percent chromium (III).

INORGANICS BELOW BACKGROUND CONCENTRATIONS REMOVED
INHALATION OF PARTICULATES FROM SURFICIAL/SUBSURFACE SOILS (0-5 FEET)
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m³/day)	ED (years)	EF (days/yr)	BW (kg)	PEF (m³/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) (mg/kg-day)⁻¹	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																
Aluminum	--	20	9	250	70	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Antimony	--	20	9	250	70	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Arsenic	--	20	9	250	70	1.32E+09	25,550	3,285	--	--	15	--	--	--	--	--
Barium	171 B	20	9	250	70	1.32E+09	25,550	3,285	3.3E-09	2.5E-08	--	0.00014	--	--	0.0002	1.4E+00
Beryllium	--	20	9	250	70	1.32E+09	25,550	3,285	--	--	8.4	0.0000057	--	--	--	--
Cadmium	--	20	9	250	70	1.32E+09	25,550	3,285	--	--	6.3	--	--	--	--	--
Calcium	15100	20	9	250	70	1.32E+09	25,550	3,285	2.9E-07	2.2E-06	--	--	--	--	--	--
Chromium (III)	22.7	20	9	250	70	1.32E+09	25,550	3,285	4.3E-10	3.4E-09	--	--	--	--	--	--
Chromium (VI)	22.7	20	9	250	70	1.32E+09	25,550	3,285	4.3E-10	3.4E-09	42	0.0000029	1.8E-08	1.0E+02	1.1E-04	8.4E-01
Cobalt	19.8	20	9	250	70	1.32E+09	25,550	3,285	3.8E-10	2.9E-09	--	--	--	--	--	--
Copper	--	20	9	250	70	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Iron	--	20	9	250	70	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Lead	18.05	20	9	250	70	1.32E+09	25,550	3,285	3.4E-10	2.7E-09	--	--	--	--	--	--
Magnesium	1420	20	9	250	70	1.32E+09	25,550	3,285	2.7E-08	2.1E-07	--	--	--	--	--	--
Manganese	1270	20	9	250	70	1.32E+09	25,550	3,285	2.4E-08	1.9E-07	--	0.000014	--	--	0.014	1.0E+02
Mercury	--	20	9	250	70	1.32E+09	25,550	3,285	--	--	--	0.0000086	--	--	--	--
Nickel	11.81	20	9	250	70	1.32E+09	25,550	3,285	2.3E-10	1.8E-09	--	--	--	--	--	--
Potassium	1560	20	9	250	70	1.32E+09	25,550	3,285	3.0E-08	2.3E-07	--	--	--	--	--	--
Selenium	--	20	9	250	70	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Silver	0.69	20	9	250	70	1.32E+09	25,550	3,285	1.3E-11	1.0E-10	--	--	--	--	--	--
Sodium	--	20	9	250	70	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Strontium	62.7	20	9	250	70	1.32E+09	25,550	3,285	1.2E-09	9.3E-09	--	--	--	--	--	--
Thallium	--	20	9	250	70	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Vanadium	--	20	9	250	70	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Zinc	70.2	20	9	250	70	1.32E+09	25,550	3,285	1.5E-09	1.2E-08	--	--	--	--	--	--
Organics																
Acetone	0.016	20	9	250	70	1.32E+09	25,550	3,285	3.0E-13	2.4E-12	--	--	--	--	--	--
Butyl Benzyl Phthalate	0.332	20	9	250	70	1.32E+09	25,550	3,285	6.3E-12	4.9E-11	--	--	--	--	--	--
Di-n-Butyl Phthalate	0.075	20	9	250	70	1.32E+09	25,550	3,285	1.3E-11	1.0E-10	--	--	--	--	--	--
Methylene Chloride	0.0061	20	9	250	70	1.32E+09	25,550	3,285	1.2E-13	9.5E-13	0.00165	0.857	2.0E-16	0.00	1.1E-12	7.9E-09
Styrene	0.0081	20	9	250	70	1.32E+09	25,550	3,285	1.5E-13	1.2E-12	--	0.286	--	--	4.2E-12	3.0E-08
Trichloroethene	0.011	20	9	250	70	1.32E+09	25,550	3,285	2.1E-13	1.6E-12	0.00595	--	1.2E-15	6.7E-06	--	--

*Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x IR x EF x ED) / (PEF*BW*AT)*
Chemical-Specific CR = CDI x SF; Total CR = Σ CR$_i$

Chemical-Specific HQ = CDI / RfD; HI = CDI$_1$/RfD$_1$ + CDI$_2$/RfD$_2$ + ... + CDI$_i$/RfD$_i$

Total Pathway Risk: 1.8E-08 Hazard Index: 0.014

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

INORGANICS BELOW BACKGROUND CONCENTRATIONS REMOVED
INHALATION OF PARTICULATES FROM SURFICIAL/SUBSURFACE SOILS (0-5 FEET)
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m3/day)	ED (years)	EF (days/yr)	BW (kg)	PEF (m^3/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) (mg/kg-day)$^{-1}$	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																
Aluminum	--	20	25	250	70	1.32E+09	25,550	9,125	--	--	--	--	--	--	--	--
Antimony	--	20	25	250	70	1.32E+09	25,550	9,125	--	--	--	--	--	--	--	--
Arsenic	--	20	25	250	70	1.32E+09	25,550	9,125	--	--	15	--	--	--	--	--
Barium	171.8	20	25	250	70	1.32E+09	25,550	9,125	9.1E-09	2.5E-08	--	0.00014	--	--	0.0002	1.4E+00
Beryllium	--	20	25	250	70	1.32E+09	25,550	9,125	--	--	8.4	0.00000057	--	--	--	--
Cadmium	--	20	25	250	70	1.32E+09	25,550	9,125	--	--	6.3	--	--	--	--	--
Calcium	15100	20	25	250	70	1.32E+09	25,550	9,125	8.0E-07	2.2E-06	--	--	--	--	--	--
Chromium (III)	22.7	20	25	250	70	1.32E+09	25,550	9,125	1.2E-09	3.4E-09	--	--	--	--	--	--
Chromium (VI)	22.7	20	25	250	70	1.32E+09	25,550	9,125	1.2E-09	3.4E-09	42	0.000029	5.0E-08	1.0E+02	1.17E-04	8.4E-01
Cobalt	10.8	20	25	250	70	1.32E+09	25,550	9,125	1.0E-09	2.9E-09	--	--	--	--	--	--
Copper	--	20	25	250	70	1.32E+09	25,550	9,125	--	--	--	--	--	--	--	--
Iron	--	20	25	250	70	1.32E+09	25,550	9,125	--	--	--	--	--	--	--	--
Lead	18.05	20	25	250	70	1.32E+09	25,550	9,125	9.6E-10	2.7E-09	--	--	--	--	--	--
Magnesium	1420	20	25	250	70	1.32E+09	25,550	9,125	7.5E-08	2.1E-07	--	--	--	--	--	--
Manganese	1270	20	25	250	70	1.32E+09	25,550	9,125	6.7E-08	1.9E-07	--	0.000014	--	--	0.014	1.0E+02
Mercury	--	20	25	250	70	1.32E+09	25,550	9,125	--	--	--	0.000086	--	--	--	--
Nickel	11.81	20	25	250	70	1.32E+09	25,550	9,125	6.3E-10	1.8E-09	--	--	--	--	--	--
Potassium	1560	20	25	250	70	1.32E+09	25,550	9,125	8.3E-08	2.3E-07	--	--	--	--	--	--
Selenium	--	20	25	250	70	1.32E+09	25,550	9,125	--	--	--	--	--	--	--	--
Silver	0.69	20	25	250	70	1.32E+09	25,550	9,125	3.7E-11	1.0E-10	--	--	--	--	--	--
Sodium	--	20	25	250	70	1.32E+09	25,550	9,125	--	--	--	--	--	--	--	--
Strontium	92.7	20	25	250	70	1.32E+09	25,550	9,125	3.3E-09	9.1E-09	--	--	--	--	--	--
Thallium	--	20	25	250	70	1.32E+09	25,550	9,125	--	--	--	--	--	--	--	--
Vanadium	--	20	25	250	70	1.32E+09	25,550	9,125	--	--	--	--	--	--	--	--
Zinc	79.2	20	25	250	70	1.32E+09	25,550	9,125	4.2E-09	1.2E-08	--	--	--	--	--	--
Organics																
Acetone	0.016	20	25	250	70	1.32E+09	25,550	9,125	8.5E-13	2.4E-12	--	--	--	--	--	--
Butyl Benzyl Phthalate	0.332	20	25	250	70	1.32E+09	25,550	9,125	1.8E-11	4.9E-11	--	--	--	--	--	--
Di-n-Butyl Phthalate	0.675	20	25	250	70	1.32E+09	25,550	9,125	3.6E-11	1.0E-10	--	--	--	--	--	--
Methylene Chloride	0.0064	20	25	250	70	1.32E+09	25,550	9,125	3.4E-13	9.5E-13	0.00165	0.857	5.6E-16	0.00	1.1E-12	7.9E-09
Styrene	0.0081	20	25	250	70	1.32E+09	25,550	9,125	4.3E-13	1.2E-12	--	0.286	--	--	4.2E-12	3.0E-08
Trichloroethene	0.011	20	25	250	70	1.32E+09	25,550	9,125	5.8E-13	1.6E-12	0.00595	--	3.5E-15	7.0E-06	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x IR x EF x ED) / (PEF*BW*AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR$_i$
Chemical-Specific HQ = CDI / RfD; HI = CDI$_1$/RfD$_1$ + CDI$_2$/RfD$_2$ + ... + CDI$_i$/RfD$_i$

Total Pathway Risk 5.0E-08 Hazard Index 0.014

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that no toxicity factor was available for this constituent.

INORGANICS BELOW BACKGROUND
INHALATION OF VOLATILES FROM SOILS
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - CENTRAL TENDENCY EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m³/day)	ED (years)	EF (days/yr)	BW (kg)	VF (m³/kg)	PEF (m³/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) (mg/kg-day)⁻¹	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																	
Aluminum	--	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Antimony	--	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Arsenic	--	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	15	--	--	--	--	--
Barium	179	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	0.00014	--	--	--	--
Beryllium	--	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	8.4	0.0000057	--	--	--	--
Cadmium	--	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	6.3	--	--	--	--	--
Calcium	5580	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Chromium (III)	19.4	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Chromium (VI)	19.4	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	42	0.000029	--	--	--	--
Cobalt	12.6	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Copper	--	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Iron	--	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Lead	13.04	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Magnesium	1540	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Manganese	1270	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	0.000014	--	--	--	--
Mercury	--	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	0.0000086	--	--	--	--
Nickel	15.8	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Potassium	917	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Selenium	--	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Silver	0.65	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Sodium	--	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Strontium	40.1	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Thallium	--	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Vanadium	--	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Zinc	54.3	20	9	250	70	--	1.32E+09	25,550	3,285	--	--	--	--	--	--	--	--
Organics																	
Acetone	0.013	20	9	250	70	5.12E+03	1.32E+09	25,550	3,285	6.4E-08	5.0E-07	--	--	--	--	--	--
Butyl Benzyl Phthalate	0.547	20	9	250	70	3.41E+06	1.32E+09	25,550	3,285	1.7E-09	1.4E-08	--	--	--	--	--	--
Di-n-Butyl Phthalate	0.373	20	9	250	70	3.27E+06	1.32E+09	25,550	3,285	2.9E-09	2.2E-08	--	--	--	--	--	--
Methylene Chloride	0.0049	20	9	250	70	1.02E+03	1.32E+09	25,550	3,285	1.2E-07	9.4E-07	0.00165	0.857	2.0E-10	6.6E-07	1.1E-06	6.5E-01
Styrene	0.0014	20	9	250	70	5.47E+03	1.32E+09	25,550	3,285	2.0E-08	1.6E-07	--	0.286	--	--	5.6E-07	3.3E-01
Trichloroethene	0.025	20	9	250	70	1.33E+03	1.32E+09	25,550	3,285	4.7E-07	3.7E-06	0.00595	--	2.8E-09	9.3E-01	--	--

Chronic Daily Intake (CDI) (Administered, mg/kg-day) = (CS x IR x FF x ED) / (VF*BW*AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR$_i$
Chemical-Specific HQ = CDI / RfD; HI = CDI$_1$/RfD$_1$ + CDI$_2$/RfD$_2$ + ... + CDI$_i$/RfD$_i$

Total Pathway Risk: 1.0E-05 Hazard Index: 0.0000017

[a] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[b] Dashes (--) indicate that an toxicity factor was unavailable for this constituent.

INORGANICS BELOW BACKGROUND CONCENTRATIONS REMOVED
INHALATION OF VOLATILES FROM SOILS
ON-SITE MAINTENANCE WORKER POPULATION
FUTURE SCENARIO - REASONABLE MAXIMUM EXPOSURE

SITE 16
LONGHORN ARMY AMMUNITION PLANT
KARNACK, TEXAS

Constituent	CS (mg/kg)	IR (m³/day)	ED (years)	EF (days/yr)	BW (kg)	VF (m³/kg)	C AT (days)	NC AT (days)	C Chronic Daily Intake (Administered) (mg/kg-day)	NC Chronic Daily Intake (Administered) (mg/kg-day)	Inhalation Slope Factor (SF) (mg/kg-day)⁻¹	Inhalation Reference Dose (RfD) (mg/kg-day)	Oral Chemical-Specific Carcinogenic Risk (CR)	% Carcinogenic Risk	Oral Chemical-Specific Noncarcinogenic Hazard Quotient (HQ)	% Noncarcinogenic Risk
Inorganics																
Aluminum	--	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Antimony	--	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Arsenic	--	20	25	250	70	--	25,550	9,125	--	--	15	--	--	--	--	--
Barium	179	20	25	250	70	--	25,550	9,125	--	--	--	0.00014	--	--	--	--
Beryllium	--	20	25	250	70	--	25,550	9,125	--	--	8.4	0.0000057	--	--	--	--
Cadmium	--	20	25	250	70	--	25,550	9,125	--	--	6.3	--	--	--	--	--
Calcium	5580	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Chromium (III)	19.4	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Chromium (VI)	19.4	20	25	250	70	--	25,550	9,125	--	--	42	0.000029	--	--	--	--
Cobalt	12.6	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Copper	--	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Iron	--	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Lead	13.05	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Magnesium	1540	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Manganese	1270	20	25	250	70	--	25,550	9,125	--	--	--	0.000014	--	--	--	--
Mercury	--	20	25	250	70	--	25,550	9,125	--	--	--	0.000086	--	--	--	--
Nickel	15.8	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Potassium	917	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Selenium	--	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Silver	0.65	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Sodium	--	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Strontium	40.1	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Thallium	--	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Vanadium	--	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Zinc	44.3	20	25	250	70	--	25,550	9,125	--	--	--	--	--	--	--	--
Organics																
Acetone	0.013	20	25	250	70	8.53E+03	25,550	9,125	1.1E-07	3.0E-07	--	--	--	--	--	--
Butyl Benzyl Phthalate	0.237	20	25	250	70	5.68E+06	25,550	9,125	2.0E-09	5.2E-09	--	--	--	--	--	--
Di-n-Butyl Phthalate	0.173	20	25	250	70	5.14E+06	25,550	9,125	4.8E-09	1.3E-08	--	--	--	--	--	--
Methylene Chloride	0.0039	20	25	250	70	1.70E+03	25,550	9,125	2.0E-07	5.6E-07	0.00165	0.857	3.3E-10	6.60	6.5E-07	6.5E+01
Styrene	0.0044	20	25	250	70	9.11E+03	25,550	9,125	3.4E-08	9.5E-08	--	0.286	--	--	3.3E-07	3.3E+01
Trichloroethene	0.025	20	25	250	70	2.22E+03	25,550	9,125	7.9E-07	2.2E-06	0.00595	--	4.7E-09	9.4E+01	--	--

Chronic Daily Intake (CDI) (Administered; mg/kg-day) = (CS x IR x EF x ED) / (VF*PEF*BW*AT)
Chemical-Specific CR = CDI x SF; Total CR = Σ CR$_i$
Chemical-Specific HQ = CDI / RfD; HI = CDI$_1$/RfD$_1$ + CDI$_2$/RfD$_2$ + ... + CDI$_i$/RfD$_i$

Total Pathway Risk: 5.0E-09 Hazard Index: 0.000001

[A] Dashes (--) indicate that this constituent was not retained as a constituent of interest in this medium.

[B] Dashes (--) indicate that no toxicity factor was available for this constituent.

ATTACHMENT 4

INTEGRATED RISK INFORMATION SYSTEM (IRIS) PRINTOUTS

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Acetone

CASRN 67-64-1

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0128
Acetone; CASRN 67-64-1

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Acetone

File On-Line 03/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	08/01/1993
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	12/01/1990

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Acetone
CASRN -- 67-64-1
Last Revised -- 08/01/1993

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

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.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Increased liver and kidney weights and nephrotoxicity Rat Oral Subchronic Study U.S. EPA, 1986	NOEL: 100 mg/kg/day LOAEL: 500 mg/kg/day	1000	1	1E-1 mg/kg/day

*Conversion Factors: Actual dose tested

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

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.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

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

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

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.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

00088305

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

Other EPA Documentation -- None

Agency Work Group Review -- 12/18/1985, 05/30/1986, 07/21/1993

Verification Date -- 05/30/1986

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Acetone
CASRN -- 67-64-1

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Acetone
CASRN -- 67-64-1
Last Revised -- 12/01/1990

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for

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Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on lack of data concerning carcinogenicity in humans or animals.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. ANIMAL CARCINOGENICITY DATA

None.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

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).

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

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None.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

None.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1988

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

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 12/06/1989

Verification Date -- 12/06/1989

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

VI. BIBLIOGRAPHY

Substance Name -- Acetone
CASRN -- 67-64-1
Last Revised -- 07/01/1990

__VI.A. ORAL RfD REFERENCES

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__VI.B. INHALATION RfD REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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_VII. REVISION HISTORY

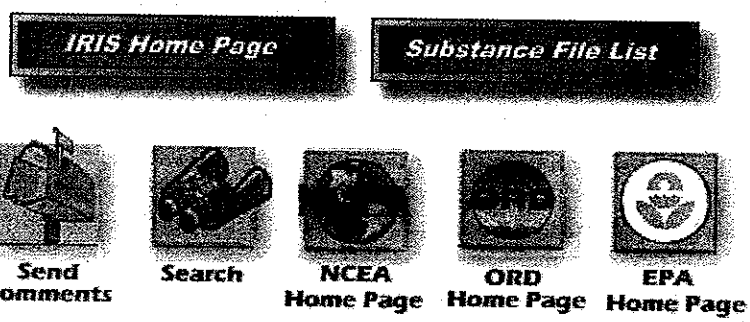
Substance Name -- Acetone
CASRN -- 67-64-1

Date	Section	Description
03/01/1988	I.A.5.	Confidence levels revised
07/01/1989	VI.	Bibliography on-line
01/01/1990	II.	Carcinogen assessment now under review
07/01/1990	II.	Carcinogen assessment on-line
07/01/1990	IV.F.1.	EPA contact changed
07/01/1990	VI.C.	Carcinogen references added
12/01/1990	I.A.2.	Text edited
12/01/1990	I.A.7.	EPA contacts changed
12/01/1990	II.A.4.	Text edited
01/01/1992	IV.	Regulatory actions updated
08/01/1993	I.A.	Oral RfD noted as pending change
08/01/1993	I.A.6.	Work group review date added

VIII. SYNONYMS

Substance Name -- Acetone
CASRN -- 67-64-1
Last Revised -- 03/31/1987

67-64-1
ACETON
Acetone
DIMETHYLFORMALDEHYDE
DIMETHYLKETAL
DIMETHYL KETONE
KETONE, DIMETHYL
KETONE PROPANE
beta-KETOPROPANE
METHYL KETONE
PROPANONE
2-PROPANONE
PYROACETIC ACID
PYROACETIC ETHER
RCRA WASTE NUMBER U002
UN 1090



Last updated: 5 May 1998
URL: http://www.epa.gov/iris/subst/0128.htm

00088311

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Aldrin

CASRN 309-00-2

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0130
Aldrin; CASRN 309-00-2

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Aldrin

File On-Line 03/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	03/01/1988
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	07/01/1993

00088312

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Aldrin
CASRN -- 309-00-2
Last Revised -- 03/01/1988

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Liver toxicity Rat Chronic Feeding Study Fitzhugh et al., 1964	NOAEL: none LOAEL: 0.5 ppm diet (0.025 mg/kg/day)	1000	1	3E-5 mg/kg/day

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

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Fitzhugh, O.G., A.A. Nelson, and M.L. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmet. Toxicol. 2: 551-562.

Groups of 24 rats (12/sex) were fed aldrin in the diet at levels of 0, 0.5, 2, 10, 50, 100, or 150 ppm for 2 years. Liver lesions characteristic of chlorinated insecticide poisoning were observed at dose levels of 0.5 ppm and greater. These lesions were characterized by enlarged centrilobular hepatic cells, with increased cytoplasmic oxyphilia, and peripheral migration of basophilic granules. A statistically significant increase in liver-to-body weight ratio was observed at all dose levels. Kidney lesions occurred at the highest dose levels. Survival was markedly decreased at dose levels of 50 ppm

and greater.

Additional data are fairly supportive. Effect and no-effect levels are similar (to those found for rats) for liver effects in dogs after 15 months' exposure to aldrin in the diet. Liver effects were observed at slightly higher doses in several other subchronic-to-chronic rat and dog studies. Short-term exposure to higher doses resulted in mortality for a number of species.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- The composite UF of 1000 encompasses the uncertainty of extrapolation from animals to humans, the uncertainty in the range of human sensitivities, and an additional uncertainty because the RfD is based on a LOAEL rather than a NOAEL.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

None.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

The principal study, designed as a carcinogenesis bioassay, is strong in histopathologic analysis but lacks other toxicologic parameters, and is therefore rated medium. The data base is fairly extensive, and generally supportive, but is rated medium because of the lack of NOELs for some studies. Also, no chronic data exist for the dog, which may be a more sensitive species than the rat. Medium confidence in the RfD follows.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

U.S. EPA. 1982. Toxicity-Based Protective Ambient Water Levels for Various Carcinogens. Environmental Criteria and Assessment Office, Cincinnati, OH. ECAO-CIN-431. Internal review draft.

The RfD has been reviewed internally by ECAO-Cin.

Agency Work Group Review -- 12/18/1985

Verification Date -- 12/18/1985

I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Aldrin
CASRN -- 309-00-2

Not available at this time.

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Aldrin
CASRN -- 309-00-2
Last Revised -- 07/01/1993

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

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Orally administered aldrin produced significant increases in tumor responses in three different strains of mice in both males and females. Tumor induction has been observed for structurally related chemicals, including dieldrin, a metabolite.

___II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. Two studies of workers exposed to aldrin and dieldrin (a metabolite of aldrin) did not find these workers to have an excess risk of cancer. Both studies, however, were limited in their ability to detect an excess of deaths from cancer. Van Raalte (1977) observed two cases of cancer (gastric and lymphosarcoma) among 166 pesticide manufacturing workers exposed 4 to 19 years and followed from 15 to 20 years. Exposure was not quantified, and workers were also exposed to other organochlorine pesticides (endrin and telodrin). A small number of workers was studied, the mean age of the cohort (47.7 years) was low, the number of expected deaths was not calculated, and the duration of exposure and of latency was relatively short.

In a retrospective mortality study, Ditraglia et al. (1981) reported no increased incidence of deaths from cancer among 1155 organochlorine pesticide manufacturing workers (31 observed vs. 37.8 expected, SMR=82). This result was not statistically significant. Workers were employed for 6 or more months and followed for 13 or more years (24,939 person-years). Workers with no exposure (for example, office workers) were included in the cohort. Vital status was not known for 112 (10%) of the workers, and these workers were assumed to be alive; therefore, additional deaths may have occurred but were not observed. Exposure was not quantified and workers were also exposed to other chemicals and pesticides (including endrin). An increased incidence of deaths from cancer was seen at several specific sites: esophagus (2 deaths observed, SMR=235), rectum (3, SMR=242); liver (2, SMR=225), and lymphatic and hematopoietic system (6, SMR=147); but these site-specific incidences were not statistically significant.

___II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Davis and Fitzhugh (1962) fed a group of 215 male and female C3HeB/Fe mice a dietary mixture containing 10 ppm aldrin for up to 2 years. The control group consisted of 217 mice. The aldrin-treated mice died 2 months earlier than controls. Intercurrent disease, pneumonia, and intestinal parasitism may have influenced the long-term survival rate. A statistically significant increase of hepatomas was reported in the treated animals as compared with controls. An independent reevaluation of the liver lesions showed most of the hepatomas to be liver carcinomas (Epstein, 1975). In a follow-up study, Davis (1965) administered aldrin at 0 or 10 ppm in the diet to 100 male and 100 female C3H mice for 2 years. The incidence of hepatic hyperplasia and benign hepatomas in the aldrin group was approximately double that of controls, whereas the number of hepatic carcinomas was about the same. Neither study provided a detailed pathologic examination or data separated by

sex.

Aldrin (95% pure) was administered in the diet to 50 male and 50 female B6C3FI mice at TWA doses of 4 and 8 ppm or 3 and 6 ppm. Treatment was for 80 weeks, and animals were observed for an additional 10 to 13 weeks (NCI, 1978). In male mice, there was a significant dose-related increase in hepatocellular carcinomas when compared with matched or pooled controls.

Treon and Cleveland (1955) administered aldrin in the diet to 40 Carworth rats/sex at concentrations of 2.5, 12.5, or 25 ppm for a period of 2 years. Forty animals/sex served as controls. Mortality of the treated rats was greater than controls, with 50% surviving in the 2.5 and 12.5 ppm groups and 40% surviving in the 25 ppm group at the end of the experiment. Cleveland (1966) reported that no apparent treatment-related tumors were present in the above study. Deichmann et al. (1970) fed 50 male and 50 female Osborne-Mendel rats aldrin (95% pure) at final concentrations of 20, 30, or 50 ppm for 31 months. Controls consisted of 100 rats/sex. There was no evidence of carcinogenic response in male or female rats fed aldrin. The NCI (1978) fed 50 Osborne-Mendel rats/sex aldrin (95% pure) at 30 or 60 ppm. Male rats were treated 111 to 113 weeks and followed for 37 to 38 weeks of observation, and female rats were treated for 80 weeks and followed for 32 to 33 weeks of observation. Aldrin produced no significant effect on the mortality of the rats of either sex. The tumors observed awere randomly distributed, with no apparent relationship to aldrin treatment. Four additional bioassays observed no carcinogenic effect of aldrin in rats, but were considered inadequate for carcinogenicity assessment.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Aldrin causes chromosomal aberrations in mouse, rat, and human cells (Georgian, 1974) and unscheduled DNA synthesis in rats (Probst et al., 1981) and humans (Rocchi et al., 1980) cells. Aldrin does not cause reverse mutations in S. typhimurium, E. coli, or S. marcesans, or mitotic gene conversion in S. cerevisiae (Fahrig, 1974).

Five compounds structurally related to aldrin--dieldrin, chlordane, heptachlor, heptachlor epoxide, and chlorendic acid--have induced malignant liver tumors in mice. Chlorendic acid has also induced liver tumors in rats.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

___II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 1.7E+1 per (mg/kg)/day

Drinking Water Unit Risk -- 4.9E-4 per (ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-1 ug/L
E-5 (1 in 100,000)	2E-2 ug/L
E-6 (1 in 1,000,000)	2E-3 ug/L

___II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- liver carcinoma
Test Animals -- mouse/C3H (Davis); mouse/B6C3F1, male (NCI)
Route -- diet
Reference -- Davis, 1965 (see table); NCI, 1978

Administered Dose (ppm)	Human Equivalent Dose (mg/kg-day)	Tumor Incidence	Reference
females			
0	0	2/53	Davis, 1965 reevaluated by Reuber (cited in Epstein, 1975)
10	0.104	72/85	
males			
0	0	22/73	
10	0.104	75/91	
0	0	3/20	NCI, 1978
4	0.04	16/49	
8	0.08	25/45	

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

Body weights for mice were assumed to be 0.03 kg for purposes of dose conversion. The above data sets were used for calculation of the following slope factors: 2.3E+1 per (mg/kg)/day for female C3H mice, 1.8E+1 per (mg/kg)/day for male C3H mice, and 1.2E+1 per (mg/kg)/day for male B6C3F1 mice. No strain or sex specificity was noted in the studies, since aldrin treatment induced liver tumors in all mouse strains tested. A geometric mean of 1.7E+1 per (mg/kg)/day was thus chosen for the quantitative estimate, since all three slope factors were very similar.

The unit risk should not be used if the water concentration exceeds 20 ug/L, since above this concentration the unit risk may not be appropriate.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

Adequate numbers of animals were treated for a large proportion of their lifetime. The route of treatment was appropriate. Slope factors calculated from three data sets from two independent assays were within a factor of 2. A slope factor for dieldrin, a major metabolite of aldrin, was determined to be 1.6E+1, essentially identical to that of aldrin.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk -- 4.9E-3 per (ug/cu.m)

Extrapolation Method -- Linearized multistage procedure, extra risk

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

II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSUR

The unit risk was calculated from the oral data presented in II.B.2.

II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE

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.

II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOS

See II.B.4.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1986

The values in the 1986 Carcinogenicity Assessment for Aldrin/Dieldrin have been reviewed by the Carcinogen Assessment Group.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 03/22/1987

Verification Date -- 03/22/1987

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Aldrin
CASRN -- 309-00-2
Last Revised -- 09/01/1989

__VI.A. ORAL RfD REFERENCES

Fitzhugh, O.G., A.A. Nelson, and M.L. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. Food Cosmet. Toxicol. 2: 551-562.

U.S. EPA. 1982. Toxicity-Based Protective Ambient Water Levels for Various Carcinogens. Environmental Criteria and Assessment Office, Cincinnati, OH. ECAO-CIN-431. Internal review draft.

__VI.B. INHALATION RfD REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Cleveland, F.P. 1966. A summary of work on aldrin and dieldrin toxicity at the Kettering Laboratory. Arch. Environ. Health. 13: 195.

Davis, K.J. 1965. Pathology report on mice fed dieldrin, aldrin, heptachlor, or heptachlor epoxide for two years. Internal FDA memorandum to Dr. A.J. Lehrman, July 19.

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Van Raalte, H.G.S. 1977. Human experience with dieldrin in perspective. Ecotoxicol. Environ. Safety. 1: 203-210.

VII. REVISION HISTORY

Substance Name -- Aldrin
CASRN -- 309-00-2

Date	Section	Description
09/30/1987	II.	Carcinogenicity section added
03/01/1988	II.B.4.	Confidence statement revised
12/01/1988	II.B.4.	Corrected slope factor in text
09/01/1989	II.A.2.	Ditraglia reference changed to Ditraglia et al.
09/01/1989	II.A.3.	Deichmann reference changed to Deichmann et al.
09/01/1989	II.B.3.	Body weight for mice corrected to kg
09/01/1989	VI.	Bibliography on-line
01/01/1991	II.	Text edited
01/01/1991	II.C.1.	Inhalation slope factor removed (global change)
01/01/1992	I.A.7.	Secondary contact changed
01/01/1992	IV.	Regulatory actions updated
07/01/1993	II.D.3.	Secondary contact's phone number changed

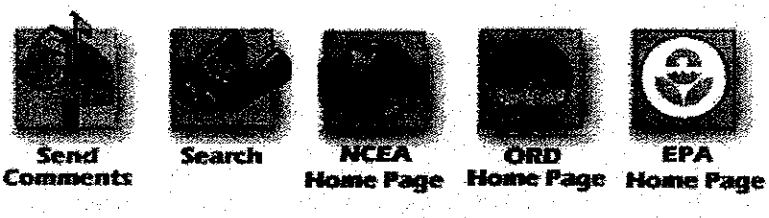
VIII. SYNONYMS

Substance Name -- Aldrin
CASRN -- 309-00-2
Last Revised -- 03/31/1987

309-00-2
Aldrex
Aldrin
Aldrite
Aldrosol
1,4:5,8-Dimethanonaphthalene, 1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-, (1 alpha, 4 alpha, 4a beta, 5 alpha, 8 alpha, 8a beta)-
1,4:5,8-Dimethanonaphthalene, 1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-
Drinox
ENT 15,949
1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-1,4,5,8-Dimethanonaphthalene
1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-1,4-endo-exo-5,8-Dimethanonaphthalene
1,2,3,4,10,10-Hexachloro-1,4,4a,5,8,8a-Hexahydro-exo-1,4-endo-5,8-Dimethanonaphthalene
Hexachlorohexahydro-endo-exo-Dimethanonaphthalene
HHDN
NCI-C00044
Octalene
Seedrin

IRIS Home Page Substance File List

00088322



Last updated: 5 May 1998
URL: http://www.epa.gov/iris/subst/0130.htm

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Benzene

CASRN 71-43-2
(10/16/1998)

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

Note: A Benzene Support Document is available for this chemical in Adobe* PDF format (57 Pages, 285 Kbytes).
Similar documents can be found in the List of Available IRIS Toxicological Reviews.
A Response to the Peer-Review is also available.

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Benzene; CASRN 71-43-2 (10/16/1998)

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Benzene
File First On-Line 03/01/1988

Category (section)	Status	Last Revised

00088324

Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	10/16/1998

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Benzene
CASRN -- 71-43-2

Not available at this time.

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Benzene
CASRN -- 71-43-2
Not available at this time.

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Benzene
CASRN -- 71-43-2
Last Revised -- 10/16/1998

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

II.A. EVIDENCE FOR HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CHARACTERIZATION

Benzene is classified as a "known" human carcinogen (Category A) under the Risk Assessment Guidelines of 1986. Under the proposed revised Carcinogen Risk Assessment Guidelines (U.S. EPA, 1996), benzene is characterized as a known human carcinogen for all routes of exposure based upon convincing human evidence as well as supporting evidence from animal studies. (U.S. EPA, 1979, 1985, 1998; ATSDR, 1997).

Epidemiologic studies and case studies provide clear evidence of a causal association between exposure to benzene and acute nonlymphocytic leukemia (ANLL) and also suggest evidence for chronic nonlymphocytic leukemia (CNLL) and chronic lymphocytic leukemia (CLL). Other neoplastic conditions that are associated with an increased risk in humans are hematologic neoplasms, blood disorders such as preleukemia and aplastic anemia, Hodgkin's lymphoma, and myelodysplastic syndrome (MDS). These human data are supported by animal studies. The experimental anima data add to the argument that exposure to benzene increases the risk of cancer in multiple species at multiple organ sites (hematopoietic, oral and nasal, liver, forestomach, preputial gland, lung, ovary, and mammary gland). It is likely that these responses are due to interactions of the metabolites of benzene with DNA (Ross, 1996; Latriano et al., 1986). Recent evidence supports the viewpoint that there are likely multiple mechanistic pathways leading to cancer and, in particular, to leukemogenesis from exposure to benzene (Smith, 1996).

II.A.2. HUMAN CARCINOGENICITY DATA

Benzene is a known human carcinogen based upon evidence presented in numerous occupational epidemiological studies. Significantly increased risks of leukemia, chiefly acute myelogenous leukemia (AML), have been reported in benzene-exposed workers in the chemical industry, shoemaking, and oil refineries.

The following epidemiologic studies briefly described are the key studies that support the weight-of-evidence classification that exposure to benzene is causally related to an increase in the risk of cancer, specifically leukemia.

Aksoy et al. (1974) reported effects of benzene exposure among 28,500 Turkish workers employed in the shoe industry. The mean duration of employment was 9.7 years (range 1 to 15 years) and the mean age was 34.2 years. Peak exposure to benzene was reported to be 210 to 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 analysis of the study (Aksoy, 1980) reported eight additional cases of leukemia as well as evidence suggestive of increases in other malignancies. This case study lacks detailed information on personal exposure to benzene and potential exposure to other chemicals, a well-defined comparison population, and control of confounding variables.

Infante et al. (1977b), in a retrospective cohort mortality study, examin the leukemogenic effects of benzene exposure in 748 white male workers exposed at least 1 day while employed in the manufacture of rubber products. Exposure occurred from 1940 to 1949 and vital status was obtained through 1975. A statistically significant increased risk of leukemia (7 observed, 1.48 expected; $p < .002$) was found by comparison of observed leukemia deaths in this cohort with those expected based upon general U.S. population death rates.

The risk of leukemia was said by the authors to be potentially understated since follow-up was only 75% complete. According to the authors, there was no evidence of solvent exposure other than benzene. No effort was made to evaluate individual exposures to benzene for the purpose of doing a dose-response analysis. The main criticism of this study, as well as its later updates, is the small size of the cohort.

In an extension and elaboration of the analysis done by Infante et al. (1977b), Rinsky et al. (1981) reported seven deaths from leukemia in this same cohort after achieving a 98% vital status ascertainment through June 1975. Forty additional deaths from all causes were reported, but no new leukemia deaths. Again, the risk of death from leukemia was statistically significant (standardized mortality ratio [SMR] was 560 based upon 7 leukemia deaths, $p < .001$). Some 437 members of the cohort were exposed for less than 1 year. Those who received 5 or more years of exposure exhibited an SMR of 2100, based upon 5 leukemia deaths versus 0.25 expected ($p < .01$). All seven leukemia cases were of the myelogenous or monocytic cell type. Four additional deaths from leukemia were also noted but could not be added to the total because they did not fit the criteria for inclusion. The authors tried to reconstruct past exposure to benzene at the two locations of this company and found that in some areas of the plants airborne benzene concentrations occasionally rose to several hundred parts per million, but most often employee 8-hour time-weighted averages (TWA) fell within the limits considered permissible at the time of exposure. No dose-response analysis was attempted.

In an updated version of the Rinsky et al. (1981) study, the same authors examined a somewhat expanded cohort of 1165 nonsalaried white men employed in the rubber hydrochloride department for at least 1 day through December 1965 and followed to December 31, 1981 (Rinsky et al., 1987). Followup was 98.6% complete Again, a statistically significant excess risk of leukemia was found for the tota cohort (9 observed, 2.7 expected; $p < 0.05$). For the first time, individual measurements of cumulative exposure in terms of ppm-years were generated for all members of the cohort utilizing the historical air-sampling data discussed above or interpolating estimates based on the existing data. SMRs for leukemia ranged from a nonsignificant 109 (2 observed, 1.83 expected) at cumulative exposures under 40 ppm-years to a statistically significant SMR of 2339 (5 observed, 0.21 expected; $p < .05$) at 200 ppm-years or more of exposure. The authors found significantly elevated risks of leukemia at cumulative exposures less than the then equivalent current standard for occupational exposure, which was 10 ppm over a 40-year working lifetime.

The Rinsky et al. (1981, 1987) study analyses, based upon the original cohort of Pliofilm rubber workers studied by Infante et al. (1977b), were selecte by the Agency as the critical study for dose-response analysis and for the quantitative estimation of cancer risk to humans. The Rinsky et al. (1981, 1987) analyses show ample power, latency, reasonably good estimates of exposure to benzene except prior to 1946, few confounders, and a wide range of exposure to benzene from low levels to high levels. Limitations include the small cohort size, reporting only nine leukemia deaths with no estimates of risk according to cell type. There remain questions about the estimation of personal exposure to benzene, especially prior to 1946 when no measurements of airborne benzene were made. And finally, at levels less than 200 ppm-years it is not possible to determine leukemia risk in this cohort because of lack of sensitivity of the data at low levels.

Ott et al. (1978) observed a nonsignificantly increased risk of leukemia (3 deaths) among 594 chemical workers exposed to benzene followed for at least 23 years in a retrospective cohort mortality study. Benzene exposures ranged from under 2 ppm to over 25 ppm 8-hour TWA. Bond et al. (1986) updated this report by following this cohort an additional 9 years to the end of 1982 and adding an additional 362 exposed workers not studied previously. The authors reported finding a nonsignificant excess risk (SMR = 194) of deaths from leukemia based upon 4 cases. All were diagnosed as myelogenous leukemias. The authors reported that this represented a significant excess (4 observed versus 0.9

expected, $p < .011$) for myelogenous leukemia based upon the International Classification of Diseases and Causes of Death. It is not stated whether these four deaths were acute or chronic. One additional death was classified as a "pneumonia" death, but on the death certificate "acute myelogenous leukemia" was noted as a significant contributing condition. Cumulative exposure estimates ranged from 18 ppm-months to a high of 4211 ppm-months. The Bond et al. (1986) study has little power to detect significant risk of leukemia at low doses. The authors also state that their data should not be used for determining unit risk estimates because of the small number of events, competing exposures to other potentially hazardous materials, and the contribution of unquantified brief exposures to benzene.

Wong (1983, 1987) reported on the mortality of male chemical workers who had been exposed to benzene for at least 6 months during the years 1946 to 1975. The study population of 4602 persons was drawn from seven chemical plants and cumulative exposures to benzene were determined for all subjects. The control subjects (3074 persons) held jobs at the same plants for at least 6 months but were never subjected to benzene exposure. Dose-dependent increases were seen in the risk of leukemia and the risk of lymphatic and hematopoietic cancer. Chemical workers with a cumulative exposure to benzene of 720 ppm-months were subject to a borderline significant relative risk of 3.93 ($p = .05$) for lymphatic and hematopoietic cancer. None of the leukemia deaths were of the acute myeloid cell type, the type that was known to be associated with exposure to benzene in other studies. The author further observed that cumulative exposure, not peak exposure, was the major variable in quantifying mortality risk from lymphopoietic cancer. The Mantel-Haenszel chi-square for upward trend in risk of leukemia with increasing cumulative exposure was significantly elevated at the 99% level of confidence. Some of the limitations of this study include imprecise historical industrial hygiene data, unusual distribution of leukemia cell types, i.e., there were no acute cases of myelogenous leukemia out of seven leukemia cases, and possible exposure of comparison subjects to potentially carcinogenic solvents other than benzene.

The National Cancer Institute of the U.S. National Institutes of Health a the Chinese Academy of Preventative Medicine have been conducting a comprehensive epidemiological study of 74,828 benzene-exposed workers employed from 1972 to 198 in 672 factories in 12 cities of China (Dosemeci et al., 1994; Hayes et al., 1996 1997; Yin et al., 1987, 1989, 1994, 1996). A comparison group of workers consisting of 35,805 employees was assembled from non-benzene-exposed units of 69 of the same factories and 40 factories elsewhere. Workers in a variety of jobs i painting, printing, footwear, rubber, chemical, and other industries were followe for vital status for an average period of time of less than 12 years. Less than 0.3% were lost to follow-up. Employee work histories were linked to benzene exposure data in order to derive individual time-specific estimates for each work (Dosemeci et al., 1994). This large cohort mortality study produced a significan elevated risk of hematologic neoplasms (RR = 2.2, 95% C.I. = 1.1-4.2) in workers exposed to benzene at an average level of less than 10 ppm. A combination of ANL and MDS produced a risk of 3.2 (95% C.I .= 1.0-10.1). For exposure to a sustaine concentration of 25 ppm benzene, the risk of ANLL and MDS increased to 7.1 (95% C.I. = 2.1-23.7). The risk of other leukemias (other than ANLL), including chronic myeloid and monocytic leukemia, was not significantly elevated (RR = 2.0) Additionally, the risk of non-Hodgkin's lymphoma was significantly elevated (RR = 4.2 with 95% C.I. = 1.1-15.9) for those with a sustained exposure to benzen that occurred at least 10 years prior to diagnosis. The authors concluded that benzene exposure "is associated with a spectrum of hematologic neoplasms and related disorders in humans and that risks for these conditions are elevated at average benzene-exposure levels of less than 10 ppm." Limitations of this study include possible concurrent exposures to many different chemicals found in the factories where the benzene exposure occurred. There is a lack of reliable exposure information in the early days of the observation period, when only 3% of the exposure estimates were based on actual measurements.

All of the epidemiological studies referred to above have some methodological problems, i.e., confounding exposures, lack of sufficient power,

and other limitations, but the consistent excess risk of leukemia across all of these studies argues that such problems could not be entirely responsible for the elevated risks of cancer. Most of these epidemiologic and case studies have been reviewed in peer-reviewed publications (IARC, 1982; ATSDR, 1997; U.S. EPA, 1998). They provide clear evidence of a causal association between exposure to benzene and ANLL. The evidence is suggestive with respect to CNLL and CLL.

The limitations of these studies, except for Rinsky et al. (1981, 1987), preclude their use in quantitative risk estimation. This is further discussed in the quantitative risk estimation sections (II.C.3 and II.C.4).

II.A.3. ANIMAL CARCINOGENICITY DATA

Although human epidemiological studies provide the bulk of the evidence reaffirming the classification of benzene as a category A, "known" human carcinogen (U.S. EPA, 1979, 1985, 1998), many experimental animal studies, both inhalation and oral, also support the evidence that exposure to benzene increases the risk of cancer in multiple organ systems including the hematopoietic system, oral and nasal cavities, liver, forestomach, preputial gland, lung, ovary, and mammary gland. The key animal studies that support the finding of an excess risk of leukemia in humans from exposure to benzene by the inhalation route are Maltoni et al. (1982, 1983, 1985, 1989), Cronkite et al. (1984, 1985, 1989), Snyder et al. (1988), and Farris et al. (1993); and by the oral route, Huff et al. (1989), NTP (1986), and Maltoni et al. (1983, 1985, 1989). The details of these studies have been reviewed (ATSDR, 1997). Studies on the carcinogenicity of benzene in rodents include inhalation exposures to Sprague-Dawley rats, C57BL/6 mice, AKR mice, CD-1 mice, and CBA mice; and gavage treatment of Sprague-Dawley rats, Wistar rats, F344 rats, RF/J mice, Swiss mice, and B6C3F1 mice (Cronkite et al., 1989; Goldstein et al., 1982; Huff et al., 1989; Maltoni et al., 1983, 1988; NTP, 1986; Snyder et al., 1980, 1982, 1984; Farris et al., 1993). Inhalation concentrations ranged from 0 to 1000 ppm and gavage doses ranged from 0 to 200 mg/kg.

It is noted that in humans the cancer induced by benzene exposure is predominantly acute nonlymphocytic leukemia, while in rodents lymphocytic leukemia was observed in two series of experiments in C57BL/6 mice (Snyder et al., 1980) and CBA/Ca mice (Cronkite et al., 1989). The difference in induction of hematopoietic cancers in mice and humans is not fully understood, but it may be related to species-specific differences in hematopoiesis. Lymphocytes make up a larger portion of the nucleated cells in mouse bone marrow than in human bone marrow (Parmley, 1988) and could simply represent a larger target cell population for benzene metabolites. The bone marrow, Zymbal gland, and Harderian gland all contain peroxidases, which can activate phenols to toxic quinones and free radicals. Sulfatases, which remove conjugated sulfate and thus reform free phenols, are also present at high levels in these target organs. The selective distribution of these two types of enzymes in the body may explain the accumulation of free phenol, hydroquinone, and catechol in the bone marrow and the resulting differences in target organ toxicity of benzene metabolites in humans and animals. The animal bioassay results may have some relevance to human leukemia, but it should be emphasized that there is no well-demonstrated and reproducible animal model for leukemia resulting from benzene exposure. The mechanism of leukemia development following exposure to benzene is not well understood (Low et al., 1989, 1995).

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

The supporting evidence for the carcinogenic effects of exposure

to benzene comes from our current understanding of the metabolism and mode of action (Stephens et al., 1994; Medinsky et al., 1996; Lee et al., 1996; Valentine et al., 1996; Rothman, 1997). This is briefly summarized below and reviewed in U.S. EPA (1998).

It is generally agreed that the toxicity of inhaled benzene results from its biotransformation to reactive species. Benzene is metabolized in the liver by cytochrome P4502E1 (CYP2E1) to its major metabolites: phenol, hydroquinone, and catechol. The intermediate benzene oxide can also undergo ring opening to trans-trans muconic acid. Although there is a scientific consensus that metabolism of benzene is required for resultant toxicity and carcinogenic response, the role of a metabolite or metabolites of benzene in producing these adverse effects is controversial and more research data are needed to better define sequelae of pathogenesis following exposure to benzene and its metabolites. Current evidence indicates that benzene-induced myelotoxicity and genotoxicity result from a synergistic combination of phenol with hydroquinone, muconaldehyde, or catechol.

Molecular targets for the action of these metabolites, whether acting alone or in concert, include tubulin, histone proteins, topoisomerase II, and other DNA-associated proteins. Damage to these proteins would potentially cause DNA strand breakage, mitotic recombination, chromosomal translocations, and malsegregation of chromosomes to produce aneuploidy. If these effects took place in stem or early progenitor cells, a leukemic clone with selective advantage to grow could arise as a result of protooncogene activation, gene fusion, and suppressor-gene inactivation. Epigenetic effects of benzene metabolites on the bone marrow stroma, and perhaps the stem cells themselves, could then foster development and survival of a leukemic clone. Since these plausible events have not been conclusively demonstrated, this remains a hypothesis (Smith, 1996).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

THIS SECTION IS BEING CURRENTLY REVIEWED FOR THE APPROPRIATENESS OF THE RECENT DATA/INFORMATION TO REVISE THE QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE TO BENZENE. THEREFORE, THIS SECTION II.B REMAINS UNCHANGED AT THIS TIME.

___II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 2.9E-2 per (mg/kg)/day

Drinking Water Unit Risk -- 8.3E-7 per (ug/L)

Extrapolation Method -- One-hit (pooled data)

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

___II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

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.

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

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.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

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.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

___II.C.1. SUMMARY OF RISK ESTIMATES

___II.C.1.1. Air Unit Risk:

A range of 2.2×10^{-6} to 7.8×10^{-6} is the increase in the lifetime risk of an individual who is exposed for a lifetime to 1 $\mu g/m^3$ benzene in air.

___II.C.1.2. Extrapolation Method:

Low-dose linearity utilizing maximum likelihood estimates (Crump, 1992, 1994).

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	13.0 to 45.0 $\mu g/m^3$
E-5 (1 in 100,000)	1.3 to 4.5 $\mu g/m^3$
E-6 (1 in 1,000,000)	0.13 to 0.45 $\mu g/m^3$

___II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSUR

Tumor Type -- Leukemia
Test Species -- Humans
Route -- Inhalation
References -- Rinsky et al., 1981, 1987; Paustenbach et al., 1993; Crump and Allen, 1984; Crump, 1992, 1994; U.S. EPA, 1998.

___II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE

The Pliofilm workers of Rinsky et al. (1981, 1987) provide the best published set of data to date for evaluating human cancer risks from exposure to benzene. Compared to the published studies of Ott et al. (1978), Bond et al. (1986), and Wong (1987), this cohort has fewer reported co-exposures to other potentially carcinogenic substances in the workplace that might confound risk analysis for benzene. This cohort also provides a greater range of exposures than those of Ott et al. (1978), Bond et al. (1986), and Wong (1987). The Rinsky et al. data were used for developing the unit cancer risk by Crump (1992, 1994). Differences in the unit risk estimates, in addition to the choice of model used, stem largely from differences in the exposure estimates and the dose-response model used.

Although the ongoing Chinese cohort studies (Dosemeci et al., 1994; Hayes et al., 1996, 1997; Yin et al., 1987, 1989, 1994, 1996) provide a large data set and perhaps may provide information in the future to better characterize risk of cancer at low dose exposure, their use in the derivation of risk estimates remains problematic at present for the reasons cited in Section II.A.2.

The two most important determinants of the magnitude of the unit risk number are the choice of extrapolation model to be used to estimate risk at environmental levels of exposure and the choice of the exposure estimates to which the Pliofilm workers (Rinsky et al., 1981, 1987) were subjected. Crump (1992, 1994) presented 96 unit risk calculation analyses by considering different combinations of the following factors: (1) different disease endpoints, (2) additive or multiplicative models, (3) linear/nonlinear exposure-response relationships, (4) two different sets of exposure measurements (Crump and Allen [1984] vs. exposure estimates by Paustenbach et al. [1993]) and (5) cumulative or weighted exposure measurements. The unit risk estimates range from 8.6×10^{-5} to 2.5×10^{-2} at 1 ppm (3200 $\mu g/m^3$) of benzene air concentration (Crump, 1992, 1994).

The risk estimates would fall into the lower range if a sublinear exposur response model were found to be more plausible. However, the shape of the exposure dose-response curve cannot be considered without a better understanding of the biological mechanism(s) of benzene-induced leukemia. Understanding of the mechanisms by which exposure to benzene and its metabolites exert their

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toxic and carcinogenic effects remains uncertain (U.S. EPA, 1998). It is likely that more than one mechanistic pathway may be responsible for the toxicity of benzene contributing to the leukemogenic process. Not enough is known to determine the shape of the dose-response curve at environmental levels of exposure and to provide a sound scientific basis to choose any particular extrapolation model to estimate human cancer risk at low doses. In fact, recent data (Hayes et al., 1997) suggest that because genetic abnormalities appear at low exposures in humans, and the formation of toxic metabolites plateaus above 25 ppm, the dose-response curve could be supralinear below 25 ppm. Given this, EPA believes that use of a linear extrapolation model as a default approach is appropriate.

When a linear model was employed, the choice of cancer unit risk estimate narrows to a range between 7.1×10^{-3} and 2.5×10^{-2} at 1 ppm (2.2×10^{-6} to 7.8×10^{-6} at 1 $\mu g/m^3$ of benzene in air), depending on which exposure measurements were used, i.e., Crump and Allen (1984) or Paustenbach et al. (1993). The choice of these limits was dictated by the following considerations: (1) use of the (1981, 1987) Rinsky cohort, (2) use of Crump's (1992, 1994) analysis of the Crump and Allen (1984) and the Paustenbach (1992, 1993) exposure measurements. The range of risks nearly includes the 1985 EPA risk estimate of 2.6×10^{-2} at 1 ppm (8.1×10^{-6} at 1 $\mu g/m^3$). The set of risk estimates falling within this interval reflects both the inherent uncertainties in the risk assessment of benzene and the limitations of the epidemiologic studie in determining dose-response and exposure data.

II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

The major conclusion of this update (U.S. EPA, 1998) is a reaffirmation within an order of magnitude of the benzene interim unit risk estimates derived in EPA's 1985 interim risk assessment (U.S. EPA, 1985), which established the probability of humans developing cancer from exposure to 1 ppm of benzene. Review of the 1985 interim risk assessment required addressing two main concerns.

The first concern was the use of the updated epidemiologic data from Rins et al.'s (1987) cohort of Pliofilm workers and selection of appropriate estimates of their exposure to benzene for the derivation of the unit risk estimate. Although numerous epidemiological studies demonstrate an association of exposure to benzene and increased risk of human cancer, these studies are not without methodological limitations. The Rinsky et al. (1981, 1987) study continues to provide the best available data for derivation of unit cancer risk estimates. This study had the least number of confounders and a wide range of exposure to benzene.

The second major concern was continued application of the low-dose linear concept to the model used to generate estimates of unit risk. It was concluded that at present there is insufficient evidence to reject this concept, and a linear extrapolation was used (U.S. EPA, 1998). If one assumes that the linear extrapolation model is the appropriate model to be used, given the uncertainties outlined, then the range of suitable estimates is defined by the choice of exposure estimates selected. The lowest unit risk among linear choices is determined by the exposure estimates of Paustenbach et al. (1993) according to th calculations of Crump (1992, 1994), simply because Paustenbach's exposure estimates for the Rinsky cohort are highest. That estimate is 7.1×10 at 1 ppm (2.2×10^{-6} at 1 $\mu g/m^3$). The highest risk number is determined by Crump (1992, 1994) using the lower exposure estimates from Crump and Allen (1984), and that is 2.5×10^{-2} at 1 ppm (7.8×10^{-6} at 1 $\mu g/m^3$).

At present, the true cancer risk from exposure to benzene cannot be ascertained, even though dose-response data are used in the quantitative cancer risk analysis, because of uncertainties in the low-dose exposure scenarios and lack of clear understanding of the mode of action. A range of estimates of risk is recommended, each having equal scientific plausibility. The range estimates

are maximum likelihood values (i.e., best statistical estimates) and were derived from observable dose responses using a linear extrapolation model to estimate low environmental exposure risks. The extrapolation range is on the order of 20-60 depending on what environmental level is of interest. This range is fairly low and thus does not suggest any unusual lack of plausibility about the estimates. The use of a linear model is a default public health protective approach and an argument both for and against recognizing supra- and sublinear relationships at low doses and nonthreshold or threshold modes of action on exposure to benzene. Therefore, the true risk could be either higher or lower. The numerical difference between the 1985 risk estimate (2.6×10^{-2} at 1 ppm or 8.1×10^{-6} at 1 $\mu g/m^3$) compared to the new high-end risk (2.5×10^{-2} at 1 ppm or 7.8×10^{-6} at 1 $\mu g/m^3$) is insignificant and no scientific inferences about the merit of one value versus the other should be made.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

II.D.1. EPA DOCUMENTATION

Source Documents -- U.S. EPA, 1998; U.S. EPA, 1985; U.S. EPA, 1979.

The EPA (1998) assessment on which this summary was based was externally peer reviewed by a panel of experts meeting held on July 16, 1997. Their comments have been evaluated carefully and incorporated in finalization of this IRIS summary. A summary record of the comments and EPA responses is included as an appendix to the benzene support document file.

The EPA 1979 and 1985 documents provide the basis for the classification of benzene as a Group A carcinogen.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Consensus Date -- 09/30/1998

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513) 569-7254 (phone), (513) 569-7159 (fax), or RIH.IRIS@EPAMAIL.EPA.GOV (Internet address).

VI. BIBLIOGRAPHY

Benzene
CASRN -- 71-43-2
Last Revised -- 10/16/1998

VI.A. ORAL RfD REFERENCES

N.A.

VI.B. INHALATION RfC REFERENCES

N.A.

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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00088336

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_VII. REVISION HISTORY

Benzene
CASRN -- 71-43-2

Date	Section	Description
12/01/1988	II.A.4.	Anderson and Richardson citation year correct
12/01/1988	II.A.4.	Kissling and Speck citation year corrected
07/01/1989	I.B.	Inhalation RfD now under review
02/01/1990	II.	Clarified citations
02/01/1990	II.A.3.	Corrected Maltoni, 1979 to Maltoni and Scarnato, 1979
02/01/1990	II.A.3.	Corrected Maltoni, 1983 to Maltoni et al., 19
02/01/1990	II.A.3.	Corrected Synder et al., 1980 to 1981
02/01/1990	VI.	Bibliography on-line

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03/01/1990	VI.C.	Clarify Maltoni et al., 1983 and NTP, 1986 references
08/01/1990	III.A.10	Primary contact changed
08/01/1990	IV.F.1.	EPA contact changed
01/01/1991	II.	Text edited
01/01/1991	II.C.1.	Inhalation slope factor removed (global chang
01/01/1992	IV.	Regulatory actions updated
04/01/1992	II.B.2.	Text revised
02/01/1994	II.D.3.	Secondary contact's phone number changed
10/16/1998	II., VI.	Revised inhalation carcinogenicity section and references

VIII. SYNONYMS

Benzene
CASRN -- 71-43-2
Last Revised -- 03/01/1988

71-43-2
Benzene
benzol
coal naphtha
cyclohexatriene
phene
phenyl hydride
polystream
pyrobenzol

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Last updated: **14 October 1998**
URL: http://www.epa.gov/iris/subst/0276.htm

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Bromodichloromethane

CASRN 75-27-4

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0213
Bromodichloromethane; CASRN 75-27-4

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Bromodichloromethane

File On-Line 09/30/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	03/01/1991
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	03/01/1993

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I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Bromodichloromethane
CASRN -- 75-27-4
Last Revised -- 03/01/1991

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Renal cytomegaly	NOAEL: None	1000	1	2E-2 mg/kg/day
Chronic Mouse Gavage Bioassay	LOAEL: 17.9 mg/kg/day			
NTP, 1986				

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

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

NTP (National Toxicology Program). 1986. Toxicology and Carcinogenesis Studies of Bromodichloromethane in F344/N Rats and B6C3F1 Mice (gavage studies). NTP Technical Report, Ser. No. 321, NIH Publ. No. 87-2537.

Bromodichloromethane (BDCM) was administered in corn oil by gavage, 5 days/week for 102 weeks, to groups of 50 male and 50 female F344/N rats at doses of 0, 50, or 100 mg/kg/day; to groups of 50 male B6C3F1 mice at doses of 0, 25, or 50 mg/kg/day; and to groups of 50 B6C3F1 female mice at doses of 0, 75, or 150 mg/kg/day. Final survival of dosed female mice was reduced compared with controls. Final mean body weights of dosed female mice and high-dose male and female rats were 75 to 91% that of vehicle controls.

Compound-related nonneoplastic lesions included cytomegaly and tubular cell hyperplasia of the kidney and fatty metamorphosis of the liver in male rats; eosinophilic cytoplasmic change, clear cell change, focal cellular change, and fatty metamorphosis of the liver and tubular cell hyperplasia of the kidney in female rats; fatty metamorphosis of the liver, renal cytomegaly, and follicular cell hyperplasia of the thyroid gland in male mice; and follicular cell hyperplasia of the thyroid gland in female mice. A LOAEL of 17.9 mg/kg/day (25 mg/kg/day x 5 days/7 days) based on renal cytomegaly in male mice, an effect considered minimal in the absence of data demonstrating renal functional impairment, is indicated by these results.

In a subchronic bioassay conducted by NTP (1986), male and female rats received doses of 19 to 300 mg/kg/day, male mice received doses of 6.25 to 100 mg/kg/day, and female mice received doses of 25 to 400 mg/kg/day for 5 days/week. Five of 10 male rats and 2/10 female rats at 300 mg/kg/day died. Final body weights of male and female rats in the 150 or 300 mg/kg/day treatment groups, male mice receiving 100 mg/kg/day and female mice receiving 200 or 400 mg/kg/day, were lower than those of controls. Centrilobular degeneration of the liver and degeneration and necrosis of the kidney were seen in high-dose male rats; liver lesions were observed in high-dose female rats and in female mice at 200 or 400 mg/kg/day, and kidney lesions were seen in male mice at 100 mg/kg/day. These data define a NOAEL of 35.7 mg/kg/day (50 mg/kg/day x 5 days/7 days), a dose above which produced kidney lesions and depressed body weight in male mice. Because the chronic study used more animals/dose, was of longer duration, and presented more complete data, more confidence is placed in the chronic LOAEL than in the subchronic NOAEL.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- A factor of 100 was employed for extrapolation from animal data and for protection of sensitive human subpopulations. An additional factor of 10 was used because the RfD was based on a LOAEL (although minimally adverse), and to account for data base deficiencies (no reproductive studies).

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

A study by Chu et al. (1982) who administered bromodichloromethane in the drinking water to rats for 90 days reported a no-effect level of 0.45 mg/kg/day. This study, however, is not considered suitable for derivation of the RfD because of difficulties in interpretation of study design and statistical methodology.

There are no published data on teratogenicity or reproductive effects of trihalomethanes.

___I.A.5. CONFIDENCE IN THE ORAL RfD

Study -- Medium
Data Base -- Medium

RfD -- Medium

Confidence in the study is rated medium because although NTP (1986) incorporated both chronic and subchronic exposures in two species using sufficient numbers of animals and measured multiple endpoints, including histopathology of most organ systems, a NOEL was not determined. Although there are some discrepancies in the dose levels producing adverse effects, there are several published subchronic studies of bromodichloromethane permitting confidence in the data base to be rated medium to low. Thus, overall confidence in the RfD is rated medium to low.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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

Other EPA Documentation -- None

Agency Work Group Review -- 12/02/1985, 02/05/1986, 05/14/1986, 07/16/1987

Verification Date -- 07/16/1987

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Bromodichloromethane
CASRN -- 75-27-4

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Bromodichloromethane
CASRN -- 75-27-4
Last Revised -- 03/01/1993

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

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Based on inadequate human data and sufficient evidence of carcinogenicity in two animal species (mice and rats) as shown by increased incidence of kidney tumors and tumors of the large intestine in male and female rats, kidney tumors in male mice, and liver tumors in female mice.

___II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. There are no epidemiologic studies of bromodichloromethane alone. Bromodichloromethane is one of several trihalomethanes (including chloroform, bromoform and dibromochloromethane) that are formed from the interaction of chlorine with organic materials found in water. A large number of other byproducts are present in chlorinated water as well. Several ecologic studies (Cantor et al., 1978; Aldrich and Peoples, 1982; Isacson et al., 1983) and case-control studies (Young and Kanarek, 1983; Cantor et al., 1987) suggest a positive correlation between drinking chlorinated water and the incidence of several human cancers, particularly bladder, rectal and colon cancer. These studies have design limitations such as lack of individual exposure information, misclassification of exposure, and lack of data to control for diet, smoking or alcohol consumption. The agreement of findings in several independent studies strengthens the association between drinking chlorinated water and cancer (Cantor, 1983; Crump, 1983). However, in all studies exposure to chlorinated water resulted in intake of a mixture of compounds, including chloroform, which is considered to be a probable human carcinogen. Thus, these data are inadequate for assessing the carcinogenic potential of bromodichloromethane in humans.

II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. In a 2-year carcinogenicity study (NTP, 1987), bromodichloromethane was administered in corn oil by gavage, 5 days/week for 102 weeks, to F344/N rats (50/sex/dose) at 0, 50 or 100 mg/kg/day. Similarly, groups of 50 male B6C3F1 mice were given oral doses of 0, 25 or 50 mg/kg/day and groups of 50 female B6C3F1 mice were administered doses of 0, 75 or 150 mg/kg/day. The study using the male rats was restarted 10.5 months into the original study because a temperature elevation killed 45/50 of the vehicle control male rats. Survival was reduced 52%, 26% and 30% in the control, low-dose and high-dose females, respectively, after week 84; the mortality was associated with ovarian abscesses.

Bromodichloromethane caused compound-related statistically significant increases in tumors of the kidney in male mice, the liver in female mice, and the kidney and large intestine in male and female rats. In male mice, the incidence of tubular cell adenomas (vehicle control, 1/46; low dose, 2/49; high dose, 6/50) and the combined incidence of tubular cell adenomas and adenocarcinomas of the kidneys were significantly increased in the high-dose (50 mg/kg/day) group (1/46, 2/49 and 9/50 in the control, low-dose and high-dose groups, respectively). In female mice, significant increases of hepatocellular adenomas occurred at 75 mg/kg/day and 150 mg/kg/day while hepatocellular carcinomas were significantly increased at 150 mg/kg/day. The combined incidence of hepatocellular adenomas or carcinomas in vehicle control, low-dose and high-dose groups were 3/50, 18/48 and 29/50, respectively.

In male and female rats, the incidences of tubular cellular adenomas, adenocarcinomas, and the combined incidence of adenomas and adenocarcinomas of the kidneys were statistically significantly increased only in the high-dose (100 mg/kg/day) groups. The combined incidence of tubular cell adenomas or adenocarcinomas in vehicle control, low-dose and high-dose groups were 0/50, 1/49 and 13/50 for males, and 0/50, 1/50 and 15/50 for females, respectively. Tumors of large intestines, namely adenocarcinomas (vehicle control, 0/50; low dose, 11/49; high dose, 38/50) and adenomatous polyps (0/50, 3/49 and 33/50 in the vechicle control, low-dose and high-dose groups, respectively) were significantly increased in male rats in a dose-dependent manner. These large intestinal tumors, however, were only observed in high-dose (100 mg/kg/day) female rats (adenocarcinomas 0/46, 0/50, 6/47; adenomatous polyps 0/46, 0/50, 7/47 in the vehicle control, low-dose and high-dose groups, respectively). The combined incidence of large intestine adenocarcinomas and/or adenomatous polyps in vehicle control, low-dose and high-dose groups were 0/50, 13/49 and 45/50 for males and 0/46, 0/50 and 12/47 for females. The combined tumor incidence of large intestine and kidney in male and female rats at control, low dose and high dose were 0/50, 13/49, 46/50 and 0/46, 1/50, 24/48, respectively. Under the conditions of this bioassay, NTP concluded there was clear evidence of carcinogenicity of bromodichloromethane in male and female F344/N rats and B6C3F1 mice.

Hepatic tumor data reported in female mice should be interpreted with caution, however, because of the possible role of the corn oil vehicle in induction of these tumors. Chloroform, a closely related structural analogue, induced hepatocellular carcinoma in mice (NCI, 1979) when administered in corn oil (NCI, 1976; Roe et al., 1976), but not in drinking water (Jorgenson et al., 1985). Based primarily on the fact that the drinking water study did not replicate hepatic tumors in female mice and on the potential role of corn oil in enhancing toxicity, the NAS Subcommittee on the Health Effects of Disinfectants and Disinfection By-Products recommended that kidney tumor data obtained from Jorgenson's study be used for estimating carcinogenic risk of chloroform (NAS, 1987; U.S. EPA, 1992a,b,c, 1993).

On October 25-26, 1990, the Science Advisory Board's Drinking Water Committee held a meeting in Washington, DC to review the Office of Water's draft Drinking Water Criteria Document for Trihalomethanes (including bromodichloromethane) (1990 version). Based on the concern of the corn oil vehicle effect cited for chloroform, the Committee concluded that hepatic tumor induction by a trihalomethane administered in an oil vehicle should be utilized only in making the weight-of-evidence judgement for carcinogenicity, and these hepatic tumor data should be disregarded in making a quantitative estimation of the carcinogenic risk of a trihalomethane. Commenting on bromodichloromethane specifically, the Committee considered the use of renal or intestinal tumor incidence for carcinogenic risk calculation to be appropriate. The Committee regarded the resulting kidney tumors to be independent from the vehicle effects (U.S. EPA, 1992d). The Committee also commented that large intestinal tumors are not commonly seen in the rat, the tumor incidence was high in males, and was observed in both sexes (U.S. EPA, 1992d).

Theiss et al. (1977) tested bromodichloromethane in a short-term lung adenoma test in strain A/St male mice. Twenty mice/group were injected intraperitoneally with 0, 20, 40 or 100 mg/kg of bromodichloromethane in tricaprylin, 3 times/week for a total of 18-24 injections (total doses were 0, 360, 960 or 2400 mg/kg, respectively). There was no effect of treatment on survival. Twenty-four weeks after the first injection, the mice were sacrificed and the lungs examined for surface adenomas. The number of pulmonary tumors per mouse appeared elevated in the high-dose animals, although the increase was not statistically significant ($p=0.062$).

In a unpublished but documented 2-year study, SPF Wistar rats (40/sex/group) were fed a diet supplemented with 0.014, 0.055 or 0.22% microencapsulated bromodichloromethane (Tobe et al., 1982). Based on reported body weights (150-475 g) and food consumption (15-20 g/day), these levels correspond to doses of about 6, 24 or 130 mg/kg/day for males and 11, 41 or 220 mg/kg/day for females. Controls (70/sex) received empty microcapsules. At 6, 12 and 18 months, 9-12/sex of the controls and 5-7/sex/group of the treated rats were sacrificed. The remainder of the animals were sacrificed at 24 months. Body weight was decreased in the high-dose animals by 25% relative to controls. Mortality was not correlated with dose in either males or females. Survival at 24 months was 77, 81, 75 and 77% in females and 58, 60, 62 and 79% in males for the control, low-, mid- and high-dose groups, respectively. No gross tumors were observed at 18 or 24 months; histopathology was not reported.

Tumasonis et al. (1985) administered 1.2 mL bromodichloromethane per liter of drinking (tap) water to male and female Wistar rats for 72 weeks, after which concentrations were halved for the remainder of the lifetime of the animals (140-180 weeks). Controls were untreated. Body weight decreased in treated animals relative to controls by approximately 35-40%. The authors estimated the treated animals consumed 150 mg/kg/day (females) or 200 mg/kg/day (males). Hepatic neoplastic nodules were significantly elevated in female rats (17/53) when compared with controls (0/18). Neoplastic nodules in males and lymphosarcomas and pituitary tumors in both sexes were reported, but did not have an significantly increased incidence relative to the controls. Of the treated animals, two males and one female were noted to have renal adenoma or adenocarcinoma, while none were reported in the control group.

Voronin et al. (1987) examined the carcinogenicity of bromodichloromethane in CBA x C57Bl/6 mice. Groups of 50-55 mice/sex were treated with bromodichloromethane in drinking water at concentrations of 0.04, 4.0 or 400 mg/L (0.0076, 0.76 or 76 mg/kg/day) for 104 weeks. An untreated control group with 75 male and 50 female mice was also maintained. Total tumor incidences, based on the number of mice surviving until detection of the first tumor, were 4/63 (6%), 3/35 (8%), 1/16 (6%) and 1/18 (9%) for males, and 3/34 (9%), 1/45 (2%), 1/18 (6%) and 1/13 (8%) for females in the control, low-, mid- and high-

dose groups, respectively. The authors concluded that the results were not statistically significant by chi square analysis, and that under the conditions of this bioassay, bromodichloromethane was not carcinogenic.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Bromodichloromethane was mutagenic in Salmonella typhimurium strain TA100 in the absence of liver homogenate in a vapor phase test performed in a desiccator. When tested in a standard Salmonella/microsomal assay, however, the compound was not mutagenic (Simmon et al., 1977). Varma et al. (1988) reported that bromodichloromethane was mutagenic in Salmonella typhimurium strain TA1537 without rat liver homogenate activation. Similar results were also reported by Ishidate et al. (1982) using Salmonella typhimurium strain TA100. Mortelmans et al. (1986) reported bromodichloromethane was not mutagenic in S. typhimurium strains TA98, TA100, TA1535 or TA1538 both with and without rat or hamster liver homogenate. Bromodichloromethane did not induce mitotic recombination in the presence or absence of liver homogenate in studies with Saccharomyces cerevisiae strain D3 (Simmon and Kauhanen, 1978). However, Nestmann and Lee (1985) observed weak mutagenic effects in S. cerevisiae strains D7 and XV185-14C following exposure to bromodichloromethane in the absence of liver homogenate.

Bromodichloromethane was not mutagenic in the mouse lymphoma L5178/TK+/- assay in the absence of rat liver homogenate, but did induce forward mutations in this system in the presence of rat liver homogenate (NTP, 1987). Morimoto and Koizumi (1983) reported that bromodichloromethane produced a significant increase in the frequency of SCEs in both cultured human peripheral blood lymphocytes treated in vitro and mouse bone marrow cells treated in vivo. Similarly, Sobti (1984) reported statistically significant increases in the frequency of SCEs in human lymphocytes and rat liver cells exposed in vitro. A statistically significant increase in the frequency of chromosomal aberrations in Chinese hamster fibroblast was observed by Ishidate et al. (1982), but only in the presence of rat liver homogenate. NTP (1987) reported no induction of chromosomal aberrations or SCEs in CHO cells following treatment with bromodichloromethane in either the presence or absence of liver homogenate. Bromodichloromethane is structurally similar to other known animal carcinogens such as dibromochloromethane and chloroform.

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 6.2E-2 per (mg/kg)/day

Drinking Water Unit Risk -- 1.8E-6 (ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

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+0 ug/L
E-6 (1 in 1,000,000)	6E-1 ug/L

II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- Kidney (tubular cell adenoma and tubular cell adenocarcinoma)
Test Animals -- B6C3F1 mice, male
Route -- gavage, corn oil
Reference -- NTP, 1987

Dose		Combined Tumor Incidence
Administered (mg/kg/day)	Human Equivalent (mg/kg/day)	
0	0	1/46
25	1.5	2/49
50	3.0	9/50

II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

Using the linearized multistage procedure, a range of oral slope factors were calculated for bromodichloromethane, based on the observed incidence of various types of tumors (large intestine, kidney, or combined) in mice or rats reported in the NTP bioassay. The resulting cancer slope factors fall between 4.9E-3 and 6.2E-2 per (mg/kg)/day (U.S. EPA, 1992a,b,c). An oral slope factor of 1.3E-1 per (mg/kg)/day was derived from the incidence of hepatic tumors in female mice (U.S. EPA, 1993). However, because of the possible role of corn oil used as gavage vehicle (in the NTP study) in induction of hepatic tumors, carcinogenic risk estimates based on the tumor incidence in the liver is considered inappropriate.

In accordance with EPA's 1986 Guidelines for Carcinogen Risk Assessment (U.S. EPA, 1986), the slope factor of the greatest sensitivity (6.2E-2 per (mg/kg)/day) is selected as the oral quantitative cancer risk estimate for bromodichloromethane (U.S. EPA, 1992b). Survival adjustments were made for the animal counts in the control and low-dose groups by subtracting 3 and 1 early death, respectively. Additional animal count adjustment was also made in the control group due to the escape of one mouse.

The unit risk should not be used if the water concentration exceeds 6E+3 ug/L, since above this concentration the slope factor may differ from that stated.

II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

Adequate numbers of animals were used for a lifetime bioassay with two

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animal species. Bromodichloromethane was administered at two dose levels. Tumors of multiple tissue types were observed in a dose-related manner. Slope factors derived from tumor incidences of kidney and large intestine are similar and within one order of magnitude in differences.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1992c

The Drinking Water Criteria Document for Trihalomethanes received Science Advisory Board (SAB) review in 1992.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 09/07/1989, 01/11/1990, 04/01/1992, 04/02/1992

Verification Date -- 04/02/1992

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

VI. BIBLIOGRAPHY

Substance Name -- Bromodichloromethane
CASRN -- 75-27-4

Last Revised -- 03/01/1993

__VI.A. ORAL RfD REFERENCES

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__VI.B. INHALATION RfD REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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_VII. REVISION HISTORY

Substance Name -- Bromodichloromethane
CASRN -- 75-27-4

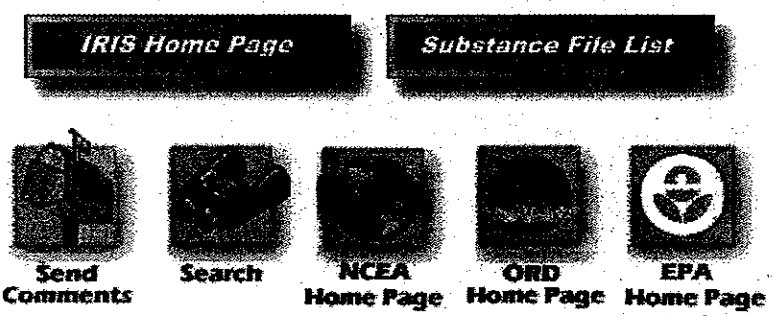
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Date	Section	Description
03/01/1988	I.A.1.	RfD exponent corrected
03/01/1988	I.A.1.	RfD exponent corrected
03/01/1988	I.A.2.	Text clarified
03/01/1988	I.A.6.	Dates corrected
06/30/1988	I.A.7.	Primary contact changed
12/01/1988	I.A.	NTP citation corrected
01/01/1989	IV.C.1.	Units corrected
08/01/1989	VI.	Bibliography on-line
10/01/1989	II.	Carcinogen assessment now under review
10/01/1990	II.	Carcinogen assessment on-line
10/01/1990	IV.F.1.	EPA contact changed
10/01/1990	VI.C.	Carcinogen references added
03/01/1991	I.A.7.	Primary contact changed
01/01/1992	IV.	Regulatory actions updated
07/01/1992	II.	Carcinogenicity assessment noted as pending change
02/01/1993	II.	Carcinogen assess. replaced; oral slope factor changed
02/01/1993	VI.C.	Carcinogenicity assessment references replaced
03/01/1993	II.A.3.	Text corrected
03/01/1993	II.D.1.	Other EPA Documentation corrected
03/01/1993	VI.C.	References corrected

VIII. SYNONYMS

Substance Name -- Bromodichloromethane
CASRN -- 75-27-4
Last Revised -- 09/30/1987

75-27-4
Bromodichloromethane
Dichlorobromomethane
Dichloromonobromomethane
Methane, bromodichloro-
Monobromodichloromethane



Last updated: 5 May 1998
URL: http://www.epa.gov/iris/subst/0213.htm

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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Butyl benzyl phthalate

CASRN 85-68-7

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0293
Butyl benzyl phthalate; CASRN 85-68-7

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Butyl benzyl phthalate

File On-Line 08/22/1988

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	02/01/1993
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	02/01/1993

00088355

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Butyl benzyl phthalate
CASRN -- 85-68-7
Last Revised -- 02/01/1993

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses	UF	MF	RfD
Significantly increased liver-to-body weight and liver-to-brain weight ratios 6-Month Rat Study Oral Exposure (diet) NTP, 1985	NOAEL: 2800 ppm (159 mg/kg/day) LOAEL: 8300 ppm (470 mg/kg/day)	1000	1	2E-1 mg/kg/day

*Conversion Factors: approximately 300 g bw and 17 g of food consumption/day from data presented in the report

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

NTP (National Toxicology Program). 1985. Twenty-six week subchronic study and modified mating trial in F344 rats. Butyl benzyl phthalate. Final Report. Project No. 12307-02, -03. Hazelton Laboratories America, Inc. Unpublished study.

NTP (1985) conducted a toxicity study in F344 rats in which 15 males/group

were administered concentrations of either 0, 0.03, 0.09, 0.28, 0.83, or 2.5% BBP in the diet for 26 weeks. Using body weight and food consumption data presented in the report these dietary levels correspond to 0, 17, 51, 159, 470, and 1417 mg/kg/day, respectively. In this study powdered rodent meal was provided in such a way that measured food consumption at the highest dose level could include significant waste and spillage rather than true food intake. For this reason a standard food consumption rate of 5% rat body weight was used in the 2.5% dose conversion. Throughout the study body weight gain was significantly depressed at the 2.5% BBP level when compared with the controls. There were no deaths attributed to BBP toxicity. All the rats given 2.5% BBP had small testes upon gross necropsy; 5/11 had soft testes and 1/11 had a small prostate and seminal vesicle. In the 0.03, 0.09, 0.28, and 0.83% dose groups there were no grossly observable effects on male reproductive organs. Terminal mean organ weight values were significantly decreased ($p<0.05$) for the heart, kidney, lungs, seminal vesicles and testes in the 2.5% group. Hematological effects at 2.5% BBP included decreased red cell mass (which the authors state is indicative of deficient hemoglobin synthesis), reduced values for hemoglobin, total RBC and hematocrit. The kidneys of six animals in the 2.5% group contained focal cortical areas of infarct-like atrophy. In addition, testicular lesions were also observed at the 2.5% dose level. Lesions were characterized by atrophy of seminiferous tubules and aspermia. At 0.83% the effects noted were significantly ($p<0.05$) increased absolute liver weight, increased liver-to-body weight and liver-to-brain weight ratios and increases in mean corpuscular hemoglobin. The 0.03, 0.09, 0.28, and 0.83% treatment groups showed no evidence of abnormal morphology in any organ. No adverse effects were observed at the 0.28% treatment level or below.

The only other information on subchronic effects is reported by Krauskopf (1973) from an unpublished study by Monsanto (1972). Rats fed diets containing 0.25% (125 mg/kg/day) and 0.5% (250 mg/kg/day) for 90 days showed no toxic effects. Liver weights were increased in animals fed diets containing 1.0, 1.5, or 2.0% (500, 750, or 1000 mg/kg/day, respectively) for 90 days, and a mild decrease in growth rate was reported for the 1.5 and 2.0% groups. No other hematologic, histopathologic or urinalysis effects were observed. When dogs were administered gelatin capsules containing doses equivalent to 1.0, 2.0, or 5.0% of the daily diet (10,000 20,000 and 50,000 ppm) for 90 days, no effect on hematological parameters, urinalysis or liver and kidney functions were observed. No further details of this study were available for review.

Similar LOAELs of 470 and 500 mg/kg/day for increased liver weight were identified in both the NTP (1985) and Monsanto (1972) studies, respectively. NOAELs differ slightly: 159 (NTP, 1985) versus 250 mg/kg/day (Monsanto, 1972). It is recommended that the NOAEL of 159 mg/kg/day from the NTP (1985) study be used to derive the RfD for two reasons: 1) the NTP (1985) study is of longer duration, and 2) The Monsanto (1972) study provides an incomplete description of methods comparing study design and clinical analysis. Treatment-related effects across similar dose ranges including liver effects in both studies support the use of 159 mg/kg/day as a NOAEL.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- 10 for intraspecies sensitivity, 10 for interspecies variability and 10 for extrapolating from subchronic to chronic NOAELs.

MF -- None

I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

Two 14-day studies support the selection of the NTP (1985) bioassay for deriving the oral RfD. Agarwal et al. (1985) administered BBP to male F344 rats in the diet for 14 consecutive days at dose levels of 0.625, 1.25, 2.5, and 5.0%. Significant increases in liver and kidney weights and kidney pathology (proximal tubular regeneration) was observed at 0.625% (375 mg/kg/day), which represents a LOAEL.

In male Sprague-Dawley rats administered 160, 480, or 1600 mg/kg/day BBP for 14 days by gastric intubation, biochemical or morphological changes in the liver as well as effects on testes weights were not observed in the 160 mg/kg/day dose group (Lake et al., 1978). However, at 480 mg/kg/day activities of ethyl morphine N-demethylase and cytochrome oxidase were significantly increased and testicular atrophy was observed in one-third Sprague-Dawley rats in the first portion of this experiment. In the second position, the 480 mg/kg/day dose induced testicular atrophy in one-sixth Sprague-Dawley rats, whereas the Wistar albino strain revealed no such effects. A NOAEL for this study would be 160 mg/kg/day based on the absence of liver and testicular effects.

In an addendum to the NTP (1985) final report, evaluation of the data revealed a significantly reduced total marrow cell count in the 2.5% dose group (NTP, 1986). The change in total cell count was comprised primarily of significant decreases in neutrophil, metamyelocytes, bands, segmeters, lymphocytes and leasophilic rubricytes. The total marrow cell counts, metamyelocyte, and leasophilic rubricyte counts were also significantly decreased in the lowest dose group, 0.03%. No statistically significant differences were noted in the middle dose groups (0.09, 0.28, or 0.83%) when compared with controls. The addendum states that decreased total marrow cell count in the 0.03 and 2.5% dose group represent change of uncertain meaning in light of the systemic effects noted in the middle dose groups. Trend analysis by the Terpstra-Jonckheere test revealed significantly ($p<0.05$%) decreasing trends in all of the previously mentioned parameters as well as an increasing trend for monocytes at 0.03 and 2.5%.

NTP (1985) also conducted a male mating trial study concomitantly with the toxicity study. Testicular atrophy was observed in male F344 rats after 10 weeks of exposure to 2.5% (2875 mg/kg/day) BBP. Throughout the study, body weight gain was significantly depressed at the 2.5% BBP level when compared with the controls.

I.A.5. CONFIDENCE IN THE ORAL RfD

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

The critical study is of adequate quality and is given a medium confidence rating. Since the critical study used only male rats and there are no adequate supporting studies of chronic duration, the data base is given a low confidence rating. Low confidence in the RfD follows.

I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1987a,b; 1988

U.S. EPA (1987a, 1988) have been both OHEA reviewed and Agency reviewed. U.S. EPA (1987b) has been OHEA reviewed.

Other EPA Documentation -- None

Agency Work Group Review -- 06/15/1989

Verification Date -- 06/15/1989

I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Butyl benzyl phthalate
CASRN -- 85-68-7

Not available at this time.

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Butyl benzyl phthalate
CASRN -- 85-68-7
Last Revised -- 02/01/1993

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity

information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- C; possible human carcinogen.

Basis -- Based on statistically significant increase in mononuclear cell leukemia in female rats; the response in male rats was inconclusive and there was no such response in mice.

___II.A.2. HUMAN CARCINOGENICITY DATA

None.

___II.A.3. ANIMAL CARCINOGENICITY DATA

Limited. A bioassay was performed by the NTP (1982) to evaluate the carcinogenic potential of orally administered butyl benzyl phthalate (BBP) to both rats and mice. Dietary levels of 0, 6000, and 12,000 ppm BBP were fed to groups of 50 male and 50 female F344 rats and 50 male and 50 female B6C3F1 mice for 103 weeks. The male rats at both dose levels experienced high mortality within the first 30 weeks of the study due to apparent internal hemorrhaging; all male rats were, thus, terminated at 30 weeks. No chronic toxicity or carcinogenic effects were observed in male or female mice. Among female rats a statistically significant increase in mononuclear cell leukemia (MCL) or lymphoma ($p=0.007$) was observed at the high dose level compared with controls with an increasing trend at $p=0.006$. The time to first tumor was 83 weeks in control as well as in the high-dose group. NTP indicated that BBP was "probably" carcinogenic in female rats. The tumor incidence was 7/49 (14%) for controls, 7/49 (14%) in low dose and 19/50 (38%) in the high dose as compared with historical control incidence in the laboratory of 19% (12-24% range).

Given the similarity of the MCL pathology in the control and the dosed female rats as well as the absence of a reduction in time to first tumor, the response is judged to be an acceleration of an old age tumor in the F344 rats. This weakens somewhat the interpretive value of the MCL response. The NTP has initiated a retest in the rats.

BBP did not induce lung adenomas in strain A mice administered 24 intraperitoneal injections of 160, 400 or 800 mg/kg (Theiss et al., 1977).

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Studies indicate that BBP is not a direct acting mutagen in the reverse mutation assay in Salmonella typhimurium (Rubin et al., 1979; Kozumbo et al., 1982; Zeiger et al., 1982) or in E. coli (NTP, 1982). Mammalian cytogenicity studies using chinese hamster ovary cells were also negative (NTP, 1982). NTP (1982) noted that additional studies on metabolites, benzyl alcohol and n-butanol was important.

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

Not available. The qualitative weaknesses of the MCL response does not provide a compelling basis to model the dose-response data.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1987

The 1987 Draft Drinking Water Quality Criteria Document received Agency review.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 08/26/1987

Verification Date -- 08/26/1987

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Butyl benzyl phthalate
CASRN -- 85-68-7
Last Revised -- 09/01/1989

__VI.A. ORAL RfD REFERENCES

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U.S. EPA. 1988. Drinking Water Criteria Document for Phthalic Acid Esters (PAEs). 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.

_VI.B. INHALATION RfD REFERENCES

None

_VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Kozumbo, W.J., R. Kroll and R.J. Rubin. 1982. Assessment of the mutagenicity of phthalate esters. Environ. Health Perspect. 45: 103-109.

NTP (National Toxicology Program). 1982. Carcinogenesis Bioassay of Butyl Benzyl Phthalate (CAS No. 85-68-7) in F344 Rats and B6C3F1 Mice (Feed Study). NTP Tech. Rep. Ser. TR No. 213, NTP, Research Triangle Park, NC. p. 98

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Zeiger, E., S. Haworth, W. Speck and V. Mortlemans. 1982. Phthalate ester testing in the National Toxicology Program's environmental mutagenesis test development program. Environ. Health Perspect. 45: 99-101.

_VII. REVISION HISTORY

Substance Name -- Butyl benzyl phthalate
CASRN -- 85-68-7

Date	Section	Description
08/22/1988	II.A.	Carcinogen summary on-line

00088363

02/01/1989	II.D.3.	Primary contact's phone number corrected
07/01/1989	I.A.	Oral RfD now under review
09/01/1989	I.A.	Oral RfD summary on-line
09/01/1989	VI.	Bibliography on-line
08/01/1991	I.A.7.	Primary and secondary contacts changed
08/01/1991	II.D.3.	Primary and secondary contacts changed
01/01/1992	IV.	Regulatory Action section on-line
02/01/1993	I.A.7.	Primary contact changed
02/01/1993	II.D.3.	Primary contact changed

VIII. SYNONYMS

Substance Name -- Butyl benzyl phthalate
CASRN -- 85-68-7
Last Revised -- 08/22/1988

85-68-7
BBP
1,2-BENZENEDICARBOXYLIC ACID, BUTYL PHENYLMETHYL ESTER
BENZYL-BUTYLESTER KYSELINY FTALOVE
BENZYL BUTYL PHTHALATE
BENZYL n-BUTYL PHTHALATE
Butyl benzyl phthalate
n-BUTYL BENZYL PHTHALATE
BUTYL PHENYLMETHYL 1,2-BENZENEDICARBOXYLATE
NCI-C54375
PALATINOL BB
PHTHALIC ACID, BENZYL BUTYL ESTER
SANTICIZER 160
SICOL 160
UNIMOLL BB

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Last updated: 5 May 1998
URL: http://www.epa.gov/iris/subst/0293.htm

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Chloroform
CASRN 67-66-3

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0025
Chloroform; CASRN 67-66-3

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Chloroform

File On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	09/01/1992
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	03/01/1991

00088365

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Chloroform
CASRN -- 67-66-3
Primary Synonym -- Trichloromethane
Last Revised -- 09/01/1992

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Fatty cyst formation in liver	NOEL: none	1000	1	1E-2 mg/kg/day
Dog, Chronic Oral Bioassay	LOAEL: 15 mg/kg/day (converted to 12.9 mg/kg/day)			
Heywood et al., 1979				

*Conversion Factors: 15 mg/kg/day x 6 days/7 days = 12.9 mg/kg/day

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Heywood, R., R.J. Sortwell, P.R.B. Noel, et al. 1979. Safety evaluation of toothpaste containing chloroform. III. Long-term study in beagle dogs. J. Environ. Pathol. Toxicol. 2: 835-851.

In this study beagle dogs were administered chloroform in a toothpaste base (0.5 mL of toothpaste base/kg/day) in gelatin capsules. A control group composed of 16 males and 16 females received the vehicle, and additional control groups of eight animals/sex were administered an alternative toothpaste or were left untreated. Experimental groups of eight male and

eight female dogs received 15 or 30 mg chloroform/kg/day for 6 days/week. Treatment was continued for 7.5 years. Fatty cysts, considered to be treatment-related, were observed in livers of some dogs in both treatment groups. Nodules of altered hepatocytes were considered treatment-related but not dose-dependent. A dose-related increase in SGPT levels was noted and a less marked increase in SGOT was noted in the high-dose animals. The LOAEL was determined to be 12.9 mg/kg/day, and an RfD was set at 0.01 mg/kg/day.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- Uncertainty factors of 10 each were applied to the LOAEL of 12.9 mg/kg/day to account for the interspecies conversion, protection of sensitive human subpopulations, and concern that the effect seen was a LOAEL and not a NOEL.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

Chloroform is considered to be highly fetotoxic, but not teratogenic (Schwetz et al., 1974; Thompson et al., 1974).

A study in rats, using only one treatment dose (Palmer et al., 1979), identified 60 mg/kg/day by gavage as a LOAEL for decreased weight gain, plasma cholinesterase and relative liver weight. Other data in the literature (Jorgenson et al., 1982) also indicate changes in liver fat to be treatment-related.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

The critical study (Heywood et al., 1979) was of chronic duration, used a fairly large number of dogs, and measured multiple endpoints; however, only two treatment doses were used and no NOEL was determined. Therefore, confidence in the study is rated medium. Confidence in the data base is considered medium to low; several studies support the choice of a LOAEL, but a NOEL was not found. Confidence in the RfD is also considered medium to low.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1985

The 1985 Drinking Water Criteria Document for Trihalomethanes is currently undergoing Agency review.

00088367

Other EPA Documentation -- None

Agency Work Group Review -- 12/02/1985, 05/15/1986

Verification Date -- 12/02/1985

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Chloroform
CASRN -- 67-66-3
Primary Synonym -- Trichloromethane

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Chloroform
CASRN -- 67-66-3
Primary Synonym -- Trichloromethane
Last Revised -- 03/01/1991

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

(Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Based on increased incidence of several tumor types in rats and three strains of mice

II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. There are no epidemiologic studies of chloroform itself. Chloroform and other trihalomethanes are formed from the interaction of chlorine with organic material found in water. Several ecological and case-control studies of populations consuming chlorinated drinking water in which chloroform was the major chlorinated organic show small significant increases in the risk of rectal bladder or colon cancer on an intermittent basis. Many other suspected carcinogens were also present in these water supplies.

II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Chloroform has been tested for carcinogenicity in eight strains of mice, two strains of rats and in beagle dogs.

In a gavage bioassay (NCI, 1976), Osborne-Mendel rats and B6C3F1 mice were treated with chloroform in corn oil 5 times/week for 78 weeks. Fifty male rats received 90 or 125 mg/kg/day; females initially were treated with 125 or 250 mg/kg/day for 22 weeks and 90 or 180 mg/kg/day thereafter. Male mice received 100 or 200, raised to 150 or 300 mg/kg/day at 18 weeks; females were dosed with 200 or 400, raised to 250 or 500 mg/kg/day. A significant increase in kidney epithelial tumors was observed in male rats and highly significant increases in hepatocellular carcinomas in mice of both sexes. Liver nodular hyperplasia was observed in low-dose male mice not developing hepatocellular carcinoma. Hepatomas have also developed in female strain A mice and NLC mice gavaged with chloroform (Eschenbrenner and Miller, 1945; Rudali, 1967).

Jorgenson et al. (1985) administered chloroform (pesticide quality and distilled) in drinking water to male Osborne-Mendel rats and female B6C3F1 mice at concentrations of 200, 400, 900, and 1800 mg/L for 104 weeks. These concentrations were reported by the author to correspond to 19, 38, 81, and 160 mg/kg/day for rats and 34, 65, 130, and 263 mg/kg/day for mice. A significant increase in renal tumors in rats was observed in the highest dose group. The increase was dose related. The liver tumor incidence in female mice was not significantly increased. This study was specifically designed to measure the effects of low doses of chloroform.

Chloroform administered in toothpaste was not carcinogenic to male C57B1, CBA, CF-1 or female ICI mice or to beagle dogs. Male ICI mice administered 60 mg/kg/day were found to have an increased incidence of kidney epithelial tumors (Roe et al., 1979; Heywood et al., 1979). A pulmonary tumor bioassay in strain A/St mice was negative as was one in which newborn C57X DBA2/F1 mice were treated s.c. on days 1 to 8 of life (Theiss et al., 1977; Roe et al., 1968).

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

The majority of tests for genotoxicity of chloroform have been negative. These negative findings include covalent binding to DNA, mutation in Salmonella, a Drosophila sex-linked recessive, tests for DNA damage a micronucleus test, and transformation of BHK cells. By contrast one study demonstrated binding of radiolabeled chloroform to calf thymus DNA following metabolism by rat liver microsomes (DiRenzo, 1982). Chloroform caused mitotic recombination in Saccharomyces (Callen et al., 1980) and sister chromatid exchange in cultured human lymphocytes and in mouse bone marrow cells exposed in vivo (Morimoto and Koizumi, 1983).

The carcinogenicity of chloroform may be a function of its metabolism to phosgene, which is known to cross-link DNA. A host-mediated assay using mice indicated that chloroform was metabolized in vivo to a form mutagenic to Salmonella strain TA1537. Likewise urine extracts from chloroform-treated mice were mutagenic (Agustin and Lim-Sylianco, 1978).

Chloroform administered to mice in drinking water promoted growth and metastasis of Ehrlich ascites cells injected i.p. (Capel et al., 1979).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

___II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor 6.1E-3 per (mg/kg)/day

Drinking Water Unit Risk -- 1.7E-7 per (ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

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

00088370

___II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- all kidney tumors
Test Animals -- rat/Osborne-Mendel, male
Route -- drinking water
Reference -- Jorgensen et al., 1985

Dose		
Administered (mg/L)	Human Equivalent (mg/kg/day)	Tumor Incidence
0	0	1/50
200	3.4	6/313
400	6.9	7/148
900	14.8	3/48
1800	28.9	7/50

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

Historical control kidney tumor incidence was 5/301.

The unit risk should not be used if the water concentration exceeds 6E+4 ug/L, since above this concentration the unit risk may not be appropriate.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

This assay was designed for detection and quantitation of effects at low dose; thus, large numbers of animals were treated and observed for their lifetime. Exposure route and vehicle is relevant to the medium for which the risk estimate was developed.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

___II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk -- 2.3E-5 per (ug/cu.m)

Extrapolation Method -- Linearized multistage procedure, extra risk

Air Concentrations at Specified Risk Levels:

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

___II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSUR

Tumor Type -- hepatocellular carcinoma
Test Animals -- mouse, B6C3F1, female
Route -- oral, gavage
Reference -- NCI, 1976

Dose		Tumor Incidence
Administered (mg/kg/day)	Human Equivalent (mg/kg/day)	
Female		
0	0	0/20
238	9.9	36/45
477	19.9	39/41
Male		
0	0	1/18
138	6.2	18/50
277	12.5	44/45

___II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE

This inhalation quantitative risk estimate is based on data from a gavage study. Above doses are TWA; body weights at the end of the assay were 35 g, males and 28 g, females. Vehicle control animals were run concurrently and housed with test animals. All treated animals experienced decreased body weight gain. Survival was reduced in high-dose males and in all treated females.

Experimental data for this compound support complete absorption of orally administered chloroform under conditions of this assay. There are no apparent species differences in this regard. Extrapolation of metabolism-dependent carcinogenic responses from mice to humans on the basis of body surface area is supported by experimental data. The incidence data for both male and female mice were used to derive slope factors of 3.3E-2 and 2.0E-1 per (mg/kg)/day, respectively. The unit risk was prepared by taking a geometric mean of the slope factor and assuming 100% for low doses of chloroform in air.

The unit risk should not be used if the air concentration exceeds 400 ug/cu.m, since above this concentration the unit risk may not be appropriate.

___II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOS

Adequate numbers of animals were treated and observed.

Risk estimates derived from male rat kidney tumor data (2.4E-2) (NCI, 1976) and studies by Roe et al. (1979) (1.0E-1) are generally supportive of the risk estimate.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

II.D.1. EPA DOCUMENTATION

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

The Health Assessment Document for Chloroform received extensive Agency and external review.

The Draft Drinking Water Criteria Document for Trihalomethanes has received external peer review.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 10/29/1986, 08/26/1987

Verification Date -- 08/26/1987

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

VI. BIBLIOGRAPHY

Substance Name -- Chloroform
CASRN -- 67-66-3
Primary Synonym -- Trichloromethane
Last Revised -- 07/01/1992

VI.A. ORAL RfD REFERENCES

Heywood, R., R.J. Sortwell, P.R.B. Noel, et al. 1979. Safety evaluation of toothpaste containing chloroform. III. Long-term study in beagle dogs. J. Environ. Pathol. Toxicol. 2: 835-851.

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U.S. EPA. 1985. Drinking Water Criteria Document for Trihalomethanes. Office of Drinking Water, Washington, DC. (External Review Draft)

VI.B. INHALATION RfC REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Augstin, J.S. and C.Y. Lim-Syliano. 1978. Mutagenic and clastogenic effects of chloroform. Bull. Phil. Biochem. Soc. 1: 17-23.

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_VII. REVISION HISTORY

Substance Name -- Chloroform
CASRN -- 67-66-3
Primary Synonym -- Trichloromethane

Date	Section	Description
03/01/1988	I.A.1.	Dose conversion clarified
03/01/1988	I.A.2.	LOAEL and RfD in text corrected
03/01/1988	I.A.4.	Text revised
03/01/1988	I.A.5.	Text revised
06/30/1988	II.	Carcinogen summary on-line
06/30/1988	I.A.7.	Primary contact changed
10/01/1989	I.B.	Inhalation RfD now under review
06/01/1990	IV.A.1.	Area code for EPA contact corrected
06/01/1990	IV.F.1.	EPA contact changed
01/01/1991	II.	Text edited
01/01/1991	II.C.1.	Inhalation slope factor removed (global change)

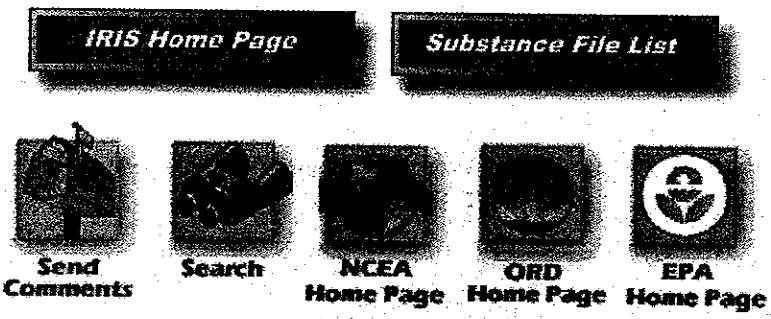
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02/01/1991	II.C.3.	Information on extrapolation process included
02/01/1991	II.C.4.	Text edited
03/01/1991	II.D.3.	Primary contact changed
01/01/1992	IV.	Regulatory actions updated
04/01/1992	IV.A.1.	CAA regulatory action withdrawn
07/01/1992	I.A.	Clarify Schwetz citation
07/01/1992	VI.C.	Oral RfD references on-line
07/01/1992	VI.C.	Carcinogenicity assessment references on-line
09/01/1992	I.A.7.	Primary contact changed

VIII. SYNONYMS

Substance Name -- Chloroform
CASRN -- 67-66-3
Primary Synonym -- Trichloromethane
Last Revised -- 01/31/1987

67-66-3
Chloroform
Formyl Trichloride
Freon 20
Methane Trichloride
Methane, Trichloro-
Methenyl Chloride
Methenyl Trichloride
Methyl Trichloride
NCI-C02686
R-20
TCM
Trichloroform
Trichloromethane



Last updated: 5 May 1998
URL: http://www.epa.gov/iris/subst/0025.htm

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

1,1-Dichloroethane

CASRN 75-34-3

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0409
1,1-Dichloroethane; CASRN 75-34-3

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR 1,1-Dichloroethane

File On-Line 10/01/1990

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	12/01/1996

00088377

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- 1,1-Dichloroethane
CASRN -- 75-34-3

Not available at this time.

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- 1,1-Dichloroethane
CASRN -- 75-34-3

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- 1,1-Dichloroethane
CASRN -- 75-34-3
Last Revised -- 12/01/1996

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for

Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- C; possible human carcinogen

Basis -- 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.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. 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).

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

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.

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

None.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

None.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1985, 1986, 1990

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

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 12/07/1989

Verification Date -- 12/07/1989

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- 1,1-Dichloroethane
CASRN -- 75-34-3
Last Revised -- 10/01/1990

__VI.A. ORAL RfD REFERENCES

None

VI.B. INHALATION RfD REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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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.

00088383

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_VII. REVISION HISTORY

Substance Name -- 1,1-Dichloroethane
CASRN -- 75-34-3

Date	Section	Description
08/01/1989	I.A.	Oral RfD now under review
05/01/1990	I.B.	Inhalation RfC now under review
10/01/1990	II.	Carcinogen assessment on-line
10/01/1990	VI.	Bibliography on-line
11/01/1991	ALL	FILES AD
01/01/1992	IV.	Regulatory Action section on-line
12/01/1996	II.D.3.	Primary contact removed

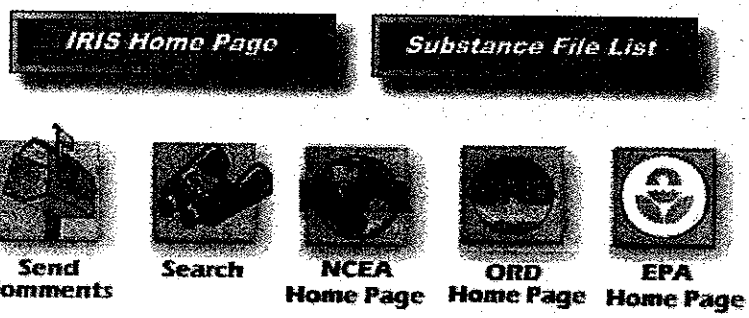
VIII. SYNONYMS

Substance Name -- 1,1-Dichloroethane
CASRN -- 75-34-3
Last Revised -- 10/01/1990

75-34-3
AETHYLIDENCHLORID [GERMAN]
CHLORINATED HYDROCHLORIC ETHER
CHLORURE D'ETHYLIDENE [FRENCH]
CLORURO DI ETILIDENE [ITALIAN]
1,1-DICHLOORETHAAN [DUTCH]
1,1-DICHLORAETHAN [GERMAN]

00088384

1,1-DICHLORETHANE
DICHLORO-1,1 ETHANE [FRENCH]
1,1-DICHLOROETHANE
1,1-DICLOROETANO [ITALIAN]
1,1-DICLOROETANO [SPANISH]
ETHANE, 1,1-DICHLORO-
ETHYLIDENE CHLORIDE
ETHYLIDENE DICHLORIDE
HSDB 64
NCI-C04535
RCRA WASTE NUMBER U076
UN 2362



Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0409.htm

United States Environmental Protection Agency

IRIS

Integrated Risk Information System

1,2-Dichloroethane

CASRN 107-06-2

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0149
1,2-Dichloroethane; CASRN 107-06-2

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR 1,2-Dichloroethane

File On-Line 03/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	01/01/1991

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- 1,2-Dichloroethane
CASRN -- 107-06-2

Not available at this time.

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- 1,2-Dichloroethane
CASRN -- 107-06-2

Not available at this time.

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- 1,2-Dichloroethane
CASRN -- 107-06-2
Last Revised -- 01/01/1991

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for

Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Based on the induction of several tumor types in rats and mice treated by gavage and lung papillomas in mice after topical application

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. ANIMAL CARCINOGENICITY DATA

1,2-Dichloroethane in corn oil was administered by gavage to groups of 50 each male and female Osborne-Mendel rats and B6C3F1 mice. Treatment was for 78 weeks followed by an additional observation period of 12-13 weeks for mice or 32 weeks for low-dose rats. TWA dosages were 47 and 95 mg/kg/day for rats, 97 and 195 mg/kg/day for male mice and 149 and 299 mg/kg/day for female mice. All high-dose male rats died after 23 weeks of observation; the last high-dose female died after 15 weeks. Male rats had significantly increased incidence of forestomach squamous-cell carcinomas and circulatory system hemangiosarcomas. Female rats and mice were observed to have significant increases in mammary adenocarcinoma incidence. Mice of both sexes developed alveolar/bronchiolar adenomas, females developed endometrial stromal polyps and sarcomas, and males developed hepatocellular carcinomas (NCI, 1978).

Inhalation exposure of Wistar, Sprague-Dawley rats and Swiss mice did not result in increased tumor incidence (Spencer et al., 1951; Maltoni et al., 1980). An elevation that was not statistically significant in lung adenomas was seen in A/st mice treated i.p. with 1,2-dichloroethane in tricaprylin (Theiss et al., 1977). ICR/Ha Swiss mice treated topically had a significant increase in benign lung papillomas, but not skin carcinomas (van Duuren et al., 1979).

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

1,2-Dichloroethane was mutagenic for Salmonella in assays wherein

excessive evaporation was prevented; exogenous metabolism by mammalian systems enhanced the response (Nestmann et al., 1980; Barber et al., 1981; Rannug et al., 1978). Both somatic cell mutations and sex-linked recessives were induced in Drosphila (Nylander et al., 1979; Shakarnis, 1969, 1970; King et al., 1979). Metabolites of 1,2-dichloroethane have been shown to form adducts with DNA after in vitro or in vivo exposures.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

___II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 9.1E-2 per (mg/kg)/day

Drinking Water Unit Risk -- 2.6E-6 per (ug/L)

Extrapolation Method -- Linearized multistage procedure with time-to-death analysis, extra risk

Drinking Water Concentrations at Specified Risk Levels:

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

___II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- hemangiosarcomas
Test Animals -- rat/Osborne-Mendel, male
Route -- gavage
Reference -- NCI, 1978

Administered Dose (mg/kg/day)	Human Equivalent Dose (mg/kg/day)	Tumor Incidence
0	0	0/40
47	4.46	9/48
95	8.23	7/27

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

Equivalent human dose was calculated using an assumed 70-kg human weight and the reported terminal rat weight of 0.5 kg. Metabolism of 1,2-dichloroethane after oral exposure is dose-dependent. Metabolism was estimated to be <50% saturation at the dose equal to the TWA for rats but near saturation for the high-dose mice in the NCI (1978) bioassay. Because of the

high mortality rate in the high-dose rats, a time-to-event analysis was used to quantitate the risk estimate. It was assumed that rats with hemangiosarcomas were killed by the tumors. The 95% upper bound of the risk was calculated using 90 weeks to approximate the lifetime risk.

The unit risk should not be used if the water concentration exceeds 4E+3 ug/L, since above this concentration the unit risk may not be appropriate.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

Adequate numbers of animals were treated and observed for the majority of their expected lifespan. The incidence of hemangiosarcoma was significantly elevated in the treated animals and was dose-related. A slope factor of 6.2E-2 (mg/kg)/day, calculated from data on hepatocellular carcinomas in male mice (NCI, 1978), is supportive of the risk estimate.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

___II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk -- 2.6E-5 per (ug/cu.m)

Extrapolation Method -- Linearized multistage procedure, extra risk

Air Concentrations at Specified Risk Levels:

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

___II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSUR

The inhalation unit risk was calculated from oral data in Section II.B.2., assuming 100% absorption and metabolism at the low dose.

___II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE

Reitz et al. (1982) found the major urinary metabolites in rats of ingested and inhaled 1,2-dichloroethane to be identical and generated in the same relative amounts.

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The unit risk should not be used if the air concentration exceeds 4E+2 ug/cu.m, since above this concentration the unit risk may not be appropriate.

___II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOS

This inhalation risk estimate was derived from the oral data presented in Section II.B.2. Based on the negative inhalation study of Maltoni et al. (1980), a 95% upper bound on risk was inferred to be 1.0E-6 per (ug/cu.m) approximately 26 times smaller than in the unit risk calculated from the rat gavage data.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1985

The Health Assessment Document for 1,2-Dichloroethane received both Agency and external review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 12/04/1986

Verification Date -- 12/04/1986

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- 1,2-Dichloroethane
CASRN -- 107-06-2

Last Revised -- 07/01/1993

VI.A. ORAL RfD REFERENCES

None

VI.B. INHALATION RfD REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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_VII. REVISION HISTORY

Substance Name -- 1,2-Dichloroethane
CASRN -- 107-06-2

Date	Section	Description
03/01/1988	II.B.4.	Confidence statement revised
03/01/1988	II.C.2.	Text added
03/01/1988	II.C.4.	Confidence statement revised
03/01/1988	III.A.	Health Advisory added
05/01/1989	IV.C.2.	All values corrected
08/01/1989	VI.	Bibliography on-line
03/01/1990	II.A.3.	Text revised (second paragraph)
06/01/1990	IV.A.1.	Area code for EPA contact corrected
06/01/1990	IV.F.1.	EPA contact changed
01/01/1991	II.	Text edited
01/01/1991	II.C.1.	Inhalation slope factor removed (global change)
01/01/1992	IV.	Regulatory actions updated
04/01/1992	IV.A.1.	CAA regulatory action withdrawn
07/01/1992	SYNONYMS	Synonyms corrected
07/01/1993	VI.C.	References alphabetized correctly

VIII. SYNONYMS

00088393

Substance Name -- 1,2-Dichloroethane
CASRN -- 107-06-2
Last Revised -- 07/01/1992

107-06-2
Ethane, 1,2-dichloro-
Ethylene Dichloride
1,2-DICHLOROETHANE
Dichloroethane, 1,2-
Aethylenchlorid [German]
AI3-01656
alpha,beta-DICHLOROETHANE
BICHLORURE D'ETHYLENE [French]
BORER SOL
Brocide
Caswell No. 440
CCRIS 225
CHLORURE D'ETHYLENE [French]
CLORURO DI ETHENE [Italian]
DESTRUXOL BORER-SOL
DI-CHLOR-MULSION
Dichlor-Mulsion
Dichloremulsion
DICHLORO-1,2-ETHANE [French]
Dichlorure d'ethylene [French]
Dicloruro de etileno [Spanish]
DUTCH LIQUID
DUTCH OIL
EDC
ENT 1,656
EPA Pesticide Chemical Code 042003
Ethane dichloride
Ethane, 1,2-Dichloro-
Ethyleendichloride [Dutch]
Ethylene chloride
Glycol dichloride
HSDB 65
NCI-C00511
RCRA WASTE NUMBER U077
sym-DICHLOROETHANE
1,2-BICHLOROETHANE
1,2-DCE
1,2-DICHLOORETHAAN [Dutch]
1,2-DICHLOR-AETHAN [German]
1,2-DICHLORETHANE
1,2-DICLOROETANO [Italian]
1,2-ETHYLENE DICHLORIDE

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00088394

Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0149.htm

00088395

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Di (2-ethylhexyl)phthalate (DEHP)

CASRN 117-81-7
03/01/1997

Contents Bis (2-ethylhexyl) Phthalate

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0014
Di(2-ethylhexyl)phthalate (DEHP); CASRN 117-81-7

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR DEHP

File On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	05/01/1991
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	02/01/1993

00088396

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Di(2-ethylhexyl)phthalate (DEHP)
CASRN -- 117-81-7
Primary Synonym -- Bis(2-ethylhexyl)phthalate
Last Revised -- 05/01/1991

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Increased relative liver weight Guinea Pig Sub-chronic-to-Chronic Oral Bioassay	NOAEL: none LOAEL: 0.04% of diet (19 mg/kg bw/day)	1000	1	2E-2 mg/kg/day

Carpenter et al., 1953

*Conversion Factors: none

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

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.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

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.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

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 (NTP, 1984). DEHP has been observed to be both fetotoxic and teratogenic (Singhe, 1972; Shiot and Nishimura, 1982).

___I.A.5. CONFIDENCE IN THE ORAL RfD

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.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

The RfD has been reviewed by the RfD Work Group. Documentation may be found in the meeting notes of 01/22/1986.

Other EPA Documentation -- None

Agency Work Group Review -- 01/22/1986

Verification Date -- 01/22/1986

00088398

I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

Substance Name -- Di(2-ethylhexyl)phthalate (DEHP)
CASRN -- 117-81-7
Primary Synonym -- Bis(2-ethylhexyl)phthalate

Not available at this time.

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Di(2-ethylhexyl)phthalate (DEHP)
CASRN -- 117-81-7
Primary Synonym -- Bis(2-ethylhexyl)phthalate
Last Revised -- 02/01/1993

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

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen.

Basis -- Orally administered DEHP produced significant dose-related increases in liver tumor responses in rats and mice of both sexes.

II.A.2. 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.

___II.A.3. 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.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

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).

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II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 1.4E-2/mg/kg/day

Drinking Water Unit Risk -- 4.0E-7 per (ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

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

II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- Mouse/B6C3F1, male
Test Animals -- hepatocellular carcinoma and adenoma
Route -- diet
Reference -- NTP, 1982

Dose		
Administered (ppm)	Human Equivalent (mg/kg)/day	Tumor Incidence
0	0	14/50
3000	32	25/48
6000	65	29/50

II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

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.

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___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

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).

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1988

The values in the 1988 Drinking Water Criteria Document for Phthalic Acid Esters (External Review Draft) have received Agency review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 08/26/1987, 10/07/1987

Verification Date -- 10/07/1987

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Di(2-ethylhexyl)phthalate (DEHP)
CASRN -- 117-81-7
Primary Synonym -- Bis(2-ethylhexyl)phthalate
Last Revised -- 05/01/1990

__VI.A. ORAL RfD REFERENCES

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.

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__VI.B. INHALATION RfC REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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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.

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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.

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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).

VII. REVISION HISTORY

Substance Name -- Di(2-ethylhexyl)phthalate (DEHP)
CASRN -- 117-81-7
Primary Synonym -- Bis(2-ethylhexyl)phthalate

Date	Section	Description
03/01/1988	I.A.2.	Text added to paragraph 1
09/07/1988	II.	Carcinogen summary on-line
02/01/1989	II.A.2.	Study description revised
02/01/1989	II.D.3.	Primary contact's phone number corrected
07/01/1989	VI.	Bibliography on-line
08/01/1989	I.A.4.	Text revised
05/01/1990	II.A.4.	Text revised
05/01/1990	VI.C.	Kozumbo et al., 1982 citation added
05/01/1991	I.A.2.	Corrected principal study title
05/01/1991	I.A.2.	2nd para, line 3 units corrected from g/kg to mg/kg
08/01/1991	II.D.3.	Primary and secondary contacts changed
08/01/1991	IV.F.1.	EPA contact changed
09/01/1991	All	Primary name changed from Bis(2-ethylhexyl)phthalate
01/01/1992	I.A.7.	Secondary contact changed
01/01/1992	IV.	Regulatory actions updated
02/01/1993	II.D.3.	Primary contact changed
05/04/1998	I.A.4.	NTP, 1984 citation added for clarification.

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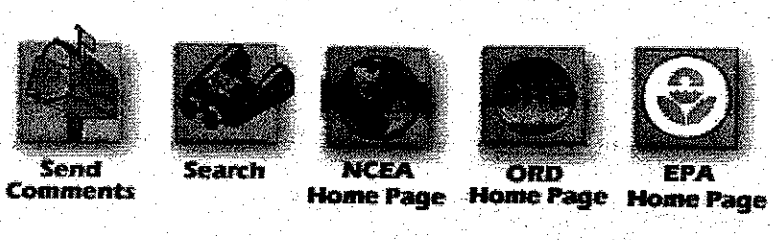
VIII. SYNONYMS

Substance Name -- Di(2-ethylhexyl)phthalate (DEHP)
CASRN -- 117-81-7
Primary Synonym -- Bis(2-ethylhexyl)phthalate
Last Revised -- 01/31/1987

117-81-7
BEHP
Bis(2-ethylhexyl)-1,2-benzene-dicarboxylate
Bis(2-ethylhexyl)phthalate
Bisoflex 81
Bisoflex DOP
Compound 889
DAF 68
DEHP
Di(2-ethylhexyl)orthophthalate
Di(2-ethylhexyl)phthalate
Dioctyl phthalate
Di-sec-octyl phthalate
DOP
Ergoplast FDO
Ethylhexyl phthalate
2-Ethylhexyl phthalate
Eviplast 80
Eviplast 81
Fleximel
Flexol DOP
Flexol plasticizer DOP
Good-Rite GP 264
Hatcol DOP
Hercoflex 260
Kodaflex DOP
Mollan O
NCI- C52733
Nuoplaz DOP
Octoil
Octyl phthalate
Palatinol AH
Phthalic acid, Bis(2-ethylhexyl) ester
Phthalic acid, dioctyl ester
Pittsburgh PX-138
Platinol DOP
RC Plasticizer DOP
RCRA waste number U028
Reomol D 79P
Reomol DOP
Sicol 150
Staflex DOP
Truflex DOP
Vestinol AH
Vinicizer 80
Witcizer 312

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00088405



Last updated: **19 May 1998**
URL: http://www.epa.gov/iris/subst/0014.htm

United States Environmental Protection Agency

IRIS
Integrated Risk Information System

cis-1,2-Dichloroethylene

CASRN 156-59-2

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0418
cis-1,2-Dichloroethylene; CASRN 156-59-2

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR cis-1,2-Dichloroethylene

File On-Line 12/01/1990

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	02/01/1995

00088407

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- cis-1,2-Dichloroethylene
CASRN -- 156-59-2

Not available at this time.

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- cis-1,2-Dichloroethylene
CASRN -- 156-59-2

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- cis-1,2-Dichloroethylene
CASRN -- 156-59-2
Last Revised -- 02/01/1995

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for

00088408

Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on no data in humans or animals and generally nonpositive results in mutagenicity assays.

___II.A.2. HUMAN CARCINOGENICITY DATA

None.

___II.A.3. ANIMAL CARCINOGENICITY DATA

None.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

cis-1,2-Dichloroethylene did not yield positive results for a Salmonella typhimurium spot test assay in the absence of mammalian liver homogenates; however, this compound did cause a dose-dependent increase in mutations in a host-mediated assay (Cerna and Kypenova, 1977). cis-1,2-Dichloroethylene at a medium concentration of 2.9 mM produced no positive results in a mutagenicity assay for Escherichia coli K12 (Greim et al., 1975). Galli et al. (1982a) reported no positive results for cis-1,2-dichloroethylene in point mutation, mitotic gene conversion and mitotic recombination assays (all for Saccharomyces cerevisiae). In addition, it did not yield positive results in an in vivo (intravenous) host-mediated mutagenicity assay (Galli et al., 1982b). cis-1,2-Dichloroethylene did not induce chromosomal aberrations in mouse bone marrow in vivo (Cerna and Kypenova, 1977).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

None.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

None.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1984

The 1984 Health Effects Assessment document has received Agency review and has been approved for publication as an EPA document.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 09/07/1989

Verification Date -- 09/07/1989

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

VI. BIBLIOGRAPHY

Substance Name -- cis-1,2-Dichloroethylene
CASRN -- 156-59-2
Last Revised -- 12/01/1990

__VI.A. ORAL RfD REFERENCES

None

__VI.B. INHALATION RfC REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Cerna, M. and H. Kypenova. 1977. Mutagenic activity of chloroethylenes analyzed by screening system tests. Mutat. Res. 46(3): 214-215.

Galli, A., C. Bauer, G. Bronzetti, et al. 1982a. Attivita genetica dell' 1,2-dichloroetilene. a) Studio in vitro. Boll. Soc. Ital. Biol. Sper. 58: 860-863. (Ital.)

Galli, A., C. Bauer, G. Bronzetti, et al. 1982b. Attivita genetica dell' 1,2-dichloroetilene. b) Studio in vivo: Effecto sugli enzimi microsomiali. Boll. Soc. Ital. Biol. Sper. 58: 864-869. (Ital.)

Greim, H., G. Bonse, Z. Radwan, D. Reichert and D. Henschler. 1975. Mutagenicity in vitro and potential carcinogenicity of chlorinated ethylenes as a function of metabolic oxirane formation. Biochem. Pharmacol. 24(21): 2013-2017.

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_VII. REVISION HISTORY

Substance Name -- cis-1,2-Dichloroethylene
CASRN -- 156-59-2

Date	Section	Description
12/01/1990	II.	Carcinogen assessment on-line

12/01/1990	VI.	Bibliography on-line
01/01/1992	IV.	Regulatory Action section on-line
02/01/1995	II.D.3.	Primary contact changed

VIII. SYNONYMS

Substance Name -- cis-1,2-Dichloroethylene
CASRN -- 156-59-2
Last Revised -- 12/01/1990

156-59-2
Ethene, 1,2-dichloro-, (Z)-
(Z)-1,2-Dichloroethene
(Z)-1,2-DICHLOROETHYLENE
cis-DICHLOROETHYLENE
CIS-1,2-DICHLORETHYLENE
CIS-1,2-DICHLOROETHENE
cis-1,2-DICHLOROETHYLENE
Ethene, 1,2-dichloro-, (Z)-
Ethylene, 1,2-dichloro-, (Z)-
HSDB 5656
NSC 6149
1,2-CIS-DICHLOROETHYLENE

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0418.htm

00088412

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

1,1-Dichloroethylene

CASRN 75-35-4
(02/01/1998)

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0039
1,1-Dichloroethylene; CASRN 75-35-4 (02/01/1998)

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR 1,1-Dichloroethylene

File On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	04/01/1989
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	02/01/1998

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I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- 1,1-Dichloroethylene
CASRN -- 75-35-4
Primary Synonym -- Vinylidene Chloride
Last Revised -- 04/01/1989

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

NOTE: The Oral RfD for 1,1,-dichloroethylene may change in the near future pending the outcome of a further review now being conducted by the Oral RfD Work Group.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Hepatic lesions	NOAEL: none	1000	1	9E-3 mg/kg/day
Rat Chronic Oral Bioassay	LOAEL: 50 ppm (converted to 9 mg/kg/day)			
Quast et al., 1983				

*Conversion Factors: Doses were calculated by the authors.

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Quast, J.F., C.G. Humiston, C.E. Wade, et al. 1983. A chronic toxicity and oncogenicity study in rats and subchronic toxicity study in dogs on ingested

vinylidene chloride. Fund. Appl. Toxicol. 3: 55-62.

Groups of 48 each male and female Sprague-Dawley rats (Spartan substrain) were administered 50, 100, or 200 ppm 1,1-dichloroethylene in drinking water for a period of 2 years. Control groups of 80 animals/sex were maintained for the same period. Daily intake was calculated by the authors to be 7, 10, or 20 mg/kg bw/day for males and 9, 14, or 30 mg/kg bw/day for females. There were no treatment-related effects on mortality, body or organ weight, clinical chemistry, urinalysis, hematology, or numbers of tumors. The only pathologic findings were of hepatic lesions, generally characterized by minimal mid-zonal hepatocellular fatty change and hepatocellular swelling. These findings were noted in rats of all female treatment groups. In male rats, only the 200 ppm treatment group showed a statistically significant increase in the incidence of hepatocellular swelling, but this trend was also observed in animals receiving 100 ppm 1,1-dichloroethylene.

As part of this same study, beagle dogs (4/sex/group) were administered 6.25, 12.5, or 25 mg/kg bw/day in gelatin capsules. Treatment for 97 days had no effect.

This study, as well as a review of the available data, indicate that the liver is the most sensitive target organ and, futhermore, that rats are the most sensitive species. The drinking water exposure reported by Quast et al. (1983) offers a more suitable model for potential human exposure to 1,1-dichloroethylene than does the NTP bioassay wherein animals were gavaged. It is, therefore, appropriate to set an RfD based on the LOAEL of 9 mg/kg bw/day for hepatic lesions in female rats.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- A factor of 10 each was used for use of a LOAEL, for interspecies variation, and for protection of sensitive human subpopulations.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

1,1-Dichloroethylene has been shown to be fetotoxic, but not teratogenic to rodents after exposure in drinking water or by inhalation (Short et al., 1977; Murray et al., 1979).

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

The study by Quast et al. (1983) was conducted using appropriate numbers of animals of two species, measured several endpoints, and was of chronic duration (rats). Since there are corroborative chronic and subchronic oral bioassays, confidence in the study, data base, and RfD are considered medium.

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___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1985

This document has received a lengthy internal review and has undergone public comments.

Other EPA Documentation -- None

Agency Work Group Review -- 01/22/1985

Verification Date -- 01/22/1985

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE

(RfC)

Substance Name -- 1,1-Dichloroethylene
CASRN -- 75-35-4
Primary Synonym -- Vinylidene Chloride

Not available at this time.

==========

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- 1,1-Dichloroethylene
CASRN -- 75-35-4
Primary Synonym -- Vinylidene Chloride
Last Revised -- 02/01/1998

Section II provides information on three aspects of the carcinogenic

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

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- C; possible human carcinogen

Basis -- Tumors observed in one mouse strain after inhalation exposure is the basis for this classification. Other studies were of inadequate design. Vinylidene chloride is mutagenic, and a metabolite is known to alkylate and to bind covalently to DNA. It is structurally related to the known human carcinogen, vinyl chloride.

II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. An epidemiologic study of 138 workers showed no carcinogenic effect associated with vinylidene chloride exposure (Ott et al., 1976). Based on power considerations, this study is inadequate for assessing cancer risk in humans.

II.A.3. ANIMAL CARCINOGENICITY DATA

Limited. Eighteen animal studies have been reported, which provide information about the carcinogenic potential of vinylidene chloride. Eleven of the studies involved inhalation exposure, five were oral, and one each was by skin application and subcutaneous injection. Most were not designed for maximum sensitivity to detect carcinogenic effects. None of the 11 inhalation exposures were for lifetime; all were 12 months or less. Three of the five oral studies were of lifetime exposures. Of all the studies, only one inhalation study produced a response as a complete carcinogen.

In the inhalation study by Maltoni et al. (1985) both sexes of Swiss

mice were exposed to 10 and 25 ppm (MTD) for 4-5 days/week for 12 months. A statistically significant increase in kidney adenocarcinoma was noted in male mice. Although statistically significant increases in mammary carcinomas in female mice and pulmonary adenomas in both sexes were reported, dose-response relationships were unclear. A second Maltoni study exposed Sprague-Dawley rats to 10, 25, 50, 100, or 150 ppm, 4-5 days/week for 12 months and observed them until spontaneous death. A statistically significant increase in total mammary tumors, but not carcinomas alone, was seen only at 10 and 100 ppm. No dose-response relationship was apparent, and the overall interpretation of the mammary tumor incidence is inconclusive.

Four gavage studies (in rats and mice) and one drinking water study in rats have been negative (Maltoni et al., 1985; Quast et al., 1983; Humiston et al., 1978; NTP, 1982; Ponomarkov and Tomatis, 1980). Only the NTP (1982) corn oil gavage study in Fischer 344 rats and female B6C3F1 mice and the drinking water study in Sprague-Dawley rats were undertaken for 2 years dosing. The NTP study was apparently not conducted at the MTD and the drinking water study did not achieve a maximum dose of metabolite. All other oral studies were limited in design and, thus, lacking in sensitivity sufficient to detect a response.

Vinylidene chloride did not act as a complete carcinogen when applied topically or s.c. to ICR/Ha mice but did serve as an initiator when followed by phorbol myristate acetate treatment (Van Duuren et al., 1979).

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Vinylidene chloride has been shown to be mutagenic for Salmonella typhimurium in multiple assays. This activity is largely dependent on the presence of microsomal enzymes. It has been used as a positive control in studies of chemicals that are gases at or near room temperature. Both conventional and host-mediated assays of Saccharomyces cerevisiae have been positive for mitotic gene conversion (Bronzetti et al., 1981). Vinylidene chloride was not mutagenic for V79 cells exposed to vapor in vitro (Drevon and Kuroki, 1979), nor did it produce chromosomal aberrations in bone marrow cells of ICR mice given single or repeated i.p. treatment in vivo (Cerna and Kypenova, 1977). CD-1 mice and Sprague-Dawley rats treated in vivo with labeled vinylidene chloride showed evidence of DNA alkylation and subsequent repair which was specific to liver and kidney. Kidney in rat and mouse had higher alkylation than liver (Reitz et al., 1980). Covalent binding of vinylidene chloride closely correlates with metabolite formation. McKenna et al. (1977) observed greater binding in kidney than liver, and greater binding in mice than in rats. Vinylidene chloride failed to induce dominant lethal mutations in mice (Anderson et al., 1977) or rats (Short et al., 1977). Vinylidene chloride is structurally related to the known carcinogen, vinyl chloride.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL

EXPOSURE

___II.B.1. SUMMARY OF RISK ESTIMATES

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Oral Slope Factor -- 6E-1 per (mg/kg)/day

Drinking Water Unit Risk -- 1.7E-5 per (ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

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

___II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- adrenal pheochromocytomas
Test Animals -- rat/F344, male
Route -- drinking water
Reference -- NTP, 1982

Administered Dose (mg/kg-day)	Human Equivalent Dose (mg/kg-day)	Tumor Incidence
0	0	6/50
0.71	0.120	5/48
3.57	0.603	13/47

Human equivalent doses were determined by adjusting the administered animal dose by the cube root of the ratio of the weight of the animal (estimated 0.43 kg) to human weight (70 kg), and adjusting for length of exposure and length of the experiment.

The unit risk estimate chosen was derived from the highest of four slope factors calculated from two studies that did not show a statistically significant increase in tumor incidence attributable to oral exposure of vinylidene chloride. The drinking water study in rats (Quast et al., 1983) produced the lowest slope factor of 0.2 per (mg/kg)/day. The highest slope factor [0.6 per (mg/kg)/day] was based on male rat adrenal tumors from NTP (1982). Use of data from this study wherein there were no statistically significant increases in tumor incidence appears justified, since the slope factor derived is within a factor of 2 of the slope factor based on data from the inhalation study of Maltoni et al. (1977, 1985).

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

Animal pharmacokinetic data show that metabolite elimination is dose-dependent and saturable at inhalation concentrations of 150-200 ppm, or approximately 50 mg/kg oral ingestion. Vinylidene chloride is rapidly absorbed, has limited solubility, and is not stored in body tissues. Pharmacokinetics and metabolism data indicate that the available assays were not of adequate design. The positive Maltoni inhalation study comes closest to achieving a maximum dose of metabolite, albeit less than lifetime exposure, but less than maximum dosing vis-a-vis metabolites. The water unit risk based on incidence data from a drinking water study was chosen

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because route of administration is appropriate to oral risk estimation.

The unit risk should not be used if the water concentration exceeds 6E+2 ug/L, since above this concentration the unit risk may not be appropriate.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

The estimate is based on a data set in which there is no significant increase in tumor incidence. The confidence that the upper limit is not greater than 0.6 per (mg/kg)/day is high, since it is the largest value by a factor of 3 from four rat data sets in two studies. If drinking water exposure alone is considered the estimates might be reduced by a factor of 3.

The slope factors for the oral quantitative estimate based on data from inhalation exposure and based on the negative oral data are within a factor of 2.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION

EXPOSURE

___II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk -- 5.0E-5 per (ug/cu.m)

Extrapolation Method -- Linearized multistage procedure, extra risk

Air Concentrations at Specified Risk Levels:

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

___II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION

EXPOSURE

Tumor Type -- kidney adenocarcinoma
Test Animals -- Mouse/Swiss, male
Route -- inhalation
Reference -- Maltoni et al., 1977, 1985

Dose		Tumor
Administered (ppm)	Human Equivalent (mg/kg/day)	Incidence
0	0	0/56

0	0	0/70
10	0.078	0/25
25	0.195	3/21
25	0.195	25/98

___II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION

EXPOSURE)

Within each same dose pair there were no statistically significant differences between incidences in the two control and the 25 ppm groups. These groups were combined for modeling. The number of animals surviving to appearance of the first kidney adenocarcinoma was used as the denominator for incidence. Human equivalent doses were determined assuming 0.035 kg as the average weight of the male mice, adjusting for continuous lifetime exposure in the mice, accounting for metabolism and pharmacokinetics for mice, and using 70 kg weight and with 1.85 sq.m surface area for humans (U.S. EPA, 1985). A slope factor of 0.2E+0 per (mg/kg)/day was calculated using estimated animal administered doses.

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

___II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION

EXPOSURE)

Sufficient numbers of animals were used for treatment and control groups. Treatment was for approximately 50% of lifetime. Only two dose points provided data suitable for modeling.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY

ASSESSMENT)

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1985

The values in the 1985 Health Assessment Document for Vinylidene Chloride received extensive peer and public review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 12/04/1986, 01/07/1987

Verification Date -- 01/07/1987

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

VI. BIBLIOGRAPHY

Substance Name -- 1,1-Dichloroethylene
CASRN -- 75-35-4
Primary Synonym -- Vinylidene Chloride
Last Revised -- 08/01/1991

VI.A. ORAL RfD REFERENCES

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

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VI.B. INHALATION RfC REFERENCES

None

00088422

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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McKenna, M.J., P.G. Watanabe and P.G. Gehring. 1977. Pharmacokinetics of vinylidene chloride in the rat. Environ. Health Perspect. 21: 99-105.

NTP (National Cancer Institute/National Toxicology Program). 1982. NTP Technical Report on the Carcinogenesis Bioassay of Vinylidene Chloride (CAS No. 75-35-4) in F344/N Rats and B6CF1/Mice (Gavage Study). NPT No. 80-82. NIH Pub. No. 82-1784. NTP Research Triangle Park, NC. and Bethesda, MD. U.S. Department of Health and Human Services, Public Health Services, National Institute of Health.

Ott, M.G., W.A. Fishbeck, J.C. Townsend and E.J. Schneider. 1976. A Health study of employees exposed to vinylidene chloride. J. Occup. Med. 18(11): 735-738.

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Short, R.D., J.M. Winston, J.L. Minor, J. Seifter and C-C. Lee. 1977. Effect

of various treatments on toxicity of inhaled vinylidene chloride. Environ. Health Perspect. 21: 125-129.

U.S. EPA. 1985. Health Assessment Document for Vinylidene Chloride. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Research Triangle Park, NC. EPA 600/8-83-031F.

Van Duuren, B., B.M. Goldschmidt, G. Loewengert, A.C. Smith, S. Melchionne, I. Seidman and D. Roth. 1979. Carcinogenicity of halogenated olefinic and aliphatic hydrocarbons in mice. J. Natl. Cancer Inst. 63(6): 1433-1439.

VII. REVISION HISTORY

Substance Name -- 1,1-Dichloroethylene
CASRN -- 75-35-4
Primary Synonym -- Vinylidene Chloride

Date	Section	Description
03/31/1987	II.	Carcinogenicity Section added
03/01/1988	I.A.1.	Dose conversion clarified
03/01/1988	I.A.7.	Contact changed
03/01/1988	II.A.2.	Text added
03/01/1988	II.B.3.	Text revised
03/01/1988	II.B.4.	Confidence statement revised
03/01/1988	II.C.3.	Text added
03/01/1988	II.C.4.	Confidence statement revised
06/30/1988	I.A.7.	Changed primary contact's telephone number
12/01/1988	II.A.3.	van Durren et al. citation year corrected
04/01/1989	I.A.	Oral RfD summary noted as pending change
12/01/1989	I.B.	Inhalation RfD now under review
03/01/1990	II.	Clarified NTP, 1982 citation
03/01/1990	VI.	Bibliography on-line
01/01/1991	II.	Text edited
01/01/1991	II.C.1.	Inhalation slope factor removed (global change)
02/01/1991	II.C.3.	Information on extrapolation process included
08/01/1991	VI.A.	References clarified
08/01/1991	VI.C.	References clarified
01/01/1992	IV.	Regulatory actions updated
02/01/1998	II.C.3.	Slope factor corrected from 1.2 to 0.2E+0

VIII. SYNONYMS

Substance Name -- 1,1-Dichloroethylene

00088424

CASRN -- 75-35-4
Primary Synonym -- Vinylidene Chloride
Last Revised -- 01/31/1987

75-35-4
CHLORURE DE VINYLIDENE
1,1-DCE
1,1-DICHLOROETHENE
Dichloroethylene, 1,1-
ETHENE, 1,1-DICHLORO-
ETHYLENE, 1,1-DICHLORO-
NCI-C54262
RCRA WASTE NUMBER U078
SCONATEX
UN 1303
VDC
Vinylidene Chloride
VINYLIDENE DICHLORIDE
VINYLIDINE CHLORIDE
□

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0039.htm

00088425

United States Environmental Protection Agency

IRIS
Integrated Risk Information System

trans-1,2-Dichloroethylene

CASRN 156-60-5

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0314
trans-1,2-Dichloroethylene; CASRN 156-60-5

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR trans-1,2-Dichloroethylene

File On-Line 09/26/1988

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	01/01/1989
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	no data	

00088426

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- trans-1,2-Dichloroethylene
CASRN -- 156-60-5
Last Revised -- 01/01/1989

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Increased serum alkaline phosphatase in male mice	NOAEL: 0.1 mg/L (17 mg/kg/day)	1000	1	2E-2 mg/kg/day
90-Day Mouse Drinking Water Study	LOAEL: 1 mg/L (175 mg/kg/day)			
Barnes et al., 1985				

*Conversion Factors: See text.

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

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

Male and female CD-1 mice were given trans-1,2-dichloroethylene (DCE) in their drinking water for 90 days at concentrations of 0.1, 1.0, or 2.0 mg/mL. Based on fluid consumption, the doses were: males - 17, 175, or 387 mg/kg/day and females - 23, 224, or 452 mg/kg/day. There were no differences in fluid consumption among the various groups of mice and at the termination of the 90-day experiment, no DCE-induced changes in terminal body weight or gross

00088427

pathology were noted in either sex at any dose level. In male mice (N=24), there were significant increases in serum alkaline phosphatase levels at the 175 and 387 mg/kg/day levels. In addition, liver glutathione concentrations were decreased at the highest dose. In females (N=24), the thymus weight, calculated as percent body weight, was significantly decreased at either 224 or 452 mg/kg/day, while the lung weight was depressed at the highest dose only. The levels of serum glutamic-pyruvic (SGPT) and glutamic-oxaloacetic (SGOT) transaminases were decreased at the two higher doses. In addition, the levels of aniline hydroxylase were decreased at all three dose levels, but the levels of cytochromes P-450 and b5, microsomal protein and aminopyrine N-demethylase were not affected by any dose of DCE. Since there is uncertainty about the toxicological significance of the decreases in SGOT, SGPT, and aniline hydroxylase, these parameters will not be used to set RfD values.

The immunotoxicity of trans-1,2-dichloroethylene (DCE) was assessed in a 90-day study by Shopp et al. (1985). Humoral immune status was assessed by measuring: 1) quantitation of spleen IgM antibody forming cells (AFC), 2) hemagglutination titers to sheep erythrocytes (sRBC), and 3) spleen response to the B cell mutagen lipopolysaccharide (LPS). Cell-mediated immunity was assayed by: 1) delayed type hypersensitivity (DTH) to SRBC, 2) popiteal lymph node proliferation in response to SRBC, and 3) spleen cell response to challenge with concanavalin A. In males on day 4 after treatment, there was a significant decrease in AFC/spleen at 17, 175, and 387 mg/kg/day does levels, but the decreases were only significant at the 175 and 387 mg/kg/day levels when the data were calculated on the basis of spleen cells. On day 5, a decrease in AFC was noted only at the 387 mg/kg/day level. No dose-dependent effects were noted in the DCE-treated female mice. In both sexes, there were no changes in hemagglutination titers after DCE treatment and spleen responsiveness to LPS was unaltered in male mice. However, females exposed to 452 mg/kg/day did have an enhanced response to LPS. In addition, lymphocyte responsiveness in the absence of the mitogen (LPS) was decreased at the 224 and 452 mg/kg/day doses in females only. In the cell-mediated immunity assays, there were no dose-related decreases seen in either sex. The significance of the decrease in AFC/spleen in male mice at all three dose levels is uncertain. This decrease is seen at only the 175 and 387 mg/kg/day levels when the data were calculated on the basis of spleen cells. Furthermore, two other measures of humoral immune status, hemagglutination titers and spleen cell response to LPS, were not affected. Accordingly, this parameter will not be used to set a RfD, but will be used as supportive data.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- The UF of 1000 allows for uncertainty in the extrapolation of dose levels from laboratory animals to humans (10A), uncertainty in the threshold for sensitive humans (10H), and uncertainty in the effect of duration when extrapolating from subchronic to chronic exposure (10S).

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

CD rats, 22 to 30 days old, were given trans-1,2-dichloroethylene (DCE) for 90 days in their drinking water at doses of 402, 1311 or 3114 mg/kg/day for males and 353, 1257 or 2809 mg/kg/day for females (Hayes et al., 1987). The authors found no compound-related effects on fluid consumption, body weights, hematology, serum chemistry or urinalyses. They did note a significant dose-

00088428

dependent decrease in kidney weight at the 1257 and 2809 mg/kg/day doses in female rats.

Wistar rats were exposed to air containing trans-1,2-dichloroethylene at 0, 200, 1000 or 2000 ppm (0 to 7940 mg/cu.m) (Freundt et al., 1977). Brief (8-hour) or prolonged (8 hours/day, 5 days/week for 1, 2, 8, or 16 weeks) exposure at 200 ppm produced slight degeneration of the liver lobule and lipid accumulation in the Kupffer cells. At 8 and 16 weeks of exposure, severe pneumonic infiltration was observed. Exposure at 1000 ppm for 8 hours resulted in significant reductions in serum albumin, urea nitrogen and alkaline phosphatase. Eight-hour exposures at both 200 and 1000 ppm produced a significant decrease in the number of leucocytes.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

The principal study was well-designed, except for dose spacing, and did establish both a NOEL and LOAEL. The data base confidence is rated low because of the lack of chronic studies and the lack of data on reproductive and developmental toxicity. Confidence in the RfD is rated low because of the data base weakness.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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

Other EPA Documentation -- None

Agency Work Group Review -- 04/20/1988

Verification Date -- 04/20/1988

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- trans-1,2-Dichloroethylene

00088429

CASRN -- 156-60-5

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- trans-1,2-Dichloroethylene
CASRN -- 156-60-5

This substance/agent has not undergone a complete evaluation and determination under US EPA's IRIS program for evidence of human carcinogenic potential.

_VI. BIBLIOGRAPHY

Substance Name -- trans-1,2-Dichloroethylene
CASRN -- 156-60-5
Last Revised -- 03/01/1991

__VI.A. ORAL RfD REFERENCES

Barnes, D.W., V.M. Sanders, K.L. White, Jr., G.M. Shopp and A.E. Munson. 1985. Toxicology of trans-1,2-dichloroethylene in the mouse. Drug Chem. Toxicol. 8: 373-392.

Freundt, J.J., G.P. Liebaldt and E. Lieberwirth. 1977. Toxicity studies on trans-1,2-dichloroethylene. Toxicology. 7: 141-153.

Hayes, J.R., L.W. Condie, J.L. Egle and J.F. Borzelleca. 1987. The acute and subacute toxicity in rats of trans-1,2-dichloroethylene in drinking water. J. Amer. College Toxicol. 6(4): 471-478.

Shopp, G.M., V.M. Sanders, K.L. White and A.E. Munson. 1985. Humoral and cell-mediated immune status of mice exposed to trans-1,2-dichloroethylene. Drug Food Chem. Toxicol. 8(5): 393-407.

__VI.B. INHALATION RfC REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

None

VII. REVISION HISTORY

Substance Name -- trans-1,2-Dichloroethylene
CASRN -- 156-60-5

Date	Section	Description
09/26/1988	I.A.	Oral RfD summary on-line
01/01/1989	I.A.7.	Contact phone number corrected
03/01/1991	VI.	Bibliography on-line
01/01/1992	IV.	Regulatory Action section on-line

VIII. SYNONYMS

Substance Name -- trans-1,2-Dichloroethylene
CASRN -- 156-60-5
Last Revised -- 09/26/1988

156-60-5
acetylene dichloride, trans-
dichloroethylene, trans-
1,2-Dichloroethylene, trans-
ethylene, 1,2-dichloro-, (E)-
RCRA waste number U079
trans-acetylene dichloride
trans-dichloroethylene
trans-1,2-Dichloroethylene

00088431

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0314.htm

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Dibutyl phthalate

CASRN 84-74-2

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0038
Dibutyl phthalate; CASRN 84-74-2

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Dibutyl phthalate

File On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	08/01/1990
Inhalation RfC Assessment (I.B.)	message	10/01/1990
Carcinogenicity Assessment (II.)	on-line	02/01/1993

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Dibutyl phthalate
CASRN -- 84-74-2
Last Revised -- 08/01/1990

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

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

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Increased mortality Rat Subchronic to Chronic, Oral Bioassay	NOAEL: 0.25% of diet (125 mg/kg/day) LOAEL: 1.25% of diet (600 mg/kg bw/day)	1000	1	1E-1 mg/kg/day

Smith, 1953

*Conversion Factors: The values of 125 mg/kg/day for 0.25% dibutyl phthalate in the diet and 600 mg/kg/day for 1.25% were estimated from a figure depicting daily intake in mg/kg in Smith (1953).

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Smith, C.C. 1953. Toxicity of butyl sterate, dibutyl sebacate, dibutyl phthalate and methoxyethyl oleate. Arch. Hyg. Occup. Med. 7: 310-318.

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Male Sprague-Dawley rats in groups of 10 were fed diets containing 0, 0.01, 0.05, 0.25, and 1.25% dibutyl phthalate for a period of 1 year. One-half of all rats receiving the highest dibutyl phthalate concentration died during the first week of exposure. The remaining animals survived the study with no apparent ill effects. There was no effect of treatment on gross pathology or hematology. While it was stated that several organs were sectioned and stained, no histopathologic evaluation was reported.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- A factor of 10 was applied to account for interspecies variation, a factor of 10 for protection of sensitive human subpopulations, and an additional factor of 10 to account for both the less-than-chronic duration of the study and deficiencies in the study, such as the use of only male animals.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

Fetotoxicity was observed when mice were fed 2100 mg/kg/day dibutyl phthalate throughout gestation (Shiota and Nishimura, 1982). An increase in terata of borderline statistical significance was observed in progeny of this treatment group. Dibutyl phthalate produces degeneration of the seminiferous tubules, probably as a result of increased urinary excretion of zinc (Gangolli, 1982).

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

The study by Smith (1953) used few animals of one sex only. It was not indicated in the paper whether the 50% mortality observed early in the study was considered treatment-related, nor was the cause of death indicated. This is the only subchronic bioassay of dibutyl phthalate reported in the literature. Confidence in the study, data base, and RfD are all rated low.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1980

The RfD in the 1980 Ambient Water Quality Criteria document received extensive peer and public review.

Other EPA Documentation -- None

Agency Work Group Review -- 01/22/1986

Verification Date -- 01/22/1986

I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Dibutyl phthalate
CASRN -- 84-74-2

The health effects data for dibutyl phthalate were 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 health effects of this chemical interested parties are referred to the EPA documentation listed below.

U.S. EPA. 1987. Drinking Water Criteria Document for Phthalic Acid Esters Prepared by the Office of Health and Environmental Assessement, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (External Review Draft)

Agency Work Group Review -- 07/26/1990

EPA Contacts:

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Dibutyl phthalate
CASRN -- 84-74-2
Last Revised -- 02/01/1993

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is

00088436

the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable.

Basis -- Pertinent data regarding carcinogenicity was not located in the available literature.

___II.A.2. HUMAN CARCINOGENICITY DATA

None.

___II.A.3. ANIMAL CARCINOGENICITY DATA

None.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

DBP 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 the absence of S9 hepatic homogenate (Kozumbo et al., 1982). It was a weak direct-acting mutagen in a forward mutation assay in Salmonella typhimurium (Seed, 1982). DBP was mutagenic in the mouse lymphoma forward mutation assay only in the presence of metabolic activation (CMA, 1986). In addition, DBP showed some evidence of clastogenic activity in Chinese hamster fibroblasts (Ishidate and Odashima, 1977) but was negative in human leukocytes (Tsuchiya and Hattori, 1977). Research indicates that DBP is hydrolyzed to monoesters (Kluwe, 1982; Rowland et al., 1977; Albro and Moore, 1974). There is evidence that DBP induces peroxisome proliferation (U.S. EPA, 1987).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

Not available.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1987

The Drinking Water Criteria Document for Phthalic Acid Esters has received OHEA review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 08/26/1987

Verification Date -- 08/26/1987

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Dibutyl phthalate
CASRN -- 84-74-2
Last Revised -- 10/01/1990

__VI.A. ORAL RfD REFERENCES

Gangolli, S.D. 1982. Testicular effects of phthalate esters. Environ. Health Perspect. 45: 77-84.

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.

Smith C.C. 1953. Toxicity of butyl sterate, dibutyl sebacate, dibutyl phthalate and methoxyethyl oleate. Arch. Hyg. Occup. Med. 7: 310-318.

U.S. EPA. 1980. Ambient Water Quality Criteria for Phthalate Esters. 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 440/5-80-067. NTIS PB 81-117780.

__VI.B. INHALATION RfC REFERENCES

U.S. EPA. 1987. Drinking Water Criteria Document for Phthalic Acid Esters Prepared by the Office of Health and Environmental Assessement, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. (External Review Draft)

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Albro, P.W. and B. Moore. 1974. Identification of the metabolites of simple phthalate diesters in rat urine. J. Chromatogr. 94: 209-218.

CMA (Chemical Manufacturers Association). 1986. Mutagenicity of 1C (di-n-butyl phthalate) in a mouse lymphoma mutation assay. Final report. Submitted to Hazleton Biotechnologies Company. HB Project No. 20989. September, 1986.

Ishidate, M., Jr. and S. Odashima. 1977. Chromosome tests with 134 compounds on Chinese hamster cells in vitro -- A screening test for chemical carcinogens. Mutat. Res. 48: 337-354.

Kluwe, W.M. 1982. Overview of phthalate ester pharmacokinetics in mammalian species. Environ. Health Perspect. 45: 3-10.

Kozumbo, W.J., R. Kroll and R.J. Rubin. 1982. Assessment of the mutagenicity of phthalate esters. Environ. Health Perspect. 45: 103-109.

Rowland, I.R., R.C. Cottrell and J.C. Phillips. 1977. Hydrolysis of phthalate esters by the gastro-intestinal contents of the rat. Food Cosmet. Toxicol. 15: 17-21.

Seed, J.L. 1982. Mutagenic activity of phthalate esters in bacterial liquid suspension assays. Environ. Health Perspect. 45: 111-114.

Tsuchiya, K. and K. Hattori. 1977. Chromosomal study on human leukocyte cultures treated with phthalic acid ester. Hokkaidoritus Eisei Kenkyusho Ho. 26: 114. (Abstract)

U.S. EPA. 1987. 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.

_VII. REVISION HISTORY

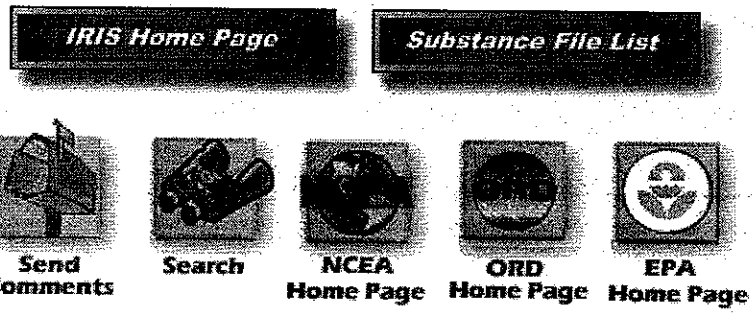
Substance Name -- Dibutyl phthalate
CASRN -- 84-74-2

Date	Section	Description
09/07/1988	II.	Carcinogen summary on-line
08/01/1989	VI.	Bibliography on-line
03/01/1990	I.A.4.	Text corrected
05/01/1990	II.A.4.	First sentence revised
08/01/1990	I.A.	Oral RfD summary noted as pending change
09/01/1990	I.B.	Not verified; data inadequate
09/01/1990	IV.F.1.	EPA contact changed
10/01/1990	I.B.	Inhalation RfC message on-line
10/01/1990	VI.B.	Inhalation RfC references added
08/01/1991	II.D.3.	Primary and secondary contacts changed
01/01/1992	I.A.7.	Secondary contact changed
01/01/1992	IV.	Regulatory actions updated
02/01/1993	II.D.3.	Primary contact changed

VIII. SYNONYMS

Substance Name -- Dibutyl phthalate
CASRN -- 84-74-2
Last Revised -- 01/31/1987

84-74-2
1,2-Benzenedicarboxylic Acid Dibutyl Ester
o-Benzenedicarboxylic Acid, Dibutyl Ester
Benzene-o-Dicarboxylic Acid Di-n-Butyl Ester
Butylphthalate
Celluflex DPB
Dibutyl 1,2-Benzene dicarboxylate
Dibutyl phthalate
Di-n-Butylphthalate
Dibutyl-o-Phthalate
DPB
Elaol
Ergoplast FDB
Genoplast B
Hexaplast M/B
N-Butylphthalate
Palatinol C
Phthalic Acid Dibutyl Ester
Polycizer DBP
PX 104
RC Plasticizer DBP



Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0038.htm

00088441

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

m-Dinitrobenzene

CASRN 99-65-0

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0318
m-Dinitrobenzene; CASRN 99-65-0

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR m-Dinitrobenzene

File On-Line 08/22/1988

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	08/22/1988
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	02/01/1993

00088442

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- m-Dinitrobenzene
CASRN -- 99-65-0
Last Revised -- 08/22/1988

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Increased splenic weight	NOAEL: 3 ppm (0.40 mg/kg/day)	3000	1	1E-4 mg/kg/day
Rat Subchronic Oral Study	LOAEL: 8 ppm			
Cody et al., 1981				

*Conversion Factors: Drinking water concentrations converted to dosages by investigators.

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Cody, T.E., S. Witherup, L. Hastings, K. Stemmer and R.T. Christian. 1981. 1,3-Dinitrobenzene: Toxic effect in vivo and in vitro. J. Toxicol. Environ. Health. 7(5): 829-847.

Groups of 20 Carworth Farm rats of each sex were exposed to 0, 3, 8, or 20 ppm m-dinitrobenzene in drinking water for 16 weeks. Hematocrit, hemoglobin levels and white cell counts were measured on selected males in each group 5, 10, and 14 weeks after the start of the study. Body weights and food and water consumption were recorded throughout the experiment. At termination

00088443

body and organ weights were measured and all major tissues were examined histopathologically. The concentration of 20 ppm m-dinitrobenzene decreased body weight gain in females, decreased hemoglobin concentrations and caused testicular atrophy in males, and was associated with splenic enlargement and hemosiderin deposits in both sexes of rats. Significantly increased spleen weights were also observed in both sexes of rats treated with 8 ppm. No effects considered to be treatment-related were found at 3 ppm m-dinitrobenzene.

Therefore, the 3 ppm dose is considered a NOAEL for oral exposure and the 8 ppm dose is a LOAEL. Based on water consumption data, Cody et al. (1981) stated that the 3 ppm dose level corresponded to a mean daily m-dinitrobenzene intake of 0.40 mg/kg in males. Therefore, the equivalent NOAEL of 0.40 mg/kg/day, when divided by an uncertainty factor of 3000, equals the RfD of 0.0001 mg/kg/day or 0.009 mg/day for a 70 kg man.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- 10 for extrapolation from subchronic to chronic exposure, 10 for extrapolation across species, 10 for sensitive human subgroups, and 3 for the lack of subchronic and reproductive toxicity data.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

In a behavioral rat study, 10 male rats/group were exposed to 0, 3, or 8 ppm m-dinitrobenzene in drinking water for 90 days. At both m-dinitrobenzene exposure levels, rats showed significantly increased running wheel activity and nonsignificant increases in activity platform counts. Because more traditional measures of toxicity were not affected by administration of 3 ppm m-dinitrobenzene, the change in running wheel activity should not, by itself, be considered an adverse effect (Cody et al., 1981).

Male and female rats were exposed to 50, 100, 200 ppm DNB in drinking water for 8-Weeks. Mortality was high at 200 ppm, hemoglobin concentrations were decreased at doses higher than 50 ppm. At 50 ppm and higher doses increased relative spleen weights and testicular (but not ovarian) atrophy, with accompanying decreased spermatogenesis. At 50 ppm or higher doses (females) and 100 ppm or higher doses (males), hexabarbital sleep time was significantly decreased, indicating acceleration of microsomal metabolism (Cody et al., 1981).

The ACGIH (1986) cited historical studies indicating that dinitrobenzene exposure is associated with methemoglobinemia, liver damage and ready skin absorption. No published reports were found either in man or animals; thus the ACGIH (1986) designated a TLV-TWA of 0.15 ppm (approximately 1 mg/cu.m) for all three dinitrobenzene isomers. They based this value on the comparative acute toxicities of similar polynitro aromatics, relative to the toxicities of mononitro derivatives.

There are currently no data on the reproductive effects of m-dinitrobenzene.

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___I.A.5. CONFIDENCE IN THE ORAL RfD

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

Study confidence was rated medium because both a NOAEL and a LOAEL were defined in subchronic drinking water studies using an adequate number of animals and suitable endpoints. Confidence in the data base is rated low because the only supporting evidence is a subchronic study at much higher concentration levels. Data base confidence would be increased if reproductive and chronic toxicity bioassays were available and if more specific details regarding human exposure were provided. The resulting confidence in the RfD is rated low.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1985

Limited peer review and extensive Agency-wide review, 1985.

Other EPA Documentation -- None

Agency Work Group Review -- 03/19/1987, 04/20/1988

Verification Date -- 04/20/1988

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- m-Dinitrobenzene
CASRN -- 99-65-0

Not available at this time.

00088445

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- m-Dinitrobenzene
CASRN -- 99-65-0
Last Revised -- 02/01/1993

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

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on no data in humans and animals.

___II.A.2. HUMAN CARCINOGENICITY DATA

None.

___II.A.3. ANIMAL CARCINOGENICITY DATA

None.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

00088446

m-Dinitrobenzene was mutagenic in Salmonella typhimurium strains TA98 and TA100 with and without metabolic activation (Spanggord et al., 1982; McGregor et al., 1980; Kawai et al., 1987). Chiu et al. (1978) found m-DNB to be mutagenic in TA98 but not in TA100. Testing was done with only three concentrations and without metabolic activation. m-DNB was mutagenic in TA1538 with and without metabolic activation (Spanggord et al, 1982; McGregor et al., 1980; Garner and Nutman, 1977). Spanggord et al. (1982) found that m-DNB was mutagenic in TA98 and TA100 but was not mutagenic in TA1535, TA1537 and TA100 NR3 (nitroreductase negative strain), with and without metabolic activation. McGregor et al. (1980) found only slight activity in TA1537 and no mutagenicity in TA1535.

Probst et al. (1981) reported that m-DNB did not increase unscheduled DNA synthesis in cultivated hepatocytes at concentrations from 0.5 to 1000 nmoles/mL. m-DNB induced mutations in S. typhimurium strains TA1538, TA98, TA100 and D3052 tested over a 10,000-fold concentration range with and without metabolic activation; the minimum concentration was 0.6 mmoles/mL.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

None.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

None.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1989

The Health Advisory for 1,3-Dinitrobenzene has received Agency and external review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 06/15/1990

Verification Date -- 06/15/1990

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

VI. BIBLIOGRAPHY

Substance Name -- m-Dinitrobenzene
CASRN -- 99-65-0
Last Revised -- 03/01/1991

VI.A. ORAL RfD REFERENCES

ACGIH (American Conference of Government Industrial Hygienists). 1986. Threshold limit values for dinitrobenzenes. ACGIH, Cincinnati, OH. p. 214.

Cody, T.E., S. Witherup, L. Hastings, K. Stemmer and R.T. Christian. 1981. 1,3-Dinitrobenzene: Toxic effects in vivo and in vitro. J. Toxicol. Environ. Health. 7(5): 829-847.

U.S. EPA. 1985. Health and Environmental Effects Profile on Dinitrobenzenes (o-, m-, p-). 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. NTIS PB 88-173638.

VI.B. INHALATION RfC REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Chiu, C.W., L.H. Lee, C.Y. Wang, G.T. Bryan. 1978. Mutagenicity of some commercially available nitro compounds for Salmonella typhimurium. Mutat. Res. 58: 11-22.

00088448

Garner, R. and C.A. Nutman. 1977. Testing of some azo dyes and their reduction products for mutagenicity using Salmonella typhimurium TA 1538. Mutat. Res. 44: 9-19.

Kawai, A., S. Goto, Y. Matsumota and H. Matsushita. 1987. Mutagenicity of aliphatic and aromatic nitro compounds. Japan J. Ind. Health. 29: 34-54.

McGregor, D.B., C.G. Riach, R.M. Hastell and J.C. Dacre. 1980. Genotoxic activity in microorganisms of tetryl, 1,3-dinitrobenzene and 1,3,5-trinitrobenzene. Environ. Mutagen. 2(4): 531-541.

Probst, G.S., R.E. McMahon, L.E. Hill, C.Z. Thompson, J.K. Epp and S.B. Neal. 1981. Chemically-induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 compounds. Environ. Mutagen. 3(1): 11-32.

Spanggord, R.J., K.E. Mortelmans, A.F. Griffin and V.F. Simmon. 1982. Mutagenicity in Salmonella typhimurium and structure-activity relationships of wastewater components emanating from the manufacture of trinitrotoluene. Environ. Mutagen. 4(2): 163-179.

U.S. EPA. 1989. Drinking Water Health Advisory for 1,3-Dinitrobenzene. Office of Drinking Water, Washington, DC. (Draft)

_VII. REVISION HISTORY

Substance Name -- m-Dinitrobenzene
CASRN -- 99-65-0

Date	Section	Description
08/22/1988	I.A.	Oral RfD summary on-line
07/01/1990	II.	Carcinogen assessment now under review
03/01/1991	II.	Carcinogenicity assessment on-line
03/01/1991	VI.	Bibliography on-line
01/01/1992	I.A.7.	Secondary contact changed
01/01/1992	IV.	Regulatory Action section on-line
02/01/1993	II.D.3.	Primary contact changed

VIII. SYNONYMS

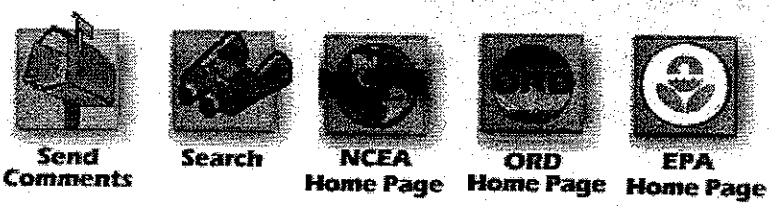
Substance Name -- m-Dinitrobenzene
CASRN -- 99-65-0
Last Revised -- 08/22/1988

99-65-0

00088449

benzene, 1,3-dinitro-
benzene, m-dinitro-
binitrobenzene
1,3-dinitrobenzene
2,4-dinitrobenzene
Dinitrobenzene, m-
1,3-dinitrobenzol
dwunitrobenzen
UN 1597

IRIS Home Page | Substance File List



Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0318.htm

00088450

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

2,4-/2,6-DINITROTOLUENE

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0397
2,4-/2,6-Dinitrotoluene mixture; no CASRN

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR 2,4-/2,6-Dinitrotoluene mixture

File On-Line 09/01/1990

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	09/01/1990

00088451

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- 2,4-/2,6-Dinitrotoluene mixture
CASRN --

Not available at this time.

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- 2,4-/2,6-Dinitrotoluene mixture
CASRN --

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- 2,4-/2,6-Dinitrotoluene mixture
CASRN --
Last Revised -- 09/01/1990

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

00088452

(Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

NOTE: The carcinogenicity assessment for dinitrotoluene mixture includes both 2,4-dinitrotoluene and 2,6-dinitrotoluene.

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Based on multiple benign and malignant tumor types at multiple sites in both sexes of rats (2 strains) and malignant renal tumors in male mice. The classification is supported by evidence of mutagenicity.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Ellis et al. (1979) tested 2,4-DNT (98% 2,4-DNT and 2% 2,6-DNT) in a chronic oral study using Charles River CD (Sprague-Dawley) rats (38/sex/dose) and CD-1 Swiss mice (58/sex/dose) for 2 years. Rats and mice were fed dietary concentrations of 0, 15, 100, and 700 ppm and 0, 100, 700, and 5000 ppm, respectively. Mortality was high in all treatment groups; the control group survival rate at 2 years was only 40-45% in rats and 20-30% in mice. In rats the test chemical induced increased incidences of hepatocellular carcinomas in high-dose males (1/25, 2/28, 2/19, 6/30) and a statistically significant increase in the same tumor type in high-dose females (0/23, 0/35, 1/27, 19/35). The incidence of hepatocellular neoplastic nodules was not considered statistically significantly elevated in any of the rat treatment groups. A statistically significant increase in the incidence of benign mammary gland tumors was observed in high-dose female rats (8/23, 9/35, 16/27, 33/35). Most male mice in the high-dose group died before 12 months and were not included in the incidence. In male mice the incidence of kidney tumors (both benign and malignant) was significantly elevated in the mid-dose group (0/20, 4/21, 15/17 for control, low and medium dose groups). No evidence of treatment-related increases in tumor frequency was noted in female mice.

In a 2-year NCI study (1978), 2,4-DNT (greater than 95% purity) was administered in the diet of Fischer 344 rats (50/sex/dose) and B6C3F1 mice (50/sex/dose) at doses of 80 and 200 ppm (rats) and 80 and 400 ppm (mice). Controls consisted of 75 rats/sex and 50 mice/sex. Rats and mice were on test for 78 weeks followed by an additional observation period of 13 to 26 weeks.

Survival was adequate in all groups, and a reduced body weight gain in high dose groups indicated that an MTD had been approached; this indicates that the study conditions were valid. Only benign tumors were noted. 2,4-DNT induced a statistically significant increase in fibromas of the skin and subcutaneous tissue in male rats (0/71, 7/49, 13/49) and fibroadenomas of the mammary gland in high-dose female rats (13/71, 12/49, 23/50). No statistically significant increase in incidence of tumors was noted in male or female mice.

A CIIT study (1982) treated F344 rats (130/sex/dose) with technical grade DNT (76% 2,4-DNT and 19% 2,6-DNT) at dietary concentrations of 0, 3.5, 10.0 and 35.0 mg/kg/day. All male and female rats in the high-dose group were sacrificed at 55 weeks because of significantly reduced survival. Histopathological studies were performed on sacrificed animals (20 rats/sex) with 100% incidence of hepatocellular carcinoma in male rats (20/20) and 55% incidence in females (11/20). Mid- and low-dose animals were kept on test for 104 weeks. The incidences of liver carcinoma in males at 104 weeks were 1/61 for the control group, 9/70 for the low-dose group, 22/23 for the mid-dose group, and 20/20 (at 55 weeks) for the high-dose group; the incidences in females at 104 weeks were 0/57 for the control group, 0/61 for low-dose group, 40/68 for mid-dose group and 11/20 (at 55 weeks) for the high-dose group. The incidence of neoplastic nodules in males was 9/61, 11/70, 16/23, and 5/20, and the incidence in females was 5/57, 12/61, 53/68, and 12/20, at 104 weeks for the control, low-, mid- and (at 55 weeks) for the high-dose groups, respectively. Cholangiocarcinomas, presumably derived from the bile duct epithelium, were also observed in three high-dose males at 55 weeks and two mid-dose males at 104 weeks.

Leonard et al. (1987) treated groups of 20 F344 male rats with either technical-grade DNT, 2,4-DNT, or 2,6-DNT in the diet for 1 year. There was an untreated control group of 20 rats. Technical DNT (76% 2,4-DNT, 19% 2,6-DNT) (35 mg/kg/day) induced hepatocellular carcinomas in 47% (9/19) of the treated males. 2,6-DNT (99.9% purity) induced hepatocellular carcinomas in 100% (19/19) of the high-dose rats (14 mg/kg/day) and 85% (17/20) of the low-dose (7 mg/kg/day). No tumors were found in controls or rats exposed to 2,4-DNT (99.9 purity) at 27 mg/kg/day. Two low-dose males receiving 2,6-DNT and two males receiving technical DNT developed cholangiocarcinoma. Although the duration of these studies was limited to 1 year and the number of animals tested was small, the data suggest that the 2,6-isomer accounts for much of the carcinogenic activity observed in previous mixed-isomer DNT bioassays.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

The mutagenicity of dinitrotoluenes has been tested in numerous systems. 2,4-DNT causes reverse and forward mutations in several strains of Salmonella typhimurium (Couch et al., 1981; Tokiwa et al., 1981). DNA repair, as measured by UDS, was shown to occur in an in vivo male F344 rat hepatocyte assay (Mirsalis and Butterworth, 1982), but negative results were obtained in in vitro assays in rat hepatocytes (Bermudez et al., 1979) and spermatocytes (Working and Butterworth, 1984). Although Lee et al. (1978) observed an increased frequency of chromosomal aberrations in CD rat lymphocyte and kidney cultures, Ellis et al. (1979) observed no increased frequency in CD rat and beagle dog bone marrow and kidney cultures.

In a series of in vivo tumor initiation-promotion tests, Leonard and coworkers (Popp and Leonard, 1983; Leonard et al., 1983, 1986) compared the development of hepatic foci by the 2,4- and 2,6-DNT isomers and technical DNT. Both 2,6- and technical DNT showed comparable initiating activity in partially-hepatectomized male F344 rats. In a promotion experiment, male F344 rats were initiated with a single dose of diethylnitrosamine prior to feeding 27 mg/kg/day 2,4-DNT or 7 mg/kg/day 2,6-DNT for 12 weeks. Positive results

were observed for both 2,4 and 2,6-DNT, with the 2,6-isomer yielding a stronger response. These findings suggest the 2,6-isomer may be a complete hepatocarcinogen and 2,4-DNT a promoter.

In a skin-painting study using SENCAR mice, 2,6-DNT and 2,4-DNT were given as initiators (1, 5, or 10 mg) followed by TPA application for 30 weeks. Increased incidence of squamous cell carcinoma (5%) was observed in the 2,6-DNT-treated mice, although these results were not statistically significant (Slaga et al., 1985). When given intraperitoneally at 10 mg/kg followed by weekly TPA applications, 2,6-DNT produced 10% incidence of carcinomas, which was not significantly greater than controls. In the lung tumor bioassay, neither 1200 mg/kg of 2,4- nor 4800 mg/kg of 2,6-DNT administered intraperitoneally 3 times a week for 8 weeks increased the incidence of lung tumors in male A/Jax mice (Slaga et al., 1985). Schut et al. (1982), Stoner et al. (1984) and Maronpot et al. (1983) also reported negative results for 2,4-DNT administered orally or ip in the lung tumor bioassay with A/Jax mice, but positive results were reported using female A/St mice (Maronpot et al., 1983).

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 6.8E-1 per (mg/kg)/day

Drinking Water Unit Risk -- 1.9E-5 per (ug/L)

Extrapolation Method -- Linearized multistage procedure

Drinking Water Concentrations at Specified Risk Levels:

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

II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- liver: hepatocellular carcinomas, neoplastic nodules; mammary gland: adenomas, fibroadenomas, fibromas, adenocarcinomas/carcinomas
Test Animals -- rat/Sprague-Dawley, female
Route -- diet
Reference -- Ellis et al., 1979

Administered Dose (ppm)	Human Equivalent Dose (mg/kg)/day	Tumor Incidence
0	0	11/23
15	0.129	12/35
100	0.927	17/27

700 7.557 34/35

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

The tumor incidences could be combined for quantitative purposes because the report by Ellis et al. (1979) provided pathology data for the individual animals. Transformed doses reflect the measured weight of the rats for each treatment period (0.425 kg control and low dose, 0.410 kg medium dose, 0.325 kg high dose).

The U.S. Army (ORNL, 1987) has calculated a quantitative risk estimate for the 2,6-isomer based on Leonard et al. (1987).

The unit risk should not be used if the water concentration exceeds 500 ug/L, since above this concentration the slope factor may differ from that stated.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

Relatively few animals were observed for a period of time approximating the lifespan of the animals. A slope factor of 3.9E-1 per (mg/kg)/day, obtained from renal tumors in male CD-1 mice (Ellis, 1979), is supportive of the risk estimate.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1986, 1987

The values in the 1986 Health and Environmental Effects Profile for Dinitrotoluene have received extensive Agency review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 04/01/1987, 04/22/1987, 05/25/1988, 11/09/1988, 11/30 05/03/1989

Verification Date -- 05/03/1989

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- 2,4-/2,6-Dinitrotoluene mixture
CASRN --
Last Revised -- 08/01/1991

__VI.A. ORAL RfD REFERENCES

None

__VI.B. INHALATION RfD REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Bermudez, E., D. Tillery and B.E. Butterworth. 1979. The effect of 2,4-Diaminotoluene and isomers of dinitrotoluene on unscheduled DNA synthesis in primary rat hepatocytes. Environ. Mutagen. 1: 391-398.

CIIT (Chemical Industry Institute of Toxicology). 1982. 104-Week Chronic Toxicity Study in Rats: Dinitrotoluene. Final Report, Vol. 1 and 2. Docket No. 12362. Research Triangle Park, NC.

Couch, D.B., P.F. Allen and D.J. Abernathy. 1981. The mutagenicity of dinitrotoluenes in Salmonella typhimurium. Mutat. Res. 90: 373-383.

Ellis, H.V. III, J.H. Hagensen, J.R. Hodgson, J.L. Minor and C.B. Hong. 1979. Mammalian Toxicity of Munitions Compounds. Phase III. Effects of Lifetime Exposure. Part I. 2,4-Dinitrotoluene. Report Order No. AD-A077692. p. 281.

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Leonard, T.B., T. Adams and J.A. Popp. 1986. Dinitrotoluene isomer-specific enhancement of the expression of diethylnitrosamine-initiated hepatocyte foci. Carcinogenesis. 7(11): 1797-1803.

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Maronpot, R.R., H.P. Witschi, L.H. Smith and J.L. McCoy. 1983. Recent experience with the strain A mouse pulmonary tumor bioassay model. In: Short-term Bioassays in the Analysis of Complex Environmental Mixtures III. p. 341-349.

Mirsalis, J.C. and B.E. Butterworth. 1982. Induction of unscheduled DNA synthesis in rat hepatocytes following in vivo treatment with dinitrotoluene. Carcinogenesis. 3(3): 241-245.

NCI (National Cancer Institute). 1978. Bioassay of 2,4-dinitrotoluene for possible carcinogenicity. Technical Report Series No. 54. U.S. Dept. Health, Education and Welfare, Washington, DC.

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Schut, H.A.J., T.R. Loeb and G.D. Stoner. 1982. Distribution, elimination and test for carcinogenicity of 2,4-dinitrotoluene in strain A mice. Toxicol. Appl. Pharmacol. 64: 213-220.

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Stoner, G.D., E.A. Greisiger, H.A.J. Schut, M.A. Pereira, T.R. Loeb, J.E. Klaunig and D.G. Branstetter. 1984. A comparision of the lung adenoma response in strain A/J mice after intraperitoneal and oral administration of carcinogens. Toxicol. Appl. Pharmacol. 72: 313-323.

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Washington, DC.

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Working, P.K. and B.E. Butterworth. 1984. An assay to detect chemically induced DNA repair in rat spermatocytes. Environ. Mutagen. 6: 273-286.

_VII. REVISION HISTORY

Substance Name -- 2,4-/2,6-Dinitrotoluene mixture
CASRN --

Date	Section	Description
09/01/1990	II.	Carcinogen assessment on-line
09/01/1990	VI.	Bibliography on-line
08/01/1991	VI.C.	Citations clarified
08/01/1991	I.A.	Oral RfD now under review
09/01/1991	I.A.	Oral RfD will be isomer-specific
01/01/1992	IV.	Regulatory Action section on-line

VIII. SYNONYMS

Substance Name -- 2,4-/2,6-Dinitrotoluene mixture
CASRN --
Last Revised -- 09/01/1990

121-14-2
BENZENE, 1-METHYL-2,4-DINITRO-
2,4-DINITROTOLUENE
2,4-DINITROTOLUOL
2,4-DNT
HSDB 1144
HSDB 2931
1-METHYL-2,4-DINITROBENZENE
NCI-C01865
NSC 7194
RCRA WASTE NUMBER U105
RCRA WASTE NUMBER U106
TOLUENE, 2,4-DINITRO-
606-20-2
2,6-DINITROTOLUENE
2,6-DNT
BENZENE, 2-METHYL-1,3-DINITRO-

00088459

2-METHYL-1,3-DINITROBENZENE
TOLUENE, 2,6-DINITRO-
□

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0397.htm

00088460

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Ethylbenzene

CASRN 100-41-4

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0051
Ethylbenzene; CASRN 100-41-4

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Ethylbenzene

File On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	06/01/1991
Inhalation RfC Assessment (I.B.)	on-line	03/01/1991
Carcinogenicity Assessment (II.)	on-line	08/01/1991

00088461

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Ethylbenzene
CASRN -- 100-41-4
Last Revised -- 06/01/1991

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

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

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

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

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

00088462

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.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- 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.

MF --None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

None.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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

00088463

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.

Agency Work Group Review -- 05/20/1985

Verification Date -- 05/20/1985

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Ethylbenzene
CASRN -- 100-41-4
Last Revised -- 03/01/1991

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is expressed in units of mg/cu.m. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (EPA/600/8-88/066F August 1989) and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994). RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

___I.B.1. INHALATION RfC SUMMARY

Critical Effect	Exposures*	UF	MF	RfC
Developmental toxicity Rat and Rabbit Developmental	NOAEL: 434 mg/cu.m (100 ppm) NOAEL(ADJ): 434 mg/cu.m NOAEL(HEC): 434 mg/cu.m	300	1	1E+0 mg/cu.m

00088464

Inhalation Studies	LOAEL: 4340 mg/cu.m (1000 ppm) LOAEL(ADJ): 4340 mg/cu.m
Andrew et al., 1981; Hardin et al., 1981	LOAEL(HEC): 4340 mg/cu.m

*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.

I.B.2. PRINCIPAL AND SUPPORTING STUDIES (INHALATION RfC)

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

00088465

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).

___I.B.3. UNCERTAINTY AND MODIFYING FACTORS (INHALATION RfC)

UF -- 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.

MF -- None

___I.B.4. ADDITIONAL STUDIES / COMMENTS (INHALATION RfC)

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

00088466

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

00088467

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

00088468

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

00088469

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

00088470

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.

___I.B.5. CONFIDENCE IN THE INHALATION RfC

Study -- Low
Data Base -- Low
RfC -- Low

The 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.

___I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

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

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

Agency Work Group Review -- 09/19/1990, 12/20/1990

Verification Date -- 12/20/1990

___I.B.7. EPA CONTACTS (INHALATION RfC)

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Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Ethylbenzene
CASRN -- 100-41-4
Last Revised -- 08/01/1991

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

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity.

Basis -- nonclassifiable due to lack of animal bioassays and human studies.

___II.A.2. HUMAN CARCINOGENICITY DATA

None.

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II.A.3. 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.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

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.

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

Not available.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

00088473

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1980, 1984, 1987

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.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 10/07/1987

Verification Date -- 10/07/1987

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

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Substance Name -- Ethylbenzene
CASRN -- 100-41-4
Last Revised -- 03/01/1991

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_VII. REVISION HISTORY

Substance Name -- Ethylbenzene
CASRN -- 100-41-4

Date	Section	Description
03/01/1988	I.A.1.	Dose conversion clarified
03/01/1988	I.A.6.	Documentation revised
03/01/1988	III.A.	Health Advisory added
09/07/1988	II.	Carcinogen summary on-line
08/01/1989	VI.	Bibliography on-line
08/01/1990	IV.F.1.	EPA contact changed
10/01/1990	I.B.	Inhalation RfC now under review
03/01/1991	I.B.	Inhalation RfC summary on-line
03/01/1991	VI.B.	Inhalation RfC references added
06/01/1991	I.A.7.	Primary contact changed
08/01/1991	II.D.3.	Secondary contact changed
01/01/1992	I.A.7.	Secondary contact changed
01/01/1992	IV.	Regulatory actions updated

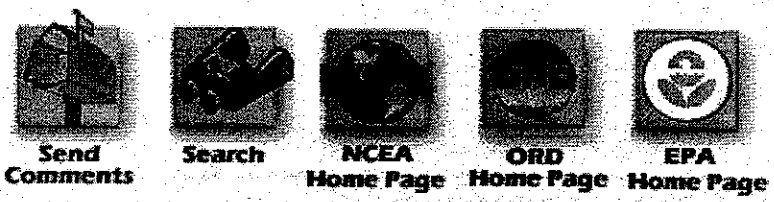
VIII. SYNONYMS

Substance Name -- Ethylbenzene
CASRN -- 100-41-4
Last Revised -- 01/31/1987

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

IRIS Home Page Substance File List

00088477



Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0051.htm

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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetr... (HMX)

CASRN 2691-41-0

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0311
Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetr... (HMX); CASRN 2691-41-0

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR HMX

File On-Line 09/26/1988

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	02/01/1993
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	02/01/1993

00088479

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX)
CASRN -- 2691-41-0
Last Revised -- 02/01/1993

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Hepatic lesions	NOAEL: 50 mg/kg/day	1000	1	5E-2 mg/kg/day
13-Week Rat Feeding Study	LOAEL: 150 mg/kg/day			
U.S. DOD, 1985a				

*Conversion Factors: None

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

U.S. Department of Defense. 1985a. AD-A171 601. Available from Defense Technical Information Center. Write to Documents, Cameron Station, Alexandria, VA 22314, or call (703)274-7633.

The subchronic (13-week) toxicity of HMX has been studied in Fischer 344 rats (U.S. DOD, 1985a). HMX was incorporated into the daily diet of 20 rats/sex/dose for 13 weeks at the following levels: 0, 50, 150, 450, 1350, and 4000 mg/kg/day for males and 0, 50, 115, 270, 620, and 1500 mg/kg/day for females. Three deaths occurred during the course of the study; the study authors considered none to be treatment-related. One male receiving 150 mg/kg/day died during week 9; a female receiving 1500 mg/kg/day died during

00088480

week 1, and a control female died at blood sampling in week 13. No treatment-related clinical signs of toxicity were observed in the HMX-treated animals. All HMX-treated animals exhibited significant ($p<0.001$ to $p<0.05$) dose-related reductions in mean body weight gain with concomitant reductions in food consumption in the early part of the study. By week 5, only animals in the two highest dose groups continued to have significantly depressed weight gains until study termination.

Histopathological examination revealed a significant incidence of toxic liver changes occurring almost exclusively in males at 4000, 1350, and 450 mg/kg/day (the three highest dose levels) and some changes at 150 mg/kg/day. These liver changes were characterized by enlarged centrilobular cells with pale nuclei and dark cytoplasm, dilation of sinusoids, and necrosis. Tubular kidney changes characterized by focal atrophy and dilation were observed almost exclusively in females at the three highest doses. These changes seemed to correlate with some of the clinical pathology changes observed, i.e., increased AP activity and altered indicators of renal function (BUN, albumin, and total protein and urine changes). Thus, it would appear that there are sexual differences in the target organ response of rats to HMX.

The following changes were observed in clinical pathology parameters in the high-dose animals (clinical pathology was not conducted in the lower doses). Hemoglobin and hematocrit were decreased in high-dose males and females at week 5 and in high-dose females at week 12. Red blood cell counts were also significantly decreased in high-dose females at week 12 when compared with controls. There was a significant ($p<0.001$) increase in serum alkaline phosphatase (AP) in high-dose males at 12 weeks and a marginal increase in high-dose females when compared with controls. The level of alkaline phosphatase in male controls was, however, lower than normal at 12 weeks; therefore, the toxicologic importance of the increase is not clear. There was a slight increase in albumin in high-dose males, but the values were within the normal range. Blood urea nitrogen (BUN) was slightly increased in females receiving 1500 mg/kg/day ($p<0.05$ at week 5 and $p<0.001$ at week 12) when compared with controls. There was an increase in urine volume and lowered pH and specific gravity in high-dose females at 12 weeks, but no urinary effects were observed in males.

There were several organ weight changes in dosed animals, although they were difficult to interpret because many reflected the overall decrease in body weight. An increase in absolute brain weight was seen in females receiving all but the lowest dose level, and an increase in brain-to-body weight ratio was increased in males receiving 1350 and 4000 mg/kg/day. Liver-to-body weight ratios, but not absolute liver weights, were increased in females receiving 620 and 1500 mg/kg/day. Kidney weights tended to be decreased in dosed males; the kidney-to-body weight ratios were increased in females at all but the lowest dose level. Adrenal weights and adrenal-to-body weight ratios were decreased in all dosed males; spleen-to-body weight ratios were decreased in all dosed females. It was reported that small changes in spleen, adrenal, testes, and ovary weights were of questionable toxicologic significance.

Based on the results of this study, a NOAEL of 50 mg/kg/day for males and 115 mg/kg/day for females can be estimated, together with a LOAEL of 150 mg/kg/day for toxic liver effects in males and 270 mg/kg/day for toxic renal effects in females.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- The UF of 1000 allows for uncertainty in the extrapolation of dose levels from laboratory animals to humans (10A), uncertainty in the threshold for sensitive humans (10H), and uncertainty in the effect of duration when

00088481

extrapolating from subchronic to chronic exposure (10S).

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

In a subchronic (14 day) study commissioned by the U.S. DOD (1985b) in B6C3F1 mice, five groups of 20 mice/sex/dose were administered HMX in the diet at the following concentrations: 0, 5, 12, 30, 75, and 200 mg/kg/day (males) and 0, 10, 30, 90, 250, and 750 mg/kg/day (females). Mortality in males was 0/20, 0/20, 0/20, 1/20, 2/20, and 13/20 for the 0-, 5-, 12-, 30-, 75-, and 200-mg/kg/day groups, respectively, and mortality in females was 1/20, 0/20, 1/20, 0/20, 12/20, and 20/20 for the 0-, 10-, 30-, 90-, 250-, and 750-mg/kg/day groups, respectively. The deaths that occurred in both sexes at 30 mg/kg/day were not thought to be related to HMX, but no reason was given for this conclusion. Despite the seemingly high mortality rates in the high-dose groups of both sexes, no treatment-related clinical signs of toxicity were noted. Similarly, no significant changes in body weight or clinical chemistry were observed in the treated mice. Except for slight increases in brain weight seen in both males receiving 200 mg/kg/day and females receiving 250 mg/kg/day that are of questionable significance, no remarkable treatment-related changes were noted at necropsy or upon histologic examination. However, only liver, kidney, spleen, and brain were examined in high-dose males and females. Dark red lungs were observed grossly, but were not examined histologically. It is therefore difficult to estimate a NOAEL or LOAEL from this study because of the lack of obvious treatment-related toxicity (except for the high mortality observed at the highest doses).

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

Confidence in the principal study is rated medium because although the study was well-designed, interpretation of some data was difficult and some endpoints were not evaluated at the lower doses. Confidence in the data base is low because of a lack of chronic, reproductive, and other specialized data. Low confidence in the RfD is due to the weakness of the data base.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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

Other EPA Documentation -- U.S. EPA, 1988

Agency Work Group Review -- 05/25/1988

Verification Date -- 05/25/1988

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX)
CASRN -- 2691-41-0

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX)
CASRN -- 2691-41-0
Last Revised -- 02/01/1993

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

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

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___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- No cancer bioassays or epidemiological studies are available.

___II.A.2. HUMAN CARCINOGENICITY DATA

None.

Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine is an explosive polynitramine commonly known as HMX (derived from High Melting Explosive). There are no human studies evaluating carcinogenicity.

___II.A.3. ANIMAL CARCINOGENICITY DATA

There are no lifetime (chronic) bioassays that evaluate carcinogenicity.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Genetic toxicology assays in the literature have been limited to microbial systems. Saturated solutions of HMX, before and after chlorination or ozonation, were not mutagenic for Salmonella typhimurium either with or without hepatic homogenates (S9) (U.S. Army, 1977). However, the concentrations assayed were low due to limited solubility of HMX in water, and the authors conceded that the findings may represent false negatives. The Saccharomyces cerevisiae mitotic gene conversion assay with untreated and with postchlorinated or ozonated samples of saturated HMX was also negative, but the limited solubility of HMX lowers confidence in the studies. Whong et al. (1980) reported that HMX (1.25 and 0.625 mg/spot in the Salmonella spot test) was negative in five strains of Salmonella typhimurium (TA1535, TA1537, TA1538, TA98, and TA100). In a plate incorporation assay with S9 activation, a negative response with all five Salmonella strains was obtained up to 2.5 mg/plate. However, the actual data on HMX were not reported in the Whong et al. (1980) paper.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

Not available.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1988

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 09/22/1988

Verification Date -- 09/22/1988

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX)
CASRN -- 2691-41-0
Last Revised -- 12/01/1990

__VI.A. ORAL RfD REFERENCES

U.S. Department of Defense. 1985a. AD-A171 601. Available from Defense Technical Information Center. Write to Documents, Cameron Station,

00088485

Alexandria, VA 22314, or call (703)274-7633.

U.S. Department of Defense. 1985b. AD-A171 602. Available from Defense Technical Information Center. Write to Documents, Cameron Station, Alexandria, VA 22314, or call (703)274-7633.

U.S. EPA. 1988. Drinking Water Health Advisory for Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX). Office of Drinking Water, Washington, DC.

__VI.B. INHALATION RfC REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

U.S. EPA. 1988. Health Advisory on Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX). Office of Drinking Water, Washington, DC.

U.S. Army Medical Research and Development Command. 1977. DAMD 17-76-C-6013. Ft. Deitrick, Frederick, MD 21701.

Whong, W.Z., N.D. Speciner and G.S. Edwards. 1980. Mutagenic activity of tetryl, a nitroaromatic explosive, in three microbial test systems. Toxicol. Lett. 5: 11-17.

_VII. REVISION HISTORY

Substance Name -- Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX)
CASRN -- 2691-41-0

Date	Section	Description
09/26/1988	I.A.	Oral RfD summary on-line
03/01/1989	II.	Carcinogen summary on-line
04/01/1989	II.A.4.	Correct citation
04/01/1989	II.D.1.	Correct citation
08/01/1990	I.A.	Text edited
08/01/1990	I.A.7.	Primary contact changed
08/01/1990	II.	Text edited
08/01/1990	II.D.3.	Primary contact changed
12/01/1990	I.A.6.	Source document statement added

00088486

12/01/1990	III.A.	Health Advisory on-line
12/01/1990	VI.	Bibliography on-line
02/01/1993	I.A.7.	Primary contact changed
02/01/1993	II.D.3.	Primary contact changed
02/01/1993	III.A.10	Primary contact changed

VIII. SYNONYMS

Substance Name -- Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine (HMX)
CASRN -- 2691-41-0
Last Revised -- 09/26/1988

2691-41-0
beta HMY
cyclotetramethylenetetranitramine
HMX
HW 4
LX 14-0
Octahydro-1,3,5,7-tetranitro-1,3,5,7-tetrazocine
octogen
oktogen
tetramethylenetetranitramine
1,3,5,7-tetrazocine, octahydro-1,3,5,7-tetranitro-
UN 0226

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0311.htm

United States Environmental Protection Agency

IRIS
Integrated Risk Information System

Methyl ethyl ketone (MEK)
CASRN 78-93-3

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

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Methyl ethyl ketone (MEK); CASRN 78-93-3

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR MEK

File On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	05/01/1993
Inhalation RfC Assessment (I.B.)	on-line	07/01/1992
Carcinogenicity Assessment (II.)	on-line	12/01/1989

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Methyl ethyl ketone (MEK)
CASRN -- 78-93-3
Last Revised -- 05/01/1993

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Decreased fetal birth weight	NOAEL: 1771 mg/kg-day (1% 2-butanol solution)	3000	1	6E-1 mg/kg-day
Multigeneration/ Developmental Rat Feeding Study	LOAEL: 3122 mg/kg-day (2% 2-butanol solution)			
Cox et al., 1975				

*Conversion Factors and Assumptions: Based on actual consumption data for the NOAEL and regression analysis for the LOAEL of exposure to 2-butanol, a metabolic intermediate of methyl ethyl ketone.

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Cox, G.E., D.E. Bailey and K. Morgareidge. 1975. Toxicity studies in rats with 2-butanol including growth, reproduction and teratologic observations. Food and Drug Research Laboratories, Inc., Waverly, NY, Report No. 91MR R 1673.

The identification of the critical effect for methyl ethyl ketone (MEK), also referred to as 2-butanone, is based on its metabolic intermediate, 2-

butanol. A detailed rationale for use of this intermediate of MEK can be found in Additional Comments/Studies.

Weanling FDRL-Wistar stock rats (30/sex/group) were given 2-butanol in drinking water at 0, 0.3, 1.0, or 3.0% solutions and a standard laboratory ration ad libitum. Weekly feed, fluid intakes and body weights were recorded to determine the efficiency of food utilization and to calculate the average daily intake of 2-butanol. The average daily intake of 2-butanol for males was 0, 538, 1644, and 5089 mg/kg-day and for females was 0, 594, 1771, and 4571 mg/kg-day for the 0, 0.3, 1.0, and 3.0% solutions, respectively. At the highest exposure level (3.0%), net weight gain was reduced compared with controls both in males (229 g vs. 269 g in controls) and females (130 g vs. 154 g in controls) during the 8 weeks of initial monitoring. However, no differences were found in the efficiency of food utilization.

After 9 weeks of exposure, parental matings were made with one male and one female from each of the respective treatment groups (P generation). Following birth of the first litter (F1A) of the parental generation, various reproduction and lactation responses were measured. This study design incorporated a multigeneration protocol with measurement of developmental toxicity endpoints. Significant effects were noted in the litters from the 3.0% 2-butanol dose group vs. the control group, including the number of pups/litter cast alive (8.46 vs. 10.3), the number of pups/litter alive at 4 days before culling (8.12 vs. 10.3), the number of pups/litter alive at 21 days (6.85 vs. 7.68), the mean body weight/pup after culling at 4 days (8.2 g vs. 10.3 g) and the mean body weight/pup at 21 days (28.4 g vs. 49.5 g).

Based on results found in the 3.0% (high-dose) 2-butanol F1A generation, the treatment of all high-dose parents and offspring was reduced to 2.0% 2-butanol for the remainder of the experimental protocol. Following a 2-week interim adaptation period to allow the F1A generation animals to attain a more normal weight, the P generation was subsequently remated to produce a second litter (F1B), and the F1A generation selected for an F2 mating. Therefore, a new high dose of 2.0% 2-butanol was calculated to be equivalent to 3384 mg/kg-day in males and 3122 mg/kg-day in females based on regression analysis of the 8 week water intake data. The F1B litters receiving 2.0% 2-butanol showed a slight reduction in average fetal weight compared with controls (3.74 g vs. 4.14 g in controls). Nidation, early fetal deaths, and late fetal deaths were not detectably affected. Skeletal findings also were reported for the F1B generation. The 2.0% group showed apparent increases in missing sternebrae, wavy ribs, and incomplete vertebrae ossification when compared with both the 0.3 and 1.0% groups. However, because of the rather high incidence in the control group for these findings, these effects could not be determined to be compound-related. The 2.0% group also showed a reduction in the mean body weight per pup at day 4 following culling (9.48 g vs. 10.0 g in the control) and in the mean body weight at 21 days (34.9 vs. 41.1 g in the control). Although these reductions were not as great in the high dose (3.0%) F1A generation, the percent body weight reduction in the F1B high dose (2.0%) was doubled at 21 days (reductions of 5% at 4 days and 11% at 21 days). This generation showed the same doubling trend in fetal weight reduction as was reported in the F1A group exposed to 3.0% 2-butanol throughout (reductions of 21% at 4 days and 43% at 21 days). These results are also presented in abstract form by Gallo et al. (1977).

At the 2.0% level of the F2 generation, there were a number of histopathologic changes noted in the kidney of the male rats only. These changes were characterized by nonreactive tubular degeneration in the outer medullary zone, tubular cast formation, foci of tubular degeneration and regeneration, microcysts in the renal papilla, glomerular fibrosis, and focal epithelial calcification. These findings are consistent with the pattern of early stages of alpha 2u-globulin-associated rat nephrotoxicity as set forth by the Risk Assessment Forum (U.S. EPA, 1991). As per the Agency's guidance, it is not appropriate to use these specie-and sex-specific renal effects to establish a critical effect.

Administration of 2-butanol resulted in the occurrence of developmental effects. Decreased pup survivability and fetal weight were seen in the 3.0% group, and in the F1 offspring (F1A and F1B, whose parents received 3.05 and 2.0% 2-butanol, respectively). The decrease in fetal weight continued to be demonstrated in the F2 generation at the 2.0% level. Therefore, based on these developmental toxicologic endpoints, a LOAEL of 3122 mg/kg-day (2.0% solution) and a NOAEL of 1771 mg/kg-day (1.0% solution) are identified.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- Four uncertainty factors of 10 each were applied, one to account for interspecies extrapolation and intraspecies variability (extrapolation to sensitive human populations); one to adjust for subchronic to chronic extrapolation since long-term effects in the dams during the exposure period were not reported in the principal study; one for incompleteness of the data base, including a lack of both subchronic and chronic oral exposure studies for MEK; and one to account for the absence of data for a second rodent specie for either MEK or 2-butanol. As is usual practice, the application of four full areas of uncertainty generally results in a total uncertainty factor of 3000, given the interrelationship among and overlap between the various areas of uncertainty described above.

MF -- None

___I.A.4. ADDITIONAL STUDIES / COMMENTS (ORAL RfD)

Data on toxic effects in humans or laboratory animals following oral exposure to MEK are restricted to a limited number of acute studies. Oral LD50 values for MEK include 5522 and 2737 mg/kg in rats (Smyth et al., 1962 and Kimura et al., 1971, respectively) and 4,044 mg/kg in mice (Tanii et al., 1986). Single gavage doses of 15 mmol/kg MEK in corn oil (1082 mg/kg) produced no deaths or histological alterations in the livers of male Fischer 344 rats, but produced tubular necrosis in kidneys (Brown and Hewitt, 1984). Pre-administration of single gavage doses of MEK (or other ketonic solvents) enhanced the liver and kidney damage produced by a 0.5 mL/kg intraperitoneal dose of carbon tetrachloride (Brown and Hewitt, 1984). This MEK potentiation of carbon tetrachloride hepatotoxicity was also observed in similar experiments with male Sprague-Dawley rats (Dietz and Traiger, 1979).

At present there are insufficient oral exposure data of MEK from which to derive an oral RfD. Availability of oral exposure data of a metabolic intermediate of MEK, i.e., 2-butanol, was used to derive the RfD of MEK. The following is an explanation of the rationale for using the oral data of 2-butanol rather than attempting a route-to-route extrapolation of the available inhalation data for MEK.

Traiger and Bruckner (1976) have estimated that approximately 96% of an administered dose of 2-butanol is oxidized in vivo to MEK. The data from the Dietz et al. (1981) study support this estimation. Administration of 1776 mg/kg 2-butanol by gavage shows peak blood 2-butanol concentrations (0.59 mg/mL) within 2 hours; the compound is barely detectable after 16 hours. As the blood concentrations of 2-butanol fall, the peak concentrations of MEK (0.78 mg/mL) and 2,3-butanediol (0.21 mg/mL) occurred at 8 and 18 hours respectively. Ultimately, 2-butanol and MEK are metabolized through the same intermediates.

Additional metabolism of the ketone occurs by oxidation to hydroxylated intermediates such as 2,3-butanediol, all of which can be eliminated in the urine (DiVincenzo et al., 1977). DiVincenzo et al. (1976) have previously identified MEK, 2-butanol, and other oxidative metabolites of MEK in the serum of guinea pigs following administration of MEK.

A pharmacokinetic model was presented by Dietz et al. (1981) to describe the biotransformation of 2-butanol and MEK in rats when given a dose calculated to produce an equivalent calculated adjusted urinary concentration. When MEK is administered by gavage at a dose of 1690 mg/kg, the detection of the formation of 2-butanol and the oxidative metabolites corresponds to that previously reported in the guinea pig. By 18 hours post administration, MEK is barely detectable in the blood (peak concentration of 0.95 mg/mL at 4 hours), but a peak blood concentration (0.26 mg/mL) of the oxidative metabolite 2,3-butanediol can be detected. Peak blood concentrations of 2-butanol (0.033 mg/mL) occurred after 6 hours.

Although it is not known if the effects reported by Cox et al. (1975) are due to either 2-butanol directly or indirectly through another common metabolite, the weight of evidence of the available data argues for using the butanol data as a surrogate approach to the development of the RfD for MEK. It is assumed in this surrogate approach that 2-butanol is not the active metabolite causing the effects reported by Cox, given the profile of metabolism for both 2-butanol and MEK. Therefore, it is appropriate to utilize data from the oral administration of 2-butanol in order to derive an RfD for MEK in lieu of appropriate oral data for MEK.

Decreased fetal body weight was also reported in pregnant mice exposed by inhalation to 0, 398, 1010, or 3020 ppm MEK (0, 1174, 2978, or 8906 mg/cu.m, respectively, assuming 25 C and 760 mm Hg) 7 hours/day during gestational days 6-15 (Schwetz et al., 1991; Mast et al., 1989). The only maternal effect observed was a concentration-related increase in relative liver and kidney weight. The difference was statistically significant in the dams exposed to 3020 ppm MEK. The biologic significance of this increase has not been ascertained. The decrease in fetal body weight was observed in the 3020-ppm group, however the difference was statistically significant only in the males. There was a statistically significant ($p<0.05$) trend with increasing concentration in the incidence of fetuses with misaligned sternebrae, but this trend was not apparent in the incidence of litters with misaligned sternebrae. Although increased incidence of fetuses and litters with malformations were observed in exposed groups compared with controls, the increases were not statistically significant. The observed fetal malformations (cleft palate, fused ribs, missing vertebrae and syndactyly) present in the exposed groups were not observed in concurrent or contemporary control groups of the same strain of mice. The Schwetz et al. (1991) study identified a NOAEL of 1126 ppm and a LOAEL of 3020 ppm for the occurrence of significant, developmental effects in mice including a decrease in fetal body weight, thereby corroborating the effects observed by Cox et al. (1975).

Sprague-Dawley rats were exposed to 0, 1126, or 2618 ppm MEK (0, 3320, or 7720 mg/cu.m, respectively, assuming 25 C and 760 mm Hg) 7 hours/day during gestational days 6-15 (Schwetz et al., 1974). The following endpoints were used to assess exposure-related effects: maternal (body weight, food intake, liver weight, SGOT levels, number of implantations/litter size) and fetal (examination for anomalies, incidence of fetal resorptions, fetal body measurements). No maternal effects or effect on the incidence of fetal resorptions were observed. A decrease in fetal body weight and crown-rump length were observed in the 1126 ppm offspring, however, these effects were not observed in the offspring of the rats exposed to 2618 ppm. There were no gross, soft tissue, or specific skeletal anomalies that occurred at a significantly increased incidence among litters of dams exposed to 1126 ppm MEK. However, the total number of litters containing fetuses with anomalous skeletons was significantly increased compared with controls. In the fetuses

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of the 2618-ppm group, there was a significantly increased number of fetuses and litters having gross anomalies (two acaudate fetuses with an imperforate anus and 2 brachygnathous fetuses). No single soft tissue anomaly occurred at a statistically significant increased incidence, but the total number of litters containing fetuses with soft tissue anomalies was significantly greater than in controls.

Groups of 25 pregnant Sprague-Dawley rats were exposed to 0, 412, 1002, or 3005 ppm MEK (0, 1215, 2956, or 8861 mg/cu.m, respectively, assuming 25 C and 760 mm Hg) 7 hours/day during gestational days 6-15 (Deacon et al., 1981). Decreased maternal body weight gain and increased water consumption were observed in the 3005 ppm group. No other maternal effects were noted. No statistically significant differences in the incidence of external or soft-tissue alterations were observed among exposed fetuses. A significant decrease in the incidence of delayed ossification of interparietal bones of the skull and an increase in the incidence of extra lumbar ribs and in the occurrence of delayed ossification of cervical centra were noted at the 3005-ppm exposure level.

Data for humans and rats support the assumption that approximately 50% of inhaled MEK is absorbed. From measurements of environmental and exhaled air concentrations, Perbellini et al. (1984) calculated pulmonary retentions of about 70% in workers exposed to concentrations less than 300 ppm for 4 hours (most of the measurements were made at environmental concentrations at or below 100 ppm). From similar measurements, Liira et al. (1988) calculated a pulmonary retention of about 53% for male human volunteers exposed to 200 ppm for 4 hours. Kessler et al. (1988) reported a pulmonary retention of 40% for rats exposed to concentrations less than or equal to 180 ppm for up to about 14 hours.

Although a route-to-route extrapolation could be attempted from the inhalation data, a number of assumptions would need to be made with regard to the inhalation and oral absorption efficiencies making a route-to-route extrapolation limited at best. The developmental effects of MEK inhalation exposure are consistent with the developmental effects seen following oral exposure to 2-butanol and therefore make such an exercise plausible. However, the calculation of an inhalation absorption efficiency for MEK is problematic. The pulmonary retention values discussed above are calculated for exposures of 200 ppm (Liira et al., 1988) and 300 ppm (Perbellini et al., 1984) for a limited 4-hour exposure in humans. The pulmonary retention data of Kessler et al. (1988) using rats, although for a longer period of time (14 hours), was at a maximum concentration of 180 ppm. The calculation of the 40% pulmonary retention value is dependent upon a low concentration that is not limited by metabolic capacity but by transport to the metabolizing enzymes. This retention value, therefore, is valid only if related to normal physiologic processes. Inhalation effects of MEK are seen at concentrations an order of magnitude greater than was used to calculate the rat pulmonary retention value. The toxicity of MEK may be a result of challenges made to a saturable enzymatic detoxification mechanism. Therefore, it cannot be assumed that the pulmonary retention value will be the same at exposures of less than 180 ppm or at greater than 1800 ppm. For this reason, it is inappropriate to estimate the pulmonary retention value at these effect levels, thereby precluding derivation of an oral RfD based upon extrapolation from inhalation effects.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

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Confidence in the principal study is low. The multigeneration/ developmental study for 2-butanol defined a critical effect that is corroborated by inhalation exposure data. Although the study employed an adequate number of animals and examined appropriate endpoints, lowering the high-dose group from 3.0 to 2.0% confounded determination of the critical effect. Confidence in the data base is low. This RfD is based on a multigeneration developmental study for a compound that is nearly completely converted to MEK in a short period of time. This compound exhibits similar developmental effects as seen by inhalation exposure to MEK. However, the lack of oral data for MEK itself and the absence of data in a second specie precludes any higher level for data base confidence. This assessment for MEK is based upon the strength of data supporting the use of the 2-butanol multigeneration study and the concurrence of developmental effects for inhalation exposure to MEK and assumes that 2-butanol was not responsible for the fetal toxicity. There is a lack of data on the metabolism of 2-butanol and MEK over time thereby decreasing the confidence in the use of this surrogate approach. Therefore, confidence in the RfD is low.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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

Other EPA Documentation -- U.S. EPA, 1984, 1985

Agency Work Group Review -- 06/24/1985, 07/08/1985, 05/16/1990, 07/17/1991, 09/23 11/05/1992

Verification Date -- 11/05/1992

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Methyl ethyl ketone (MEK)
CASRN -- 78-93-3
Last Revised -- 07/01/1992

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is expressed in units of mg/cu.m. In general, the RfC is an estimate (with uncertainty

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spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (EPA/600/8-88/066F August 1989) and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994). RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

___I.B.1. INHALATION RfC SUMMARY

Critical Effect	Exposures*	UF	MF	RfC
Decreased fetal birth weight Mouse Developmental Study Schwetz et al., 1991; Mast et al., 1989	NOAEL: 2978 mg/cu.m (1010 ppm, nominal) NOAEL(ADJ): 2978 mg/cu.m NOAEL(HEC): 2978 mg/cu.m LOAEL: 8906 mg/cu.m (3020 ppm, nominal) LOAEL(ADJ): 8906 mg/cu.m LOAEL(HEC): 8906 mg/cu.m	1000	3	1E+0 mg/cu.m

*Conversion Factors: MW = 72.1 Assuming 25 C and 760 mm Hg. NOAEL (mg/cu.m) = 1010 ppm x 72.1/24.45 = 2978 mg/cu.m. Currently, the approach for dose-response analysis of developmental endpoints does not duration-adjust exposure concentrations. NOAEL(ADJ) = NOAEL = 2978 mg/cu.m. The NOAEL(HEC) was calculated for a gas:extrarespiratory effect assuming periodicity was attained. Since the b:a lambda values are unknown for the experimental animal species (a) and human (h), a default value of 1.0 is used for this ratio. NOAEL(HEC) = NOAEL(ADJ) x (b:a lambda(a)/b:a lambda(h)) = 2978 mg/cu.m.

___I.B.2. PRINCIPAL AND SUPPORTING STUDIES (INHALATION RfC)

Schwetz, B.A., T.J. Mast, R.J. Weigel, J.A. Dill and R.E. Morrissey. 1991. Developmental toxicity of inhaled methyl ethyl ketone in mice. Fund. Appl. Toxicol. 16: 742-748.

Mast, T.J., J.A. Dill, J.J. Evanoff, R.L. Rommereim, R.J. Weigel and R.B. Westerberg. 1989. Inhalation developmental toxicology studies: Teratology study of methyl ethyl ketone in mice. Final Report. Prepared by Pacific Northwest Laboratory, Battelle Memorial Institute, for the National Toxicology Program, Washington, DC. PNL-6833 UC-408.

Pregnant Swiss mice were exposed to 0, 398, 1010 or 3020 ppm methyl ethyl ketone (0, 1174, 2978 or 8906 mg/cu.m, respectively, assuming 25 C and 760 mm Hg) 7 hours/day during gestational days 6-15. The Schwetz et al. (1991) and the Mast et al. (1989) reports are the same developmental mouse study presented in different formats and are hence considered as one single study. The number of dams exposed to methyl ethyl ketone ranged from 23-28 mice depending upon exposure group. The dams were killed on gestational day 18. The only maternal effect observed was a concentration-related increase in relative liver and kidney weight. The difference was statistically

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significant only in the dams exposed to 3020 ppm methyl ethyl ketone. The biological significance of this increase has not been ascertained. A decrease in fetal body weight was also observed in the 3020 ppm exposed group; however, the difference was significant only in the males. There was a significant ($p<0.05$) trend in the incidence of misaligned sternebrae present in fetuses but not reflected in a similar analysis of litters of the animals exposed to 3020 ppm. Additionally, although no significant increase of any single malformation was found, there were several malformations (cleft palate, fused ribs, missing vertebrae and syndactyly) present at low incidences in exposed groups.

Neither maternal nor developmental toxicity was observed at exposures at or less than 1010 ppm (2978 mg/cu.m). At 3020 ppm (8906 mg/cu.m), an equivocal maternal effect was reported; however, mild developmental effects (decreased fetal body weight and misaligned sternebrae) were found. Based on the absence of both maternal and developmental toxic effects, a NOAEL of 1126 ppm (HEC=2978 mg/cu.m) is established. The LOAEL is established at 3020 ppm (HEC=8906 mg/cu.m) based on the appearance of mild, but significant developmental effects.

Groups of 21-23 pregnant Sprague-Dawley rats were exposed to 0, 1126 or 2618 ppm methyl ethyl ketone (0, 3320 or 7720 mg/cu.m, respectively, assuming 25 C and 760 mm Hg) 7 hours/day during gestational days 6-15 (Schwetz et al., 1974). The following endpoints were used to assess exposure-related effects: maternal body weight, food intake, liver weight, SGOT levels, and number of implantations/litter size; and fetal anomalies, incidence of resorptions, and fetal body measurements.

No maternal effects or effects on the incidence of fetal resorptions were observed. A decrease in fetal body weight and crown-to-rump length was observed in the offspring exposed to 1126 ppm; however, these effects were not observed in the offspring of the rats exposed to 2618 ppm There were no gross, soft tissue or specific skeletal anomalies that occurred at a significantly increased incidence among litters of dams exposed to 1126 ppm methyl ethyl ketone. However, the total number of litters containing fetuses with anomalous skeletons was increased significantly compared to controls. In the fetuses exposed to 2618 ppm methyl ethyl ketone, there was a significantly increased number of fetuses and litters having gross anomalies (two acaudate fetuses with an imperforate anus and two brachygnathous fetuses) compared to the incidence among controls. No single soft tissue anomaly occurred at a statistically significant increased incidence, but the total number of litters containing fetuses with soft tissue anomalies was significantly greater than in controls.

The study by Deacon et al. (1981) was conducted to determine the repeatability of the fetotoxic effects observed in the Schwetz et al. (1974) study. Groups of 25 pregnant Sprague-Dawley rats were exposed to 0, 412, 1002 or 3005 ppm methyl ethyl ketone (0, 1215, 2955 or 8861 mg/cu.m, respectively, assuming 25 C and 760 mm Hg) 7 hours/day during gestational days 6-15. Decreased maternal body weight gain and increased water consumption were observed in the group exposed to 3005 ppm. No other maternal effects were noted. No statistically significant differences in the incidence of external or soft-tissue alterations were observed among exposed fetuses. A significant decrease in the incidence of delayed ossification of interparietal bones of the skull and an increase in the incidence of extra lumbar ribs and in the occurrence of delayed ossification of cervical centra were noted at the 3005 ppm exposure level.

I.B.3. UNCERTAINTY AND MODIFYING FACTORS (INHALATION RfC)

UF -- An uncertainty factor of 1000 reflects factors of 10 to account for interspecies extrapolation, sensitive individuals, and incomplete data base including a lack of chronic and reproductive toxicity studies.

MF -- A modifying factor of 3 was used to address the lack of unequivocal data for respiratory tract (portal-of-entry) effects.

___I.B.4. ADDITIONAL STUDIES/COMMENTS (INHALATION RfC)

Slight nose and throat irritation was observed in 10 human volunteers exposed to 100 ppm methyl ethyl ketone (HEC=295 mg/cu.m) for 5 minutes. Exposure to 300 ppm (HEC=885 mg/cu.m) was judged to be intolerable by the subjects (Nelson et al., 1943).

Male and female volunteers (12-13/sex) were exposed to 186 ppm (HEC=549 mg/cu.m, assuming 25 C and 760 mm Hg) for 4 hours. No statistically significant changes in neurobehavioral performance were observed (Dick et al., 1988).

Groups of male and female Fischer 344 rats (n=15) were exposed to 0, 1254, 2518 or 5041 ppm methyl ethyl ketone (0, 3698, 7425 or 14,865 mg/cu.m, respectively, assuming 25 C and 760 mm Hg) 6 hours/day, 5 days/week for 90 days (Cavender et al., 1983; Toxigenics, 1981). For each exposure group, 10 animals were used for general histopathology and 5 animals for neuropathologic examination. Tissues examined included nasal cavity, trachea and bronchi, lung, liver and kidney. The prevalence of chronic respiratory disease in the pulmonary region was 100% across control and all exposure groups. There were also high prevalences of nasal inflammation, including submucosal lymphocytic infiltration and lumenal exudate, across control and all exposure groups. These latter lesions, however, varied slightly across the nasal cavity section examined and with exposure level tested. Although the authors suggest that the pulmonary lesions are secondary to mycoplasma infection, no infectious agent was cultured. In other investigations utilizing a model for Mycoplasma pulmonis, pulmonary lesions showed a concentration-response indicative of upper airway irritation (Broderson et al., 1976). The high prevalences of both the nasal and pulmonary lesions in this study preclude unequivocal interpretation regarding respiratory effects. No histopathologic alterations of other major tissues or of neuropathology were observed. An increase in absolute and relative liver weight and relative kidney weight was observed in the animals of both sexes exposed to 5041 ppm. An increase in serum alkaline phosphatase levels was also observed in the female rats exposed to 5041 ppm methyl ethyl ketone. At the highest concentration, females were found to have decreased absolute and increased relative brain weight, however, no pathological changes were observed in the medulla oblongata or the sciatic and tibial nerves. No functional tests (pulmonary or neurologic) were performed in these studies. Based on toxicity remote to the respiratory tract described above, this study provisionally identifies a NOAEL of 2518 ppm (HEC=1326 mg/cu.m) and a LOAEL of 5041 ppm (HEC=2654 mg/cu.m).

Groups of 5 male Wistar rats were exposed to 10,000 ppm methyl ethyl ketone (29,489 mg/cu.m, assuming 25 C and 760 mm Hg) 8 hours/day, 7 days/week for 15 weeks. Because of severe upper respiratory tract irritation, the concentration was lowered to 6000 ppm (17,693 mg/cu.m). The exposed rats died after 7 weeks of exposure; the cause of death was bronchopneumonia. Severe hindlimb weakness was observed in all animals given a methyl ethyl ketone/n-hexane mixture by the 9th week of exposure, which progressed to severe paresis. These effects were observed again when the experiment was repeated. However, these effects were not observed when methyl ethyl ketone was administered alone (Altenkirch et al., 1978).

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A group of 25 adult rats (strain not given) was exposed to 235 ppm (693 mg/cu.m) 7 hours/day, 5 days/week for 12 weeks (LaBelle and Brieger, 1955). The dose duration-adjusted concentration is equivalent to 144 mg/cu.m. A control group was included, but the number of animals was not given. At the end of the study, 15 rats were examined microscopically (organs not given) and hematologically (hemoglobin, erythrocyte, leukocyte, neutrophil, lymphocyte, and monocyte counts). The remaining 10 rats were reserved for growth studies. No adverse effects were observed, but only a few critical parameters were measured for a single exposure concentration. Similarly, 15 guinea pigs were exposed to methyl ethyl ketone at the same concentration as were the rats. No adverse effects were reported for the exposed guinea pigs. However, this information is only presented qualitatively in the report. Furthermore, it was reported that the guinea pigs experienced a vitamin deficiency possibly contributing to two deaths during the exposure period.

Male Wistar rats (8/group) were exposed to 0 or 200 ppm methyl ethyl ketone (0 or 590 mg/cu.m, assuming 25 C and 760 mm Hg) 12 hours/day, 7 days/week for 24 weeks. A slight increase in motor nerve conduction velocity and mixed nerve conduction velocity and a decrease in distal motor latency were observed at 4 weeks of exposure. However, no difference was observed after 4 weeks [NOEL(HEC)=295 mg/cu.m] (Takeuchi et al., 1983).

Groups of 12 Sprague-Dawley rats were continuously exposed to 1125 ppm methyl ethyl ketone (3318 mg/cu.m, assuming 25 C and 760 mm Hg) for 16-55 days. Pathological examination of the sciatic nerve and intrinsic foot muscles revealed no peripheral neuropathy [NOEL(HEC)=3318 mg/cu.m] (Saida et al., 1976).

Four baboons were exposed to 100 ppm methyl ethyl ketone (295 mg/cu.m, assuming 25 C and 760 mm Hg) continuously for 7 days. Operant conditioning behavior conducted during exposure was compared to pre-exposure test scores. The operant behavior selected was a match-to-sample discrimination task and the experimental protocol allowed the performance of each baboon during exposure to be compared to his performances during a clean air exposure in the same chamber immediately prior to each exposure. There was no significant effect on accuracy; however, there was a decrease in mean response time and response during delay (Geller et al., 1979). The authors (Geller et al., 1979) suggested that this could be an early manifestation of the incoordination and narcosis observed in guinea pigs after acute exposure to 10,000 ppm methyl ethyl ketone (29,489 mg/cu.m, assuming 25 C and 760 mm Hg) (Patty et al., 1935). However, it should be pointed out that each baboon in this study was exposed to four different chemicals of which methyl ethyl ketone was given second.

Swiss mice were exposed to various levels of methyl ethyl ketone. A decrease in the immobility developed in a behavioral despair swimming test was observed. A 50% decrease in immobility (ID50) was observed at 2065 ppm (6089 mg/cu.m, assuming 25 C and 760 mm Hg) (DeCeaurriz et al., 1983).

Although the role of methyl ethyl ketone in potentiating peripheral neuropathies caused by potent neurotoxins such as hexane and methyl n-butylketone is documented (Altenkirch et al., 1978; Saida et al., 1976), current experimental data indicate that, by itself, methyl ethyl ketone has little if any neurotoxic potential. Several studies examining neurotoxicity of methyl ethyl ketone have employed neurohistopathology to evaluate toxicity. No peripheral neurohistopathological changes were noted in rats exposed continuously to 1125 ppm methyl ethyl ketone for up to 5 months (Saida et al., 1976). No treatment-related central or peripheral neurohistopathology was observed in rats exposed for 90 days (6 hours/day, 5 days/week) to methyl ethyl ketone concentrations up to 5041 ppm (Toxigenics, 1981; also reported in Cavender et al., 1983). In a study combining experiments of methyl ethyl ketone and n-hexane, Altenkirch et al. (1978) reported that all 10 male rats exposed to 6000 ppm methyl ethyl ketone (8 hours/day, 7 days/week) died in the 7th week without neurological symptoms or pathology although central and

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extensive peripheral neurohistopathology was observed in animals concurrently exposed to n-hexane. The authors also reported that animals in all exposure groups (both methyl ethyl ketone and hexane) were somnolent during exposure. A few studies have attempted to examine neurological deficits through functional testing in addition to histopathology. Takeuchi et al. (1983) reported an alteration in nerve conduction velocities but only in the 4th week of a 24-week exposure to 200 ppm methyl ethyl ketone; the alteration was not corroborated by histopathology and was in the opposite direction to that predicted by peripheral neurotoxins such as n-hexane. Results from other functional-deficit studies in humans (Dick et al., 1988) have been negative and in baboons (Geller et al., 1979), inconclusive, perhaps due to experimental design problems. At present, there is no convincing experimental evidence that methyl ethyl ketone, by itself, is neurotoxic to either experimental animals or humans other than possibly inducing CNS depression at high exposure levels.

___I.B.5. CONFIDENCE IN THE INHALATION RfC

Study -- Medium
Data Base -- Low
RfC -- Low

Confidence in the principal study is medium. This and other developmental studies were well-designed and tested several exposure concentrations and several endpoints of toxicity. Although the principal and supporting studies corroborate an effect level for developmental toxicity endpoints, there are insufficient data presented for possible respiratory effects. Confidence in the data base is low. There are no multigenerational studies and only one subchronic study. Furthermore, these studies do not adequately address portal-of-entry effects given that short-term exposure to higher concentrations than that established for the LOAEL cause nasal and throat irritation in both human and experimental animal species. Reflecting medium confidence in the principal study and low confidence in the data base, confidence in the RfC is low.

___I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

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

Other EPA Documentation -- None

Agency Work Group Review -- 04/21/1988, 05/26/1988, 03/22/1990, 05/16/1990, 07/17

Verification Date -- 07/17/1991

___I.B.7. EPA CONTACTS (INHALATION RfC)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

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_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Methyl ethyl ketone (MEK)
CASRN -- 78-93-3
Last Revised -- 12/01/1989

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

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on no human carcinogenicity data and inadequate animal data.

___II.A.2. HUMAN CARCINOGENICITY DATA

None.

___II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. No data were available to assess the carcinogenic potential of methyl ethyl ketone by the oral or inhalation routes. In a skin

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carcinogenesis study, two groups of 10 male C3H/He mice received dermal applications of 50 mg of a solution containing 25 or 29% methyl ethyl ketone in 70% dodecylbenzene twice a week for 1 year. No skin tumors developed in the group of mice treated with 25% methyl ethyl ketone. After 27 weeks, a single skin tumor developed in 1 of 10 mice receiving 29% methyl ethyl ketone (Horton et al., 1965).

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Methyl ethyl ketone was not mutagenic for Salmonella typhimurium strains TA98, TA100, TA1535, or TA1537 with or without rat hepatic homogenates (Florin et al., 1980; Douglas et al., 1980). Methyl ethyl ketone induced aneuploidy in the diploid D61, M strain of Saccharomyces cerevisiae (Zimmermann et al., 1985). Low levels of methyl ethyl ketone combined with low levels of nocodazole (another inducer of aneuploidy), also produced significantly elevated levels of aneuploidy in the system (Mayer and Goin, 1987).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

None.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

None.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

U.S. EPA. 1985. Health and Environmental Effects Profile for Methyl Ethyl Ketone. 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. 1988. Updated Health Effects Assessment for Methyl Ethyl Ketone. 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.

The 1988 Updated Health Effects Assessment for Methyl Ethyl Ketone has

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received Agency review.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 05/30/1989

Verification Date -- 05/30/1989

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

VI. BIBLIOGRAPHY

Substance Name -- Methyl ethyl ketone (MEK)
CASRN -- 78-93-3
Last Revised -- 06/01/1993

VI.A. ORAL RfD REFERENCES

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36: 511-522.

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_VII. REVISION HISTORY

Substance Name -- Methyl ethyl ketone (MEK)
CASRN -- 78-93-3

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Date	Section	Description
03/31/1987	I.A.6.	Documentation corrected
03/01/1988	I.A.2.	Paragraph 2 clarified
07/01/1989	II.	Carcinogen assessment now under review
07/01/1989	VI.	Bibliography on-line
12/01/1989	II.	Carcinogen assessment on-line
12/01/1989	VI.C.	Carcinogen references added
04/01/1990	I.B.	Inhalation RfC now under review
06/01/1990	I.A.	Oral RfD summary noted as pending change
06/01/1990	IV.F.1.	EPA contact changed
08/01/1991	I.A.	Withdrawn pending further review
08/01/1991	VI.A.	Oral RfD references withdrawn
01/01/1992	IV.	Regulatory Action section on-line
07/01/1992	I.B.	Inhalation RfC on-line
07/01/1992	VI.B.	Inhalation RfC references on-line
08/01/1992	VI.B.	Inhalation RfC references clarified
10/01/1992	I.A.	Work group review date added
12/01/1992	I.A.	Work group review date added
05/01/1993	I.A.	Oral RfD summary replaced; RfD changed
05/01/1993	VI.A.	Oral RfD references replaced
06/01/1993	VI.C.	Minor correction

VIII. SYNONYMS

Substance Name -- Methyl ethyl ketone (MEK)
CASRN -- 78-93-3
Last Revised -- 01/31/1987

78-93-3
aethylmethylketon
2-butanone
butanone-2
ethyl methyl cetone
ethylmethylketon
ethyl methyl ketone
ketone, ethyl methyl
meetco
MEK
methyl acetone
Methyl Ethyl Ketone
metiletilchetone
metyloetyloketon
RCRA waste number U159
UN 1193
UN 1232

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0071.htm

EPA United States Environmental Protection Agency

IRIS
Integrated Risk Information System

Methylene Chloride

Dichloromethane

CASRN 75-09-2

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0070
Dichloromethane; CASRN 75-09-2

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Dichloromethane

File On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	03/01/1988
Inhalation RfC Assessment (I.B.)	no data	09/01/1991
Carcinogenicity Assessment (II.)	on-line	02/01/1995

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I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Dichloromethane
CASRN -- 75-09-2
Primary Synonym -- Methylene Chloride
Last Revised -- 03/01/1988

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Liver toxicity 2-Year Rat Drinking Water Bioassay National Coffee Association, 1982	NOAEL: 5.85 and 6.47 mg/kg/day for males and females, respectively LOAEL: 52.58 and 58.32 mg/kg/day for males and females, respectively	100	1	6E-2 mg/kg/day

*Conversion Factors: Doses reflect actual values and not nominal ones.

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

National Coffee Association. 1982. 24-Month chronic toxicity and oncogenicity study of methylene chloride in rats. Final Report. Prepared by Hazleton Laboratories America, Inc., Vienna, VA. (Unpublished)

The chosen study appears to have been very well conducted, with 85 rats/ sex at each of four nominal dose groups (i.e., 5, 50, 125 and 250 mg/kg/day) for 2 years. A high-dose recovery group of 25 rats/sex, as well as two control

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groups of 85 and 50 rats/sex, was also tested. Many effects were monitored. Treatment related histological alterations of the liver were evident at nominal doses of 50 mg/kg/day or higher. The low nominal dose of 5 mg/kg/day was a NOAEL.

The supporting data base is limited. A NOAEL of 87 mg/cu.m was reported in one inhalation study (Haun et al., 1972). [The equivalent oral dose is about 28 mg/kg bw/day (i.e., 87 mg/cu.m x 0.5 x 0.223 cu.m/day/0.35 kg; these exposure values are for rats).]

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- (10a x 10h) The 100-fold factor accounts for both the expected intra- and interspecies variability to the toxicity of this chemical in lieu of specific data.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

None.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

The study is given a high confidence rating because a large number of animals of both sexes were tested in four dose groups, with a large number of controls. Many effects were monitored and a dose-related increase in severity was observed. The data base is rated medium to low because only a few studies support the NOAEL. Medium confidence in the RfD follows.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1985

Other EPA Documentation -- None

Agency Work Group Review -- 06/24/1985, 07/08/1985, 11/06/1985

Verification Date -- 11/06/1985

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I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Dichloromethane
CASRN -- 75-09-2
Primary Synonym -- Methylene Chloride

Not available at this time.

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Dichloromethane
CASRN -- 75-09-2
Primary Synonym -- Methylene Chloride
Last Revised -- 02/01/1995

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

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

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___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification --B2; probable human carcinogen

Basis -- Based on inadequate human data and sufficient evidence of carcinogenicity in animals; increased incidence of hepatocellular neoplasms and alveolar/bronchiolar neoplasms in male and female mice, and increased incidence of benign mammary tumors in both sexes of rats, salivary gland sarcomas in male rats and leukemia in female rats. This classification is supported by some positive genotoxicity data, although results in mammalian systems are generally negative.

___II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. Neither of two studies of chemical factory workers exposed to dichloromethane showed an excess of cancers (Ott et al., 1983; Friedlander et al., 1978; Hearne and Friedlander, 1981). The Ott et al. (1983) study was designed to examine cardiovascular effects, and consequently the study period was too short to allow for latency of site-specific cancers. In the Friedlander et al. (1978) study, exposures were low, but the data provided some suggestion of an increased incidence of pancreatic tumors. This study was recently updated to include a larger cohort, followed through 1984, and an investigation of possible confounding factors (Hearne et al., 1986, 1987). A nonsignificant excess in pancreatic cancer deaths was observed, which was interpreted by EPA (1987a) as neither clear evidence of carcinogenicity in humans, nor evidence of noncarcinogenicity. An update of the Ott et al. (1983) study, based on longer follow-up, indicated possible elevation of liver and biliary tract cancers (TSCA section 8(e) submission no. 8eHQ-0198-0772 FLWP et seq., 1989).

___II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Dichloromethane administered in the drinking water induced a significant increase in combined hepatocellular carcinoma and neoplastic nodules in female F344 rats and a nonsignificant increase in combined hepatocellular carcinoma and neoplastic nodules in male B6C3F1 mice (NCA, 1982, 1983). Two inhalation studies with dichloromethane have shown an increased incidence of benign mammary tumors in both sexes of Sprague-Dawley (Burek et al., 1984) and F344 (NTP, 1986) rats. Male Sprague-Dawley rats had increased salivary gland sarcoma (Burek et al., 1984) and female F344 rats had increased leukemia incidence (NTP, 1986). Both sexes of B6C3F1 mice developed liver and lung tumors after dichloromethane treatment (NTP, 1986).

In a 2-year study by the National Coffee Association (1982, 1983), groups of 85 F344 rats/sex/dose received 5, 50, 125, or 250 (mg/kg)/day of dichloromethane in the drinking water. Control groups consisted of 135 rats/sex. In female rats the incidence of combined hepatocellular carcinoma and neoplastic nodules was statistically significantly increased in the 50 and 250 mg/kg dose groups when compared with matched controls (0/134, 1/85, 4/83, 1/85, and 6/85 in the five dose groups 0, 5, 50, 125, and 250 (mg/kg)/day, respectively). The incidence of hepatocellular carcinoma alone was not significantly increased (0/134, 0/85, 2/83, 0/85, 2/85). The combined incidence of hepatocellular carinoma and neoplastic nodules in controls and

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the 4 dose groups (472 rats: 4 with carcinoma and 8 with neoplastic nodules) was similar to that for historical controls (419 rats; 5 with carcinoma, 19 with neoplastic nodules). Male rats showed no increase in liver tumors.

In the same National Coffee Association study (1982, 1983), B6C3F1 mice received 0, 60, 125, 185, or 250 (mg/kg)/day of dichloromethane in drinking water. Treatment groups consisted of 50 female mice and 200, 100, 100, and 125 male mice (low to high dose). One hundred females and 125 males served as controls. Male mice had an increased incidence of combined neoplastic nodules and hepatocellular carcinoma (24/125, 51/200, 30/100, 31/99, 35/125). The increase was not dose-related, but the pairwise comparisons for the two mid-dose groups were reported to be statistically significant (U.S. EPA, 1985a). The hepatocellular carcinoma incidence alone for male mice (which was about 55 to 65% of the total) was not significantly elevated. Female mice did not have increased liver tumor incidence. The EPA (1985b) regarded this study as suggestive but not conclusive evidence for carcinogenicity of dichloromethane.

A gavage bioassay of dichloromethane conducted by NTP (1982) has not been published because of high mortality, much of which was attributed to gavage accidents.

Inhalation exposure of 107 to 109 Syrian hamsters/sex/dose to 0, 500, 1500, or 3500 ppm of dichloromethane for 6 hours/day, 5 days/week for 2 years did not induce neoplasia (Burek et al., 1984). Sprague-Dawley rats (129/sex/dose) were exposed under the same conditions. Female rats administered the highest dose experienced significantly reduced survival from 18-24 months. Female rats showed a dose-related increase in the average number of benign mammary tumors per rat (1.7, 2.3, 2.6, 3.0), although the numbers of rats with tumors were not significantly increased. A similar response was observed in male rats, but to a lesser degree. In the male rats there was a statistically significant positive trend in the incidence of sarcomas of the salivary gland (1/93, 0/94, 5/91, 11/88); the incidence was significantly elevated at the high dose. There is a question as to whether these doses reached the MTD, particularly in the hamsters and the male rats. In another study (Dow Chemical Co., 1982), 90 Sprague-Dawley rats/sex were exposed by inhalation to 0, 50, 200, or 500 ppm dichloromethane for 20 months (male) or 24 months (female). No salivary tumors were observed, but there was an exposure-related increase in the total number of benign mammary tumors in female rats, although the increase was not statistically significant in any individual exposure group.

Groups of 50 each male and female F344/N rats and B6C3F1 mice were exposed to dichloromethane by inhalation, 6 hours/day, 5 days/week for 2 years (NTP, 1986). Exposure concentrations were 0, 1000, 2000, or 4000 ppm for rats and 0, 2000, or 4000 ppm for mice. Survival of male rats was low; however, this apparently was not treatment-related. Survival was decreased in a treatment-related fashion for male and female mice and female rats. Mammary adenomas and fibroadenomas were significantly increased in male and female rats after survival adjustment, as were mononuclear cell leukemias in female rats. Among treated mice of both sexes there were significantly increased incidences of hepatocellular adenomas and carcinomas, and of alveolarbronchiolar adenomas and carcinomas, by life table tests. Adenomas and carcinomas were significantly increased alone as well as in combination. In addition, there were significant dose-related increases in the number of lung tumors per animal multiplicity in both sexes of mice.

Two inhalation assays using dogs, rabbits, guinea pigs, and rats showed no tumors, but were not conducted for the lifetime of the animals (Heppel et al., 1944; MacEwen et al., 1972). Theiss et al., (1977) injected Strain A male mice intraperitoneally with 0, 160, 400, or 800 mg/kg of dichloromethane 16 to 17 times, over 5 to 6 weeks. Survival of the animals was poor. The animals remaining 24 weeks after the first treatment were killed and examined for lung tumors; pulmonary adenomas were found.

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II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Dichloromethane was mutagenic for Salmonella typhimurium with or without the addition of hepatic enzymes (Green, 1983) and produced mitotic recombination in yeast (Callen et al., 1980). Results in cultured mammalian cells have generally been negative, but dichloromethane has been shown to transform rat embryo cells and to enhance viral transformation of Syrian hamster embryo cells (Price et al., 1978; Hatch et al., 1983). Although chlorinated solvents have often been suspected of acting through a nongenotoxic mechanism of cell proliferation, Lefevre and Ashby (1989) found methylene chloride to be unable to induce hepatocellular division in mice.

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 7.5E-3 per (mg/kg)/day

Drinking Water Unit Risk -- 2.1E-7 per (ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

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

II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- hepatocellular adenomas or carcinomas (NTP) and hepatocellular cancer and neoplastic nodules (NCA)
Test Animals -- mouse/B6C3F1 (female, NTP; male, NCA)
Route -- inhalation (NTP); drinking water (NCA)
Reference -- NTP, 1986; National Coffee Association (NCA), 1983

Dose				
Administered (ppm)	mg/kg/day	Human Equivalent (mg/kg)/day	Tumor Incidence	Reference
0	0	0	3/50	NTP, 1986
2000	1582	122	16/48	
4000	3162	244	40/48	

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0	0	24/125	NCA, 1983
60	4.5	51/200	
125	9.4	30/100	
185	14.0	31/99	
250	18.9	35/125	

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

The slope factor is an arithmetic mean of slope factors derived from NTP(1986) and the National Coffee Association (1983) data, 2.6E-3 per (mg/kg)/day and 1.2E-2 per (mg/kg)/day, respectively. The use of liver tumor data from the NTP inhalation bioassay was considered valid since dichloromethane is rapidly absorbed following either inhalation or ingestion.

Dose conversions used the mean body weight for female mice at the midpoint of the bioassay, and an estimated inhalation rate of 0.0407 cu.m/day. To obtain estimates of unit risk for humans, an inhalation rate of 20 cu.m/day was assumed. Dichloromethane was considered to be well-absorbed as a vapor at low doses. No pharmacokinetic or metabolism data have been used to modify the oral unit risk estimate, because such analyses have not yet been carried out.

The unit risk should not be used if the water concentration exceeds 5E+4 ug/L, since above this concentration the unit risk may not be appropriate.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

Adequate numbers of animals were used in both assays. Risk estimates were based on the more sensitive sex in each study. The two risk estimates were within a factor of 5.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

___II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk -- 4.7E-7 per (ug/cu.m)

Extrapolation Method -- Linearized multistage procedure, extra risk

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+1 ug/cu.m
E-6 (1 in 1,000,000)	2E+0 ug/cu.m

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___II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSUR

Tumor Type -- combined adenomas and carcinomas
Test Animals -- mouse/B6C3F1, female
Route -- inhalation
Reference -- NTP, 1986

	Dose			
Tumor Type	Administered (ppm)	Transformed Animal (mg/kg)/day	Human Equivalent (mg/kg)/day	Tumor Incidence
Liver	0	0	0	3/45
	2000	1582	356	16/46
	4000	3162	712	40/46
Lung	0	0	0	3/45
	2000	1582	356	30/46
	4000	3162	712	41/46

___II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE

The unit risk of 4.7E-7 per (ug/cu.m), which incorporates information on pharmacokinetics and metabolism of dichloromethane, is approximately nine-fold lower than the previous applied dose estimate (U.S. EPA, 1987a,b). Internal dose estimates were based on the metabolism of dichloromethane by the glutathione-s-transferase pathway, as estimated by the model developed by Andersen et al. (1987). The internal dose was corrected for interspecies differences in sensitivity by using the surface area correction factor.

Calculation of a slope factor from the unit risk is inappropriate when pharmacokinetic models are used. (When dose-response relationships are figured on the basis of internal or metabolized dose, a slope factor in terms of per (mg/kg)/day represents a back calculation using different absorption assumptions than the pharmacokinetic models. This introduces possible contradictions.)

The unit risk should not be used if the air concentration exceeds 2E+4 ug/cu.m, since above this concentration the unit risk may differ from that stated. Since the unit risk is based on a pharmacokinetic model, the risk may change with alterations in exposure patterns. Thus, the unit risk presented here may not be applicable to acute, high exposures.

___II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOS

Adequate numbers of animals were observed and tumor incidences were significantly increased in a dose-dependent fashion. Analysis excluding animals that died before observation of the first tumors produced similar

00088516

risk estimates, as did time-to-tumor analysis. The use of animal and human metabolism and pharmacokinetic data reduces some of the uncertainty typically associated with dose-risk extrapolation. A great deal of uncertainty still exists, however, in the estimates of internal dose generated by the model of Andersen et al. (1987). Important uncertainties remain regarding the pharmacokinetics, pharmacodynamics, and mechanisms of carcinogenicity for dichloromethane.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1985a,b, 1987a,b

The Addendum to the Health Assessment Document, the Update to the Health Assessment Document and Addendum, and the Technical Analysis of New Methods and Data for dichloromethane have received Agency and external review, including a review by the Science Advisory Board (SAB). Although the last two documents are not yet finalized and the SAB comments are not yet incorporated, these do not alter this document's analyses or conclusions.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 11/12/1986, 12/04/1986, 04/06/1989

Verification Date -- 04/06/1989

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Dichloromethane
CASRN -- 75-09-2
Primary Synonym -- Methylene Chloride
Last Revised -- 08/01/1991

00088517

__VI.A. ORAL RfD REFERENCES

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None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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00088518

and volatile chlorinated methanes and ethanes. Cancer Res. 43: 1945-1950.

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82/004FF.

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U.S. EPA. 1987b. Technical Analysis of New Methods and Data Regarding Dichloromethane Hazard Assessments. Review Draft. Office of Health and Environmental Assessment, Washington, DC. EPA/600/8- 87/029A.

_VII. REVISION HISTORY

Substance Name -- Dichloromethane
CASRN -- 75-09-2
Primary Synonym -- Methylene Chloride

Date	Section	Description
04/20/1987	II.C.1.	Unit Risk corrected from 4.1E-4 to 4.1E-6
05/21/1987	II.A.2.	Missing text replaced in 3rd paragraph
03/01/1988	I.A.1.	Dose conversion clarified
03/01/1988	I.A.2.	Text revised
03/01/1988	II.B.3.	Text revised
03/01/1988	II.B.4.	Confidence statement revised
03/01/1988	II.C.3.	Text revised
03/01/1988	II.C.4.	Confidence statement revised
03/01/1988	II.D.3.	Primary contact changed
03/01/1988	III.A.	Health Advisory added
01/01/1989	II.	Carcinogen summary noted as pending change
10/01/1989	II.B.3.	Inhalation rate corrected in paragraph 1
10/01/1989	II.C.2.	Dose corrections in mg/kg/day
10/01/1989	II.C.3.	Inhalation rate corrected in paragraph 1
10/01/1989	II.D.3.	Contacts phone number changed
08/01/1990	IV.F.1.	EPA contact changed
09/01/1990	II.	Carcinogen assessment revised following re-evaluation
09/01/1990	II.C.1.	Inhalation unit risk changed
09/01/1990	VI.	Bibliography on-line
01/01/1991	II.C.1.	Paragraph moved to II.C.3.
01/01/1991	II.C.1.	Inhalation slope factor removed (global change)
08/01/1991	VI.C.	Citations clarified
09/01/1991	I.B.	Inhalation RfC now under review
01/01/1992	IV.	Regulatory actions updated
02/01/1995	II.D.3.	Primary contact changed

VIII. SYNONYMS

Substance Name -- Dichloromethane

00088520

CASRN -- 75-09-2
Primary Synonym -- Methylene Chloride
Last Revised -- 01/31/1987

75-09-2
Aerothene MM
Chlorure de methylene
DCM
Dichlormethan, uvasol
Dichloromethane
1,1-Dichloromethane.
Freon 30
Methane dichloride
Methane, dichloro-
Methylene bichloride
Methylene Chloride
Methylene dichloride
Metylenu chlorek
Narkotil
NCI-C50102
R 30
Solaesthin
Solmethine
WLN: G1G

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0070.htm

00088521

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Nitrobenzene

CASRN 98-95-3

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0079
Nitrobenzene; CASRN 98-95-3

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Nitrobenzene

File On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	01/01/1991
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	02/01/1995

00088522

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Nitrobenzene
CASRN -- 98-95-3
Last Revised -- 01/01/1991

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

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

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Hematologic, adrenal, renal and hepatic lesions Rat/Mouse Subchronic Inhalation Study	NOAEL: none LOAEL: 25 mg/cu.m (mice) converted to 4.6 mg/kg/day	10,000	1	5E-4 mg/kg/day

CIIT, 1984

*Conversion Factors: 6 hour/24 hour, 5 days/7 days, 0.039 cu.m/day/0.03 kg (mice breathing rate/body weight) and 0.8 absorption factor; thus, 25 mg/cu.m x 6 hour/24 hour x 5 days/7 days x 0.039 cu.m/day / 0.03 kg x 0.8 = 4.6 mg/kg/day

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

CIIT (Chemical Industry Institute of Toxicology). 1984. Ninety-day inhalation toxicity study of nitrobenzene in F344 rats and B6C3F1 mice.

00088523

Research Triangle Park, NC. FYI-OTS-0874-0333.

The CIIT 90-day inhalation study provides the most appropriate data currently available to derive an RfD. Ten animals/sex/species/dose group were administered nitrobenzene at 1 of 3 doses. Other than increased incidence of hemolytic anemia in rats at 25 mg/cu.m and vacuolization of adrenal cortical cells in female mice at 25 mg/cu.m and higher, adverse effects of nitrobenzene exposure in mice and rats were comparable to unexposed controls at this dose. Mice and rats exposed to nitrobenzene at 81 mg/cu.m showed increased incidence and severity of liver and kidney lesions.

Data regarding the effects of nitrobenzene in humans are limited to symptoms and observations in workers, including headaches, vertigo, and methemoglobinemia (ACGIH, 1980). The potential "safe" level derived from the TLV appears adequate to protect workers from such adverse effects; however, the effects of occupational exposure to nitrobenzene on the liver and/or kidneys have not been adequately evaluated. The CIIT (1984) study indicates that the liver and kidney may be target organs of chronic/subchronic nitrobenzene exposure, and an acceptable level based on the TLV may not be protective for the toxic effects of nitrobenzene on the liver and/or kidney. Therefore, until more definitive chronic data are available, the RfD of 0.0005 mg/kg/day is recommended to protect against adverse health effects of nitrobenzene.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- The uncertainty factor of 10,000 represents two 10-fold factors for both intra- and interspecies variability to the toxicity of this chemical in lieu of specific data, a 10-fold factor for estimating a chronic effect level, from its subchronic equivalent, and a 10-fold factor for estimating an RfD from a LOAEL rather than a NOAEL.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

None.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

Medium to low confidence in the study is recommended because it is not an oral study, a limited number of animals/sex/dose were tested, and a NOAEL for the critical toxic effect (i.e., adrenal toxicity) was not determined, although two species were used and many parameters were measured. Low confidence in the data base is recommended because chronic reproductive and teratology data are missing. Low confidence in the RfD follows.

00088524

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1985

Other EPA Documentation -- None

Agency Work Group Review -- 06/24/1985, 07/08/1985, 11/06/1985

Verification Date -- 07/08/1985

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Nitrobenzene
CASRN -- 98-95-3

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Nitrobenzene
CASRN -- 98-95-3
Last Revised -- 02/01/1995

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1

00088525

in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

NOTE: The carcinogenicity assessment for nitrobenzene 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.

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on no data concerning carcinogenicity in humans or animals.

___II.A.2. HUMAN CARCINOGENICITY DATA

None.

___II.A.3. ANIMAL CARCINOGENICITY DATA

None.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

In Salmonella/microsomal mutagenicity assays with strains TA92, TA94, TA97, TA98, TA100, TA1535, TA1537 and TA1538, nitrobenzene was not positive (Garner and Nutman, 1977; Chiu et al., 1978; Shimizu et al., 1983; Ho et al., 1981; Haworth et al., 1983; Anderson and Styles, 1978; Miyata et al., 1981). These assays were conducted with and without liver homogenates by plate incorporation, spot test and in one study (Hughes et al., 1984), by the vapor exposure method.

Nitrobenzene was mutagenic in the presence of liver homogenate and norharman in S. typhimurium strain TA98 but not in strain TA100 (Suzuki et al., 1983). Nitrobenzene did not cause an increase in unscheduled DNA synthesis in hepatocytes isolated from gavage-treated rats (Mirsalis et al., 1982). Fel'dt (1985) reported that oral administration of nitrobenzene did

00088526

not produce micronucleus or chromosome aberrations in bone marrow cells or dominant lethal mutations in mice.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

None.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

None.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

U.S. EPA. 1985. Health and Environmental Effects Profile for Nitrobenzene. 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. 1987. Health Effects Assessment for Nitrobenzene. Prepared by the Office of Health and Environmental Assessment, Environmental and Criteria and Assessment Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Washington, DC.

The 1985 Health and Environmental Effects Profile for Nitrobenzene and the 1987 Health Effects Assessment for Nitrobenzene have received OHEA review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 11/08/1989, 12/07/1994

Verification Date -- 11/08/1989

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

00088527

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Nitrobenzene
CASRN -- 98-95-3
Last Revised -- 12/01/1990

__VI.A. ORAL RfD REFERENCES

ACGIH (American Conference of Governmental Industrial Hygienists). 1980. Documentation of the Threshold Limit Values. 4th ed. Cincinnati, OH. p. 303.

CIIT (Chemical Industry Institute of Toxicology). 1984. Ninety-day inhalation toxicity study of nitrobenzene in F344 rats, CD rats, and B6C3F1 mice. Research Triangle Park, NC. FYI-OTS-0784-0333.

U.S. EPA. 1985. Health and Environmental Effects Profile for Nitrobenzene. 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.

__VI.B. INHALATION RfC REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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00088528

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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.

Miyata, R., T. Nomi, K. Yoshikawa and M. Ishidate. 1981. Metabolic activation of p- nitrotoluene and trichloroethylene by rat liver S9 or mouse liver S9 fractions in Salmonella typhimurium strains. Eisei Shikensho Hokoku. 99: 60-65.

Shimizu, M., Y. Yasui and N. Matsumoto. 1983. Structural specificity of aromatic compounds with special reference to mutagenic activity in Salmonella typhimurium: A series of chloro- or fluoro-nitrobenzene derivatives. Mutat. Res. 116: 217-238.

Suzuki, J., T. Koyami and S. Suzuki. 1983. Mutagenicities of mono-nitrobenzene derivatives in the presence of norharman. Mutat. Res. 120: 105-110.

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U.S. EPA. 1987. Health Effects Assessment for Nitrobenzene. Prepared by the Office of Health and Environmental Assessment, Environmental and Criteria and Assessment Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Washington, DC.

_VII. REVISION HISTORY

Substance Name -- Nitrobenzene
CASRN -- 98-95-3

Date	Section	Description

00088529

03/01/1988	I.A.2.	Text deleted
03/01/1988	I.A.4.	Text deleted
03/01/1988	I.A.5.	Confidence levels revised
03/01/1988	I.A.7.	Documentation corrected
06/30/1988	I.A.3.	UF corrected in text
07/01/1989	I.B.	Inhalation RfD now under review
08/01/1989	I.A.	Oral RfD summary noted as pending change
12/01/1989	II.	Carcinogen assessment now under review
06/01/1990	IV.F.1.	EPA contact changed
06/01/1990	VI.	Bibliography on-line
12/01/1990	II.	Carcinogen assessment on-line
12/01/1990	VI.C.	Carcinogen assessment references added
01/01/1991	I.A.	Text edited
01/01/1992	I.A.7.	Primary contact changed
01/01/1992	IV.	Regulatory actions updated
01/01/1995	II.D.2.	Work group review date added
02/01/1995	II.	Carcinogenicity assessment noted as pending change

VIII. SYNONYMS

Substance Name -- Nitrobenzene
CASRN -- 98-95-3
Last Revised -- 01/31/1987

98-95-3
Benzene, Nitro-
Essence of Mirbane
Essence of Myrbane
Mirbane Oil
NCI-C60082
Nitrobenzene
Nitrobenzol
Oil of Mirbane
Oil of Myrbane

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0079.htm

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00088531

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX)

CASRN 121-82-4

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0313
Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX); CASRN 121-82-4

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR RDX

File On-Line 09/26/1988

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	02/01/1993
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	07/01/1993

00088532

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX)
CASRN -- 121-82-4
Last Revised -- 02/01/1993

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Inflammation of the prostate 2-Year Rat Feeding Study U.S. DOD, 1983	NOEL: 0.3 mg/kg/day LOAEL: 1.5 mg/kg/day	100	1	3E-3 mg/kg/day

*Conversion Factors: None

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

U.S. Department of Defense. 1983. AD-A160-774. Available from Defense Technical Center. Write to Documents, Cameron Station, Alexandria, VA 22314, or call (703)274-7633.

The U.S. Department of Defense (U.S. DOD, 1983) commissioned a study to evaluate the chronic effects of RDX in groups of 85 male and female Fischer 344 rats fed doses of 0, 0.3, 1.5, 8.0, or 40.0 mg/kg/day for 24 months. RDX purity ranged from 89.2 to 98.7%, and particle size ranged from <22 um (51.7%) to 440 um. Mortality was increased in high-dose males and females throughout the study (e.g., 88 and 41%, respectively, at week 88 as compared with 32 and

00088533

33% for respective controls). Tremors and convulsions were frequently observed prior to deaths of high-dose males and females beginning at week 25. Behavioral hypersensitivity to stimuli resulted in fighting among cohabited high-dose males. Histologic evaluation failed to detect lesions of the central nervous system. The incidence of cataracts was significantly increased ($p<0.05$) in high-dose females at weeks 78 and 104; the eyes of high-dose males appeared normal. These observations were considered to be treatment-related. Hepatotoxicity, primarily at 40 mg/kg/day, was evidenced by hepatomegaly (although histological changes were not reported to be apparent), hypocholesteremia, hypotriglyceridemia, reduced serum albumin/total protein levels, and increased lactic dehydrogenase (LDH) levels. Compound-induced renal toxicity was found primarily in high-dose males. Absolute kidney weights were significantly increased in high-dose females, and relative kidney weights were significantly increased in high-dose males throughout the study. Absolute and relative kidney weights were sporadically increased in males and females receiving 8 mg/kg/day at 12 months or in any controls. In males receiving 1.5, 8.0, and 40.0 mg/kg/day, there was increased pigment in the spleen (possibly a hematopoietic response and not adverse) and suppurative inflammation of the prostate. The only significant ($p<0.05$) histologic change in females was an increase in lenticular cataracts (32/48 in the high-dose group compared with 15/53 in controls). Based on suppurative inflammation of the prostate of males receiving 1.5 mg/kg/day and above, the LOAEL was 1.5 mg/kg/day and the NOEL was 0.3 mg/kg/day.

Schneider et al. (1978) dosed 30 Sprague-Dawley rats (sex not specified) orally with RDX in an isotonic saline slurry at 20 mg/kg/day for 90 days. Eight dosed rats died between days 42 and 77, apparently from exacerbation of chronic respiratory disease.

Groups of 10 male and 12 female mice were fed 0, 40, 60, or 80 mg/kg/day RDX in the diet for 2 weeks, followed by 0, 80, 160, or 320 mg/kg/day RDX, respectively, for 11 weeks. The LOAEL for the study based on anemia in males was 160 mg/kg/day, and the NOAEL was 80 mg/kg/day. In the 13-week study with Fischer 344 rats (U.S. DOD, 1980), 60 male and 60 female rats were divided into six groups, each consisting of 10 males and 10 females. The groups were fed diets that provided an RDX intake of 0, 10, 14, 20, 28, or 40 mg/kg/day. Based on anemia, the LOAEL was 28 mg/kg/day and the NOAEL was 20 mg/kg/day.

Levine et al. (1981) fed RDX in the diet to groups of 10 male and 10 female Fischer 344 rats for 13 weeks at doses of 0, 10, 30, 100, 300, or 600 mg/kg/day. Based on effects on liver weights, the LOAEL was 100 mg/kg/day and the NOAEL 30 mg/kg/day.

Von Oettingen et al. (1949) conducted short-term toxicity studies with RDX in rats (strain and sex not specified), and dogs (breed not specified). Groups of 15 rats received RDX in the feed at doses of 0, 15, 50, or 100 mg/kg/day for 10 weeks. Based on CNS effects, the LOAEL was 50 mg/kg/day and the NOAEL was 15 mg/kg/day. In a follow-up study, groups of 20 rats were administered RDX in the diet at doses to provide an intake of 0, 15, 25, or 50 mg/kg RDX/day for 12 weeks. The LOAEL based on mortality and body weight loss was 25 mg/kg/day, and the NOAEL was 15 mg/kg/day. In the dog study, seven healthy females were force-fed 50 mg/kg/day RDX (molded in a moistened pellet), 6 days/week for 6 weeks. One treated dog that died at the end of week 5 had many congested areas on the walls of the small intestines. Treatment caused hyperirritability, convulsions, and weight loss. The gross and microscopic changes in the organs and tissues were negligible.

Groups of three male and three female dogs were fed 0, 0.1, 1, or 10 mg/kg/day RDX for 90 days (U.S. DOD, 1974a). No signs of toxicity, except for temporary episodes of emesis, were seen.

Groups of three male and three female monkeys were administered oral gavage doses of 0, 0.1, 1, or 10 mg/kg RDX in a 1% aqueous solution of methylcellulose daily for 90 days (U.S. DOD, 1974b). Based on effects on the

00088534

CNS, the LOAEL was 10 mg/kg/day and the NOAEL was 1 mg/kg/day.

Male and female Sprague-Dawley rats were fed RDX at doses of 0, 1.0, 3.1, or 10 mg/kg/day for 24 months (U.S. DOD, 1976). Survival was comparable to controls in high-dose males and females. The LOAEL is 3.1 mg/kg/day and the NOAEL is 1 mg/kg/day based on decreased body weights in females.

The chronic effects of RDX were evaluated for groups of 85 male and female B6C3F1 mice fed doses of 0, 1.5, 7.0, 35.0, or 100 mg/kg/day for 24 months (U.S. DOD, 1984). The major toxic effects included weight loss, increased liver weights, and testicular degeneration. Based on testicular degeneration in males, the LOAEL for systemic toxicity was 35 mg/kg/day, and the NOAEL was 7.0 mg/kg/day.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- The UF of 100 allows for uncertainty in the extrapolation of dose levels from laboratory animals to humans (10A) and uncertainty in the threshold for sensitive humans (10H).

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

A two-generation reproduction study was conducted with Fischer 344 rats (U.S. DOD, 1980). The initial group of parental animals (F0), consisting of 22 males and 22 females, was fed RDX in the diet at nominal doses of 0, 5, 16, or 50 mg/kg/day for 13 weeks. After treatment, the animals were mated and the dams were allowed to litter. After weaning, groups of 26 males and 26 females (F1) were fed the same dietary concentrations of RDX as their parents for 13 weeks and were allowed to mate following completion of treatment. The LOAEL for reproductive effects was 50 mg/kg/day, and the NOAEL was 16 mg/kg/day. The LOAEL for developmental toxicity was 16 mg/kg/day, and the NOAEL was 5 mg/kg/day.

Developmental toxicity studies were conducted with Fischer 344 rats and New Zealand rabbits (U.S. DOD, 1980). In the study with rats, groups of 24 or 25 rats received 0, 0.2, 2, or 20 mg/kg/day RDX, by gavage on days 6 through 19 of gestation. Teratogenicity was not demonstrated at any of the dose levels tested. Based on embryotoxicity and maternal toxicity, the LOAEL was 20 mg/kg/day and the NOAEL was 2 mg/kg/day. In the study with rabbits, dams (number not reported) were dosed with 0, 0.2, 2, or 20 mg/kg/day RDX, by gavage, on days 7 through 29 of gestation, and 11 to 12 litters/group were delivered by cesarean section on day 30. Based on maternal toxicity and developmental effects, the LOAEL was 20 mg/kg/day and the NOAEL was 2.0 mg/kg/day.

Angerhofer et al. (1986) investigated the developmental toxic effects of RDX in rats. The authors conducted a range-finding study using groups of six pregnant rats treated with RDX, by gavage, at dose levels of 0, 10, 20, 40, 80, or 120 mg/kg/day on gestational days 6 through 15. No teratogenic effects were noted in this study. Based on maternal deaths and toxicity and a decrease in fetal weight length, the LOAEL was 20 mg/kg/day and the NOAEL was 6 mg/kg/day.

00088535

I.A.5. CONFIDENCE IN THE ORAL RfD

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

The principal study was a well-designed and well-executed long-term study with a large number of dose groups using an adequate number of animals. A clear NOEL and LOAEL were established. The data base is extensive, covering all important toxicological endpoints, but lacking a chronic study for a nonrodent species. However, short-term data suggests that dogs and monkeys are not more sensitive than rats. High confidence in the RfD follows.

I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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

Other EPA Documentation --U.S. EPA, 1988

Agency Work Group Review -- 04/20/1988

Verification Date -- 04/20/1988

I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX)
CASRN -- 121-82-4

Not available at this time.

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

00088536

Substance Name -- Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX)
CASRN -- 121-82-4
Last Revised -- 07/01/1993

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

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- C; possible human carcinogen

Basis -- Hepatocellular adenomas and carcinomas in female B6C3F1 mice.

___II.A.2. HUMAN CARCINOGENICITY DATA

None.

Hexahydro-1,3,5-trinitro-1,3,5-triazine, an explosive polynitramine, is commonly known as RDX (British code name for Research Department Explosive or Royal Demolition Explosive). Epidemiological studies of munitions workers have not been conducted. There is no carcinogenicity information in the human health effects data base.

___II.A.3. ANIMAL CARCINOGENICITY DATA

The carcinogenic potential of RDX has been evaluated in Fischer 344 rats [U.S. Department of Defense (U.S. DOD, 1983), Sprague-Dawley rats (Hart, 1976) and B6C3F1 mice (U.S. DOD, 1984). RDX was not found to be carcinogenic when

00088537

fed to either strain of rats. It was found to produce significant increases in the combined hepatocellular adenomas/carcinomas in B6C3F1 female mice.

RDX was not found to be oncogenic in male and female Sprague-Dawley rats (100 rats/sex/dose) fed RDX in the diet at doses of 1.0, 3.1, or 10 mg/kg/day for 24 months (Hart, 1976). The percent survival among the groups was comparable (the range was between 60% and 73%).

U.S. DOD (1983) evaluated the carcinogenicity of RDX in male and female Fischer 344 rats (85 rats/sex/dose) fed doses of 0.3, 1.5, 8.0, or 40.0 mg/kg/day RDX for 24 months. Ten rats/sex/dose were killed at 6 and 12 months and the remaining animals killed after 24 months of treatment. The major toxic effects observed included anemia with secondary splenic lesions, hepatotoxicity, and urogenital lesions. Based on adverse systemic effects at 1.5 mg/kg/day, the MTD was achieved. RDX was not found to be oncogenic in this study.

U.S. DOD (1984) evaluated the incidence of tumors in groups of 85 male and female B6C3F1 mice fed doses of 1.5, 7.0, 35.0, or 100 mg/kg/day RDX for 24 months. The combined incidence of hepatocellular carcinomas and adenomas was statistically significantly increased in females receiving 7.0, 35.0, and 100.0 mg/kg RDX (14.1, 18.8, and 19.4%, respectively) when compared with concurrent (1.5%) or historical (7.9%) controls. These findings were considered to be compound-related in females. Historically, the combined incidence of hepatocellular adenomas and carcinomas in untreated male B6C3F1 mice is 31.6% as compared with 7.9% for untreated B6C3F1 females (Haseman et al., 1984). In addition, the incidence of hepatocellular adenomas in females receiving 7.0 and 35.0, but not 100 mg/kg/day, was statistically significantly increased when compared with historical controls. The incidence of combined hepatocellular adenomas and carcinomas in high-dose males was 48.1% as compared with 33.3% for the concurrent control group; the increase was not statistically significant. The incidence of benign and malignant tumors was not distinguished. Increases in alveolar and bronchiolar carcinomas in high-dose males and females, and in lung histiocytes of high-dose females were not statistically significant. Malignant lymphoma of the kidney was reported to be slightly increased (not statistically significant) in males receiving 1.5, 7.0, and 35.0 mg/kg when compared with concurrent controls. This finding was not seen in female mice. The high dose was lowered from 175 mg/kg/day to 100 mg/kg/day during week 11 because of the high mortality in both sexes. This mortality substantially reduced the number of high-dose animals. The RDX used contained 3 to 10% octahydro-1,3,5,7-tetranitro-1,3,5,7-toluene (HMX). The increase in the incidence of hepatocellular adenomas or carcinomas was not statistically significant (when compared with concurrent controls) in dosed females when analyzed separately, but was significant only when the two groups were combined. Percent survival for the groups is not stated. Mean survival time was statistically significantly lower for the highest male (14.6 months) and female (20.6 months) dose groups and for the male dose group receiving 1.5 mg/kg/day (21 months). The mean survival times for the male and female control groups were 22.3 months and 22 months, respectively.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

RDX gave negative results in all genotoxicity studies cited. The mutagenic potential of RDX was evaluated in two studies using the Salmonella/microsomal preincubation assay. Salmonella strains TA98, TA100, TA1535, and TA1538 were exposed to RDX at concentrations of 1, 10, 100, 300, or 1000 ug/plate in one study (U.S. DOD, 1980), and at 0.625 or 1.25 mg/plate in the second study (Whong et al., 1980). The assays were conducted with and without the addition of hepatic homogenates. In both studies, RDX was not mutagenic.

00088538

U.S. DOD (1977) tested the mutagenicity of several munitions wastewater chemicals before and after chlorination or ozone treatment. RDX was evaluated in Salmonella strains TA98, TA100, TA1535, TA1537, and TA1538 at several concentrations ranging from 0.24 to 14 ug/plate, and in Saccharomyces cerevisiae strain D3 at concentrations ranging from 0.00004 to 0.0023%. The assays were conducted with or without hepatic homogenates. RDX was not mutagenic before or after chlorination in these assays.

RDX gave negative results in an unscheduled DNA synthesis (UDS) assay using WI-38 (human fibroblasts) when tested at a maximum concentration of 4000 ug/mL, with or without the addition of hepatic homogenates (U.S. DOD, 1978).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

___II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 1.1E-1 per (mg/kg)/day

Drinking Water Unit Risk -- 3.1E-6 per (ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

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

___II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- liver, hepatocellular carcinoma, and adenomas (combined)
Test Animals -- mouse/B6C3F1, female
Route -- diet
Reference -- U.S. DOD, 1984

Administered Dose (mg/kg)/day	Human Equivalent Dose (mg/kg)/day	Tumor Incidence
0.0	0.0	1/65
1.5	0.13	5/62
7.0	0.58	9/64
35.0	2.90	12/64
100.0	8.30	6/31

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

00088539

The animal study dose is divided by the ratio of the human weight (70 kg) to the mouse weight (0.040 kg) raised to the 1/3 power. The high-dose data was not used in the slope factor calculation since it was lowered from 175 mg/kg/day to 100 mg/kg/day during week 11 due to low survival.

The unit risk should not be used if the water concentration exceeds 3E+3 ug/L, since above this concentration the slope factor may differ from that stated.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

RDX was found to be carcinogenic in the female B6C3F1 mice. This study had some previously discussed technical problems but is clearly positive for liver carcinomas and adenomas combined. Adequate number of animals were treated for a period of time approximating lifetime.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1988

The risk assessment in the 1988 Health Advisory on Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX) is a final document and has received Agency review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 08/15/1988, 11/30/1988

Verification Date -- 11/30/1988

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

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Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX)
CASRN -- 121-82-4
Last Revised -- 12/01/1990

__VI.A. ORAL RfD REFERENCES

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VI.B. INHALATION RfD REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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VII. REVISION HISTORY

Substance Name -- Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX)
CASRN -- 121-82-4

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00088542

Date	Section	Description
09/26/1988	I.A.	Oral RfD summary on-line
05/01/1989	II.	Carcinogen assessment now under review
01/01/1990	VI.	Bibliography on-line
08/01/1990	I.A.	Text edited
08/01/1990	II.	Carcinogen assessment on-line
08/01/1990	VI.C.	Carcinogen references added
12/01/1990	I.A.6.	Source document statement added
12/01/1990	III.A.	Health Advisory on-line
12/01/1990	VI.D.	Health Advisory references added
02/01/1993	I.A.7.	Primary contact changed
02/01/1993	II.D.3.	Primary contact changed
02/01/1993	III.A.10	Primary contact changed
07/01/1993	II.D.3.	Secondary contact's phone number changed

VIII. SYNONYMS

Substance Name -- Hexahydro-1,3,5-trinitro-1,3,5-triazine (RDX)
CASRN -- 121-82-4
Last Revised -- 09/26/1988

121-82-4
cyclonite
cyclotrimethylenenitramine
cyclotrimethylenetrinitramine
CYKLONIT
ESAIDRO-1,3,5-TRINITRO-1,3,5-TRIAZINA
HEKSOGEN
HEXAHYDRO-1,3,5-TRINITRO-1,3,5-TRIAZIN
Hexahydro-1,3,5-trinitro-1,3,5-triazine
HEXAHYDRO-1,3,5-TRINITRO-s-TRIAZINE
HEXOGEEN
HEXOGEN
HEXOGEN 5W
HEXOLITE
HEXOLITE, dry or containing, by weight, less than 15% water
PBX(AF) 108
PBXW 108(E)
RDX
T4
1,3,5-TRIAZINE, HEXAHYDRO-1,3,5-TRINITRO-
s-triazine, hexahydro-1,3,5-trinitro-
TRIMETHYLEENTRINITRAMINE
TRIMETHYLENETRINITRAMINE
sym-TRIMETHYLENETRINITRAMINE
TRINITROCYCLOTRIMETHYLENE TRIAMINE
1,3,5-TRINITROHEXAHYDRO-s-TRIAZINE
1,3,5-TRINITRO-1,3,5-TRIAZACYCLOHEXANE
UN 0072
UN 0118

00088543

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0313.htm

00088544

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Styrene

CASRN 100-42-5

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0104
Styrene; CASRN 100-42-5

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Styrene

File On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	09/01/1990
Inhalation RfC Assessment (I.B.)	on-line	07/01/1993
Carcinogenicity Assessment (II.)	no data	

00088545

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Styrene
CASRN -- 100-42-5
Last Revised -- 09/01/1990

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

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

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Red blood cell and liver effects Dog Subchronic Oral Study Quast et al., 1979	NOAEL: 200 mg/kg-day LOAEL: 400 mg/kg-day	1000	1	2E-1 mg/kg-day

*Conversion Factors: none

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Quast, J.F., C.G. Humiston, R.Y. Kalnins, et al. 1979. Results of a toxicity study of monomeric styrene administered to beagle dogs by oral intubation for 19 months. Toxicology Research Laboratory, Health and Environmental Sciences, DOW Chemical Co., Midland, MI. Final Report.

Four beagle dogs/sex were gavaged with doses of 0, 200, 400, or 600 mg

00088546

styrene/kg bw/day in peanut oil for 560 days. No adverse effects were observed for dogs administered styrene at 200 mg/kg-day. In the higher dose groups, increased numbers of Heinz bodies in the RBCs, decreased packed cell volume, and sporadic decreases in hemoglobin and RBC counts were observed. In addition, increased iron deposits and elevated numbers of Heinz bodies were found in the livers. Marked individual variations in blood cell parameters were noted for animals at the same dose level. Other parameters examined were body weight, organ weights, urinalyses, and clinical chemistry. The NOAEL in this study is 200 mg/kg-day and the LOAEL is 400 mg/kg-day.

Long-term studies (120 weeks) in rats and mice (Ponomarkov and Tomatis, 1978) showed liver, kidney, and stomach lesions for rats (dosed weekly with styrene at 500 mg/kg) and no significant effects for mice (dosed weekly with 300 mg/kg). Rats receiving an average daily oral dose of 95 mg styrene/kg bw for 185 days showed no adverse effects, while those receiving 285 or 475 mg/kg-day showed reduced growth and increased liver and kidney weights (Wolf et al., 1956). Other subchronic rat feeding studies found LOAELs in the 350-500 mg/kg-day range and NOAELs in the range of 100-400 mg/kg-day.

The lifetime studies in rats and mice (Ponomarkov and Tomatis, 1978) are not appropriate for risk assessment of chronic toxicity because of the dosing schedule employed. The Wolf et al. (1956) study is of insufficient duration (185 days) to be considered chronic.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- 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 subchronic effects to chronic effects.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

None.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

The principal study is well done and the effect levels seem reasonable, but the small number of animals/sex/dose prevents a higher confidence than medium at this time. The data base offers strong support, but lacks a bona fide full-term chronic study; thus, it is also considered to have medium confidence. Medium confidence in the RfD follows.

00088547

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

U.S. EPA. 1984. Health and Environmental Effects Profile for Styrene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste, Washington, DC.

U.S. EPA. 1985. Drinking Water Criteria Document for Styrene. 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 ADI in the 1984 Health and Environmental Effects Profile document has received an Agency review with the help of two external scientists.

Agency Work Group Review -- 10/09/1985, 11/06/1985, 10/09/1985

Verification Date -- 10/09/1985

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Styrene
CASRN -- 100-42-5
Last Revised -- 07/01/1993

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is expressed in units of mg/cu.m. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (EPA/600/8-88/066F August 1989) and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994). RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

00088548

I.B.1. INHALATION RfC SUMMARY

Critical Effect	Exposures*	UF	MF	RfC
CNS effects Occupational Study Mutti et al. (1984)	NOAEL: 94 mg/cu.m (25 ppm = 150 mmole urinary styrene metabolites/mole creatinine adjusted to lower 95% confidence limit = 22 ppm) NOAEL(HEC): 34 mg/cu.m LOAEL: >94 mg/cu.m (>22 ppm derived as in NOAEL listing)	30	1	1E+0 mg/cu.m

*Conversion Factors: MW = 104.15. Assuming 25 C and 760 mmHg, NOAEL (mg/cu.m) = NOAEL (ppm) x MW/24.45 = 94 mg/cu.m. The NOAEL exposure level is based on a back extrapolation from worker urinary concentration of styrene metabolites reported in the principal study and adjusted to the lower 95% confidence limit listed in Guillemin et al. (1982), which was 88%, 25 ppm x 0.88 = 22 ppm. The NOAEL(HEC) is calculated using an 8-hour TWA occupational exposure. MVho = 10 cu.m/day, MVh = 20 cu.m/day. NOAEL(HEC) = 94 mg/cu.m x MVho/MVh x 5 days/7 days = 34 mg/cu.m. The feasibility of applying the exposure model of Perbellini et al. (1988) for extrapolation of the values in the principal study is currently being investigated. Application of this model may result in changes in the NOAEL(HEC) value and, therefore, the RfC.

I.B.2. PRINCIPAL AND SUPPORTING STUDIES (INHALATION RfC)

Mutti, A., A. Mazzucchi, P. Rusticelli, G. Frigeri, G. Arfini, and I. Franchini. 1984. Exposure-effect and exposure-response relationships between occupational exposure to styrene and neuropsychological functions. Am. J. Ind. Med. 5: 275-286.

In a cross-sectional study, Mutti et al. (1984) examined the neuro-psychological function in 50 workers whose mean duration of styrene exposure was 8.6 (SD of 4.5) years. Styrene exposure was assessed by the authors to correspond to air concentrations ranging from 10-300 ppm as a mean daily exposure. These concentrations were estimated from the summation of the principal urinary metabolites of styrene, mandelic acid (MA) and phenylglyoxylic acid (PGA). Urinary metabolite levels are considered as reliable biological indicators of styrene exposure (ACGIH, 1986; WHO, 1983), and several laboratories have determined collectively that the specific method used in this study, the summation of the principal metabolites collected in next-morning urine, is the most reliable and representative of actual air exposure concentrations (Guillemin et al., 1978, 1982; Ikeda et al., 1982; Franchini et al., 1983). Workers with absence of metabolic and neurologic disorders, smoking habits of <20 cigarettes/day, and an alcohol intake of <80 mL of ethanol/day were chosen. These same eligibility criteria were used to select a control group of 50 workers that was matched for age, sex, and educational level. The exposed workers were further segregated into four subgroups (n = 9-14) according to increasing levels of urinary styrene metabolites. A battery of neuropsychological tests was conducted on the same day as the urine collection and included exams evaluating visuo-motor speed, memory, and intellectual function. No other endpoints were considered. Correlation analysis of the test results and urinary metabolite levels showed a clear concentration response in at least three of eight tests, including block design (intellectual function), digit-symbol (memory), and reaction times (visuo-motor speed). Evidence of a concentration-response relationship

00088549

was also present for short- and long-term logical memory and embedded figures (impaired visual perception). When the results were analyzed using duration of exposure as a covariate, increases in reaction times and a decrease in digit symbol (memory, concentration) were apparent. The only test showing results in the lowest exposure group, short-term verbal memory loss, exhibited no concentration-response relationship. The neuropsychological results from this study are from established tests for CNS dysfunction, are present when compared against a stringently matched control population, and show concentration-response relationships. Also, the deficiencies noted in the reaction-times corroborate the results presented by Moller et al. (1990) and others discussed below.

The concentration-response relationship between urinary metabolite concentration (mandelic acid and phenylglyoxylic acid levels normalized to creatinine in "morning-after" urine) and test results indicated a significant effect level in the subgroup whose urine contained 150-299 mmole urinary metabolites/mole creatinine. Workers with metabolite concentrations of up to 150 mmoles/mole appeared to have no significant effects, and this level is therefore designated as the NOAEL in this study. The authors state that this level of urinary metabolites corresponds to a mean daily 8-hour exposure to air styrene of 25 ppm (106 mg/cu.m). Derivation of this air level is from the creatinine-normalized, combined concentration of the styrene metabolites, MA and PGA, in urine collected from the workers on Saturday mornings. Guillemin et al. (1982) demonstrated a logarithmic relationship (r = 0.871) between the summation of urinary metabolites (MA + PGA, next morning) and air concentrations of styrene (ppm x hours). Guillemin calculated the mean combined urinary metabolite concentration (next morning) for an 8-hour exposure to 100 ppm. This relationship was used by both Mutti et al. (1984) and Guillemin and Berode (1988) in a proportional manner to obtain styrene air levels at lower urinary metabolite concentrations. The 95% confidence interval was also calculated for an 8-hour exposure at 100 ppm, the lower limit of the confidence calculation being 88% of the mean styrene exposure. This factor was applied directly to the NOAEL of 25 ppm [25 ppm x 0.88 = 22 ppm (94 mg/cu.m)]. Due to the construction of the subgroups, designation of a LOAEL was the lower limit of the subgroup in which adverse effects were observed [i.e., greater than the NOAEL of 22 ppm (94 mg/cu.m)].

___I.B.3. UNCERTAINTY AND MODIFYING FACTORS (INHALATION RfC)

UF -- A partial UF of 3 was used for data base inadequacy, including the lack of concentration-response information on respiratory tract effects. A partial UF of 3 instead of 10 was used for intraspecies variability since the lower 95% confidence limit of the exposure extrapolation was used and because Perbellini et al. (1988) demonstrated that this biological exposure index (i.e., urinary metabolites) accounts for differences in pharmacokinetic/ physiologic parameters such as alveolar ventilation rate. A partial UF of 3 instead of 10 was also evoked for lack of information on chronic studies as the average exposure duration of the principal study of Mutti et al. (1984) was not long enough (8.6 years) to be considered chronic. The total uncertainty is therefore 30 (three times the one-half logarithm of 10).

MF -- None

___I.B.4. ADDITIONAL STUDIES / COMMENTS (INHALATION RfC)

Central nervous system effects caused by exposure to styrene have been

00088550

reported by several investigators in studies since the early 1970s. Gotell et al. (1972) noted significantly increased reaction times in a group of six polyester plant workers exposed to >150 ppm styrene as compared with unexposed controls and another group exposed to <150 ppm. Gamberle et al. (1975) noted longer and more irregular reaction times in 106 exposed workers (estimated at 13-101 ppm styrene for an average of 2.7 years) as compared with a control population. Cherry et al. (1980) found no alteration in reaction times among 27 workers exposed to a mean of 92 ppm styrene (duration unspecified) and no difference in performance on four behavioral tests of memory and vigilance. However, a follow-up study conducted 21 months later by these authors on 8 of the same 27 individuals (Cherry et al., 1981) found that the three men with the highest urinary mandelic acid concentrations all improved reaction times significantly ($p < 0.05$) as compared with their earlier values. These alterations were correlated with those individuals having slow clearance of mandelic acid. Among 98 male laminating workers (mean styrene exposure of 5.1 years), Lindstrom et al. (1976) reported weak correlations of CNS-behavioral testing (poor psychomotor performance and visuomotor inaccuracy) with levels of styrene exposure estimated to be between 25 and 75 ppm based on urinary mandelic acid levels. The study of Mutti et al. (1984) is one of few in which extensive CNS-behavioral testing was carried out. The recent studies of Moller et al. (1990) and Flodin et al. (1989) couple functional decrements with adverse behavioral effects in a chronically exposed worker population. The effect of styrene on peripheral nerves has also been reported, although not to the extent of central effects. These studies are briefly described in the recent study of Murata et al. (1991) in which data on a small number of workers suggest that styrene may differentially affect sensory nerves.

The studies of Moller et al. (1990) and Flodin et al. (1989) both examined central effects of styrene in the same worker population. The Flodin et al. (1989) study on neuropsychiatric effects of styrene exposure provides documentation of the styrene levels to which these workers were exposed.

In a cross-sectional occupational study, Moller et al. (1990) studied 18 male Swedish boatbuilders exposed to styrene for an average of 10.8 years (range of 6-15 years). Personal sampling (8-hour TWA concentrations) available for 7/10 years showed that the workers had been exposed to 50-140 mg/cu.m styrene. The exposure data is discussed at length in the text of the Flodin et al. (1989) study below. These workers were not further subgrouped into high-and low-exposure groups as in the Flodin et al. (1989) study. Two reference groups were used for evaluation of the tests; both were unexposed to industrial solvents and matched to the exposure group with respect to smoking and alcohol consumption. These workers were subsequently given a thorough otoneurological examination, evaluating auditory, visual, and vestibular systems, as well as coordination of vestibular sensations with compensatory eye movements manifest in the vestibuloocular reflex (VOR). The test showing the greatest and most consistent deviation in performance between the exposed workers and the reference population was the visual suppression of the VOR. Execution of this test required the subjects to fixate on a target that moved with movement of the subjects' chair. The normal VOR functions to maintain steady gaze of the eyes despite movements of the head. However, when the target moves with the subject, this reflex must be suppressed in order to follow the target. Quantitation of a subject's capacity to suppress VOR is measured as the ratio between eye and target velocity (gain) and in the temporal relationship between eye and target velocity (phase). Abnormal functioning would be manifest by an increase in gain and a decrease in phase, if the values exceeded the mean +/- 2 SD units of the corresponding measurement in a reference population. Abnormal phase shifts were recorded for 4/18 workers; in each of the four cases, the gain was also abnormal. These differences were reflected in the group values that showed a higher mean gain ($p < 0.001$) and a decrease in angle of phase lead ($p < 0.001$) in examinations carried out with predictable (sinusoidal frequency sweep) and unpredictable (pseudorandomized) conditions. The deficits shown in these tests suggest lesions in the brainstem or cerebellar regions, because these findings are in accordance with findings in patients with known brainstem or

00088551

cerebellar disorders (Odkvist et al., 1982, 1987). Other tests reported in this study also gave indications of neurological deficits. The posturography test demonstrated increases in sway area of subjects with their eyes closed, which are consistent with vestibular deficits. The overall results of this test are, however, internally inconsistent as more of these workers showed abnormal scores with their eyes open than with their eyes closed. Although examination of saccadic eye movements (brief, fast movements occurring with change in fixation point) did reveal abnormal scores in latency for 7/18 subjects, none of the 18 were found to have abnormal velocity. This also is an internal inconsistency within the exam, making these results equivocal. A similar internal inconsistency was noted in the results of the smooth-pursuit eye movement during which the subject is asked to visually track a slowly moving stimulus. Seven of the 18 subjects had abnormal results in lag time (phase), whereas none of the 18 tested abnormal for gain. Disturbances were found in the central auditory pathways of seven workers but the significance of these effects were not evaluated (see discussion of Pryor et al., 1987). This study provides credible toxicological information on the possible long-term consequences of human exposure to styrene. No effect levels are assigned based on these data as the number of subjects is small and no indication is given of a dose-response relationship among these chronically exposed workers.

Twenty-one male Swedish workers were exposed to styrene for an average of 11.6 years (range of 6-21 years) as a consequence of their occupation, boatbuilding (Flodin et al., 1989). Personal sampling (8-hour average concentrations) had been carried out in 9 of the last 12 years, with the number of sampling days for each year ranging from 3 to 27; the data presented indicate an exposure range of around 40-140 mg/cu.m during this time. In one year, 30-minute sampling times were performed on most tasks and showed that peak exposures were exceptional, with only about 1% of all measurements of styrene >300 mg/cu.m; about 44% of the samples were <25 mg/cu.m, and about 65% were <50 mg/cu.m. Based on these sampling data, the workers were classified into two groups: those exposed to about 50 mg/cu.m styrene for the past 7 years and those exposed to about 25 mg/cu.m. The higher exposed workers were primarily involved in lamination processes. Workers were examined by a clinical interview that included a detailed inquiry about neuropsychiatric symptoms; psychometrical tests and some clinical chemistry were also included. The workers were examined twice, the first time (21 workers examined) occurring when they had not been exposed for 1 week. The second examination (17 workers examined; nine in the higher exposure group and eight in the lower exposure group) occurred after the workers had not been exposed for a minimum of 3 months because the factory had gone bankrupt. Two of the remaining four workers refused participation in the second interview, and results from two others were disallowed as one was diagnosed as having pulmonary thrombosis and the other psycho-organic syndrome (POS) (see discussion of Moller et al., 1989). No concurrent control group was used. According to the questionnaire, the symptoms at the first examination were distributed in a exposure-related manner, with the higher exposure group having an average of 10.4 symptoms/worker and the lower exposure group having an average of 5.3 symptoms/worker. On the second examination, the average symptoms/worker decreased to 1.9 in both exposure groups. The claim that the higher exposure group performed significantly worse in one psychometrical test (manual dexterity) is not interpretable due to lack of specifics in the text. Internal inconsistencies of the results and the lack of a concurrent matched control group cause the results from this study to be equivocal. No effect levels were designated in this study.

The levels of styrene reported in the study of Mutti et al. (1984) were estimated from levels of urinary styrene metabolites. As already discussed, this method is considered as a biological indicator of exposure and has been shown to reliably reflect the total concentration of styrene to which individuals have been exposed. The relationships between styrene exposure and urinary metabolite concentrations have been established by a number of studies including those of Guillemin et al. (1982) and Ikeda et al. (1982) and reviewed by the WHO (1983). The work of Perbellini et al. (1988) has shown

00088552

that the variability of urinary metabolite levels observed in styrene-exposed worker populations may reflect physiologic and metabolic variability inherent in humans. These characteristics are not shared by the extensive studies on styrene levels reported by Lemasters et al. (1985a) and of Jensen et al. (1990), both of which are historical and are based on area and personal sampling procedures.

A diagnosis of psycho-organic syndrome has been used to describe the symptomatology observed in workers heavily exposed to a variety of organic solvents. Examples of specific effects include neurasthenia, personality alterations, unsteadiness, dizziness, and vertigo. Moller et al. (1989) examined nine men who had been diagnosed as having POS, subjecting them to a battery of audiological and vestibular-oculomotor tests, the latter of which measure the capacity to transform signals from the inner ear to compensatory eye movements for purposes such as maintenance of equilibrium. The men were described as having been exposed to various mixtures of alcoholic, aromatic, and aliphatic industrial solvents during their working careers of 8-30 years (mean = 21 years). Seven of them had been granted disability pensions at the time of the examinations. Abnormal static posture was noted in 4/9 members of the POS group ($p < 0.001$). Voluntary saccades (rapid intermittent movements of the eye) were abnormal (both prolonged latency and decreased maximum speed) in 5/9 of the POS group, but in only 2/9 matched controls. As these functions are considered to be controlled centrally in the area of the cerebellum, these findings indicate that cerebellar lesions may occur from chronic exposures to a variety of organic solvents. The results presented by Moller et al. (1990) for these tests in workers exposed to styrene indicate this chemical to be capable of eliciting this toxicity.

A duration-response between length of exposure to solvents and incidence of altered cerebellar functioning (as evidenced by effects on hearing and the vestibular-oculomotor system) is indicated in the study of Odkvist et al. (1987). This study examined 23 workers, all of whom had been extensively exposed to aliphatic and aromatic solvents. Sixteen of the 23 workers were diagnosed with POS. The average length of exposure of these 16 workers was 27 years (range 9-40 years), considerably more than the average of 21 years (range 5-30 years) for the remaining seven individuals. In the battery of 11 vestibular-oculomotor tests, five (including saccade, visual-suppression, Romberg's test, and electronystagmography) showed a dose-response relationship with the percentage incidences being higher in the group diagnosed with POS; the other six tests either were not conducted in one or the other group or exhibited no change. Thus, both the number of workers diagnosed with POS and incidence of cerebellar dysfunctioning were correlated with duration of exposure to organic solvents.

The specific capacity of styrene to cause alterations in cerebellar function in humans under short-term acute exposure conditions was experimentally shown by Odkvist et al. (1982). Ten people (5/sex) were exposed to 370-591 mg/cu.m styrene for 80 minutes. A battery of six vestibular-oculomotor tests was administered before, during, and after the exposure. Visual suppression and saccade tests both showed statistically significant alterations in 8/10 subjects. Results between exposed subjects and controls did not differ for the optovestibular, optokinetic, and slow pursuit movement test or the sinusoidal swing test. These results indicate that acute exposures to high concentrations of styrene may affect processes within the cerebellum. It should be noted, however, that the results obtained by Moller et al. (1990) and Flodin et al. (1989) were obtained in workers that had not been exposed to styrene for a minimum of 3 months.

Larsby et al. (1978) investigated the relationship between vestibulo-oculomotor function and styrene levels in arterial blood and cerebrospinal fluid. Rabbits (n = 16) were cannulated and infused with a 10% solution of styrene introduced at 3.1-12.6 mg/minute. Vestibular function was evaluated using electronystagmography in response to rotary acceleration. Styrene concentration was monitored in both arterial (ear) and cerebrospinal fluid.

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During the exposure, positional nystagmus (i.e., involuntary eye movement response to rotary movement evident only when the animal is lying on one side or the other, not prone) was observed in 10/11 rabbits tested. In addition, a paradoxical rotary response was observed in 6/16 rabbits. This phenomenon is described as involuntary eye movement opposite to the direction of rotation (left-beating eye movements when rotated clockwise and vice versa). Both observations indicate that vestibular function was affected. Results also showed that, at an infusion rate of 4.9 mg/minute, arterial blood levels reached a constant level only after about 2 hours. Styrene concentrations in cerebrospinal fluid had the same shape as the arterial curve and was constantly 5-10% of the corresponding arterial concentration.

A number of other occupational studies investigating the effects of styrene on workers are available in the literature. Most of these reports examine either central or peripheral nervous function, although blood effects (Stengel et al., 1990), nephrotoxicity (Viau et al., 1987), and liver effects (Hotz et al., 1980) have also been examined. Nearly all of these reports suffer from one or more deficiencies, the most common being lack of exposure information. Almost all, however, indicate that styrene affects central processes in humans. An overview of several of these studies follows.

In addition to the auditory data listed in the Moller et al. (1990) study, two other studies have reported hearing loss, one in humans and the other in laboratory animals. Muijser et al. (1988) evaluated hearing thresholds up to 16 kHz in 59 workers exposed to airborne styrene. Air samples (4-hour mean averages, 6-16 samples/group work area) were taken in the breathing zone during 3 consecutive days in work areas where 31 workers were directly exposed to styrene (mean = 138 mg/cu.m) and 28 workers were indirectly exposed to styrene (mean = 61 mg/cu.m). The duration of employment for the combined group was 8.6 +/- 6.5 years. The control population consisted of 88 individuals not exposed to styrene or other chemicals but who were comparable in age and socio-economic status to the exposed workers. Audiometric analyses were corrected for age. Comparison of hearing thresholds between the controls and both exposed groups revealed no differences in hearing thresholds at frequencies up to and including 16 kHz. Comparison between the experimental groups, however, did reveal that the directly exposed workers had higher thresholds for frequencies at 8-16 kHz than did the indirectly exposed workers (p value from multivariate analysis of covariance = 0.012). Quantitative and qualitative differences in the background noise between the control and study plants may have compromised the control population in this study. Although difficulties with the control population prevent definite assignment of effect levels, human exposure to 138 mg/cu.m styrene appears to have resulted in high-frequency hearing loss [LOAEL(HEC) = 69 mg/cu.m]. Pryor et al. (1987) exposed male Fisher 344/N rats (12/group) to 0 (clean air), 800, 1000, or 1200 ppm (0, 3408, 4260, or 5112 mg/cu.m, respectively) styrene for 14 hours/day, 7 days/week for 3 weeks. The duration-adjusted values are 0, 1988, 2485, or 2982 mg/cu.m, respectively. Auditory response thresholds were determined by both behavioral and electrophysiologic methods, apparently some weeks after the last exposure. Increases in auditory thresholds were recorded at 8-20 kHz with both methods at the lowest concentration used [LOAEL(HEC) = 1988 mg/cu.m].

Several occupational studies have reported adverse health effects on workers whose exposure was at or near 25 ppm (110 mg/cu.m) styrene. Lindstrom et al. (1976) reported that the visuomotor accuracy of styrene-exposed workers (n = 98, average exposure 4.9 years, range 0.5-14 years) was significantly poorer ($p < 0.05$) than that of the nonexposed workers. Moreover, this deficit was shown to be related to concentration in two subgroups whose exposures (estimated from urinary mandelic acid) were 25 and 75 ppm. Seppalainen and Harkonen (1976) conducted a cross-sectional study on 96 styrene workers, all of whom received EEG examinations 24 hours after termination of exposure. Mean exposure to styrene was estimated from urinary mandelic acid to be 808 mg/L, approximately 36 ppm in air. Twenty-three of 96 (24%) EEG's were abnormal in the exposed group as compared with a normal population (indicated

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as about 10% in the text), a difference that is statistically significant ($p < 0.05$).

Human irritation from styrene exposure has been characterized in a limited study by Stewart et al. (1968). Nine male volunteers were exposed to air concentrations of styrene of 50-375 ppm (213-1597 mg/cu.m) for periods of 1-7 hours. Urinalysis, hematology, and blood chemistry studies were conducted prior to exposure and at 16 and 72 hours postexposure; subjective symptoms were also recorded. Within 15 minutes following a 60-minute exposure to 375 ppm styrene, 4/5 volunteers complained of mild eye and nasal irritation. (Throughout the remainder of the exposure, all subjects noted a progressive loss of their ability to perceive the odor of styrene.) It is not clear from the text whether or not the irritation remained throughout the exposure. After 45 minutes of exposure, one of the subjects reported being nauseated and two others complained of feeling slightly inebriated. At 216 ppm styrene, 1/3 subjects noted nasal irritation 20 minutes into a 60-minute exposure. No adverse symptoms were reported by the three subjects exposed to 52 ppm styrene for 1 hour. Six subjects were then exposed to 99 ppm styrene for a total of 7 hours. Three of the six subjects complained of mild eye and throat irritation 20 minutes after the start of the exposure; in two of these subjects, the eye irritation persisted for 30 minutes before subsiding. At the end of the exposure, none of the subjects reported nausea, headache, or eye, nose, or throat irritation. The clinical studies were all normal. None of the six individuals exposed to 99 ppm (422 mg/cu.m) styrene for 7 hours or 216 ppm for 1 hour experienced any subjective symptoms of significant consequence. Although these results suggest no symptoms of consequence in humans exposed to styrene concentrations as high as 216 ppm (920 mg/cu.m), the population tested was small (only three subjects), and the duration was minimal.

Considerable success has been attained in modeling levels of inhaled styrene in biological systems. The physiologically based pharmacokinetic model for styrene of Ramsey and Andersen (1984) allows simulation over a wide range of concentrations on the time course of styrene distributed to four main tissue groups: (1) highly perfused organs, (2) moderately perfused organs (predominately muscle), (3) slowly perfused tissue (predominately fat), and (4) liver. When applied to actual rat data, this model accurately predicted blood styrene levels of 80-1200 ppm (340-5112 mg/cu.m). Then the behavior of inhaled styrene in humans was simulated successfully by substitution of human physiological parameters. These authors were able to demonstrate that blood concentrations of inhaled styrene in rats, mice, and humans were nearly identical at air concentrations of less than or equal to 200 ppm (852 mg/cu.m) but differed widely at higher concentrations. Perbellini et al. (1988) developed a physiologically based mathematical model for human exposure to airborne styrene that accounts for metabolism, subsequent synthesis, transfer, and urinary excretion of the principal metabolites MA and PGA. The model comprises eight compartments: (1) lung; (2) the richly perfused tissues of heart, brain, and kidney; (3) muscle; (4) fat; (5) liver tissue for catabolism of styrene; (6) liver tissue for transfer of metabolites; (7) body water in which the metabolites are distributed; and (8) urine in which the metabolites are excreted. Simulation results using this model were in agreement with reported urinary metabolite concentrations measured in various studies of worker populations, including that of Guillemin et al. (1982). Further simulations demonstrated that the use of urinary metabolites as a biological exposure index can accurately account for variability in pharmacokinetic/physiologic parameters such as the alveolar ventilation rate. Simulations using an alveolar ventilation rate of 6-12 L/minute resulted in less than a three-fold range in model output (urinary metabolite concentration).

Jersey et al. (1978) exposed Sprague-Dawley rats (96/sex/group) to 0, 600, or 1200 ppm (0, 2556, or 5112 mg/cu.m, respectively) of 99.5% styrene for 6 hours/day, 5 days/week for up to 20 months. The exposure concentration of the 1200 ppm exposure group was reduced to 1000 ppm (4260 mg/cu.m) after 2 months because the males showed signs of toxicity (narcosis leading to anesthesia and excessive weight loss) with death coming to three animals. Exposures were

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terminated when mortality reached 50% for one exposure group of each sex; this was at 18.3 months for males and 20.7 months for females. All surviving rats were euthanized at the end of 2 years with interim group samplings at 6 and 12 months. Hematology, clinical chemistry, body weights, gross anatomical and histopathological analysis, and cage-side observations were performed for evaluation of toxicity. The respiratory tract (including the lungs, trachea, and nasal turbinates) was not examined in all animals; the nasal turbinates, for example, were examined only in a portion of the controls and high-exposure animals (14/28 animals, sexes combined). The number of sections examined in the nasal turbinates, trachea, or lungs is not indicated in the text. No exposure-related increase in mortality was noted in either sex. An inverse relationship between mortality and exposure was noted in male rats. A high incidence of murine pneumonia was associated with an increased mortality, but only in the control and high-exposure animals; no dose-response relationship was apparent from the data. Average body weights of both females and males were decreased at both dose levels at various times throughout the experiment. However, only the body weights of the males exposed to the highest concentration were decreased more than 10% (14% maximum), consistently only during treatment days 82-263. The only concentration-dependent alteration in organ weights observed was in absolute and relative liver weights in females sacrificed at 6 months. At the terminal sacrifice, an increase in absolute liver weights was observed only in the females exposed to the highest concentration; no histopathology accompanied this alteration. The only histological result considered to be concentration-dependent was an increase in incidence of alveolar histiocytosis (areas containing lipid-laden alveolar macrophages) that corresponded to grossly visible subpleural pale foci in the lungs of the females exposed to the highest concentration. No concentration-related effects were reported in any groups for hematology, clinical chemistry, or urinalysis. Deficiencies in this study preclude assigning effect levels.

Conti et al. (1988) exposed Sprague-Dawley rats (30/sex/dose) to 0, 25, 50, 100, 200, or 300 ppm (0, 106, 213, 426, 852, or 1278 mg/cu.m, respectively) styrene for 4 hours/day, 5 days/week for 52 weeks. The animals were kept under observation until spontaneous death. Histopathologic examinations were performed on each animal; tissues examined included brain, liver, kidneys, gonads, spleen, and pancreas. The lungs were apparently the only portion of the respiratory tract examined in this study. No noncancer results were reported or discussed in this study. No effect levels could be assigned in this study.

Effects of styrene on the respiratory tract have been addressed in mouse subchronic studies by the NTP (NTP, 1991a). B6C3F1 mice (10/sex/group) were exposed to 0, 62.5, 125, 250, or 500 ppm (0, 266, 532, 1065, or 2130 mg/cu.m, respectively) styrene for 6 hours/day, 5 days/week for 13 weeks. The duration-adjusted values for this exposure regime are 0, 47.5, 95, 190, or 380 mg/cu.m. Body weight changes, hematology, serum chemistry, sperm morphology, vaginal cytology, gross pathology, and histopathology (including the entire respiratory tract) were monitored for toxicity. Death occurred in the first week of exposure but only in the males exposed to 250 ppm; no deaths at the highest concentration were noted. Histopathology of these animals showed evidence of thymic and renal cortical necrosis. In exposed female mice, the average liver to body weight ratio was increased in the animals at the two highest concentrations. Histopathology revealed concentration-related increases in centrilobular liver cytomegaly, karyomegaly, and necrosis at these same concentrations with no effects being recorded at 125 ppm styrene or below. The lung to body weight ratios were increased at all levels relative to the control values. Histopathology of the respiratory tract revealed that the incidence of metaplasia and degeneration of the olfactory epithelium of the nasal cavity were already total (10/10 mice) in females at the lowest concentration, with necrosis being observed at higher concentrations. Likewise, bronchiolar regeneration was present in all female animals at all concentrations. The incidence of epithelial hyperplasia of the forestomach was also maximal at the lowest concentration. Similar results were noted for

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the males. No NOAEL for either respiratory or extrarespiratory effects was achieved in this study. The LOAEL(ADJ) for this study would be 47.5 mg/cu.m. The effects described occurred in both the nasal cavity [extrathoracic (ET)] and bronchiolar region [tracheobronchiolar (TB)]. The LOAEL(HEC) would therefore be based on effects in these regions with an RGDR of 1.55 = 74 mg.cu.m.

In a rat subchronic study (NTP, 1991b), F344/N rats (10/sex/group) were exposed to 0, 125, 250, 500, 1000, or 1500 ppm (0, 532, 1065, 2130, 4260, or 6390 mg/cu.m, respectively) styrene for 6 hours/day, 5 days/week for 13 weeks. The duration-adjusted values for this exposure regime are 0, 95, 190, 380, 761, or 1141 mg/cu.m. The same toxicological endpoints as in the mouse study were monitored. No deaths were recorded. Liver to body weight ratios were elevated at the three highest exposure levels in males and the two highest exposure levels in females, although no histopathology accompanied this alteration. Concentration-related goblet-cell hypertrophy was noted in the nasopharyngeal duct starting at 125 ppm styrene in males and at 250 ppm in females. Concentration-related degeneration of the olfactory epithelium (described as minimal to mild) was noted starting at 1000 ppm styrene in females and at 1500 ppm in males. Degeneration of the olfactory epithelium was noted also at the two highest exposure levels in both sexes. A NOAEL of 500 ppm is designated for extrathoracic effects [NOAEL(ADJ) = 380 mg/cu.m x RGDR of 0.107 = NOAEL(HEC) = 41 mg/cu.m].

The effect of styrene on the trachea of rats was addressed in two studies conducted by Ohashi et al. (1985, 1986). Both ciliary activity and histopathology were evaluated. Male Sprague-Dawley rats (10/exposure group and 10 controls) were exposed to styrene at 30 or 800 ppm for 8 consecutive weeks or at 150 or 1000 ppm for 3 consecutive weeks. Nasal and tracheal mucosa were examined by electron microscopy, immediately and several weeks after cessation of exposure. The 8-week study (Ohashi et al., 1985) found that there was a strong increase in mucus secretion, with an increase in number of dense bodies in the nasal, but not tracheal, mucosal cells after exposure to 30 ppm styrene. There was an increase in secretory granules in goblet cells at 3-weeks postexposure. At the higher concentration in the 8-week exposure, there was a marked increase in mucus secretion, vacuolation, and sloughing of epithelial cells in both the nasal and tracheal epithelium. Compound cilia were observed, together with nuclear pyknosis, vacuolization of epithelial cells, and changes in electron density in goblet cells, which also exhibited cores with high electron density. Sloughing of epithelial cells from the basement membrane also persisted at 3-weeks postexposure. Severe ciliary denudation was observed in the high-exposure group (1000 ppm styrene) in the 3-week exposure, with ciliary activity in the nose disabled and decreased to 18% of control values in the trachea. No effect levels were designated from these studies as the quantitative relationship between ciliary activity and mucus transport is not clear. In his review of mucociliary transport, Wanner (1977) suggests considerable functional reserve of this system; in chicken trachea 30-50% of particle transport activity was present at a time when only 10% of the epithelium was ciliated.

In two studies, Lemasters et al. (1985b, 1989) examined the reproductive outcomes of female workers involved in plastics manufacturing. In the 1985 study, data from a total of 174 styrene-exposed and 449 unexposed women were collected and analyzed. No increased prevalence in menstrual disorders was observed in subgroups of the workers exposed to either 13 or 52 ppm styrene. In the 1989 study, the authors examined the relationship between styrene exposure and lowered birth weights. During the study, the authors collected and analyzed data from 819 no-, 154 low- (2-29 ppm), and 75 high- (30-82 ppm) exposed pregnancies. There was not a statistically significant concentration-response relationship in decreasing average birth weights. In women who worked at the most highly exposed jobs (estimated at 82 ppm), however, a 4% reduction in average birth weight that approached statistical significance ($p = 0.08$), despite the small sample size ($n = 50$), was detected.

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Murray et al. (1978) exposed pregnant Sprague-Dawley rats and New Zealand rabbits to inhaled styrene at concentrations of 0, 300, or 600 ppm (0, 1278, or 2556 mg/cu.m., respectively) for 7 hours/day from gestation days 6-15 (rats) and 6-18 (rabbits). No concentration-related developmental toxicity was evident in either species by either route. Adverse maternal effects (decreased food consumption and a $p < 0.05$ decrease in weight gain only during the first 3 days of exposure) were noted. This study identifies a freestanding NOAEL for developmental effects of 2556 mg/cu.m.

Beliles et al. (1985) conducted a three-generation reproductive study concomitantly with a 2-year chronic study of exposure of rats to styrene in their drinking water. Sprague-Dawley rats were treated with monomeric styrene in their drinking water at 0, 125, or 250 ppm. These doses corresponded to 8-14 mg/kg/day for males and 12-21 mg/kg/day for females. After animals were dosed for 90 days, 20 females and 10 males from the styrene groups and 30 females and 15 males from the controls were used for the F0 generation and then returned to the chronic study. Representatives of these pups, the F1 generation, were then exposed until they were 110 days of age, at which time they were mated to produce the F2 generation. The F3 generation was produced in the same manner. Each generation was evaluated for fertility (male and female), litter size, pup viability, pup survival, sex ratio, pup body weight, weanling liver and kidney weight, physical and behavioral abnormalities on each day of lactation, and marrow cytogenetics. Reduction in the gestation and 1-, 7-, and 14-day survival indices of the high dose F2 pups was observed. A reduction in survival was also noted among the high dose F1 pups, but only at 21 days. No other evidences of fetotoxicity were noted. Although the authors claim these effects to be due to extensive losses in only 1 or 2 litters, only data on individual fetuses is presented. The high-dose level is designated as a NOAEL for reproductive effects.

Kankaanpaa et al. (1980) exposed pregnant BMR/T6T6 mice (15 controls and 13 exposed) to 250 ppm (1065 mg/cu.m) >99% pure styrene on gestation days 6-16 for 6 hours/day. Parameters monitored included number of litters and fetuses (total, live, dead, and malformed). No description of maternal toxicity is given, although narrative is provided on two preliminary experiments, one conducted at 500 ppm (2130 mg/cu.m) in which 2/6 pregnant mice and the surviving females had a fetal death rate of 47%. The other experiment was conducted at 700 ppm (2982 mg/cu.m) in which 3/5 pregnant animals died, with the surviving dams having a 95% fetal death rate. No dams died during the 250 ppm experiment, and the difference in the fetal death rate between controls and exposed dams was not statistically significant (27% vs. 18% in the controls; $p < 0.10$). The number of malformed fetuses was also increased in the exposed vs. the control mice (2.9% vs. 0.9%), but no statistical analysis was performed. A steep concentration response is indicated by this study: 500 ppm bringing death to both dams and fetuses, whereas 250 ppm appears to be without effect [NOAEL(HEC) = 1065 mg/cu.m]. These authors also exposed pregnant Chinese hamsters (2-7/treatment group and 15 controls) to 0, 300, 500, 750, or 1000 ppm (1278, 2130, 3195, or 4260 mg/cu.m, respectively) styrene for 6 hours/day on gestation days 6-18. Although the small number of animals limits the interpretation of this study, the highest concentration appears to be an effect level [LOAEL(HEC) = 4260 mg/cu.m], as the number of dead or resorbed fetuses was 66% as compared with 26% in the controls. There were no incidences of malformed fetuses in any treatment group or in the controls.

___I.B.5. CONFIDENCE IN THE INHALATION RfC

Study -- Medium
Data Base -- Medium
RfC -- Medium

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The study of Mutti et al. (1984) documents concentration-response relationships of CNS effects in a relatively small worker population. However, the results of this study are consistent with a number of other studies showing central effects in chronically exposed worker populations, most notably that of Moller et al. (1990). The urinary metabolites, MA and PGA, are direct biological indicators of exposure to styrene. Numerous studies have demonstrated the relationship between urinary metabolites and air levels of styrene to be reliable and quantitative. Physiologically based pharmacological modeling of this exposure methodology demonstrates that it reflects and incorporates at least a portion of intrahuman variability related to pharmacokinetics. The study is therefore assigned a medium confidence level. The data base can be considered medium to high as chronic laboratory animal studies addressing noncancer endpoints are not yet available, but a number of human exposure studies support the choice of critical effect. Preliminary information in mice indicate that styrene is a respiratory tract irritant in mice at concentrations lower than 47.5 mg/cu.m. The RfC is assigned an overall confidence rating of medium.

___I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

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

Other EPA Documentation -- U.S. EPA 1984a,b, 1985, 1989, 1991

Agency Work Group Review -- 09/20/1989, 03/26/1992

Verification Date -- 03/26/1992

___I.B.7. EPA CONTACTS (INHALATION RfC)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Styrene
CASRN -- 100-42-5

Not available at this time.

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_VI. BIBLIOGRAPHY

Substance Name -- Styrene
CASRN -- 100-42-5
Last Revised -- 11/01/1992

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VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

None

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_VII. REVISION HISTORY

Substance Name -- Styrene
CASRN -- 100-42-5

Date	Section	Description
06/30/1988	I.A.7.	Contacts changed
10/01/1989	I.B.	Inhalation RfD now under review
02/01/1990	VI.	Bibliography on-line
05/01/1990	I.A.	Oral RfD summary noted as pending change
05/01/1990	II.	Carcinogen assessment now under review
09/01/1990	I.A.	Text edited
09/01/1990	III.A.	Health Advisory on-line
09/01/1990	VI.D.	Health Advisory references added
01/01/1992	IV.	Regulatory actions updated
11/01/1992	I.B.	Inhalation RfC summary on-line
11/01/1992	VI.B.	Inhalation RfC references on-line
07/01/1993	I.B.1.	'E' notation added

VIII. SYNONYMS

Substance Name -- Styrene
CASRN -- 100-42-5
Last Revised -- 01/31/1987

100-42-5
BENZENE, VINYL-
CINNAMENE
CINNAMENOL
CINNAMOL
DIAREX HF 77
ETHENYLBENZENE
ETHYLENE, PHENYL-
NCI-C02200
PHENETHYLENE
PHENYLETHENE
PHENYLETHYLENE
STIROLO
STYREEN
STYREN
Styrene
STYRENE, MONOMER
STYROL
STYROLE
STYROLENE
STYROPOL

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STYROPOR
UN 2055
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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0104.htm

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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Tetrachloroethylene

CASRN 127-18-4

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0106
Tetrachloroethylene; CASRN 127-18-4

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Tetrachloroethylene

File On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	03/01/1988
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	no data	

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I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Tetrachloroethylene
CASRN -- 127-18-4
Last Revised -- 03/01/1988

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. 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)			

*Conversion Factors: Doses have been adjusted for treatment schedule (5 days/week)

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

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

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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.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- 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.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

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).

___I.A.5. CONFIDENCE IN THE ORAL RfD

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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.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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.

Agency Work Group Review -- 05/20/1985, 08/05/1986, 09/17/1987

Verification Date -- 09/17/1987

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Tetrachloroethylene
CASRN -- 127-18-4

Not available at this time.

00088569

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Tetrachloroethylene
CASRN -- 127-18-4

Not available at this time.

_VI. BIBLIOGRAPHY

Substance Name -- Tetrachloroethylene
CASRN -- 127-18-4
Last Revised -- 07/01/1989

__VI.A. ORAL RfD REFERENCES

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.

Carpenter, C.P. 1937. The chronic toxicity of tetrachloroethylene. J. Ind. Hyg. Toxicol. 19(7): 323-336.

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.

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.

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.

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.

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.

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.

U.S. EPA. 1985. Health Assessment Document for Tetrachloroethylene (perchloroethylene). Prepared by the Office of Health and Environmental

00088570

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.

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.

_VI.B. INHALATION RfC REFERENCES

None

_VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

None

_VII. REVISION HISTORY

Substance Name -- Tetrachloroethylene
CASRN -- 127-18-4

Date	Section	Description
12/23/1987	I.A.	RfD withdrawn pending further review
03/01/1988	I.A.	Revised Oral RfD sumary added - RfD changed
03/01/1988	III.A.	Health Advisory added
07/01/1989	VI.	Bibliography on-line
05/01/1990	II.	Carcinogen assessment now under review
06/01/1990	IV.A.1.	Area code for EPA contact corrected
06/01/1990	IV.F.1.	EPA contact changed
01/01/1992	IV.	Regulatory actions updated
04/01/1992	IV.	Regulatory action section withdrawn

VIII. SYNONYMS

00088571

Substance Name -- Tetrachloroethylene
CASRN -- 127-18-4
Last Revised -- 01/31/1987

127-18-4
Ankilostin
Antisal 1
Antisol 1
Carbon bichloride
Carbon dichloride
Czterochloroetylen
Dee-Solv
Didakene
Didokene
Dowclene EC
Dow-Per
ENT 1,860
Ethene, tetrachloro-
Ethylene tetrachloride
Ethylene, tetrachloro-
Fedal-Un
NCI-C04580
Nema
PCE
PER
Perawin
PERC
Perchloorethyleen, per
Perchlor
Perchloraethylen, per
Perchlorethylene
Perchlorethylene, per
Perchloroethylene
Perclene
Percloroetilene
Percosolv
Percosolve
PERK
Perklone
Persec
Tetlen
Tetracap
Tetrachlooretheen
Tetrachloraethen
Tetrachlorethylene
Tetrachloroethene
Tetrachloroethylene
1,1,2,2-Tetrachloroethylene.
Tetracloroetene
Tetraguer
Tetraleno
Tetralex
Tetravec
Tetroguer
Tetropil
WLN: GYGUYGG

00088572

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0106.htm

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

1,1,2-Trichloroethane

CASRN 79-00-5

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0198
1,1,2-Trichloroethane; CASRN 79-00-5

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR 1,1,2-Trichloroethane

File On-Line 03/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	02/01/1995
Inhalation RfC Assessment (I.B.)	no data	12/01/1992
Carcinogenicity Assessment (II.)	on-line	02/01/1994

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I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- 1,1,2-Trichloroethane
CASRN -- 79-00-5
Last Revised -- 02/01/1995

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Clinical serum chemistry	NOAEL: 20 mg/L (drinking water) (3.9 mg/kg/day)	1000	1	4E-3 mg/kg/day
Mouse Subchronic Drinking Water Study	LOAEL: 200 mg/L (drinking water) (44 mg/kg/day)			
White et al., 1985 Sanders et al., 1985				

*Conversion Factors: None

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Sanders, V.M., K.L. White, Jr., G.M. Shopp, Jr. and A.E. Munson. 1985. Humoral and cell-mediated immune status of mice exposed to 1,1,2-trichloroethane. Drug Chem. Toxicol. 8(5): 357-372.

White, K.L., Jr., V.M. Sanders, V.W. Barnes, G.M. Shopp, Jr. and A.E. Munson. 1985. Toxicology of 1,1,2-trichloroethane in the mouse. Drug Chem. Toxicol. 8(5): 333-355.

The studies by White et al. (1985) and Sanders et al. (1985) have been

selected as the basis for the derivation of the oral RfD because they are adequate studies in which mice of both sexes were exposed to 1,1,2-trichloroethane in drinking water for 90 days. Concentrations provided were 0, 20, 200, or 2000 mg/L, which resulted in intakes of 0, 4.4, 46, and 305 mg/kg/day for males and 0, 3.9, 44, and 384 mg/kg/day for females. Clinical chemistry indications of adverse effects on the liver occurred in both sexes at 2000 mg/L. Effects on the erythrocytes occurred only in females and depressed humoral immune status occurred in both sexes at 200 and 2000 mg/L. The concentration of 20 mg/L, corresponding to 3.9 mg/kg/day for female rats, (White et al., 1985; Sanders et al., 1985) is the NOAEL at which significant adverse effects were not observed and is chosen as the basis for the derivation of an oral RfD.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- The UF includes the standard uncertainty factors for interspecies and intrahuman variability, and a factor of 10 for extrapolation to lifetime exposure from an intermediate exposure duration.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

The previous oral RfD (2E-1 mg/kg/day) was based on the NCI (1978) study in rats. This study's major weakness was its lack of reporting of noncancer effects. Also, doses far below the NCI (1978) NOAEL (65.7 mg/kg/day) have been shown to alter levels of clinical serum chemistries, which are indicative of systemic tissue damage.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

The critical study is given a medium confidence for balanced strengths (clinical chemistries) and weaknesses (lack of histopathology and a NOAEL). The supporting data base and the RfD are rated medium because of the general lack of appropriate chronic toxicologic data.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

U.S. EPA. 1987. Drinking Water Health Advisory for 1,1,2-Trichloroethane. 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-W027.

U.S. EPA. 1980. Ambient Water Quality Criteria for Chlorinated Ethanes. 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 440/5-80-029. NTIS PB 81-117400.

Agency Work Group Review -- 12/18/1985, 05/15/1986, 05/26/1988

Verification Date -- 05/26/1988

I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- 1,1,2-Trichloroethane
CASRN -- 79-00-5

Not available at this time.

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- 1,1,2-Trichloroethane
CASRN -- 79-00-5
Last Revised -- 02/01/1994

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for

00088577

Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- C; possible human carcinogen

Basis -- Hepatocellular carcinomas and pheochromocytomas in one strain of mice forms the basis for this classification. Carcinogenicity was not shown in rats. 1,1,2-Trichloroethane is structurally related to 1,2-dichloroethane, a probable human carcinogen.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. ANIMAL CARCINOGENICITY DATA

In a bioassay conducted by NCI (1978) technical grade (92.7% pure) 1,1,2-trichloroethane was administered by gavage in corn oil to Osborne-Mendel rats and B6C3F1 mice: (50/species/sex/dose) for each of 2 doses and 20 animals/species/sex for each of 2 control groups. Administration was 5 times/week for 78 weeks during which time doses for rats were increased from 70 and 30 mg/kg/ day to 100 and 50 mg/kg/day and doses for mice were increased from 300 and 150 mg/kg/day to 400 and 200 mg/kg/day. By two statistical tests, treatment of mice was found to be associated with increased incidence of hepatocellular carcinomas. A dose-related increase in pheochromocytomas was also confirmed in female mice. Tumors found in treated but not control rats included adrenal cortical carcinomas; transitional-cell carcinomas of kidney; renal tubular adenomas; and hemangiosarcomas of spleen, pancreas, abdomen and subcutaneous tissue. There was, however, no statistically significant increase in tumor incidence in rats as a function of treatment.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

1,1,2-Trichloroethane was found to be nonmutagenic for Salmonella typhimurium (Simmon et al., 1977). In rats and mice acutely exposed to 1,1,2-trichloroethane by inhalation and intraperitoneal injection, trichloroacetic acid, trichloroethanol, chloroacetic acid and thiodiacetic acid were among the urinary metabolites identified (Yllner, 1971; Ikeda and Ohtsuji, 1972). 1,1,2-Trichloroethane is structurally related to 1,2-

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dichloroethane, a probable human carcinogen.

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 5.7E-2 per (mg/kg)/day

Drinking Water Unit Risk -- 1.6E-6 (ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

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+0 ug/L
E-6 (1 in 1,000,000)	6E-1 ug/L

II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- hepatocellular carcinoma
Test Animals -- Mouse/B6C3F1
Route -- gavage
Reference -- NCI, 1978

Administered Dose (mg/kg)/day	Human Equivalent Dose (mg/kg)/day	Tumor Incidence
0	0	2/20
139	9.3	18/49
279	18.6	37/49

II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

Doses are TWAs adjusted for frequency of exposure (5/7 days). Weight of the mice was assumed to be 0.033 kg.

The unit risk should not be used if the water concentration exceeds 6E+3 ug/L, since above this concentration the unit risk may not be appropriate.

II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

Dose-related increases in hepatocellular carcinomas were observed in adequate numbers or mice of both sexes. Background incidence of this tumor type is generally high. Modeling was done on only one data set.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

___II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk -- 1.6E-5 per (ug/cu.m)

Extrapolation Method -- Linearized multistage procedure, extra risk

Air Concentrations at Specified Risk Levels:

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

___II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSUR

This inhalation risk estimate was calculated from the oral exposure data in Section II.B.2.

___II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE

The unit risk should not be used if the air concentration exceeds 6E+2 ug/cu.m, since above this concentration the unit risk may not be appropriate.

___II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOS

See II.B.4.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1980

The values in the Ambient Water Quality Criteria Document for Chlorinated Ethanes received extensive peer and public review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 07/23/1986

Verification Date -- 07/23/1986

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- 1,1,2-Trichloroethane
CASRN -- 79-00-5
Last Revised -- 08/01/1990

__VI.A. ORAL RfD REFERENCES

NCI (National Cancer Institute). 1978. Bioassay of 1,1,2-trichloroethane for possible carcinogenicity. U.S. DHEW Tech. Rep. Ser. 74. Publ. No. NIH 78-1324.

Sanders, V.M., K.L. White, Jr., G.M. Shopp, Jr. and A.E. Munson. 1985. Humoral and cell-mediated immune status of mice exposed to 1,1,2-trichloroethane. Drug Chem. Toxicol. 8(5): 357-372.

U.S. EPA. 1980. Ambient Water Quality Criteria for Chlorinated Ethanes. 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 440/5-80-029. NTIS PB 81-117400.

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U.S. EPA. 1987. Drinking Water Health Advisory for 1,1,2-Trichloroethane. 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-W027.

White, K.L., Jr., V.M. Sanders, D.W. Barnes, G.M. Shopp, Jr. and A.E. Munson. 1985. Toxicology of 1,1,2-trichloroethane in the mouse. Drug Chem. Toxicol. 8(5): 333-355.

VI.B. INHALATION RfD REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Ikeda, M. and H. Ohtsuji. 1972. A comparative study of the excretion of Fujiwara reaction-positive substances in urine of humans and rodents given trichloro- or tetrachloro-derivatives of ethane and ethylene. Brit. J. Industr. Med. 29: 99-104.

NCI (National Cancer Institute). 1978. Bioassay of 1,1,2-trichloroethane for possible carcinogenicity. U.S. DHEW Tech. Rep. Ser. 74. Publ. No. NCI-CG-TR-74.

Simmon, V.F., K. Kauhanon and R.G. Tardiff. 1977. Mutagenic activity of chemicals identified in drinking water. In: Progress in Genetic Toxicology, D. Scott et al., Ed. Elsevier/North Holland Biomedical Press, Amsterdam. p. 249-258.

Yllner, S. 1971. Metabolism of 1,1,2-Trichloroethane - 1,2-14C in the mouse. Acta Pharmacol. et Toxicol. 30: 248-256. From the National Institute of Occupational Health, Stockholm, Sweden.

U.S. EPA. 1980. Ambient Water Quality Criteria for Chlorinated Ethanes. 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 440/5-80-029. NTIS PB 81-117400.

VII. REVISION HISTORY

Substance Name -- 1,1,2-Trichloroethane
CASRN -- 79-00-5

00088582

Date	Section	Description
09/30/1987	IV.	Regulatory Action section on-line
03/01/1988	II.B.4.	Confidence statement revised
03/01/1988	II.C.4.	Confidence statement revised
09/26/1988	I.A.	Oral RfD replaced
03/01/1990	I.B.	Inhalation RfC now under review
06/01/1990	IV.F.1.	EPA contact changed
06/01/1990	VI.	Bibliography on-line
08/01/1990	I.A.	Text edited
08/01/1990	II.	Text edited
08/01/1990	III.A.	Health Advisory on-line
08/01/1990	VI.D.	Health Advisory references added
01/01/1991	II.	Text edited
01/01/1991	II.C.1.	Inhalation slope factor removed (global change)
01/01/1992	I.A.7.	Secondary contact changed
01/01/1992	IV.	Regulatory actions updated
12/01/1992	I.B.	Work group review date added
02/01/1994	II.D.3.	Secondary contact's phone number changed
02/01/1995	I.A.7.	Secondary contact's name changed

VIII. SYNONYMS

Substance Name -- 1,1,2-Trichloroethane
CASRN -- 79-00-5
Last Revised -- 03/31/1987

79-00-5
ETHANE TRICHLORIDE
ETHANE, 1,1,2-TRICHLORO-
NCI-C04579
RCRA WASTE NUMBER U227
RCRA WASTE NUMBER U359
beta-T
1,1,2-TRICHLORETHANE
1,1,2-Trichloroethane
1,2,2-TRICHLOROETHANE
Trichloroethane, 1,1,2-
beta-TRICHLOROETHANE
TROJCHLOROETAN(1,1,2)
VINYL TRICHLORIDE

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00088583


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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0198.htm

00088584

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Trichloroethylene

CASRN 79-01-6

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0199
Trichloroethylene; CASRN 79-01-6

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Trichloroethylene

File On-Line 03/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	08/01/1992
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	withdrawn	07/01/1994

00088585

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Trichloroethylene
CASRN -- 79-01-6

Not available at this time.

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Trichloroethylene
CASRN -- 79-01-6

Not available at this time.

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Trichloroethylene
CASRN -- 79-01-6

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 -- 12/04/1986, 04/06/1989, 05/30/1989, 09/22/1993, 06/09

EPA Contacts:

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Trichloroethylene
CASRN -- 79-01-6

Not available at this time.

_VII. REVISION HISTORY

Substance Name -- Trichloroethylene
CASRN -- 79-01-6

Date	Section	Description
03/01/1988	II.B.3.	Text revised
03/01/1988	II.B.4.	Confidence statement revised
03/01/1988	II.C.2.	Text added
03/01/1988	II.C.4.	Confidence statement revised
03/01/1988	II.D.4.	Documentation corrected
05/01/1989	II.	Carcinogen assessment summary noted as pending change
06/01/1989	II.D.3.	Primary contact changed
07/01/1989	II.	Withdrawn; new assessment verified (in preparation)
12/01/1989	I.B.	Inhalation RfD now under review
06/01/1990	IV.A.1.	Area code for EPA contact corrected
06/01/1990	IV.F.1.	EPA contact changed
01/01/1992	IV.	Regulatory actions updated
04/01/1992	IV.A.1.	CAA regulatory action withdrawn
07/01/1992	II.	EPA contact changed; work group review dates added
08/01/1992	I.A.	Oral RfD now under review
11/01/1993	II.	Work group review date added
07/01/1994	II.	Work group review date added

VIII. SYNONYMS

Substance Name -- Trichloroethylene
CASRN -- 79-01-6
Last Revised -- 03/31/1987

79-01-6
ACETYLENE TRICHLORIDE
ALGYLEN

00088587

ANAMENTH
BENZINOL
BLACOSOLV
BLANCOSOLV
CECOLENE
CHLORILEN
1-CHLORO-2,2-DICHLOROETHYLENE
CHLORYLEA
CHLORYLEN
CHORYLEN
CIRCOSOLV
CRAWHASPOL
DENSINFLUAT
1,1-DICHLORO-2-CHLOROETHYLENE
DOW-TRI
DUKERON
ETHINYL TRICHLORIDE
ETHYLENE TRICHLORIDE
ETHYLENE, TRICHLORO-
FLECK-FLIP
FLOCK FLIP
FLUATE
GEMALGENE
GERMALGENE
LANADIN
LETHURIN
NARCOGEN
NARKOGEN
NARKOSOID
NCI-C04546
NIALK
PERM-A-CHLOR
PERM-A-CLOR
PETZINOL
PHILEX
RCRA WASTE NUMBER U228
TCE
THRETHYLEN
THRETHYLENE
TRETHYLENE
TRI
TRIAD
TRIAL
TRIASOL
TRICHLOORETHEEN
TRICHLOORETHYLEEN, TRI
TRICHLORAETHEN
TRICHLORAETHYLEN, TRI
TRICHLORAN
TRICHLOREN
TRICHLORETHENE
TRICHLORETHYLENE
TRICHLORETHYLENE, TRI
TRICHLOROETHENE
Trichloroethylene
1,1,2-TRICHLOROETHYLENE
1,2,2-TRICHLOROETHYLENE
TRI-CLENE
TRICLORETENE
TRICLOROETILENE
TRIELENE
TRIELIN
TRIELINA
TRIKLONE

00088588

TRILEN
TRILENE
TRILINE
TRIMAR
TRIOL
TRI-PLUS
TRI-PLUS M
UN 1710
VESTROL
VITRAN
WESTROSOL

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0199.htm

00088589

United States Environmental Protection Agency

IRIS
Integrated Risk Information System

Trichlorofluoromethane

CASRN 75-69-4

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0120
Trichlorofluoromethane; CASRN 75-69-4

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Trichlorofluoromethane

File On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	08/01/1992
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	no data	

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Trichlorofluoromethane
CASRN -- 75-69-4
Last Revised -- 08/01/1992

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Survival and histopathology	NOAEL: none	1000	1	3E-1 mg/kg/day
Cancer Bioassay Studies in Rats and Mice	LOAEL: 488 mg/kg/day converted to 349 mg/kg/day			

NCI, 1978

*Conversion Factors: 5 days/7 days; thus, 488 mg/kg/day x 5 days/7 days = 349 mg/kg/day

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

NCI (National Cancer Institute). 1978. Bioassay of trichlorofluoromethane for possible carcinogenicity. Report. No. 106, PHS/NIH, DHEW Publ. No. 78-1356.

The NCI bioassay was performed on rats and mice exposed to various doses of trichloromonofluoromethane by gavage over a period of 78 weeks (50 animals/species/sex/dose for each of two doses with 20 animals/species/sex for each of two control groups). A statistically significant positive association

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between increased dosage and accelerated mortality by the Tarone test in male and female rats and female mice were observed. In treated rats of both sexes there were also elevated incidences of pleuritis and pericarditis not seen in controls. Inhalation studies which employed multispecies exposures to higher levels of the compound than used by NCI (Leuschner et al., 1983) reported no adverse clinical/pathologic signs of toxicity due to subchronic or short-term exposures.

The LOAEL of 488 mg/kg/day (based on mortality in rats) was converted to 349 mg/kg/day on a 7-day exposure basis.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- An uncertainty factory of 1000 (10 for LOAEL, 10 for species conversion, and 10 for sensitive human population), results in an RfD of 0.3 mg/kg/day.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

None.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

The chosen study is given a medium confidence rating because large numbers of animals/sex were tested in two doses for chronic exposures, but the study did not establish a NOEL. The data base is given a medium confidence rating because of the support of chronic data, but the lack of reproductive data. Medium confidence in the RfD follows.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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

Other EPA Documentation -- None

Agency Work Group Review -- 05/20/1985, 05/31/1985

Verification Date -- 05/31/1985

00088592

I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Trichlorofluoromethane
CASRN -- 75-69-4

Not available at this time.

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Trichlorofluoromethane
CASRN -- 75-69-4

This substance/agent has not undergone a complete evaluation and determination under US EPA's IRIS program for evidence of human carcinogenic potential.

VI. BIBLIOGRAPHY

Substance Name -- Trichlorofluoromethane
CASRN -- 75-69-4
Last Revised -- 07/01/1992

VI.A. ORAL RfD REFERENCES

Leuschener, F., B.W. Neumann and F. Hubscher. 1983. Report on the subacute toxicological studies with several fluorocarbons in rats and dogs by inhalation. Arzneim-Forsch. 33(10): 1475-1476.

NCI (National Cancer Institute). 1978. Bioassay of trichlorofluoromethane for possible carcinogenicity. Report No. 106, PHS/NIH, DWEH, Publ. No. 78-1356.

VI.B. INHALATION RfC REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

None

VII. REVISION HISTORY

Substance Name -- Trichlorofluoromethane
CASRN -- 75-69-4

Date	Section	Description
03/31/1987	I.A.6.	Documentation corrected
03/01/1988	I.A.5.	Confidence levels revised
08/01/1990	I.A.	Text edited
08/01/1990	IV.F.1.	EPA contact changed
01/01/1992	I.A.7.	Primary contact changed
01/01/1992	IV.	Regulatory actions updated
07/01/1992	I.A.2.	Citations removed
07/01/1992	VI.A.	Oral RfD references on-line
08/01/1992	I.A.3.	Minor text change

VIII. SYNONYMS

Substance Name -- Trichlorofluoromethane
CASRN -- 75-69-4
Last Revised -- 01/31/1987

00088594

75-69-4
ALGOFRENE TYPE 1
ARCTON 9
ELECTRO-CF 11
ESKIMON 11
F 11
FC 11
FLUOROCARBON NO. 11
FLUOROTRICHLOROMETHANE
FLUOROTROJCHLOROMETAN
FREON 11
FREON 11A
FREON 11B
FREON HE
FREON MF
FRIGEN 11
GENETRON 11
HALOCARBON 11
ISCEON 131
ISOTRON 11
LEDON 11
MONOFLUOROTRICHLOROMETHANE
NCI-C04637
RCRA WASTE NUMBER U121
Trichlorofluoromethane
TRICHLOROMONOFLUOROMETHANE
UCON FLUROCARBON 11
UCON REFRIGERANT 11

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URL: http://www.epa.gov/iris/subst/0120.htm

00088595

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

1,3,5-Trinitrobenzene;

CASRN 99-35-4
10/01/1997

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

Note: ***A Support Document*** is available for this chemical in Adobe* PDF format (35 Pages, 94 Kbytes)
Similar documents can be found in the List of Available IRIS Toxicological Reviews

0316
1,3,5-Trinitrobenzene; CASRN 99-35-4 (10/01/1997)

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR 1,3,5-Trinitrobenzene

File On-Line 09/07/1988

Category (section)	Status	Last Revised

Oral RfD Assessment (I.A.)	on-line	10/01/1997
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	no data	10/01/1991

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- 1,3,5-Trinitrobenzene
CASRN -- 99-35-4
Last Revised -- 10/01/1997

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Dose -- 2.68 mg/kg-day (1N)
UF -- 100
MF -- 1
RfD -- 3E-2 mg/kg-day

Critical Study 1

Critical Effect -- Methemoglobinemia and spleen-erythroid cell hyperplasia

Study Type -- Rat 2-Year Dietary Study

Reference -- Reddy et al., 1996, 1997

NOAEL -- 2.68 mg/kg-day
NOAEL(ADJ) --

LOAEL -- 13.31 mg/kg-day
LOAEL(ADJ) --

Conversion Factors and Assumptions -- Based on food consumption data, the authors calculated the intake of TNB from dietary concentrations of 0, 5, 60 and 300 ppm: 0, 0.23, 2.68 and 13.31 mg/kg/day (females) and 0, 0.22, 2.64 and 13.44 mg/kg/day (males).

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

00088597

Reddy, T.V., F.B. Daniel, G.R. Olson, B. Wiechman and G. Reddy. 1996. Chronic toxicity studies of 1,3,5-trinitrobenzene in Fischer 344 rats. U.S. Army, Fort Detrick, MD. (Final Report)

Reddy, G., T.V. Reddy, H. Choudhury, F.B. Daniel and G. Leach. 1997. Assessments of environmental hazards of 1,3,5-trinitrobenzene (TNB). J. Toxicol.Environ. Health 52:101-114.

Chronic toxic effects of 1,3,5-TNB in male and female Fisher 344 rats were evaluated by feeding powdered certified laboratory chow diet supplemented with varied concentrations of TNB for 2 years. Based on food consumption, the average TNB intake was calculated for both males and females.

The study was conducted in accordance with the U.S. EPA guidelines for chronic toxicity studies as required by the GLP standards. One of the unique features of this study is that 10 animals/sex were sacrificed at the end of 90 days, 6 months and 1 year, and 25 or more rats were sacrificed at 2 years; complete toxicological evaluations were performed during these periods.

High-dose animals showed decreased body weight gains associated with decreased food consumption. Relative organ weight changes for the brain (increase), spleen (increase), liver (increase) and testes (decrease in 90- and 180-day periods) were reported for all treated animals dosed with TNB at levels higher than 3 mg/kg/day; adverse hematological findings (decreased hematocrit and hemoglobin) and increased methemoglobulin) were consistently reported in all animals treated at these levels. Histopathological findings in the 1-year study revealed extramedullary hematopoiesis in rats treated with TNB at doses of 3 mg/kg-day or higher. In the 2-year study, these effects were seen only in rats dosed with TNB at the high dosage level (13.23 mg/kg/day). The adverse effects, such as increased methemoglobin, erythroid cell hyperplasia, and increased relative organ weights, observed during interim sacrifices in rats receiving 60 ppm TNB did not persist and were not detected in rats fed 60 ppm TNB for 2 years, suggesting that an adaptive mechanism has taken place in order to compensate adverse effects observed during interim sacrifices.

Results of this study exhibited clear evidence of toxicity of the hematopoietic system as has been reported for other nitroaroniatics such as, dinitrobenzene and trinitrotoluene. The NOAEL for this study is 2.68 mg/kg/day and the LOAEL for hematological effects is 13.31 mg/kg/day.

I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- 100. Ten for inter- and 10 for intra-species extrapolation. The previous RfD developed in 1988 was based on analogy to dinitrobenzene with a UF of 10,000. Subsequently, several subchronic, chronic, reproductive and developmental toxicity studies were conducted in rats and mice through the collaborative efforts of the U.S. EPA and the U.S. Army; thus reducing uncertainties in the studies and data base for TNB. The data in these studies provided adequate evidence that rats were more sensitive than mice in terms of both systemic and developmental toxicity. The RfD, based on 2-year oral toxicity study, is supported by additional subchronic studies in mice and rats, reproductive, developmental and single-generation studies where adverse effects were observed at doses higher than that reported in the 2-year study; thus precluding uncertainties in database.

MF -- None.

I.A.4. ADDITIONAL STUDIES / COMMENTS (ORAL RfD)

Male and female Fisher 344 rats were evaluated by feeding certified laboratory chow diets containing different doses of TNB for 90 days. The estimated doses of TNB were 0, 4, 23 and 44 mg/kg/day for males and 0, 4, 25 and

49 mg/kg/day for females (Reddy et al., 1994a,b).

Toxicological evaluation of data indicated the following adverse effects: (a) increased relative spleen and brain weights and decreased testis weight in rats dosed with 23-49 mg/kg/day TNB. Liver weights were increased in the high-dose animals; (b) decreased RBC, hematocrit, and serum alkaline phosphatase and increased methemoglobulin levels in the mid- and high-dose groups: (c) Histopathological examinations showed extramedullary hematopoiesis and testicular degeneration in the mid- and high-dose groups. The data indicated a NOAEL of 4 mg/kg/day and a LOAEL of 23 mg/kg/day for hematopoietic effects (methemoglobulinemia) and testicular degeneration. Hyaline droplets were observed in all dosed male animals but the clinical significance of this lesion cannot be ascertained in terms of human renal effects.

Kinkead et al. (1994, 1995) in their 90-day reproductive toxicity study dosed male and female Sprague-Dawley rats for 14 days prior to mating, and continued through a total of a 90-day dosing period. One group of male rats was treated with the high dose for 90 days followed by a 60-day recovery period to ascertain the reversibility of the testicular toxicity. The dosing regimen for females included premating, gestation and lactation and 4 weeks post-weaning; thus achieving a 90-day total exposure period. The consumed doses of TNB were 2, 9 and 19 mg/kg/day for males and 3, 14 and 29 mg/kg/day for females.

Toxicological evaluation of data indicated adverse effects on the spleen and testis. The high dose males showed testicular degeneration during the 90-day exposure, which continued through the recovery period. Increased methemoglobin levels and hemosiderosis were observed in all rats exposed to 10 mg/kg/day or higher doses of TNB. This study indicated 3 mg/kg/day as the NOAEL and 10 mg/kg/day as the LOAEL for hematological (spleen) effects.

In the single generation study (Kinkead et al., 1994), male and female Sprague-Dawley rats were dosed with TNB during the 14-day premating period and continuing through the end of lactation. The target administered doses were 0, 2, 23 and 51 mg/kg/day for males and 0, 3, 30 and 60 mg/kg/day for females.

In this study, both mid- and high-dose males showed testicular degeneration; high-dose females showed CNS toxicity; and neonatal mortality were observed during lactation period in pups born to high-dose animals.

This study indicated a NOAEL of 3 mg/kg/day for females and a LOAEL of 23 mg/kg/day for testicular degeneration.

In the rat developmental toxicity study (Cooper and Caldwell, 1995) pregnant female Sprague-Dawley rats were gavaged daily with 0, 11.25, 22.50, 45.0 and 90.0 mg/kg-day TNB during 20 days of their gestation period. Complete morphological and microscopical examinations of fetuses on gestation day 20 was performed; material toxicological evaluations were also conducted. This study indicated maternal toxicity and skeletal abnormalities and decreased fetal body weight in the high dosage group, resulting in a NOAEL of 45 mg/kg/day and LOAEL of 90 mg/kg/day.

A subchronic toxicity study was conducted in the White-footed mouse dosed with 0, 150, 375 and 750 mg/kg diet. The calculated doses were 0, 20, 65 and 108 mg/kg/day (females) and 0, 23.5, 67.4 and 113.50 mg/kg/day (males) of TNB during a 90-day period.

Results of this study indicated erythroid cell hyperplasia (spleen), increased reticulocytes, increased relative spleen weight, and testicular degeneration in mice exposed to high dose TNB; no other toxicological effects were observed in this study; thus the White-footed mouse was considered to be less sensitive to TNB exposure (Reddy et al., 1995).

I.A.5. CONFIDENCE IN THE ORAL RfD

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

The RfD is based on a well-conducted 2-year study that includes interim sacrifices at 3, 6 and 12 months and is supported by subchronic reproductive and developmental toxicity data in rats and subchronic data in mice. High confidence is recommended for the study. The data base contains adequate subchronic studies in rats and mice, reproductive, developmental and chronic studies in rat and lacks additional developmental studies in other species. Medium confidence is therefore recommended for the data base and the RfD.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1997

This assessment was peer reviewed by external scientists. These comments have been evaluated and incorporated in the finalization of this IRIS summary. A record of these comments is included as an appendix to U.S. EPA, 1997.

Other EPA Documentation -- U.S. EPA, 1989

Agency Work Group Review -- 03/24/1988, 04/20/1988, 05/26/1988

Verification Date -- 08/27/1997

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX), or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

Substance Name -- 1,3,5-Trinitrobenzene
CASRN -- 99-35-4

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- 1,3,5-Trinitrobenzene
CASRN -- 99-35-4

Not available at this time.

00088600

VI. BIBLIOGRAPHY

Substance Name -- 1,3,5-Trinitrobenzene
CASRN -- 99-35-4
Last Revised -- 10/01/1997

VI.A. ORAL RfD REFERENCES

Cooper, K.R. and D.J. Caldwell. 1995. Developmental toxicity evaluation of 1,3,5-trinitrobenzene in Sprague-Dawley rats. Final Report, U.S. Army, Wright-Patterson AFB, OH.

Kinkead, E.R., R.E. Wolfe, C.D. Flemming, D.J. Caldwell, C.R. Miller and G.B. Marit. 1994. Reproductive toxicity screen of 1,3,5-trinitrobenzene administered in the diet of Sprague-Dawley rats. Al/OE-TR-1994-0144, WRAIR-TR-1994-0016. U.S. Army, Wright-Patterson AFB, OH.

Kinkead, E.R., R.E. Wolfe, C.D. Flemming, D.J. Caldwell, C.R. Miller and G.B. Marit. 1995. Reproductive toxicity screen of 1,3,5-trinitrobenzene administered in the diet of Sprague-Dawley rats. Toxicol. Ind. Health. 11: 309-323.

Reddy, T.V., F.B. Daniel, M. Robinson, G.R. Olson, B. Wiechman and G. Reddy. 1994a. Subchronic toxicity studies on 1,3,5-trinitrobenzene, 1,3-dinitrobenzene and tetryl in rats: Subchronic toxicity evaluation of 1,3,5-trinitrobenzene in Fischer 344 rats. ADA 283663. U.S. Army Project Order MIPR No. 92MM2525. U.S. Environmental Protection Agency, Cincinnati, OH.

Reddy, T.V., J.A. Torsella, F.B. Daniel, G.R. Olson, B. Wiechman and G. Reddy. 1994b. Subchronic toxicity evaluation of 1,3,5-trinitrobenzene (TNB) in Fischer 344 rats. Toxicologist. 14(1): 117. (Abstract)

Reddy, T.V., J. Torsell, F.B. Daniel, G.R. Olson, B. Wiechman and G. Reddy. 1995. Ninety-day Toxicity Evaluation of 1,3,5-Trinitrobenzene (TNB) in Peromyscus leucopus. Second Society of Environmental Toxicology and Chemistry World Congress. November 5-9, 1995, Vancouver, British Columbia, Canada, (Abstract), p. 189.

Reddy, T.V., F.B. Daniel, G.R. Olson, B. Wiechman and G. Reddy. 1996. Chronic toxicity studies of 1,3,5-trinitrobenzene in Fischer 344 rats. Final Report, U.S. Army, Fort Detrick, MD.

Reddy G., T.V. Reddy, H. Choudhury, F.B. Daniel and G. Leach. 1997. Assessment of environmental hazards of 1,3,5-trinitrobenzene (TNB). J. Toxicol. Environ. Health 52:101-114.

U.S. EPA. 1989. Health and Environmental Effects Document for Trinitrobenzenes (o-, m-, p-). 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, 1997. Support Document for Trinitrobenzene. National Center for Environmental Assessment, Cincinnati, OH. Contact the Risk Information Hotline at (513)569-7254 (phone), (513)569-7159 (FAX), or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

VI.B. INHALATION RfD REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

None

VII. REVISION HISTORY

Substance Name -- 1,3,5-Trinitrobenzene
CASRN -- 99-35-4

Date	Section	Description
09/07/1988	I.A.	Oral RfD summary on-line
08/01/1991	VI.	Bibliography on-line
10/01/1991	II.	Carcinogenicity assessment now under review
01/01/1992	IV.	Regulatory Action section on-line
02/01/1995	I.A.7.	Secondary contact's name changed
10/01/1997	I.A.	Oral RfD summary replaced; new assessment
10/01/1997	VI.A.	Oral RfD bibliography updated

VIII. SYNONYMS

Substance Name -- 1,3,5-Trinitrobenzene
CASRN -- 99-35-4
Last Revised -- 09/07/1988

99-35-4
BENZENE, 1,3,5-TRINITRO-
RCRA WASTE NUMBER U234
TNB
TRINITROBENZEEN
TRINITROBENZENE
1,3,5-Trinitrobenzene
Trinitrobenzene, 1,3,5-
TRINITROBENZOL

Note: ***A Support Document*** is available for this chemical in Adobe* PDF format (35 Pages, 94 Kbytes)
Similar documents can be found in the List of Available IRIS Toxicological Reviews

Get Acrobat Reader * Some of the documents provided by EPA are Adobe Acrobat PDF (Portable Document Format) files. They can be viewed, and printed, with the use of an Adobe Acrobat Reader. The Adobe Acrobat's Reader is available, free, for Windows (NT, '98, '95, and 3.1), OS/2, Unix, and Mac OS operating systems. Click this button to download the latest version of Adobe Acrobat. The readers are available directly from Adobe.

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Last updated: **16 July 1998**
URL: http://www.epa.gov/iris/subst/0316.htm

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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

2,4,6-Trinitrotoluene (TNT)

CASRN 118-96-7

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0269
2,4,6-Trinitrotoluene (TNT); CASRN 118-96-7

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR TNT

File On-Line 09/26/1988

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	02/01/1993
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	07/01/1993

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- 2,4,6-Trinitrotoluene (TNT)
CASRN -- 118-96-7
Last Revised -- 02/01/1993

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Liver effects	NOEL: none	1000	1	5E-4 mg/kg/day
26-Week Dog Feeding Study	LOAEL: 0.5 mg/kg/day			
U.S. DOD, 1983				

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

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

U.S. Department of Defense (DOD). 1983. AD-A157 002. Available from Defense Technical Center. Write to Documents, Cameron Station, Alexandria, VA 22314, or call (703)274-7633.

The U.S. Department of Defense (U.S. DOD, 1983) commissioned a study to determine the effects of TNT (approximately 99% pure) administered daily by gelatin capsule, containing a mix of TNT with Purina Certified Rodent Chow, to groups of six beagle dogs/sex at 0, 0.5, 2, 8, or 32 mg/kg/day for 25 weeks. Animals were approximately 6.5 months old at the start of the TNT dosing schedule. Animals were observed several times daily, before and after dosing, for toxic signs and were examined weekly by palpation for detectable masses.

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Body weight and food intakes were recorded weekly. Other toxicologic endpoints included a comprehensive clinical chemistry and hematological evaluation, urinalyses, and periodic electrocardiography (ECG) and ophthalmic examinations. During week 27 all animals were fasted for 16 to 18 hours and were sacrificed by injection of intravenous pentobarbital sodium. Major organs were weighed and all organs were collected and fixed for microscopic examination. Statistical analyses were performed.

Several indications of liver injury were observed upon gross and histologic examination. Male (8 and 32 mg/kg/day) and female (32 mg/kg/day) dogs had significant increases in relative and/or absolute liver weight accompanied by moderate to marked hepatocytic cloudy swelling and hepatocytomegaly. The hepatic swelling and hepatocytomegaly was observed at all dose levels, but to a greater degree in the high-dose group; lesions at the low dose (0.5 mg/kg/day) were described as trace to mild. No such lesions were seen in the control animals. Microscopic evidence of cirrhosis was seen, primarily in males, at the 8 and 32 mg/kg/day dose levels. Hemosiderosis of the liver was seen in the majority of dogs at 2 and 8 mg/kg/day (the two highest levels) as well as in one female at the 2 mg/kg/day level. None of these microscopic lesions were seen in the two females necropsied prior to termination of this study. The 0.5 mg/kg/day test level is the LOAEL for liver effects. The histopathology at this level is trace to mild and is unsupported by effects on the liver enzymes and organ weight.

In a rat study (U.S. DOD, 1984a), groups of 75 animals/sex (approximately 6 to 7 weeks old) received TNT (about 99% pure), mixed in a diet of Purina rodent chow meal, at dose levels of 0.0, 0.4, 2, 10, or 50 mg/kg/day for 24 months. A NOAEL of 0.4 mg/kg/day is based on the absence of systemic effects of TNT on the spleen, kidney, bone marrow, and bladder.

The effects of feeding experiments with TNT was studied by the U.S. DOD (1978) in Swiss-Webster mice, Sprague-Dawley rats, and beagle dogs. Dogs appeared to be the most sensitive species tested during the subchronic studies, with 0.2 mg/kg/day having no observable effects and 2 mg/kg/day showing some effects. In rats, a concentration in feed giving 7.4 mg/kg/day to females and 7 mg/kg/day to males caused toxic effects. No observable toxic effects were found at 1.4 mg/kg/day for female or male rats. In mice, a dose in feed giving approximately 37.8 mg/kg/day and 35.7 mg/kg/day to females and males, respectively, caused toxic effects. No observable effects were found at 8 mg/kg/day and 7.5 mg/kg/day for female and male mice, respectively.

I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- The UF of 1000 allows for uncertainties in laboratory animal-to-man dose extrapolation, interindividual sensitivity, subchronic-to-chronic extrapolation, and LOAEL-to-NOAEL extrapolation.

MF -- None

I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

The U.S. DOD (1981) conducted a 13-week study in which Fischer 344 rats (10/sex/dose level) were administered TNT (about 99% pure) in the diet at 1, 5, 25, 125, or 300 mg/kg/day. Thirty animals/sex were used as controls and received the same rodent chow used to prepare the test diets. A NOAEL of 5 mg/kg/day is indicated by the absence of testicular degeneration and effects

on the spleen at this dose level.

The U.S. DOD (1974a) conducted a 90-day study with cynomolgus monkeys to evaluate the toxicity of TNT administered by gastric intubation as a suspension in a 1% aqueous solution of methyl cellulose. Daily dosages were 0.02, 0.1, or 1 mg/kg. A NOAEL or LOAEL could not be determined for this study because of the small numbers of animals evaluated and the lack of statistical evaluation.

The U.S. DOD (1974b) also conducted a 90-day toxicity study in purebred beagle dogs administered TNT in the diet (consisting of ground dog chow supplemented with commercial canned dog food) at dosage levels of 0.02, 0.1, or 1 mg/kg/day. Three dogs/sex/dosage level were used. A slight increase in hemosiderosis of the bone marrow in the high-dose group could not be properly assessed because of the small group size. The small number of animals evaluated precludes the determination of a NOAEL or LOAEL.

In a 24-month study conducted by the U.S. DOD (1984b) in B6C3F1 hybrid mice, TNT (>99% pure) was administered in a diet of ground Purina chow to groups of 75 mice/sex/group at dosage levels of 0.0, 1.5, 10, or 70 mg/kg/day. No noncarcinogenic effects were seen at the LDT. Noncancer effects included anemia and hepatomegaly without microscopic alterations at the high-dose level.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

The principal study is a well-designed subchronic dog study, but the method of administration (capsule) is not ideal and a NOAEL was not established. Other studies (dog subchronic, rat chronic) are somewhat supportive of the magnitude of the RfD, but effects on the hematopoetic system are observed in other species at generally higher doses. Data on reproductive toxicology are lacking. The RfD is therefore given a medium confidence rating for these reasons.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1988

Other EPA Documentation -- None

Agency Work Group Review -- 06/11/1986, 01/20/1988, 04/20/1988

Verification Date -- 04/20/1988

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this

assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- 2,4,6-Trinitrotoluene (TNT)
CASRN -- 118-96-7

Not available at this time.

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- 2,4,6-Trinitrotoluene (TNT)
CASRN -- 118-96-7
Last Revised -- 07/01/1993

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

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- C; possible human carcinogen

Basis -- Evidence of human carcinogenicity is inadequate. Urinary bladder papilloma and carcinoma were observed in female Fischer 344 rats. Mutagenic activity was observed in Salmonella with and without metabolic activation.

___II.A.2. HUMAN CARCINOGENICITY DATA

None. There are extensive human toxicity data, but they are not useful in the evaluation of carcinogenicity.

___II.A.3. ANIMAL CARCINOGENICITY DATA

Limited. The carcinogenic potential of TNT was evaluated in 24-month studies in Fischer 344 rats (U.S. DOD, 1984a) and in hybrid B6C3F1 mice (U.S. DOD, 1984b).

In the rat, TNT was administered at 0, 0.4, 2, 10, and 50 mg/kg/day by diet to groups of 75 rats/sex. Ten rats/sex/dose were sacrificed following 6 and 12 months on test, and surviving animals were sacrificed after 24 months of treatment. Based on the observation of splenic congestion, increased amounts of pigment deposition in the kidneys, bone marrow fibrosis and decrease in body weight gain at doses of 2.0 mg/kg/day or greater, the MTD was achieved. Toxic effects on the urogenital system, primarily seen for high dose (50 mg/kg/day) animals, include hyperplasia of the renal pelvis with lymphocytic infiltration of renal tissue, and for females, urinary bladder hyperplasia, papilloma and carcinoma. The tumor incidence for combined transitional cell papilloma and carcinoma of the urinary bladder in females was 0/54, 0/54, 0/55, 1/55, and 17/55 for the control, 0.4, 2.0, 10.0, and 50.0 mg/kg/day dose groups, respectively. In addition to the above mentioned neoplasms, hepatocellular (male rats), renal and urinary bladder hyperplasia (female rats) seen at doses of 10 mg/kg/day or greater support the conclusion that TNT is a carcinogen in F344 rats under the conditions of the study. Neoplastic lesions in the urinary bladder were considered rare by the study authors.

In the mouse study, TNT was administered in the diet for up to 24 months. Groups of 75 mice/sex received TNT at doses of 0, 1.5, 10, or 70 mg/kg/day. Ten mice/sex/dose were killed following 6 and 12 months on test; surviving animals killed after 24 months of treatment. The major systemic effects observed in the high-dose group included anemia with hepatotoxicity, indicating the MTD was achieved. The study authors reported that the incidence of all types of malignant lymphoma combined with lymphocytic and granulocytic leukemia in the spleen of females increased with dose and was statistically significant at 70 mg/kg/day. However, when all types of malignant lymphomas and lymphocytic leukemia were counted in all animal tissues combined rather than for a single organ (McConnell et al., 1986), the incidence of tumors by sex or for both sexes combined was not statistically significantly elevated nor was there a significant trend. These neoplasms were, therefore, not considered to be treatment-related.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Mutagenic activity for TNT was reported by the U.S. DOD (1978a). As little as 10 ug/plate dissolved in DMSO, with or without metabolic activation, was mutagenic in Salmonella typhimurium strains TA98, TA1538 and TA1537. At 30 ug/plate TNT was mutagenic in TA100 as well as the other three strains. In vivo cytogenetic analyses on bone marrow from Sprague-Dawley rats treated for 28 days with TNT at 190.4 or 1.8 mg/kg/day in feed were negative for genetic damage (U.S. DOD, 1978b). Ashby et al. (1985) reported TNT gave negative response in the mouse bone marrow micronucleus assay and in an in vivo/in vitro rat liver assay for unscheduled DNA synthesis (UDS).

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 3.0E-2/mg/kg/day

Drinking Water Unit Risk -- 9.0E-7/ug/L

Extrapolation Method -- Linearized multistage procedure, extra risk

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)	1 ug/L

II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- urinary bladder, transitional cell papilloma and transitional squamous cell carcinomas
Test Animals -- rat/Fischer 344, female
Route -- diet
Reference -- U.S. DOD, 1984a

Administered Dose (ppm)	Human Equivalent Dose (mg/kg)/day	Tumor Incidence
0.0	0	0/54
0.4	0.065	0/54
2.0	0.325	0/55
10.0	1.623	1/55
50.0	8.117	17/55

II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

Five of the 17 urinary bladder tumors were benign neoplastic changes (i.e., papillomas). The human equivalent dose was determined using a standard surface area correction factor. The animal study dose is divided by the ratio of the human weight (70 kg) to the rat weight (0.30 kg) raised to the 1/3 power.

The unit risk should not be used if the water concentration exceeds 1E+4 ug/L. Above this concentration the slope factor may differ from that stated.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

Both rat and mouse studies (U.S. DOD, 1984a,b), were well conducted with appropriate number of animals per sex per dose.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1988

The Health Advisory for trinitrotoluene received Agency review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 07/27/1988, 08/15/1988, 09/22/1988

Verification Date -- 09/22/1988

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- 2,4,6-Trinitrotoluene (TNT)
CASRN -- 118-96-7
Last Revised -- 12/01/1990

__VI.A. ORAL RfD REFERENCES

U.S. Department of Defense. 1974a. AD-A044 650/0. Available from Defense Technical Center. Write to Documents, Cameron Station, Alexandria, VA 22314, or call (703)274-7633.

U.S. Department of Defense. 1974b. AD-A035 717. Available from Defense Technical Center. Write to Documents, Cameron Station, Alexandria, VA 22314, or call (703)274-7633.

U.S. Department of Defense. 1978. AD-A080 957. Available from Defense Technical Center. Write to Documents, Cameron Station, Alexandria, VA 22314, or call (703)274-7633.

U.S. Department of Defense. 1981. AD-A108 447. Available from Defense Technical Center. Write to Documents, Cameron Station, Alexandria, VA 22314, or call (703)274-7633.

U.S. Department of Defense. 1983. AD-A157 002. Available from Defense Technical Center. Write to Documents, Cameron Station, Alexandria, VA 22314, or call (703)274-7633.

U.S. Department of Defense. 1984a. AD-A168 637. Available from Defense Technical Center. Write to Documents, Cameron Station, Alexandria, VA 22314, or call (703)274-7633.

U.S. Department of Defense. 1984b. AD-A168 754. Available from Defense Technical Center. Write to Documents, Cameron Station, Alexandria, VA 22314, or call (703)274-7633.

U.S. EPA. 1988. Drinking Water Health Advisory for 2,4,6-Trinitrotoluene. Office of Drinking Water, Washington, DC. (Draft)

__VI.B. INHALATION RfD REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Ashby, J., B. Burlinson, P.A. Levre and J. Topham. 1985. Non-genotoxicity of 2,4,6-trinitrotoluene (TNT) to the mouse bone marrow and the rat liver. Implications for its carcinogenicity. Arch. Toxicol. 58: 14-19.

McConnell, E.E., H.A. Solleveld, J.A. Swenberg and G.A. Boorman. 1986. Guidelines for combining neoplasms for evaluation of rodent carcinogenesis studies. J. Natl. Cancer Inst. 76(2): 283-289.

U.S. DOD (U.S. Department of Defense). 1978a. AD-A069 333. Available from Defense Technical Center. Write to Documents, Cameron Station, Alexanderia, VA 22314, or call (703) 274-7633.

U.S. DOD (U.S. Department of Defense). 1978b. AD-A080 957. Available from Defense Technical Center. Write to Documents, Cameron Station, Alexanderia, VA 22314, or call (703) 274-7633.

U.S. DOD (U.S. Department of Defense). 1984a. AD-A168637. Available from Defense Technical Center. Write to Documents, Cameron Station, Alexanderia, VA 22314, or call (703) 274-7633.

U.S. DOD (U.S. Department of Defense). 1984b. AD-A168754. Available from Defense Technical Center. Write to Documents, Cameron Station, Alexanderia, VA 22314, or call (703) 274-7633.

U.S. EPA. 1988. Health Advisory on Trinitrotoluene. Office of Drinking Water, Washington, DC.

VII. REVISION HISTORY

Substance Name -- 2,4,6-Trinitrotoluene (TNT)
CASRN -- 118-96-7

Date	Section	Description
09/26/1988	I.A.	Oral RfD summary on-line
06/01/1989	I.A.1.	NOAEL - reference to ppm removed
06/01/1989	II.	Carcinogen summary on-line
06/01/1989	VI.	Bibliography on-line
08/01/1990	I.A.	Text edited
08/01/1990	I.A.7.	Primary contact changed
12/01/1990	III.A.	Health Advisory on-line
12/01/1990	VI.D.	Health Advisory references added
09/01/1992	II.D.3.	Primary contact changed
02/01/1993	I.A.7.	Primary contact changed
02/01/1993	II.D.3.	Primary contact changed
02/01/1993	III.A.10	Primary contact changed
07/01/1993	II.D.3.	Secondary contact's phone number changed

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VIII. SYNONYMS

Substance Name -- 2,4,6-Trinitrotoluene (TNT)
CASRN -- 118-96-7
Last Revised -- 09/26/1988

118-96-7
BENZENE, 2-METHYL-1,3,5-TRINITRO-
ENTSUFON
NCI-C56155
TNT
alpha-TNT
TNT-TOLITE
TOLIT
TOLITE
2,4,6-TRINITROTOLUEEN
TRINITROTOLUENE
2,4,6-Trinitrotoluene
Trinitrotoluene, 2,4,6-
s-TRINITROTOLUENE
sym-TRINITROTOLUENE
2,4,6-TRINITROTOLUOL
s-TRINITROTOLUOL
sym-TRINITROTOLUOL
TRITOL
TRITON
TROJNITROTOLUEN
TROTYL
TROTYL OIL
UN 0209

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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Toluene

CASRN 108-88-3

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0118
Toluene; CASRN 108-88-3

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Toluene

File On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	04/01/1994
Inhalation RfC Assessment (I.B.)	on-line	08/01/1992
Carcinogenicity Assessment (II.)	on-line	02/01/1994

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Toluene
CASRN -- 108-88-3
Last Revised -- 04/01/1994

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. 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

*Conversion Factors: Dose adjusted for gavage schedule of 5 days/week.

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

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.

I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

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.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

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.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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

Other EPA Documentation -- None

Agency Work Group Review -- 05/20/1985, 08/05/1985, 08/05/1986, 05/17/1990, 06/20

Verification Date -- 06/20/1990

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Toluene
CASRN -- 108-88-3
Last Revised -- 08/01/1992

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is expressed in units of mg/cu.m. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation

Reference Doses (EPA/600/8-88/066F August 1989) and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994). RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

___I.B.1. INHALATION RfC SUMMARY

Critical Effect	Exposures*	UF	MF	RfC
Neurological effects	NOAEL: None	300	1	4E-1 mg/cu.m
Occupational Study Foo et al., 1990	LOAEL: 332 mg/cu.m (88 ppm) LOAEL(ADJ): 119 mg/cu.m LOAEL(HEC): 119 mg/cu.m			
Degeneration of nasal epithelium 2-Year Rat Chronic Inhalation Study NTP, 1990	NOAEL: None LOAEL: 2261 mg/cu.m (600 ppm) LOAEL(ADJ): 437 mg/cu.m LOAEL(HEC): 79 mg/cu.m			

*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 MVhc/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.

___I.B.2. PRINCIPAL AND SUPPORTING STUDIES (INHALATION RfC)

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

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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.

___I.B.3. UNCERTAINTY AND MODIFYING FACTORS (INHALATION RfC)

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.

MF -- None

___I.B.4. ADDITIONAL STUDIES / COMMENTS (INHALATION RfC)

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%

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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

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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].

___I.B.5. CONFIDENCE IN THE INHALATION RfC

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.

___I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

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

Other EPA Documentation -- U.S. EPA, 1984, 1985

Agency Work Group Review -- 04/21/1988, 05/26/1988, 02/16/1989, 03/21/1989, 05/18 08/15/1991, 12/11/1991

Verification Date -- 05/18/1989, 12/11/1991

I.B.7. EPA CONTACTS (INHALATION RfC)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Toluene
CASRN -- 108-88-3
Last Revised -- 02/01/1994

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

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classified

Basis -- No human data and inadequate animal data. Toluene did not produce positive results in the majority of genotoxic assays.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. 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 carcinogenic 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.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

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).

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

Not available.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1987

The values in the 1987 Drinking Water Criteria Document for Toluene have received peer and administrative review.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 09/15/1987

Verification Date -- 09/15/1987

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

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CASRN -- 108-88-3
Last Revised -- 08/01/1992

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_VII. REVISION HISTORY

00088636

Substance Name -- Toluene
CASRN -- 108-88-3

Date	Section	Description
03/01/1988	I.A.4.	Text revised
09/07/1988	II.	Carcinogen summary on-line
02/01/1989	II.D.3.	Secondary contact's phone number corrected
07/01/1989	I.B.	Inhalation RfD now under review
03/01/1990	VI.	Bibliography on-line
04/01/1990	VI.C.	Combs et al., 1973 citation corrected
06/01/1990	IV.A.1.	Area code for EPA contact corrected
06/01/1990	IV.F.1.	EPA contact changed
07/01/1990	I.A.	Withdrawn; new RfD verified (in preparation)
07/01/1990	VI.A.	Oral RfD references withdrawn
08/01/1990	I.A.	Oral RfD summary replaced; RfD changed
08/01/1990	II.	Text edited
08/01/1990	VI.A.	Oral RfD references revised
09/01/1990	III.A.	Health Advisory on-line
09/01/1990	VI.D.	Health Advisory references added
08/01/1991	VI.C.	Litton Bionetics, Inc., 1981 reference title clarified
01/01/1992	IV.	Regulatory actions updated
04/01/1992	IV.A.1.	CAA regulatory action withdrawn
08/01/1992	I.B.	Inhalation RfC on-line
08/01/1992	VI.B.	Inhalation references on-line
02/01/1994	II.D.3.	Secondary contact's phone number changed
04/01/1994	I.A.7.	Primary contact changed

VIII. SYNONYMS

Substance Name -- Toluene
CASRN -- 108-88-3
Last Revised -- 01/31/1987

108-88-3
ANTISAL 1a
BENZENE, METHYL
METHACIDE
METHYL-BENZENE
METHYLBENZOL
NCI-C07272
PHENYL-METHANE
RCRA WASTE NUMBER U220
TOLUEEN
TOLUEN
Toluene
TOLUOL
TOLUOLO
TOLU-SOL
UN 1294

00088637

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0118.htm

00088638

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Xylenes

CASRN 1330-20-7

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0270
Xylenes; CASRN 1330-20-7

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Xylenes

File On-Line 09/30/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	09/30/1987
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	03/01/1991

00088639

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Xylenes
CASRN -- 1330-20-7
Last Revised -- 09/30/1987

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Hyperactivity, decreased body weight and increased mortality (males) Chronic Rat Gavage Study NTP, 1986	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

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

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

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 hyperactivity, 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.

I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

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

MF -- None

I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

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.

I.A.5. CONFIDENCE IN THE ORAL RfD

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

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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.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1986, 1985, 1984

The Health and Environmental Effects Profile for Xylenes (o-, m-, p-) has received limited peer review and extensive agency-wide review, 1986.

The Drinking Water Criteria Document for Xylenes has received extensive peer and agency-wide review.

The Health Effects Assessment for Xylenes has received ECAO internal review and limited agency review.

Other EPA Documentation -- None

Agency Work Group Review -- 12/05/1985, 03/19/1987

Verification Date -- 03/19/1987

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Xylenes
CASRN -- 1330-20-7

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

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Substance Name -- Xylenes
CASRN -- 1330-20-7
Last Revised -- 03/01/1991

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

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity.

Basis -- Orally administered technical xylene mixtures did not result in significant increases in incidences in tumor responses in rats or mice of both sexes.

___II.A.2. HUMAN CARCINOGENICITY DATA

None.

___II.A.3. 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.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

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).

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

Not available.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

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II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1987

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

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 12/02/1987

Verification Date -- 12/02/1987

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

VI. BIBLIOGRAPHY

Substance Name -- Xylenes
CASRN -- 1330-20-7
Last Revised -- 07/01/1989

VI.A. ORAL RfD REFERENCES

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97-105.

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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.

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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

VI.B. INHALATION RfD REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Berenblum, I. 1941. The cocarcinogenic action of croton resin. Cancer Res. 1: 44-48.

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.

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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.

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.

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.

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.

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.

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.

Pound, A.W. 1970. Induced cell proliferation and the initiation of skin tumor formation in mice by ultraviolet light. Pathology. 2: 269-275.

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_VII. REVISION HISTORY

Substance Name -- Xylenes
CASRN -- 1330-20-7

Date	Section	Description
09/26/1988	II.	Carcinogen summary on-line
07/01/1989	I.B.	Inhalation RfD now under review
07/01/1989	VI.	Bibliography on-line
03/01/1991	II.D.3.	Primary contact changed
03/01/1991	IV.F.1.	EPA contact changed
01/01/1992	I.A.7.	Secondary contact changed
01/01/1992	IV.	Regulatory actions updated

VIII. SYNONYMS

Substance Name -- Xylenes
CASRN -- 1330-20-7
Last Revised -- 09/30/1987

108-38-3
1330-20-7
2106-42-3

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95-47-6
dimethylbenzene
1,2-dimethylbenzene
1,3-dimethylbenzene
1,4-dimethylbenzene
mixed xylenes
m-xylene
meta-xylene
o-xylene
ortho-xylene
p-xylene
para-xylene
Xylenes

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0270.htm

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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Antimony

CASRN 7440-36-0

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0006
Antimony; CASRN 7440-36-0

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Antimony

File On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	02/01/1991
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	no data	

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Antimony
CASRN -- 7440-36-0
Last Revised -- 02/01/1991

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. 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				

*Conversion Factors: 5 mg/L (5 ppm) given as 0.350 mg/kg/day in the discussion section of the critical study

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

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.

I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- 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.

MF -- None

I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

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).

I.A.5. CONFIDENCE IN THE ORAL RfD

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.

I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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.

Agency Work Group Review -- 11/06/1985

Verification Date -- 11/06/1985

I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Antimony
CASRN -- 7440-36-0

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Antimony
CASRN -- 7440-36-0

This substance/agent has not undergone a complete evaluation and determination under US EPA's IRIS program for evidence of human carcinogenic potential.

_VI. BIBLIOGRAPHY

Substance Name -- Antimony
CASRN -- 7440-36-0
Last Revised -- 02/01/1991

__VI.A. ORAL RfD REFERENCES

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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.

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)

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.

Sunagawa, S. 1981. Experimental studies on antimony poisoning. Igaku Kenkyu. 51(3): 129-142. (Jap.) (CA 096/080942D)

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.

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.

__VI.B. INHALATION RfC REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

None

_VII. REVISION HISTORY

Substance Name -- Antimony
CASRN -- 7440-36-0

Date	Section	Description

08/01/1989	I.A.	Gross et al. (1955) citation clarified
08/01/1989	VI.	Bibliography on-line
02/01/1991	I.A.1.	Conversion factor revised
02/01/1991	I.A.2.	Text added
02/01/1991	I.A.4.	Paragraph 2 added
02/01/1991	VI.A.	Dunn, 1928 and Monier-Williams, 1934 added
01/01/1992	I.A.7.	Primary contact changed
01/01/1992	IV.	Regulatory actions updated

VIII. SYNONYMS

Substance Name -- Antimony
CASRN -- 7440-36-0
Last Revised -- 01/31/1987

7440-36-0
Antimony
ANTIMONY BLACK
ANTIMONY POWDER
ANTIMONY, REGULUS
ANTYMON
C.I. 77050
STIBIUM
UN 2871





Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0006.htm

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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Arsenic, inorganic

CASRN 7440-38-2
04/10/1998

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0278

Arsenic, inorganic; CASRN 7440-38-2 (04/10/1998)

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Arsenic, inorganic

File On-Line 02/10/1988

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	02/01/1993
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	04/10/1998

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Arsenic, inorganic
CASRN -- 7440-38-2
Last Revised -- 02/01/1993

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

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.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Hyperpigmentation, keratosis and possible vascular complications	NOAEL: 0.009 mg/L converted to 0.0008 mg/kg-day	3	1	3E-4 mg/kg-day
Human chronic oral exposure Tseng, 1977; Tseng et al., 1968	LOAEL: 0.17 mg/L converted to 0.014 mg/kg-day			

*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.

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

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

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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)

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

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.

MF -- None

___I.A.4. ADDITIONAL STUDIES / COMMENTS (ORAL RfD)

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.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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 11/15/1990. This assessment was discussed by the Risk Assessment Council of EPA on 11/15/1990 and verified through a series of meetings during the 1st, 2nd and 3rd quarters of FY91.

Other EPA Documentation -- U.S. EPA, 1984, 1988

Agency Work Group Review -- 03/24/1988, 05/25/1988, 03/21/1989, 09/19/1989, 08/22/1990, 09/20/1990

Verification Date -- 11/15/1990

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

Substance Name -- Arsenic, inorganic
CASRN -- 7440-38-2

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Arsenic, inorganic
CASRN -- 7440-38-2
Last Revised -- 04/10/1998

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

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- A: human carcinogen

Basis -- based on sufficient evidence from human data. An increased lung cancer mortality was observed in multiple human populations exposed primarily through inhalation. Also, increased mortality from multiple internal organ cancers (liver, kidney, lung, and bladder) and an increased incidence of skin cancer were observed in populations consuming drinking water high in inorganic arsenic.

___II.A.2. HUMAN CARCINOGENICITY DATA

Sufficient. 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 corroborated 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; Tseng, 1977). Although this study demonstrated an association between arsenic exposure and development of skin cancer, it has several weaknesses and uncertainties, including poor nutritional status of the exposed populations, their genetic susceptibility, and their exposure to inorganic arsenic from non-water sources, that limit the study's usefulness in risk estimation. Dietary inorganic arsenic was not considered nor was the potential confounding by contaminants other than arsenic in drinking water. There may have been bias of examiners in the original study since no skin cancer or preneoplastic lesions were seen in 7500 controls; prevalence rates rather than mortality rates are the endpoint; and furthermore there is concern of the applicability of extrapolating data from Taiwanese to the U.S. population because of different background rates of cancer, possibly genetically determined, and differences in diet other than arsenic (e.g., low protein and fat and high carbohydrate) (U.S. EPA, 1988).

A prevalence study of skin lesions was conducted in two towns in Mexico, one with 296 persons exposed to drinking water with 0.4 mg/L arsenic and a similar group with exposure at 0.005 mg/L. The more exposed group had an increased incidence of palmar keratosis, skin hyperpigmentation and hypopigmentation, and four skin cancers (histologically unconfirmed) (Cebrian et al. (1983). The association between skin cancer and arsenic is weak because of the small number of cases, small cohort size, and short duration follow-up; also there was no unexposed group in either town. No excess skin cancer incidence has been observed in U.S. residents consuming relatively high levels of arsenic in drinking water but the numbers of exposed persons were low (Morton et al., 1976; Southwick et al., 1981). Therapeutic use of Fowler's solution (potassium arsenite) has also been associated with development of skin cancer and hyperkeratosis (Sommers and McManus, 1953; Fierz, 1965); several case reports implicate exposure to Fowler's solution in skin cancer development (U.S. EPA, 1988).

Several follow-up studies of the Taiwanese population exposed to inorganic arsenic in drinking water showed an increase in fatal internal organ cancers as well as an increase in skin cancer. Chen et al. (1985) found that the standard mortality ratios (SMR) and cumulative mortality rates for cancers of the bladder, kidney, skin, lung and liver were significantly greater in the Blackfoot disease endemic area of Taiwan when compared with the age adjusted rates for the general population of Taiwan. Blackfoot disease (BFD, an endemic peripheral artery disease) and these cancers were all associated with high levels of arsenic in drinking water. In the endemic area, SMRs were greater in villages that used only artesian well water (high in arsenic) compared with villages that partially or completely used surface well water (low in arsenic). However, dose-response data were not developed (Chen et al. 1985).

A retrospective case-control study showed a significant association between duration of consuming high-arsenic well water and cancers of the liver, lung and bladder (Chen et al., 1986). In this study, cancer deaths in the Blackfoot disease endemic area between January 1980 and December 1982 were chosen for the case group. About 90% of the 86 lung cancers and 95 bladder cancers in the registry were histologically or cytologically confirmed and over 70% of the liver cancers were confirmed by biopsy or à-fetoprotein presence with a positive liver x-ray image. Only confirmed cancer cases were included in the study. A control group of 400 persons living in the same area was frequency-matched with cases by age and sex. Standardized questionnaires of the cases (by proxy) and controls determined the history of artesian well water use, socioeconomic variables, disease history, dietary habits, and lifestyle. For the cancer cases, the age-sex adjusted odds ratios were increased for bladder (3.90), lung (3.39), and liver (2.67) cancer for persons who had used artesian well water for 40 or more years when compared with controls who had never used artesian well water. Similarly, in a 15-year study of a cohort of 789 patients of Blackfoot

disease, an increased mortality from cancers of the liver, lung, bladder and kidney was seen among BFD patients when compared with the general population in the endemic area or when compared with the general population of Taiwan. Multiple logistic regression analysis to adjust for other risk factors including cigarette smoking did not markedly affect the exposure-response relationships or odds ratios (Chen et al., 1988).

A significant dose-response relationship was found between arsenic levels in artesian well water in 42 villages in the southwestern Taiwan and age- adjusted mortality rates from cancers at all sites, cancers of the bladder, kidney, skin, lung, liver and prostate (Wu et al., 1989). An ecological study of cancer mortality rates and arsenic levels in drinking water in 314 townships in Taiwan also corroborated the association between arsenic levels and mortality from the internal cancers (Chen and Wang, 1990).

Chen et al.(1992) conducted a recent analysis of cancer mortality data from the arsenic-exposed population to compare risk of various internal cancers and compare risk between males and females. The study area and population have been described by Wu et al. (1989). It is limited to 42 southwestern coastal villages where residents have used water high in arsenic from deep artesian wells for more than 70 years. Arsenic levels in drinking water ranged from 0.010 to 1.752 ppm. The study population had 898,806 person-years of observation and 202 liver cancer, 304 lung cancer, 202 bladder cancer and 64 kidney cancer deaths. The study population was stratified into four groups according to median arsenic level in well water (< 0.10 ppm, 0.10- 0.29 ppm, 0.30-0.59 ppm and 60+ ppm), and also stratified into four age groups (< 30 years, 30-49 years, 50-69 years and 70+ years). Mortality rates were found to increase significantly with age for all cancers and significant dose- response relationships were observed between arsenic level and mortality from cancer of the liver, lung, bladder and kidney in most age groups of both males and females. The data generated by Chen et al. (1992) provide evidence for an association of the levels of arsenic in drinking water and duration of exposure with the rate of mortality from cancers of the liver, lung, bladder, and kidney. Dose-response relationships are clearly shown by the tabulated data (Tables II-V of Chen et al., 1992). Previous studies summarized in U.S. EPA (1988) showed a similar association in the same Taiwanese population with the prevalence of skin cancers (which are often non-fatal). Bates et al. (1992) and Smith et al. (1992) have recently reviewed and evaluated the evidence for arsenic ingestion and internal cancers.

___II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. There has not been consistent demonstration of carcinogenicity in test animals for various chemical forms of arsenic administered by different routes to several species (IARC, 1980). Furst (1983) has cited or reviewed animal carcinogenicity testing studies of nine inorganic arsenic compounds in over nine strains of mice, five strains of rats, in dogs, rabbits, swine and chickens. Testing was by the oral, dermal, inhalation, and parenteral routes. All oxidation states of arsenic were tested. No study demonstrated that inorganic arsenic was carcinogenic in animals. Dimethylarsonic acid (DMA), the end metabolite predominant in humans and animals, has been tested for carcinogenicity in two strains of mice and was not found positive (Innes et al., 1969); however, this was a screening study and no data were provided. The meaning of non-positive data for carcinogenicity of inorganic arsenic is uncertain, the mechanism of action in causing human cancer is not known, and rodents may not be a good model for arsenic carcinogenicity testing. There are some data to indicate that arsenic may produce animal lung tumors if retention time in the lung can be increased (Pershagen et al., 1982, 1984).

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

A retrospective cohort mortality study was conducted on 478 British patients treated between 1945-1969 with Fowler's solution (potassium arsenite). The mean duration of treatment

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was 8.9 months and the average total oral consumption of arsenic was about 1890 mg (daily dose x duration). In 1980, 139 deaths had occurred. No excess deaths from internal cancers were seen after this 20-year follow-up. Three bladder cancer deaths were observed (1.19 expected, SMR 2.5) (Cuzick et al., 1982). A recent follow-up (Cuzick et al., 1992) indicated no increased mortality from all cancers but a significant excess from bladder cancer (5 cases observed/1.6 expected; SMR of 3.07). A subset of the original cohort (143 persons) had been examined by a dermatologist in 1970 for signs of arsenicism (palmar keratosis). In 1990, there were 80 deaths in the subcohort and 11 deaths from internal cancers. All 11 subjects had skin signs (keratosis-10, hyperpigmentation-5 and skin cancer-3). A case-control study of the prevalence of palmar keratoses in 69 bladder cancer patients, 66 lung cancer patients and 218 hospital controls (Cuzick et al., 1984), indicated an association between skin keratosis (as an indicator of arsenic exposure) and lung and bladder cancer. Above the age of 50, 87% of bladder cancer patients and 71% of lung cancer patients but only 36% of controls had one or more keratoses. Several case reports implicate internal cancers with arsenic ingestion or specifically with use of Fowler's solution but the associations are tentative (U.S. EPA, 1988).

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). Jacobson-Kram and Montalbano (1985) have reviewed the mutagenicity of inorganic arsenic and concluded that inorganic arsenic is inactive or very weak for induction of gene mutations in vitro but it is clastogenic with trivalent arsenic being an order of magnitude more potent than pentavalent arsenic.

Both the pentavalent and trivalent forms of inorganic arsenic are found in drinking water. In both animals and humans, arsenate (As+5) is reduced to arsenite (As+3) and the trivalent form is methylated to give the metabolites mononomethylarsinic acid (MMA) and dimethylarsonic acid (DMA) (Vahter and Marafante, 1988). The genotoxicity of arsenate (As+5) and arsenite (As+3) and the two methylated metabolites, MMA and DMA were compared in the thymidine kinase forward mutation assay in mouse lymphoma cells (Harrington-Brock et al. 1993; Moore et al., 1995, in press). Sodium arsenite (+3) and sodium arsenate (+5) were mutagenic at concentration of 1-2 ug/mL and 10-14 ug/mL, respectively, whereas MMA and DMA were significantly less potent, requiring 2.5-5 mg/mL and 10 mg/mL, respectively, to induce a genotoxic response. Based on small colony size the mutations induced were judged chromosomal rather than point mutations. The authors have previously shown that for chemicals having clastogenic activity (i.e., cause chromosomal mutations), the mutated cells grow more slowly than cells with single gene mutations and this results in small colony size. In the mouse lymphoma assay, chromosomal abberations were seen at approximately the same arsenic levels as TK forward mutations. Arsenate, arsenite and MMA were considered clastogenic but the abberation response with DMA was insufficient to consider it a clastogen. Since arsenic exerts its genotoxicity by causing chromosomal mutations, it has been suggested by the above authors that it may act in a latter stage of carcinogenesis as a progressor, rather than as a classical initiator or promotor (Moore et al., 1994). A finding which supports this process is that arsenate (8-16 uM) and arsenite (3 uM) have been shown to induce 2-10 fold amplification of the dihydrofolate reductase gene in culture in methotrexate resistant 3T6 mouse cells (Lee et al., 1988). Although the mechanism of induction in rodent cells is not known, gene amplification of oncogenes is observed in many human tumors. Inorganic arsenic has not been shown to mutate bacterial strains, it produces preferential killing of repair deficient strains (Rossman, 1981). Sodium arsenite (As+3) induces DNA-strand breaks which are associated with DNA-protein crosslinks in cultured human fibroblasts at 3 mM but not 10 mM (Dong and Luo, 1993) and it appears that arsenite inhibits the DNA repair process by inhibiting both excision and ligation (Jha et al., 1992; Lee-Chen et al., 1993).

The inhibitory effect of arsenite on strand-break rejoining during DNA repair was found to be reduced by adding glutathione to cell cultures (Huang et al., 1993). The cytotoxic effects of sodium arsenite in Chinese hamster ovary cells also has also found to correlate with the intracellular glutathione levels (Lee et al., 1989).

In vivo studies in rodents have shown that oral exposure of rats to arsenate (As+5) for 2-3 weeks resulted in major chromosomal abnormalities in bone marrow (Datta et al., 1986) and exposure of mice to As (+3) in drinking water for 4 weeks (250 mg As/L as arsenic trioxide) caused chromosomal aberrations in bone marrow cells but not spermatogonia (Poma et al., 1987); micronuclei in bone marrow cells were also induced by intraperitoneal dosing of mice with arsenate (DeKnudt et al., 1986; Tinwell et al., 1991). Chromosomal aberrations and sister chromatid exchange have been seen in patients exposed to arsenic from treatment with Fowler's solution (Burgdorf et al., 1977) and subjects exposed occupationally (Beckman et al., 1977) but no increase in either endpoint was seen in lymphocytes of subjects exposed to arsenic in drinking water (Vig et al., 1984).

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 1.5E+0 per (mg/kg)/day

Drinking Water Unit Risk -- 5E-5 per (ug/L)

Extrapolation Method -- Time- and dose-related formulation of the multistage model (U.S. EPA, 1988)

Drinking Water Concentrations at Specified Risk Levels:

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

II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

The Risk Assessment Forum has completed a reassessment of the carcinogenicity risk associated with ingestion of inorganic arsenic (U.S. EPA, 1988). The data provided in Tseng et al., 1968 and Tseng, 1977 on about 40,000 persons exposed to arsenic in drinking water and 7500 relatively unexposed controls were used to develop dose-response data. The number of persons at risk over three dose intervals and four exposure durations, for males and females separately, were estimated from the reported prevalence rates as percentages. It was assumed that the Taiwanese persons had a constant exposure from birth, and that males consumed 3.5 L drinking water/day and females consumed 2.0 L/day. Doses were converted to equivalent doses for U.S. males and females based on differences in body weights and differences in water consumption and it was assumed that skin cancer risk in the U.S. population would be similar to the Taiwanese population. The multistage model with time was used to predict dose-specific and age-specific skin cancer prevalance rates associated with ingestion of inorganic arsenic; both linear and quadratic model fitting of the data were conducted. The maximum likelihood estimate (MLE) of skin cancer risk for a 70 kg person drinking 2 L of water per day ranged from 1E-3 to 2E-3 for an

arsenic intake of 1 ug/kg/day. Expressed as a single value, the cancer unit risk for drinking water is 5E-5 per (ug/L). Details of the assessment are in U.S. EPA (1988).

Dose response data have not been developed for internal cancers for the Taiwanese population. The data of Chen et al. (1992) are considered inadequate at present.

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

Eastern Research Group, under contract to EPA, convened an Expert Panel on Arsenic Carcinogenicity on May 21 and 22, 1997 (Eastern Research Group, 1997). The Expert Panel believed that, "it is clear from epidemiological studies that arsenic is a human carcinogen via the oral and inhalation routes (p. 20)." They also concluded, "that one important mode of action is unlikely to be operative for arsenic". The panel agreed that arsenic and its metabolites do not appear to directly interact with DNA (pp. 30-31)." In addition, the panel agreed that, "for each of the modes of action regarded as plausible, the dose-response would either show a threshold or would be nonlinear (p. 31)". The panel agreed, however, "that the dose-response for arsenic at low doses would likely be truly nonlinear, i.e., with a decreasing slope as the dose decreased. However, at very low doses such a curve might be linear but with a very shallow slope, probably indistinguishable from a threshold (p. 31)."

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

This assessment is based on prevalence of skin cancer rather than mortality because the types of skin cancer studied are not normally fatal. However, competing mortality from Blackfoot disease in the endemic area of Taiwan would cause the risk of skin cancer to be underestimated. Other sources of inorganic arsenic, in particular those in food sources have not been considered because of lack of reliable information. There is also uncertainty on the amount of water consumed/day by Taiwanese males (3.5 L or 4.5 L) and the temporal variability of arsenic concentrations in specific wells was not known. The concentrations of arsenic in the wells was measured in the early 1960s and varied between 0.01 and 1.82 ppm. For many villages 2 to 5 analyses were conducted on well water and for other villages only one analysis was performed; ranges of values were not provided. Since tap water was supplied to many areas after 1966, the arsenic-containing wells were only used in dry periods. Because of the study design, particular wells used by those developing skin cancer could not be identified and arsenic intake could not be assigned except by village. Several uncertainties in exposure measurement reliability existed and subsequent analysis of drinking water found fluorescent substances in water that are possible confounders or caused synergistic effects. Uncertainties have been discussed in detail in U.S. EPA (1988). Uncertainties in exposure measurement can affect the outcome of dose- response estimation.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

___II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk -- 4.3E-3 per (ug/cu.m)

Extrapolation Method -- absolute-risk linear model

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	2E-2 per (ug/cu.m)
E-5 (1 in 100,000)	2E-3 per (ug/cu.m)
E-6 (1 in 1,000,000)	2E-4 per (ug/cu.m)

II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSURE

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 (per (ug/cu.m)

Exposure Source	Study	Unit Risk	Geometric Mean Unit Risk	Final Estimates Unit Risk
Anaconda smelter	Brown and Chu, 1983a,b,c	1.25E-3		
	Lee-Feldstein, 1983	2.80E-3	2.56E-3	
	Higgins, 1982; Higgins et al., 1982; Welch et al., 1982	4.90E-3		4.29E-3
ASARCO smelter	Enterline and Marsh, 1982	7.6E-3	6.81E-3	7.19E-3

II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

A geometric mean was obtained for data sets obtained with 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.

II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

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.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

II.D.1. EPA DOCUMENTATION

U.S. EPA. 1984, 1988, 1993

A draft of the 1984 Health Assessment Document for Inorganic Arsenic was independently reviewed in public session by the Environmental Health Committee of the U.S. EPA Science Advisory Board on September 22-23, 1983. A draft of the 1988 Special Report on Ingested Inorganic Arsenic; Skin Cancer; Nutritional Essentiality was externally peer reviewed at a two-day workshop of scientific experts on December 2-3, 1986. A draft of the Drinking Water Criteria Document for Arsenic was reviewed by the Drinking Water Committee of the U.S. EPA Science Advisory Board on March 10, 1993. The comments from these reviews were evaluated and considered in the revision and finalization of these reports.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 01/13/1988, 12/07/1989, 02/03/1994

Verification Date -- 02/03/1994

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

VI. BIBLIOGRAPHY

Substance Name -- Arsenic, inorganic
CASRN -- 7440-38-2
Last Revised -- 04/10/1998

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VI.B. INHALATION RfD REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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_VII. REVISION HISTORY

Substance Name -- Arsenic, inorganic
CASRN -- 7440-38-2

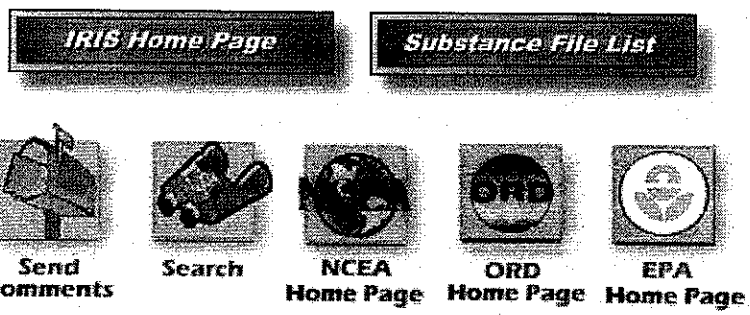
Date	Section	Description
06/30/1988	II.B.	Revised last paragraph
06/30/1988	II.C.1.	Inhalation slope factor changed
06/30/1988	II.C.3.	Paragraph 2 added
09/07/1988	II.B.	Major text changes
12/01/1988	II.A.2.	Mabuchi et al. citation year corrected
12/01/1988	II.A.3.	Pershagen et al. citation year corrected
09/01/1989	II.C.2.	Citations added to anacondor smelter
09/01/1989	VI.	Bibliography on-line
06/01/1990	II.A.2.	2nd & 3rd paragraph - Text revised
06/01/1990	II.A.4.	Text corrected
06/01/1990	II.C.1.	Inhalation slope factor removed (format change)
06/01/1990	IV.F.1.	EPA contact changed
06/01/1990	VI.C.	References added
12/01/1990	II.B.	Changed slope factor to "unit risk", 2nd para, 1st sen
02/01/1991	II.C.3.	Text edited
09/01/1991	I.A.	Oral RfD summary now on-line
09/01/1991	I.A.	Oral RfD bibliography added
10/01/1991	I.A.1.	Conversion factor text clarified

10/01/1991 IV.B.1.	MCLG noted as pending change
01/01/1992 IV.	Regulatory actions updated
08/01/1992 II.	Note added to indicate text in oral quant. estimate
10/01/1992 VI.C.	Missing reference added to bibliography
02/01/1993 I.A.4.	Citations added to second paragraph
02/01/1993 VI.A.	References added to bibliography
03/01/1993 VI.A.	Corrections to references
03/01/1994 II.D.2.	Work group review date added
06/01/1994 II.	Carcinogen assessment noted as pending change
01/01/1995 II.	Pending change note revised
01/01/1995 II.B.	Dates and document no. added to oral quant. estimate
06/01/1995 II.	Carcinogenicity assessment replaced
06/01/1995 VI.C.	Carcinogenicity references replaced
07/01/1995 II.D.1.	Documentation year corrected; review statement revised
07/01/1995 VI.C.	U.S. EPA, 1994 corrected to 1993
04/10/1998 II.B.3	Added discussion on expert panel workshop

_VIII. SYNONYMS

Substance Name -- Arsenic, inorganic
CASRN -- 7440-38-2
Last Revised -- 02/10/1988

7440-38-2
Arsenic
Arsenic, inorganic
gray-arsenic



Last updated: **29 May 1998**
URL: http://www.epa.gov/iris/subst/0278.htm

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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Beryllium and compounds

CASRN 7440-41-7
04/03/1998

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

Note: A *TOXICOLOGICAL REVIEW* is available for this chemical in Adobe* PDF format (94 Pages, 320 Kbytes)
Similar documents can be found in the List of Available IRIS Toxicological Reviews

0012
Beryllium and compounds; CASRN 7440-41-7; 04/03/1998

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices, Regional Offices, and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Beryllium and compounds

File First On-Line 01/31/1997

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Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	On-line	04/03/1998
Inhalation RfC Assessment (I.B.)	On-line	04/03/1998
Carcinogenicity Assessment (II.)	On-line	04/03/1998

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Beryllium and compounds
CASRN -- 7440-41-7
Last Revised -- 04/03/1998

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Small intestinal lesions	BMD_{10}: 0.46 mg/kg-day	300	1	.002E-3 mg/kg-day

Dog Dietary Study
Morgareidge et al., 1976

*Conversion Factors and Assumptions -- mg/kg-day doses determined from administered doses (ppm), reported food intake of 300 g/day and TWA body weights for males and females in each dose group. This benchmark dose (BMD_{10}) is the dose at the 95% confidence limit of the dose-response model corresponding to a 10% increase in incidence of these effects compared with controls. A 10% increase was chosen as the benchmark response and calculated as probability (P) of extra risk over controls at dose (d), [P(d) - P(0)/1- P(0)]. An exponential polynomial model was used as the dose-response model. There was good fit of the model to the data (p = 0.94), and the maximum likelihood estimate (MLE) at 10% increase in incidence is 1.4 mg/kg-day.

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Morgareidge, K; Cox, GE; Gallo, MA. (1976) Chronic feeding studies with beryllium in dogs. Food and Drug Research Laboratories, Inc. Submitted to the Aluminum Company of America, Alcan Research & Development, Ltd., Kawecki-Berylco Industries, Inc., and Brush-Wellman, Inc.

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Morgareidge et al. (1976) conducted a long-term feeding study in which groups of 5 male and 5 female beagle dogs (aged 8 to 12 mo) were fed diets (for 1 h per day) containing 0, 5, 50, or 500 ppm beryllium as beryllium sulfate tetrahydrate for 172 weeks. Because of overt signs of toxicity, the 500 ppm group was terminated at 33 weeks. At this time, a group of 5 male and 5 female dogs was added to the study and fed a diet containing 1 ppm beryllium; duration of exposure for this group was 143 weeks. Using estimated TWA body weights and the reported average food intake of 300 g/day, the 1, 5, 50, and 500 ppm concentrations correspond to doses of 0.023, 0.12, 1.1, and 12.2 mg/kg-day for male dogs and 0.029, 0.15, 1.3, and 17.4 mg/kg-day for females. The following parameters were used to assess toxicity: daily observations, food consumption, body weight, hematology and serum clinical chemistry, urinalysis, organ weights (heart, liver, kidney, brain, spleen, pituitary, thyroids, adrenals, and gonads), and comprehensive histopathology.

Two moribund animals in the 500 ppm group were sacrificed during week 26; the remainder of the animals in the 500 ppm group were killed during week 33. Overt signs of toxicity observed in the 500 ppm group included lassitude, weight loss, anorexia, and visibly bloody feces, indicating that the MTD is <500 ppm. Four other animals died during the course of the study or were killed moribund: two dogs died during parturition, and one male and one female dog in the 50 ppm group died. The appearance, behavior, food intake, and body weight gain of the animals in the other beryllium groups did not differ from controls. No beryllium-related hematological, serum chemistry, or urinalysis alterations were observed in the 1, 5, or 50 ppm groups. In the 500 ppm group, a slight anemia (slight decreases in erythrocyte, hemoglobin, and hematocrit; statistical analysis not reported), more apparent in the females than in the males, was observed after 3 and 6 mo of exposure; however, there were no alterations in the bone marrow and none of the animals was seriously affected. No alterations in organ weights were observed. All animals in the 500 ppm group showed fairly extensive erosive (ulcerative) and inflammatory lesions in the gastrointestinal tract. These occurred predominantly in the small intestine and to a lesser extent in the stomach and large intestine, and were regarded by the authors as treatment related. This conclusion is supported by independent review of the study report; the lesions are not considered to be related to some other cause such as intestinal worms (Goodman, 1997). All of the animals with stomach or large intestinal lesions also had lesions in the small intestine except for one animal with stomach lesions only. This animal had stomach lesions that were very localized and not very severe. Lesions in the small intestine (4/5 males and 5/5 females) considered treatment related include: desquamation of the epithelium, edema, fibrin thrombi, acute inflammation, subacute/chronic inflammation, necrosis and thinning/atrophy of the epithelium, and ulceration (Goodman, 1997). High-dose animals also showed moderate to marked erythroid hypoplasia of the bone marrow, which the authors also considered treatment related (Goodman, 1997). Bile stasis and vasculitis in the liver and acute inflammation in the lymph nodes occurring in these animals is attributed to a likely systemic bacterial invasion through the damaged intestinal mucosa. A generalized low-grade septicemia likely initiated kidney damage.

In the 50 ppm group, one female dog died after 70 weeks of treatment. This animal showed gastrointestinal lesions, but less severe, occurring in the same locations and appearing to be the same types of lesions as those in dogs administered 500 ppm. The authors stated the cause of death of this animal appeared to be related to beryllium administration. Other animals in this treatment group survived until study termination and had no remarkable gross or microscopic findings.

Dose-response modeling of the data for small intestinal lesions in male and female dogs (0/10, 0/10, 0/10, 1/10, 9/10) was conducted to derive a benchmark dose for beryllium. A BMD_{10} (the lower 95% confidence limit on the dose from the maximum likelihood estimate [MLE] of a 10% relative change) of 0.46 mg/kg-day (MLE = 1.4 mg/kg-day) was derived for this lesion and used for further quantitation in this assessment (U.S. EPA, 1995, and Conversion Factors and Assumptions, above).

An oral chronic study via drinking water was carried out by Schroeder and Mitchener

00088679

(1975a) using Long-Evans rats (52 weanling rats of each sex). In addition to control animals, rats of both genders were exposed to 5 ppm beryllium as beryllium sulfate over a lifetime. No adverse effects were observed in the beryllium-exposed rats compared to controls with respect to lifespan, various organs (heart, kidney, liver, and spleen), urinalysis, serum glucose, cholesterol, or uric acid. With respect to cancer, the incidence of gross or malignant tumors in the control and beryllium-exposed rats was not significantly different. This chronic oral study served as the basis for the previous IRIS RfD of 5E-3 mg/kg-day. The new RfD derived from the Morgareidge et al. (1976) study of 2E-3 is not significantly different.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 300.

The following uncertainty factors are applied: 10 for extrapolation for interspecies differences, 10 for consideration of intraspecies variation, and 3 for database deficiencies. A partial uncertainty factor for database deficiencies is applied because while there are several chronic oral animal studies, human toxicity data by the oral route are lacking, and reproductive/developmental and immunotoxicologic endpoints have not been adequately assessed in animals. Database gaps include lack of adequate studies for evaluation of reproductive and developmental toxicity (including multigenerational studies, studies on male reproductive toxicity, teratology, and postnatal development) owing to the possible crossing of the placenta and greater absorption of beryllium in young animals. In addition, oral studies examining immunologic endpoints, the most sensitive endpoint by the inhalation route, are lacking. Since the principal study is of chronic duration and a benchmark dose is used, there are no uncertainty factors for duration or NOAEL/LOAEL extrapolation. No modifying factor is proposed for this assessment.

MF = 1.

___I.A.4. ADDITIONAL STUDIES/COMMENTS (ORAL RfD)

No human information on the oral toxicity of this compound was located.

While each of the chronic animal studies appears to have limitations (e.g., multiple elements in drinking water, no randomization of animals, not published in peer-reviewed journal, no doses showing an adverse effect, and/or lack of histological examination of all animals), collectively they establish the range of doses that is unlikely to evoke noncancer toxicity.

In a chronic toxicity study by Morgareidge et al. (1975, 1977), groups of Wistar albino rats were fed diets containing 5, 50, or 500 ppm beryllium as beryllium sulfate tetrahydrate. The rats were administered the beryllium-containing diet from 4 weeks of age through maturation, mating, gestation, and lactation. Fifty male and 50 female offspring were then placed on the same diets as the parents and fed the beryllium-containing diet for 104 weeks. Using estimated TWA body weights of 0.467, 0.478, and 0.448 kg for males in the 5, 50, and 500 ppm groups and 0.294, 0.302, and 0.280 kg for the females, respectively, and U.S. EPA's (1988) allometric equation of food intake, doses of 0.36, 3.6, and 37 mg/kg-day for males in the 5, 50, and 500 ppm groups and 0.42, 4.2, and 43 mg/kg-day for females in the 5, 50, and 500 ppm groups, respectively, were calculated. Clinical observations, body weight, food consumption, organ weights (liver, kidney, testes, ovaries, thyroid, pituitary, adrenal), gross necropsy, and histopathological examination of most tissues and organs (25 to 26 tissues examined) were used to assess the toxicity and carcinogenicity of beryllium in the offspring; it does not appear that the P_0 rats were examined. Tissues from 20 rats/sex/group in the control and 500 ppm groups were examined microscopically (the study authors did not state whether the animals undergoing histopathological examination were randomly selected), as well as all tissues with gross abnormalities (all groups) and tissues (excluding bone marrow, eyes, and skin) from animals found dead or sacrificed moribund (all

00088680

groups).

No overt signs of toxicity were observed, and mortality appeared to be similar in the controls (30/50 males and 28/50 females died) and beryllium groups at 5 ppm (30/50 and 24/50, respectively), 50 ppm (31/50 and 18/50), and 500 ppm (24/50 and 17/50) at the end of the 104 weeks of the study. During the first 40 to 50 weeks of the study, exposure to beryllium did not appear to affect growth; slight decreases in growth (body weights of males and females in the 500 ppm group were within 10% of control body weights) were observed in the latter part of the study; however, no statistically significant alterations were observed. Alterations in organ weights were limited to statistically significant ($p < 0.05$) increases in relative kidney weight in males exposed to 50 ppm, decreases in relative kidney and adrenal weights in 500 ppm females, and decreases in relative testes weights in 5 and 50 ppm males. Histological examination of the major organs and tissues did not reveal beryllium-related noncarcinogenic alterations. This study has a NOAEL at the highest dose tested (37 mg/kg-day).

Groups of 52 male and 52 female Long-Evans rats were maintained on a low-metal diet and given drinking water containing 0 or 5 ppm beryllium as beryllium sulfate (presumably tetrahydrate) from weanling to natural death (Schroeder and Mitchener, 1975a). The water also contained 5 ppm chromium III, 50 ppm zinc, 5 ppm copper, 10 ppm manganese, 1 ppm cobalt, and 1 ppm molybdenum. Doses of 0.63 and 0.71 mg/kg-day were calculated for male and female rats, respectively. The following parameters were used to assess toxicity: body weights; blood glucose, cholesterol, and uric acid; urine protein, pH, and glucose; heart weight; gross pathology; and histopathology of heart, lung, kidney, liver, spleen, and tumors. Twenty male and eight female rats in the beryllium group died at 20 mo of age from pneumonia; a similar number of animals in the control group also died from pneumonia. At 30 days, the male and female rats exposed to beryllium weighed significantly more than the control animals. At 60, 90, 120, and 180 days, the beryllium-exposed male rats weighed significantly less than the controls; no significant alterations in body weight were observed at the other time intervals (150, 360, or 540 days). No significant alterations in mortality or longevity were observed. The results of the histological examination were not reported. Alterations in serum glucose and cholesterol levels and urine glucose levels in beryllium-treated animals were not considered adverse because the magnitude of the alterations was not sufficiently large to suggest an impairment in organ function. The NOAEL for this study is 0.63 mg/kg-day, the highest dose tested.

In a lifetime exposure study, groups of 54 male and 54 female Swiss mice were administered 0 or 5 ppm beryllium as beryllium sulfate in the drinking water from weaning to natural death (Schroeder and Mitchener, 1975b). The mice were fed low-metal diets and the drinking water was supplemented with 50 ppm zinc, 10 ppm manganese, 5 ppm copper, 5 ppm chromium III, 1 ppm cobalt, and 1 ppm molybdenum. The 5 ppm water concentration is equivalent to doses of 1.2 mg/kg-day for the male and female mice. In the beryllium group, statistically significant alterations in body weight were observed; the alterations included heavier male mice at 30 days and lighter female mice at 90 and 120 days. No significant alterations in mortality or survival were observed in the beryllium-exposed mice. A NOAEL of 1.2 mg/kg-day is established for this study.

Shorter term oral studies in rats fed up to 3% beryllium carbonate in the diet have shown beryllium rickets (Kay and Skill, 1934) and decreases in serum phosphate and serum alkaline phosphatase activity (480 mg/kg-day; Matsumoto et al., 1991). One hypothesis for the development of rickets has been the deprivation of phosphate in the intestines by precipitation as beryllium phosphate and not to any direct effects due to beryllium per se.

There are limited data on the reproductive and developmental toxicity of beryllium compounds following oral exposure. In the chronic dog oral exposure study conducted by Morgareidge et al. (1976), the male and female dogs exposed to 1, 5, or 50 ppm beryllium sulfate in the diet (0.024, 0.11, and 1.1 mg/kg-day for males and 0.025, 0.15, and 1.3 mg/kg-day for the females) were housed together, allowed to mate, and wean their pups (with the exception of the first litter, which was killed 5 days after whelping). Beryllium did not appear to adversely affect

00088681

reproductive or developmental endpoints, but stillborn or cannibalized pups dying within the first few postnatal days were not examined. The authors reported no gross or skeletal abnormalities in the surviving first litter pups, but data were not shown. In addition, there was no visceral evaluation for terata.

Several parenteral studies (as reviewed by U.S. EPA, 1991) have observed developmental effects (increased fetal mortality, decreased fetal body weight, internal abnormalities, and delayed neurodevelopment) in the offspring of rodents following intratracheal or intraperitoneal administration of beryllium chloride, beryllium oxide, or beryllium sulfate during gestation. Clary et al. (1975) conducted a continuous breeding experiment in which male and female Sprague-Dawley rats received a single intratracheal administration of 200 µg beryllium as beryllium oxide (calcined at 960C in the first experiment and calcined at 500C in the second experiment). There were no adverse effects of beryllium in either experiment. Mathur et al. (1987) administered intravenous injections of 0.021 mg/kg beryllium as beryllium nitrate to mated Sprague Dawley rats (n = 5-8/group) (1/10th the LD_{50}) and performed laparotomies; all pups died 2 to 3 days after birth, but these effects may have been due to the repeated surgeries.

___I.A.5. CONFIDENCE IN THE ORAL RfD

Study -- Medium
Database -- Low to Medium
RfD -- Low to Medium

The overall confidence in this RfD assessment is low to medium. The confidence in the principal study is medium. Beryllium was administered by a relevant route (oral) at multiple dose levels for a chronic duration, effects were demonstrated at two dose levels, and relatively comprehensive histopathologic evaluations were conducted. However, there were small groups of animals (5/sex/dose), early mortality at the high dose level, no evidence of randomization or control for potential litter effects, and no measure of immune response or function, the critical endpoint by the inhalation. Confidence in the database is low to medium because there is only one chronic study in dogs showing adverse effect levels; other chronic studies in rodents demonstrated NOAELs at the highest doses tested. Confidence in this assessment is improved over the earlier version on IRIS because of the inclusion of additional chronic studies in rats and dogs.

The major areas of scientific uncertainty in this assessment are the lack of chronic oral studies establishing LOAELs, the lack of a chronic oral study examining immunologic endpoints, the lack of a critical effect in humans by the inhalation route as identified in dogs and the lack of sensitive indicators for rickets, the lack of reproductive and developmental studies (including multigenerational studies or male reproductive toxicity), and the lack of human toxicity information. The uncertainty factors above compensate for these areas of uncertainty. The RfD determined from the BMD_{10} (0.46 mg/kg-day) of small intestinal lesions in dogs (2E-3 mg/kg-day) is almost identical to the previous IRIS RfD (5E-3 mg/kg-day) derived from the NOAEL (0.5 mg/kg-day) in the Schroeder and Mitchener (1975a) rat study. However, to substantiate the significance of the similarities between these two RfDs, additional dose-response rat studies would be needed to establish a LOAEL and NOAEL.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1998

This assessment was peer reviewed by external scientists. Their comments have been evaluated carefully and incorporated in finalization of this IRIS summary. A record of these comments is included as an appendix to the Toxicological Review of Beryllium and Compounds in support of Summary Information on the Integrated Risk Information System (IRIS) (U.S. EPA, 1998).

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Other EPA Documentation -- U.S. EPA, 1987; U.S. EPA, 1991

Agency Consensus Date -- 03/26/1998

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS in general at (513) 569-7254 (phone), (513) 569-7159 (fax) or RIH.IRIS@EPAMAIL.EPA.GOV (Internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION (RfC)

Beryllium and compounds
CASRN -- 7440-41-7
Last Revised -- 04/03/1998

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is generally expressed in units of mg/m³. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (U.S. EPA, 1994). RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

___I.B.1. INHALATION RfC SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfC
Beryllium sensitization and progression to CBD Occupational study Kreiss et al., 1996	LOAEL: 0.55 µg/m³ LOAEL(HEC): 0.20 µg/m³	10	1	2E-2 µg/m³ .02 µg → .00002 mg/m³
Community exposure study Eisenbud et al., 1949	NOAEL: 0.01-0.1 µg/m³ NOAEL(HEC): 0.01-0.1 µg/m³ LOAEL: None			

*Conversion Factors and Assumptions: MW -- 9. The occupational LOAEL is based on 8-h TWA exposure level. Mvo = 10 m³/day, Mvh = 20 m³/day. LOAEL(ADJ) = LOAEL(HEC) = 0.55 µg/m³ × (MVo/MVh × 5 days/7 days) = 0.20 µg/m³.

___I.B.2. PRINCIPAL AND SUPPORTING STUDIES (INHALATION RfC)

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The RfC is based on beryllium sensitization and progression to chronic beryllium disease (CBD) identified in the co-principal studies by Kreiss et al. (1996) and Eisenbud et al. (1949). The Kreiss et al. (1996) occupational exposure study identified a LOAEL for beryllium sensitization in workers exposed to 0.55 μg/m³ (median of average concentrations). The Eisenbud et al. (1949) study, using relatively insensitive screening methods, suggests a NOAEL of 0.01-0.1 μg/m³ in community residents living near a beryllium plant. The LOAEL from the Kreiss et al. study was used for the operational derivation of the RfC because the screening method used in the Eisenbud et al. (1949) study was less sensitive than the method used in the Kreiss et al. (1996) study.

Chronic beryllium disease is a chronic inflammatory lung lesion that can result from inhalation exposure to beryllium. It is characterized by the formation of granulomas (pathologic clusters of immune cells) and involves a beryllium-specific immune response. Beryllium sensitization of the immune system can be demonstrated using the beryllium lymphocyte transformation test (BeLT) (reviewed by Newman, 1996). In this test, lymphocytes obtained from bronchoalveolar lavage (BAL) fluid or from peripheral blood are cultured in vitro and then exposed to soluble beryllium sulfate to stimulate lymphocyte proliferation. The observation of beryllium-specific proliferation indicates beryllium sensitization.

The criteria for diagnosis of CBD have evolved with time, as more advanced diagnostic technology became available. These varying definitions of CBD should be considered in comparing results from different studies. More recent criteria have both higher specificity than earlier methods and higher sensitivity identifying preclinical effects. Recent studies typically use the following criteria: (1) history of beryllium exposure; (2) histopathological evidence of noncaseating granulomas or mononuclear cell infiltrates; and (3) a positive blood or bronchoalveolar lavage (BAL) lymphocyte transformation test (Newman et al., 1989).

A cross-sectional study (Kreiss et al., 1996) was conducted of 136/139 of the then-current beryllium workers in a plant that made beryllia ceramics from beryllium oxide powder. Measurements from 1981 and later were reviewed and included area samples, process breathing-zone samples, and personal lapel samples (the last year only). Quarterly daily-weighted average (DWA) exposures were calculated using a formula based on all of these measurements for each job title. General area and breathing zone samples were not recorded until the last quarter of 1985, soon after machining production was transferred to that plant, even though a limited amount of machining had been conducted since 1982. The median breathing zone measurements for machining was 0.6 μg/m³, and 0.3 μg/m³ for other processes. The frequency of excursions to higher exposure levels decreased with time, with the percentage of machining breathing zone measurements above 5 μg/m³ falling from 7.7% during early sampling years to 2.1% during later sampling years.

Beryllium lymphocyte transformation tests were performed by two different laboratories on blood samples collected from 136 employees. Positive results from one or both laboratories were confirmed by analyzing a subsequent blood sample. Of 136 tested employees, 5 had consistently abnormal blood BeLT results from the two laboratories and were diagnosed with CBD based on observation of granulomas in lung biopsy samples. An additional two employees had abnormal blood results from one of the two laboratories and had no granulomas in lung biopsy samples. Both employees developed abnormal blood results in the other laboratory within 2 years. One of these two employees also developed symptoms of CBD. The other employee declined clinical follow-up. An additional case of CBD was found during the study in an employee hired in 1991, who had a nonhealing granulomatous response to a beryllium-contaminated skin wound. This subject had a confirmed abnormal blood test, and after several additional months developed lung granulomas. Only one CBD case had an abnormal chest X-ray. An additional 11 former employees had CBD, for a total prevalence of 19/709. Beryllium-sensitized cases were similar to nonsensitized ones in terms of age, ethnic background, or smoking status, but did have significantly fewer pack-years of smoking. There was also no significant difference in percentage exposed to beryllium dust or mist in an accident or unusual incident, or those in areas with a posted high air count. Of the eight sensitized workers, seven had worked in machining at some

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point, while one case had never worked in a production job. The beryllium sensitization rate was 14.3% among the machinists, compared to 1.2% among all other employees. The individual average exposures for the six CBD cases and two sensitized cases among current employees ranged from 0.2 to 1.1 μg/m³, and the cumulative exposure ranged from 92.6 to 1.945 μg/m³-days. Two of the seven beryllium-sensitized machinists started machining prior to the systematic environmental monitoring, and exposure estimates for these two subjects may have been underestimated. The median of estimated average beryllium exposure for the sensitized cases was about 0.55 μg/m³. The sensitized cases without disease did not have lower exposures than the CBD cases. Machinists may have been more susceptible than other groups because of their higher overall exposure or because the particles produced during machining were primarily respirable in size, while other exposures were to particles larger than the respirable range (< 10 μm). Other characteristics of the machining exposure, such as the particle morphology, particle surface properties, and adjuvants in machining fluids, may also have affected sensitization. The study authors noted that median breathing zone levels tended to be lower than the DWAs derived from these levels because much of the day was typically spent in high-exposure tasks. This study identified a LOAEL of 0.55 μg/m³ and a LOAEL(HEC) adjusted for an occupational exposure of 0.19 μg/m³.

Eisenbud et al. (1949) evaluated beryllium exposure for 11 community cases of CBD. CBD was defined based on limited radiographic and pathologic examination. Radiologic screening of 10,000 residents was conducted, with questionable cases undergoing clinical evaluation. CBD was diagnosed based on radiologic and clinical findings from a consensus of specialists, but no sensitization information. One case was exposed to beryllium dust on worker clothes and will not be discussed further. Of the other cases, five lived within 0.25 mile of a beryllium production plant and all lived within 0.75 mile of the plant. A follow-up to this study reported three additional cases at less than 0.75 miles from the plant, but no additional cases of CBD at greater than 0.75 miles (Sterner and Eisenbud, 1951). Measurements downwind from the plant found that the beryllium concentration at 0.75 miles was about 0.045 μg/m³, and continuous sampling stations found that the average concentration at about 700 feet from the plant (the furthest distance within the affected area) was 0.05 μg/m³(range 0 to 0.46 μg/m³). The emitted beryllium was primarily as beryllium oxide, although beryllium fluoride and beryl (beryllium ore) were also present. The study authors also calculated an estimated exposure, based on emissions levels, stack heights, and wind speed data. These estimates were generally in good agreement with the downwind data. Based on these calculations, the authors estimated that the average exposure levels at 0.75 miles from the plant during the period of exposure monitoring were 0.004 to 0.02 μg/m³. Averaging this value to 0.01 μg/m³, and noting that both plant production and emissions were about 10-fold higher in earlier years, the authors estimated that the concentration at 0.75 mile was 0.01 to 0.1 μg/m³. The similar prevalence of CBD in the community compared to workers exposed to much higher levels (100 to 1,000 μg/m³) was attributed to the smaller particle size of beryllium emitted to the outside air compared to beryllium particles inside the plant. This study, although limited by classification of CBD, suggest a NOAEL(HEC) of 0.01 to 0.1 μg/m³ for the development of CBD in a population exposed to beryllium in ambient air.

Few data are available on the particle characteristics of beryllium under occupational exposure conditions. However, Hoover et al. (1990) found that 5.7% of the particles released during sawing beryllium metal had aerodynamic diameters smaller than 25 μm but larger than 5 μm, and 0.3% were smaller than 5 μm. For milling of beryllium metal, 12% to 28% of the particles had aerodynamic diameters between 5 and 25 μm, and 4% to 9% were smaller than 5 μm, depending on the milling depth. More than 99% of the particles generated from operations conducted with beryllium alloys were larger than 25 μm.

I.B.3. UNCERTAINTY AND MODIFYING FACTORS (INHALATION RfC)

UF = 10.

The available data suggest that only a small percentage of the population (1% to 5%)

appears to be susceptible to CBD (Kreiss et al., 1994). Because individuals developing beryllium sensitization and CBD are the most sensitive subpopulation, an uncertainty factor of 1 was used to account for human variability. An uncertainty factor of 1 was also used to adjust for the less-than-chronic exposure duration of the Kreiss et al. (1996) study; use of this uncertainty factor is supported by the evidence that the occurrence of CBD does not appear to be related to exposure duration. Because the screening method used in the Kreiss et al. (1996) study was more sensitive than the methods used in the Eisenbud et al. (1949) study, the RfC was derived from the LOAEL (Kreiss et al., 1996) with an uncertainty factor of 3 to account for the sensitive nature of the subclinical endpoint (beryllium sensitization). A database uncertainty factor of 3 was used to account for the poor quality of exposure monitoring in the co-principal studies and other epidemiology studies that assessed the incidence of beryllium sensitization and CBD among exposed workers and community residents. Although there are no developmental studies or 2-generation reproduction studies, a limited continuous breeding study found that beryllium does not cause reproductive or developmental effects following intratracheal administration (Clary et al., 1975). In addition, systemic distribution of beryllium is less than 1% (U.S. EPA, 1987), and any systemic effects would be expected to occur at exposure levels much above the very low levels at which CBD is observed.

MF = 1.

___I.B.4. ADDITIONAL STUDIES/COMMENTS (INHALATION RfC)

A. SUPPORTING STUDIES

Cullen et al. (1987) detected five likely cases of CBD (using the beryllium case registry definition of CBD) in workers who were exposed to beryllium oxide fumes at a precious metals refinery for 4 to 8 years before the development of symptoms. Time-weighted average personal air samples for beryllium ranged from 0.22 to 42.3 $\mu g/m^3$ throughout the plant, and 10% of the samples were greater than 2.0 $\mu g/m^3$. However, four of the cases worked predominantly in the furnace area, where beryllium exposure was measured at 0.52 ± 0.44 $\mu g/m^3$ (maximum measurement 1.7 $\mu g/m^3$). No additional cases were found in the screening of current workers, but a fifth was identified after the screen. This subject worked as a crusher, where exposure was to beryllium metal dust at 2.7 ± 7.2 μg Be/m^3. The CBD cases had the classic signs of CBD, including radiologically visible hilar adenopathy, noncaseating granuloma and pulmonary fibrosis in biopsy samples, and decreased carbon monoxide diffusing capacity (DLCO). Symptoms progressed even after removal from exposure. Beryllium sensitization was shown in vitro with BAL lymphocytes. Three of the cases were considered to have CBD, while diagnosis of two (both in the furnace area) was complicated by a history of hilar enlargement in one; the other had schistosomiasis and no BAL stimulation data. The study authors also analyzed beryllium exposure levels by job classification and screened 45 of 70 current workers for CBD, using interviews and analysis of spirometry data and chest radiographs from routine testing. No in vitro screening for beryllium sensitization was conducted on the general worker population. Noting that the prevalence of CBD was highest at a task with lower exposure levels, the study authors suggested that the beryllium oxide fumes to which workers were exposed in the furnace area were more toxic than the beryllium metal dust to which workers were exposed at other tasks. Alternative explanations for the development of disease following low-level exposure were considered unlikely. Although sampling efficiency was low for particles < 0.8 microns, these small particles were not considered to contribute significantly to the overall mass; however, because of their great surface area, they may have been much more toxic than larger particles. Although the study authors note that there were no significant changes in work practices during the previous 20 years, it is possible that the retrospective sampling conducted in a 2-week period may not accurately reflect past and present exposure conditions. The LOAEL in this study was 0.52 $\mu g/m^3$, with a LOAEL(HEC) after adjusting for occupational exposure of 0.18 $\mu g/m^3$.

CBD has resulted in death, especially in earlier years, when exposure was much higher than it is now. In a cohort mortality study of 689 patients with CBD who were included in the case

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registry, there was a high rate of deaths due to pneumoconiosis, primarily CBD (SMR = 34.23, 158 deaths) (Steenland and Ward, 1991). Similar results were observed in an earlier analysis of deaths due to nonneoplastic respiratory disease in the beryllium case registry (Infante et al., 1980). Deaths have also been reported in community cases of CBD, including in a 10-year-old girl (Lieben and Williams, 1969). Some of these cases have been confirmed based on histological evidence of CBD and evidence of beryllium in the lungs.

In addition, CBD has been reported in people not occupationally exposed to beryllium, including people living in communities near beryllium plants (Chesner, 1950; Dattoli et al., 1964; Lieben and Metzner, 1959) and in families of beryllium workers who wore contaminated clothing at home. These cases have been markedly reduced by better hygiene, including mandatory work clothes exchange, but nonoccupational CBD still has been reported following low-level episodic exposure of family members (Newman and Kreiss, 1992).

The onset of CBD from initial exposure (latency) ranges from a few months to an average of 23.7 years (Kreiss et al., 1993); however, at the present time there is no clear relationship between duration of exposure and the development of the disease. Some studies suggest that early stages of CBD can be reversed (Rom et al., 1983; Sprince et al., 1978), although these conclusions are weakened by methodological limitations of these studies (Kanarek et al., 1973; Kriebel et al., 1988b; Rom et al., 1983); however, beryllium sensitization was observed to progress to CBD even in the absence of continued exposure, suggesting that abnormal BeLT test findings are predictive of future development of CBD (Kreiss et al., 1994).

A number of earlier studies have characterized the signs and symptoms of CBD. Clinical, radiographic, and traditional spirometric signs of CBD are less sensitive than histologic findings and immunologic screens using the BeLT. Computed tomography (CT) can identify some CBD cases missed by chest radiography, but even CT missed 25% of histologically confirmed cases (Newman et al., 1994). Initial symptoms of early cases of CBD typically include dyspnea, cough, fatigue, weight loss, and chest pain (Aronchik, 1992; Hasan and Kazemi, 1974; Kriebel et al., 1988c; Meyer, 1994; Sterner and Eisenbud, 1951; Williams, 1993). Other symptoms included bibasilar crackles, clubbing of the fingers and skin lesions, heart failure, and an enlarged liver or spleen. Prominent diagnostic findings are diminished vital capacity, diffuse infiltrates, and radiographically visible hilar adenopathy. Fibrosis occurs at late stages in the disease. Granulomatous inflammation also has been reported in extrapulmonary organs, such as extrapulmonary lymph nodes, skin, liver, spleen, kidney, bone, myocardium, central nervous system, and skeletal muscle.

The development of fiberoptic bronchoscopy and transbronchial biopsy methods has allowed the identification of subclinical cases of CBD. Newman et al. (1989) evaluated respiratory symptoms and physical examination results in 12 cases of newly identified CBD based on (1) a history of beryllium exposure, (2) histopathological evidence of noncaseating granulomas or mononuclear cell infiltrates, and (3) a positive blood or BAL BeLT. Based on these findings, the authors suggested that CBD be classified into (1) sensitization, (2) subclinical beryllium disease (sensitized subjects with histopathological evidence, but no clinical signs), and (3) beryllium lung disease (as for [2], but with respiratory symptoms, changes on chest radiographs, or altered pulmonary physiology).

The BeLT has allowed the identification of an exposure-response relationship for beryllium sensitization (Kreiss et al., 1993a). Kreiss et al. (1989) used the peripheral blood BeLT to screen occupationally exposed populations for CBD, and found that 6/51 (11.8%) of the currently exposed workers were sensitized. No sensitized cases who had not yet developed CBD were identified, perhaps because of the long latency period. Although subjects identified based on BeLT test results may not have clinical signs of CBD, many do exhibit functional impairment (Pappas and Newman, 1993).

B. GENETICS OF BERYLLIUM SENSITIVITY

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Evidence from a variety of sources shows that genetic susceptibility plays a role in the development of CBD. Early occupational studies proposed that CBD was an immune reaction with a genetic component, based on the extreme sensitivity of certain individuals and the lack of CBD in others who are exposed to levels several orders of magnitude higher (Sterner and Eisenbud, 1951). Animal studies support these results. Immune granulomas are observed in strain 2 guinea pigs, but not in strain 13 guinea pigs, which differ from strain 2 only at the MHC Ia locus (Barna et al., 1984a). Similarly, beryllium inhalation caused immune granulomas in A/J mice, but not in BALB/c or C57B1 mice, which have different MHC class 2 genes (Huang et al., 1992). These studies suggest that differences in CBD susceptibility are related to differences at the MHC locus. Saltini et al. (1989) demonstrated that T cells only respond to the antigen (in this case, beryllium or beryllium plus some protein) in association with MHC class II molecules on the surface of the antigen-presenting cell. Granuloma formation has been hypothesized to result from a cytokine amplification loop involving macrophages, lymphocytes, and other factors (Newman, 1996).

Recent studies have identified a genetic marker linked to CBD susceptibility. The MHC class II region includes the HLA-DR, DQ, and DP genes. Richeldi et al. (1993) and Stubbs et al. (1996) reported associations between the presence of MHC class II alleles and the development of CBD in beryllium-exposed workers. Both studies found a biased distribution of HLA-DPB1 alleles (glutamine in position 69) in beryllium-sensitized subjects. They also found a biased distribution of the MHC class II HLA-DR gene, but found no association with specific amino acid changes. Thus, neither of these markers are completely specific for CBD, but the data do support a strong genetic contribution to CBD susceptibility, and the markers may be useful for screening for sensitive workers. It is also not clear if the association between either allele and CBD is a causal one. However, Richeldi et al. (1993) noted that structure-function studies of MHC class II molecules indicate that the amino acid change associated with CBD may affect an amino acid that plays a critical role in antigen binding. The more common allele of HLA-DP1 has a positively charged amino acid (lysine) at position 69, while the glutamate-69 variant is negatively charged at this site and could directly interact with the beryllium ion. Nonetheless, the high percentage (~ 30%) of exposed workers without CBD who had this allele suggests that other factors also contribute to the development of CBD. The beryllium exposure level plays at least some role, since the overall prevalence of CBD in exposed workers is 2% to 5%, while the prevalence at certain highly exposed tasks is as much as 15% (Kreiss et al., 1993a, 1996).

C. CHRONIC ANIMAL STUDIES AND MODELS

Although a number of chronic studies in laboratory animals have been conducted with beryllium compounds, few have been done using modern criteria for high-quality toxicology studies. In addition, whereas several laboratory animal species (such as mice, dogs, and monkeys) respond to beryllium exposure with several features of human CBD, no laboratory animal model fully mimics all features of human CBD. In particular, the animal models fail to demonstrate a model that has a progressive granulomatous pulmonary response with a concomitant beryllium-specific immune response. In addition, no chronic studies are available on nonneoplastic effects of beryllium oxide, the most environmentally relevant form.

Considerable research has investigated the mechanism of CBD and attempted to identify an appropriate animal model for CBD. An appropriate animal model for CBD is one that forms immune granulomas following the inhalation of beryllium dust or fume. These immune granulomas are distinct from granulomas formed by foreign-body reactions (Haley, 1991). Immune granulomas result from persistent antigenic stimulation, while foreign-body granulomas result from persistent irritation. Histologically, foreign-body granulomas consist predominantly of macrophages and monocytes and small numbers of lymphocytes. By contrast, immune granulomas are characterized by larger numbers of lymphocytes, primarily T lymphocytes, known as T-cells. The studies (Haley et al., 1990; Finch et al., 1994; Haley, 1991; Sendelbach et al., 1986; Hart et al., 1984; Sanders et al., 1975) show that acute beryllium disease occurs in the rat, but the rat is not an appropriate model for CBD because it does not mount an immune response to inhaled beryllium. Mice may be an appropriate model for CBD, although not all aspects of the

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disease have been replicated in this species in these studies (Haung et al.. 1992; Finch et al., 1996). The guinea pig and beagle dog may be good models for CBD based on the studies by Barna et al. (1981, 1984a,b) with respect to the guinea pig and Haley et al. (1989, 1992) with respect to the dog. In the case of guinea pigs, intratracheal instillation of beryllium oxide can induce both immune granulomas containing a T lymphocyte component and a beryllium-specific immune response. However, the effect has not yet been demonstrated under physiological conditions (inhalation exposure), and specificity for beryllium over other metals has not been demonstrated. In the case of the beagle dog, granulomatous lesions and lung lymphocyte responses consistent with those observed in humans with CBD were observed following a single exposure (perinasally; 0.01 μg beryllium/m^3 for 5 to 40 min) to beryllium oxide aerosol generated from beryllium oxide calcined at 500 or 1,000C. However, Conradi et al. (1971) found no exposure-related histological alterations in the lungs of six beagle dogs exposed to a range of 3,300 to 4,380 μg Be/m^3 as beryllium oxide calcined at 1,400C for 30 min, once per month for 3 mo.

Conradi et al. (1971) found no effect in monkeys (*Macaca irus*) exposed via whole-body inhalation for three 30-min monthly exposures to a range of 3,300 to 4,380 μg Be/m^3 as beryllium oxide calcined at 1,400C. On the other hand, Haley et al. (1994) showed that beryllium can induce immune granulomas and beryllium sensitization to beryllium metal and not BeO in monkeys, although this was not shown using a physiologically relevant route, namely, intrabronchiolar instillation.

___I.B.5. CONFIDENCE IN THE INHALATION RfC

Study -- Medium
Database -- Medium
RfC -- Medium

The overall confidence in this RfC assessment is medium. The RfC is based on an occupational inhalation study performed with a moderate to large group size (136 subjects screened) in which sensitive measures were used to identify the affected population (Kreiss et al., 1996). No NOAEL was identified in this study, but a NOAEL slightly below the LOAEL(HEC) was suggested in a study using less sensitive methods of diagnosing CBD in a population exposed to high levels of beryllium in ambient air (Eisenbud et al., 1949). The poor quality of the exposure monitoring in the co-principal studies decreases the confidence in the principal studies to medium. The confidence in the database is also medium. A common limitation in the database is the lack of adequate exposure monitoring in the epidemiology studies and some uncertainty regarding the mechanism (and beryllium exposure levels) associated with the progression to CBD in beryllium-sensitized individuals. Several human and animal studies are currently being conducted that may provide additional information on the mechanisms of action and data that would be useful for dose-response assessment. In particular, several investigators are conducting screening and monitoring studies of workers at the Rocky Flats Environmental Technology site and Oak Ridge site. Although no inhalation developmental or multigenerational reproductive studies were available for beryllium, no reproductive effects were observed in an intratracheal reproduction study in animals at exposure levels above those causing CBD (Clary et al., 1975). In addition, the unusually low level at which CBD occurs, together with the low systemic distribution of inhaled beryllium, mean that any developmental effects would occur at levels much higher than those causing CBD. Reflecting the medium confidence in the principal studies and database, confidence in the RfC is medium.

___I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

Source Document -- U.S. EPA, 1998

This assessment was peer reviewed by external scientists. Their comments have been evaluated

carefully and incorporated in finalization of this IRIS summary. A record of these comments is included as an appendix to the Toxicological Review of Beryllium in support of Summary Information on the Integrated Risk Information System (IRIS) (U.S. EPA, 1998).

Other EPA Documentation -- U.S. EPA, 1987; U.S. EPA, 1991

Agency Consensus Date-- 03/26/1998

___I.B.7. EPA CONTACTS (INHALATION RfC)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS in general at (513) 569-7254 (phone), (513) 569-7159 (fax) or RIH.IRIS@EPAMAIL.EPA.GOV (Internet address).

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Beryllium and compounds
CASRN -- 7440-41-7
Last Revised -- 04/03/1998

Section II provides information on three aspects of the carcinogenic assessment for the substance in question: the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per µg/L drinking water or risk per µg/m^3 air breathed. The third form in which risk is presented is a concentration of the chemical in drinking water or air providing cancer risks of 1 in 10,000, 1 in 100,000, or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in the Risk Assessment Guidelines of 1986 (U.S. EPA, 1986) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (U.S. EPA, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II.A. EVIDENCE FOR HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CHARACTERIZATION

B1; probable human carcinogen. Based on the limited evidence of carcinogenicity in humans exposed to airborne beryllium (lung cancer) and sufficient evidence of carcinogenicity in animals (lung cancer in rats and monkeys inhaling beryllium, lung tumors in rats exposed to beryllium via intratracheal instillation, and osteosarcomas in rabbits and possibly mice receiving intravenous or intramedullary injection), beryllium is reclassified from a B2 (inadequate human data) to a B1 probable human carcinogen (limited human data) using criteria of the 1986 Guidelines for Carcinogen Risk Assessment. Using the 1996 proposed Guidelines for Carcinogen Risk Assessment, inhaled beryllium would be characterized as a "likely" carcinogen in humans, and the human carcinogenic potential of ingested beryllium cannot be determined.

Studies regarding the potential carcinogenicity of ingested beryllium to humans were not available. Increases in lung cancer mortality have been observed in cohort mortality studies of beryllium processing workers (Ward et al., 1992; Wagoner et al., 1980; Mancuso, 1979, 1980)

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and in studies of entrants on the BCR (Steenland and Ward, 1991; Infante et al., 1980). No increases in other types of cancer were found, but increases in deaths from nonmalignant respiratory disease were also observed. Newer studies, particularly the occupational study of Ward et al. (1992), have been considered as the basis for a dose-response assessment, but share a limitation with the Wagoner et al. (1980) study--lack of individual exposure monitoring or job history data that would support a more definitive exposure assessment. NIOSH has recently completed a lung cancer case-control study nested within a cohort mortality study of beryllium manufacturing workers at the Reading beryllium processing facility. The study developed an exposure matrix and calculated airborne beryllium exposure concentrations and thus may provide the best available basis for a quantitative cancer estimate. Rather than calculate an interim quantitative estimate based on the Ward et al. (1992) data and poorly defined exposure estimates, the existing unit risk based on the Wagoner et al. (1980) study is retained until the NIOSH assessment can be evaluated as the basis for a quantitative estimate.

Chronic oral studies of the potential carcinogenicity of beryllium in animals were conducted at dose levels below the MTD, and therefore are inadequate for the assessment of carcinogenicity. Beryllium has been shown to induce lung cancer in rats exposed to beryllium by both inhalation and intratracheal instillation and in monkeys by inhalation. Osteosarcomas have been produced in rabbits and possibly in mice by intravenous and intramedullary injection using a variety of beryllium compounds and beryllium metal. No tumors were produced by intracutaneous or percutaneous injections of beryllium compounds.

The majority of studies do not induce gene mutations in bacterial assays with or without metabolic activation. Gene mutations have been observed in mammalian cells cultured with beryllium chloride. Culturing mammalian cells with beryllium chloride, beryllium sulfate, or beryllium nitrate has resulted in clastogenic alterations.

___II.A.2. HUMAN CARCINOGENICITY DATA

Limited. The most recently published studies are cohort mortality studies of beryllium processing workers (Ward et al., 1992) and of entrants to the beryllium case registry (Steenland and Ward, 1991). The Ward et al. (1992) is a follow-up to the studies by Mancuso (1979, 1980) and Wagoner et al. (1980) and the Steenland and Ward (1991) is a follow-up to the study by Infante et al. (1980).

Wagoner et al. (1980) conducted a cohort mortality study of 3,055 white males employed between 1942 and 1967 at a beryllium extraction, processing, and fabrication facility in Reading, Pennsylvania. The study cohort was followed through 1975. The total number of deaths (875) was not significantly different from the number expected on the basis of age and calendar time period for the general white male U.S. population (vital statistics data for the period 1965-1967 were assumed to be those of 1968-1975). Significant ($p < 0.05$) increases in the number of deaths due to malignant neoplasm of trachea, bronchus, and lung (47 deaths observed versus 34.29 expected, SMR = 1.37), heart disease (SMR = 1.13), and nonneoplastic respiratory disease (excluding influenza and pneumonia) (SMR = 1.65) were observed in the study cohort. When deaths from lung cancer were segregated by latency (interval since onset of employment) and duration of employment, significant increases were observed for workers with a 25-year latency employed for < 5 years (17 observed versus 9.07 expected, SMR = 1.87) and across all employment durations (20 observed versus 10.79 expected, SMR = 1.87). (It should be noted that 83% of the cohort was employed for < 5 years.) When lung cancer mortalities were partitioned based on initial date of employment, lung cancer deaths were significantly higher in workers hired before 1950 (SMR = 1.35; $p < 0.05$); an increase in deaths in workers hired after 1950 was also found, but it was not statistically significant (SMR = 1.52). (Prior to 1950, beryllium exposures were not controlled and it is likely that the workers were exposed to high concentrations of beryllium.) Similar findings were reported when nonneoplastic respiratory disease mortalities were segregated; a significant increase in mortality was observed in the workers in the 25-year latency and < 5-year employment category (SMR = 2.13). In the workers who were hired prior to 1950, a significant increase in

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mortality from nonneoplastic respiratory disease was observed (SMR = 1.85). This was not observed for workers whose initial date of employment was after 1950 (0 deaths observed versus 2.03 expected).

Wagoner et al. (1980) compared cigarette smoking histories of the cohort and the U.S. population using smoking habit information collected during a medical survey in 1968 and cigarette smoking data for white males from a Public Health Service Survey conducted in 1964-1965. A smaller percentage of the cohort were current smokers (49.6% never smoked or were former smokers versus 45.2% in the U.S. population), but the percentage of current smokers smoking more than one pack per day was higher (21.4% versus 15.3%).

Limitations of the study include the following:

1. The use of U.S. white male mortality data for the period of 1941 to 1967, which resulted in an underestimation of the number of expected lung cancer deaths because lung cancer death rates in the United States were increasing during the period 1968-1975. The expected number of lung cancer deaths should have been 10% to 11% higher (U.S. EPA, 1987; Saracci, 1985).
2. The inclusion of one lung cancer death of an individual who was paid for the pre-employment physical but was not hired (U.S. EPA, 1987).
3. The exclusion of approximately 300 white males employed at the Reading facility in similar jobs as the workers included in the cohort (U.S. EPA, 1987).
4. The inadequate discussion of confounding effects from other potential lung carcinogens (U.S. EPA, 1987).

These limitations tended to exaggerate the risk of lung cancer in this population of workers potentially exposed to beryllium (MacMahon, 1994; U.S. EPA, 1987).

EPA (1987) adjusted the lung cancer SMRs from the Wagoner et al. (1980) study. The expected lung cancer deaths were increased by 11% to account for the underestimation that occurred from using older U.S. vital statistics and by 4.1% to account for differences in smoking habits between the beryllium cohort and the U.S. population. The one ineligible lung cancer death was removed from the observed deaths. Although the SMRs for latency 25 years remained elevated after this adjustment (1.42 for < 5 years employment and 1.36 across all durations of employment), they were no longer statistically significant at $p < 0.05$.

Ward et al. (1992) conducted a retrospective cohort mortality study of 9,225 men (5,681 alive and 3,240 dead) employed for at least 2 days between January 1, 1940, and December 31, 1969, and followed through December 31, 1988, at any one of seven beryllium processing facilities located in Reading, PA, Hazleton, PA, Lorain, OH, Cleveland, OH (data for Perkins and St. Clair plants combined), Lucky, OH, and Elmore, OH. Cohort members were identified from quarterly earning reports from the Social Security Administration and compared to personnel files. Workers identified from quarterly earning reports without personnel files were only included in the cohort if they appeared on at least two quarterly earning reports. Workers who worked at more than one facility were placed into a seventh category termed "multiple plant." Vital statistics for the workers were obtained from the Social Security Administration, Internal Revenue Service, post office cards mailed to the last known address, Veterans Administration, Health Care Financing Administration, and the National Death Index. Vital statistics were not located for 304 (3.3%) individuals, and death certificates were not obtained for 46 (0.4%) individuals known to be deceased.

The workers at the beryllium processing facilities were involved in the extraction of beryllium hydroxide from beryl ore; the production of beryllium oxide, pure beryllium metal, and beryllium copper alloy; and the machining of beryllium-containing products. The beryllium compounds the workers were potentially exposed to included beryllium sulfate mists and fumes, beryllium oxide dusts, beryllium ammonium fluoride and beryllium fluoride dusts, beryllium metal, and beryllium copper alloy dusts and fumes. In addition to exposure to beryllium, the workers also were potentially exposed to ore dust, silicon dioxide fumes, lead sulfide, copper

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sulfide, sulfur trioxide, acid fluoride mists, hydrogen fluoride, and ammonium fluoride. In addition, according to the Beryllium Industry Scientific Advisory Committee (BISAC) (1997), exposure to sulfuric acid mists and fumes occurred in the Lorain facility. Because no occupational history data other than starting and ending dates of employment were coded, and no individual monitoring data were available, the study could not address the relationship of degree of beryllium exposure or type of beryllium compound to lung cancer risk. Ward et al. (1992) noted that prior to 1949 when controls were not mandated, air concentrations of beryllium were very high, frequently exceeding 1,000 $\mu g/m^3$.

When mortality from all causes in the entire cohort was compared to mortality rates from the U.S. population, a significant ($p < 0.05$) increase in risk was observed (SMR of 1.05, 95% confidence interval [CI] of 1.01-1.08). Excess deaths were also observed for malignant neoplasm of the trachea, bronchus, and lung (SMR = 1.26, 95% CI = 1.12-1.42), ischemic heart disease (SMR = 1.08, 95% CI = 1.01 to 1.14), pneumoconiosis and other respiratory disease (SMR = 1.48, 95% CI = 1.21-1.80), and chronic and unspecified nephritis, renal failure, and other renal sclerosis (SMR = 1.49, 95% CI = 1.00-2.12). Examination of the cause of death on a per plant basis revealed that only the Lorain and Reading facilities (the two oldest plants) had significant excesses in lung cancer: total SMRs of 1.69 and 1.24, respectively. In addition, the Cleveland and Hazleton facilities had nonsignificant excesses in lung cancer (total SMRs > 1).

A significant excess of pneumoconiosis and other respiratory disease was also observed at the Lorain facility (SMR = 1.94). Increased employment duration was not associated with an increase in lung cancer SMR; when lung cancer mortalities were stratified by employment duration category, the only significant increase in lung cancer SMRs was for workers employed < 1 year. However, there was a tendency for lung cancer SMRs to increase with increasing latency; SMRs were statistically significantly elevated in the > 30-year latency category for all employment durations combined (SMR = 1.46) and for workers employed for < 1 year (SMR = 1.52), and in the 25- to 30-year latency category for workers employed for < 1 year. Additionally, decade of hire influenced lung cancer mortality. The SMR (1.42) was statistically elevated in workers hired before 1950; this was mainly influenced by mortality in the Lorain plant, which closed in 1948. Of the other facilities in operation before 1950 (Reading and Cleveland), an increased lung cancer rate was found at the Reading facility (SMR = 1.26 for workers hired before 1950). With the exception of those hired before 1950 (total SMR = 1.42), no other significant increases in lung cancer deaths were observed when workers were grouped by decade of hire. Nonsignificant increases were seen for the 1950s decade at the Reading (SMR = 1.42), Cleveland (SMR = 1.32), Elmore (SMR = 1.42), and Hazleton (SMR = 1.86) facilities. Regression analysis (controlling for age, race, and calendar period of risk) showed that decade of hire was independent of potential latency (time since first employment).

The influence of geographic variation in lung cancer mortality was evaluated comparing lung cancer mortality found in the beryllium cohort to lung cancer rates for the cohort where most of the workers resided. Lung cancer mortality was significantly elevated in workers at the Lorain (SMR = 1.60, 95% CI = 1.21-2.08) and Reading (SMR = 1.42, 95% CI = 1.18-1.69) facilities as compared to residents of Lorain County and Berks County, respectively. The investigators noted that county residents may not serve as a better referent group than the U.S. population because the percentage of workers at the Lorain and Reading facilities residing in an urban area was approximately 3 times higher than the percentage of county residents living in urban areas.

Data on the smoking habits of the entire beryllium cohort were not available. Some information was available from a 1968 Public Health Service survey conducted at the Reading, Hazleton, Elmore, and St. Clair facilities (it included 15.9% of the cohort members). These data were compared to smoking habits of the United States population obtained from averaging smoking surveys conducted in 1965 (National Center for Health Statistics) and 1970 (Office of Health Research, Statistics, and Technology). The estimated relative risk ratio for lung cancer for the beryllium cohort to the United States population was calculated using estimated risks of 1 for nonsmokers, 6.5 for current smokers of 1 pack per day, 13.8 for smokers of > 1 pack per day, and 6.2 for former smokers. The relative risk ratio or smoking adjustment was 1.1323, which indicates

that smoking alone could account for an SMR of 1.13.

Using the smoking adjustment factor, smoking-adjusted SMRs were calculated for the entire cohort and the Lorain and Reading facilities. The resultant SMRs were 1.12, 1.49, and 1.09, respectively. One process exposure, to which BISAC (1997) attributed the excess cancer (SMR 1.49) after adjustment for smoking, and which was briefly mentioned by Ward et al. (1992), is exposure to mists and vapors from sulfuric acid and the related sulfur oxide gases. Such exposure, according to BISAC (1997), was very high in the Lorain plant. This was the only plant that used a sulfuric acid-dependent process with limited ventilation (Kjellgren, 1946); at the time, the occupational inhalation risks associated with airborne beryllium (acute pneumonitis) and airborne sulfuric acid (respiratory cancer) had not yet been established. Because the 1968 survey data are the only information available on the smoking habits of the beryllium cohort, an assumption was made that the smoking habit difference between the cohort and the United States population found in the late 1960s was the same in the 1940s and 1950s. Other investigators have shown that increased smoking is unlikely to account for SMRs greater than 1.3 for lung cancer and other smoking-related diseases (Siemiatycki et al., 1988).

Ward et al. (1992) examined the beryllium case registry (BCR) mortality study file to determine how many of the members of the cohort were registered (i.e., had a history of beryllium disease). The Lorain plant had highest percentage of registrants--8.2% (98 of 1,192 workers); 93% of these were listed as having had acute beryllium disease, which is associated with very high exposure (Eisenbud and Lisson, 1983). The lung cancer SMR for the Lorain workers on the BCR was 3.33 (95% CI = 1.66-5.95) compared to 1.51 (95% CI = 1.11-2.02) for the remaining Lorain workers.

Ward et al. (1992) concluded that a plausible explanation for the observed increased lung cancer rates is occupational exposure to beryllium. Although the results of this study are suggestive that occupational exposure to beryllium can result in an increase in lung cancer mortality, interpretation of this study is limited by a number of factors:

1. No data (including job history data) were available to associate beryllium exposure levels, exposure to specific beryllium compounds, or concomitant exposure to other chemicals with members of the cohort.
2. Because of the lack of job history data, it is possible that the cohort contained salaried workers and other nonproduction personnel who may not have been exposed to beryllium.
3. The limitations in the available smoking habit data, as discussed above, may have led to an over- or underestimation of the contribution of smoking to the lung cancer rates.
4. A large percentage (73.1%) of the workers were employed in the beryllium industry for 5 years. This is particularly true at the Lorain facility, where 84.6% of the workers were employed for < 1 year. EPA (1987) points out that there is a possibility that the workers were exposed to other potential carcinogens at jobs held before or after the beryllium job; the two facilities with the highest cancer rates (Lorain and Reading) are located in or near heavily industrialized areas.

Regardless of the shortcomings of the epidemiological studies of beryllium exposure, the results of the follow-up mortality studies on the same cohort and of the BCR cohort studies are suggestive of a causal relationship between beryllium exposure and an increased risk of lung cancer. The increased incidences of lung cancers among workers with acute beryllium disease (presumably these workers were exposed to very high concentrations of beryllium), the higher incidences of lung cancers among workers first employed when exposure levels were very high, a consistent finding of lung cancer excesses in six of seven beryllium processing facilities, and the occurrence of the highest risks for lung cancer in plants where the risk for nonmalignant respiratory disease is the highest strengthen this conclusion.

II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Lung tumors have been induced via inhalation and intratracheal administration

to rats and monkeys, and osteosarcomas have been induced via intravenous and intramedullary injection in rabbits and possibly in mice. The chronic oral studies did not report increased incidences of tumors in rodents, but these were conducted at doses below the MTD.

In a chronic toxicity study by Morgareidge et al. (1975, 1977), groups of Wistar albino rats were fed diets containing 5, 50, or 500 ppm beryllium as beryllium sulfate tetrahydrate. The rats were administered the beryllium containing diet from 4 weeks of age through maturation, mating, gestation, and lactation. Fifty male and 50 female offspring were then placed on the same diets as the parents and fed the beryllium-containing diet for 104 weeks. Using estimated TWA body weights of 0.467, 0.478, and 0.448 kg for males in the 5, 50, and 500 ppm groups and 0.294, 0.302, and 0.280 kg for the females, respectively, and U.S. EPA's (1988) allometric equation of food intake, doses of 0.36, 3.6, and 37 mg/kg-day for males in the 5, 50, and 500 ppm groups and 0.42, 4.2, and 43 mg/kg-day for females in the 5, 50, and 500 ppm groups, respectively, were calculated. Clinical observations, body weight, food consumption, organ weights (liver, kidney, testes, ovaries, thyroid, pituitary, adrenal), gross necropsy, and histopathological examination of most tissues and organs (25 to 26 tissues examined) were used to assess the toxicity and carcinogenicity of beryllium in the offspring; it does not appear that the P_0 rats were examined. Tissues from 20 rats/sex/group in the control and 500 ppm groups were examined microscopically (the study authors did not state whether the animals undergoing histopathological examination were randomly selected), as well as all tissues with gross abnormalities (all groups) and tissues (excluding bone marrow, eyes, and skin) from animals found dead or sacrificed moribund (all groups).

No overt signs of toxicity were observed, and mortality appeared to be similar in the controls (30/50 males and 28/50 females died) and beryllium groups at 5 ppm (30/50 and 24/50, respectively), 50 ppm (31/50 and 18/50), and 500 ppm (24/50 and 17/50) at the end of the 104 weeks of the study. During the first 40 to 50 weeks of the study, exposure to beryllium did not appear to affect growth; slight decreases in growth (body weights of males and females in the 500 ppm group were within 10% of control body weights) were observed in the latter part of the study; however, no statistically significant alterations were observed. Alterations in organ weights were limited to statistically significant ($p < 0.05$) increases in relative kidney weight in males exposed to 50 ppm, decreases in relative kidney and adrenal weights in 500 ppm females, and decreases in relative testes weights in 5 and 50 ppm males. Histological examination of the major organs and tissues did not reveal beryllium-related noncarcinogenic alterations. These data suggest that the maximum tolerated dose (MTD) was not reached.

Reticulum cell sarcomas were observed in a number of tissues examined including the lungs, lymph nodes, spleen, liver, kidneys, and pancreas; the highest incidence was in the lungs. Because lymphomas (reticulum cell sarcoma is a type of lymphoma) are almost always detected grossly, reticulum cell sarcoma incidences were calculated based on the number of tissues grossly examined (all gross diagnoses were confirmed histopathologically) rather than on the number of tissues microscopically examined. In most organs, the incidence of reticulum cell sarcomas was not significantly higher in the beryllium-exposed rats, as compared to controls. In the lung, the incidences of reticulum cell sarcoma were 10/50, 17/50, 16/50, and 12/50 in males and 5/50, 7/50, 7/50, and 5/50 in females exposed to 0, 5, 50, or 500 ppm beryllium, respectively. The incidences of lung reticulum cell sarcomas in the beryllium-exposed rats were not significantly different from controls. The incidences of reticulum cell sarcoma-bearing rats in the 0, 5, 50, and 500 ppm groups were 12/50, 18/50, 16/50, and 13/50, respectively, for males and 8/50, 11/50, 7/50, and 8/50 for the females; no significant increases in tumor-bearing rats was found. No other treatment-related increases in tumor incidence were observed.

In a lifetime exposure study, groups of 52 male and 52 female Long-Evans rats were maintained on a low-metal diet and given drinking water containing 0 or 5 ppm beryllium as beryllium sulfate (presumably tetrahydrate) from weaning to natural death (Schroeder and Mitchener, 1975a). The water also contained 5 ppm chromium III, 50 ppm zinc, 5 ppm copper, 10 ppm manganese, 1 ppm cobalt, and 1 ppm molybdenum. Doses of 0.63 and 0.71 mg/kg-day were

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calculated for male and female rats, respectively, using estimated TWA body weights of 0.42 and 0.26 kg and U.S. EPA's (1988) allometric equation for water consumption. The following parameters were used to assess toxicity: body weights (animals weighed at weekly and monthly intervals for the first year and at 3-mo intervals thereafter), blood glucose, cholesterol, and uric acid (blood samples collected from 12 rats/sex after an 18-h fast), urine protein, pH, and glucose, heart weight, gross pathology, and histopathology of heart, lung, kidney, liver, spleen, and tumors. Twenty male and eight female rats in the beryllium group died at 20 mo of age from pneumonia; a similar number of animals in the control group also died from pneumonia. At 30 days, the male and female rats exposed to beryllium weighed significantly more than the control animals. At 60, 90, 120, and 180 days, the beryllium-exposed male rats weighed significantly less than the controls; no significant alterations in body weight were observed at the other time intervals (150, 360, or 540 days). Overall, the weight loss of less than 10% indicates that the MTD was not achieved. No significant alterations in mortality or longevity were observed. Glycosuria (females only) and alterations in serum glucose levels were observed in the beryllium-exposed rats. The alterations in serum glucose levels consisted of significantly lower levels in males aged 475 days and higher levels in males and females aged 719 days. It should be noted that the control rats were at least 50 days older than the beryllium-exposed rats when blood samples were collected, and in the controls, blood glucose levels declined with increasing age. Significantly increased serum cholesterol levels were observed in female rats exposed to beryllium at ages 475 and 719 days. The results of the histological examination were not reported. The alterations in serum glucose and cholesterol levels and urine glucose levels were not considered adverse because the magnitude of the alterations was not sufficiently large to suggest an impairment in organ function. The incidence of gross tumors was 4/26 (15%) and 17/24 (70%) in the male and female control rats and 9/33 (27%) and 14/17 (82%) in the male and female rats exposed to beryllium. The incidences of malignant tumors (tumors were considered malignant if there were multiple tumors in the same animal) were 2/26 (7.7%) and 8/24 (33%) in the male and female controls and 4/33 (12%) and 8/57 (14%) in the male and female beryllium-exposed rats. The incidences of gross or malignant tumors in the control and beryllium-exposed groups was not significantly different. It should be noted that in an unpublished report (Schroeder and Nason, 1976), the incidence of gross tumors in the male and female beryllium-exposed rats was 4/25 and 13/20 (control data the same as reported in published paper). In the published paper, this is the tumor incidence for the tungsten-exposed male and female rats, respectively. It is difficult to determine which is the correct tumor incidence data for the beryllium-exposed rats; however, neither set of incidence data is statistically significantly different than controls.

In a similar lifetime exposure study, groups of 54 male and 54 female Swiss mice were administered 0 or 5 ppm beryllium as beryllium sulfate in the drinking water from weaning to natural death (Schroeder and Mitchener, 1975b). The mice were fed low-metal diets and the drinking water was supplemented with 50 ppm zinc, 10 ppm manganese, 5 ppm copper, 5 ppm chromium III, 1 ppm cobalt, and 1 ppm molybdenum. The 5 ppm water concentration is equivalent to doses of 1.2 mg/kg-day for the male and female mice, using an estimated TWA body weight of 0.042 and 0.035 kg and EPA's (1988) allometric equation for water consumption. In the beryllium group, statistically significant alterations in body weight were observed; the alterations included heavier male mice at 30 days and lighter female mice at 90 and 120 days. Overall, the decrease in body weight was less than 10%, indicating that the MTD was not reached. No significant alterations in mortality or survival were observed in the beryllium-exposed mice. No alterations in tumor incidence were observed.

Reeves et al. (1967) exposed 150 male and 150 female Sprague-Dawley rats for 7 h/day, 5 days/week to 34.25 μg Be/m^3 as beryllium sulfate aerosol (average particle size was 0.118 μm, electron microscopy) for up to 72 weeks, with 3 of each sex sacrificed monthly during exposure. An equal number of control rats were exposed to distilled water aerosol. Lung weights were markedly increased in the exposed rats, and an inflammatory lung response (characterized as a marked accumulation of histiocytic elements and thickened and distorted alveolar septa) was noted, accumulation of alveolar macrophages. A proliferative response was also noted, progressing from hyperplasia to alveolar adenocarcinomas in 100% of the exposed rats at 13 months, compared to 0% of the controls. Histopathologic examination was limited to the lungs.

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The authors noted that 8 male and 4 female rats in the control group and 9 male and 17 females in the beryllium group died during the course of the study. The plateau body weight in the beryllium-exposed female rats was approximately 25% less than found in the controls (statistical significance not reported).

Vorwald and Reeves (1959) exposed Sherman rats (number and sex not reported) via the inhalation route to aerosols of beryllium sulfate at 6 and 54.7 μg Be/m³ for 6 h/day, 5 days/week for an unspecified duration. Animals were sacrificed periodically and examined histopathologically. Initially, inflammation consisted of histiocytes, lymphocytes, and plasma cells scattered throughout the lung parenchyma. Following more prolonged exposures, more focal lesions consisting primarily of histiocytes were observed. Multinucleated giant cells were also observed. Thickened alveolar walls and fibrotic changes were also observed. Lung tumors, primarily adenomas and squamous cell cancers, were observed in the animals sacrificed after 9 months of this exposure regime.

Similar results to those of Vorwald and Reeves (1959) were observed in a study by Reeves and Deitch (1969, as reviewed by U.S. EPA, 1987). In this study, groups of 20 to 25 Charles River CD rats were exposed to 35.66 μg Be/m³ as beryllium sulfate for 35 h/week; the mean particle size was 0.21 μm (d_{ae}). The exposure durations were 800 h (5 groups), 1,600 h (2 groups), and 2,400 h (1 group). Age at the initiation of exposure appeared to be a more important variable for tumor development than was exposure duration. The lung tumor incidence (19/22, 86%) for young rats exposed for 3 mo was the same as in rats exposed for 18 mo (13/15, 86%), but was higher than in older rats exposed for 3 mo (3-10/20-25, 15-40%). Tumors were typically observed after a latency period of 9 mo. In the beryllium-exposed rats, the epithelial hyperplasia observed at 1 mo progressed to metaplasia at 5 to 6 mo, and anaplasia by 7 to 8 mo.

In a study to test the carcinogenicity of beryllium ores, Wagner et al. (1969) exposed groups of 12 male squirrel monkeys (*Saimiri sciurea*), 60 male CR-CD rats, 30 male Greenacres Controlled Flora (GA) rats, and 48 male Golden Syrian hamsters to 0 or 15 mg/m³ bertrandite or beryl for 6 h/day, 5 days/week for 17 mo (rats and hamsters) or 23 mo (monkeys). The test atmospheres generated from the bertrandite ore ($Be_4Si_2O_7[OH]_2$; 1.4% beryllium) and beryl ore ($B_3Al_2Si_6O_{18}$; 4.14% beryllium) contained 210 and 620 μg Be/m³, respectively, and the geometric mean diameters of the particles were 0.27 μm (geometric standard deviation of 2.4) and 0.64 μm (geometric standard deviation of 2.5). Both ores contained very high silicon dioxide levels (63.9% by weight). Exposed and control monkeys, rats, and hamsters were serially sacrificed on completion of 6 and 12 mo of exposure; rats and hamsters at 17 months, and monkeys at 23 mo. Five control rats and five rats from the 12- and 17-mo exposure groups were sacrificed in order to determine the free-silica content of the lung tissue. At exposure termination, beryllium concentrations in the lungs were 18.0 and 83 μg/g fresh tissue in the bertrandite- and beryl-exposed rats, 14.1 and 77.4 μg/g fresh tissue in the bertrandite- and beryl-exposed hamsters, and 33 and 280 μg/g fresh tissue in the bertrandite- and beryl-exposed monkeys. Free silica (SiO_2) levels in the rat lungs were 30 to 100 times higher in the beryllium ore-exposed rats than in the controls. Increased mortality was observed in the monkeys (11%), rats (13%), and hamsters (25%) exposed to either bertrandite or beryl ore, with the highest mortality rates in the bertrandite ore-exposed animals (no further details provided). No significant alterations in body weight gain were observed in the monkeys or hamsters.

In the rats, decreased body weight gains (terminal body weights were 15% lower compared to controls) were observed beginning after 6 mo of exposure, and from 12 mo to exposure termination at 17 mo. In the beryl-exposed rats, small foci of squamous metaplasia or tiny epidermoid tumors were observed in the lungs of 5/11 rats killed after 12 mo of exposure. At exposure termination, lung tumors were observed in 18/19 rats (18 had bronchiolar alveolar cell tumors, 7 had adenomas, 9 had adenocarcinomas, and 4 had epidermoid tumors). Additional alterations in the lungs included loose collections of foamy macrophages and cell breakdown products, lymphocyte infiltrates around the bronchi, and polymorphonuclear leukocytes and lymphocytes present in most of the bronchiolar-alveolar cell tumors. In the bertrandite-exposed

rats, granulomatous lesions composed of several large, tightly packed, dust-laden macrophages were observed in all rats exposed for 6, 12, or 17 mo. No tumors were observed. Neoplastic or granulomatous pulmonary lesions were not observed in the control rats. In the beryl- and bertrandite-exposed monkeys, the histological alterations consisted of aggregates of dust-laden macrophages, lymphocytes, and plasma cells near respiratory bronchioles and small blood vessels. No tumors were found. In the bertrandite-exposed hamsters, granulomatous lesions consisting of tightly packed, dust-laden macrophages were observed after 6 mo, and the number did not increase after 17 mo. These alterations were not observed in the beryl-exposed or control hamsters. Atypical proliferation and lesions, which were considered bronchiolar alveolar cell tumors except for their size, were observed in the hamsters after 12 mo of exposure to beryl or bertrandite. After 17 mo of exposure, these lesions became larger and more adenomatous in the beryl-exposed hamsters. It should be noted that silicosis was not observed in any of the animals exposed to the beryllium ores, which contained a large amount of free silica. No significant gross or histologic alterations were observed in the thymus, spleen, liver, or kidneys of the beryllium-exposed rats, hamsters, and monkeys.

In a monkey carcinogenicity study (Vorwald, 1968), a group of 7 male and 9 female rhesus monkeys (*Macaca mulatta*) (aged 18 months) were exposed to 35 μg Be/m³ beryllium sulfate mist 6 h/day, 5 days/week. The author notes that the "exposure was interrupted, often for considerable periods of time, in order to maintain the best possible overall well-being of the animal, to prevent a threatening acute beryllium pneumonitis, and to favor survival to old age or at least long enough for the inhaled beryllium to exert its maximal chronic effects in terms of epithelial proliferation, metaplasia, and cancer." The exposure schedule was presented in a figure, but it was difficult to determine the exposure protocol from this figure. The longest exposure was for 4,070 h, with most of the exposure occurring during the first 4.5 years of the study with an approximate 6-mo exposure 2.5 years later. Four animals died within the first 2 mo of the study; the cause of death was acute chemical pneumonitis. Lung cancer was observed in 8 of the 12 remaining animals. The first tumor was observed in a monkey 8 years of age exposed for 3,241 hours. The tumors were described as a gross mass located in either the hilar area or more peripheral portions of the lung or as small and large tumors scattered irregularly throughout the pulmonary tissue.

A single-exposure inhalation study of beryllium metal in F344/N rats resulted in a 64% incidence of lung carcinomas over the lifetime of the animals (Nickell-Brady et al., 1994). Groups of 30 males and 30 females were administered a single, nose-only exposure to a beryllium metal aerosol (MMAD = 1.4 μm, GSD = 1.9) at 500 mg/m³ for 8 min, 410 mg/m³ for 30 min, 830 mg/m³ for 48 min, or 980 mg/m³ for 39 min. Control rats were exposed to filtered air alone. Mean lung burdens resulting from these exposures were 40, 110, 360, and 430 μg of beryllium, respectively. Tumors became apparent by 14 mo after exposure, and the incidence (apparently for all groups combined) was 64% over the lifetime of the rats. Multiple tumors were frequently found, the majority were adenocarcinomas, and some were > 1 cm.

Lung tumors have been observed in rats following a single intratracheal instillation of beryllium metal, passivated beryllium metal (99% beryllium, < 1% chromium), beryllium-aluminum alloy (62% beryllium), and beryllium hydroxide. Beryllium alloys containing 4% beryllium did not result in increases in lung tumors. Lung tumor incidences of 11% to 51% were observed in rats following intratracheal instillation of beryllium oxide fired at high, low, and medium temperatures. The types of lung tumors found in animals receiving intratracheal instillations of beryllium included adenocarcinomas, adenomas, squamous cell carcinoma, and malignant lymphoma. Osteosarcomas have been induced by intravenous and intramedullary injection of various beryllium compounds into rabbits, and possibly mice. The osteosarcomas in rabbits are histologically similar to human osteosarcomas (U.S. EPA, 1987, 1991).

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

The genotoxicity of beryllium has been previously reviewed by EPA (1987) and recently

reviewed by IARC (1993). Most studies have found that beryllium chloride, beryllium nitrate, beryllium sulfate, and beryllium oxide did not induce gene mutations in bacterial assays with or without metabolic activation. In the case of beryllium sulfate, all mutagenicity studies (Ames [Simmon, 1979; Dunkel et al., 1984; Arlauskas et al., 1985; Ashby et al., 1990]; E. coli pol A [Rosenkranz and Poirer, 1979]; E. coli WP2 uvr A [Dunkel et al., 1984]) and *Saccharomyces cerevisiae* (Simmon, 1979) were negative with the exception of results reported for Bacillus subtilis rec assay (Kada et al., 1980; Kanematsu et al., 1980) and E. coli rec assay (Dylevoi, 1990). Beryllium sulfate did not induce unscheduled DNA synthesis in primary rat hepatocytes and was not mutagenic when injected intraperitoneally in adult mice in a host-mediated assay using *Salmonella typhimurium* (Williams et al., 1982).

Beryllium nitrate was negative in the Ames assay (Tso and Fung, 1981; Kuroda et al., 1991) but positive in a Bacillus subtilis rec assay (Kuroda et al., 1991). Beryllium chloride was negative in a variety of studies (Ames [Ogawa et al., 1987; Kuroda et al., 1991]; E. coli WP2 uvr A [Rossman and Molina, 1986]; and Bacillus subtilis rec assay [Nishioka, 1975]). In addition, beryllium chloride failed to induce SOS repair in E. coli (Rossman et al., 1984). However, positive results were reported for Bacillus subtilis rec assay using spores (Kuroda et al., 1991), E. coli KMBL 3835; lacI gene (Zakour and Glickman, 1984), and hprt locus in Chinese hamster lung V79 cells. Beryllium oxide was negative in the Ames assay and Bacillus subtilis rec assays (Kuroda et al., 1991).

Gene mutations have been observed in mammalian cells (V79 and CHO) cultured with beryllium chloride (Miyaki et al., 1979; Hsie et al., 1979a,b), and culturing of mammalian cells with beryllium chloride (Vegni-Talluri and Guiggiani, 1967), beryllium sulfate (Brooks et al., 1989; Larramendy et al., 1981), or beryllium nitrate has resulted in clastogenic alterations.

Data on the in vivo genotoxicity of beryllium are limited to a single study that found beryllium sulfate (1.4 and 2.3 g/kg, 50% and 80% of median lethal dose) administered by gavage did not induce micronuclei in the bone marrow of CBA mice, although a marked depression of erythropoiesis suggestive of bone marrow toxicity was evident 24 h after dosing. No mutations were seen in *p53* or c-*raf* -1 and only weak mutations were detected in K-*ras* in lung carcinomas from F344/N rats given a single nose-only exposure to beryllium metal (Nickell-Brady et al., 1994). The authors concluded that the mechanisms for the development of lung carcinomas from inhaled beryllium in the rat do not involve gene dysfunctions commonly associated with human non-small-cell lung cancer.

Regarding human exposure, beryllium particles produced from anthropogenic processes almost always enter the atmosphere as BeO. Depending on the particle size, they can be deposited near the source or transported long distances. The inhalation toxicity of insoluble beryllium oxide, which appears to be the chemical form more likely than beryllium salts to be present in the atmosphere, depends to a great extent on its physical and chemical properties, which can be altered considerably, depending on production conditions. It is well known that the toxicity of beryllium oxide is dependent on the particle size, with smaller particles (< 10 μm, d_{ae}) able to penetrate beyond the larynx.

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

The oral database is considered inadequate for the assessment of carcinogenicity. The chronic oral studies did not report increased incidences of tumors in rodents, but were conducted at doses below the MTD. The oral database, including the Schroeder and Mitchener study (1975a) previously used in the development of the oral slope factor on IRIS, is considered inadequate for the assessment of carcinogenicity. The basis for not using the Schroeder and Mitchener rat study (1975a) is that the incidences of gross or malignant tumors in the control and beryllium-exposed groups were not significantly different.

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II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

The cancer dose-response assessment based on the occupational study of Wagoner et al. (1980) that is presented in this section was derived by EPA (1987), was verified in 1988, and loaded on IRIS. Newer studies, particularly the occupational study of Ward et al. (1992), have been considered as the basis for a dose-response assessment, but share a limitation with the Wagoner et al. (1980) study--lack of individual exposure monitoring or job history data that would support a more definitive exposure assessment. NIOSH has recently completed a lung cancer case-control study nested within a cohort mortality study of beryllium manufacturing workers at the Reading beryllium processing facility. The study developed an exposure matrix and calculated airborne beryllium exposure concentrations and thus may provide the best available basis for a quantitative cancer estimate. The study is currently in peer review. Rather than calculate an interim quantitative estimate based on the Ward et al. (1992) data and poorly defined exposure estimates, it is recommended that the existing unit risk based on the Wagoner et al. (1980) study be retained until the NIOSH assessment can be evaluated as the basis for a quantitative estimate.

II.C.1. SUMMARY OF RISK ESTIMATES

II.C.1.1. Air Unit Risk -- 2.4E-3 per ($\mu g/m^3$)*

II.C.1.2 Extrapolation Method -- Relative risk

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	4E-2 per ($\mu g/m^3$)
E-5 (1 in 100,000)	4E-3 per ($\mu g/m^3$)
E-6 (1 in 1,000,000)	4E-4 per ($\mu g/m^3$)

*The unit risk should not be used if the air concentration exceeds 4 $\mu g/m^3$, since above this concentration the unit risk may not be appropriate.

II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSURE

Tumor Type -- Lung cancer
Test Animals -- Human, male
Route -- Inhalation, occupational exposure
Reference -- Wagoner et al., 1980

Beryllium concentration in workplace ($\mu g/m^3$)	Ratio of years of exposure to years at risk (f/L)	Effective dose ($\mu g/m^3$)	95 percent upper-bound estimate of relative risk	Unit risk per ($\mu g/m^3$)
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
1,000	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

II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

The epidemiology study by Wagoner et al. (1980) is used to estimate the lifetime cancer risk from exposure to beryllium oxide based on the estimated lower and upper bounds of exposure estimated by NIOSH; namely, 100 and 1,000 $\mu g/m^3$. The effective dose was determined by adjusting for duration of daily (8/24 h) and annual (240/365) exposure and the ratio of exposure duration to duration at risk, i.e., f years out of a period of L years at risk (from onset of employment to termination of follow-up). Two values of f/L were used in the calculations, f/L = 1 and 0.25. An f/L of 1.0 would avoid overestimating the risk (but could underestimate the risk) if the observation by Reeves and Deitch (1969)--that tumor yield depends not on the length of exposure but on age at exposure--is valid. For a given "effective" dose d and a relative risk R, the carcinogenic potency (q1*) is calculated by the formula B = $(R - 1) \times 0.036/d$, where 0.036 is the estimated lung cancer mortality rate in the U.S. population. 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 ($p > 0.05$) and 1.44 ($p > 0.05$) were derived and the 95% upper confidence limits of these estimates, 1.98 and 2.09, respectively, were used to estimate the lifetime cancer risk (unit risk).

With the possible exception of the Wagner et al. (1969) study, the results of the animal carcinogenicity studies are incompletely reported and the studies are not of sufficient quality to be used as the basis of quantitative cancer risk estimates. Because Wagner et al. (1969) exposed the rats to beryllium ores with relatively low beryllium levels and high levels of silica dioxide, this study would not be an appropriate basis for a risk estimate for general population exposure to beryllium.

II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

The estimate for risk for inhalation was based on an epidemiologic study having several confounding variables. The estimate of exposure levels and duration of exposure by NIOSH were also somewhat uncertain. While a quantitative estimate based on several animal studies (U.S. EPA, 1987) resulted in a similar estimate of risk, the epidemiological data are considered a better basis for quantitating risk; the assessment based on the animal data is, however, supportive of the

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assessment based on the human data.

The results of the epidemiological studies have been criticized (BISAC, 1997; MacMahon, 1994; U.S. EPA, 1987; Saracci, 1985). EPA (1987, 1991) considered the studies conducted prior to 1987 to be insufficient to assess the carcinogenic potential of beryllium in humans. Although the design of the Ward et al. (1992) study corrected a number of the shortcomings of the older studies, the interpretation of the study results is limited by the assumption used to account for lung cancer deaths due to cigarette smoking, the lack of job history data that would support quantitative exposure assessment, the lack of control for potential exposure to other carcinogens, including coexposure to sulfuric or hydrofluoric acid mists during employment in the beryllium industry or nonconcurrent exposure to other carcinogens during employment outside of the beryllium industry, and the relatively small increases in lung cancer risks. Exposure to sulfuric acid mists, however, has not been strongly associated with lung cancer, but rather with laryngeal cancer (IARC, 1992; Sathiakumar et al., 1997). Limitations in the evidence for an association between exposure to sulfuric acid mist and lung cancer include poor or no quantitation of exposure, possible confounding by other occupational exposures and smoking, and low SMRs. The majority of lung cancer SMRs in the studies that reported a positive association between exposure to sulfuric acid mists and lung cancer were in the range of 1.18 to 1.39. The studies of lung cancer in workers exposed occupationally to beryllium and/or sulfuric acid or other acid mists do not, for the most part, categorize the type of cancer. Thus, the data are insufficient to determine whether different types of lung cancer may be associated with beryllium exposure versus sulfuric acid exposure. Exposures to hydrofluoric acid and hydrogen fluoride are potential confounders at some of the beryllium processing facilities, including the Reading plant (Ward et al., 1992; BISAC, 1997). Information regarding the potential carcinogenicity of these compounds was not available. IARC (1992) considered hydrofluoric acid to be a weak inorganic acid and did not assess it in the monograph on strong inorganic acids.

IARC (1993; Vainio and Rice, 1997) considered the epidemiological data as sufficient evidence in humans for the carcinogenicity of beryllium and compounds. IARC (1993) concluded that the issue of adjustments for smoking had been handled adequately, and stated that a limitation of the most recent cohort studies was the absence of discussion of potential exposure to other lung carcinogens, although "there is no evidence that other lung carcinogens were present." The U.S. EPA, however, considers that the issues of incomplete smoking data and exposure to other potential lung carcinogens are not completely resolvable with the data currently available, and therefore concludes that the evidence of carcinogenicity of beryllium and compounds is limited in humans.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1998

This assessment was peer reviewed by external scientists. Their comments have been evaluated carefully and incorporated in finalization of this IRIS summary. A record of these comments is included as an appendix to the Toxicological Review of Beryllium in support of Summary Information on the Integrated Risk Information System (IRIS) (U.S. EPA, 1998).

Other EPA Documentation -- U.S. EPA, 1987, 1991

II.D.2. EPA REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Consensus Date -- 03/26/1998

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II.D.3. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS in general at (513) 569-7254 (phone), (513) 569-7159 (fax), or RIH.IRIS@EPAMAIL.EPA.GOV (Internet address).

III. [RESERVED]

IV. [RESERVED]

V. [RESERVED]

VI. BIBLIOGRAPHY

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CASRN -- 7440-41-7
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_VII. REVISION HISTORY

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Beryllium and compounds
CASRN 7440-41-7

Date	Section	Description
03/01/1988	I.A.1.	Reference dose table clarified
03/01/1988	I.A.2.	Text added
09/07/1988	II.	Carcinogen summary on-line
01/01/1990	II.A.2.	References clarified
01/01/1990	II.A.3.	Text revised
01/01/1990	II.B.	Quantitative estimate for oral exposure section added
01/01/1990	II.C.3.	Text revised
01/01/1990	II.D.2.	Work group review dates and verification date added
01/01/1990	VI.	Bibliography on-line
02/01/1990	VI.A.	Puzanova et al. 1978 citation corrected
02/01/1990	VI.C.	Wagner et al. 1969 citation corrected
09/01/1990	I.A.	Morgareidge ref. now Cox (same study-authors reversed)
09/01/1990	IV.F.1.	EPA contact changed
09/01/1990	VI.A.	Morgareidge ref. now Cox (same study-authors reversed)
01/01/1991	II.	Text edited
01/01/1991	II.C.1.	Inhalation slope factor removed (global change)
01/01/1992	IV.	Regulatory actions updated
09/01/1992	II.A.3.	U.S. EPA citation year corrected, paragraph 3
09/01/1992	II.D.1.	Source document year corrected
09/01/1992	II.D.1.	Review statement revised
09/01/1992	VI.C.	U.S. EPA reference year corrected
02/01/1993	I.A.7.	Primary contact changed
04/03/1998	I.A.	Oral RfD Assessment
04/03/1998	I.B.	Inhalation RfC Assessment
04/03/1998	II.	Carcinogenicity Assessment

_VIII. SYNONYMS

Beryllium and Compounds
CASRN -- 7440-41-7
Last Revised -- 04/03/1998

7440-41-7
Beryllium
Beryllium-9
Glucinum
RCRA waste number P015
UN 1567

Note: A *TOXICOLOGICAL REVIEW* is available for this chemical in Adobe* PDF format (94 Pages, 320 Kbytes)

00088711

Similar documents can be found in the List of Available IRIS Toxicological Reviews

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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Cadmium

CASRN 7440-43-9

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0141
Cadmium; CASRN 7440-43-9

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Cadmium

File On-Line 03/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	02/01/1994
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	06/01/1992

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Cadmium
CASRN -- 7440-43-9
Last Revised -- 02/01/1994

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

___I.A.1. 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

*Conversion Factors: See text for discussion

___I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

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 ian 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).

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

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.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

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.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1985

Other EPA Documentation -- None

Agency Work Group Review -- 05/15/1986, 08/19/1986, 09/17/1987, 12/15/1987, 01/20
05/25/1988

Verification Date -- 05/25/1988

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Cadmium
CASRN -- 7440-43-9

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Cadmium
CASRN -- 7440-43-9
Last Revised -- 06/01/1992

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

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B1; probable human carcinogen

Basis -- 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.

II.A.2. 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.

II.A.3. 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.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

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; Oberly 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).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

Not available. There are no positive studies of orally ingested cadmium suitable for quantitation.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

___II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk -- 1.8E-3 per (ug/cu.m)

Extrapolation Method -- Two stage; only first affected by exposure; extra risk

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

___II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSUR

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 1E+3 x 8/24 x 1/365 x 240/365.

___II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE

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.

___II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOS

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).

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1985

The Addendum to the Cadmium Health Assessment has received both Agency and external review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 11/12/1986

Verification Date -- 11/12/1986

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Cadmium
CASRN -- 7440-43-9
Last Revised -- 10/01/1989

__VI.A. ORAL RfD REFERENCES

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VI.B. INHALATION RfD REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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_VII. REVISION HISTORY

Substance Name -- Cadmium
CASRN -- 7440-43-9

Date	Section	Description
05/21/1987	II.C.	Slope factor corrected
03/01/1988	II.A.1.	Text added
03/01/1988	II.C.3.	Text revised
03/01/1988	II.C.4.	Confidence statement revised
03/01/1988	II.D.3.	Secondary contact changed
01/01/1989	IV.C.1.	Water quality human health criteria added
01/01/1989	IV.C.2.	Corrected marine acute criterion
08/01/1989	VI.	Bibliography on-line
10/01/1989	I.A.	Oral RfD summary on-line
10/01/1989	VI.A.	Oral RfD references added
12/01/1989	I.B.	Inhalation RfD now under review
06/01/1990	IV.A.1.	Area code for EPA contact corrected
06/01/1990	IV.F.1.	EPA contact changed
08/01/1990	II.A.1.	Basis statement revised
08/01/1990	II.A.2.	Text revised, paragraph 1
08/01/1990	II.B.	Text revised
01/01/1991	II.	Text edited
01/01/1991	II.C.1.	Inhalation slope factor removed (global change)
03/01/1991	II.A.1.	Text revised
03/01/1991	II.B.	Text revised
01/01/1992	IV.	Regulatory actions updated
04/01/1992	IV.A.1.	CAA regulatory action withdrawn
05/01/1992	II.C.2.	Number correction in data table
06/01/1992	II.A.2.	Text revised, paragraph 1
06/01/1992	II.A.3.	Text clarified
02/01/1994	I.A.7.	Secondary contact changed

VIII. SYNONYMS

00088722

Substance Name -- Cadmium
CASRN -- 7440-43-9
Last Revised -- 03/31/1987

7440-43-9
C.I. 77180
Cadmium
KADMIUM

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0141.htm

00088723

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Chromium(III), insoluble salts

CASRN 16065-83-1
(09/03/1998)

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

Note: ***A TOXICOLOGICAL REVIEW*** is available for this chemical in Adobe* PDF format (44 Pages, 134 Kbytes)
Similar documents can be found in the List of Available IRIS Toxicological Reviews

0028
Chromium(III), insoluble salts; CASRN 16065-83-1; 09/03/1998

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices, Regional Offices, and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Chromium(III), insoluble salts

File First On-Line 01/31/1987

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Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	On-line	09/03/1998
Inhalation RfC Assessment (I.B.)	On-line	09/03/1998
Carcinogenicity Assessment (II.)	On-line	09/03/1998

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Chromium(III), insoluble salts
CASRN -- 16065-83-1
Last Revised -- 09/03/1998

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*		UF	MF	RfD
No effects observed	NOAEL:	5% Cr_2O_3 in diet 5 days/week for 600 feedings (1,800 g/kg bw average total dose)	100	10	1.5E+0 mg/kg-day
	NOAEL(ADJ):	1,468 mg/kg-day			
	LOAEL:	none			
	LOAEL(ADJ):	none			

Rat chronic feeding study
Ivankovic and Preussman, 1975

*Conversion Factors and Assumptions -- 1,800 g Cr_2O_3/kg bw × 1,000 mg/g × 0.6849 g Cr/g Cr_2O_3/600 feeding days × 5 feeding days/7 days = 1,468 mg/kg-day.

___I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Ivankovic, S; Preussmann, R. (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 (Cr_2O_3) 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 Cr_2O_3. Body weight and food consumption were monitored. The average total amounts of ingested Cr_2O_3 were given as 360, 720, and 1,800 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 deSRCibed for the accompanying subchronic study (see below). No effects due to Cr_2O_3 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% Cr_2O_3 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 1,400 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.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

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.

MF = 10.

An additional 10-fold modifying factor is applied to reflect database deficiencies including the lack of a study in a nonrodent mammal, lack of unequivocal data evaluating reproductive impacts, and the concern regarding potential reproductive effects raised by the study of Elbetieha and Al-Hamood (1997). The value of the modifying factor has not been changed from the previous IRIS

entry. The study of Elbetieha and Al-Hamood (1997) provides strong support for the use of a 10-fold modifying factor to reflect uncertainty regarding potential reproductive effects of Cr(III). The following additional uncertainties relate to the NOAEL derived from the Ivankovic and Preussman (1975) study: 1) the effects observed in the 90-day study were not explicitly addressed in the 2-year study; 2) the effect of the vehicle (baked bread) on absorption of chromium is uncertain, and the relevance of this dosing regimen to exposures in the environment is unclear; 3) animals were allowed to die naturally after feeding stopped (2 years) and only then was histology performed. Application of the 100-fold uncertainty factor and 10-fold modifying factor to the adjusted NOAEL of 1,468 mg/kg-day gives the reference dose of 1.5 mg/kg-day.

___I.A.4. ADDITIONAL STUDIES/COMMENTS (ORAL RfD)

This RfD is limited to metallic chromium (III) of insoluble salts. Examples of insoluble salts include chromic III oxide (Cr_2O_3) and chromium (III) sulfate ($Cr_2[SO_4]_3$).

Trivalent chromium is an essential element that potentiates insulin action in peripheral tissue and is essential for lipid, protein, and fat metabolism in animals and human beings. Chromium deficiency causes changes in the metabolism of glucose and lipids and may be associated with maturity-onset diabetes, cardiovascular diseases, and nervous system disorders (Anderson, 1993, 1995). The National Research Council has identified an estimated safe and adequate daily dietary intake (ESADDI) for chromium of 50-200 µg/d (NRC, 1989), corresponding to 0.71-2.9 µg/kg/d for a 70 kg adult. FDA has selected a Reference Daily Intake for chromium of 120 µg/d (DHHS, 1995).

Very limited data suggest that Cr(III) may have respiratory effects on humans (see Section I.B). No data on chronic or subchronic effects of inhaled Cr(III) in animals can be found. Adequate developmental toxicity data do not exist, and there are inadequate data on reproductive effects.

Elbetieha and Al-Hamood (1997) reported impacts on fertility following high doses (2,000-5,000 ppm in the drinking water) of chromium chloride in mice; however, many of the observed effects did not occur in a clear dose-dependent fashion. The authors did not indicate the amount of water ingested by the animals, and only stated that water ingestion was reduced in the treatment groups relative to the controls. Zahid et al. (1990) fed mice trivalent chromium at concentrations of 100, 200, and 400 ppm for 35 days in food and reported ambiguous levels of degeneration in the outermost cellular layers of the seminiferous tubules, reduced spermatogonia per tubule, reduced sperm count, and increased percentage of morphologically abnormal sperms at all dose levels. Serious questions have been raised regarding the design and conduct of this study (Finley et al., 1993; NTP, 1996a,b, 1997). The methods utilized by Zahid et al. were considered to be insufficient to identify spermatogonia, likely generated nonreproducible counts of epididymal sperm, and resulted in the biologically implausible conclusion of reduction in spermatogonia numbers concurrent with unchanged spermatocyte and spermatid numbers. Additional questions have been raised with regard to the groupings of animals used and the statistical analysis of the data. The uncertainties preclude the use of the Elbetieha and Al-Hamood (1997) and Zahid et al. (1990) studies in the risk assessment for trivalent chromium.

The 1998 IRIS assessment updates the previous RfD of 1E+0 mg/kg-day.

___I.A.5. CONFIDENCE IN THE ORAL RfD

Study -- Low
Database -- Low
RfD -- Low

The overall confidence in this RfD assessment is low. The principal study is rated low because of the lack of explicit detail on study protocol and results. Low confidence in the database 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 that might lower the RfD.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1998

This assessment was peer reviewed by external scientists. Their comments have been evaluated carefully and incorporated in finalization of this IRIS summary. A record of these comments is included as an appendix to the Toxicological Review of Trivalent Chromium in support of Summary Information on the Integrated Risk Information System (IRIS) (U.S. EPA, 1998).

Other EPA Documentation -- U.S. EPA, 1984

Agency Consensus Date -- 4/28/98

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (fax), or RIH.IRIS@EPAMAIL.EPA.GOV (Internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

Chromium(III), insoluble salts
CASRN -- 16065-83-1
Last Revised -- 09/03/1998

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is generally expressed in units of mg/m^3. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

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Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (U.S. EPA, 1989) and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (U.S. EPA, 1994). RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

___I.B.1. INHALATION RfC SUMMARY

Not available.

___I.B.2. PRINCIPAL AND SUPPORTING STUDIES (INHALATION RfC)

Not available.

___I.B.3. UNCERTAINTY AND MODIFYING FACTORS (INHALATION RfC)

Not available.

___I.B.4. ADDITIONAL STUDIES/COMMENTS (INHALATION RfC)

Data are considered to be inadequate for development of an RfC due to the lack of a relevant toxicity study addressing respiratory effects of Cr(III). Data from animal studies have identified the respiratory tract as the primary target of chromium toxicity following inhalation of hexavalent chromium and these data have been used for development of an RfC for hexavalent chromium particulates. However, these data do not demonstrate that the effects observed following inhalation of hexavalent chromium particulates are relevant to inhalation of trivalent chromium, and these data are considered to be inappropriate for development of an RfC for trivalent chromium. The following discussion of issues related to development of an RfC for Cr(III) has been added in 1998.

Occupational exposure to trivalent chromium and other chromium compounds by inhalation has been studied in the chromate manufacturing and ferrochromium industries; however, exposures all include mixed exposures to both Cr(III) and Cr(VI). A number of epidemiological studies have demonstrated an association between inhalation of Cr(VI) and noncarcinogenic endpoints, including upper respiratory irritation and atrophy, changes in lung function, and renal toxicity. These studies have been used to support the development of an RfC for chromic acid mists (VI) and soluble chromates (VI). Data addressing exposures to Cr(III) alone are not available, and the occupational studies are considered to be unsuitable for development of an RfC for Cr(III).

Several animal studies have been performed to assess the carcinogenic potential of Cr(III) by inhalation, either by natural routes, intrapleural injection, or intrabronchial implantation (Baetjer et al., 1959; Hueper and Payne, 1962; Levy and Venitt, 1975; Levy and Martin, 1983). These studies did not provide detailed reports of noncarcinogenic effects associated with inhalation exposure to Cr(III).

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Data from subchronic animal studies identify the respiratory tract as the primary target of chromium toxicity following inhalation. Johansson et al. (1986) exposed rabbits to aerosols of hexavalent (0.9 mg/m^3 Na_2CrO_4) or trivalent (0.6 mg/m^3 $Cr(NO_3)_3$) chromium for 5 days/week, 6 hours/day for 4 to 6 weeks. The number of macrophages obtained from the lungs of the rabbits exposed to Cr(VI) was significantly increased. While the numbers of macrophages from rabbits exposed to Cr(III) were not increased, striking morphological changes were observed, including round dark chromium-rich inclusions in the cytoplasm, an increased number of cells with a smooth inactive cell surface, enlarged Golgi apparatus, and a tendency toward elongated cell shape. The macrophages from rabbits exposed to Cr(VI) showed less marked morphological changes than those exposed to Cr(III). This study did not focus on endpoints that are considered suitable for development of an RfC for Cr(III).

Johansson et al. (1980) exposed groups of four rabbits to chromium dust at concentrations of 3.1 mg/m^3 and 0.6 mg/m^3 for 5 days/week, 6 hours/day for 4 weeks. Macrophages collected from rabbits exposed to the higher concentration of chromium phagocytized significantly more chromium particles than the controls, though the number of nonviable macrophages was less than 3%. This study utilized small groups and did not focus on endpoints that are considered to be suitable for development of an RfC for Cr(III).

Akatsuka and Fairhall (1934) exposed cats to chromium carbonate dust and found no effects in terms of gross or microscopic pathology upon termination of the experiment. Only two cats were exposed, however, and neither the doses nor the durations of exposure were precisely defined; therefore, these data cannot be used in quantitative risk assessment.

___I.B.5. CONFIDENCE IN THE INHALATION RfC

Not available.

___I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

Source Document -- U.S. EPA, 1998

This assessment was peer reviewed by external scientists. Their comments have been evaluated carefully and incorporated in finalization of this IRIS summary. A record of these comments is included as an appendix to the Toxicological Review of Trivalent Chromium in support of Summary Information on the Integrated Risk Information System (IRIS) (U.S. EPA, 1998).

___I.B.7. EPA CONTACTS (INHALATION RfC)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (fax), or RIH.IRIS@EPAMAIL.EPA.GOV (Internet address).

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_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Chromium(III), insoluble salts
CASRN -- 16065-83-1
Last Revised -- 09/03/1998

Section II provides information on three aspects of the carcinogenic assessment for the substance in question, the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per mg/kg-day. The unit risk is the quantitative estimate in terms of either risk per µg/L drinking water or risk per µg/m³ air breathed. The third form in which risk is presented is a concentration of the chemical in drinking water or air associated with cancer risks of 1 in 10,000, 1 in 100,000, or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are deSRCibed in the Risk Assessment Guidelines of 1986 (U.S. EPA, 1986) and in the IRIS Background Document. IRIS Summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (U.S. EPA, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II.A. EVIDENCE FOR HUMAN CARCINOGENICITY

The following discussion of issues related to potential Cr(III) carcinogenicity has been updated in 1998.

___II.A.1. WEIGHT-OF-EVIDENCE CHARACTERIZATION

Applying the criteria for evaluating the overall weight of evidence for carcinogenicity to humans outlined in EPA's guidelines for carcinogen risk assessment (U.S. EPA, 1986), trivalent chromium is most appropriately designated a Group D -- Not classified as to its human carcinogenicity. Using the Proposed Guidelines for Carcinogen Risk Assessment (EPA, 1996), there are inadequate data to determine the potential carcinogenicity of trivalent chromium, as discussed below. However, the classification of hexavalent chromium as a known human carcinogen raises a concern for the carcinogenic potential of trivalent chromium.

___II.A.2. HUMAN CARCINOGENICITY DATA

Occupational exposure to trivalent chromium and other chromium compounds by inhalation has been studied in the chromate manufacturing and ferrochromium industries; however, exposures all include mixed exposures to both Cr(III) and Cr(VI). The Cr(VI) species is the likely etiological agent in reports of excess cancer risk in chromium workers. Data addressing exposures to Cr(III) alone are not available, and data are inadequate for an evaluation of human carcinogen potential.

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Two oral studies located in the available literature (Schroeder et al., 1965; Ivankovic and Preussman, 1975) reported negative results for rats and mice. Several animal studies have been performed to assess the carcinogenic potential of Cr(III) by inhalation. These studies have not found an increased incidence of lung tumors following exposure either by natural routes, intrapleural injection, or intrabronchial implantation (Baetjer et al., 1959; Hueper and Payne, 1962; Levy and Venitt, 1975; Levy and Martin, 1983).

___II.A.3. ANIMAL CARCINOGENICITY DATA

The data from oral and inhalation exposures of animals to trivalent chromium do not support determination of the carcinogenicity of trivalent chromium. IARC (1990) concluded that animal data are inadequate for the evaluation of the carcinogenicity of Cr(III) compounds. Furthermore, although there is sufficient evidence of respiratory carcinogenicity associated with exposure to chromium, the relative contributions of Cr(III), Cr(VI), metallic chromium, or soluble versus insoluble chromium to carcinogenicity cannot be elucidated. In general, trivalent chromium was not mutagenic in bacterial assays when tested with or without a mammalian activation system (Venitt and Levy, 1974; Petrilli and Deflora, 1977, 1978a,b). In one study, trivalent chromium was mutagenic in *Baccillus subtilis* , but this activity was low compared with compounds of hexavalent chromium (Nakamuro et al., 1978). Taken together, these studies do not provide adequate data to support determination of the carcinogenicity of Cr(III), and a quantitative estimate of potential Cr(III) carcinogenicity has not been generated.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

Not available.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1998

00088732

This assessment was peer reviewed by external scientists. Their comments have been evaluated carefully and incorporated in finalization of this IRIS summary. A record of these comments is included as an appendix to the Toxicological Review of Trivalent Chromium in support of Summary Information on the Integrated Risk Information System (IRIS) (U.S. EPA, 1998).

___II.D.2. EPA REVIEW (CARCINOGENICITY ASSESSMENT)

Not available.

___II.D.3. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (fax), or RIH.IRIS@EPAMAIL.EPA.GOV (Internet address).

_III. [reserved]
_IV. [reserved]
_V. [reserved]

_VI. BIBLIOGRAPHY

Chromium(III), insoluble salts
CASRN -- 16065-83-1
Last Revised -- 09/03/1998

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00088736

_VII. REVISION HISTORY

Chromium(III), insoluble salts
CASRN -- 16065-83-1

Date	Section	Description
03/01/1988	I.A.1.	Critical effect added
03/01/1988	III.A.	Health Advisory added
08/01/1989	VI.	Bibliography on-line
08/01/1990	IV.F.1.	EPA contact changed
10/01/1990	I.B.	Inhalation RfC now under review
11/01/1990	III.A.	Full Health Advisory summary added
01/01/1992	I.A.7.	Secondary contact changed
01/01/1992	IV.	Regulatory actions updated
05/01/1992	II.	Carcinogenicity assessment now under review
09/01/1994	File	File name changed to include "insoluble salts"
09/03/1998	I, II, VI	Revised RfD, RfC, carcinogenicity sections, refs.

_VIII. SYNONYMS

Chromium(III), insoluble salts
CASRN -- 16065-83-1
Last Revised -- 09/03/1998

16065-83-1
7440-47-3
Chromic ion
Chromium
Chromium (III)
Chromium (III) ion
Chromium, ion

Note: ***A TOXICOLOGICAL REVIEW*** is available for this chemical in Adobe* PDF format (50 Pages, 151 Kbytes)
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00088737

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00088738

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Chromium(VI)

CASRN 18540-29-9
09/03/1998

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

Note: ***A TOXICOLOGICAL REVIEW*** is available for this chemical in Adobe* PDF format (70 Pages, 189 Kbytes)
Similar documents can be found in the List of Available IRIS Toxicological Reviews

0144
Chromium(VI); CASRN 18540-29-9; 09/03/1998

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices, Regional Offices, and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Chromium(VI)

File First On-Line 03/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	On-line	09/03/1998
Inhalation RfC Assessment (I.B.)	On-line	09/03/1998

Carcinogenicity Assessment (II.) On-line 09/03/1998

Conversion Factors and Assumptions -- Drinking water consumption = 0.1 L/kg-day (reported).

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Chromium(VI)
CASRN -- 18540-29-9
Last Revised -- 09/03/1998

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
None reported	NOAEL: 25 mg/L of chromium as K_2CrO_4 2.5 mg/kg-day (adj.)	300	3	3E-3 mg/kg-day
	LOAEL: None			

Rat, 1-year drinking study
MacKenzie et al., 1958

*Conversion Factors and Assumptions -- Drinking water consumption = 0.1 L/kg-day (reported).

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

MacKenzie, RD; Byerrum, RU; Decker, CF, et al. (1958) Chronic toxicity studies. I Hexavalent and trivalent chromium administered in drinking water to rats. Am Med Assoc Arch Ind Health 18:232-234.

Groups of eight male and eight female Sprague-Dawley rats were supplied with drinking water containing 0.45-11.2 ppm (0.45-11.2 mg/L) hexavalent chromium (as K_2CrO_4) for 1 year. The control group (10/sex) received distilled water. A second experiment involved three groups of 12 male and 9 female rats. One grou

was given 25 ppm (25 mg/L) chromium (as K_2CrO_4), a second received 25 ppm chromium in the form of chromic chloride, and the controls again received distilled water. No significant adverse effects were seen in appearance, weight gain, or food consumption, and there were no pathologic changes in the blood or other tiss in any treatment group. The rats receiving 25 ppm of chromium (as K_2CrO_4) showed approximate 20% reduction in water consumption. Based on the body weight of the rat (0.35 kg) and the average daily drinking water consumption for the rat (0.035 l/day), this dose can be converted to give an adjusted NOAEL of 2.5 mg/kg-chromium(VI).

For rats treated with 0-11 ppm (in drinking water), blood was examined monthly, and tissues (livers, kidneys, and femurs) were examined at 6 mo and 1 year. Spleens were also examined at 1 year. The 25 ppm groups (and corresponding controls) were examined similarly, except that no animals were killed at 6 mo. An abrupt rise in tissue chromium concentrations was noted in rats treated with more than 5 ppm. The authors stated that "apparently, tissues can accumulate considerable quantities of chromium before pathological changes result." In the 25 ppm treatment groups, tissue concentrations of chromium were approximately 9 times higher for those treated with hexavalent chromium than for the trivalent group. Similar no-effect levels have been observed in dogs. Anwar et al. (1961) observed no significant effects in female dogs (2/dose group) given up to 11.2 ppm chromium(VI) (as K_2CrO_4) in drinking water for 4 years. The calculated doses were 0.012-0.30 mg/kg of chromium(VI).

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF = 300.

The uncertainty factor of 300 represents two 10-fold decreases in dose to account for both the expected interhuman and interspecies variability in the toxicity of the chemical in lieu of specific data, and an additional factor of 3 to compensate for the less-than-lifetime exposure duration of the principal stu

MF = 3.

The modifying factor of 3 is to account for concerns raised by the study of Zhang and Li (1987).

___I.A.4. ADDITIONAL STUDIES/COMMENTS (ORAL RfD)

This RfD is limited to soluble salts of hexavalent chromium. Examples of soluble salts include potassium dichromate ($K_2Cr_2O_7$), sodium dichromate ($Na_2Cr_2O_7$), potassium chromate ($K_2Cr_2O_4$), and sodium chromate (Na_2CrO_4). Trivalent chromium is an essential nutrient. There is evidence to indicate that hexavalent chromium is reduced in part to trivalent chromium in vivo (Petrilli and DeFlora, 1977, 1978; Gruber and Jennette, 1978).

In 1965, a study of 155 subjects exposed to drinking water at concentrations of approximately 20 mg/L was conducted outside Jinzhou, China. Subjects were observed to have sores in the mouth, diarrhea, stomachache, indigestion, vomiting elevated white blood cell counts with respect to controls, and a higher per capit rate of cancers, including lung cancer and stomach cancer. Precise exposure concentrations, exposure durations, and confounding factors were not discussed, a this study does not provide a NOAEL for the observed effects. However, the study suggests that gastrointestinal effects may occur in humans following exposures to hexavalent chromium at levels of 20 ppm in drinking water (Zhang and Li, 1987).

Zahid et al. (1990) fed BALB/C albino Swiss mice trivalent (chromium disulfate) and hexavalent (potassium dichromate) chromium at concentrations of 10 200, and 400 ppm for 35 days in the diet. The author concluded that a small but significant increase of hexavalent chromium in the testes of fed animals induced significant degeneration. The National Toxicology Program (1996a,b, 1997) recent conducted a three-part study to investigate the potential reproductive toxicity o hexavalent chromium in rats and mice. The study included oral administration of potassium dichromate in Sprague-Dawley rats, a repeat of the study of Zahid et al. (1990) using BALB/C mice, and a reproductive assessment by continuous breedin study in BALB/C mice. The reproductive assessment indicated that potassium dichromate administered at 15-400 ppm in the diet is not a reproductive toxicant either sex of BALB/C mice or Sprague-Dawley rats.

Several reports of possible fetal damage caused by chromium compounds were located in the literature. High doses (250-1,000 ppm) of orally administered chromium (VI) compounds have been reported to cause developmental toxicity in mic (Trivedi et al., 1989). The authors observed significant increases in preimplantation and postimplantation losses and dose-dependent reductions in tota weight and crown-rump length in the lower dose groups. Additional effects included treatment-related increases in abnormalities in the tail and wrist forelimbs, and subdermal hemorrhagic patches in the offspring.

Junaid et al. (1996) and Kanojia et al. (1996) exposed female Swiss albino mice and female Swiss albino rats, respectively, to 250, 500, or 750 ppm potassiu dichromate in drinking water to determine the potential embryotoxicity of hexavalent chromium during days 6-14 of gestation. The authors reported retarded fetal development and embryo- and fetotoxic effects including reduced fetal weigh reduced number of fetuses (live and dead) per dam, and higher incidences of stillbirths and post-implantation loss in the 500 and 750 ppm dosed mothers. Significantly reduced ossification in bones was also observed in the medium- and high-dose groups. Based on the body weight and the drinking water ingested by th animals in the 250 ppm dose group, the exposure levels in the 250 ppm groups can identified as 67 mg/kg-day and 37 mg/kg-day in mice and rats, respectively.

The Junaid et al. (1996) and Kanojia et al. (1996) studies utilized doses approximately 10-fold higher than those used in Mackenzie et al (1958), but neith of the reproductive studies identified a clear NOAEL for the embryotoxic effects hexavalent chromium. On the basis of the body weight and the drinking water ingested by the animals in the low-dose groups (250 ppm), the LOAELs of 67 mg/kg-day and 37 mg/kg-day can be identified from Junaid et al. (1996) and Kanojia et al. (1996) in mice and rats, respectively. Application of 10-fold uncertainty factor to extrapolate from LOAELs to NOAELs in these studies would generate NOAELs of 6.7 mg/kg-day and 3.7 mg/kg-day, respectively. These extrapolated NOAEL values are similar to, and support the use of, the NOAEL of 2. mg/kg-day identified from the study of MacKenzie et al. (1958) for development of the reference dose.

Elbetieha and Al-Hamood (1997) reported impacts on fertility following potassium dichromate exposures in mice; however, many of the observed effects did not occur in a clear dose-dependent fashion. The authors did not indicate the amount of water ingested by the animals, and stated only that water ingestion was reduced in the treatment groups relative to the controls.

Chromium is one of the most common contact sensitizers in males in industrialized countries and is associated with occupational exposures to numerou materials and processes, including chrome plating baths, chrome colors and dyes, cement, tanning agents, wood preservatives, anticorrosive agents, welding fumes, lubricating oils and greases, cleaning materials, and textiles and furs (Burrows and Adams, 1990; Polak et al., 1973). Solubility and pH appear to be the primary determinants of the capacity of individual chromium compounds to elicit an allerg response (Fregert, 1981; Polak et al., 1973). The low solubility chromium (III) compounds are much less efficient contact allergens than chromium (VI) (Spruit an van Neer, 1966).

Dermal exposure to chromium has been demonstrated to produce irritant and allergic contact dermatitis (Bruynzeel et al., 1988; Polak, 1983; Cronin, 1980; Hunter, 1974). Primary irritant dermatitis is related to the direct cytotoxic properties of chromium, while allergic contact dermatitis is an inflammatory response mediated by the immune system. Allergic contact dermatitis is a cell-mediated immune response that occurs in a two-step process. In the first step (induction), chromium is absorbed into the skin and triggers an immune response (sensitization). Sensitized individuals will exhibit an allergic dermatitis response when exposed to chromium above a threshold level (Polak, 1983). Inducti is generally considered to be irreversible. Concentrations of hexavalent chromiu in environmental media that are protective of carcinogenic and noncarcinogenic effects are likely to be lower than the concentrations required to cause inductio of allergic contact dermatitis. However, these concentrations may not be lower than concentrations required to elicit an allergic response in individuals who ha been induced.

The RfD was updated in 1998. The RfD is similar to the previous value on IR but now incorporates a threefold uncertainty factor to account for the less-than-lifetime exposure in the principal study and a threefold modifying fact to account for uncertainties related to reports of gastrointestinal effects following drinking water exposures in a residential population in China.

___I.A.5. CONFIDENCE IN THE ORAL RfD

Study -- Low
Database -- Low
RfD -- Low

The overall confidence in this RfD assessment is low. Confidence in the chosen study is low because of the small number of animals tested, the small numb of parameters measured, and the lack of toxic effect at the highest dose tested.

Confidence in the database is low because the supporting studies are of equally low quality and the developmental toxicity endpoints are not well studied

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1998

This assessment was peer reviewed by external scientists. Their comments ha been evaluated carefully and incorporated in finalization of this IRIS summary. record of these comments is included as an appendix to the Toxicological Review o Hexavalent Chromium in support of Summary Information on the Integrated Risk Information System (IRIS) (U.S. EPA, 1998).

Other EPA Documentation --

U.S. EPA. (1984) Health effects assessment for hexavalent chromium. 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. (1985) Drinking water health advisory for chromium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Drinking Water, Washington, (Draft).

Agency consensus date -- 04/28/1998

I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning thi assessment or IRIS, in general, at (513) 569-7254 (phone), (513) 569-7159 (fax), RIH.IRIS@EPAMAIL.EPA.GOV (Internet address).

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE

(RfC)

Chromium(VI)
CASRN -- 18540-29-9
Last Revised -- 09/03/1998

The inhalation reference concentration (RfC) is analogous to the oral RfD an is likewise based on the assumption that thresholds exist for certain toxic effec such as cellular necrosis. The inhalation RfC considers toxic effects for both th respiratory system (portal-of-entry) and for effects peripheral to the respirator system (extrarespiratory effects). It is generally expressed in units of mg/m^3. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (U.S. EPA, 1989 and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (U.S. EPA, 1994). RfCs ca also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.B.1. INHALATION RfC SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfC
(1) Chromic acid mists and dissolved Cr (VI) aerosols:				
Nasal septum atrophy Human subchronic occupational study Lindberg and Hedenstierna, 1983	NOAEL: none LOAEL: 2E-3 mg/m^3 7.14 E-4 mg/m^3 (adj.)	90	1	8E-6 mg/m^3
(2) Cr(VI) particulates:				
Lactate dehydrogenase in bronchioalveolar lavage fluid Rat subchronic study Glaser et al., 1990 Malsch et al., 1994	BMD: 0.016 mg/m^3 0.034 mg/m^3 (adj.)	300	1	1E-4 mg/m^3

*Conversion Factors and Assumptions -- Breathing rate for 8-hour occupational exposure = 10 m^3; breathing rate for 24-hour continuous exposure = 20 m^3; occupational exposure = 5 days/week; continuous exposure = 7 days/week. RDDR

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(regional deposited dose ratio for particulates to account for differences betwee rats and humans) = 2.16

Nasal mucosal irritation, atrophy, and perforation have been widely reported following occupational exposures to chromic acid mists and dissolved hexavalent chromium aerosols. However, there is uncertainty regarding the relevance of occupational exposures to chromic acid mists and dissolved hexavalent chromium aerosols to exposures to Cr(VI) dusts in the environment. Lower respiratory effects have been reported in laboratory animals following exposures to Cr(VI) dusts. However, these studies have not reported on nasal mucosal effects following the exposures. The uncertainties in the database have been addressed through the development of two RfCs; one based on nasal mucosal atrophy following occupational exposures to chromic acid mists and dissolved hexavalent chromium aerosols, and a second based on lower respiratory effects following inhalation of Cr(VI) particulates in rats.

___I.B.2. PRINCIPAL AND SUPPORTING STUDIES (INHALATION RfC)

(1) Chromic acid mists and dissolved Cr (VI) aerosols

Three studies have focused on nasal mucosal irritation, atrophy, and perforation following occupational exposures to chromic acid mists (Cohen et al., 1974; Lucas and Kramkowski, 1975; Lindberg and Hedenstierna, 1983). Of these, th study of Lindberg and Hedenstierna provides the most information on exposure leve and symptoms reported by exposed workers. Respiratory symptoms, lung function, a changes in nasal septum were studied in 104 workers (85 males, 19 females) expose in chrome plating plants. Workers were interviewed using a standard questionnair for the assessment of nose, throat, and chest symptoms. Nasal inspections and pulmonary function testing were performed as part of the study.

The median exposure time for the entire group of exposed subjects (104) in t study was 4.5 years (0.1-36 years). A total of 43 subjects exposed almost exclusively to chromic acid experienced a mean exposure time of 2.5 years (0.2-23 years). The subjects exposed almost exclusively to chromic acid were divided int a low-exposure group (8-hr TWA below 0.002 mg/m^3, N=19) and a high-exposure group (8-hr TWA above 0.002 mg/m^3, N=24). Exposure measurements using personal air samplers were performed for 84 subjects in the study on 13 different days. Exposure for the remaining 20 workers was assumed to be similar to that measured for workers in the same area. Nineteen office employees were used as controls fo nose and throat symptoms. A group of 119 auto mechanics whose lung function had been evaluated by similar techniques was selected as controls for lung function measurements. Smoking habits of workers were evaluated as part of the study.

At mean exposures below 0.002 mg/m^3, 4/19 workers from the low-exposure group of subjective nasal symptoms. Atrophied nasal mucosa were reported in 4/19 subjects from this group and 11/19 had smeary and crusty septal mucosa, which was statistically higher than controls. No one exposed to levels below 0.001 mg/m^3 complained of subjective symptoms. At mean concentrations of 0.002 mg/m^3 or above approximately one-third of the subjects had reddened, smeary, or crusty nasal mucosa. Atrophy was seen in 8/24 workers, which was significantly different from controls. Eight subjects had ulcerations in the nasal mucosa and five had perforations of the nasal septum. Atrophied nasal mucosa was not observed in any of the 19 controls, but smeary and crusty septal mucosa occurred in 5/19 controls

Short-term effects on pulmonary function were evaluated by comparing results of tests taken on Monday and Thursday among exposed groups and controls. No significant changes were seen in the low-exposure group or the control group. Nonsmokers in the high-exposure group experienced significant differences in pulmonary function measurements from the controls, but the results were within normal limits.

The authors concluded that 8-hour mean exposures to chromic acid above 0.002 mg/m^3 may cause a transient decrease in lung function, and that short-term exposures to greater than 0.02 mg/m^3 may cause septal ulceration and perforation. Based on the results of this study, a LOAEL of 0.002 mg/m^3 can be identified for incidence of nasal septum atrophy following exposure to chromic acid mists in chromeplating facilities. At TWA exposures greater than 0.002 mg/m^3, nasal septum ulceration and perforations occurred in addition to the atrophy reported at lower concentrations. The LOAEL is based on an 8-hour TWA occupational exposure. The LOAEL is adjusted to account for continuous exposure according to the following equation:

$$LOAELc = 0.002\ mg/m^3 \times (MVho/MVh) \times 5\ days/7\ days$$

where:

LOAELc is the LOAEL for continuous exposure
MVho is the breathing volume for an 8 hour occupational exposure (10 m^3)
MVh is the breathing volume for a 24 hour continuous exposure (20 m^3)

The LOAEL of 0.002 mg/m^3 based on a TWA exposure to chromic acid is converted to a LOAEL for continuous exposure of 7.14 E-4 mg/m^3. An uncertainty factor of 3 is applied to the LOAEL to extrapolate from a subchronic to a chronic exposure, a uncertainty factor of 3 is applied to account for extrapolation from a LOAEL to a NOAEL, and an uncertainty factor of 10 is applied to the LOAEL to account for interhuman variation. The total uncertainty factor applied to the LOAEL is 90. Application of the uncertainty factor of 90 to the LOAEL of 7.14E-4 mg/m^3 generates an RfC of 8 E-6 mg/m^3 for upper respiratory effects caused by chromic acid mists and dissolved hexavalent chromium aerosols.

(2) Cr (VI) Particulates:

Two studies provide high-quality data on lower respiratory effects following exposures to chromium particulates (Glaser et al., 1985, 1990). Glaser et al. (1990) exposed 8-week-old male Wistar rats to sodium dichromate at 0.05 - 0.4 mg Cr(VI)/m^3 22 hr/day, 7 days/wk for 30-90 days. Chromium-induced effects occurred in a strong dose-dependent manner. The authors observed obstructive respiratory dyspnea and reduced body weight following subacute exposure at the higher dose levels. The mean white blood cell count was increased at all doses ($p < 0.05$) and was related to significant dose-dependent leukocytosis following subacute exposures. Mean lung weights were significantly increased at exposure levels of 0.1 mg/m^3 following both the subacute and subchronic exposures. Accumulation of macrophages was seen in all of the exposure groups and was postulated to be a chromium-specific irritation effect that accounted for the observed increases in lung weights. Focal inflammation was observed in the upper airways following the subchronic exposure, and albumin and lactate dehydrogenase (LDH) in bronchioalveolar lavage fluid (BALF) were increased following the exposure. The authors concluded that chromium inhalation induced pneumocyte toxicity and suggested that inflammation is essential for the induction of most chromium inhalation effects and may influence the carcinogenicity of Cr(VI) compounds.

Glaser et al. (1985) exposed 5-week-old male Wistar rats to aerosols of sodi dichromate at concentrations ranging from 0.025 to 0.2 mg Cr(VI)/m^3, 22 hr/day in subacute (28 day) or subchronic (90 day) protocols. Chromium-induced effects occurred in a dose-dependent manner. Lung and spleen weights were significantly increased ($p < 0.005$) after both subacute and subchronic exposures at concentrations greater than 0.025 mg/m^3. Differences in the mean total serum immunoglobulin were also significant at exposures above 0.025 mg/m^3, while exposures to aerosol concentrations greater than 0.1 mg/m^3 resulted in depression of the immune system stimulation. The immune stimulating effect of subchronic exposure was not reversed after 2 mo of fresh air regeneration. Bronchoalveolar

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lavage (BAL) cell counts were significantly decreased following subchronic exposu to levels above 0.025 mg/m^3 chromium. The number of lymphocytes and granulocytes showed a slight but significant increase in the lavage fluids of the subacute and subchronically exposed groups. At subacute exposure concentrations up to 0.05 mg/m^3 the phagocytic activity of the alveolar macrophages increased; however, subchronic exposure at 0.2 mg/m^3 decreased this function significantly. The splee T-lymphocyte subpopulation was stimulated by subchronic exposure to 0.2 mg/m^3 chromium, and serum contents of triglycerides and phospholipids differed significantly from controls ($p < 0.05$) at this concentration.

Together, these studies provide useful information on chromium exposure-related impacts including lung and spleen weight, LDH in BALF, protein in BALF, a albumin in BALF. The cellular content of BALF is considered representative of initial pulmonary injury and chronic lung inflammation, which may lead to the ons of pulmonary fibrosis (Henderson, 1988). While these studies present dose-dependent results on sensitive indicators of lower respiratory toxicity, potential upper respiratory impacts resulting from the exposures were not addressed. Glaser et al. (1990) state that the upper respiratory tract was examined, but these data were not reported.

One approach for development of an RfC using the data of Glaser et al. (1985 1990) was offered by Malsch et al. (1994), who generated an inhalation RfC for chromium dusts using a benchmark concentration (BMC) approach. The Agency developed its RfC for particulates based on this approach. After excluding exposures for periods of less than 90 days from the BMC analysis, Malsch et al. (1994) developed BMCs for lung weight, LDH in BALF, protein in BALF, albumin in BALF, and spleen weight. The Malsch et al. (1994) analysis defined the benchmark concentration as the 95% lower confidence limit on the dose corresponding to a 10 relative change in the endpoint compared to the control. Dose-effect data were adjusted to account for discontinuous exposure (22 hr/day) and the maximum likelihood model was used to fit continuous data to a polynomial mean response regression, yielding maximum likelihood estimates of 0.036 - 0.078 mg/m^3 and BMCs of 0.016 - 0.067 mg/m^3. Malsch et al. (1994) applied dosimetric adjustments and uncertainty factors to determine a RfC based on the following equation:

$$RfC = \frac{BMC \times RDDR}{UF_A \times UF_F \times UF_H}$$

where:

RfC	is the inhalation reference concentration
BMC	is the benchmark concentration (lower 95% confidence limit on the dose corresponding to a 10% relative change in the endpoint compa to the control)
RDDR	is the regional deposited dose ratio to account for pharmacokinet differences between species
UF_A	is a threefold uncertainty factor to account for pharmacodynamic differences not addressed by the RDDR
UF_F	is a threefold uncertainty factor to account for extrapolating fro subchronic to chronic exposures; and
UF_H	is a 10-fold uncertainty factor to account for the variation in sensitivity among members of the human population

The RDDR factor is incorporated to account for differences in the deposition pattern of inhaled hexavalent chromium dusts in the respiratory tract of humans a the Wistar rat test animals (Jarabek et al., 1990). The RDDR of 2.1576 was determined based on the mass median aerodynamic diameter (0.28 µm for dose levels of 0.05-0.1 mg/m^3 and 0.39 for dose levels of 0.1-0.4 mg/m^3) and the geometric standard deviation (1.63 for dose levels of 0.05-0.1 mg/m^3 and 1.72 for dose level of 0.1-0.4 mg/m^3) of the particulates reported in Glaser et al. (1990).
A 3.16-fold uncertainty factor (midpoint between 1 and 10 on a log scale) was incorporated to account for the pharmacodynamic differences not accounted for by

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the RDDR. An additional 3.16-fold uncertainty factor was incorporated to account for the less-than-lifetime exposure in Glaser et al. (1990), and a 10-fold uncertainty factor was applied to account for variation in the human population. total uncertainty factor of 100 was applied to the BMC in addition to the RDDR.

Glaser et al. (1990) reported that LDH in BALF increased in a dose-dependent fashion from 0.05 to 0.4 mg/m^3 sodium dichromate, and this endpoint generated the lowest BMC (0.016 mg/m^3) and RfC (3.4 E-4 mg/m^3). LDH in BALF is considered the among the most sensitive indicators of potential lung toxicity (Henderson, 1984, 1985, 1988; Beck et al., 1982; Venet et al., 1985), as LDH is found extracellular after cell damage and BALF is the closest site to the original lung injury. LDH BALF may also reflect chronic lung inflammation, which may lead to pulmonary fibrosis through prevention of the normal repair of lung tissue (Henderson, 1988)

Several uncertainties must be addressed with regard to the BMC and RfC developed by Malsch et al. (1994). Potentially important endpoints, including upper airway effects and potential renal or immunological toxicity, were not addressed in the Glaser et al. (1985, 1990) studies and could not be included in the BMC analysis. While LDH in BALF resulted in the lowest BMC and RfC, all of t effects noted in Glaser et al. (1985, 1990) can be considered indicative of an inflammatory response, and might be equally suited to development of the RfC. LD in BALF did not generate the best fit on the regression curve of the endpoints considered in the BMC analysis. In addition, the threefold uncertainty factor accounting for the use of a subchronic study may not be sufficiently protective f long-term effects. While the analysis acknowledged the importance of particle si and airway deposition in the development of the RDDR, the potential impact of different particle sizes in respiratory toxicity by hexavalent chromium particulates was not addressed.

Several of these uncertainties were conservatively addressed in the analysis of Malsch et al. (1994). LDH in BALF generated the lowest estimate of the BMC fr the effects noted by Glaser et al. (1985, 1990). This effect can be considered t be indicative of cell damage that occurs prior to fibrosis, as LDH appears in BAL following cell lysis. While the Malsch et al. (1994) analysis demonstrated a relatively poor curve fit for this endpoint, the model generated a conservative f in the data that is unlikely to overestimate the BMC. LDH in BALF as reported in Glaser et al. (1990) is considered to be an acceptable endpoint for development o an RfC for inhalation of hexavalent chromium particulates, and Malsch et al. (199 used a reasonable approach for development of a BMC based on this endpoint.

The threefold uncertainty factor used by Malsch et al. (1994) to account for the subchronic study is insufficient for development of the RfC for inhalation of chromium particulates. Glaser et al. (1985) demonstrated that at the end of the 90-day exposure period, chromium was still accumulating in the lung tissue of the test animals, suggesting that lower long-term exposures might lead to accumulatio of a critical concentration in the lung. Subchronic studies also may not adequately predict the presence of inflammatory effects from lower long-term exposures. The Agency has therefore determined that a 10-fold uncertainty factor accounting for the use of a subchronic study is more appropriate in this case for the development of an RfC for inhalation of chromium particulates.

Selection of a threefold uncertainty factor to account for the pharmacodynam differences not accounted for by the RDDR, an additional 10-fold uncertainty fact to account for the less-than-lifetime exposure in Glaser et al. (1990), and a 10-fold uncertainty factor to account for variation in the human population generates a total uncertainty factor of 300. Application of the total uncertaint factor of 300 and the RDDR of 2.1576 to the BMC generated by Malsch et al. (1994 based on LDH in BALF (Glaser et al., 1990) results in an RfC of 1 E-4 mg/m^3 for inhalation of hexavalent chromium particulates.

___I.B.3. UNCERTAINTY AND MODIFYING FACTORS (INHALATION RfC)

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See discussion above.

(1) Chromic acid mists and dissolved Cr (VI) aerosols:
UF = 90.
MF = 1.

(2) Chromium (VI) particulates:
UF = 300.
MF = 1

___I.B.4. ADDITIONAL STUDIES/COMMENTS (INHALATION RfC)

There is considerable uncertainty with regard to the relevance of the nasal septum atrophy endpoint observed in the chromeplating industry to exposure to hexavalent chromium in the environment. The effects were observed in chromeplate who were exposed to chromic acid mists near the plating baths. Environmental exposures would most likely occur through contact with hexavalent chromium dusts, and exposures to chromic acid mists in the environment is considered to be unlikely. An additional uncertainty is related to the determination of dose in t Lindberg and Hedenstierna study. Nasal septum atrophy in this study was related TWA exposures to chromic acid. The most significant effects (nasal septum perforation) were observed in workers who experienced peak excursions to levels considerably greater than the TWA. It is uncertain whether the peak excursion da or the TWAs are more appropriate for the determination of dose in this study. Th RfC based on the data of Lindberg and Hedenstierna (1983) should only be used to address exposures to chromic acid and dissolved hexavalent chromium aerosols.

Nasal mucosal irritation, atrophy, and perforation have been widely reported following occupational exposures to chromic acid mists and dissolved hexavalent chromium aerosols. Glaser et al. (1990) did not report on upper respiratory effects following exposure of rats to sodium dichromate. The RfC based on the da of Glaser et al. should only be used to address inhalation of Cr(VI) particulates

The RfCs in this IRIS Summary were added in 1998. The previous RfC section for hexavalent chromium in IRIS was empty.

___I.B.5. CONFIDENCE IN THE INHALATION RfC

(1) Chromic acid mists and dissolved Cr (VI) aerosols:

Study -- Low
Database -- Low
RfC -- Low

The overall confidence in this RfC assessment is low. Confidence in the chosen study is low because of uncertainties regarding the exposure characterization and the role of direct contact for the critical effect. Confiden in the database is low because the supporting studies are equally uncertain regarding the exposure characterization.

(2) Chromium (VI) particulates:

Study -- Medium
RfC -- Medium

The overall confidence in this RfC assessment is medium. Confidence in the chosen study is medium because of uncertainties regarding upper respiratory, reproductive, and renal effects resulting from the exposures.

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I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

Source Document -- U.S. EPA, 1998

This assessment was peer reviewed by external scientists. Their comments ha been evaluated carefully and incorporated in finalization of this IRIS summary. record of these comments is included as an appendix to the Toxicological Review o Hexavalent Chromium in support of Summary Information on the Integrated Risk Information System (IRIS) (U.S. EPA, 1998).

Agency Consensus Date -- 04/28/1998

I.B.7. EPA CONTACTS (INHALATION RfC)

Please contact the Risk Information Hotline for all questions concerning thi assessment or IRIS, in general, at (513) 569-7254 (phone), (513) 569-7159 (fax), RIH.IRIS@EPAMAIL.EPA.GOV (Internet address).

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name: Chromium(VI)
CASRN: 18540-29-9
Last Revised -- 09/03/1998

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates o risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk pe (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per µg/L drinking water or risk per µg/m3 air breathed. The third form in which risk is presented is a concentration of the chemical in drinking water or air associated with cancer risks of 1 in 10,000, 1 in 100,000, or 1 in 1,000,000. Th rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA' more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. EVIDENCE FOR HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CHARACTERIZATION

Under the current guidelines (EPA, 1986), Cr(VI) is classified as Group A - known human carcinogen by the inhalation route of exposure. Carcinogenicity by t oral route of exposure cannot be determined and is classified as Group D.

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Under the proposed guidelines (EPA, 1996), Cr(VI) would be characterized as known human carcinogen by the inhalation route of exposure on the following basis

Hexavalent chromium is known to be carcinogenic in humans by the inhalation route of exposure. Results of occupational epidemiologic studies of chromium-exposed workers are consistent across investigators and study population Dose-response relationships have been established for chromium exposure and lung cancer. Chromium-exposed workers are exposed to both Cr(III) and Cr(VI) compounds Because only Cr(VI) has been found to be carcinogenic in animal studies, however, it was concluded that only Cr(VI) should be classified as a human carcinogen.

Animal data are consistent with the human carcinogenicity data on hexavalent chromium. Hexavalent chromium compounds are carcinogenic in animal bioassays, producing the following tumor types: intramuscular injection site tumors in rats and mice, intrapleural implant site tumors for various Cr(VI) compounds in rats, intrabronchial implantation site tumors for various Cr(VI) compounds in rats, and subcutaneous injection site sarcomas in rats.

In vitro data are suggestive of a potential mode of action for hexavalent chromium carcinogenesis. Hexavalent chromium carcinogenesis may result from the formation of mutagenic oxidatitive DNA lesions following intracellular reduction the trivalent form. Cr(VI) readily passes through cell membranes and is rapidly reduced intracellularly to generate reactive Cr(V) and Cr(IV) intermediates and reactive oxygen species. A number of potentially mutagenic DNA lesions are forme during the reduction of Cr(VI). Hexavalent chromium is mutagenic in bacterial assays, yeasts, and V79 cells, and Cr(VI) compounds decrease the fidelity of DNA synthesis in vitro and produce unscheduled DNA synthesis as a consequence of DNA damage. Chromate has been shown to transform both primary cells and cell lines.

II.A.2. HUMAN CARCINOGENICITY DATA

Occupational exposure to chromium compounds has been studied in the chromate production, chromeplating and chrome pigment, ferrochromium production, gold mining, leather tanning, and chrome alloy production industries.

Workers in the chromate industry are exposed to both trivalent and hexavalen compounds of chromium. Epidemiological studies of chromate production plants in Japan, Great Britain, West Germany, and the United States have revealed a correlation between occupational exposure to chromium and lung cancer, but the specific form of chromium responsible for the induction of cancer was not identified (Machle and Gregorius, 1948; Baejter, 1950a,b; Bidstrup, 1951; Mancuso and Hueper, 1951; Brinton et al., 1952; Bidstrup and Case, 1956; Todd, 1962; Taylor, 1966; Enterline, 1974; Mancuso, 1975; Ohsaki et al., 1978; Sano and Mitohara, 1978; Hayes et al., 1979; Hill and Ferguson, 1979; Alderson et al., 198 Haguenor et al., 1981; Satoh et al., 1981; Korallus et al., 1982; Frentzel-Beyme, 1983; Langard and Vigander, 1983; Watanabe and Fukuchi, 1984; Davies, 1984; Mancuso, 1997).

Mancuso and Hueper (1951) conducted a proportional mortality study of a coho of chromate workers (employed for > 1 year from 1931-1949 in a Painesville, OH chromate plant) in order to investigate lung cancer associated with chromate production. Of the 2,931 deaths of males in the county where the plant is locate 34 (1.2%) were due to respiratory cancer. Of the 33 deaths among the chromate workers, however, 6 (18.2%) were due to respiratory cancer. Within the limitation of the study design, this report strongly suggested an increased incidence of respiratory cancer in the chromate-production plant.

In an update of the Mancuso and Hueper (1951) study, Mancuso (1975) followed 332 of the workers employed from 1931-1951 until 1974. By 1974, > 50% of this cohort had died. Of these men, 63.6%, 62.5%, and 58.3% of the cancer deaths for men employed from 1931-1932, 1933-1934, and 1935-1937, respectively, were due to

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lung cancer. Lung cancer death rates increased by gradient of exposure to total chromium, and significant deposition of chromium was found in the lungs of worker long after the exposure ceased. Mancuso (1975) reported that these lung cancer deaths were related to insoluble (trivalent), soluble (hexavalent), and total chromium exposure, but the small numbers involved make identification of the specific form of chromium responsible for the lung cancer uncertain.

Mancuso (1997) recently updated this study, following the combined cohort of 332 workers until 1993. Of 283 deaths (85% of the cohort identified), 66 lung cancers were found (23.3% of all deaths and 64.7% of all cancers). Lung cancer rates clearly increased by gradient level of exposure to total chromium. The relationship between gradient level of exposure and lung cancer rates is less cle for trivalent and hexavalent chromium. The rates of lung cancer within the cohor are consistent with those reported in Mancuso (1975), and provide further support for the cancer risk assessment based on those data.

Studies of chrome pigment workers in the United States (Hayes et al., 1989), England (Davies, 1984, 1979, 1978), Norway (Langard and Vigander, 1983; Langard a Norseth, 1975), and in the Netherlands and Germany (Frentzel-Beyme, 1983) have consistently demonstrated an association between occupational chromium exposure (predominantly to Cr [VI]) and lung cancer.

Several studies of the chromeplating industry have demonstrated a positive relationship between cancer and exposure to chromium compounds (Royle, 1975; Franchini et al., 1983; Sorahan et al., 1987).

___II.A.3. ANIMAL CARCINOGENICITY DATA

Animal data are consistent with the findings of human epidemiological studie of hexavalent chromium. Hexavalent chromium compounds were carcinogenic in anima assays producing the following tumor types: lung tumors following inhalation of aerosols of sodium chromate and pyrolized Cr(VI)/Cr(III) oxide mixtures in rats (Glaser et al., 1986), lung tumors following intratracheal administration of sodi dichromate in rats (Steinhoff et al., 1983), intramuscular injection site tumors Fischer 344 and Bethesda Black rats and in C57BL mice (Furst et al., 1976; Malton 1974, 1976; Payne, 1960a; Hueper and Payne, 1959); intrapleural implant site tumors for various Cr(VI) compounds in Sprague-Dawley and Bethesda Black rats (Payne, 1960b; Hueper 1961; Hueper and Payne, 1962), intrabronchial implantation site tumors for various Cr(VI) compounds in Wistar rats (Levy and Martin, 1983; Laskin et al., 1970; Levy, as quoted in NIOSH, 1975), and subcutaneous injection site sarcomas in Sprague-Dawley rats (Maltoni, 1974, 1976). Inflammation is considered to be essential for the induction of most chromium respiratory effects and may influence the carcinogenicity of Cr(VI) compounds (Glaser et al., 1985).

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Metabolism and genotoxicity. Hexavalent chromium is rapidly taken up by cel through the sulfate transport system (Sugiyama, 1992). Once inside the cell, Cr(VI) is quickly reduced to the trivalent form by cellular reductants, including ascorbic acid, glutathione and flavoenzymes (cytochrome P-450 and glutathione reductase), and riboflavin (De Flora et al., 1989; De Flora et al., 1990; Sugiyam 1992). The intracellular reduction of Cr(VI) generates reactive Cr(V) and Cr(IV) intermediates as well as hydroxyl free radicals (OH) and singlet oxygen ($^1O_2$) (Kawanishi et al., 1986). A variety of DNA lesions are formed during the reduction of Cr(VI) to Cr(III), including DNA strand breaks, alkali-labile sites, DNA-protein and DNA-DNA crosslinks, and oxidative DNA damage, such as 8-oxo-deoxyguanosine (Klein et al., 1992; Klein et al., 1991; De Flora et al., 1990). The relative importance of the different chromium complexes and oxidative DNA damage in the toxicity of Cr(VI) is unknown.

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A large number of chromium compounds have been assayed with in vitro genetic toxicology assays. In general, hexavalent chromium is mutagenic in bacterial assays whereas trivalent chromium is not (Lofroth, 1978; Petrilli and DeFlora, 1977, 1978). Likewise Cr(VI), but not Cr(III), was mutagenic in yeasts (Bonatti al., 1976) and in V79 cells (Newbold et al., 1979). Cr(III) and (VI) compounds decrease the fidelity of DNA synthesis in vitro (Loeb et al., 1977), while Cr(VI) compounds inhibit replicative DNA synthesis in mammalian cells (Levis et al., 197 and produce unscheduled DNA synthesis, presumably repair synthesis, as a consequence of DNA damage (Raffetto, 1977). Chromate has been shown to transform both primary cells and cell lines (Fradkin et al., 1975; Tsuda and Kato, 1977; Casto et al., 1979). Chromosomal effects produced by treatment with chromium compounds have been reported by a number of authors; for example, both Cr(VI) and Cr(III) salts were clastogenic for cultured human leukocytes (Nakamuro et al., 1978).

In dogs (2/group) exposed to potassium dichromate in drinking water at concentrations up to 11.2 ppm for 4 years, gross and microscopic examination of a major organs revealed no treatment-related lesions (Anwar et al., 1961). The sma number of animals and the relatively short exposure duration relative to the lifespan of the dog precludes a conclusion regarding a possible carcinogenic response. There are no other long-term studies of ingested Cr(VI). Cr(VI) is readily converted to Cr(III) in vivo, but there is no evidence that Cr(III) is oxidized to Cr(VI) in vivo. Cr(III) is an essential trace element.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

The oral carcinogenicity of Cr(VI) cannot be determined. No data were locat in the available literature that suggested that Cr(VI) is carcinogenic by the ora route of exposure.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

___II.C.1. SUMMARY OF RISK ESTIMATES

____II.C.1.1. Air Unit Risk -- 1.2E-2 ($\mu g/m^3$)

Source: Mancuso, 1975

____II.C.1.2. Extrapolation Method -- Multistage, extra risk

Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	8E-3 ($\mu g/m^3$)
E-5 (1 in 100,000)	8E-4 ($\mu g/m^3$)
E-6 (1 in 1,000,000)	8E-5 ($\mu g/m^3$)

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II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSURE

Tumor type -- lung cancer
Test animals -- human
Route -- inhalation, occupational exposure
Source -- Mancuso, 1975

Subject age (years)	Exposure level midrange (μg/m^3)	Deaths from lung cancer	Person-years
50	5.66	3	1,345
	25.27	6	931
	46.83	6	299
60	4.68	4	1,063
	20.79	5	712
	39.08	5	211
70	4.41	2	401
	21.29	4	345

II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE

Mancuso (1997) recently updated the study of Mancuso (1975), following the combined cohort of 332 workers until 1993. Of 283 deaths (85% of the cohort identified), 66 lung cancers were found (23.3% of all deaths and 64.7% of all cancers). Lung cancer rates clearly increased by gradient level of exposure to total chromium. The relationship between gradient level of exposure and lung cancer rates is less clear for trivalent and hexavalent chromium. The rates of lung cancer within the cohort are consistent with those reported in Mancuso (1975 and provide further support for the cancer risk assessment based on those data.

The cancer mortality in Mancuso (1975) was assumed to be due to Cr(VI), whic was further assumed to be no less than one-seventh of total chromium. It was als assumed that the smoking habits of chromate workers were similar to those of the U.S. white male population.

Trivalent chromium compounds have not been reported as carcinogenic by any route of administration.

The unit risk should not be used if the air concentration exceeds 8E-1 μg/m3 since above this concentration the unit risk may not be appropriate.

The carcinogenicity section of this IRIS Summary was updated in 1998; howeve the quantitative results have not been modified.

II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

Results of studies of chromium exposure are consistent across investigators and countries. A dose relationship for lung tumors has been established. The assumption that the ratio of Cr(III) to Cr(VI) is 6:1 may lead to a sevenfold underestimation of risk. The use of 1949 hygiene data (Bourne and Yee, 1950), which may underestimate worker exposure, may result in an overestimation of risk. Further overestimation of risk may be due to the implicit assumption that the smoking habits of chromate workers were similar to those of the general white mal population, since it is generally accepted that the proportion of smokers is high for industrial workers than for the general population.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1998

This assessment was peer reviewed by external scientists. Their comments ha been evaluated carefully and incorporated in finalization of this IRIS summary. record of these comments is included as an appendix to U.S. EPA, 1998.

Other EPA Documentation -- U.S. EPA. (1984) Health assessment document for chromium. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA/600/8-83-014F.

II.D.2. EPA REVIEW (CARCINOGENICITY ASSESSMENT)

Agency consensus date -- 04/28/1998

II.D.3. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning thi assessment or IRIS, in general, at (513) 569-7254 (phone), (513) 569-7159 (fax), RIH.IRIS@EPAMAIL.EPA.GOV (Internet address).

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VI. BIBLIOGRAPHY

Chromium(VI)
CASRN -- 18540-29-9
Last Revised -- 09/03/1998

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_VII. REVISION HISTORY

Chromium(VI)
CASRN --18540-29-9

Date	Section	Description
09/30/1987	II.A.1.	Citation corrected
03/01/1988	II.C.4.	Confidence statement revised
03/01/1988	II.D.3.	Contacts switched
03/01/1988	III.A.	Health Advisory added
12/01/1989	I.B.	Inhalation RfD now under review
06/01/1990	II.A.1.	Basis - Text revised
06/01/1990	II.B.	Text revised
06/01/1990	IV.A.1.	Area code for EPA contact corrected
06/01/1990	IV.F.1.	EPA contact changed
06/01/1990	VI.	Bibliography on-line
01/01/1991	II.	Text edited
01/01/1991	II.C.1.	Inhalation slope factor removed (global chan
03/01/1991	II.A.1.	Text revised
03/01/1991	II.A.4.	Text revised
03/01/1991	II.B.	Text revised
01/01/1992	I.A.7.	Secondary contact changed
01/01/1992	IV.	Regulatory actions updated
04/01/1992	IV.A.1.	CAA regulatory action withdrawn
06/01/1992	All	CASRN corrected from 7440-47-3 to 18540-29-9
09/01/1994	I.A.6.	Work group review date added
02/01/1995	I.A.	RfD noted as pending change: Work Grp Mtg on 08/03/1994
12/01/1996	I.A.7.	Secondary contact removed
09/03/1998	I,II,VI	Revised RfD, RfC, carcinogenicity assessment refs.

00088761

_VIII. SYNONYMS

Chromium(VI)
CASRN --18540-29-9
Last Revised -- 03/31/1987

18540-29-9
7440-47-3
Chromic ion
Chromium
Chromium, ion
Chromium (VI)
Chromium (VI) ion

Note: ***A TOXICOLOGICAL REVIEW*** is available for this chemical in Adobe* PDF format (76 Pages, 235 Kbytes)
Similar documents can be found in the List of Available IRIS Toxicological Reviews

Get Acrobat Reader * Some of the documents provided by EPA are Adobe Acrobat PDF (Portable Docume Format) files. They can be viewed, and printed, with the use of an Adobe Acrobat Read The Adobe Acrobat's Reader is available, free, for Unix, Macintosh, IBM DOS, and IBM Windows operating systems. Click this button to download the latest version of Adobe Acrobat.
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Last updated: **9 September, 1998**
URL: http://www.epa.gov/iris/subst/0144.htm

00088762

United States Environmental Protection Agency

IRIS

Integrated Risk Information System

Copper

CASRN 7440-50-8

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0368
Copper; CASRN 7440-50-8

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Copper

File On-Line 09/07/1988

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	08/01/1991

00088763

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Copper
CASRN -- 7440-50-8

Not available at this time.

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Copper
CASRN -- 7440-50-8

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Copper
CASRN -- 7440-50-8
Last Revised -- 08/01/1991

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for

00088764

Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classified

Basis -- There are no human data, inadequate animal data from assays of copper compounds, and equivocal mutagenicity data.

___II.A.2. HUMAN CARCINOGENICITY DATA

None.

___II.A.3. 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.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

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.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

Not available.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1987

The values in the 1987 Drinking Water Criteria Document for Copper have received peer and administrative review.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 09/15/1987

Verification Date -- 09/15/1987

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

VI. BIBLIOGRAPHY

Substance Name -- Copper
CASRN -- 7440-50-8
Last Revised -- 08/01/1991

VI.A. ORAL RfD REFERENCES

None

VI.B. INHALATION RfD REFERENCES

None

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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inorganic metal salts. Cancer Res. 39: 193-198.

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_VII. REVISION HISTORY

Substance Name -- Copper
CASRN -- 7440-50-8

Date	Section	Description
09/07/1988	II.	Carcinogen summary on-line

08/01/1991	II.A.4.	Law citation year corrected
08/01/1991	VI.	Bibliography on-line
01/01/1992	IV.	Regulatory Action section on-line

VIII. SYNONYMS

Substance Name -- Copper
CASRN -- 7440-50-8
Last Revised -- 09/07/1988

7440-50-8
Copper

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0368.htm

00088769

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Lead and compounds (inorganic)

CASRN 7439-92-1

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0277
Lead and compounds (inorganic); CASRN 7439-92-1

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Lead and compounds (inorganic)

File On-Line 03/01/1988

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	message	02/01/1991
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	11/01/1993

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Lead and compounds (inorganic)
CASRN -- 7439-92-1

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 (07/08/1985 and 07/22/1985) 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).

EPA Contacts:

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Lead and compounds (inorganic)
CASRN -- 7439-92-1

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

00088771

Substance Name -- Lead and compounds (inorganic)
CASRN -- 7439-92-1
Last Revised -- 11/01/1993

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

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- 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.

___II.A.2. 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.

00088772

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.

II.A.3. 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

00088773

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).

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

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).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

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.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

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.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 05/04/1988

Verification Date -- 05/04/1988

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Lead and compounds (inorganic)
CASRN -- 7439-92-1
Last Revised -- 11/01/1993

__VI.A. ORAL RfD REFERENCES

None

__VI.B. INHALATION RfD REFERENCES

None

00088775

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

Anderson, E.L., and CAG (Carcinogenic Assessment Group). 1983. Quantitative approaches in use to assess cancer risk. Risk Analysis. 3: 277-295.

Azar, A., H.J. Trochimowicz and M.E. Maxfield. 1973. Review of lead studies in animals carried out at Haskell Laboratory - Two year feeding study and response to hemorrhage study. In: Barth D., A. Berlin, R. Engel, P. Recht and J. Smeets, Ed. Environmental health aspects of lead: Proceedings International Symposium; October 1972; Amsterdam, The Netherlands. Commission of the European Communities, Luxemberg. p. 199-208.

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_VII. REVISION HISTORY

Substance Name -- Lead and compounds (inorganic)
CASRN -- 7439-92-1

Date	Section	Description
09/26/1988	II.	Carcinogen summary on-line
02/01/1989	IV.B.1.	Effect level corrected in discussion
06/01/1989	II.D.3.	Primary contact changed
06/01/1989	IV.A.1.	Reference corrected - changed number for part in CFR
12/01/1989	II.A.3.	Last paragraph - Correct Van Esch 1969 citation
12/01/1989	VI.	Bibliography on-line
07/01/1990	I.A.	Changed contact J. Cohen's office and telephone number
07/01/1990	IV.F.1.	EPA contact changed
02/01/1991	I.A.	Message revised to include new EPA document
02/01/1991	I.A.	EPA contacts changed
05/01/1991	II.A.	Text edited
01/01/1992	IV.	Regulatory actions updated
06/01/1992	IV.B.2.	MCL monitoring reqs. and BAT corrected
07/01/1993	II.D.3.	Secondary contact's phone number changed
07/01/1993	VI.C.	References alphabetized correctly
11/01/1993	II.D.1.	U.S. EPA 1987 replaced with 1989; rev. state. revised
11/01/1993	VI.C.	U.S. EPA 1987 deleted; U.S. EPA 1989 added

VIII. SYNONYMS

Substance Name -- Lead and compounds (inorganic)
CASRN -- 7439-92-1
Last Revised -- 03/01/1988

7439-92-1
Lead
Lead and compounds
plumbum

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0277.htm

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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Manganese

CASRN 7439-96-5

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0373
Manganese; CASRN 7439-96-5

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Manganese

File On-Line 09/26/1988

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	05/01/1996
Inhalation RfC Assessment (I.B.)	on-line	12/01/1993
Carcinogenicity Assessment (II.)	on-line	12/01/1996

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I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Manganese
CASRN -- 7439-96-5
Last Revised -- 05/01/1996

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

NOTE: This reference dose is for the total oral intake of manganese. As discussed in the Principal and Supporting Studies and Uncertainty and Modifying Factors Sections, it is recommended that a modifying factor of 3 be applied if this RfD is used for assessments involving nondietary exposures.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
CNS effects Human Chronic Ingestion Data NRC, 1989; Freeland-Graves et al., 1987; WHO, 1973;	NOAEL (food): 0.14 mg/kg-day LOAEL: None	1	1	1.4E-1 mg/kg-day

*Conversion Factors and Assumptions -- 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 several studies.

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Freeland-Graves, J.H., C.W. Bales and F. Behmardi. 1987. Manganese

requirements of humans. In: Nutritional Bioavailability of Manganese, C. Kies, ed. American Chemical Society, Washington, DC. p. 90-104.

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.

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.

Manganese is a ubiquitous element that is essential for normal physiologic functioning in all animal species. Several disease states in humans have been associated with both deficiencies and excess intakes of manganese. Thus any quantitative risk assessment for manganese must take into account aspects of both the essentiality and the toxicity of manganese. In humans, many data are available providing information about the range of essentiality for manganese. In addition, there are many reports of toxicity to humans exposed to manganese by inhalation; much less is known, however, about oral intakes resulting in toxicity. As discussed in the Additional Studies / Comments Section, rodents do not provide a good experimental model for manganese toxicity, and only one limited study in primates by the oral route of exposure is available. The following assessment, therefore, focuses more on what is known to be a safe oral intake of manganese for the general human population. Finally, it is important to emphasize that individual requirements for, as well as adverse reactions to, manganese may be highly variable. The reference dose is estimated to be an intake for the general population that is not associated with adverse health effects; this is not meant to imply that intakes above the reference dose are necessarily associated with toxicity. Some individuals may, in fact, consume a diet that contributes more than 10 mg Mn/day without any cause for concern.

The Food and Nutrition Board of the National Research Council (NRC, 1989) determined an "estimated safe and adequate daily dietary intake" (ESADDI) of manganese to be 2-5 mg/day for adults. The lower end of this range was based on a study by McLeod and Robinson (1972), who reported equilibrium or positive balances at intakes of 2.5 mg Mn/day or higher. The range of the ESADDI also includes an "extra margin of safety" from the level of 10 mg/day, which the NRC considered to be safe for an occasional intake.

While the NRC determined an ESADDI for manganese of 2-5 mg/day, some nutritionists feel that this level may be too low. Freeland-Graves et al. (1987) have suggested a range of 3.5-7 mg/day for adults based on a review of human studies. It is noted that dietary habits have evolved in recent years to include a larger proportion of meats and refined foods in conjunction with a lower intake of whole grains. The net result of such dietary changes includes a lower intake of manganese such that many individuals may have suboptimal manganese status. This is discussed in more detail in the Additional Studies / Comments Section.

The World Health Organization (WHO, 1973) 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.

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From this information taken together, EPA concludes that an appropriate reference dose for manganese is 10 mg/day (0.14 mg/kg-day). In applying the reference dose for manganese to a risk assessment, it is important that the assessor consider the ubiquitous nature of manganese, specifically that most individuals will be consuming about 2-5 mg Mn/day in their diet. This is particularly important when one is using the reference dose to determine acceptable concentrations of manganese in water and soils.

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).

The individuals examined in the Kondakis study also had exposure to manganese in their diet. This was originally estimated to be 10-15 mg/day because of the high intake of vegetables (Kondakis, 1990). This estimate was subsequently lowered to 5-6 mg/day (Kondakis, 1993). Because of the uncertainty in the amount of manganese in the diet and the amount of water consumed, it is impossible to estimate the total oral intake of manganese in this study. These limitations preclude the use of this study to determine a quantitative dose-response relationship for the toxicity of manganese in humans.

This study, nevertheless, raises significant concerns about possible adverse neurological effects at doses not far from the range of essentially. Because of this concern, it is recommended that a modifying factor of 3 be applied when assessing risk from manganese in drinking water or soil. This is discussed more fully in the Uncertainty and Modifying Factors Section.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

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UF -- The information used to determine the RfD for manganese 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 variation in the manganese content of the diet. The 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.

MF -- When assessing exposure to manganese from food, the modifying factor is 1; however, when assessing exposure to manganese from drinking water or soil, a modifying factor of 3 is recommended. As discussed more fully in the Additional Studies/Comments Section, there are four reasons for this recommendation. First, while the data suggest that there is no significant difference between absorption of manganese as a function of the form in which it is ingested (i.e., food versus water), there is some degree of increased uptake of manganese from water in fasted individuals. Second, the study by Kondakis et al. (1989) raises some concern for possible adverse health effects associated with a lifetime consumption of drinking water containing about 2 mg/L of manganese. Third, although toxicity has not been demonstrated, there is concern for infants fed formula that typically has a much higher concentration of manganese than does human milk. If powdered formula is made with drinking water, the manganese in the water would represent an additional source of intake. Finally, there is some evidence that neonates absorb more manganese from the gastrointestinal tract, that neonates are less able to excrete absorbed manganese, and that in the neonate the absorbed manganese more easily passes the blood-brain barrier. These findings may be related to the fact that manganese in formula is in a different ionic form and a different physical state than in human milk. These considerations concerning increased exposure in an important population group, in addition to the likelihood that any adverse neurological effects of manganese are likely to be irreversible and not manifested for many years after exposure, warrant caution until more definitive data are available.

___I.A.4. ADDITIONAL STUDIES / COMMENTS (ORAL RfD)

The biochemical role of manganese is to serve as an activator of several enzymes including hydrolases, kinases, decarboxylases and transferases. It is also required for the activity of three metalloenzymes: arginase, pyruvate carboxylase and mitochondrial superoxide dismutase. A review of the biochemical and nutritional roles of manganese in human health, as well as a list of disease states related to manganese deficiency or excess, is provided by Wedler (1994).

Because of the ubiquitous nature of manganese in foodstuffs, actual manganese deficiency has not been observed in the general population. There are, however, only two reports in the literature of experimentally induced manganese deficiency in humans. The first was a report by Doisy (1972), who inadvertently omitted manganese from a formulated diet. One of two subjects developed a slight reddening of the hair, a scaly transient dermatitis, marked hypocholesterolemia, and moderate weight loss. The diet was subsequently determined to contribute 0.34 mg Mn/day, a level that resulted in manganese deficiency. The second report was a metabolic balance study conducted by Friedman et al. (1987) in which seven male volunteers were fed a semipurified diet containing 0.11 mg Mn/day for 39 days. Transient dermatitis developed in five of the seven subjects by the 35th day, which the authors speculate was a result of decreased activity of glycosyltransferases or prolidase, manganese-requiring enzymes that are necessary for dermal maintenance. Hypocholesterolemia was also observed in this study, which the authors suggest was a result of the need for manganese at several steps in the cholesterol

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biosynthesis pathway.

While an outright manganese deficiency has not been observed in the general human population, suboptimal manganese status may be more of a concern. As reviewed by Freeland-Graves and Llanes (1994), several disease states have been associated with low levels of serum manganese. These include epilepsy, exocrine pancreatic insufficiency, multiple sclerosis, cataracts, and osteoporosis. In addition, several inborn errors of metabolism have been associated with poor manganese status (e.g., phenylketonuria, maple syrup urine disease). While a correlation has been shown for low levels of serum manganese and these disease states, a causal relationship has not been demonstrated, and this remains an area in which additional research is needed.

To better understand the consequences of manganese deficiency, several animal models have been studied. These have also been reviewed by Freeland-Graves and Llanes (1994). Experiments in several species have shown a deficiency in dietary manganese to result in disorders in lipid and carbohydrate metabolism, impaired growth and reproductive function, and ataxia and skeletal abnormalities in neonates.

While manganese is clearly an essential element, it has also been demonstrated to be the causative agent in a syndrome of neurologic and psychiatric disorders that has been described in manganese miners. Donaldson (1987) provides a summary of this documented toxicity of manganese to humans, which has been primarily limited to workers exposed by inhalation. In contrast to inhaled manganese, ingested manganese has rarely been associated with toxicity. A review of manganese toxicity in humans and experimental animals has been provided by Keen and Zidenberg-Cherr (1994).

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 observed 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. No information regarding dietary levels of manganese was available in this study.

A few 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

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to an oral intake, the difference between the direct intravenous exposure and the relatively low level of absorption of manganese from the GI 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.

A third case study involved an 8-year old girl with Alagille's syndrome (an autosomal dominant disorder manifested principally by neonatal cholestasis and intrahepatic bile duct paucity) and end-stage liver disease (Devenyi et al., 1994). The patient had a stable peripheral neuropathy and for 2 months manifested with episodic, dystonic posturing and cramping of her hands and arms. Whole blood manganese was elevated (27 ug/L; normal range: 4-14 ug/L) and cranial T1-weighted magnetic resonance imaging (MRI) revealed symmetric hyperintense globus pallidi and subthalamic nuclei. These were taken as indications of manganese neurotoxicity. Following liver transplantation, the patient's manganese levels returned to normal, neurological symptoms improved and MRI appeared normal. It appeared, then, that the progression of liver dysfunction had resulted in inadequate excretion of manganese into the bile, ultimately leading to neurotoxicity. With restoration of liver function, this was remedied. This case study suggests that for individuals with impaired liver function, intakes of manganese that would otherwise be safe may present a problem.

Although conclusive evidence is lacking, some investigators have also linked increased intakes of manganese with violent behavior. Gottschalk et al. (1991) found statistically significant 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).

Only 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. While it is clear that neurotoxicity resulting from excessive exposure to manganese is of primary concern, the exact mechanism is not clear. Histopathologically, the globus pallidus and substantia nigra appear to be most affected. Biochemically, deficiencies of striatal dopamine and norepinephrine appear to be fundamental. As reviewed by Aschner and Aschner (1991), multiple pathways that contribute to manganese-induced neurotoxicity are likely.

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) (Chandra and Shukla, 1981; Lai et al., 1981, 1982; Leung et al., 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

00088785

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.

EPA initiated an investigation of the literature to determine the relative bioavailability of manganese in food and water (Ruoff, 1995). The conclusions from this research were that under a wide variety of exposure scenarios in humans, the bioavailability of manganese ingested in water was essentially equal to the bioavailability of manganese in food. Total diet, rather than the actual medium of exposure, appears to be more of a determining factor for the uptake of manganese from the GI tract. Specifically, the relative bioavailability of manganese from food compared with that from drinking water was determined to be 0.7, and not statistically significantly different. When the data were reanalyzed to include only the ingestion of manganese in drinking water by fasted individuals, the relative bioavailability was 0.5, indicating roughly a 2-fold greater uptake of manganese from drinking water compared with uptake from food.

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. Various dietary factors as well as the form of manganese can have a significant bearing on the dose absorbed from the GI tract. Many constituents of a vegetarian diet (e.g., tannins, oxalates, phytates, fiber) have been found to inhibit manganese absorption presumably by forming insoluble complexes in the gut. In addition, high dietary levels of calcium or phosphorus have been reported to decrease manganese absorption. 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. The acetate and carbonate groups, however, 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 chemical species 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 GI tract and a decreased ability to excrete absorbed manganese. 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.

No reports of actual manganese toxicity or deficiency have been reported for infants. As with adults, however, the potential for effects resulting from excess manganese or suboptimal manganese appears to exist (reviewed by Lonnerdal, 1994). In particular, suboptimal manganese may be a problem for preterm infants given calcium supplementation, which is known to inhibit the absorption of manganese. Because manganese is required for adequate bone mineralization, it is suggested that insufficient absorption of manganese in preterm infants may contribute to poor bone growth. On the other hand, excess manganese may be a problem for infants with low iron status, as this is known to increase the absorption of manganese.

An additional concern for infants has been expressed because of the often high levels of manganese in infant formulas, particularly compared with breast milk. Also, manganese in human milk is in the trivalent form bound to lactoferrin, the major iron-binding protein. Lactoferrin receptors are located in the brush border membranes of epithelial cells throughout the length of the small intestine, thus allowing for regulation of the uptake of manganese. In infant formulas, however, because manganese is in the divalent state, absorption through the GI tract cannot be regulated by lactoferrin receptors. Collipp et al. (1983) found that hair manganese levels in newborn infants increased 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 much higher (50-300 ug/L). 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).

I.A.5. CONFIDENCE IN THE ORAL RfD

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

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. There is no single 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

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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, liver function, general nutritional status) can significantly influence an individual's manganese status. As discussed in the Additional Studies / Comments Section, there is significant variability in the absorption and elimination of manganese by humans. Confidence in the data base is medium and confidence in the dietary RfD for manganese is also medium.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- This assessment is not presented in any existing EPA documentation.

This summary has been peer reviewed by three external scientists and has also received internal EPA review. The review was completed on 07/06/1995. The comments of the reviewers have been carefully evaluated and considered in the revision and finalization of this IRIS summary. A record of these comments is included in the IRIS documentation files.

Other EPA Documentation -- U.S. EPA, 1984

Agency Work Group Review -- 05/17/1990, 06/21/1990, 06/24/1992, 09/22/1992, 03/31 12/14/1993, 05/12/1995

Verification Date -- 05/12/1995

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Manganese
CASRN -- 7439-96-5
Last Revised -- 12/01/1993

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is expressed in units of mg/cu.m. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without

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an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (EPA/600/8-88/066F August 1989) and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994). RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

___I.B.1. INHALATION RfC SUMMARY

Critical Effect	Exposures*	UF	MF	RfC
Impairment of neuro-behavioral function Occupational exposure to manganese dioxide Roels et al., 1992	NOAEL: None LOAEL: 0.15 mg/cu.m LOAEL(ADJ): 0.05 mg/cu.m LOAEL(HEC): 0.05 mg/cu.m	1000	1	5E-5 mg/cu.m
Impairment of neuro-behavioral function Occupational exposure to manganese oxides and salts Roels et al., 1987	NOAEL: None LOAEL: 0.97 mg/cu.m LOAEL(ADJ): 0.34 mg/cu.m LOAEL(HEC): 0.34 mg/cu.m			

*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.

___I.B.2. PRINCIPAL AND SUPPORTING STUDIES (INHALATION RfC)

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 (MnO2) 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 MnO2 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

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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;

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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-

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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.

___I.B.3. UNCERTAINTY AND MODIFYING FACTORS (INHALATION RfC)

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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MF -- None

I.B.4. ADDITIONAL STUDIES / COMMENTS (INHALATION RfC)

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

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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 MnO_2 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 $MnCl_2$ and manganese sesquioxide (Mn_2O_3) in healthy adults. Drown et al. (1986) found that following intratracheal instillation of $MnCl_2$ and Mn_3O_4 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 MnO_2 were taken up to a significantly greater degree in cerebral cortex than the more soluble forms of $MnCl_2$ and manganese acetate [$Mn(CH_3COO)_2$]; however, the corpus striatal binding characteristics of the +4 valence state of Mn in MnO_2 were not substantially different from those of the divalent forms in $MnCl_2$, $Mn(CH_3COO)_2$, 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 (Mn_2O_3 and Mn_3O_4) 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

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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 MnO_2-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 (MnO_2 vs. mixed Mn oxides and salts) to which the workers in the two studies were exposed make it difficult to compare these

00088798

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 MnO_2, 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.

___I.B.5. CONFIDENCE IN THE INHALATION RfC

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.

___I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

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

Other EPA Documentation -- U.S. EPA, 1984

00088801

Agency Work Group Review -- 08/23/1990, 09/19/1990, 09/23/1993

Verification Date -- 09/23/1993

___I.B.7. EPA CONTACTS (INHALATION RfC)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Manganese
CASRN -- 7439-96-5
Last Revised -- 12/01/1996

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

NOTE: Manganese is an element considered essential to human health.

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Existing studies are inadequate to assess the carcinogenicity of manganese.

II.A.2. HUMAN CARCINOGENICITY DATA

None.

II.A.3. 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.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

None.

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

Not available.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1984, 1988

The Drinking Water Criteria Document for Manganese has received OHEA review.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

00088804

Agency Work Group Review -- 05/25/1988

Verification Date -- 05/25/1988

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

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CASRN -- 7439-96-5
Last Revised -- 11/01/1995

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VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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_VII. REVISION HISTORY

Substance Name -- Manganese
CASRN -- 7439-96-5

Date	Section	Description
09/26/1988	II.	Carcinogen summary on-line
09/01/1989	VI.	Bibliography on-line
06/01/1990	I.A.	Oral RfD now under review
08/01/1990	I.A.	Oral RfD summary on-line
08/01/1990	II.	Text edited
08/01/1990	VI.	Oral RfD references added
09/01/1990	I.B.	Inhalation RfC now under review
12/06/1990	I.B.	Inhalation RfC on-line
12/06/1990	VI.B.	Inhalation RfC references added
01/01/1992	IV.	Regulatory Action section on-line
06/01/1992	VI.B.	Iregren, 1990 and Nishiyama et al., 1975 pages corrected
08/01/1992	I.A.	Oral RfD noted as pending change
08/01/1992	I.A.6.	Work group review date added
10/01/1992	I.A.	Oral RfD withdrawn; new summary in preparation
10/01/1992	I.A.6.	Work group review date added
10/01/1992	VI.A.	Oral RfD references withdrawn
01/01/1993	I.A.	Oral RfD replaced (RfD changed)
01/01/1993	VI.A.	Oral RfD references replaced
05/01/1993	I.A.	Work group review date added
07/01/1993	II.A.3.	'Inadequate' added to 1st paragraph
07/01/1993	II.D.1.	EPA Documentation clarified
11/01/1993	I.B.	Inhalation RfC noted as pending changed
11/01/1993	I.B.6.	Work group review date added
12/01/1993	I.B.	Inhalation RfC replaced; RfC changed
12/01/1993	VI.B.	Inhalation RfC references replaced
01/01/1994	I.A.	Oral RfD noted as pending change
01/01/1994	I.A.6.	Work group review date added
03/01/1994	II.D.3.	Primary contact changed
04/01/1994	I.A.2.	Text revised

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04/01/1994	I.A.3.	Text revised
04/01/1994	I.A.4.	Text revised
04/01/1994	I.A.5.	Text revised
04/01/1994	VI.A.	Oral RfD references revised
06/01/1995	I.A.	Oral RfD noted as pending change
06/01/1995	I.A.6.	Work group review date added
11/01/1995	I.A.	Oral RfD assessment replaced
11/01/1995	VI.A.	Oral RfD references replaced
05/01/1996	I.A.7.	Secondary contact's phone number changed
12/01/1996	II.D.3.	Primary contact removed

VIII. SYNONYMS

Substance Name -- Manganese
CASRN -- 7439-96-5
Last Revised -- 09/26/1988

7439-96-5
COLLOIDAL MANGANESE
MAGNACAT
MANGAN
Manganese
MANGAN NITRIDOVANY
TRONAMANG

IRIS Home Page | Substance File List

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0373.htm

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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Mercury, elemental

CASRN 7439-97-6

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0370
Mercury, elemental; CASRN 7439-97-6

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Mercury, elemental

File On-Line 09/07/1988

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	on-line	06/01/1995
Carcinogenicity Assessment (II.)	on-line	05/01/1995

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I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Mercury, elemental
CASRN -- 7439-97-6

Not available at this time.

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Mercury, elemental
CASRN -- 7439-97-6
Last Revised -- 06/01/1995

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is expressed in units of mg/cu.m. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs were derived according to the Interim Methods for Development of Inhalation Reference Doses (EPA/600/8-88/066F August 1989) and subsequently, according to Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry (EPA/600/8-90/066F October 1994). RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.B.1. INHALATION RfC SUMMARY

Critical Effect	Exposures*	UF	MF	RfC
Hand tremor; increases in memory disturbances; slight subjective and objective evidence of	NOAEL: None LOAEL: 0.025 mg/cu.m (converted to LOAEL [ADJ]	30	1	3E-4 mg/cu.m

autonomic dysfunction	of 0.009 mg/cu.m	
Human occupational inhalation studies	Fawer et al., 1983; Piikivi and Tolonen, 1989; Piikivi and Hanninen, 1989; Piikivi, 1989; Ngim et al., 1992; Liang et al., 1993	

*Conversion Factors and Assumptions: This is an extrarespiratory effect of a vapor (gas). 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) = 0.025 mg/cu.m x MVho/MVh x 5 days/7 days = 0.009 mg/cu.m. Air concentrations (TWA) were measured in the Fawer et al. (1983), Ngim et al. (1992), and Liang et al. (1993) studies. Air concentrations were extrapolated from blood levels based on the conversion factor of Roels et al. (1987) as described in the Additional Comments section for the studies of Piikivi and Tolonen (1989), Piikivi and Hanninen (1989), and Piikivi (1989).

I.B.2. PRINCIPAL AND SUPPORTING STUDIES (INHALATION RfC)

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Ngim, C.H., S.C. Foo, K.W. Boey and J. Jeyaratnam. 1992. Chronic neurobehavioral effects of elemental mercury in dentists. Br. J. Ind. Med. 49: 782-790.

Liang, Y-X., R-K. Sun, Y. Sun, Z-Q. Chen and L-H. Li. 1993. Psychological effects of low exposure to mercury vapor: Application of a computer-administered neurobehavioral evaluation system. Environ. Res. 60: 320-327.

Fawer et al. (1983) used a sensitive objective electronic measure of intention tremor (tremors that occur at the initiation of voluntary movements) in 26 male workers (mean age of 44 years) exposed to low levels of mercury vapor in various occupations: fluorescent tube manufacture (n=7), chloralkali plants (n=12), and acetaldehyde production (n=7). Controls (n=25; mean age of 44.6 years) came from the same factories but were not exposed occupationally. Personal air samples (two per subject) were used to characterize an average exposure concentration of 0.026 mg/cu.m. It should be noted that it is likely that the levels of mercury in the air varied during the period of exposure and historical data indicate that previous exposures may have been higher. Exposure measurements for the control cohort were not performed. The average duration of exposure was 15.3 years. The measures of tremor were significantly increased in the exposed compared to control cohorts, and were shown to correspond to exposure and not to chronologic age. These findings

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are consistent with neurophysiological impairments that might result from accumulation of mercury in the cerebellum and basal ganglia. Thus, the TWA of 0.026 mg/cu.m was designated a LOAEL. Using the TWA and adjusting for occupational ventilation rates and workweek, the resultant LOAEL(HEC) is 0.009 mg/cu.m.

Piikivi and Tolonen (1989) used EEGs to study the effects of long-term exposure to mercury vapor in 41 chloralkali workers exposed for a mean of 15.6 +/- 8.9 years as compared with matched referent controls. They found that the exposed workers, who had mean blood Hg levels of 12 ug/L and mean urine Hg levels of 20 ug/L, tended to have an increased number of EEG abnormalities when analyzed by visual inspection only. When the EEGs were analyzed by computer, however, the exposed workers were found to have significantly slower and attenuated brain activity as compared with the referents. These changes were observed in 15% of the exposed workers. The frequency of these changes correlated with cortical Hg content (measured in other studies); the changes were most prominent in the occipital cortex less prominent in the parietal cortex, and almost absent in the frontal cortex. The authors extrapolated an exposure level associated with these EEG changes of 0.025 mg/cu.m from blood levels based on the conversion factor calculated by Roels et al. (1987).

Piikivi and Hanninen (1989) studied the subjective symptoms and psychological performances on a computer-administered test battery in 60 chloralkali workers exposed to mercury vapor for a mean of 13.7 +/- 5.5 years as compared with matched referent controls. The exposed workers had mean blood Hg levels of 10 ug/L and mean urine Hg levels of 17 ug/L. A statistically significant increase in subjective measures of memory disturbance and sleep disorders was found in the exposed workers. The exposed workers also reported more anger, fatigue and confusion. No objective disturbances in perceptual motor, memory or learning abilities were found in the exposed workers. The authors extrapolated an exposure level associated with these subjective measures of memory disturbance of 0.025 mg/cu.m from blood levels based on the conversion factor calculated by Roels et al. (1987).

Both subjective and objective symptoms of autonomic dysfunction were investigated in 41 chloralkali workers exposed to mercury vapor for a mean of 15.6 +/- 8.9 years as compared with matched referent controls (Piikivi, 1989). The quantitative non-invasive test battery consisted of measurements of pulse rate variation in normal and deep breathing, in the Valsalva maneuver and in vertical tilt, as well as blood pressure responses during standing and isometric work. The exposed workers had mean blood levels of 11.6 ug/L and mean urine levels of 19.3 ug/L. The exposed workers complained of more subjective symptoms of autonomic dysfunction than the controls, but the only statistically significant difference was an increased reporting of palpitations in the exposed workers. The quantitative tests revealed a slight decrease in pulse rate variations, indicative of autonomic reflex dysfunction, in the exposed workers. The authors extrapolated an exposure level associated with these subjective and objective measures of autonomic dysfunction of 0.030 mg/cu.m from blood levels based on the conversion factor calculated by Roels et al. (1987).

Two more recent studies in other working populations corroborate the neurobehavioral toxicity of low-level mercury exposures observed in the Fawer et al. (1983), Piikivi and Tolonen (1989), Piikivi and Hanninen (1989), and Piikivi (1989) studies.

Ngim et al. (1992) assessed neurobehavioral performance in a cross-sectional study of 98 dentists (38 female, 60 male; mean age 32, range 24-49 years) exposed to TWA concentrations of 0.014 mg/cu.m (range 0.0007 to 0.042 mg/cu.m) versus 54 controls (27 female, 27 male; mean age 34, range 23-50 years) with no history of occupational exposure to mercury. Air concentrations were measured with personal sampling badges over typical working hours (8-10 hours) and converted to an 8-hour TWA. No details on the number of exposure samples or exposure histories were provided. Blood samples

from the exposed cohort were also taken and the data supported the correspondence calculated by Roels et al. (1987). Based on extrapolation of the average blood mercury concentration (9.8 ug/L), the average exposure concentration would be estimated at 0.023 mg/cu.m. The average duration of practice of the exposed dentists was 5.5 years. Exposure measurements of the control cohort were not performed. The exposed and control groups were adequately matched for age, amount of fish consumption, and number of amalgam dental fillings. The performance of the dentists was significantly worse than controls on a number of neurobehavioural tests measuring motor speed (finger tapping), visual scaning, visumotor coordination and concentration, visual memmory, and visuomotor coordination speed. These neurobehavioral effects are consistent with central and peripheral neurotoxicity and the TWA is considered a LOAEL. Using the TWA and adjusting for occupational ventilation rates and the reported 6-day workweek, the resultant LOAEL(HEC) is 0.006 mg/cu.m.

Liang et al. (1993) investigated workers in a fluorescent lamp factory with a computer-adminstered neurobehavioral evaluation system and a mood inventory profile. The exposed cohort (mean age 34.2 years) consisted of 19 females and 69 males exposed to ninterruptedly for at least 2·years prior to the study. Exposure was monitored with area samplers and ranged from 0.008 to 0.085 mg/cu.m across worksites. No details on how the exposure profiles to account for time spent in different worksites were constructed. The average exposure was estimated at 0.033 mg/cu.m. (range 0.005 to 0.19 mg/cu.m). The average duration was of working was 15.8 years for the exposed cohort. Urinary excretion was also monitored and reported to average 0.025 mg/L. The control cohort (mean age 35.1 years) consisted of 24 females and 46 males recruited from an embroidery factory. The controls were matched for age, education, smoking and drinking habits. Exposure measurements for the control cohort were not performed. The exposed cohort performed significantly worse than the control on tests of finger tapping, mental arithmetic, two-digit searches, switiching attention, and visual reaction time. The effect on performance persisted after the confounding factor of chronological age was controlled. Based on these neurobehavioral effects, the TWA of 0.033 mg/cu.m is designated as LOAEL. Using the TWA and adjusting for occupational ventilation rates and workweek, the resultant LOAEL(HEC) is 0.012 mg/cu.m.

The above studies were taken together as evidence for a LOAEL based on neurobehavioral effects of low-level mercury exposures. The LOAEL(HEC) levels calculated on measured air concentration levels of the Ngim et al. (1992) and the Liang et al. (1993) studies bracket that calculated based on the air concentrations measured by Fawer et al. (1983) as a median HEC level. Extrapolations of blood levels, used as biological monitoring that accounts for variability in exposure levels, also converge at 0.025 mg/cu.m as a TWA which results in the same HEC level. Thus, the TWA level of 0.025 mg/cu.m was used to represent the exposure for the synthesis of the studies described above. Using this TWA and taking occupational ventilation rates and workweek into account results in a LOAEL(HEC) of 0.009 mg/cu.m.

___I.B.3. UNCERTAINTY AND MODIFYING FACTORS (INHALATION RfC)

UF -- An uncertainty factor of 10 was used for the protection of sensitive human subpopulations (including concern for acrodynia - see Additional Comments section) together with the use of a LOAEL. An uncertainty factor of 3 was used for lack of data base, particularly developmental and reproductive studies.

MF -- None

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I.B.4. ADDITIONAL COMMENTS (INHALATION RfC)

Probably the most widely recognized form of hypersensitivity to mercury poisoning is the uncommon syndrome known as acrodynia, also called erythredema polyneuropathy or pink disease (Warkany and Hubbard, 1953). Infantile acrodynia was first described in 1828, but adult cases have also since been reported. While acrodynia has generally been associated with short-term exposures and with urine levels of 50 ug/L or more, there are some cases in the literature in which mercury exposure was known to have occurred, but no significant (above background) levels in urine were reported. There could be many reasons for this, but the most likely is that urine levels are not a simple measure of body burden or of target tissue (i.e., brain levels); however, they are the best means available for assessing the extent of exposure. It was felt that the RfC level estimated for mercury vapor based on neurotoxicity of chronic exposure in workers is adequate to protect children from risk of acrodynia because such exposures of long duration would be expected to raise urine levels by only 0.12 ug/L against a background level of up to 20 ug/L (i.e., such exposures would not add significantly to the background level of mercury in those exposed).

Roels et al. (1987) investigated the relationships between the concentrations of metallic mercury in air and levels monitored in blood or urine in workers exposed during manufacturing of dry alkaline batteries. Breathing zone personal samples were used to characterize airborne mercury vapors. Total mercury in blood and urine samples were analyzed using atomic absorption. The investigation controlled for several key factors including the use of reliable personal air monitoring, quality control for blood and urine analyses, standardization of the urinary mercury concentration for creatinine concentration, and stability of exposure conditions (examined subjects were exposed to mercury vapor for at least 1 year). Strong correlations were found between the daily intensity of exposure to mercury vapor and the end of workshift levels in blood ($r=0.86$; $n=40$) or urine ($r=0.81$; $n=34$). These relationships indicated a conversion factor of 1:4.5 (air:blood) and 1:1.22 (air:urine as ug/g creatinine). These factors were used to extrapolate blood or urine levels associated with effects in the reported studies to airborne mercury levels.

Sensory and motor nerve conduction velocities were studied in 18 workers from a mercury cell chlorine plant (Levine et al., 1982). Time-integrated urine Hg levels were used as an indicator of mercury exposure. Using linearized regression analysis, the authors found that motor and sensory nerve conduction velocity changes (i.e., prolonged distal latencies correlated with the time-integrated urinary Hg levels in asymptomatic exposed workers) occurred when urinary Hg levels exceeded 25 ug/L. This study demonstrates that mercury exposure can be associated with preclinical evidence of peripheral neurotoxicity.

Singer et al. (1987) studied nerve conduction velocity of the median motor, median sensor and sural nerves in 16 workers exposed to various inorganic mercury compounds (e.g., mercuric oxides, mercurial chlorides, and phenyl mercuric acid) for an average of 7.3 +/- 7.1 years as compared with an unexposed control group using t-tests. They found a slowing of nerve conduction velocity in motor, but not sensory, nerves that correlated with increased blood and urine Hg levels and an increased number of neurologic symptoms. The mean mercury levels in the exposed workers were 1.4 and 10 ug/L for blood and urine, respectively. These urine levels are 2-fold less than those associated with peripheral neurotoxicity in other studies (e.g., Levine et al., 1982). There was considerable variability in the data presented by Singer et al. (1987), however, and the statistical analyses (t-test) were not as rigorous as those employed by Levine et al. (1982) (linearized regression analysis). Furthermore, the subjects in the Levine et al. (1982) study were asymptomatic at higher urinary levels than those reported to be associated

with subjective neurological complaints in the workers studied by Singer et al. (1987). Therefore, these results are not considered to be as reliable as those reported by Levine et al. (1982).

Miller et al. (1975) investigated several subclinical parameters of neurological dysfunction in 142 workers exposed to inorganic mercury in either the chloralkali industry or a factory for the manufacture of magnetic materials. They reported a significant increase in average forearm tremor frequency in workers whose urinary Hg concentrations exceeded 50 ug/L as compared with unexposed controls. Also observed were eyelid fasciculation, hyperactive deep-tendon reflexes and dermatographia, but there was no correlation between the incidence of these findings and urinary Hg levels.

Roels et al. (1985) examined 131 male and 54 female workers occupationally exposed to mercury vapor for an average duration of 4.8 years. Urinary mercury (52 and 37 ug/g creatinine for males and females, respectively) and blood mercury levels (14 and 9 ug/L for males and females, respectively) were recorded, but atmospheric mercury concentration was not provided. Symptoms indicative of CNS disorders were reported but not related to mercury exposure. Minor renal tubular effects were detected in mercury-exposed males and females and attributed to current exposure intensity rather (urinary Hg >50 ug/g creatinine) than exposure duration. Male subjects with urinary mercury levels of >50 ug/g creatinine exhibited preclinical signs of hand tremor. It was noted that females did not exhibit this effect and that their urinary mercury never reached the level of 50 ug/g creatinine. A companion study (Roels et al., 1987) related air mercury (Hg-air)levels to blood mercury (Hg-blood) and urinary mercury (Hg-U) values in 10 workers in a chloralkali battery plant. Duration of exposure was not specified. A high correlation was reported for Hg-air and Hg-U for preshift exposure ($r=0.70$, $p<0.001$) and post-shift ($r=0.81$, $p<0.001$) measurements. Based on these data and the results of their earlier (1985) study, the investigators suggested that some mercury-induced effects may occur when Hg-U levels exceed 50 ug/g creatinine, and that this value corresponds to a mercury TWA of about 40 ug/cu.m.

A survey of 567 workers at 21 chloralkali plants was conducted to ascertain the effects of mercury vapor inhalation (Smith et al., 1970). Mercury levels ranged from <0.01 to 0.27 mg/cu.m and chlorine concentrations ranged from 0.1 to 0.3 ppm at most of the working stations of these plants. Worker exposure to mercury levels (TWA) varied, with 10.2% of the workers being exposed to <0.01 mg/cu.m, 48.7% exposed to 0.01 to 0.05 mg/cu.m, 25.6% exposed to 0.06 to 0.10 mg/cu.m and 4.8% exposed to 0.24 to 0.27 mg/cu.m (approximately 85% were exposed to Hg levels less than or equal to 0.1 mg/cu.m). The duration of employment for the examined workers ranged from one year (13.3%) to >10 years (31%), with 55.7% of the workers being employed for 2 or 9 years. A group of 600 workers not exposed to chlorine served as a control group for assessment of chlorine effects, and a group of 382 workers not exposed to either chlorine or mercury vapor served as the reference control group. A strong positive correlation ($p<0.001$) was found between the mercury TWAs and the reporting of subjective neuropsychiatric symptoms (nervousness, insomnia), occurrence of objective tremors, and weight and appetite loss. A positive correlation ($p<0.001$) was also found between mercury exposure levels and urinary and blood mercury levels of test subjects. No adverse alterations in cardiorespiratory, gastrointestinal, renal or hepatic functions were attributed to the mercury vapor exposure. Additionally, biochemical (hematologic data, enzyme activities) and clinical measurements (EKG, chest X-rays) were no different between the mercury-exposed and non-exposed workers. No significant signs or symptoms were noted for individuals exposed to mercury vapor concentrations less than or equal to 0.1 mg/cu.m. This study provides data indicative of a NOAEL of 0.1 mg Hg/cu.m and a LOAEL of 0.18 mg Hg/cu.m. In a followup study conducted by Bunn et al. (1986), however, no significant differences in the frequency of objective or subjective findings such as weight loss and appetite loss were observed in workers exposed to mercury at levels that ranged between 50 and 100 ug/L. The study by Bunn et al. (1986) was limited, however, by the lack of information

provided regarding several methodological questions such as quality assurance measures and control of possible confounding variables.

The mercury levels reported to be associated with preclinical and symptomatic neurological dysfunction are generally lower than those found to affect kidney function, as discussed below.

Piikivi and Ruokonen (1989) found no evidence of glomerular or tubular damage in 60 chloralkali workers exposed to mercury vapor for an average of 13.7 +/- 5.5 years as compared with their matched referent controls. Renal function was assessed by measuring urinary albumin and N-acetyl-beta-glucosaminidase (NAG) activity. The mean blood Hg level in the exposed workers was 14 ug/L and the mean urinary level was 17 ug/L. The authors extrapolated the NOAEL for kidney effects based on these results of 0.025 mg/cu.m from blood levels using the conversion factor calculated by Roels et al. (1987).

Stewart et al. (1977) studied urinary protein excretion in 21 laboratory workers exposed to 10-50 ug/cu.m of mercury. Their urinary level of mercury was about 35 ug/L. Increased proteinuria was found in the exposed workers as compared with unexposed controls. When preventive measure were instituted to limit exposure to mercury, proteinuria was no longer observed in the exposed technicians.

Lauwerys et al. (1983) found no change in several indices of renal function (e.g., proteinuria, albuminuria, urinary excretion of retinol-binding protein, aminoaciduria, creatinine in serum, beta-2-microglobulin in serum) in 62 workers exposed to mercury vapor for an average of 5.5 years. The mean urinary Hg excretion in the exposed workers was 56 ug/g creatinine, which corresponds to an exposure level of about 46 ug/cu.m according to a conversion factor of 1:1.22 (air:urine [ug/g creatinine]) (Roels et al., 1987). Despite the lack of observed renal effects, 8 workers were found to have an increased in serum anti-laminin antibodies, which can be indicative of immunological effects. In a followup study conducted by Bernard et al. (1987), however, there was no evidence of increased serum anti-laminin antibodies in 58 workers exposed to mercury vapor for an average of 7.9 years. These workers had a mean urinary Hg excretion of 72 ug/g creatinine, which corresponds to an exposure levels of about 0.059 mg/cu.m.

Stonard et al. (1983) studied renal function in 100 chloralkali workers exposed to inorganic mercury vapor for an average of 8 years. No changes in the following urinary parameters of renal function were observed at mean urinary Hg excretion rates of 67 ug/g creatinine: total protein, albumin, alpha-1-acid glycoprotein, beta-2-microglobulin, NAG, and gamma-glutamyl transferase. When urinary Hg excretion exceeded 100 ug/g creatinine, a small increase in the prevalence of higher activities of NAG and gamma-glutamyl transferase was observed.

The mercury levels reported to be associated with preclinical and symptomatic neurological dysfunction and kidney effects are lower than those found to pulmonary function, as discussed below.

McFarland and Reigel (1978) described the cases of 6 workers who were acutely exposed (4-8 hours) to calculated metallic mercury vapor levels of 1.1 to 44 mg/cu.m. These men exhibited a combination of chest pains, dyspnea, cough, hemoptysis, impairment of pulmonary function (reduced vital capacity), diffuse pulmonary infiltrates and evidence of interstitial pneumonitis. Although the respiratory symptoms resolved, all six cases exhibited chronic neurological dysfunction, presumably as a result of the acute, high-level exposure to mercury vapor.

Lilis et al. (1985) described the case of a 31-year-old male who was acutely exposed to high levels of mercury vapor in a gold-extracting facility. Upon admission to the hospital, the patient exhibited dyspnea, chest pain with

deep inspiration, irregular infiltrates in the lungs and reduced pulmonary function (forced vital capacity [FVC]). The level of mercury to which he was exposed is not known, but a 24-hour urine collection contained 1900 ug Hg/L. Although the patient improved gradually over the next several days, 11 months after exposure he still showed signs of pulmonary function abnormalities (e.g., restriction and diffusion impairment).

Levin et al. (1988) described four cases of acute high-level mercury exposure during gold ore purification. The respiratory symptoms observed in these four cases ranged from minimal shortness of breath and cough to severe hypoxemia. The most severely affected patient exhibited mild interstitial lung disease both radiographically and on pulmonary function testing. One patient had a urinary Hg level of 245 ug/L upon hospital admission. The occurrence of long-term respiratory effects in these patients could not be evaluated since all but one refused follow-up treatment.

Ashe et al. (1953) reported that there was no histopathological evidence of respiratory damage in 24 rats exposed to 0.1 mg Hg/cu.m 7 hr/day, 5 days/week for 72 weeks. This is equivalent to a NOAEL[HEC] of 0.07 mg/cu.m.

Kishi et al. (1978) observed no histopathological changes in the lungs of rats exposed to 3 mg/cu.m of mercury vapor 3 hours/day, 5 days/week for 12-42 weeks.

Beliles et al. (1967) observed no histopathological changes in the lungs of pigeons exposed to 0.1 mg/cu.m of mercury vapor 6 hours/day, 5 days/week for 20 weeks.

Neurological signs and symptoms (i.e., tremors) were observed in 79 workers exposed to metallic mercury vapor whose urinary mercury levels exceeded 500 ug/L. Short-term memory deficits were reported in workers whose urine levels were less than 500 ug/L (Langolf et al., 1978).

Impaired performance in mechanical and visual memory tasks and psychomotor ability tests was reported by Forzi et al. (1978) in exposed workers whose urinary Hg levels exceeded 100 ug/L.

Decreased strength, decreased coordination, increased tremor, decreased sensation and increased prevalence of Babinski and snout reflexes were exhibited by 247 exposed workers whose urinary Hg levels exceeded 600 ug/L. Evidence of clinical neuropathy was observed at urinary Hg levels that exceeded 850 ug/L (Albers et al., 1988).

Preclinical psychomotor dysfunction was reported to occur at a higher incidence in 43 exposed workers (mean exposure duration of 5 years) whose mean urinary excretion of Hg was 50 ug/L. Workers in the same study whose mean urinary Hg excretion was 71 ug/L had a higher incidence of total proteinuria and albuminuria (Roels et al., 1982).

Postural and intention tremor was observed in 54 exposed workers (mean exposure duration of 7.7 years) whose mean urinary excretion of Hg was 63 ug/L (Roels et al., 1989).

Verbeck et al. (1986) observed an increase in tremor parameters with increasing urinary excretion of mercury in 21 workers exposed to mercury vapor for 0.5-19 years. The LOAEL for this effect was a mean urinary excretion of 35 ug/g creatinine.

Rosenman et al. (1986) evaluated routine clinical parameters (physical exams, blood chemistry, urinalysis), neuropsychological disorders, urinary NAG, motor nerve conduction velocities and occurrence of lenticular opacities in 42 workers of a chemical plant producing mercury compounds. A positive correlation ($p<0.05$ to $p<0.001$) was noted between urinary mercury (levels ranged from 100-250 ug/L) and the number of neuropsychological symptoms, and

NAG excretions and the decrease in motor nerve conduction velocities.

Evidence of renal dysfunction (e.g., increased plasma and urinary concentrations of beta-galactosidase, increased urinary excretion of high-molecular weight proteins and a slightly increased plasma beta-2-microglobulin concentration) was observed in 63 chloralkali workers. The incidence of these effects increased in workers whose urinary Hg excretion exceeded 50 ug/g creatinine (Buchet et al., 1980).

Increased urinary NAG levels were found in workers whose urinary Hg levels exceeded 50 ug/L (Langworth et al., 1992).

An increase in the concentration of urinary brush border proteins (BB-50) was observed in 20 workers whose mean urinary Hg excretion exceeded 50 ug/g creatinine (Mutti et al., 1985).

Foa et al. (1976) found that 15 out of 81 chloralkali workers exposed to 60-300 ug/cu.m mercury exhibited proteinuria.

An increased excretion of beta-glutamyl transpeptidase, indicative of renal dysfunction, was found in 509 infants dermally exposed to phenylmercury via contaminated diapers (Gotelli et al., 1985).

Berlin et al. (1969) exposed rats, rabbits and monkeys to 1 mg/cu.m of mercury vapor for 4 hours and measured the uptake and distribution of mercury in the brain as compared with animals injected intravenously with the same doses of mercury as mercuric salts. Mercury accumulated in the brain following inhalation exposure to metallic mercury vapor at levels that were 10 times higher than those observed following intravenous injection of the same dose of mercury as mercuric salts. These results demonstrate that mercury is taken up by the brain following inhalation of the vapor at higher levels than other forms of mercury and that this occurs in all species studied.

Limited animal studies concerning inhalation exposure to inorganic mercury are available. The results of a study conducted by Baranski and Szymczyk (1973) were reported in an English abstract. Adult female rats were exposed to metallic mercury vapor at 2.5 mg/cu.m for 3 weeks prior to fertilization and during gestation days 7-20. A decrease in the number of living fetuses was observed in the dams compared with unexposed controls, and all pups born to the exposed dams died by the sixth day after birth. However, no difference in the occurrence of developmental abnormalities was observed between exposed and control groups. The cause of death of the pups in the mercury-exposed group was unknown, although an unspecified percentage of the deaths was attributed by the authors to a failure of lactation in the dams. Death of pups was also observed in another experiment where dams were only exposed prior to fertilization (to 2.5 mg/cu.m), which supports the conclusion that the high mortality in the first experiment was due at least in part to poor health of the mothers. Without further information, this study must be considered inconclusive regarding developmental effects.

The only other study addressing the developmental toxicology of mercury is the one reported in abstract form by Steffek et al. (1987) and, as such, is included as a supporting study. Sprague-Dawley rats (number not specified) were exposed by inhalation to mercury vapor at concentrations of 0.1, 0.5 or 1.0 mg/cu.m throughout the period of gestation (days 1-20) or during the period of organogenesis (days 10-15). The authors indicated the exposure protocols to be chronic and acute exposure, respectively. At either exposure protocol, the lowest mercury level produced no detectable adverse effect. At 0.5 mg/cu.m, an increase in the number of resorptions (5/41) was noted for the acute group, and two of 115 fetuses exhibited gross cranial defects in the chronic group. At 1.0 mg/cu.m, the number of resorptions was increased in acute (7/71) and chronic (19/38) groups and a decrease in maternal and fetal weights also was detected in the chronic exposure group. No statistical analysis for these data was provided. A LOAEL of 0.5 mg/cu.m is provided

based on these data.

Mishinova et al. (1980) investigated the course of pregnancy and parturition in 349 women exposed via inhalation to metallic mercury vapors (unspecified concentrations) in the workplace as compared to 215 unexposed women. The authors concluded that the rates of pregnancy and labor complication were high among women exposed to mercury and that the effects depended on "the length of service and concentration of mercury vapors." Lack of sufficient details preclude the evaluation of dose-response relationships.

In a questionnaire that assessed the fertility of male workers exposed to mercury vapor, Lauwerys et al. (1985) found no statistically significant change in the observed number of children born to the exposed group compared with a matched control group. The urinary excretion of mercury in the exposed workers ranged from 5.1 to 272.1 ug/g creatinine.

Another study found that exposure to metallic mercury vapor caused prolongation of estrus cycles in animals. Baranski and Szymczyk (1973) reported that female rats exposed via inhalation to mercury vapor at an average of 2.5 mg/cu.m, 6 hours/day, 5 days/week for 21 days experienced longer estrus cycles than unexposed animals. In addition, estrus cycles during mercury exposure were longer than normal estrus cycles in the same animals prior to exposure. Although the initial phase of the cycle was protracted, complete inhibition of the cycle did not occur. During the second and third weeks of exposure, these rats developed signs of mercury poisoning including restlessness, seizures and trembling of the entire body. The authors speculated that the effects on the estrus cycle were caused by the action of mercury on the CNS (i.e., damage to the hypothalamic regions involved in the control of estrus cycling).

Renal toxicity has been reported following oral exposure to inorganic mercury salts in animals, with the Brown-Norway rat appearing to be uniquely sensitive to this effect. These mercury-induced renal effects in the Brown-Norway rat are the basis for the oral RfD for mercurial mercury. Several investigators have produced autoimmune glomerulonephritis by administering HgCl2 to Brown-Norway rats (Druet et al., 1978).

The current OSHA standard for mercury vapor is 0.05 mg/cu.m. NIOSH recommends a TWA Threshold Limit Value of 0.05 mg/cu.m for mercury vapor.

___I.B.5. CONFIDENCE IN THE INHALATION RfC

Study -- Medium
Data Base -- Medium
RfC -- Medium

Due to the use of a sufficient number of human subjects, the inclusion of appropriate control groups, the exposure duration, the significance level of the reported results and the fact that exposure levels in a number of the studies had to be extrapolated from blood mercury levels, confidence in the key studies is medium. The LOAEL values derived from these studies can be corroborated by other human epidemiologic studies. The adverse effects reported in these studies are in accord with the well-documented effects of mercury poisoning. The lack of human or multispecies reproductive/ developmental studies precludes assigning a high confidence rating to the data base and inadequate quantification of exposure levels. Based on these considerations, the RfC for mercury is assigned a confidence rating of medium.

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I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

Source Document -- U.S. EPA, 1995

This IRIS summary is included in The Mercury Study Report to Congress which was reviewed by OHEA and EPA's Mercury Work Group in November 1994. An interagency review by scientists from other federal agencies took place in January 1995. The report was also reviewed by a panel of non-federal external scientists in January 1995 who met in a public meeting on January 25-26. All reviewers comments have been carefully evaluated and considered in the revision and finalization of this IRIS summary. A record of these comments is summarized in the IRIS documentation files.

Other EPA Documentation -- None

Agency Work Group Review -- 11/16/1989, 03/22/1990, 04/19/1990

Verification Date -- 04/19/1990

I.B.7. EPA CONTACTS (INHALATION RfC)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Mercury, elemental
CASRN -- 7439-97-6
Last Revised -- 05/01/1995

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

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II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Mercury, elemental
CASRN -- 7439-97-6
Preparation Date -- 5/24/94

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on inadequate human and animal data. Epidemiologic studies failed to show a correlation between exposure to elemental mercury vapor and carcinogenicity; the findings in these studies were confounded by possible or known concurrent exposures to other chemicals, including human carcinogens, as well as lifestyle factors (e.g., smoking). Findings from genotoxicity tests are severely limited and provide equivocal evidence that mercury adversely affects the number or structure of chromosomes in human somatic cells.

II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. A number of epidemiological studies were conducted that examined mortality among elemental mercury vapor-exposed workers. Conflicting data regarding a correlation between mercury exposure and an increased incidence of cancer mortalities have been obtained. All of the studies have limitations that complicate interpretation of their results for associations between mercury exposure and induction of cancer; increased cancer rates were attributable to other concurrent exposures or lifestyle factors.

A retrospective cohort study examined mortality among 5663 white males who worked between 1953 and 1963 at a plant in Oak Ridge, Tennessee, where elemental mercury was used for lithium isotope separation (Cragle et al., 1984). The workers were divided into three cohorts: exposed workers who had been monitored on a quarterly basis for mercury levels in urine (n=2,133); workers exposed in the mercury process section for whom urinalysis monitoring data were not collected (n=270); and unexposed workers from other sections of the nuclear weapons production facility (n=3260). The study subjects worked at least 4 months during 1953-1958 (a period when mercury exposures were likely to be high); mortality data from death certificates were followed through the end of 1978. The mean age of the men at first employment at the facility was 33 years, and the average length of their employment was >16 years with a mean of 3.73 years of estimated mercury exposure. Air mercury levels were monitored beginning in 1955; during 1955 through the third quarter of 1956, air mercury levels were reportedly above 100 ug/cu.m in 30-80% of the samples. Thereafter, air mercury levels decreased to concentrations below 100 ug/cu.m. The mortality experience (i.e., the SMR) of each group was compared with the age-adjusted mortality experience of the U.S. white male population. Among exposed and monitored workers, no significant increases in mortality from cancer at any site were reported, even after the level or length of exposure was considered. A significantly lower mortality from all causes was

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observed. An excessive number of deaths was reportedly due to lung cancer in the exposed and monitored workers (42 observed, 31.36 expected), but also in the unexposed workers (71 observed, 52.93 expected). The SMR for each group was 1.34; the elevated incidence of lung cancer deaths was, therefore, attributed to some other factor at the plant and/or to lifestyle factors (e.g., smoking) common to both the exposed and unexposed groups. Study limitations include small cohort sizes for cancer mortality, which limited the statistical stability of many comparisons.

Barregard et al. (1990) studied mortality and cancer morbidity between 1958 and 1984 in 1190 workers from eight Swedish chloralkali plants that used the mercury cell process in the production of chlorine. The men included in the study had been monitored for urinary or blood mercury for more than one year between 1946 and 1984. Vital status and cause of death were ascertained from the National Population Register and the National Bureau of Statistics. The cancer incidence of the cohort was obtained from the Swedish Cancer Register. The observed total mortality and cancer incidences were compared with those of the general Swedish male population. Comparisons were not made between exposed and unexposed workers. Mean urinary mercury levels indicated a decrease in exposure between the 1950s and 1970s; the mean urinary mercury level was 200 ug/L during the 1950s, 150 ug/L during the 1960s and 50 ug/L in the 1970s. Mortality from all causes was not significantly increased in exposed workers. A significant increase in deaths from lung tumors was observed in exposed workers 10 years or more after first exposure (rate ratio, 2.0; 95% CI, 1.0-3.8). Nine of the 10 observed cases of lung cancer occurred among workers (457 of the 1190) possibly exposed to asbestos as well as to mercury. No dose response was observed with respect to mercury exposure and lung tumors. This study is limited because no quantitation was provided on smoking status, and results were confounded by exposure to asbestos.

Ahlbom et al. (1986) examined the cancer mortality during 1961-1979 of cohorts of Swedish dentists and dental nurses aged 20-64 and employed in 1960 (3454 male dentists, 1125 female dentists, 4662 female dental nurses). Observed incidences were compared with those expected based on cancer incidence during 1961-1979 among all Swedes employed during 1960 and the proportion of all Swedes employed as dentists and dental nurses. Data were stratified by sex, age (5-year age groups) and county. The incidence of glioblastomas among the dentists and dental nurses combined was significantly increased compared to survival rates (SMR, 2.1; 95% CI, 1.3-3.4); the individual groups had apparently elevated SMRs (2.0-2.5), but the 95% confidence intervals of these groups included unity. By contrast, physicians and nurses had SMRs of only 1.3 and 1.2, respectively. Exposure to mercury could not be established as the causative factor because exposure to other chemicals and X-rays was not ruled out.

Amandus and Costello (1991) examined the association between silicosis and lung cancer mortality between 1959 and 1975 in 9912 white male metal miners employed in the United States between 1959 and 1961. Mercury exposures were not monitored. Exposures to specific metals among the silicotic and nonsilicotic groups were analyzed separately. Lung cancer mortality in both silicotic and nonsilicotic groups was compared with rates in white males in the U.S. population. Both silicotic (n=11) and nonsilicotic mercury miners (n=263) had significantly increased lung cancer mortality (SMR, 14.03; 95% CI, 2.89-40.99 for silicotics. SMR, 2.66; 95% CI, 1.15-5.24 for nonsilicotics). The analysis did not focus on mercury miners, and confounders such as smoking and radon exposure were not analyzed with respect to mercury exposure. This study is also limited by the small sample size for non-silicotic mercury miners.

A case-control study of persons admitted to a hospital in Florence, Italy, with lung cancer between 1981-1983 was performed to evaluate occupational risk factors (Buiatti et al., 1985). Cases were matched with one or two controls (persons admitted to the hospital with diagnoses other than lung cancer or suicide) with respect to sex, age, date of admission and smoking status.

Women who had "ever worked" as hat makers had a significantly increased risk of lung cancer. The duration of employment as a hat maker averaged 22.2 years, and latency averaged 47.8 years. Workers in the Italian hat industry were known to be occupationally exposed to mercury; however, the design of this study did not allow evaluation of the relationship between cumulative exposure and cancer incidence. In addition, interpretation of the results of this study is limited by the small sample size (only 6/376 cases reported this occupation) and by exposure of hat makers to other pollutants including arsenic, a known lung carcinogen.

Ellingsen et al. (1992) examined the total mortality and cancer incidence among 799 workers employed for more than 1 year in two Norwegian chloralkali plants. Mortality incidence between 1953 and 1988 and cancer incidence between 1953 and 1989 were examined. Mortality and cancer incidence were compared with that of the age-adjusted general male Norwegian population. No increase in total cancer incidence was reported, but lung cancer was significantly elevated in the workers (rate ratio, 1.66; 95% CI, 1.0-2.6). No causal relationship can be drawn from the study between mercury exposure and lung cancer because no correlation existed between cumulative mercury dose, years of employment or latency time. Also, the prevalence of smoking was 10 20% higher in the exposed workers, and many workers were also exposed to asbestos.

___II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. Druckrey et al. (1957) administered 0.1 mL of metallic mercury to 39 male and female rats (BD III and BD IV strains) via intraperitoneal injection. Among the rats surviving longer than 22 months, 5/12 developed peritoneal sarcomas. The increase in the incidence of sarcomas was observed only in those tissues that had been in direct contact with the mercury. Although severe kidney damage was reported in all treated animals, no renal tumors or tumors at any site other than the peritoneal cavity were observed.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Cytogenetic monitoring studies of workers occupationally exposed to mercury by inhalation provide very limited evidence that mercury adversely affects the number or structure of chromosomes in human somatic cells. Popescu et al. (1979) compared four men exposed to elemental mercury vapor with an unexposed group and found a statistically significant increase in the incidence of chromosome aberrations in the WBCs from whole blood. Verschaeve et al. (1976) found an increase in aneuploidy after exposure to low concentrations of vapor, but results could not be repeated in later studies (Verschaeve et al., 1979). Mabille et al. (1984) did not find increases in structural chromosomal aberrations of lymphocytes of exposed workers. Similarly, Barregard et al. (1991) found no increase in the incidence or size of micronuclei and no correlation between micronuclei and blood or urinary mercury levels of chloralkali workers. A statistically significant correlation was observed between cumulative exposure to mercury and micronuclei induction in T lymphocytes in exposed workers, suggesting a genotoxic effect.

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II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

None.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

None.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

II.D.1. EPA DOCUMENTATION

Source document -- U.S. EPA, 1995

This IRIS summary is included in The Mercury Study Report to Congress which was reviewed by OHEA and EPA's Mercury Work Group in November 1994. An interagency review by scientists from other federal agencies took place in January 1995. The report was also reviewed by a panel of non-federal external scientists in January 1995 who met in a public meeting on January 25-26. All reviewers comments have been carefully evaluated and considered in the revision and finalization of this IRIS summary. A record of these comments is summarized in the IRIS documentation files.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 01/13/1988, 03/03/1994

Verification Date -- 03/03/1994

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

00088830

_VI. BIBLIOGRAPHY

Substance Name -- Mercury, elemental
CASRN -- 7439-97-6
Last Revised -- 06/01/1995

__VI.A. ORAL RfD REFERENCES

None

__VI.B. INHALATION RfC REFERENCES

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_VII. REVISION HISTORY

Substance Name -- Mercury, elemental
CASRN -- 7439-97-6

Date	Section	Description
09/07/1988	II.	Carcinogen summary on-line
09/01/1989	VI.	Bibliography on-line
12/01/1989	I.B.	Inhalation RfD now under review
05/01/1991	II.A.3.	Text edited
01/01/1992	IV.	Regulatory Action section on-line
04/01/1994	II.	Carcinogenicity assessment noted as pending change
04/01/1994	II.D.2.	Work group review date added
05/01/1995	All	Name changed from mercury (inorganic)
05/01/1995	II.	Carcinogen assessment replaced
05/01/1995	VI.C.	Carcinogen assessment references replaced
06/01/1995	I.B.	Inhalation RfC summary on-line
06/01/1995	VI.B.	Inhalation RfC references on-line

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VIII. SYNONYMS

Substance Name -- Mercury, elemental
CASRN -- 7439-97-6
Last Revised -- 05/01/1995

7439-97-6
hydragyrum
Mercury
Mercury, elemental
Mercury, inorganic
Mercury, metallic
Mercury (organo) alkyl compounds
Caswell No. 546
COLLOIDAL MERCURY
EPA Pesticide Chemical Code 052301
KWIK [Dutch]
Liquid Silver
Mercure [French]
Mercurio [Italian]
Mercurio [Spanish]
Mercury compounds
Mercury vapor
NCI-C60399
Quecksilber [German]
Quicksilver

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0370.htm

00088836

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Nickel

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0271
Nickel, soluble salts; CASRN Various

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Nickel, soluble salts

File On-Line 09/30/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	12/01/1996
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	message	08/01/1994

00088837

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Nickel, soluble salts
CASRN -- Various
Last Revised -- 12/01/1996

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. 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				

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

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

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.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

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.

The Ni dietary study by Ambrose et al. (1976) identifying a NOAEL of 100 ppm (5 mg/kg/day) is supported by the subchronic gavage study in water (ABC, 1986), which indicated the same NOAEL (5 mg/kg/day).

MF -- None

I.A.4. ADDITIONAL STUDIES / COMMENTS (ORAL RfD)

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.

00088842

___I.A.5. CONFIDENCE IN THE ORAL RfD

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.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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

Agency Work Group Review -- 04/16/1987, 05/20/1987, 07/16/1987, 05/17/1990, 08/14

Verification Date -- 07/16/1987

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Nickel, soluble salts
CASRN -- Various

Not available at this time.

00088843

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Nickel, soluble salts
CASRN -- Various

The U.S. EPA has not evaluated soluble salts of nickel, as a class of compounds, for potential human carcinogenicity. However, nickel refinery dust and specific nickel compounds - nickel carbonyl and nickel subsulfide - have been evaluated. Summaries of these evaluations are on IRIS.

_VI. BIBLIOGRAPHY

Substance Name -- Nickel, soluble salts
CASRN -- Various
Last Revised -- 01/01/1992

__VI.A. ORAL RfD REFERENCES

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VI.B. INHALATION RfD REFERENCES

None

00088845

VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

None

VII. REVISION HISTORY

Substance Name -- Nickel, soluble salts
CASRN -- Various

Date	Section	Description
03/01/1988	I.A.2.	Text clarified paragraph 1
12/01/1989	I.B.	Inhalation RfD now under review
04/01/1990	I.A.	ABC, 1986 corrected to U.S. EPA, 1986
04/01/1990	VI.	Bibliography on-line
06/01/1990	I.A.	Oral RfD summary noted as pending change
06/01/1990	IV.F.1.	EPA contact changed
09/01/1991	I.A.	Oral RfD no longer pending change; no changes made
12/01/1991	I.A.3.	Text revised; additional study added
12/01/1991	I.A.4.	Text significantly revised; additional studies added
12/01/1991	VI.A.	Oral RfD references revised to reflect new text
01/01/1992	I.A.4.	Citation year corrected for Schnegg and Kirchgessner
01/01/1992	IV.	Regulatory actions updated
01/01/1992	VI.A.	Citation year corrected for Schnegg and Kirchgessner
03/01/1994	II.	Carcinogenicity assessment now under review
08/01/1994	II.	Message added
08/01/1994	II.	Work group review date added
05/01/1995	All	CAS Registry Number changed to Various
12/01/1996	I.A.7.	Primary contact removed

VIII. SYNONYMS

Substance Name -- Nickel, soluble salts
CASRN -- Various
Last Revised -- 09/30/1987

7440-02-0
C.I. 77775
NICHEL
Nickel
Nickel, soluble salts

00088846

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0271.htm

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Selenium and Compounds

CASRN 7782-49-2

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0472
Selenium and Compounds; CASRN 7782-49-2

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Selenium and Compounds

File On-Line 03/01/1991

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	09/01/1991
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	07/01/1993

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Selenium and Compounds
CASRN -- 7782-49-2
Last Revised -- 09/01/1991

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Clinical selenosis	NOAEL: 0.015 mg/kg/day	3	1	5E-3 mg/kg/day
Human Epidemiological Study	LOAEL: 0.023 mg/kg/day			
Yang et al., 1989b				

*Conversion Factors: NOAEL (0.853 mg/day) and LOAEL (1.261 mg/day) calculated from regression analysis (log Y = 0.767log X - 2.248, where Y = blood selenium and X = selenium intake) as detailed in Yang et al. (1989a) based upon the correlation (r = 0.962) between dietary selenium intake and blood selenium level for data showing incidence of clinical selenosis in adults based on an average adult body weight of 55 kg (Yang et al., 1989b).

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Yang, G., S. Yin, R. Zhou, et al. 1989b. Studies of safe maximal daily dietary Se-intake in a seleniferous area in China. II. Relation between Se-intake and the manifestation of clinical signs and certain biochemical alterations in blood and urine. J. Trace Elem. Electrolytes Health Dis. 3(2): 123-130.

Yang et al. (1989b), in a follow-up to an earlier study (Yang et al., 1983), studied a population of approximately 400 individuals living in an area of China with unusually high environmental concentrations of selenium (Se). The subjects were evaluated for clinical and biochemical signs of Se intoxication. Three geographical areas with low, medium and high selenium levels in the soil and food supply were chosen for comparison in the studies. The earlier Yang et al. (1983) study was conducted in response to endemic selenium intoxication in two separate areas with sample sizes of only 6 and 3. Comparisons were then made to a selenium-adequate area (n=8) and low- selenium area (n=13). The Yang et al. (1989a,b) studies provide a much larger sample size and include additional analysis of tissue selenium levels. This allows a more accurate estimation of the dose-response relationship observed for selenium toxicity. Selenium levels in soil and approximately 30 typical food types commonly eaten by the exposed population showed a positive correlation with blood and tissue Se levels. The daily average Se intakes, based on lifetime exposure, 70, 195 and 1438 ug for adult males and 62, 198 and 1238 ug for adult females in the low-, medium- and high-selenium areas, respectively. Significant correlations demonstrated between Se concentrations of various tissues were used to estimate the minimal daily Se intake values that elicited various alterations in biochemical parameters indicative of possible Se-induced liver dysfunction (i.e., prolongation of clotting time and serum glutathione titer) and clinical signs of selenosis (i.e., hair or nail loss, morphological changes of the nails, etc.). In this manner, a marginal safe level of daily Se intake was estimated.

Analysis of the results indicated that perisistent clinical signs of selenosis were observed only in 5/349 adults, a potentially sensitive subpopulation. The blood selenium concentration in this group ranged from 1.054 to 1.854 mg/L with a mean of 1.346 mg/L. Clinical signs observed included the characteristic "garlic odor" of excess selenium excretion in the breath and urine, thickened and brittle nails, hair and nail loss, lowered hemoglobin levels, mottled teeth, skin lesions and CNS abnormalities (peripheral anesthesia, acroparesthesia and pain in the extremities). Alterations in the measured biochemical parameters occurred at dietary intake levels of 750-850 ug/day. These alterations were described as a delay in prothrombin time, i.e., increase in blood coagulation time and reduction in blood glutathione concentration. However, these indicators were poorly characterized and are not typically used as an index for clinical selenosis resulting from chronic exposure to selenium (NAS, 1989). Based upon the blood selenium levels shown to reflect clinical signs of selenium intoxication, a whole blood selenium concentration of 1.35 mg/L corresponding to 1.261 mg of daily selenium intake is indicative of the lowest correlative selenium intake causing overt signs of selenosis. The next lowest whole blood selenium concentration of 1.0 mg/L, corresponding to 0.853 mg selenium/day, produces no clinical signs of selenosis. The NOAEL for this study is 0.85 mg Se/day and the LOAEL is 1.26 mg Se/day.

A group of 142 volunteers in South Dakota and Wyoming were recruited by Longnecker et al. (1991) at random from households listed in a telephone directory or from ranches with suspected high selenium intake based on previous cases of livestock selenosis. The geographical areas were chosen because of known seleniferous topsoil and high concentrations of selenium in plants and food. The subjects were followed for 1 year and completed health questionnaires, underwent physical examinations, provided blood samples for clinical assessment, and provided blood, urine, toenails and duplicate-plate food collections for selenium analysis. The average selenium intake was 239 ug/day, approximately 2-3 times higher than the national average. The concentration of selenium in whole blood, serum, urine and toenails and the amount in diet were highly correlated. Blood selenium concentration was highly correlated with selenium intake. The correlation was very similar to that reported by Yang et al. (1989a). Liver function (prothrombin time and alanine aminotransferase, aspartate aminotransferase, gamma glutamyltransferase and alkaline phosphatase), hematologic functon (leukocyte count, hemoglobin and hematocrit) and clinical chemistry (sodium, potassium

and chloride concentration) were not found to be altered as a result of selenium intake. High regression coefficient predictor variables for selenium toxicity (muscle twitching, paresthesia, nail loss, nail lines, hair loss and garlic breath) were not found in increased frequency for this population. No signs of selenium toxicity were found in this population, including individuals whose selenium intake was as high as 724 ug/day. This report corroborates that of Yang et al. (1989b), which showed that a selenium intake of up to 853 ug/day is not associated with characteristic nail or hair loss typical of selenium intoxication.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- An uncertainty factor of 3 was applied to the NOAEL to account for sensitive individuals. A full factor of 10 was not deemed necessary since similar NOAELs were identified in two moderately-sized human populations exposed to selenium levels in excess of the RDA throughout a lifetime without apparent clinical signs of selenosis.

MF -- None

___I.A.4. ADDITIONAL COMMENTS (ORAL RfD)

The essentiality for selenium has been well-documented in livestock based upon the alleviation of specific deficiency conditions by selenium supplementation of the diet (Combs and Combs, 1986). Selenium has been clearly demonstrated to be a cofactor of glutathione peroxidase, a hydrogen and lipid peroxide reducing enzyme and is therefore essential (Rotruck et al., 1973). Human requirements for selenium were not conclusively established until 1979 when an association was made between low selenium status and cardiomyopathy (Keshan disease) in China for young children and women of child-bearing age (Keshan Disease Research Group, 1979a,b). More recently, iatrogenic episodes of selenium deficiency have been reported in patients receiving intravenous total parenteral administration of feeding solutions devoid of selenium. Symptoms included low glutathione peroxidase activity and low selenium levels in erythrocytes (Levander and Burk, 1986), muscular weakness and discomfort (van Rij et al., 1979) and cardiomyopathy (Johnson et al., 1981). It is important to note that glutathione peroxidase activity is a valid indicator of human selenium status only in populations with relatively low selenium intakes, since the enzyme activity plateaus at adequate selenium intake levels (Whanger et al., 1988), thereby precluding the use of this biochemical indicator under excessive selenium intake situations.

The NAS (1989) has determined the recommended dietary allowance for selenium to be 0.87 ug/kg, or approximately 70 and 55 ug/day for the reference adult North American male and female, respectively. Requirements for selenium increase during pregnancy to 65 ug/day and for lactation to 75 ug/day. Selenium requirements for infants and children vary according to age. However, based on the reference weights of NHANES II, these populations demonstrate an increased requirement per unit weight relative to adults. For infants, the selenium requirement is 1.67 ug/kg and for children the requirement ranges from 1.07-1.53 ug/kg. It should be noted that the most recent RDA for selenium did not consider the 1989 results of Yang et al. (1989a,b) discussed above, but an earlier preliminary report by the same authors (Yang et al., 1983).

Yang et al. (1983) reported clinical signs of selenosis (i.e., loss of hair

and nails) in approximately 50% of a population of 248 inhabitants living in Enshi County, Hubei Province of the People's Republic of China. Selenosis was reported in the highest selenium contaminated area where the average daily Se intake was 5.0 mg/day (range 3.2-6.7), but no selenosis occurred when the average intake was 0.750 mg/day (range 0.240-1.51). These estimates, however, were based upon estimates of intake from only 6 and 3 inhabitants in the high and low contaminated areas, respectively. Yang et al. (1989b) reported prolonged clotting time and serum glutathione and these biochemical changes were indicated as adverse effects of selenium exposure. Glutathione is a strong nucleophile that reacts well with soft electrophiles and is an important conjugate-forming compound for the detoxification and excretion of electrophilic metabolites and metabolically produced oxidizing agents. If glutathione is depleted or markedly reduced in the liver, the hepatotoxicity of these compounds would likewise be expected to be enhanced (Ketterer et al., 1983). However, the significance of decreased serum glutathione is not well characterized and should not be used in this context as a biochemical marker of selenium toxicity. Likewise, there is no indication that prothrombin activity is affected by excess selenium administration (Longnecker et al., 1991). Furthermore, the description of this effect in Yang et al. (1989b) was based on a population for which there is insufficient documentation of normal clotting times in the general Chinese population.

Selenium toxicity has been clinically described according to three types: acute selenosis, subacute selenosis and chronic selenosis. The acute condition is caused by consuming relatively high amounts of selenium over a short period of time. After the onset of this condition, walking becomes unsteady, cyanosis of the mucous membranes occurs and labored breathing is usually seen sometimes resulting in death. Pathological findings include congestion of the liver, endocarditis and myocarditis, degeneration of the sooth musculature of the gastrointestinal tract, gallbladder and bladder, and erosion of the long bones (Francke and Moxon, 1936).

Subacute selenosis occurs from exposure to large doses of Se over a longer period of time resulting in neurological dysfunction (impaired vision, ataxia, disorientation) and respiratory distress. It is typically seen most frequently in grazing livestock feeding upon Se-accumulating plants and has been referred to as "blind staggers" (Rosenfeld and Beath, 1964).

Prolonged exposure to more moderate levels of selenium result in skin lesions involving alopecia, hoof necrosis and loss, emaciation and increased serum transaminases and alkaline phosphatase in animals. In man, the condition is characterized by chronic dermatitis, fatigue, anorexia, gastroenteritis, hepatic degeneration, enlarged spleen and increased concentrations of Se in the hair and nails (Harr and Muth, 1972).

Selenium exists naturally in a number of oxidation states, thereby accounting for the different forms of selenium important to living organisms by oral ingestion. In the -2 oxidation state, selenium can be found as hydrogen selenide (H2Se), sodium selenide (Na2Se), di-[(CH3)2Se] and trimethyl selenium [(CH3)3Se] and various selenoamino acids such as selenomethionine, selenocysteine, Se-methyl selenocysteine, selenocystathionine and selenotaurine. Elemental selenium and the dipeptide selenodiglutathione have an oxidation state of 0. In the +4 oxidation state, selenium can exist as selenium dioxide (SeO2), selenious acid (H2SeO3) or as sodium selenite (Na2SeO3). Finally, in its most oxidized state (+6), selenium can be found as selenic acid (H2SeO4) or as sodium selenate (Na2SeO4).

The toxicity of selenium has been consistently well documented. However, some early studies reported that selenium may be a carcinogen. Nelson et al. (1943) showed that rats fed diets containing Se as seleniferous wheat developed hepatic tumors and low-grade carcinomas in 11/53 animals. This work has subsequently been criticized due to low-protein content and relatively high levels of Se in the diet (5, 7 or 10 ppm Se), a poorly characterized source of selenium, and in general poor experimental design.

The authors reported no encapsulation or metastases and in fact noted their own difficulty in determining the difference between hyperplasia and tumor. Another early investigation by Seifter et al. (1946) reported several thyroid tumors and adenomatous hyperplasia in livers of rats fed 0.05% bis-4-acetylaminophenyl selenium dihydroxide for 105 days. This organic selenium compound was suspected of having goitrogenic properties but its carcinogenic effect has not been further confirmed to be attributable to the selenium in the molecule.

The first animal experiment which demonstrated anticarcinogenic effects of selenium was performed by Clayton and Baumann (1949). An approximate 50% reduction in dimethylaminoazobenzene-induced tumor incidence occurred in rats fed a diet supplemented with 5 ppm Se as selenite. Additional evidence subsequently reported, further illustrated the inhibitory effect of selenium on transplantable tumors in rats (Weisberger and Suhrland, 1956a) and leukemia in humans (Weisberger and Suhrland, 1956b). The National Cancer Institute sponsored an extensive study on selenium toxicity in rats in order to resolve the issue of selenium carcinogenicity. Diets containing up to 8 ppm selenium did not increase tumor incidence (Tinsley et al., 1967; Harr et al., 1967). Since 1970, there has been an increased interest in characterizing the anti-carcinogenic and anti-tumorigenic properties of selenium. The number of reports characterizing these properties are too numerous to discuss in detail here. The reader is referred to a review by Milner and Fico (1987) for a more comprehensive treatment of the data base.

The essentiality and toxicity of selenium varies according to the valence state of selenium when incorporated into biomolecules and the form in which selenium is fed or administered. This is especially true when comparing the LD50 value as an index of toxicity for the various selenium compounds. Although it is difficult to make an assessment for several selenium compounds by a similar mode of administration in a common species, there is general agreement that sodium selenite, sodium selenate, selenomethionine and selenoglutathione are among the more toxic species (Combs and Combs, 1986). The relative potency of systemic toxicity for selenium compounds is also similar in experiments examining potency of anti-tumorigenic activity. In vitro examination of potency of effect of selenium compounds on incubated Ehrlich ascites tumor cells (EATC) showed that sodium selenite is more efficacious in significantly reducing EATC viability than an equivalent concentration of sodium selenate. Although selenium dioxide, selenomethionine and selenocystine ultimately decreased viability of the EATC, nearly 50% more incubation time was required for the same effect (Poirier and Milner, 1979). The same authors investigated the relative potency of various selenium compounds administered intraperitoneally on EATC growth in vivo. Sodium selenite and selenodiglutathione (an intermediate of selenium metabolism) were the most effective forms of selenium in preventing EATC propagation. Sodium selenide, dimethyl selenide and selenocystine were not effective in inhibiting EATC growth (Poirier and Milner, 1983). Similar relative potency results have been reported in in vitro systems for canine mammary cells (Fico et al., 1986) and human mammary cells (Watrach et al., 1984).

Since selenium has been reported to cause growth retardation, decreased fertility, embryotoxicity, fetotoxicity and teratogenic effects in animals, Yang et al. (1989b) made the following observations: Malformation in chickens hatched from locally produced eggs did occur; however, teratogeneic effects in human infants were never seen in this area although Se has been reported to be transmitted through the placenta to the fetus in animals. These findings confirm those reported by Yang et al. (1983) in which chicken eggs from this same area were reported to have very low hatchability and some deformed embryos in those that did hatch.

The developmental toxicity of selenomethionine was investigated by Tarantal et al. (1991) in non-human primates. Forty pregnant long-tailed macaques were dosed daily by nasogastric intubation with 0, 0.025, 0.150 or 0.3 mg selenium/kg as selenomethionine through gestational days 20-50. Dams were

examined clinically and the pregnancies of two to three dams within each test group were followed to term (gestational day 165). All other dams were hysterectomized on gestational day 100. Neonates delivered at term were examined for morphometric, neurologic, behavioral and ophthalmologic effects on days 1, 8, 15, 22 and 30. Pregnancy loss among treated animals was not significantly different from concurrent or historical controls. No statistically significant treatment-related effects were observed at necropsy on gestational day 100. There were no significant maternal or fetal developmental effects or teratogenesis found up to 0.3 mg/kg selenium, the highest dose tested.

Halverson et al. (1966) fed 60-70 g male, post-weanling Sprague-Dawley rats selenium as selenite or seleniferous wheat ad libitum at 1.6, 3.2, 4.8, 6.4, 8.0, 9.6 or 11.2 ppm of selenium (13, 27, 40, 67, 81 or 94 ug/kg/day, respectively). Levels of selenium up to 4.8 ppm showed no effect. At 8.0 ppm selenium as seleniferous wheat, there was an observed decrease in liver weight, increase in spleen weight, and decrease in hemoglobin. Mortality was observed in the groups fed 8.0, 9.6 and 11.2 ppm selenium as seleniferous wheat at incidences of 1/8, 5/8 and 8/8, respectively. The incidences of mortality reported for groups fed 8.0 and 9.6 ppm selenium as selenite were 1/8 and 1/10, respectively. A significant growth reduction was reported for both selenium sources at 6.4 ppm and higher, although feed utilization was not decreased. No other effects were reported for the rats fed sodium selenite.

Schroeder and Mitchener (1971) administered 3 ppm selenium as selenate (390 ug/kg/day) to CD mice through four generations. Maternal effects were not observed. There was a significant increase in young deaths in the F1 generation and an increase in numbers of runts in generations F1 through F3. By the F3 generation there was also a decrease in breeding events.

Rosenfeld and Beath (1954) administered selenium as potassium selenate to sires and pregnant rats through five breeding cycles at 1.5, 2.5 or 7.5 ppm selenium (75, 125 or 375 ug/kg/day). No effect was observed on reproduction, the number of young reared or on the reproduction of two successive generations of dams and sires in groups receiving 1.5 ppm selenium. In the group receiving 2.5 ppm selenium, the was a 50% reduction in the number of young reared. At 7.5 ppm there was a decrease in fertility of the females but not males, a decrease in the number of survivors and a reduction in the rate of growth in the young.

Nobunaga et al. (1979) administered 3 or 6 ppm selenium (390 or 780 ug/kg/day, respectively) as selenite to IVCS mice for 30 days prior to mating and throughout gestation. On day 18 of gestation, maternal mice were sacrificed and the embryos removed. Number of litters, total implants, total implants per dam, dead fetuses, dead embryos, resorptions, surviving fetuses (% to total implants), litter size, gross malformations and skeletal anomalies were not significantly different for either selenium-treated or control mice. The only significant effect noted was a decrease in the body weight of surviving fetuses in mice given 6 ppm selenium.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

Confidence in the chosen principal study is medium. Although this is a human epidemiological study in which a sizable population with sensitive subpopulations was studied, there are still several possible interactions that were not fully accounted for, e.g., fluoride intake and protein status. Also,

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except for clinical signs of selenosis there are no other reliable indicators, biochemical or clinical, of selenium toxicity. Confidence in the data base is high because many animal studies and epidemiologic studies (reviewed by Combs and Combs, 1986) support the principal study. An additional human study with a freestanding NOAEL (Longnecker et al., 1991) strongly corroborates the NOAEL identified in the principal study. Therefore, high confidence in the RfD is selected based upon support of the critical study and the high level of confidence in the data base.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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

Other EPA Documentation -- U.S. EPA, 1985

Agency Work Group Review -- 01/20/1988, 03/22/1989, 09/21/1989, 11/14/1990, 03/27

Verification Date -- 03/27/1991

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Selenium and Compounds
CASRN -- 7782-49-2

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Selenium and Compounds
CASRN -- 7782-49-2
Last Revised -- 07/01/1993

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

NOTE: This assessment is for the following compounds: Selenium (CASRN 7782-49-2); sodium selenate (CASRN 13410-01-0); sodium selenite (CASRN 10102-18-8); selenious acid (CASRN 7783-00-8); selenic acid (CASRN 7783-08-6); sodium selenide (CASRN 1313-85-5).

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to carcinogenicity in humans

Basis -- Based on inadequate human data and inadequate evidence of carcinogenicity in animals. The evidence for various selenium compounds in animal and mutagenicity studies is conflicting and difficult to interpret; however, evidence for selenium sulfide is sufficient for a B2 (probable human carcinogen) classification.

___II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. Data on the potential carcinogenicity of selenium and various selenium compounds in humans are inadequate. Epidemiological studies have evaluated selenium in blood and cancer death rates in areas of high vs. low naturally-occurring selenium. However, these studies have limited value because they do not assess specific selenium compounds or correlate exposure with cancer risk.

Several investigators have studied the association between serum selenium and the risk of cancer through prospective, case-control and nested case-control studies. Analysis of blood serum levels indicated that patients with cancer, particularly gastrointestinal cancer, prostatic cancer, or Hodgkin's lymphoma, had significantly lower blood selenium levels in blood than healthy patients (Shamberger et al., 1973; Salonen et al., 1984; Kok et al., 1987; Willet et al., 1983; Willet and Stampfer, 1986). The risk of cancer for men

(Kok et al., 1987) or for all subjects (Willet et al., 1983) in the lowest quintile of serum selenium was twice that of subjects with higher levels.

Geographic correlational studies have compared cancer mortality in areas of high vs. low levels of naturally-occurring selenium. In an ecological study Shamberger and Frost (1969) reported that an inverse relationship existed between cancer death rates and the selenium concentrations in foliage plants of several Canadian provinces. The human cancer death rate in provinces with selenium-containing plants was 122.2 +/- 7.8 (presumably per 100,000 population although this was not specified), while in the provinces devoid of these plants, the human death rate was 139.9 +/- 4.0.

In an ecological study Shamberger and Willis (1971) reported that there was a correlation between decreased cancer death rates in humans and an increase in the selenium in the forage crops in California. In high-selenium areas (selenium 0.11 ppm of forage crops) the cancer death rate per 100,000 was 141.2. In the medium-selenium areas (0.05-0.10 ppm) the cancer death rate was 190.1. In low-selenium areas (0.02-0.05 ppm) the cancer death rate was 233.0. Shamberger and Willis (1971) also investigated the ratio of observed to expected cancer death rates by anatomic site for men in 17 paired cities including high- and low-selenium areas. The anatomic sites that would come into contact with dietary selenium, such as pharynx, esophagus, stomach, bladder and intestine, showed a substantially lower rate ratio in the high-selenium cities than in the low-selenium cities. Other ecological and prospective studies have correlated an increased incidence of colon, breast and other forms of cancer in humans in geographic areas where selenium is deficient and a lowered cancer incidence with higher selenium concentrations (Schrauzer and Ishmael, 1974; Shamberger, 1976; Schrauzer et al., 1976; Jansson et al., 1978; Yang et al., 1983).

In a study of approximately 300 employees exposed to selenium (form not specified) in a rectifier (electronics) process over a 26-year period, only 17 deaths occurred, 6 of which were because of cancer (Glover, 1970). This number, however, is not statistically different from the 5.1 deaths expected based on national mortality rates. The source of the mortality rates was not specified. Several toxic effects including pulmonary irritation, epigastric pain and dermal irritation and dermatitis were associated with selenium exposure in men, but no carcinogenic effect was reported.

II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. The carcinogenicity of selenium compounds has been evaluated in several animal studies. However, the data are conflicting and difficult to interpret because of apparent anticarcinogenic activity and high toxicity of some selenium salts. In addition, comparison of the available data is difficult because several different salts with varying degrees of bioavailability were used in the assays.

In a 2-year dietary study reported by Nelson et al. (1943), Osborne-Mendel rats (sex not specified) were fed selenium in the form of seleniferous corn or wheat or ammonium potassium selenide at 5-10 ppm. Survival was lower in the treated rats; 53/126 (42%) rats fed selenium survived 18 months or longer compared with 14/18 (78%) control rats. Of the 53 surviving selenium-treated rats, 43 (81%) developed liver cirrhosis and 11 (21%) developed hepatocellular adenoma or carcinoma. All 11 animals with tumors also had liver cirrhosis. None of the 14 control animals surviving 2 years developed liver tumors. Only pooled group data were reported and no statistical analysis was reported.

No tumors developed in a total of 1437 Wistar rats fed sodium selenite or sodium selenate in the diet at levels of 0.5-16 ppm for their lifetime (Harr

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et al., 1967; Tinsley et al., 1967). Nonneoplastic liver effects such as hyperemia, cellular degeneration, binucleation, and mild proliferation of hepatocytes were observed at concentrations of 4 ppm and higher.

Long-Evans rats (approximately 50/sex/group at study initiation) received 2 ppm (as selenium) sodium selenate or sodium selenite in drinking water for 1 year, then 3 ppm for the remainder of the study (Schroeder and Mitchener, 1971). The treatment of the control group was not discussed. The animals were observed for the duration of their natural lifespan, approximately 36 months, although one selenate-treated female lived for 5 years. Selenite produced 50% mortality in males by 58 days. At this time, 2 ppm selenate was substituted for selenite in the male group. The concentration of selenium was raised to 3 ppm in this group when the animals were 1 year old; however, the high mortality rendered the group size too small for further statistical analysis. Selenite produced 50% mortality in females by 348 days; selenite-treated females were sacrificed at 23 months due to high mortality. Selenate produced 50% mortality in females by 1014 days and in males by 962 days. In the control groups 50% mortality was achieved by 872 and 853 days in females and males, respectively. Survival of rats receiving selenate was comparable to controls and median lifespan was increased by >100 days. Body weights of treated males were comparable to controls throughout the study. Body weights of females fed selenate were significantly greater than controls at 24 and 36 months; body weights of females fed selenite were significantly less than controls at all times but 18 months.

Incidence of all tumors and of malignant tumors was significantly increased in the selenate-treated rats compared with the controls. Incidence of all tumors in controls, selenate- and selenite-treated rats was 20/65 (30.8%), 30/48 (62.5%) and 4/32 (12.5%), respectively. Incidence of malignant tumors in the same groups was 11/65 (16.9%), 20/48 (41.7%) and 4/32 (12.5%), respectively. The earliest tumor occurred on day 833 in the control males, on day 633 in the control females, on day 344 in selenate males and on day 633 in selenate females. The shortened survival time of the selenite groups was thought to be responsible for the small number of tumors. This study is considered inadequate because only the heart, lung, liver, kidney and spleen tissues from animals necropsied were examined histologically, and an increase in longevity was observed in selenate-treated female rats.

Schroeder and Mitchener (1972) administered 3 ppm sodium selenate or sodium selenite in drinking water to Swiss mice (50/sex/group). Body weights of selenate-treated animals were comparable to controls. Body weights of males fed selenite were significantly increased compared with controls, but body weights of females fed selenite were significantly decreased compared with controls. Longevity in males fed selenate was increased compared with controls. Longevity in females fed selenate increased, but longevity in females fed selenite decreased compared with controls. When compared to controls, there was no significant increase in total tumor incidence or malignant tumor incidence observed in selenium- (form not specified) treated mice. In the control group 23/119 (19%) had tumors (10/119 (8%) malignant tumors). Selenium-fed mice showed 13/88 (15%) tumors (all were malignant). In selenium-treated group 8/13 malignancies were lymphoma or leukemia, 4/13 were papillary or alveologenic adenocarcinoma and 1/13 an osteosarcoma. In the control group there were two incidences of lymphoma or leukemia, 7 of lung carcinoma and 1 carcinoma of unknown origin. The 13 benign tumors included breast and ovary tumors.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Selenium is an essential micronutrient for several species, including humans, and is part of several enzymes such as glutathione peroxidase, an

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enzyme involved in cellular defense against oxidative damage, and heme oxidase. While low doses of selenium are essential, high doses of selenium or a deficiency of dietary selenium may cause a toxic response. Additionally, selenium may be protective against tumor development. The greatest daily exposure to selenium is via food. Bioavailability of selenium is dependent on numerous factors, including the intake levels, chemical form and nutritional status. Organic forms of selenium are more bioavailable than inorganic forms; selenates and selenites are the inorganic forms more readily absorbed. Sodium selenate and selenite are soluble in water, but the extent to which they are absorbed dermally or through the gastrointestinal tract has not been fully elucidated (U.S. EPA, 1989).

Shamberger (1985) reported that the oral administration of 0.1-6 ppm or dermal application of 0.005% of selenium reduced incidences of skin, liver, tracheal, intestinal and lung tumors induced by several carcinogens in rats, mice and hamsters. Shamberger theorized that selenium may reduce cellular damage caused by peroxidation of fat. In another study, natural killer (NK) cell activity was significantly increased in female rats administered 0.5 or 2.0 ppm selenium (sodium selenate) in the drinking water for 10 weeks (Koller et al., 1986), suggesting to the authors that NK-sensitive tumors may be prevented by using selenium therapy.

Data on the mutagenicity of selenium and its compounds are equivocal. Selenate and selenite (12 uM) were mutagenic in a reverse mutation assay using Salmonella typhimurium strains TA98, TA100 and TA1537 (Noda et al., 1979) in the absence of rat hepatic homogenates. In a second assay, sodium selenate, but not sodium selenite, was mutagenic; the S. typhimurium strains used were not reported (Lofroth and Ames, 1978). Selenite (selenious acid and sodium selenite) produced DNA damage in Bacillus subtilis strains 17A and 45T; however, selenate (selenic acid and sodium selenate) was negative in the Rec assay (Nakamuro et al., 1976).

Sodium selenide, sodium selenite, and sodium selenate (in order of decreasing activity) caused an increase in unscheduled DNA synthesis in the presence or absence of glutathione in Chinese hamster ovary cells at concentrations of 1.0E-4 M (Whiting et al., 1980). Increased chromosomal aberrations were also produced by sodium selenite at E-5 M in rat lymphocytes (Newton and Lilly, 1986) and by sodium selenite, selenious acid, selenic acid, and selenium oxide at 2.6E-6 M in human lymphocytes (Nakamuro et al., 1976). Sodium selenite produced an increase in chromosomal aberrations in the bone marrow of rats administered a total of 10-12 mg/kg intravenously (near-lethal doses) (Newton and Lilly, 1986). Selenium (elemental), selenium dioxide, sodium selenide, and sodium selenite (in order of decreasing activity) induced an increase in SCEs in human whole-blood cultures; sodium selenate was not mutagenic in this assay (Ray and Altenburg, 1980).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

None.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

None.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1980, 1984, 1989

The 1989 Health and Environmental Effects Document on Selenium and Compounds has received OHEA review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 11/09/1989, 03/07/1990

Verification Date -- 03/07/1990

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

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CASRN -- 7782-49-2
Last Revised -- 06/01/1991

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None

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VII. REVISION HISTORY

Substance Name -- Selenium and Compounds
CASRN -- 7782-49-2

Date	Section	Description
03/01/1991	II.	Carcinogenicity assessment on-line
03/01/1991	VI.	Bibliography on-line
06/01/1991	II.A.2.	Para 4: Correct Shamber&Willis to Shamberger&Willis
06/01/1991	I.A.	Oral RfD summary on-line
06/01/1991	VI.A.	Oral RfD references added
09/01/1991	I.A.2.	Paragraph 2, line 2: number corrected to 5/349
09/01/1991	I.A.3.	Text revised
09/01/1991	I.A.5.	Confidence in RfD changed; text revised
01/01/1992	IV.	Regulatory Action section on-line
07/01/1993	II.D.3.	Primary contact's phone number changed

VIII. SYNONYMS

Substance Name -- Selenium and Compounds
CASRN -- 7782-49-2
Last Revised -- 03/01/1991

7782-49-2
Selenium
C.I. 77805
Caswell No. 732
ELEMENTAL SELENIUM
EPA Pesticide Chemical Code 072001
HSDB 4493
SELEN [Polish]
Selenio [Spanish]
Selenium
SELENIUM ALLOY
SELENIUM BASE
SELENIUM DUST
SELENIUM ELEMENTAL
SELENIUM HOMOPOLYMER
UN 2658
13410-01-0
Selenic acid, disodium salt
Caswell No. 791
Disodium selenate
EPA Pesticide Chemical Code 072002
Natriumseleniat [German]
NSC 378348
Selenic acid, disodium salt
Sodium selenate
10102-18-8

00088865

Selenious acid, disodium salt
DISODIUM SELENITE
DISODIUM SELENIUM TRIOXIDE
HSDB 768
Natriumselenit [German]
SELENIOUS ACID, DISODIUM SALT
SODIUM SELENITE
UN 2630
7783-00-8
Selenious acid
HSDB 6065
MONOHYDRATED SELENIUM DIOXIDE
Selenious Acid
7783-08-6
Selenic acid
Acide selenique [French]
Acido selenico [Spanish]
HSDB 675
Selenic acid
UN 1905
1313-85-5
Sodium selenide [Na2Se]
Disodium monoselenide
Sodium selenide

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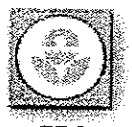

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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Silver

CASRN 7440-22-4

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0099
Silver; CASRN 7440-22-4

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Silver

File On-Line 01/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	12/01/1996
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	06/01/1989

00088867

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Silver
CASRN -- 7440-22-4
Last Revised -- 12/01/1996

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Argyria	NOEL: None	3	1	5E-3 mg/kg/day
2- to 9-Year Human i.v. Study Gaul and Staud, 1935	LOAEL: 1 g (total dose); converted to an oral dose of 0.014 mg/kg/day			

*Conversion Factors: Based on conversion from the total i.v. dose to a total oral dose of 25 g (i.v. dose of 1 g divided by 0.04, assumed oral retention factor; see Furchner et al., 1968 in Additional Comments section) and dividing by 70 kg (adult body weight) and 25,500 days (a lifetime, or 70 years).

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Gaul, L.E. and A.H. Staud. 1935. Clinical spectroscopy. Seventy cases of generalized argyrosis following organic and colloidal silver medication. J. Am. Med. Assoc. 104: 1387-1390.

The critical effect in humans ingesting silver is argyria, a medically benign but permanent bluish-gray discoloration of the skin. Argyria results from the

deposition of silver in the dermis and also from silver-induced production of melanin. Although silver has been shown to be uniformly deposited in exposed and unexposed areas, the increased pigmentation becomes more pronounced in areas exposed to sunlight due to photoactivated reduction of the metal. Although the deposition of silver is permanent, it is not associated with any adverse health effects. No pathologic changes or inflammatory reactions have been shown to result from silver deposition. Silver compounds have been employed for medical uses for centuries. In the nineteenth and early twentieth centuries, silver arsphenamine was used in the treatment of syphillis; more recently it has been used as an astringent in topical preparations. While argyria occurred more commonly before the development of antibiotics, it is now a rare occurrence. Greene and Su (1987) have published a review of argyria.

Gaul and Staud (1935) reported 70 cases of generalized argyria following organic and colloidal silver medication, including 13 cases of generalized argyria following intravenous silver arsphenamine injection therapy and a biospectrometric analysis of 10 cases of generalized argyria classified according to the quantity of silver present. In the i.v. study, data were presented for 10 males (23-64 years old) and for two females (23 and 49 years old) who were administered 31-100 i.v. injections of silver arsphenamine (total dose was 4-20 g) over a 2- to 9.75-year period. Argyria developed after a total dose of 4, 7 or 8 g in some patients, while in others, argyria did not develop until after a total dose of 10, 15 or 20 g. In the biospectrometric analysis of skin biopsies from 10 cases of generalized argyria, the authors confirmed that the degree of the discoloration is directly dependent on the amount of silver present. The authors concluded that argyria may become clinically apparent after a total accumulated i.v. dose of approximately 8 g of silver arsphenamine. The book entitled "Argyria. The Pharmacology of Silver" reached the same conclusion, that a total accumulative i.v. dose of 8 gm silver arsphenamine is the limit beyond which argyria may develop (Hill and Pillsbury, 1939). However, since body accumulates silver throughout life, it is theoretically possible for amounts less than this (for example, 4 g silver arsphenamine) to result in argyria. Therefore, based on cases presented in this study, the lowest i.v. dose resulting in argyria in one patient, 1 g metallic silver (4 g silver arsphenamine x 0.23, the fraction of silver in silver arsphenamine) is considered to be a minimal effect level for this study.

Blumberg and Carey (1934) reported argyria in an emaciated chronically ill (more than 15 years) 33-year-old female (32.7 kg) who had ingested capsules containing silver nitrate over a period of 1 year. The patient reported ingesting 16 mg silver nitrate three times a day (about 30 mg silver/day) for alternate periods of 2 weeks. Spectrographic analysis of blood samples revealed a blood silver level of 0.5 mg/L 1 week after ingestion of silver nitrate capsules ceased, and there was only a small decrease in this level after 3 months. The authors noted that this marked argyremia was striking because even in cases of documented argyria, blood silver levels are not generally elevated to this extent. Normal levels for argyremic patients were reported to range from not detected to 0.005 mg Ag/l blood. Heavy traces of silver in the skin, moderate amounts in the urine and feces, and trace amounts in the saliva were reported in samples tested 3 months after ingestion of the capsules stopped; however, despite the marked argyremia and detection of silver in the skin, the argyria at 3 months was quite mild. No obvious dark pigmentation was seen other than gingival lines which are considered to be characteristic of the first signs of argyria. The authors suggested that this may have been because the woman was not exposed to strong light during the period of silver treatment. This study is not suitable to serve as the basis for a quantitative risk assessment for silver because it is a clinical report on only one patient of compromised health. Furthermore, the actual amount of silver ingested is based on the patient's recollection and cannot be accurately determined.

In a case reported by East et al. (1980), argyria was diagnosed in a 47-year-

old woman (58.6 kg) who had taken excessively large oral doses of anti-smoking lozenges containing silver acetate over a period of 2.5 years. No information was provided as to the actual amount of silver ingested. Symptoms of argyria appeared after the first 6 months of exposure. Based on whole body neutron activation analysis, the total body burden of silver in this female was estimated to be 6.4 (plus or minus 2) g. Both the total body burden and concentration of silver in the skin were estimated to be 8000 times higher than normal. In a separate 30-week experiment, the same subject retained 18% of a single dose of orally-administered silver, a retention level much higher than that reported by other investigators. East et al. (1980) cited other studies on this particular anti-smoking formulation (on the market since 1973) which demonstrated that "within the limits of experimental error, no silver is retained after oral administration." However, this may not hold true for excessive intakes like that ingested by this individual. As with the study by Blumberg and Carey (1934), this study is not suitable to serve as the basis for a quantitative risk assessment. It is a clinical report on only one patient and the actual amount of silver ingested can only be estimated.

I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- An uncertainty factor of 3 is applied to account for minimal effects in a subpopulation which has exhibited an increased propensity for the development of argyria. The critical effect observed is a cosmetic effect, with no associated adverse health effects. Also, the critical study reports on only 1 individual who developed argyria following an i.v. dose of 1 g silver (4 g silver arsphenamine). Other individuals did not respond until levels five times higher were administered. No uncertainty factor for less than chronic to chronic duration is needed because the dose has been apportioned over a lifetime of 70 years.

MF -- None

I.A.4. ADDITIONAL STUDIES/COMMENTS (ORAL RfD)

In the study by East et al. (1980) (see section I.A.2.), one human was found to retain 18% of a single oral dose. However, the authors acknowledge that this high level of retention is not consistent with data published in other laboratories. For ethical reasons, the experiment could be not repeated to determine the validity of the results.

Humans are exposed to small amounts of silver from dietary sources. The oral intake of silver from a typical diet has been estimated to range from 27-88 ug/day (Hamilton and Minski, 1972/1973; Kehoe et al., 1940). Tipton et al. (1966) estimated a lesser intake of 10-20 ug/day in two subjects during a 30-day observation period. Over a lifetime, a small but measurable amount of silver is accumulated by individuals having no excessive exposure. Gaul and Staud (1935) estimated that a person aged 50 years would have an average retention of 0.23-0.48 g silver (equivalent to 1-2 g silver arsphenamine). Petering et al. (1991) estimated a much lower body burden of 9 mg over a 50-year period based on estimated intake, absorption, and excretion values; however, it is not clear how the final estimate was calculated. Furchner et al. (1968) studied the absorption and retention of ingested silver (as silver nitrate, amount not specified) in mice, rats, monkeys and dogs. In all four species, very little silver was absorbed from the GI tract. Cumulative excretion ranged from 90 to 99% on the second day after ingestion, with <1% of the dose being retained in <1 week in monkeys, rats and mice. Dogs had a

slightly greater retention. The authors used the data from the dog to estimate how much silver ingested by a 70 kg human would be retained. An "equilibrium factor" of 4.4% was determined by integrating from zero to infinity a retention equation which assumes a triphasic elimination pattern for silver with the initial elimination of 90% coming from the dog data. The first elimination half-time of 0.5 days was used "arbitrarily"; subsequent half-times of 3.5 days and 41 days were taken from a metabolic study by Polachek et al. (1960). Furchner et al. (1968) considered their calculated equilibrium factor of 4.4% to be a conservative estimate for the amount of silver which would be retained by a 70 kg human. This figure was rounded to 4% and was used in the dose conversion (i.v. dose converted to oral intake) for the calculation of the RfD.

In addition to silver arsphenamine, any silver compound (silver nitrate, silver acetate, argyrol, Neosilvol and Collargol, etc.), at high dose, can cause argyria. Another important factor predisposing to the development of argyria is the exposure of the skin to light.

Argyria, the critical effect upon which the RfD for silver is based, occurs at levels of exposure much lower than those levels associated with other effects of silver. Argyrosis, resulting from the deposition of silver in the eye, has also been documented, but generally involves the use of eye drops or make-up containing silver (Greene and Su, 1987). Silver has been found to be deposited in the cornea and the anterior capsule of the lens. The same deposition pattern was seen in the eyes of male Wistar rats following administration of a 0.66% silver nitrate solution to the eyes for 45 days (Rungby, 1986). No toxicological effects were reported.

Toxic effects of silver have been reported primarily for the cardiovascular and hepatic systems. Olcott (1950) administered 0.1% silver nitrate in drinking water to rats for 218 days. This exposure (about 89 mg/kg/day) resulted in a statistically significant increase in the incidence of ventricular hypertrophy. Upon autopsy, advanced pigmentation was observed in body organs, but the ventricular hypertrophy was not attributed to silver deposition.

Hepatic necrosis and ultrastructural changes of the liver have been induced by silver administration to vitamin E and/or selenium deficient rats (Wagner et al., 1975; Diplock et al., 1967; Bunyan et al., 1968). Investigators have hypothesized that this toxicity is related to a silver-induced selenium deficiency that inhibits the synthesis of the seleno-enzyme glutathione peroxidase. In animals supplemented with selenium and/or vitamin E, exposures of silver as high as 140 mg/kg/day (100 mg Ag/L drinking water) were well-tolerated (Bunyan et al., 1968).

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

The critical human study rates a medium confidence. It is an old study (1935) which offers fairly specific information regarding the total dose of silver injected over a stated period of time. One shortcoming of the study is that only patients developing argyria are described; no information is presented on patients who received multiple injections of silver arsphenamine without developing argyria. Therefore, it is difficult to establish a NOAEL. Also, the individuals in the study were being treated for syphilis and may have been of compromised health.

Confidence in the data base is considered to be low because the studies used to support the RfD were not controlled studies. For clinical case studies of argyria (such as Blumberg and Carey, 1934; East et al., 1980), it is especially difficult to determine the amount of silver that was ingested.

Confidence in the RfD can be considered low-to-medium because, while the critical effect has been demonstrated in humans following oral administration of silver, the quantitative risk estimate is based on a study utilizing intravenous administration and thus necessitates a dose conversion with inherent uncertainties.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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

Other EPA Documentation -- None

Agency Work Group Review -- 10/09/1985, 02/05/1986, 04/18/1990, 02/20/1991, 07/18

Verification Date -- 07/18/1991

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Silver
CASRN -- 7440-22-4

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Silver
CASRN -- 7440-22-4
Last Revised -- 06/01/1989

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

II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classified as to human carcinogenicity

Basis -- In animals, local sarcomas have been induced after implantation of foils and discs of silver. However, the interpretation of these findings has been questioned due to the phenomenon of solid-state carcinogenesis in which even insoluble solids such as plastic have been shown to result in local fibrosarcomas.

II.A.2. HUMAN CARCINOGENICITY DATA

No evidence of cancer in humans has been reported despite frequent therapeutic use of the compound over the years.

II.A.3. ANIMAL CARCINOGENICITY DATA

Inadequate. Local sarcomas have been induced after subcutaneous (s.c.) implantation of foils and discs of silver and other noble metals. Furst (1979, 1981), however, cited studies showing that even insoluble solids such as smooth ivory and plastic result in local fibrosarcomas and that tin when crumbled will not. He concluded that i.p. and s.c. implants are invalid as indicators of carcinogenicity because a phenomenon called solid-state carcinogenesis may complicate the interpretation of the cause of these tumors. It is difficult to interpret these implantation site tumors in laboratory animals in terms of exposure to humans via ingestion. Within these

constraints there are two studies given below in which silver per se appeared to induce no carcinogenic response.

Schmahl and Steinhoff (1960) reported, in a study of silver and of gold, that colloidal silver injected both i.v. and s.c. into rats resulted in tumors in 8 of 26 rats which survived longer than 14 months. In 6 of the 8, the tumor was at the site of the s.c. injection. In about 700 untreated rats the rate of spontaneous tumor formation of any site was 1 to 3%. No vehicle control was reported.

Furst and Schlauder (1977) evaluated silver and gold for carcinogenicity in a study designed to avoid solid-state carcinogenesis. Metal powder was suspended in trioctanoin and injected monthly, i.m., into 50 male and female Fischer 344 rats per group. The dose was 5 mg each for 5 treatments and 10 mg each for 5 more treatments for a total dose of 75 mg silver. The treatment regimen included a vehicle control (a reportedly inert material), and cadmium as a positive control. Injection site sarcomas were found only in vehicle control (1/50), gold (1/50) and cadmium (30/50); no tumors (0/50) appeared at the site of injection in the silver-treated animals. A complete necropsy was performed on all animals. The authors mentioned the existence of spontaneous tumors in Fischer 344 rats, but reported only injection site tumors. They concluded that finely divided silver powder injected i.m. does not induce cancer.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Further support for the lack of silver's ability to induce or promote cancer stems from the finding that, despite long standing and frequent therapeutic usage in humans, there are no reports of cancer associated with silver. In a recent Proceedings of a Workshop/Conference on the Role of Metals in Carcinogenesis (1981) containing 24 articles on animal bioassays, epidemiology, biochemistry, mutagenicity, and enhancement and inhibition of carcinogenesis, silver was not included as a metal of carcinogenic concern.

No evidence of the mutagenicity of silver was shown in two available studies. Demerec et al. (1951) studied silver nitrate for the possible induction of back-mutations from streptomycin dependence to nondependence in Eschericha coli. Silver nitrate was considered nonmutagenic in this assay. Nishioka (1975) screened silver chloride with other chemicals for mutagenic effects using a method called the rec-assay. Silver chloride was considered nonmutagenic in this assay.

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

Not available.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1988

The 1988 Drinking Water Criteria Document for Silver has received Agency review.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 09/22/1988

Verification Date -- 09/22/1988

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

VI. BIBLIOGRAPHY

Substance Name -- Silver
CASRN -- 7440-22-4
Last Revised -- 12/01/1991

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_VII. REVISION HISTORY

Substance Name -- Silver
CASRN -- 7440-22-4

Date	Section	Description
03/01/1988	I.A.4.	Text revised
03/01/1988	I.A.7.	Secondary contact changed
06/30/1988	I.A.7.	Primary contact changed
06/01/1989	II.	Carcinogen summary on-line
06/01/1989	VI.	Bibliography on-line
08/01/1989	VI.A.	Oral RfD references added
03/01/1991	I.A.	Oral RfD summary noted as pending change
08/01/1991	I.A.	Withdrawn; new oral RfD verified (in preparation)
08/01/1991	VI.A.	Oral RfD references withdrawn
12/01/1991	I.A.	Oral RfD summary replaced; RfD changed
12/01/1991	VI.A.	Oral RfD references replaced
01/01/1992	IV.	Regulatory actions updated
12/01/1996	I.A.7.	Secondary contact removed

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VIII. SYNONYMS

Substance Name -- Silver
CASRN -- 7440-22-4
Last Revised -- 06/01/1989

7440-22-4
ARGENTUM CREDE
COLLARGOL
Silver

IRIS Home Page | Substance File List

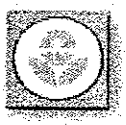

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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Strontium

CASRN 7440-24-6

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0550
Strontium; CASRN 7440-24-6

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Strontium

File On-Line 10/01/1992

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	12/01/1996
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	no data	

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Strontium
CASRN -- 7440-24-6
Last Revised -- 12/01/1996

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
Rachitic bone 20-Day, 9-Week, and 3-Year Oral Studies in Young and Adult Rats Storey, 1961; Marie et al., 1985; Skoryna, 1981	NOAEL: 0.19% Sr (as SrCO3) (190 mg Sr/kg/day) LOAEL: 0.38% Sr (as SrCO3) (380 mg/kg/day)	300	1	6E-1 mg/kg/day

*Conversion Factors: 0.19% Sr = 1900 mg Sr/kg diet. Assuming young rats consume food equivalent to 10% of their body weight/day, the actual intake is calculated to be 190 mg Sr/kg bw/day.

I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

Storey, E. 1961. Strontium "rickets": bone calcium and strontium changes. Austral. Ann. Med. 10: 213-222.

Marie, P.J., M.T. Garba, M. Hott and L. Miravet. 1985. Effect of low doses of stable Sr on bone metabolism in rats. Miner. Electrolyte Metab. 11: 5-13.

Skoryna, S.C. 1981. Effects of oral supplementation with stable strontium. Can. Med. Assoc. J. 125(7): 703-712.

Storey (1961) fed young (40-60 g) and adult (200-250 g) female rats (strain unspecified; 3/group) diets with adequate calcium (1.6%), phosphorous (0.9%) and vitamin D for 20 days. The dietary levels of strontium (as strontium carbonate) given to both adult and young rats were 0.19, 0.38, 0.75, 1.0 (young rats only), 1.5 and 3.0%. Assuming young rats consume 10% and adult rats consume 5% of their body weight in food per day, these doses correspond to 190, 380, 750, 1000, 1500 and 3000 mg/kg-day for young rats and 95, 190, 375, 750 and 1500 mg/kg-day for adult rats. Rats were examined for changes in bone mineralization and defects in cartilage. They were weighed at the onset and end of the experiment. Young rats were found to be affected more severely at lower dietary Sr levels than were adult rats. In young rats at 0.38% (380 mg/kg-day) the epiphyseal plate was irregular and slightly widened; however, at 0.75% (750 mg/kg-day) this plate was so irregular that measurements were unreliable. Changes observed with the dose of 0.38% and higher were inhibition of calcification, as evidenced by increasing width of epiphyseal cartilage, presence of uncalcified bone matrix and decreased ash weight of bone. In adults, the first obvious bone change occurred at the 1.5% dietary strontium level (750 mg/kg-day) and included slightly wider than normal epiphyseal cartilage plate and metaphyseal osteoid seams, which were irregularly increased in extent and width. At the 3% strontium level in adult animals (1500 mg/kg-day), the cartilage plate was much larger. For young rats, the dietary level of 0.19% strontium (190 mg/kg-day) was a NOAEL and 0.38% strontium (380 mg/kg-day) was a LOAEL. For adult rats, the dietary level of 0.75% strontium (375 mg/kg-day) was a NOAEL and 1.5% strontium (750 mg/kg-day) was a LOAEL.

Marie et al. (1985) administered stable strontium to weanling male Spraque-Dawley rats. The purpose of this study was to determine the effect of low doses of stable strontium on mineral homeostasis and bone histology. Rats were divided into groups (8/group) receiving 0, 0.19, 0.27, 0.34 and 0.40% of SrCl2 in distilled water for 9 weeks. The diet contained 0.5% calcium. Based on body weight and water consumption data, the authors estimated average strontium intakes of 0, 316, 425, 525 and 633 mg/kg-day. The authors concluded that an oral dose lower than 0.40% (633 mg/kg-day) did not produce adverse effects on body growth or on bone mineralization. Rats in the 0.40% (633 mg/kg-day) dose group showed signs of increased mineralization lag time; excessive osteoid thickness associated with a decline in the rate of calcification, which resulted in slow growth rate; and a decreased double-labeled osteoid surface, which frequently resulted in defective long bone growth. This study identified a NOAEL of 525 mg/kg and a LOAEL of 633 mg/kg-day.

Skoryna (1981) investigated the oral toxicity of stable strontium in male adult RVH hooded rats. The rats (12/group, starting weight of 250 g) were fed ad libitum a standard laboratory diet and divided into four groups, which were administered 0.002, 900, 1900 or 3400 ppm strontium chloride (55% strontium) in their drinking water for 3 years. Assuming that an adult rat consumes water at a rate of 49 mL/day, the experimental doses correspond to 70, 147 and 263 mg/kg Sr/day. The control and experimental groups received adequate amounts of calcium (0.35 ppm) and magnesium (0.0682 ppm) in their drinking water. The animals were weighed and examined weekly. Histologic examinations of bone and observation of body weight changes in rats receiving strontium in drinking water revealed no abnormalities (Skoryna and Fuskova, 1981). The animal tissues from different organs (kidney, lungs, adrenal, brain, heart and muscle) were examined on gross and histologic levels. No evidence of changes in morphology was observed; organs were not weighed. The concentration of strontium in tissues was determined by heated graphite atomization. In addition, strontium levels in the animals' serum were analyzed by standard atomic absorption spectrophotometry. Except for bone, no organ predilection for strontium was observed in either group. A

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chronic NOAEL of 263 mg/kg-day was identified from this study.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

UF -- The uncertainty factor of 300 includes 10 for species-to-species extrapolation and 10 for an incomplete data base (including a lack of developmental and reproductive data) and to account for uncertainties in using data for strontium carbonate to derive a risk estimate that may apply to other salts of strontium. An uncertainty factor of 3 was applied for sensitive subpopulations; a factor of 10 was not warranted because the critical study was performed in young animals, a recognized sensitive subpopulation.

MF -- None

___I.A.4. ADDITIONAL STUDIES / COMMENTS (ORAL RfD)

Pertinent data to derive an oral RfD based on the toxicity of stable strontium in humans were not located in the available literature. Estimates of dietary strontium intake range from 0.98-2.2 mg/day for adults, with milk providing about one-third of this (Snyder et al., 1975). Absorption of strontium from the gastrointestinal tract varies greatly, ranging from 9-63% (average of 38%) (Snyder et al., 1975). The bioavailability of strontium was estimated to be 20% in 6 healthy adult males administered 2.5 mmol of strontium chloride (Leeuwenkamp et al., 1990). Deficiency of dietary calcium leads to an increased absorption of strontium (Stokinger, 1981).

Use of strontium in the treatment of patients with osteoporosis has been reported. McCaslin and Janes (1959) reported treating 72 patients with daily doses of 1.7 g strontium (as strontium lactate) for periods ranging from 3 months to 3 years. Of the 32 patients who were available for follow-up, 84% experienced marked improvement. Assuming an average body weight of 70 kg, the supplementation to these patients was about 24 mg Sr/kg/day. Skoryna (1981) also reported subjective improvement in patients with osteoporosis receiving 274-1750 mg Sr/day as the gluconate, carbonate or lactate. No adverse side effects were reported in either study. Although these cases have been reported, strontium is not recognized as a standard therapy for osteoporosis (Krane, 1977).

Ingested strontium is distributed in the body in three compartments: plasma extracellular fluid; soft tissue and superficial zone of bone tissue; and bone itself (El Solh and Rousselet, 1981). The average adult is estimated to have a body burden of 320 mg strontium, 99% of which is in the bones (Snyder et al., 1975; Stokinger, 1981). The toxic effect of excessive strontium intakes is inhibition of calcification of epiphyseal cartilage and deformities of long bones at high doses. Strontium causes adverse effects on bone by substituting for calcium in the hydroxyapatite crystal during bone calcification or by displacing calcium from existing calcified matrix (Skoryna, 1984; Kshirsagar, 1985).

As opposed to calcium, which is under homostatic regulation, strontium appears to be passively absorbed (Comar and Wasserman, 1964). However, several factors may affect the bioavailability of ingested strontium, for example, age and species, the form of strontium, and the composition of the diet, especially with regard to phosphorus, vitamin D and calcium levels. These factors are reviewed in U.S. EPA (1990, 1992).

The adequacy of calcium nutrition is a critical factor regarding strontium toxicity; rachitic changes are exacerbated by inadequate calcium levels (El Solh and Rousselet, 1981). The effect of dietary calcium on strontium toxicity was also demonstrated by Engfeldt and Hjerquist (1969). Rachitic changes were observed in weanling Sprague-Dawley rats fed a diet containing 0.95% strontium (950 mg/kg-day) and "optimal" 0.69% calcium for 4 weeks. When dietary calcium was raised to 1.6%, no rachitic changes were seen at the same dose of strontium.

Because their bones are actively growing, young animals are more sensitive than adult animals to excessive strontium intakes. In addition to the information presented in the critical study (Storey, 1961), the greater sensitivity of young animals was also demonstrated by Storey (1962). Both young (50-70 g) and adult (200-250 g) rats of both sexes (strain not specified) were provided a diet containing 1.8% strontium as strontium carbonate. The exposure continued for up to 7 months with several interim sacrifices. After only 3 weeks of exposure, the young rats exhibited a "rachitic gait" with the most obvious changes occurring in the distal end of the femur and the proximal end of the tibia. The epiphyseal plate was reported to be "grossly widened" and the "metaphysis was a mass of soft white tissue." Conversely, it was 3 months before any change was observed in the adult rats, this being the appearance of fine traverse lines in the upper tibial metaphysis. The author goes on to portray significant differences in the effects seen in young animals vs. adults provided the same dietary concentration of strontium. Because young rats consume more food per kg body weight, it is difficult to ascertain how much more sensitive young animals would be at a dose adjusted on a mg/kg bw/day basis.

Relatively little information is available regarding the potential for developmental toxicity resulting from exposure to strontium. Pregnant female Wistar rats (3/group) were administered subcutaneous doses of 0, 25, 50, 100 or 200 mg/kg of strontium nitrate (10.3, 20.7, 41.4 or 82.8 mg Sr/kg/day, respectively) during gestational days 9-19 (Lansdown et al., 1972). No effects were seen on the size or body weight of fetuses, litter sizes or the number of resorption sites. Skeletons and zones of calcification were normal and no histologic changes were seen in soft tissues. Although this study reported no teratogenic effects of strontium, the small number of dams exposed and fetuses examined preclude a definite evaluation of the results.

In addition to the information available in rats, Marie and Hott (1986) studied the effects of strontium on weanling mice. Eleven male C57BL/6J mice were provided with drinking water containing 0.27% strontium chloride from 21 to 50 days of age. Another group of 13 untreated mice served as controls. The dose of strontium was based on the earlier study by Marie et al. (1985), which determined this level of strontium to be effective in stimulating bone formation without affecting bone mineralization in rats. In mice, no significant effects were observed in bone formation parameters except an increase in the osteoid surface and a decrease in the number of osteoclasts involved in bone resorption. No effect was seen on total calcified bone volume.

Skeletal abnormalities have also been observed in dogs administered oral doses of strontium (1-3 g strontium phosphate/day) in conjunction with low levels of dietary calcium (Lehnerdt, 1910). substitute for calcium in

In addition to the effects exerted on bones, strontium can also physiologic processes such as heart and other skeletal muscle contraction, and ionic transport across red blood cell membranes and nerve cells (reviewed in U.S. EPA, 1990, 1992). However, these effects are reported following intravenous infusion of large doses of strontium, which is of questionable relevance to oral exposures.

Initially, the primary concern of most investigators was the retention and absorption of radioactive strontium from water and food sources. Radioactive

strontium is generally used as a tracer element to evaluate toxicokinetic properties (absorption, distribution and excretion). The actual dose of radioactive strontium used for this purpose is frequently unreported. The kinetics of trace amounts of radioisotopes and of stable isotopes, which are usually administered in much higher quantities, may differ.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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

Confidence in the critical studies is rated as medium because together they determine the critical effect and suggest a sensitive population but have difficulties with incomplete reporting of experimental details (e.g., number of animals, experimental protocol). The data base is rated as medium to low because although several studies exist to support these critical studies, they are all in one species and little information is available on reproductive or developmental effects. Also, little is known about the speciation of strontium (e.g., how the toxicity of $SrCO_3$ relates to other strontium compounds). The confidence in the RfD is medium, reflecting the confidence in the study and the data base.

___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

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

Other EPA Documentation -- U.S. EPA, 1990, 1992

Agency Work Group Review -- 07/18/1991, 06/23/1992

Verification Date -- 06/23/1992

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Strontium
CASRN -- 7440-24-6

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Not available at this time.

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Strontium
CASRN -- 7440-24-6

This substance/agent has not undergone a complete evaluation and determination under US EPA's IRIS program for evidence of human carcinogenic potential.

VI. BIBLIOGRAPHY

Substance Name -- Strontium
CASRN -- 7440-24-6
Last Revised -- 10/01/1992

VI.A. ORAL RfD REFERENCES

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VI.B. INHALATION RfD REFERENCES

None

00088886

_VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

None

_VII. REVISION HISTORY

Substance Name -- Strontium
CASRN -- 7440-24-6

Date	Section	Description
08/01/1991	I.A.	Oral RfD now under review
08/01/1992	I.A.6.	Work group review dates added
10/01/1992	I.A.	Oral RfD summary on-line
10/01/1992	VI.A.	Oral RfD references on-line
12/01/1996	I.A.7.	Primary contact removed

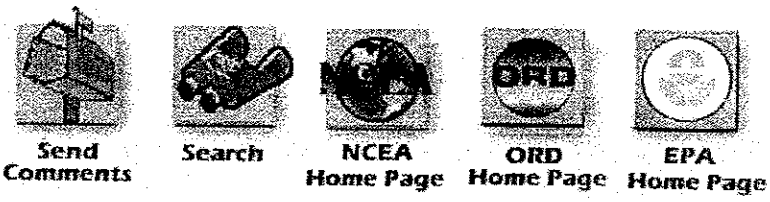
VIII. SYNONYMS

Substance Name -- Strontium
CASRN -- 7440-24-6
Last Revised -- / /

7440-24-6
strontium
stable strontium
HSDB 2545



00088887



Last updated: 5 May 1998
URL: http://www.epa.gov/iris/subst/0550.htm

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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Zinc and Compounds

CASRN 7440-66-6

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0426
Zinc and Compounds; CASRN 7440-66-6

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Zinc and Compounds

File On-Line 02/01/1991

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	on-line	10/01/1992
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	02/01/1991

I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Zinc and Compounds
CASRN -- 7440-66-6
Last Revised -- 10/01/1992

The oral Reference Dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis. It is expressed in units of mg/kg-day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Please refer to the Background Document for an elaboration of these concepts. RfDs can also be derived for the noncarcinogenic health effects of substances that are also carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

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.

I.A.1. ORAL RfD SUMMARY

Critical Effect	Experimental Doses*	UF	MF	RfD
47% Decrease in erythrocyte superoxide dismutase (ESOD) concentration in adult	NOAEL: None LOAEL = 59.72 mg/day (1.0 mg/kg/day)	3	1	3E-1 mg/kg/day

females after 10 weeks of zinc exposure

Human Diet Supplement Study

Yadrick et al., 1989

*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).

___I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

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 ($p \geq 0.05$) 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.

___I.A.3. UNCERTAINTY AND MODIFYING FACTORS (ORAL RfD)

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.

MF -- None

___I.A.4. ADDITIONAL STUDIES / COMMENTS (ORAL RfD)

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 pg/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.

___I.A.5. CONFIDENCE IN THE ORAL RfD

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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___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document -- U.S. EPA, 1990

The Drinking Water Health Advisory for Zinc has received internal Office of Water review.

Other EPA Documentation -- None

Agency Work Group Review -- 09/21/1989, 08/15/1991, 09/11/1991, 11/06/1991

Verification Date -- 11/06/1991

___I.A.7. EPA CONTACTS (ORAL RfD)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Zinc and Compounds
CASRN -- 7440-66-6

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Zinc and Compounds
CASRN -- 7440-66-6
Last Revised -- 02/01/1991

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m

air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on inadequate evidence in humans and animals.

___II.A.2. 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.

II.A.3. 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.

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

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.

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II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSU

None.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

None.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1980, 1984, 1987, 1988

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.

II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 11/08/1989, 06/15/1990

Verification Date -- 06/15/1990

II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

VI. BIBLIOGRAPHY

Substance Name -- Zinc and Compounds

00088900

CASRN -- 7440-66-6
Last Revised -- 10/01/1992

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None

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_VII. REVISION HISTORY

Substance Name -- Zinc and Compounds
CASRN -- 7440-66-6

Date	Section	Description
02/01/1991	II.	Carcinogenicity assessment on-line
02/01/1991	VI.	Bibliography on-line
01/01/1992	IV.	Regulatory Action section on-line
10/01/1992	I.A.	Oral Rfd summary on-line
10/01/1992	VI.A.	Oral RfD references on-line

VIII. SYNONYMS

Substance Name -- Zinc and Compounds
CASRN -- 7440-66-6
Last Revised -- 02/01/1991

7440-66-6
Zinc
Asarco L 15
Blue powder
Cinc [Spanish]
EMANAY ZINC DUST
GRANULAR ZINC
HSDB 1344

00088904

JASAD
Lead refinery vacuum zinc
Merrillite
UN 1436
Zinc
ZINC DUST
ZINC POWDER
ZINC, ashes
ZINC, powder or dust, non-pyrophoric
ZINC, powder or dust, pyrophoric

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