SFIM-AEC-RP-CR-97006



U.S. ARMY ENVIRONMENTAL CENTER

Tooele Army Depot

Revised Final Remedial Investigation Addendum Report for Operable Units 4, 8, and 9

Volume IV (Appendices K through R)

February 1997

Rust Environment and Infrastructure
Grand Junction, Colorado 81506

Prepared for
U.S. Army Environmental Center
Aberdeen Proving Ground, Maryland 21010-5401
under
Contract No. DAAA15-90-D-0007

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APPENDIX K

GWM-1 SPREADSHEETS AND MULTIMED MODELING
OUTPUT TABLES

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SECTION 1

Modeling for COPC Breakthrough Times

Note: All units in the MULTIMED output tables are in years for time and mg/l for concentrations.

SWMU 6

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : Antimony

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	45	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	407.6	[-] (retardation coefficient)
Vc	=	3.53E-04	[cm/day] (contaminant travel velocity)
CTt	=	63690	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	1.49E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.49E+00	[ppm] (pore water contam. conc. at WT)
AL	=	1.7E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	.199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	9.22E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	2835	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	6.06E+01	[ppm] (total soil concentration)
Slb	=	1.50E+00	[ppm] (chemical solubility)
Pwc	=	1.49E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	45	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	159.0	[-] (retardation coefficient)
T	=	14836	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : Aluminum

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1500	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	13553.9	[-] (retardation coefficient)
Vc	=	1.06E-05	[cm/day] (contaminant travel velocity)
CTt	=	2117944	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	2.08E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	2.08E+01	[ppm] (pore water contam. conc. at WT)
AL	=	2.3E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.28E+01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	94274	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.81E+04	[ppm] (total soil concentration)
Slb	=	2.09E+01	[ppm] (chemical solubility)
Pwc	=	2.08E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1500	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5268.4	[-] (retardation coefficient)
T	=	491512	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : Arsenic

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	9.52E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	9.52E+01	[ppm] (pore water contam. conc. at WT)
AL	=	1.1E+03	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	5.88E+01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	9.52E+01	[ppm] (total soil concentration)
Slb	=	9.53E+01	[ppm] (chemical solubility)
Pwc	=	9.52E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : Barium

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	60	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	543.1	[-] (retardation coefficient)
Vc	=	2.65E-04	[cm/day] (contaminant travel velocity)
CTt	=	84868	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	4.25E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	4.25E+01	[ppm] (pore water contam. conc. at WT)
AL	=	4.7E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.63E+01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	3778	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.30E+03	[ppm] (total soil concentration)
Slb	=	4.26E+01	[ppm] (chemical solubility)
Pwc	=	4.25E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	60	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	211.7	[-] (retardation coefficient)
T	=	19750	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : Boron

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	3	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	28.1	[-] (retardation coefficient)
Vc	=	5.12E-03	[cm/day] (contaminant travel velocity)
CTt	=	4392	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	7.75E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	7.75E+00	[ppm] (pore water contam. conc. at WT)
AL	=	8.6E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	4.79E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	195	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.17E+01	[ppm] (total soil concentration)
Slb	=	7.76E+00	[ppm] (chemical solubility)
Pwc	=	7.75E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	3	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	11.5	[-] (retardation coefficient)
T	=	1076	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : Cadmium

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.3	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	12.7	[-] (retardation coefficient)
Vc	=	1.13E-02	[cm/day] (contaminant travel velocity)
CTt	=	1992	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	3.66E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	3.66E+01	[ppm] (pore water contam. conc. at WT)
AL	=	4.1E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.26E+01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	89	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.65E+01	[ppm] (total soil concentration)
Slb	=	3.67E+01	[ppm] (chemical solubility)
Pwc	=	3.66E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.3	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.6	[-] (retardation coefficient)
T	=	519	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : Cromium

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.2	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	11.8	[-] (retardation coefficient)
Vc	=	1.21E-02	[cm/day] (contaminant travel velocity)
CTt	=	1850	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	1.86E+02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.86E+02	[ppm] (pore water contam. conc. at WT)
AL	=	2.1E+03	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.15E+02	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	82	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.20E+02	[ppm] (total soil concentration)
Slb	=	1.87E+02	[ppm] (chemical solubility)
Pwc	=	1.86E+02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.2	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.2	[-] (retardation coefficient)
T	=	486	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : Copper

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.4	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	13.6	[-] (retardation coefficient)
Vc	=	1.05E-02	[cm/day] (contaminant travel velocity)
CTt	=	2133	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	7.35E+03	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	7.35E+03	[ppm] (pore water contam. conc. at WT)
AL	=	8.1E+04	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	4.54E+03	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	95	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.00E+04	[ppm] (total soil concentration)
Slb	=	7.36E+03	[ppm] (chemical solubility)
Pwc	=	7.35E+03	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.4	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.9	[-] (retardation coefficient)
T	=	552	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
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Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : Fluoride

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	1.24E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.24E+01	[ppm] (pore water contam. conc. at WT)
AL	=	1.4E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	7.66E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.24E+01	[ppm] (total soil concentration)
Slb	=	1.25E+01	[ppm] (chemical solubility)
Pwc	=	1.24E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
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EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : Lead

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	4.5	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	41.7	[-] (retardation coefficient)
Vc	=	3.45E-03	[cm/day] (contaminant travel velocity)
CTt	=	6510	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	4.10E+03	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	4.10E+03	[ppm] (pore water contam. conc. at WT)
AL	=	4.5E+04	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	1.70E+04	[ppm] (total soil concentration)
Slb	=	4.11E+03	[ppm] (chemical solubility)
Pwc	=	4.10E+03	[ppm] (pore water concentration)
			(assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.53E+03	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	290	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	4.5	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	16.8	[-] (retardation coefficient)
T	=	1568	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
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EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : Mercury

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	10	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	91.4	[-] (retardation coefficient)
Vc	=	1.57E-03	[cm/day] (contaminant travel velocity)
CTt	=	14275	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	7.76E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	7.76E-02	[ppm] (pore water contam. conc. at WT)
AL	=	8.6E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	4.79E-02	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	635	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	7.06E-01	[ppm] (total soil concentration)
Slb	=	7.77E-02	[ppm] (chemical solubility)
Pwc	=	7.76E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	10	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	36.1	[-] (retardation coefficient)
T	=	3369	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : Nickel

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	150	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	1356.3	[-] (retardation coefficient)
Vc	=	1.06E-04	[cm/day] (contaminant travel velocity)
CTt	=	211935	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	8.14E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	8.14E-01	[ppm] (pore water contam. conc. at WT)
AL	=	9.0E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	5.03E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	9434	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.10E+02	[ppm] (total soil concentration)
Slb	=	8.15E-01	[ppm] (chemical solubility)
Pwc	=	8.14E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	150	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	527.7	[-] (retardation coefficient)
T	=	49235	[years] (contam. travel time)

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EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : Nitrate

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	1.14E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.14E+01	[ppm] (pore water contam. conc. at WT)
AL	=	1.3E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	7.04E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.14E+01	[ppm] (total soil concentration)
Slb	=	1.15E+01	[ppm] (chemical solubility)
Pwc	=	1.14E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

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EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : Thallium

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	3200	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	28913.9	[-] (retardation coefficient)
Vc	=	4.97E-06	[cm/day] (contaminant travel velocity)
CTt	=	4518104	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	1.20E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.20E-01	[ppm] (pore water contam. conc. at WT)
AL	=	1.3E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	7.41E-02	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	201111	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.47E+02	[ppm] (total soil concentration)
Slb	=	1.21E-01	[ppm] (chemical solubility)
Pwc	=	1.20E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	3200	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	11238.2	[-] (retardation coefficient)
T	=	1048452	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : Vanadium

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1000	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	9036.3	[-] (retardation coefficient)
Vc	=	1.59E-05	[cm/day] (contaminant travel velocity)
CTt	=	1412015	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	3.86E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	3.86E-02	[ppm] (pore water contam. conc. at WT)
AL	=	4.3E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.38E-02	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	62852	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.48E+01	[ppm] (total soil concentration)
Slb	=	3.87E-02	[ppm] (chemical solubility)
Pwc	=	3.86E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1000	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	3512.6	[-] (retardation coefficient)
T	=	327706	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : ZINC

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	1.10E+04	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.10E+04	[ppm] (pore water contam. conc. at WT)
AL	=	1.2E+05	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	6.79E+03	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.10E+04	[ppm] (total soil concentration)
Slb	=	1.11E+04	[ppm] (chemical solubility)
Pwc	=	1.10E+04	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : RDX

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	9.41E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	9.41E+00	[ppm] (pore water contam. conc. at WT)
AL	=	1.0E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	5.81E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	9.41E+00	[ppm] (total soil concentration)
Slb	=	9.42E+00	[ppm] (chemical solubility)
Pwc	=	9.41E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : 2,4-Dinitrotoluene

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	3.40E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	3.40E+01	[ppm] (pore water contam. conc. at WT)
AL	=	3.8E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.10E+01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.40E+01	[ppm] (total soil concentration)
Slb	=	3.50E+01	[ppm] (chemical solubility)
Pwc	=	3.40E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

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Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : 2,6-Dinitrotoluene

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	7.80E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	7.80E-01	[ppm] (pore water contam. conc. at WT)
AL	=	8.6E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	4.82E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	7.80E-01	[ppm] (total soil concentration)
Slb	=	7.85E-01	[ppm] (chemical solubility)
Pwc	=	7.80E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

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Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : 1,3,5-Trinitrobenzene

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	1.70E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.70E+01	[ppm] (pore water contam. conc. at WT)
AL	=	1.9E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.05E+01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.70E+01	[ppm] (total soil concentration)
Slb	=	1.71E+01	[ppm] (chemical solubility)
Pwc	=	1.70E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

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Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : 2,4,6 Trinitrotoluene

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	1.60E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.60E+01	[ppm] (pore water contam. conc. at WT)
AL	=	1.8E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	9.88E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.60E+01	[ppm] (total soil concentration)
Slb	=	1.70E+01	[ppm] (chemical solubility)
Pwc	=	1.60E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

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MULTIMED OUTPUT (SWMU 6): Antimony (mg/L)

TIME	CONCENTRATION
0.500E+02	0.00000E+00
0.205E+04	0.00000E+00
0.405E+04	0.00000E+00
0.605E+04	0.00000E+00
0.805E+04	0.00000E+00
0.100E+05	0.00000E+00
0.120E+05	0.00000E+00
0.140E+05	0.00000E+00
0.160E+05	0.00000E+00
0.180E+05	0.00000E+00
0.200E+05	0.00000E+00
0.220E+05	0.00000E+00
0.240E+05	0.00000E+00
0.260E+05	0.00000E+00
0.280E+05	0.70556E-01
0.300E+05	0.16994E+00
0.320E+05	0.25009E+00
0.340E+05	0.29788E+00
0.360E+05	0.30899E+00
0.380E+05	0.29103E+00
0.400E+05	0.25319E+00
0.420E+05	0.20491E+00
0.440E+05	0.15346E+00
0.460E+05	0.10408E+00
0.480E+05	0.59757E-01

MULTIMED OUTPUT (SWMU 6): Aluminum (mg/L)

TIME	CONCENTRATION
0.500E+04	0.00000E+00
0.860E+04	0.00000E+00
0.122E+05	0.00000E+00
0.158E+05	0.00000E+00
0.194E+05	0.00000E+00
0.230E+05	0.00000E+00
0.266E+05	0.00000E+00
0.302E+05	0.00000E+00
0.338E+05	0.00000E+00
0.374E+05	0.00000E+00
0.410E+05	0.00000E+00
0.446E+05	0.00000E+00
0.482E+05	0.00000E+00
0.518E+05	0.00000E+00
0.554E+05	0.00000E+00
0.590E+05	0.00000E+00
0.626E+05	0.00000E+00
0.662E+05	0.00000E+00
0.698E+05	0.00000E+00
0.734E+05	0.00000E+00
0.770E+05	0.00000E+00
0.806E+05	0.00000E+00
0.842E+05	0.00000E+00
0.878E+05	0.00000E+00
0.914E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 6): Arsenic (mg/L)

TIME	CONCENTRATION
0.500E+02	0.00000E+00
0.100E+03	0.00000E+00
0.150E+03	0.00000E+00
0.200E+03	0.00000E+00
0.250E+03	0.00000E+00
0.300E+03	0.00000E+00
0.350E+03	0.00000E+00
0.400E+03	0.00000E+00
0.450E+03	0.00000E+00
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.80948E+00
0.700E+03	0.78668E+01
0.750E+03	0.14149E+02
0.800E+03	0.18297E+02
0.850E+03	0.20139E+02
0.900E+03	0.19684E+02
0.950E+03	0.17569E+02
0.100E+04	0.14516E+02
0.105E+04	0.11036E+02
0.110E+04	0.76261E+01
0.115E+04	0.45373E+01
0.120E+04	0.18647E+01
0.125E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 6): Barium (mg/L)

TIME	CONCENTRATION
0.500E+02	0.00000E+00
0.205E+04	0.00000E+00
0.405E+04	0.00000E+00
0.605E+04	0.00000E+00
0.805E+04	0.00000E+00
0.100E+05	0.00000E+00
0.120E+05	0.00000E+00
0.140E+05	0.00000E+00
0.160E+05	0.00000E+00
0.180E+05	0.00000E+00
0.200E+05	0.00000E+00
0.220E+05	0.00000E+00
0.240E+05	0.00000E+00
0.260E+05	0.00000E+00
0.280E+05	0.00000E+00
0.300E+05	0.00000E+00
0.320E+05	0.17588E+01
0.340E+05	0.46372E+01
0.360E+05	0.72670E+01
0.380E+05	0.91534E+01
0.400E+05	0.10186E+02
0.420E+05	0.10398E+02
0.440E+05	0.99186E+01
0.460E+05	0.89105E+01
0.480E+05	0.75996E+01

MULTIMED OUTPUT (SWMU 6): Boron (mg/L)

TIME	CONCENTRATION
0.500E+02	0.00000E+00
0.250E+03	0.00000E+00
0.450E+03	0.00000E+00
0.650E+03	0.00000E+00
0.850E+03	0.00000E+00
0.105E+04	0.00000E+00
0.125E+04	0.00000E+00
0.145E+04	0.00000E+00
0.165E+04	0.00000E+00
0.185E+04	0.84278E-01
0.205E+04	0.85497E+00
0.225E+04	0.14258E+01
0.245E+04	0.16073E+01
0.265E+04	0.14618E+01
0.285E+04	0.11258E+01
0.305E+04	0.73710E+00
0.325E+04	0.37529E+00
0.345E+04	0.88346E-01
0.365E+04	0.00000E+00
0.385E+04	0.00000E+00
0.405E+04	0.00000E+00
0.425E+04	0.00000E+00
0.445E+04	0.00000E+00
0.465E+04	0.00000E+00
0.485E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 6): Cadmium (mg/L)

TIME	CONCENTRATION
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.00000E+00
0.850E+03	0.11894E+01
0.900E+03	0.32832E+01
0.950E+03	0.51928E+01
0.100E+04	0.66184E+01
0.105E+04	0.74643E+01
0.110E+04	0.77306E+01
0.115E+04	0.75193E+01
0.120E+04	0.69189E+01
0.125E+04	0.60474E+01
0.130E+04	0.50460E+01
0.135E+04	0.40008E+01
0.140E+04	0.29773E+01
0.145E+04	0.20349E+01
0.150E+04	0.12029E+01
0.155E+04	0.46472E+00
0.160E+04	0.00000E+00
0.165E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 6): Chromium (mg/L)

TIME	CONCENTRATION
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.86000E+01
0.850E+03	0.19655E+02
0.900E+03	0.29170E+02
0.950E+03	0.35602E+02
0.100E+04	0.38748E+02
0.105E+04	0.38547E+02
0.110E+04	0.35850E+02
0.115E+04	0.31429E+02
0.120E+04	0.26076E+02
0.125E+04	0.20404E+02
0.130E+04	0.14877E+02
0.135E+04	0.97160E+01
0.140E+04	0.52755E+01
0.145E+04	0.14767E+01
0.150E+04	0.00000E+00
0.155E+04	0.00000E+00
0.160E+04	0.00000E+00
0.165E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 6): Copper (mg/L)

TIME	CONCENTRATION
0.500E+02	0.00000E+00
0.100E+03	0.00000E+00
0.150E+03	0.00000E+00
0.200E+03	0.00000E+00
0.250E+03	0.00000E+00
0.300E+03	0.00000E+00
0.350E+03	0.00000E+00
0.400E+03	0.00000E+00
0.450E+03	0.00000E+00
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.00000E+00
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.00000E+00
0.850E+03	0.00000E+00
0.900E+03	0.14892E+02
0.950E+03	0.54446E+02
0.100E+04	0.90688E+02
0.105E+04	0.12089E+03
0.110E+04	0.14190E+03
0.115E+04	0.15282E+03
0.120E+04	0.15380E+03
0.125E+04	0.14699E+03

MULTIMED OUTPUT (SWMU 6): Fluoride (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.16059E+00
0.703E+03	0.10753E+01
0.753E+03	0.18791E+01
0.803E+03	0.24054E+01
0.853E+03	0.26248E+01
0.903E+03	0.25528E+01
0.953E+03	0.22671E+01
0.100E+04	0.18610E+01
0.105E+04	0.14102E+01
0.110E+04	0.96867E+00
0.115E+04	0.57011E+00
0.120E+04	0.22485E+00

MULTIMED OUTPUT (SWMU 6): Lead (mg/L)

TIME	CONCENTRATION
0.200E+04	0.00000E+00
0.220E+04	0.00000E+00
0.240E+04	0.00000E+00
0.280E+04	0.11735E+03
0.300E+04	0.39434E+03
0.320E+04	0.63232E+03
0.340E+04	0.78723E+03
0.360E+04	0.84977E+03
0.380E+04	0.82760E+03
0.400E+04	0.74225E+03
0.420E+04	0.61973E+03
0.440E+04	0.48166E+03
0.460E+04	0.34383E+03
0.480E+04	0.21648E+03
0.500E+04	0.10679E+03
0.520E+04	0.16289E+02
0.540E+04	0.00000E+00
0.560E+04	0.00000E+00
0.580E+04	0.00000E+00
0.600E+04	0.00000E+00
0.620E+04	0.00000E+00
0.640E+04	0.00000E+00
0.660E+04	0.00000E+00
0.680E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 6): Mercury (mg/L)

TIME	CONCENTRATION
0.500E+04	0.00000E+00
0.520E+04	0.00000E+00
0.540E+04	0.00000E+00
0.560E+04	0.00000E+00
0.580E+04	0.00000E+00
0.600E+04	0.30701E-03
0.620E+04	0.27383E-02
0.640E+04	0.51696E-02
0.660E+04	0.74719E-02
0.680E+04	0.96531E-02
0.700E+04	0.11629E-01
0.720E+04	0.13209E-01
0.740E+04	0.14464E-01
0.760E+04	0.15413E-01
0.780E+04	0.15873E-01
0.800E+04	0.16029E-01
0.820E+04	0.15884E-01
0.840E+04	0.15468E-01
0.860E+04	0.14798E-01
0.880E+04	0.13948E-01
0.900E+04	0.12949E-01
0.920E+04	0.11855E-01
0.940E+04	0.10694E-01
0.960E+04	0.95056E-02
0.980E+04	0.83040E-02

MULTIMED OUTPUT (SWMU 6): Nickel (mg/L)

TIME	CONCENTRATION
0.500E+04	0.00000E+00
0.850E+04	0.00000E+00
0.120E+05	0.00000E+00
0.155E+05	0.00000E+00
0.190E+05	0.00000E+00
0.225E+05	0.00000E+00
0.260E+05	0.00000E+00
0.295E+05	0.00000E+00
0.330E+05	0.00000E+00
0.365E+05	0.00000E+00
0.400E+05	0.00000E+00
0.435E+05	0.00000E+00
0.470E+05	0.00000E+00
0.505E+05	0.00000E+00
0.540E+05	0.00000E+00
0.575E+05	0.00000E+00
0.610E+05	0.00000E+00
0.645E+05	0.00000E+00
0.680E+05	0.00000E+00
0.715E+05	0.00000E+00
0.750E+05	0.00000E+00
0.785E+05	0.00000E+00
0.820E+05	0.00000E+00
0.855E+05	0.00000E+00
0.890E+05	0.66420E-04

MULTIMED OUTPUT (SWMU 6): Nitrate (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.14764E+00
0.703E+03	0.98862E+00
0.753E+03	0.17275E+01
0.803E+03	0.22114E+01
0.853E+03	0.24131E+01
0.903E+03	0.23469E+01
0.953E+03	0.20843E+01
0.100E+04	0.17109E+01
0.105E+04	0.12965E+01
0.110E+04	0.89055E+00
0.115E+04	0.52413E+00
0.120E+04	0.20672E+00

MULTIMED OUTPUT (SWMU 6): Thallium (mg/L)

TIME	CONCENTRATION
0.500E+04	0.00000E+00
0.850E+04	0.00000E+00
0.120E+05	0.00000E+00
0.155E+05	0.00000E+00
0.190E+05	0.00000E+00
0.225E+05	0.00000E+00
0.260E+05	0.00000E+00
0.295E+05	0.00000E+00
0.330E+05	0.00000E+00
0.365E+05	0.00000E+00
0.400E+05	0.00000E+00
0.435E+05	0.00000E+00
0.470E+05	0.00000E+00
0.505E+05	0.00000E+00
0.540E+05	0.00000E+00
0.575E+05	0.00000E+00
0.610E+05	0.00000E+00
0.645E+05	0.00000E+00
0.680E+05	0.00000E+00
0.715E+05	0.00000E+00
0.750E+05	0.00000E+00
0.785E+05	0.00000E+00
0.820E+05	0.00000E+00
0.855E+05	0.00000E+00
0.890E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 6): Vanadium (mg/L)

TIME	CONCENTRATION
0.500E+04	0.00000E+00
0.870E+04	0.00000E+00
0.124E+05	0.00000E+00
0.161E+05	0.00000E+00
0.198E+05	0.00000E+00
0.235E+05	0.00000E+00
0.272E+05	0.00000E+00
0.309E+05	0.00000E+00
0.346E+05	0.00000E+00
0.383E+05	0.00000E+00
0.420E+05	0.00000E+00
0.457E+05	0.00000E+00
0.494E+05	0.00000E+00
0.531E+05	0.00000E+00
0.568E+05	0.00000E+00
0.605E+05	0.00000E+00
0.642E+05	0.00000E+00
0.679E+05	0.00000E+00
0.716E+05	0.00000E+00
0.753E+05	0.00000E+00
0.790E+05	0.00000E+00
0.827E+05	0.00000E+00
0.864E+05	0.00000E+00
0.901E+05	0.00000E+00
0.938E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 6): Zinc (mg/L)

TIME	CONCENTRATION
0.500E+02	0.00000E+00
0.100E+03	0.00000E+00
0.150E+03	0.00000E+00
0.200E+03	0.00000E+00
0.250E+03	0.00000E+00
0.300E+03	0.00000E+00
0.350E+03	0.00000E+00
0.400E+03	0.00000E+00
0.450E+03	0.00000E+00
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.93532E+02
0.700E+03	0.90897E+03
0.750E+03	0.16348E+04
0.800E+03	0.21141E+04
0.850E+03	0.23270E+04
0.900E+03	0.22744E+04
0.950E+03	0.20300E+04
0.100E+04	0.16773E+04
0.105E+04	0.12752E+04
0.110E+04	0.88116E+03
0.115E+04	0.52427E+03
0.120E+04	0.21546E+03
0.125E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 6): RDX (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.800E+01	0.00000E+00
0.130E+02	0.00000E+00
0.180E+02	0.00000E+00
0.230E+02	0.00000E+00
0.280E+02	0.00000E+00
0.330E+02	0.00000E+00
0.380E+02	0.00000E+00
0.430E+02	0.00000E+00
0.480E+02	0.00000E+00
0.530E+02	0.00000E+00
0.580E+02	0.60982E+00
0.630E+02	0.20394E+01
0.680E+02	0.37608E+01
0.730E+02	0.58465E+01
0.780E+02	0.79395E+01
0.830E+02	0.96966E+01
0.880E+02	0.11069E+02
0.930E+02	0.12196E+02
0.980E+02	0.12961E+02
0.103E+03	0.13435E+02
0.108E+03	0.13704E+02
0.113E+03	0.14073E+02
0.118E+03	0.14360E+02
0.123E+03	0.14175E+02

MULTIMED OUTPUT (SWMU 6): 2,4-Dinitrotoluene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.130E+02	0.00000E+00
0.230E+02	0.00000E+00
0.330E+02	0.00000E+00
0.430E+02	0.00000E+00
0.530E+02	0.00000E+00
0.630E+02	0.00000E+00
0.730E+02	0.51869E+00
0.830E+02	0.30375E+01
0.930E+02	0.63233E+01
0.103E+03	0.96587E+01
0.113E+03	0.11960E+02
0.123E+03	0.13609E+02
0.133E+03	0.14744E+02
0.143E+03	0.14271E+02
0.153E+03	0.11794E+02
0.163E+03	0.83065E+01
0.173E+03	0.50576E+01
0.183E+03	0.25978E+01
0.193E+03	0.90078E+00
0.203E+03	0.38963E-01
0.213E+03	0.00000E+00
0.223E+03	0.00000E+00
0.233E+03	0.00000E+00
0.243E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 6): 2,6-Dinitrotoluene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.130E+02	0.00000E+00
0.230E+02	0.00000E+00
0.330E+02	0.00000E+00
0.430E+02	0.00000E+00
0.530E+02	0.00000E+00
0.630E+02	0.00000E+00
0.730E+02	0.40458E-01
0.830E+02	0.23692E+00
0.930E+02	0.49322E+00
0.103E+03	0.75338E+00
0.113E+03	0.93285E+00
0.123E+03	0.10615E+01
0.133E+03	0.11500E+01
0.143E+03	0.11131E+01
0.153E+03	0.91996E+00
0.163E+03	0.64791E+00
0.173E+03	0.39449E+00
0.183E+03	0.20263E+00
0.193E+03	0.70261E-01
0.203E+03	0.30391E-02
0.213E+03	0.00000E+00
0.223E+03	0.00000E+00
0.233E+03	0.00000E+00
0.243E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 6): 1,3,5-Trinitrobenzene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.130E+02	0.00000E+00
0.230E+02	0.00000E+00
0.330E+02	0.00000E+00
0.430E+02	0.00000E+00
0.530E+02	0.00000E+00
0.630E+02	0.00000E+00
0.730E+02	0.00000E+00
0.830E+02	0.93952E-04
0.930E+02	0.31627E+01
0.103E+03	0.76782E+01
0.113E+03	0.12629E+02
0.123E+03	0.16829E+02
0.133E+03	0.20443E+02
0.143E+03	0.23230E+02
0.153E+03	0.23884E+02
0.163E+03	0.21596E+02
0.173E+03	0.17364E+02
0.183E+03	0.12504E+02
0.193E+03	0.80710E+01
0.203E+03	0.45978E+01
0.213E+03	0.20700E+01
0.223E+03	0.47190E+00
0.233E+03	0.00000E+00
0.243E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 6): 2,4,6 Trinitrotoluene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.280E+02	0.00000E+00
0.530E+02	0.00000E+00
0.780E+02	0.00000E+00
0.103E+03	0.00000E+00
0.128E+03	0.00000E+00
0.153E+03	0.00000E+00
0.178E+03	0.00000E+00
0.203E+03	0.00000E+00
0.228E+03	0.00000E+00
0.253E+03	0.00000E+00
0.278E+03	0.00000E+00
0.303E+03	0.00000E+00
0.328E+03	0.00000E+00
0.353E+03	0.00000E+00
0.378E+03	0.12905E-03
0.403E+03	0.15534E+01
0.428E+03	0.31552E+01
0.453E+03	0.46263E+01
0.478E+03	0.57354E+01
0.503E+03	0.63234E+01
0.528E+03	0.64501E+01
0.553E+03	0.61603E+01
0.578E+03	0.55785E+01
0.603E+03	0.48247E+01

SWMU 7- Bullet Stop

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / BULLET STOP
Analyte : ARSENIC

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	429200.0	[m^2] (contaminated area of the land)
C init	=	1.76E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	17.60	[ppm] (pore water contam. conc. at WT)
AL	=	6.6E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	367	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	7613	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	0.88	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	20.0408	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.76E+01	[ppm] (total soil concentration)
Slb	=	1.77E+01	[ppm] (chemical solubility)
Pwc	=	1.76E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / BULLET STOP
Analyte : CADMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.3	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	12.7	[-] (retardation coefficient)
Vc	=	1.13E-02	[cm/day] (contaminant travel velocity)
CTt	=	1992	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	429200.0	[m^2] (contaminated area of the land)
C init	=	1.23E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.23	[ppm] (pore water contam. conc. at WT)
AL	=	4.6E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	367	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	7613	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	0.88	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.3986	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	74	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.56E+00	[ppm] (total soil concentration)
Slb	=	1.24E+00	[ppm] (chemical solubility)
Pwc	=	1.23E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.3	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.6	[-] (retardation coefficient)
T	=	519	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / BULLET STOP
Analyte : CHROMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.2	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	11.8	[-] (retardation coefficient)
Vc	=	1.21E-02	[cm/day] (contaminant travel velocity)
CTt	=	1850	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	429200.0	[m^2] (contaminated area of the land)
C init	=	2.20E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	22.03	[ppm] (pore water contam. conc. at WT)
AL	=	8.3E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	367	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	7613	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	0.88	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	25.0881	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	69	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.60E+01	[ppm] (total soil concentration)
Slb	=	2.21E+01	[ppm] (chemical solubility)
Pwc	=	2.20E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.2	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.2	[-] (retardation coefficient)
T	=	486	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / BULLET STOP
Analyte : LEAD

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	4.5	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	41.7	[-] (retardation coefficient)
Vc	=	3.45E-03	[cm/day] (contaminant travel velocity)
CTt	=	6510	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	429200.0	[m^2] (contaminated area of the land)
C init	=	7.18E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	71.79	[ppm] (pore water contam. conc. at WT)
AL	=	2.7E+03	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	367	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	7613	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	0.88	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	81.7414	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	242	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.98E+02	[ppm] (total soil concentration)
Slb	=	7.19E+01	[ppm] (chemical solubility)
Pwc	=	7.18E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	4.5	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	16.8	[-] (retardation coefficient)
T	=	1568	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / BULLET STOP
Analyte : THALLIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	3200	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	28913.9	[-] (retardation coefficient)
Vc	=	4.97E-06	[cm/day] (contaminant travel velocity)
CTt	=	4518104	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	429200.0	[m^2] (contaminated area of the land)
C init	=	1.40E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.01	[ppm] (pore water contam. conc. at WT)
AL	=	5.3E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	367	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	7613	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	0.88	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0159	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	167941	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.02E+01	[ppm] (total soil concentration)
Slb	=	1.41E-02	[ppm] (chemical solubility)
Pwc	=	1.40E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	3200	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	11238.2	[-] (retardation coefficient)
T	=	1048452	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / BULLET STOP
Analyte : VANADIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1000	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	9036.3	[-] (retardation coefficient)
Vc	=	1.59E-05	[cm/day] (contaminant travel velocity)
CTt	=	1412015	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	429200.0	[m^2] (contaminated area of the land)
C init	=	3.59E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.04	[ppm] (pore water contam. conc. at WT)
AL	=	1.4E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	367	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	7613	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	0.88	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0408	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	52486	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.23E+01	[ppm] (total soil concentration)
Slb	=	3.60E-02	[ppm] (chemical solubility)
Pwc	=	3.59E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1000	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	3512.6	[-] (retardation coefficient)
T	=	327706	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / BULLET STOP
Analyte : ZINC

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	429200.0	[m^2] (contaminated area of the land)
C init	=	9.90E+03	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	9900.00	[ppm] (pore water contam. conc. at WT)
AL	=	3.7E+05	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	367	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	7613	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	0.88	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	11272.9623	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	9.90E+03	[ppm] (total soil concentration)
Slb	=	9.91E+03	[ppm] (chemical solubility)
Pwc	=	9.90E+03	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 7, Bullet Stop): Arsenic (mg/L)

TIME	CONCENTRATION
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.27606E+00
0.700E+03	0.26829E+01
0.750E+03	0.47456E+01
0.800E+03	0.60488E+01
0.850E+03	0.65695E+01
0.900E+03	0.63362E+01
0.950E+03	0.55589E+01
0.100E+04	0.44911E+01
0.105E+04	0.33207E+01
0.110E+04	0.21845E+01
0.115E+04	0.11621E+01
0.120E+04	0.28448E+00
0.125E+04	0.00000E+00
0.130E+04	0.00000E+00
0.135E+04	0.00000E+00
0.140E+04	0.00000E+00
0.145E+04	0.00000E+00
0.150E+04	0.00000E+00
0.155E+04	0.00000E+00
0.160E+04	0.00000E+00
0.165E+04	0.00000E+00
0.170E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 7, Bullet Stop): Cadmium (mg/L)

TIME	CONCENTRATION
0.800E+03	0.00000E+00
0.850E+03	0.73974E-01
0.900E+03	0.20367E+00
0.950E+03	0.31820E+00
0.100E+04	0.40105E+00
0.105E+04	0.44761E+00
0.110E+04	0.45880E+00
0.115E+04	0.44147E+00
0.120E+04	0.40138E+00
0.125E+04	0.34560E+00
0.130E+04	0.28322E+00
0.135E+04	0.21885E+00
0.140E+04	0.15665E+00
0.145E+04	0.98558E-01
0.150E+04	0.48066E-01
0.155E+04	0.49551E-02
0.160E+04	0.00000E+00
0.165E+04	0.00000E+00
0.170E+04	0.00000E+00
0.175E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 7, Bullet Stop): Chromium (mg/L)

TIME	CONCENTRATION
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.00000E+00
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.19037E+01
0.850E+03	0.43194E+01
0.900E+03	0.63398E+01
0.950E+03	0.76552E+01
0.100E+04	0.82462E+01
0.105E+04	0.81169E+01
0.110E+04	0.74692E+01
0.115E+04	0.64500E+01
0.120E+04	0.52599E+01
0.125E+04	0.40123E+01
0.130E+04	0.28070E+01
0.135E+04	0.16947E+01
0.140E+04	0.71722E+00
0.145E+04	0.00000E+00
0.150E+04	0.00000E+00
0.155E+04	0.00000E+00
0.160E+04	0.00000E+00
0.165E+04	0.00000E+00
0.170E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 7, Bullet Stop): Lead (mg/L)

TIME	CONCENTRATION
0.250E+04	0.00000E+00
0.255E+04	0.00000E+00
0.260E+04	0.00000E+00
0.265E+04	0.00000E+00
0.270E+04	0.00000E+00
0.275E+04	0.15101E+01
0.280E+04	0.38141E+01
0.285E+04	0.61181E+01
0.290E+04	0.84221E+01
0.295E+04	0.10726E+02
0.300E+04	0.12752E+02
0.305E+04	0.14750E+02
0.310E+04	0.16747E+02
0.315E+04	0.18745E+02
0.320E+04	0.20175E+02
0.325E+04	0.21574E+02
0.330E+04	0.22973E+02
0.335E+04	0.23991E+02
0.340E+04	0.24805E+02
0.345E+04	0.25619E+02
0.350E+04	0.26003E+02
0.355E+04	0.26340E+02
0.360E+04	0.26456E+02
0.365E+04	0.26432E+02
0.370E+04	0.26222E+02

MULTIMED OUTPUT (SWMU 7, Bullet Stop): Thallium (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.390E+04	0.00000E+00
0.770E+04	0.00000E+00
0.115E+05	0.00000E+00
0.153E+05	0.00000E+00
0.191E+05	0.00000E+00
0.229E+05	0.00000E+00
0.267E+05	0.00000E+00
0.305E+05	0.00000E+00
0.343E+05	0.00000E+00
0.381E+05	0.00000E+00
0.419E+05	0.00000E+00
0.457E+05	0.00000E+00
0.495E+05	0.00000E+00
0.533E+05	0.00000E+00
0.571E+05	0.00000E+00
0.609E+05	0.00000E+00
0.647E+05	0.00000E+00
0.685E+05	0.00000E+00
0.723E+05	0.00000E+00
0.761E+05	0.00000E+00
0.799E+05	0.00000E+00
0.837E+05	0.00000E+00
0.875E+05	0.00000E+00
0.913E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 7, Bullet Stop): Vanadium (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.390E+04	0.00000E+00
0.770E+04	0.00000E+00
0.115E+05	0.00000E+00
0.153E+05	0.00000E+00
0.191E+05	0.00000E+00
0.229E+05	0.00000E+00
0.267E+05	0.00000E+00
0.305E+05	0.00000E+00
0.343E+05	0.00000E+00
0.381E+05	0.00000E+00
0.419E+05	0.00000E+00
0.457E+05	0.00000E+00
0.495E+05	0.00000E+00
0.533E+05	0.00000E+00
0.571E+05	0.00000E+00
0.609E+05	0.00000E+00
0.647E+05	0.00000E+00
0.685E+05	0.00000E+00
0.723E+05	0.00000E+00
0.761E+05	0.00000E+00
0.799E+05	0.00000E+00
0.837E+05	0.00000E+00
0.875E+05	0.00000E+00
0.913E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 7, Bullet Stop): Zinc (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.23652E+03
0.703E+03	0.15816E+04
0.753E+03	0.27190E+04
0.803E+03	0.34313E+04
0.853E+03	0.36946E+04
0.903E+03	0.35450E+04
0.953E+03	0.30937E+04
0.100E+04	0.24875E+04
0.105E+04	0.18287E+04
0.110E+04	0.11925E+04
0.115E+04	0.62260E+03
0.120E+04	0.13587E+03

SWMU 7- New Trench Area

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / NEW TRENCH AREA
Analyte : ALUMINUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1500	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	13553.9	[-] (retardation coefficient)
Vc	=	1.06E-05	[cm/day] (contaminant travel velocity)
CTt	=	2117944	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	38900.0	[m^2] (contaminated area of the land)
C init	=	3.14E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	31.39	[ppm] (pore water contam. conc. at WT)
AL	=	1.1E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	110	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	2378	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	2.92	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	10.7616	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	78726	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.24E+04	[ppm] (total soil concentration)
Slb	=	3.15E+01	[ppm] (chemical solubility)
Pwc	=	3.14E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1500	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	8500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5288.4	[-] (retardation coefficient)
T	=	491512	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / NEW TRENCH AREA
Analyte : BARIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	158	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	52	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	470.8	[-] (retardation coefficient)
Vc	=	3.05E-04	[cm/day] (contaminant travel velocity)
CTt	=	73573	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	38900.0	[m^2] (contaminated area of the land)
C init	=	7.62E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	7.62	[ppm] (pore water contam. conc. at WT)
AL	=	2.6E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	110	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	2378	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	2.92	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.6122	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	2735	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.12E+02	[ppm] (total soil concentration)
Slb	=	7.62E+00	[ppm] (chemical solubility)
Pwc	=	7.62E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	52	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	183.6	[-] (retardation coefficient)
T	=	17129	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / NEW TRENCH AREA
Analyte : CADMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.3	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	12.7	[-] (retardation coefficient)
Vc	=	1.13E-02	[cm/day] (contaminant travel velocity)
CTt	=	1992	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	38900.0	[m^2] (contaminated area of the land)
C init	=	3.91E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	3.91	[ppm] (pore water contam. conc. at WT)
AL	=	1.3E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	110	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	2378	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	2.92	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.3387	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	74	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.9600	[ppm] (total soil concentration)
Slb	=	3.92E+00	[ppm] (chemical solubility)
Pwc	=	3.91E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.3	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.6	[-] (retardation coefficient)
T	=	519	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / NEW TRENCH AREA
Analyte : CHROMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.2	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	11.8	[-] (retardation coefficient)
Vc	=	1.21E-02	[cm/day] (contaminant travel velocity)
CTt	=	1850	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	38900.0	[m^2] (contaminated area of the land)
C init	=	3.25E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	32.46	[ppm] (pore water contam. conc. at WT)
AL	=	1.1E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	110	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	2378	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	2.92	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	11.1260	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	69	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.83E+01	[ppm] (total soil concentration)
Slb	=	3.26E+01	[ppm] (chemical solubility)
Pwc	=	3.25E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.2	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.2	[-] (retardation coefficient)
T	=	486	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / NEW TRENCH AREA
Analyte : LEAD

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	4.5	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	41.7	[-] (retardation coefficient)
Vc	=	3.45E-03	[cm/day] (contaminant travel velocity)
CTt	=	6510	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	38900.0	[m^2] (contaminated area of the land)
C init	=	8.74E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	8.74	[ppm] (pore water contam. conc. at WT)
AL	=	3.0E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	110	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	2378	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	2.92	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.9976	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	242	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.63E+01	[ppm] (total soil concentration)
Slb	=	8.75E+00	[ppm] (chemical solubility)
Pwc	=	8.74E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	4.5	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	8500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	16.8	[-] (retardation coefficient)
T	=	1568	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / NEW TRENCH AREA
Analyte : MANGANESE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	38900.0	[m^2] (contaminated area of the land)
C init	=	7.32E+02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	732.00	[ppm] (pore water contam. conc. at WT)
AL	=	2.5E+03	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	110	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	2378	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	2.92	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	250.9336	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	7.32E+02	[ppm] (total soil concentration)
Slb	=	7.33E+02	[ppm] (chemical solubility)
Pwc	=	7.32E+02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	8500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / NEW TRENCH AREA
Analyte : NICKEL

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	150	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	1356.3	[-] (retardation coefficient)
Vc	=	1.06E-04	[cm/day] (contaminant travel velocity)
CTt	=	211935	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	38900.0	[m^2] (contaminated area of the land)
C init	=	2.00E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.20	[ppm] (pore water contam. conc. at WT)
AL	=	6.8E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	110	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	2378	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	2.92	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0685	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	7878	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.70E+01	[ppm] (total soil concentration)
Slb	=	2.01E-01	[ppm] (chemical solubility)
Pwc	=	2.00E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	150	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	527.7	[-] (retardation coefficient)
T	=	49235	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / NEW TRENCH AREA
Analyte : THALLIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	3200	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	28913.9	[-] (retardation coefficient)
Vc	=	4.97E-06	[cm/day] (contaminant travel velocity)
CTt	=	4518104	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	38900.0	[m^2] (contaminated area of the land)
C init	=	1.92E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.02	[ppm] (pore water contam. conc. at WT)
AL	=	6.5E-02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	110	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	2378	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	2.92	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0066	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	187941	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	5.53E+01	[ppm] (total soil concentration)
Slb	=	1.93E-02	[ppm] (chemical solubility)
Pwc	=	1.92E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	3200	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	8500	[m] (cistance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	11238.2	[-] (retardation coefficient)
T	=	1048452	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / NEW TRENCH AREA
Analyte : VANADIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1000	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	9036.3	[-] (retardation coefficient)
Vc	=	1.59E-05	[cm/day] (contaminant travel velocity)
CTt	=	1412015	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	38900.0	[m^2] (contaminated area of the land)
C init	=	5.49E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.05	[ppm] (pore water contam. conc. at WT)
AL	=	1.9E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	110	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	2378	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	2.92	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0188	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	52486	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.94E+01	[ppm] (total soil concentration)
Slb	=	5.50E-02	[ppm] (chemical solubility)
Pwc	=	5.49E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1000	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	3512.6	[-] (retardation coefficient)
T	=	327706	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 7, Trench Area): Aluminum (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.390E+04	0.00000E+00
0.770E+04	0.00000E+00
0.115E+05	0.00000E+00
0.153E+05	0.00000E+00
0.191E+05	0.00000E+00
0.229E+05	0.00000E+00
0.267E+05	0.00000E+00
0.305E+05	0.00000E+00
0.343E+05	0.00000E+00
0.381E+05	0.00000E+00
0.419E+05	0.00000E+00
0.457E+05	0.00000E+00
0.495E+05	0.00000E+00
0.533E+05	0.00000E+00
0.571E+05	0.00000E+00
0.609E+05	0.00000E+00
0.647E+05	0.00000E+00
0.685E+05	0.00000E+00
0.723E+05	0.00000E+00
0.761E+05	0.00000E+00
0.799E+05	0.00000E+00
0.837E+05	0.00000E+00
0.875E+05	0.00000E+00
0.913E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 7, Trench Area): Barium (mg/L)

TIME	CONCENTRATION
0.250E+05	0.00000E+00
0.260E+05	0.00000E+00
0.270E+05	0.00000E+00
0.280E+05	0.00000E+00
0.290E+05	0.00000E+00
0.300E+05	0.00000E+00
0.310E+05	0.19295E-01
0.320E+05	0.14819E+00
0.330E+05	0.27709E+00
0.340E+05	0.39816E+00
0.350E+05	0.50978E+00
0.360E+05	0.61447E+00
0.370E+05	0.69257E+00
0.380E+05	0.75970E+00
0.390E+05	0.80506E+00
0.400E+05	0.82976E+00
0.410E+05	0.83888E+00
0.420E+05	0.83161E+00
0.430E+05	0.81083E+00
0.440E+05	0.77681E+00
0.450E+05	0.73374E+00
0.460E+05	0.68157E+00
0.470E+05	0.62472E+00
0.480E+05	0.56415E+00
0.490E+05	0.50187E+00

MULTIMED OUTPUT (SWMU 7, Trench Area): Cadmium (mg/L)

TIME	CONCENTRATION
0.800E+03	0.00000E+00
0.850E+03	0.70808E-01
0.900E+03	0.19495E+00
0.950E+03	0.30458E+00
0.100E+04	0.38389E+00
0.105E+04	0.42845E+00
0.110E+04	0.43917E+00
0.115E+04	0.42258E+00
0.120E+04	0.38420E+00
0.125E+04	0.33081E+00
0.130E+04	0.27110E+00
0.135E+04	0.20948E+00
0.140E+04	0.14995E+00
0.145E+04	0.94341E-01
0.150E+04	0.46009E-01
0.155E+04	0.47430E-02
0.160E+04	0.00000E+00
0.165E+04	0.00000E+00
0.170E+04	0.00000E+00
0.175E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 7, Trench Area): Chromium (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.150E+03	0.00000E+00
0.200E+03	0.00000E+00
0.250E+03	0.00000E+00
0.300E+03	0.00000E+00
0.350E+03	0.00000E+00
0.400E+03	0.00000E+00
0.450E+03	0.00000E+00
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.00000E+00
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.84683E+00
0.850E+03	0.19214E+01
0.900E+03	0.28201E+01
0.950E+03	0.34052E+01
0.100E+04	0.36682E+01
0.105E+04	0.36106E+01
0.110E+04	0.33225E+01
0.115E+04	0.28692E+01
0.120E+04	0.23398E+01
0.125E+04	0.17848E+01
0.130E+04	0.12487E+01

MULTIMED OUTPUT (SWMU 7, Trench Area): Lead (mg/L)

TIME	CONCENTRATION
0.250E+04	0.00000E+00
0.255E+04	0.00000E+00
0.260E+04	0.00000E+00
0.265E+04	0.00000E+00
0.270E+04	0.00000E+00
0.275E+04	0.55351E-01
0.280E+04	0.13980E+00
0.285E+04	0.22425E+00
0.290E+04	0.30870E+00
0.295E+04	0.39315E+00
0.300E+04	0.46742E+00
0.305E+04	0.54064E+00
0.310E+04	0.61386E+00
0.315E+04	0.68707E+00
0.320E+04	0.73948E+00
0.325E+04	0.79077E+00
0.330E+04	0.84205E+00
0.335E+04	0.87937E+00
0.340E+04	0.90920E+00
0.345E+04	0.93903E+00
0.350E+04	0.95310E+00
0.355E+04	0.96546E+00
0.360E+04	0.96972E+00
0.365E+04	0.96885E+00
0.370E+04	0.96114E+00

MULTIMED OUTPUT (SWMU 7, Trench Area): Manganese (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.52659E+01
0.703E+03	0.35213E+02
0.753E+03	0.60536E+02
0.803E+03	0.76396E+02
0.853E+03	0.82259E+02
0.903E+03	0.78928E+02
0.953E+03	0.68879E+02
0.100E+04	0.55382E+02
0.105E+04	0.40715E+02
0.110E+04	0.26551E+02
0.115E+04	0.13862E+02
0.120E+04	0.30251E+01

MULTIMED OUTPUT (SWMU 7, Trench Area): Nickel (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.390E+04	0.00000E+00
0.770E+04	0.00000E+00
0.115E+05	0.00000E+00
0.153E+05	0.00000E+00
0.191E+05	0.00000E+00
0.229E+05	0.00000E+00
0.267E+05	0.00000E+00
0.305E+05	0.00000E+00
0.343E+05	0.00000E+00
0.419E+05	0.00000E+00
0.457E+05	0.00000E+00
0.495E+05	0.00000E+00
0.533E+05	0.00000E+00
0.571E+05	0.00000E+00
0.609E+05	0.00000E+00
0.647E+05	0.00000E+00
0.685E+05	0.00000E+00
0.723E+05	0.00000E+00
0.761E+05	0.00000E+00
0.799E+05	0.00000E+00
0.837E+05	0.00000E+00
0.875E+05	0.00000E+00
0.913E+05	0.28305E-02

MULTIMED OUTPUT (SWMU 7, Trench Area): Thallium (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.390E+04	0.00000E+00
0.770E+04	0.00000E+00
0.115E+05	0.00000E+00
0.153E+05	0.00000E+00
0.191E+05	0.00000E+00
0.229E+05	0.00000E+00
0.267E+05	0.00000E+00
0.305E+05	0.00000E+00
0.343E+05	0.00000E+00
0.381E+05	0.00000E+00
0.419E+05	0.00000E+00
0.457E+05	0.00000E+00
0.495E+05	0.00000E+00
0.533E+05	0.00000E+00
0.571E+05	0.00000E+00
0.609E+05	0.00000E+00
0.647E+05	0.00000E+00
0.685E+05	0.00000E+00
0.723E+05	0.00000E+00
0.761E+05	0.00000E+00
0.799E+05	0.00000E+00
0.837E+05	0.00000E+00
0.913E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 7, Trench Area): Vanadium (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.390E+04	0.00000E+00
0.770E+04	0.00000E+00
0.115E+05	0.00000E+00
0.153E+05	0.00000E+00
0.191E+05	0.00000E+00
0.229E+05	0.00000E+00
0.267E+05	0.00000E+00
0.305E+05	0.00000E+00
0.343E+05	0.00000E+00
0.381E+05	0.00000E+00
0.419E+05	0.00000E+00
0.457E+05	0.00000E+00
0.495E+05	0.00000E+00
0.533E+05	0.00000E+00
0.571E+05	0.00000E+00
0.609E+05	0.00000E+00
0.647E+05	0.00000E+00
0.685E+05	0.00000E+00
0.723E+05	0.00000E+00
0.761E+05	0.00000E+00
0.799E+05	0.00000E+00
0.837E+05	0.00000E+00
0.875E+05	0.00000E+00
0.913E+05	0.00000E+00

SWMU 7/Firing Point

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / FIRING POINT
Analyte : ARSENIC

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contamminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4600.0	[m^2] (contaminated area of the land)
C init	=	1.75E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	17.50	[ppm] (pore water contam. conc. at WT)
AL	=	7.1E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	829	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	829	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.4886	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.75E+01	[ppm] (total soil concentration)
Slb	=	1.78E+01	[ppm] (chemical solubility)
Pwc	=	1.75E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / FIRING POINT
Analyte : CADMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.3	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	12.7	[-] (retardation coefficient)
Vc	=	1.13E-02	[cm/day] (contaminant travel velocity)
CTt	=	1992	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4600.0	[m^2] (contaminated area of the land)
C init	=	1.70E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	17.01	[ppm] (pore water contam. conc. at WT)
AL	=	6.9E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0018	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	829	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	829	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.4184	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	74	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.16E+01	[ppm] (total soil concentration)
Slb	=	1.71E+01	[ppm] (chemical solubility)
Pwc	=	1.70E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.3	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.6	[-] (retardation coefficient)
T	=	519	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assessment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / FIRING POINT
Analyte : CHROMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	158	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.2	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	11.8	[-] (retardation coefficient)
Vc	=	1.21E-02	[cm/day] (contaminant travel velocity)
CTt	=	1850	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4600.0	[m^2] (contaminated area of the land)
C init	=	2.15E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	21.52	[ppm] (pore water contam. conc. at WT)
AL	=	8.7E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	829	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	829	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	3.0608	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	69	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.54E+01	[ppm] (total soil concentration)
Slb	=	2.16E+01	[ppm] (chemical solubility)
Pwc	=	2.15E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.2	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.2	[-] (retardation coefficient)
T	=	486	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / FIRING POINT
Analyte : COPPER

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.4	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	13.6	[-] (retardation coefficient)
Vc	=	1.05E-02	[cm/day] (contaminant travel velocity)
CTt	=	2133	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4600.0	[m^2] (contaminated area of the land)
C init	=	7.32E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	73.23	[ppm] (pore water contam. conc. at WT)
AL	=	3.0E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0018	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	829	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	829	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	10.4132	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	79	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	9.96E+01	[ppm] (total soil concentration)
Slb	=	7.33E+01	[ppm] (chemical solubility)
Pwc	=	7.32E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.4	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.9	[-] (retardation coefficient)
T	=	552	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / FIRING POINT
Analyte : LEAD

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	4.5	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	41.7	[-] (retardation coefficient)
Vc	=	3.45E-03	[cm/day] (contaminant travel velocity)
CTt	=	6510	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4600.0	[m^2] (contaminated area of the land)
C init	=	1.17E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	11.68	[ppm] (pore water contam. conc. at WT)
AL	=	4.7E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	829	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	829	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.6614	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	242	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.85E+01	[ppm] (total soil concentration)
Slb	=	1.18E+01	[ppm] (chemical solubility)
Pwc	=	1.17E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	4.5	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	16.8	[-] (retardation coefficient)
T	=	1568	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / FIRING POINT
Analyte : VANADIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1000	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	9036.3	[-] (retardation coefficient)
Vc	=	1.59E-05	[cm/day] (contaminant travel velocity)
CTt	=	1412015	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4600.0	[m^2] (contaminated area of the land)
C init	=	3.32E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.03	[ppm] (pore water contam. conc. at WT)
AL	=	1.3E-02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	829	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	829	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0047	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	52486	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.99E+01	[ppm] (total soil concentration)
Slb	=	3.33E-02	[ppm] (chemical solubility)
Pwc	=	3.32E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1000	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	3512.6	[-] (retardation coefficient)
T	=	327706	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 7 / FIRING POINT
Analyte : ZINC

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4600.0	[m^2] (contaminated area of the land)
C init	=	1.20E+04	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	12000.00	[ppm] (pore water contam. conc. at WT)
AL	=	4.8E+03	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	829	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	829	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.71E+03	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.20E+04	[ppm] (total soil concentration)
Sib	=	1.21E+04	[ppm] (chemical solubility)
Pwc	=	1.20E+04	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name :Tooele North - SWMU 7 / FIRING POINT
Analyte :HEXACHLOROBENZENE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.94	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	18.5	[-] (retardation coefficient)
Vc	=	7.76E-03	[cm/day] (contaminant travel velocity)
CTt	=	2895	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4600.0	[m^2] (contaminated area of the land)
C init	=	1.84E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	ERR	[1/day]
C at GW	=	0.18	[ppm] (pore water contam. conc. at WT)
AL	=	7.4E-02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	829	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	829	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.62E-02	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	304.8	[cm] (thickness of contamin. soil zone)
P. d.	=	108	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.40E-01	[ppm] (total soil concentration)
Slb	=	1.85E-01	[ppm] (chemical solubility)
Pwc	=	1.84E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.94	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	7.8	[-] (retardation coefficient)
T	=	729	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 7, Firing Point): Arsenic (mg/L)

TIME	CONCENTRATION
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.28417E-01
0.700E+03	0.27616E+00
0.750E+03	0.48849E+00
0.800E+03	0.62263E+00
0.850E+03	0.67623E+00
0.900E+03	0.65222E+00
0.950E+03	0.57220E+00
0.100E+04	0.46229E+00
0.105E+04	0.34181E+00
0.110E+04	0.22486E+00
0.115E+04	0.11963E+00
0.120E+04	0.29283E-01
0.125E+04	0.00000E+00
0.130E+04	0.00000E+00
0.135E+04	0.00000E+00
0.140E+04	0.00000E+00
0.145E+04	0.00000E+00
0.150E+04	0.00000E+00
0.155E+04	0.00000E+00
0.160E+04	0.00000E+00
0.165E+04	0.00000E+00
0.170E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 7, Firing Point): Cadmium (mg/L)

TIME	CONCENTRATION
0.800E+03	0.00000E+00
0.850E+03	0.10584E+00
0.900E+03	0.29141E+00
0.950E+03	0.45528E+00
0.100E+04	0.57383E+00
0.105E+04	0.64044E+00
0.115E+04	0.63166E+00
0.120E+04	0.57430E+00
0.125E+04	0.49449E+00
0.130E+04	0.40523E+00
0.135E+04	0.31313E+00
0.140E+04	0.22414E+00
0.150E+04	0.68774E-01
0.155E+04	0.70898E-02
0.160E+04	0.00000E+00
0.165E+04	0.00000E+00
0.170E+04	0.00000E+00
0.175E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 7, Firing Point): Copper (mg/L)

TIME	CONCENTRATION
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.00000E+00
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.00000E+00
0.850E+03	0.00000E+00
0.900E+03	0.28413E+00
0.950E+03	0.10388E+01
0.100E+04	0.17101E+01
0.105E+04	0.22569E+01
0.110E+04	0.26236E+01
0.115E+04	0.27987E+01
0.120E+04	0.27896E+01
0.125E+04	0.26393E+01
0.130E+04	0.23762E+01
0.135E+04	0.20499E+01
0.140E+04	0.16872E+01
0.145E+04	0.13193E+01
0.150E+04	0.96355E+00
0.155E+04	0.63032E+00
0.160E+04	0.33826E+00
0.165E+04	0.79785E-01
0.170E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 7, Firing Point): Lead (mg/L)

TIME	CONCENTRATION
0.250E+04	0.00000E+00
0.255E+04	0.00000E+00
0.260E+04	0.00000E+00
0.265E+04	0.00000E+00
0.270E+04	0.00000E+00
0.275E+04	0.25475E-01
0.280E+04	0.64342E-01
0.285E+04	0.10321E+00
0.290E+04	0.14208E+00
0.295E+04	0.18094E+00
0.300E+04	0.21512E+00
0.305E+04	0.24882E+00
0.310E+04	0.28252E+00
0.315E+04	0.31621E+00
0.320E+04	0.34033E+00
0.325E+04	0.36394E+00
0.330E+04	0.38754E+00
0.335E+04	0.40472E+00
0.340E+04	0.41845E+00
0.345E+04	0.43218E+00
0.350E+04	0.43865E+00
0.355E+04	0.44434E+00
0.360E+04	0.44630E+00
0.365E+04	0.44590E+00
0.370E+04	0.44235E+00

MULTIMED OUTPUT (SWMU 7, Firing Point): Vanadium (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.360E+04	0.00000E+00
0.710E+04	0.00000E+00
0.106E+05	0.00000E+00
0.141E+05	0.00000E+00
0.176E+05	0.00000E+00
0.211E+05	0.00000E+00
0.246E+05	0.00000E+00
0.281E+05	0.00000E+00
0.316E+05	0.00000E+00
0.351E+05	0.00000E+00
0.386E+05	0.00000E+00
0.456E+05	0.00000E+00
0.491E+05	0.00000E+00
0.526E+05	0.00000E+00
0.561E+05	0.00000E+00
0.596E+05	0.00000E+00
0.631E+05	0.00000E+00
0.666E+05	0.00000E+00
0.701E+05	0.00000E+00
0.736E+05	0.00000E+00
0.771E+05	0.00000E+00
0.806E+05	0.00000E+00
0.841E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 7, Firing Point): Zinc (mg/L)

TIME	CONCENTRATION
0.100E+02	0.00000E+00
0.600E+02	0.00000E+00
0.110E+03	0.00000E+00
0.160E+03	0.00000E+00
0.210E+03	0.00000E+00
0.260E+03	0.00000E+00
0.310E+03	0.00000E+00
0.360E+03	0.00000E+00
0.410E+03	0.00000E+00
0.460E+03	0.00000E+00
0.510E+03	0.00000E+00
0.560E+03	0.00000E+00
0.610E+03	0.00000E+00
0.660E+03	0.53462E+02
0.710E+03	0.21913E+03
0.760E+03	0.35568E+03
0.810E+03	0.43903E+03
0.860E+03	0.46342E+03
0.910E+03	0.43851E+03
0.960E+03	0.37850E+03
0.101E+04	0.30078E+03
0.106E+04	0.21801E+03
0.111E+04	0.13903E+03
0.116E+04	0.69017E+02
0.121E+04	0.99795E+01

MULTIMED OUTPUT (SWMU 7, Firing Point): Hexachlorobenzene (mg/L)

TIME	CONCENTRATION
0.100E+02	0.00000E+00
0.600E+02	0.00000E+00
0.110E+03	0.00000E+00
0.160E+03	0.00000E+00
0.210E+03	0.00000E+00
0.260E+03	0.00000E+00
0.310E+03	0.00000E+00
0.360E+03	0.00000E+00
0.410E+03	0.00000E+00
0.460E+03	0.00000E+00
0.510E+03	0.00000E+00
0.560E+03	0.00000E+00
0.610E+03	0.00000E+00
0.660E+03	0.00000E+00
0.710E+03	0.00000E+00
0.760E+03	0.00000E+00
0.810E+03	0.00000E+00
0.860E+03	0.00000E+00
0.910E+03	0.00000E+00
0.960E+03	0.00000E+00
0.101E+04	0.00000E+00
0.106E+04	0.00000E+00
0.111E+04	0.00000E+00
0.116E+04	0.00000E+00
0.121E+04	0.16401E-03

SWMU 8

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 8
Analyte : ALUMINUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1500	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	13553.9	[-] (retardation coefficient)
Vc	=	1.06E-05	[cm/day] (contaminant travel velocity)
CTt	=	2755394	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4528.0	[m^2] (contaminated area of the land)
C init	=	21.32	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	21.32	[ppm] (pore water contam. conc. at WT)
AL	=	8.5E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	823	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	823	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	3.0321	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	7873	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.88E+04	[ppm] (total soil concentration)
Slb	=	2.14E+01	[ppm] (chemical solubility)
Pwc	=	2.13E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1500	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5268.4	[-] (retardation coefficient)
T	=	491512	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 8
Analyte : ARSENIC

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2040	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4528.0	[m^2] (contaminated area of the land)
C init	=	27.00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	27.00	[ppm] (pore water contam. conc. at WT)
AL	=	1.1E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	823	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	823	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	3.8393	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	6	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	27.0000	[ppm] (total soil concentration)
Slb	=	2.71E+01	[ppm] (chemical solubility)
Pwc	=	2.70E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 8
Analyte : BARIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	52	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	470.8	[-] (retardation coefficient)
Vc	=	3.05E-04	[cm/day] (contaminant travel velocity)
CTt	=	95717	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4528.0	[m^2] (contaminated area of the land)
C init	=	7.78	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	7.78	[ppm] (pore water contam. conc. at WT)
AL	=	3.1E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	823	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	823	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.1062	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	273	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.65E+02	[ppm] (total soil concentration)
Slb	=	7.79E+00	[ppm] (chemical solubility)
Pwc	=	7.78E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	52	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	183.6	[-] (retardation coefficient)
T	=	17129	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 8
Analyte : BORON

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	3	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	28.1	[-] (retardation coefficient)
Vc	=	5.12E-03	[cm/day] (contaminant travel velocity)
CTt	=	5714	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4528.0	[m^2] (contaminated area of the land)
C init	=	6.89	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	6.89	[ppm] (pore water contam. conc. at WT)
AL	=	2.7E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	823	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	823	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.9799	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	16	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.93E+01	[ppm] (total soil concentration)
Slb	=	6.90E+00	[ppm] (chemical solubility)
Pwc	=	6.89E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	3	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	11.5	[-] (retardation coefficient)
T	=	1076	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 8
Analyte : CADMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.3	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	12.7	[-] (retardation coefficient)
Vc	=	1.13E-02	[cm/day] (contaminant travel velocity)
CTt	=	2591	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4528.0	[m^2] (contaminated area of the land)
C init	=	1.13	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.13	[ppm] (pore water contam. conc. at WT)
AL	=	4.5E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	823	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	823	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.1601	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	7	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.43E+00	[ppm] (total soil concentration)
Slb	=	1.14E+00	[ppm] (chemical solubility)
Pwc	=	1.13E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.3	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.6	[-] (retardation coefficient)
T	=	519	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 8
Analyte : CHROMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.2	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	11.8	[-] (retardation coefficient)
Vc	=	1.21E-02	[cm/day] (contaminant travel velocity)
CTt	=	2407	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4528.0	[m^2] (contaminated area of the land)
C init	=	34.07	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	34.07	[ppm] (pore water contam. conc. at WT)
AL	=	1.4E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	823	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	823	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	4.8440	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	7	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.02E+01	[ppm] (total soil concentration)
Slb	=	3.42E+01	[ppm] (chemical solubility)
Pwc	=	3.41E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.2	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	8500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.2	[-] (retardation coefficient)
T	=	486	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 8
Analyte : COPPER

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.4	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	13.6	[-] (retardation coefficient)
Vc	=	1.05E-02	[cm/day] (contaminant travel velocity)
CTt	=	2775	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4528.0	[m^2] (contaminated area of the land)
C init	=	1249.87	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1249.87	[ppm] (pore water contam. conc. at WT)
AL	=	5.0E+02	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	1.70E+03	[ppm] (total soil concentration)
Slb	=	1.26E+03	[ppm] (chemical solubility)
Pwc	=	1.25E+03	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	823	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	823	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	177.7254	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	8	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	1.4	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.9	[-] (retardation coefficient)
T	=	552	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 8
Analyte : NICKEL

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	150	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	1356.3	[-] (retardation coefficient)
Vc	=	1.06E-04	[cm/day] (contaminant travel velocity)
CTt	=	275722	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4528.0	[m^2] (contaminated area of the land)
C init	=	0.1776	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.18	[ppm] (pore water contam. conc. at WT)
AL	=	7.1E-02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	823	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	823	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0253	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	788	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.40E+01	[ppm] (total soil concentration)
Slb	=	1.79E-01	[ppm] (chemical solubility)
Pwc	=	1.78E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	150	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	527.7	[-] (retardation coefficient)
T	=	49235	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 8
Analyte : LEAD

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	4.5	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	41.7	[-] (retardation coefficient)
Vc	=	3.45E-03	[cm/day] (contaminant travel velocity)
CTt	=	8469	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4528.0	[m^2] (contaminated area of the land)
C init	=	7949.45	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	7949.45	[ppm] (pore water contam. conc. at WT)
AL	=	3.2E+03	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	823	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	823	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1130.3720	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	24	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.30E+04	[ppm] (total soil concentration)
Slb	=	7.96E+03	[ppm] (chemical solubility)
Pwc	=	7.95E+03	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	4.5	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	16.8	[-] (retardation coefficient)
T	=	1568	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 8
Analyte : ANTIMONY

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	45	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	407.6	[-] (retardation coefficient)
Vc	=	3.53E-04	[cm/day] (contaminant travel velocity)
CTt	=	82859	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4528.0	[m^2] (contaminated area of the land)
C init	=	3.52	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	3.52	[ppm] (pore water contam. conc. at WT)
AL	=	1.4E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	823	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	823	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.5006	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	237	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.43E+02	[ppm] (total soil concentration)
Slb	=	3.53E+00	[ppm] (chemical solubility)
Pwc	=	3.52E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	45	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	159.0	[-] (retardation coefficient)
T	=	14836	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 8
Analyte : SELENIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2040	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4528.0	[m^2] (contaminated area of the land)
C init	=	1.93	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.93	[ppm] (pore water contam. conc. at WT)
AL	=	7.7E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	823	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	823	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.2744	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	6	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.93E+00	[ppm] (total soil concentration)
Slb	=	1.94E+00	[ppm] (chemical solubility)
Pwc	=	1.93E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 8
Analyte : VANADIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1000	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	9036.3	[-] (retardation coefficient)
Vc	=	1.59E-05	[cm/day] (contaminant travel velocity)
CTt	=	1836997	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	4528.0	[m^2] (contaminated area of the land)
C init	=	0.0460	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.05	[ppm] (pore water contam. conc. at WT)
AL	=	1.8E-02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	38	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	823	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	823	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.03	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0065	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	5249	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.14E+01	[ppm] (total soil concentration)
Slb	=	4.61E-02	[ppm] (chemical solubility)
Pwc	=	4.60E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1000	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	3512.6	[-] (retardation coefficient)
T	=	327706	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 8): ALUMINUM (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.395E+04	0.00000E+00
0.790E+04	0.00000E+00
0.119E+05	0.00000E+00
0.158E+05	0.00000E+00
0.198E+05	0.00000E+00
0.237E+05	0.00000E+00
0.277E+05	0.00000E+00
0.316E+05	0.00000E+00
0.356E+05	0.00000E+00
0.395E+05	0.00000E+00
0.435E+05	0.00000E+00
0.474E+05	0.00000E+00
0.514E+05	0.00000E+00
0.553E+05	0.00000E+00
0.593E+05	0.00000E+00
0.632E+05	0.00000E+00
0.672E+05	0.00000E+00
0.711E+05	0.00000E+00
0.751E+05	0.00000E+00
0.790E+05	0.00000E+00
0.830E+05	0.00000E+00
0.869E+05	0.00000E+00
0.909E+05	0.00000E+00
0.948E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 8): Arsenic (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.54053E-04
0.853E+03	0.26964E-01
0.903E+03	0.52258E-01
0.953E+03	0.74598E-01
0.100E+04	0.90075E-01
0.105E+04	0.97764E-01
0.110E+04	0.98799E-01
0.115E+04	0.94146E-01
0.120E+04	0.85284E-01

MULTIMED OUTPUT (SWMU 8): Barium (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.395E+04	0.00000E+00
0.790E+04	0.00000E+00
0.119E+05	0.00000E+00
0.158E+05	0.00000E+00
0.198E+05	0.00000E+00
0.237E+05	0.00000E+00
0.277E+05	0.00000E+00
0.316E+05	0.00000E+00
0.356E+05	0.00000E+00
0.395E+05	0.31944E-02
0.435E+05	0.14789E-01
0.474E+05	0.23630E-01
0.514E+05	0.27046E-01
0.553E+05	0.25693E-01
0.593E+05	0.21276E-01
0.632E+05	0.15653E-01
0.672E+05	0.10140E-01
0.711E+05	0.53985E-02
0.751E+05	0.16986E-02
0.790E+05	0.00000E+00
0.830E+05	0.00000E+00
0.869E+05	0.00000E+00
0.909E+05	0.00000E+00
0.948E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 8): Boron (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.203E+03	0.00000E+00
0.403E+03	0.00000E+00
0.603E+03	0.00000E+00
0.803E+03	0.00000E+00
0.100E+04	0.00000E+00
0.120E+04	0.00000E+00
0.140E+04	0.00000E+00
0.160E+04	0.00000E+00
0.180E+04	0.00000E+00
0.200E+04	0.00000E+00
0.220E+04	0.00000E+00
0.240E+04	0.55837E-02
0.260E+04	0.14059E-01
0.300E+04	0.23472E-01
0.320E+04	0.23237E-01
0.340E+04	0.20634E-01
0.360E+04	0.16754E-01
0.380E+04	0.12488E-01
0.400E+04	0.84272E-02
0.420E+04	0.49007E-02
0.440E+04	0.20291E-02
0.460E+04	0.00000E+00
0.480E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 8): Cadmium (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.00000E+00
0.853E+03	0.00000E+00
0.903E+03	0.00000E+00
0.953E+03	0.00000E+00
0.100E+04	0.00000E+00
0.105E+04	0.46460E-03
0.110E+04	0.12655E-02
0.115E+04	0.20106E-02
0.120E+04	0.27202E-02

MULTIMED OUTPUT (SWMU 8): Chromium (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.00000E+00
0.853E+03	0.00000E+00
0.903E+03	0.00000E+00
0.953E+03	0.15879E-02
0.100E+04	0.29657E-01
0.105E+04	0.56993E-01
0.110E+04	0.81886E-01
0.115E+04	0.10127E+00
0.120E+04	0.11499E+00

MULTIMED OUTPUT (SWMU 8): Copper (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.00000E+00
0.853E+03	0.00000E+00
0.903E+03	0.00000E+00
0.953E+03	0.00000E+00
0.100E+04	0.00000E+00
0.105E+04	0.00000E+00
0.110E+04	0.91144E-01
0.115E+04	0.97197E+00
0.120E+04	0.18528E+01

MULTIMED OUTPUT (SWMU 8): Nickel (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.395E+04	0.00000E+00
0.790E+04	0.00000E+00
0.119E+05	0.00000E+00
0.158E+05	0.00000E+00
0.198E+05	0.00000E+00
0.237E+05	0.00000E+00
0.277E+05	0.00000E+00
0.316E+05	0.00000E+00
0.356E+05	0.00000E+00
0.395E+05	0.00000E+00
0.435E+05	0.00000E+00
0.474E+05	0.00000E+00
0.514E+05	0.00000E+00
0.553E+05	0.00000E+00
0.593E+05	0.00000E+00
0.632E+05	0.00000E+00
0.672E+05	0.00000E+00
0.711E+05	0.00000E+00
0.751E+05	0.00000E+00
0.790E+05	0.00000E+00
0.830E+05	0.00000E+00
0.869E+05	0.00000E+00
0.909E+05	0.00000E+00
0.948E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 8): Lead (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.203E+03	0.00000E+00
0.403E+03	0.00000E+00
0.603E+03	0.00000E+00
0.803E+03	0.00000E+00
0.100E+04	0.00000E+00
0.120E+04	0.00000E+00
0.140E+04	0.00000E+00
0.160E+04	0.00000E+00
0.180E+04	0.00000E+00
0.200E+04	0.00000E+00
0.220E+04	0.00000E+00
0.240E+04	0.00000E+00
0.260E+04	0.00000E+00
0.280E+04	0.00000E+00
0.300E+04	0.00000E+00
0.320E+04	0.00000E+00
0.340E+04	0.58151E+00
0.360E+04	0.76465E+01
0.380E+04	0.14346E+02
0.400E+04	0.20415E+02
0.420E+04	0.24718E+02
0.440E+04	0.27102E+02
0.460E+04	0.27638E+02
0.480E+04	0.26793E+02

MULTIMED OUTPUT (SWMU 8): Antimony (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.395E+04	0.00000E+00
0.790E+04	0.00000E+00
0.119E+05	0.00000E+00
0.158E+05	0.00000E+00
0.198E+05	0.00000E+00
0.237E+05	0.00000E+00
0.277E+05	0.00000E+00
0.316E+05	0.00000E+00
0.356E+05	0.36217E-02
0.395E+05	0.92332E-02
0.435E+05	0.12089E-01
0.474E+05	0.11843E-01
0.514E+05	0.96122E-02
0.553E+05	0.66553E-02
0.593E+05	0.38296E-02
0.632E+05	0.15295E-02
0.672E+05	0.00000E+00
0.711E+05	0.00000E+00
0.751E+05	0.00000E+00
0.790E+05	0.00000E+00
0.830E+05	0.00000E+00
0.869E+05	0.00000E+00
0.909E+05	0.00000E+00
0.948E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 8): Selenium (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.38638E-05
0.853E+03	0.19275E-02
0.903E+03	0.37354E-02
0.953E+03	0.53324E-02
0.100E+04	0.64387E-02
0.105E+04	0.69883E-02
0.110E+04	0.70623E-02
0.115E+04	0.67297E-02
0.120E+04	0.60962E-02

MULTIMED OUTPUT (SWMU 8): Vanadium (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.395E+04	0.00000E+00
0.790E+04	0.00000E+00
0.119E+05	0.00000E+00
0.158E+05	0.00000E+00
0.198E+05	0.00000E+00
0.237E+05	0.00000E+00
0.277E+05	0.00000E+00
0.316E+05	0.00000E+00
0.356E+05	0.00000E+00
0.395E+05	0.00000E+00
0.435E+05	0.00000E+00
0.474E+05	0.00000E+00
0.514E+05	0.00000E+00
0.553E+05	0.00000E+00
0.593E+05	0.00000E+00
0.632E+05	0.00000E+00
0.672E+05	0.00000E+00
0.711E+05	0.00000E+00
0.751E+05	0.00000E+00
0.790E+05	0.00000E+00
0.830E+05	0.00000E+00
0.869E+05	0.00000E+00
0.909E+05	0.00000E+00
0.948E+05	0.00000E+00

SWMU 13

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 13
Analyte : CHROMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt.	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.2	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	11.8	[-] (retardation coefficient)
Vc	=	1.21E-02	[cm/day] (contaminant travel velocity)
CTt	=	1850	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	119000.0	[m^2] (contaminated area of the land)
C init	=	2.25E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	2.25E+01	[ppm] (pore water contam. conc. at WT)
AL	=	2.3E+02	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	2.65E+01	[ppm] (total soil concentration)
Slb	=	2.26E+01	[ppm] (chemical solubility)
Pwc	=	2.25E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	193	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4102	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.67	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.35E+01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	82	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	1.2	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.2	[-] (retardation coefficient)
T	=	486	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 13
Analyte : LEAD

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	4.5	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	41.7	[-] (retardation coefficient)
Vc	=	3.45E-03	[cm/day] (contaminant travel velocity)
CTt	=	6510	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	119000.0	[m^2] (contaminated area of the land)
C init	=	5.54E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	5.54E+00	[ppm] (pore water contam. conc. at WT)
AL	=	5.8E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	193	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4102	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.67	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	3.32E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	290	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.30E+01	[ppm] (total soil concentration)
Slb	=	5.55E+00	[ppm] (chemical solubility)
Pwc	=	5.54E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	4.5	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	16.8	[-] (retardation coefficient)
T	=	1568	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 13
Analyte : BIS(2-ETHYLHEXY)PHTHALATE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	50	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	452.8	[-] (retardation coefficient)
Vc	=	3.18E-04	[cm/day] (contaminant travel velocity)
CTt	=	70749	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	119000.0	[m^2] (contaminated area of the land)
C init	=	4.43E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	4.43E-01	[ppm] (pore water contam. conc. at WT)
AL	=	4.6E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	193	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4102	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.67	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.66E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	3149	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.00E+01	[ppm] (total soil concentration)
Slb	=	4.44E-01	[ppm] (chemical solubility)
Pwc	=	4.43E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	50	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	176.6	[-] (retardation coefficient)
T	=	16474	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 13
Analyte : CHLOROMETHANE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	119000.0	[m^2] (contaminated area of the land)
C init	=	7.00E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	7.00E-01	[ppm] (pore water contam. conc. at WT)
AL	=	7.3E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	193	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4102	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.67	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	4.20E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	7.00E-01	[ppm] (total soil concentration)
Slb	=	7.10E-01	[ppm] (chemical solubility)
Pwc	=	7.00E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 13
Analyte : DICHLOROBENZENE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	119000.0	[m^2] (contaminated area of the land)
C init	=	3.30E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	3.30E+00	[ppm] (pore water contam. conc. at WT)
AL	=	3.4E+01	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	3.30E+00	[ppm] (total soil concentration)
Slb	=	3.31E+00	[ppm] (chemical solubility)
Pwc	=	3.30E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	193	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4102	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.67	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.98E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 13
Analyte : DIETHYL PHTHALATE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	119000.0	[m^2] (contaminated area of the land)
C init	=	6.00E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	6.00E+01	[ppm] (pore water contam. conc. at WT)
AL	=	6.3E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	193	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4102	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.67	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	3.60E+01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	6.00E+01	[ppm] (total soil concentration)
Slb	=	6.01E+01	[ppm] (chemical solubility)
Pwc	=	6.00E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 13
Analyte : 1,1,1-TRICHLOROETHANE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	119000.0	[m^2] (contaminated area of the land)
C init	=	2.10E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	2.10E-01	[ppm] (pore water contam. conc. at WT)
AL	=	2.2E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	193	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4102	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.67	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.26E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.10E-01	[ppm] (total soil concentration)
Slb	=	2.11E-01	[ppm] (chemical solubility)
Pwc	=	2.10E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 13): Chromium (mg/L)

TIME	CONCENTRATION
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.10110E+01
0.850E+03	0.23107E+01
0.900E+03	0.34292E+01
0.950E+03	0.41854E+01
0.100E+04	0.45552E+01
0.105E+04	0.45316E+01
0.110E+04	0.42145E+01
0.115E+04	0.36948E+01
0.120E+04	0.30655E+01
0.125E+04	0.23987E+01
0.130E+04	0.17489E+01
0.135E+04	0.11422E+01
0.140E+04	0.62018E+00
0.145E+04	0.17360E+00
0.150E+04	0.00000E+00
0.155E+04	0.00000E+00
0.160E+04	0.00000E+00
0.165E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 13): Lead

TIME	CONCENTRATION
0.700E+03	0.00000E+00
0.800E+03	0.00000E+00
0.900E+03	0.00000E+00
0.100E+04	0.00000E+00
0.110E+04	0.00000E+00
0.130E+04	0.00000E+00
0.140E+04	0.00000E+00
0.150E+04	0.00000E+00
0.160E+04	0.00000E+00
0.170E+04	0.00000E+00
0.180E+04	0.00000E+00
0.190E+04	0.00000E+00
0.200E+04	0.00000E+00
0.210E+04	0.00000E+00
0.220E+04	0.00000E+00
0.230E+04	0.00000E+00
0.240E+04	0.00000E+00
0.250E+04	0.00000E+00
0.260E+04	0.00000E+00
0.270E+04	0.00000E+00
0.280E+04	0.15410E+00
0.290E+04	0.34027E+00
0.300E+04	0.51783E+00
0.310E+04	0.68500E+00

MULTIMED OUTPUT (SWMU 13): bis(2-Ethylhexyl) phthalate (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.370E+04	0.00000E+00
0.730E+04	0.00000E+00
0.109E+05	0.00000E+00
0.145E+05	0.00000E+00
0.181E+05	0.00000E+00
0.217E+05	0.00000E+00
0.253E+05	0.00000E+00
0.289E+05	0.00000E+00
0.325E+05	0.38127E-01
0.361E+05	0.76297E-01
0.397E+05	0.88824E-01
0.433E+05	0.79273E-01
0.469E+05	0.57758E-01
0.505E+05	0.34007E-01
0.541E+05	0.13526E-01
0.577E+05	0.00000E+00
0.613E+05	0.00000E+00
0.649E+05	0.00000E+00
0.685E+05	0.00000E+00
0.721E+05	0.00000E+00
0.757E+05	0.00000E+00
0.793E+05	0.00000E+00
0.829E+05	0.00000E+00
0.865E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 13): Chloromethane (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.180E+02	0.00000E+00
0.330E+02	0.00000E+00
0.480E+02	0.00000E+00
0.630E+02	0.54523E-01
0.780E+02	0.42044E+00
0.930E+02	0.76905E+00
0.108E+03	0.95120E+00
0.123E+03	0.10338E+01
0.138E+03	0.85389E+00
0.153E+03	0.44281E+00
0.168E+03	0.13035E+00
0.183E+03	0.00000E+00
0.198E+03	0.00000E+00
0.213E+03	0.00000E+00
0.228E+03	0.00000E+00
0.243E+03	0.00000E+00
0.258E+03	0.00000E+00
0.273E+03	0.00000E+00
0.288E+03	0.00000E+00
0.303E+03	0.00000E+00
0.318E+03	0.00000E+00
0.333E+03	0.00000E+00
0.348E+03	0.00000E+00
0.363E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 13): Dichlorobenzene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.180E+02	0.00000E+00
0.330E+02	0.00000E+00
0.480E+02	0.00000E+00
0.630E+02	0.00000E+00
0.780E+02	0.00000E+00
0.930E+02	0.00000E+00
0.108E+03	0.38457E+00
0.123E+03	0.14600E+01
0.138E+03	0.25792E+01
0.153E+03	0.36504E+01
0.168E+03	0.41713E+01
0.183E+03	0.37995E+01
0.198E+03	0.28333E+01
0.213E+03	0.17934E+01
0.228E+03	0.92267E+00
0.243E+03	0.33770E+00
0.258E+03	0.31674E-01
0.273E+03	0.00000E+00
0.288E+03	0.00000E+00
0.303E+03	0.00000E+00
0.318E+03	0.00000E+00
0.333E+03	0.00000E+00
0.348E+03	0.00000E+00
0.363E+03	0.00000E+00

MULTIMED OUTPUT(SWMU 13): Diethyl phthalate (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.180E+02	0.00000E+00
0.330E+02	0.00000E+00
0.480E+02	0.00000E+00
0.630E+02	0.00000E+00
0.780E+02	0.59096E+01
0.930E+02	0.31272E+02
0.108E+03	0.58760E+02
0.123E+03	0.76164E+02
0.138E+03	0.85786E+02
0.153E+03	0.73417E+02
0.168E+03	0.44583E+02
0.183E+03	0.19493E+02
0.198E+03	0.48014E+01
0.213E+03	0.00000E+00
0.228E+03	0.00000E+00
0.243E+03	0.00000E+00
0.258E+03	0.00000E+00
0.273E+03	0.00000E+00
0.288E+03	0.00000E+00
0.303E+03	0.00000E+00
0.318E+03	0.00000E+00
0.333E+03	0.00000E+00
0.348E+03	0.00000E+00
0.363E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 13): 1,1,1-TCE (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.180E+02	0.00000E+00
0.330E+02	0.00000E+00
0.480E+02	0.00000E+00
0.630E+02	0.00000E+00
0.780E+02	0.00000E+00
0.930E+02	0.00000E+00
0.108E+03	0.35439E-01
0.123E+03	0.10573E+00
0.138E+03	0.17737E+00
0.153E+03	0.24348E+00
0.168E+03	0.26826E+00
0.183E+03	0.23602E+00
0.198E+03	0.16819E+00
0.213E+03	0.99916E-01
0.228E+03	0.48222E-01
0.243E+03	0.15552E-01
0.258E+03	0.00000E+00
0.273E+03	0.00000E+00
0.288E+03	0.00000E+00
0.303E+03	0.00000E+00
0.318E+03	0.00000E+00
0.333E+03	0.00000E+00
0.348E+03	0.00000E+00
0.363E+03	0.00000E+00

SWMU 22

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 22
Analyte : Cadmium

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	12000	[cm] (vadose zone thickness)
Tt	=	229	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.3	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	12.7	[-] (retardation coefficient)
Vc	=	1.13E-02	[cm/day] (contaminant travel velocity)
CTt	=	2915	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	115.0	[m^2] (contaminated area of the land)
C init	=	1.21E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.21E+00	[ppm] (pore water contam. conc. at WT)
AL	=	1.2E-02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	6	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	132	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	132	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.71E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	74	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.54E+00	[ppm] (total soil concentration)
S!b	=	1.22E+00	[ppm] (chemical solubility)
Pwc	=	1.21E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.3	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.6	[-] (retardation coefficient)
T	=	519	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 22
Analyte : CHROMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	12000	[cm] (vadose zone thickness)
Tt	=	229	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.2	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	11.8	[-] (retardation coefficient)
Vc	=	1.21E-02	[cm/day] (contaminant travel velocity)
CTt	=	2708	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	115.0	[m^2] (contaminated area of the land)
C init	=	6.27E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	6.27E+01	[ppm] (pore water contam. conc. at WT)
AL	=	6.3E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	6	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	132	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	132	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	8.86E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	69	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	7.40E+01	[ppm] (total soil concentration)
Slb	=	6.28E+01	[ppm] (chemical solubility)
Pwc	=	6.27E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.2	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.2	[-] (retardation coefficient)
T	=	486	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 22
Analyte : COPPER

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	12000	[cm] (vadose zone thickness)
Tt	=	229	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.4	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	13.6	[-] (retardation coefficient)
Vc	=	1.05E-02	[cm/day] (contaminant travel velocity)
CTt	=	3121	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	115.0	[m^2] (contaminated area of the land)
C init	=	5.44E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	5.44E+01	[ppm] (pore water contam. conc. at WT)
AL	=	5.5E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	6	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	132	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	132	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	7.68E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	79	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	7.40E+01	[ppm] (total soil concentration)
Slb	=	5.45E+01	[ppm] (chemical solubility)
Pwc	=	5.44E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.4	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.9	[-] (retardation coefficient)
T	=	552	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 22
Analyte : CYANIDE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	12000	[cm] (vadose zone thickness)
Tt	=	229	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2295	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	115.0	[m^2] (contaminated area of the land)
C init	=	1.75E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.75E+01	[ppm] (pore water contam. conc. at WT)
AL	=	1.8E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	6	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	132	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	132	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.47E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.75E+01	[ppm] (total soil concentration)
Slb	=	1.76E+01	[ppm] (chemical solubility)
Pwc	=	1.75E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
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EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 22
Analyte : LEAD

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	12000	[cm] (vadose zone thickness)
Tt	=	229	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	4.5	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	41.7	[-] (retardation coefficient)
Vc	=	3.45E-03	[cm/day] (contaminant travel velocity)
CTt	=	9526	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	115.0	[m^2] (contaminated area of the land)
C init	=	2.41E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	2.41E+01	[ppm] (pore water contam. conc. at WT)
AL	=	2.4E-01	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	1.00E+02	[ppm] (total soil concentration)
Slb	=	2.42E+01	[ppm] (chemical solubility)
Pwc	=	2.41E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	6	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	132	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	132	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	3.40E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	242	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	4.5	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	16.8	[-] (retardation coefficient)
T	=	1568	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
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EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 22
Analyte : NICKEL

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	12000	[cm] (vadose zone thickness)
Tt	=	229	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	150	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	1356.3	[-] (retardation coefficient)
Vc	=	1.06E-04	[cm/day] (contaminant travel velocity)
CTt	=	310149	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	115.0	[m^2] (contaminated area of the land)
C init	=	9.62E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	9.62E-01	[ppm] (pore water contam. conc. at WT)
AL	=	9.7E-03	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	6	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	132	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	132	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.36E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	7883	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.30E+02	[ppm] (total soil concentration)
Slb	=	9.63E-01	[ppm] (chemical solubility)
Pwc	=	9.62E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	150	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	527.7	[-] (retardation coefficient)
T	=	49235	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
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EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 22
Analyte : NITRATE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	12000	[cm] (vadose zone thickness)
Tt	=	229	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2295	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	115.0	[m^2] (contaminated area of the land)
C init	=	4.08E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	4.08E+00	[ppm] (pore water contam. conc. at WT)
AL	=	4.1E-02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	6	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	132	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	132	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	5.76E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.08E+00	[ppm] (total soil concentration)
Slb	=	4.09E+00	[ppm] (chemical solubility)
Pwc	=	4.08E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 22
Analyte : NITRITE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	12000	[cm] (vadose zone thickness)
Tt	=	229	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2295	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	115.0	[m^2] (contaminated area of the land)
C init	=	4.37E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	4.37E+00	[ppm] (pore water contam. conc. at WT)
AL	=	4.4E-02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	6	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	132	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	132	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	6.17E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.37E+00	[ppm] (total soil concentration)
Slb	=	4.38E+00	[ppm] (chemical solubility)
Pwc	=	4.37E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 22
Analyte : VANADIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	12000	[cm] (vadose zone thickness)
Tt	=	229	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1000	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	9036.3	[-] (retardation coefficient)
Vc	=	1.59E-05	[cm/day] (contaminant travel velocity)
CTt	=	2066363	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	115.0	[m^2] (contaminated area of the land)
C init	=	4.19E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	4.19E-02	[ppm] (pore water contam. conc. at WT)
AL	=	4.2E-04	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	6	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	132	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	132	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	5.92E-03	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	52520	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.77E+01	[ppm] (total soil concentration)
Slb	=	4.20E-02	[ppm] (chemical solubility)
Pwc	=	4.19E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1000	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	3512.6	[-] (retardation coefficient)
T	=	327706	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 22
Analyte : HMX

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	12000	[cm] (vadose zone thickness)
Tt	=	229	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2295	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	115.0	[m^2] (contaminated area of the land)
C init	=	5.80E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	5.80E+01	[ppm] (pore water contam. conc. at WT)
AL	=	5.8E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	6	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	132	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	132	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	8.19E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	5.80E+01	[ppm] (total soil concentration)
Slb	=	5.81E+01	[ppm] (chemical solubility)
Pwc	=	5.80E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 22
Analyte : RDX

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	12000	[cm] (vadose zone thickness)
Tt	=	229	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2295	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	115.0	[m^2] (contaminated area of the land)
C init	=	1.60E+03	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.60E+03	[ppm] (pore water contam. conc. at WT)
AL	=	1.6E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	6	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	132	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	132	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.26E+02	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.60E+03	[ppm] (total soil concentration)
Slb	=	1.61E+03	[ppm] (chemical solubility)
Pwc	=	1.60E+03	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd.	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 22
Analyte : 2,4-DINITROTOLUENE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	12000	[cm] (vadose zone thickness)
Tt	=	229	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2295	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	115.0	[m^2] (contaminated area of the land)
C init	=	3.53E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	3.53E+00	[ppm] (pore water contam. conc. at WT)
AL	=	3.6E-02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	6	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	132	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	132	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	4.99E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.53E+00	[ppm] (total soil concentration)
Slb	=	3.54E+00	[ppm] (chemical solubility)
Pwc	=	3.53E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 22
Analyte : 1,3,5-TRINITROBENZENE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	12000	[cm] (vadose zone thickness)
Tt	=	229	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2295	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	115.0	[m^2] (contaminated area of the land)
C init	=	2.96E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	2.96E+00	[ppm] (pore water contam. conc. at WT)
AL	=	3.0E-02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	6	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	132	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	132	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	4.18E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.96E+00	[ppm] (total soil concentration)
Slb	=	2.97E+00	[ppm] (chemical solubility)
Pwc	=	2.96E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 22): Cadmium (mg/L)

TIME	CONCENTRATION
0.500E+02	0.00000E+00
0.150E+03	0.00000E+00
0.250E+03	0.00000E+00
0.350E+03	0.00000E+00
0.450E+03	0.00000E+00
0.550E+03	0.00000E+00
0.650E+03	0.00000E+00
0.750E+03	0.00000E+00
0.850E+03	0.00000E+00
0.950E+03	0.00000E+00
0.105E+04	0.00000E+00
0.115E+04	0.00000E+00
0.125E+04	0.11694E-01
0.135E+04	0.26919E-01
0.145E+04	0.38257E-01
0.155E+04	0.43496E-01
0.165E+04	0.42847E-01
0.175E+04	0.38155E-01
0.185E+04	0.31232E-01
0.195E+04	0.23632E-01
0.205E+04	0.16458E-01
0.215E+04	0.10236E-01
0.225E+04	0.51833E-02
0.235E+04	0.15423E-02
0.245E+04	0.11528E-02

MULTIMED OUTPUT (SWMU 22): Chromium (mg/L)

TIME	CONCENTRATION
0.500E+02	0.00000E+00
0.150E+03	0.00000E+00
0.250E+03	0.00000E+00
0.350E+03	0.00000E+00
0.450E+03	0.00000E+00
0.550E+03	0.00000E+00
0.650E+03	0.00000E+00
0.750E+03	0.00000E+00
0.850E+03	0.00000E+00
0.950E+03	0.00000E+00
0.105E+04	0.00000E+00
0.115E+04	0.52078E+00
0.125E+04	0.13795E+01
0.135E+04	0.20105E+01
0.145E+04	0.22720E+01
0.155E+04	0.21982E+01
0.165E+04	0.18944E+01
0.175E+04	0.14869E+01
0.185E+04	0.10642E+01
0.195E+04	0.67902E+00
0.205E+04	0.36156E+00
0.215E+04	0.12439E+00
0.225E+04	0.66202E-01
0.235E+04	0.44367E-01
0.245E+04	0.22531E-01

MULTIMED OUTPUT (SWMU 22): Copper (mg/L)

TIME	CONCENTRATION
0.500E+02	0.00000E+00
0.150E+03	0.00000E+00
0.250E+03	0.00000E+00
0.350E+03	0.00000E+00
0.450E+03	0.00000E+00
0.550E+03	0.00000E+00
0.650E+03	0.00000E+00
0.750E+03	0.00000E+00
0.850E+03	0.00000E+00
0.950E+03	0.00000E+00
0.105E+04	0.00000E+00
0.115E+04	0.00000E+00
0.125E+04	0.00000E+00
0.135E+04	0.58911E+00
0.145E+04	0.12216E+01
0.155E+04	0.16991E+01
0.165E+04	0.19293E+01
0.175E+04	0.19379E+01
0.185E+04	0.17714E+01
0.195E+04	0.14991E+01
0.205E+04	0.11857E+01
0.215E+04	0.87335E+00
0.225E+04	0.58928E+00
0.235E+04	0.35151E+00
0.245E+04	0.16433E+00

MULTIMED OUTPUT (SWMU 22): Cyanide (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.00000E+00
0.853E+03	0.00000E+00
0.903E+03	0.00000E+00
0.953E+03	0.91137E-01
0.100E+04	0.23793E+00
0.105E+04	0.37578E+00
0.110E+04	0.49130E+00
0.115E+04	0.57488E+00
0.120E+04	0.62165E+00

MULTIMEAD OUTPUT (SWMU 22): Lead (mg/L)

TIME	CONCENTRATION
0.200E+04	0.00000E+00
0.230E+04	0.00000E+00
0.260E+04	0.00000E+00
0.290E+04	0.00000E+00
0.320E+04	0.00000E+00
0.350E+04	0.00000E+00
0.380E+04	0.00000E+00
0.410E+04	0.19767E+00
0.440E+04	0.47214E+00
0.470E+04	0.69794E+00
0.500E+04	0.82940E+00
0.530E+04	0.86011E+00
0.560E+04	0.81078E+00
0.590E+04	0.70685E+00
0.620E+04	0.57696E+00
0.650E+04	0.44161E+00
0.680E+04	0.31469E+00
0.710E+04	0.20380E+00
0.770E+04	0.35827E-01
0.800E+04	0.24166E-01
0.830E+04	0.17192E-01
0.860E+04	0.10218E-01
0.890E+04	0.32444E-02
0.920E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 22): Nickel (mg/L)

TIME	CONCENTRATION
0.500E+04	0.00000E+00
0.860E+04	0.00000E+00
0.122E+05	0.00000E+00
0.158E+05	0.00000E+00
0.194E+05	0.00000E+00
0.230E+05	0.00000E+00
0.266E+05	0.00000E+00
0.302E+05	0.00000E+00
0.338E+05	0.00000E+00
0.374E+05	0.00000E+00
0.410E+05	0.00000E+00
0.446E+05	0.00000E+00
0.482E+05	0.00000E+00
0.518E+05	0.00000E+00
0.554E+05	0.00000E+00
0.590E+05	0.00000E+00
0.626E+05	0.00000E+00
0.662E+05	0.00000E+00
0.698E+05	0.00000E+00
0.734E+05	0.00000E+00
0.770E+05	0.00000E+00
0.806E+05	0.00000E+00
0.842E+05	0.00000E+00
0.878E+05	0.00000E+00
0.914E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 22): Nitrate (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.00000E+00
0.853E+03	0.00000E+00
0.903E+03	0.00000E+00
0.953E+03	0.21248E-01
0.100E+04	0.55472E-01
0.105E+04	0.87611E-01
0.110E+04	0.11454E+00
0.115E+04	0.13403E+00
0.120E+04	0.14493E+00

MULTIMED OUTPUT (SWMU 22): Nitrite (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.00000E+00
0.853E+03	0.00000E+00
0.903E+03	0.00000E+00
0.953E+03	0.22758E-01
0.100E+04	0.59414E-01
0.105E+04	0.93839E-01
0.110E+04	0.12269E+00
0.115E+04	0.14356E+00
0.120E+04	0.15524E+00

MULTIMED OUTPUT (SWMU 22): Vanadium (mg/L)

TIME	CONCENTRATION
0.500E+04	0.00000E+00
0.860E+04	0.00000E+00
0.122E+05	0.00000E+00
0.158E+05	0.00000E+00
0.194E+05	0.00000E+00
0.230E+05	0.00000E+00
0.266E+05	0.00000E+00
0.302E+05	0.00000E+00
0.338E+05	0.00000E+00
0.374E+05	0.00000E+00
0.410E+05	0.00000E+00
0.446E+05	0.00000E+00
0.482E+05	0.00000E+00
0.518E+05	0.00000E+00
0.554E+05	0.00000E+00
0.590E+05	0.00000E+00
0.626E+05	0.00000E+00
0.662E+05	0.00000E+00
0.698E+05	0.00000E+00
0.734E+05	0.00000E+00
0.770E+05	0.00000E+00
0.806E+05	0.00000E+00
0.842E+05	0.00000E+00
0.878E+05	0.00000E+00
0.914E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 22): HMX (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.130E+02	0.00000E+00
0.230E+02	0.00000E+00
0.330E+02	0.00000E+00
0.430E+02	0.00000E+00
0.530E+02	0.00000E+00
0.630E+02	0.00000E+00
0.730E+02	0.00000E+00
0.830E+02	0.00000E+00
0.930E+02	0.26914E+01
0.103E+03	0.71362E+01
0.113E+03	0.11373E+02
0.123E+03	0.15551E+02
0.133E+03	0.18730E+02
0.143E+03	0.19021E+02
0.153E+03	0.16343E+02
0.163E+03	0.12293E+02
0.173E+03	0.79106E+01
0.183E+03	0.42358E+01
0.193E+03	0.15552E+01
0.203E+03	0.00000E+00
0.213E+03	0.00000E+00
0.223E+03	0.00000E+00
0.233E+03	0.00000E+00
0.243E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 22): RDX (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.130E+02	0.00000E+00
0.230E+02	0.00000E+00
0.330E+02	0.00000E+00
0.430E+02	0.00000E+00
0.530E+02	0.00000E+00
0.630E+02	0.00000E+00
0.730E+02	0.00000E+00
0.830E+02	0.00000E+00
0.930E+02	0.74247E+02
0.103E+03	0.19686E+03
0.113E+03	0.31373E+03
0.123E+03	0.42900E+03
0.133E+03	0.51668E+03
0.143E+03	0.52472E+03
0.153E+03	0.45083E+03
0.163E+03	0.33912E+03
0.173E+03	0.21822E+03
0.183E+03	0.11685E+03
0.193E+03	0.42902E+02
0.203E+03	0.00000E+00
0.213E+03	0.00000E+00
0.223E+03	0.00000E+00
0.233E+03	0.00000E+00
0.243E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 22): 2,4-Dinitrotoluene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.130E+02	0.00000E+00
0.230E+02	0.00000E+00
0.330E+02	0.00000E+00
0.430E+02	0.00000E+00
0.530E+02	0.00000E+00
0.630E+02	0.00000E+00
0.730E+02	0.00000E+00
0.830E+02	0.00000E+00
0.930E+02	0.00000E+00
0.103E+03	0.00000E+00
0.113E+03	0.65325E-01
0.123E+03	0.23145E+00
0.133E+03	0.45609E+00
0.143E+03	0.70756E+00
0.153E+03	0.91197E+00
0.163E+03	0.10135E+01
0.173E+03	0.98774E+00
0.183E+03	0.85694E+00
0.193E+03	0.68378E+00
0.203E+03	0.48672E+00
0.213E+03	0.31667E+00
0.223E+03	0.17696E+00
0.233E+03	0.74273E-01
0.243E+03	0.36457E-02

MULTIMED OUTPUT (SWMU 22): 1,3,5-Trinitrobenzene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.130E+02	0.00000E+00
0.230E+02	0.00000E+00
0.330E+02	0.00000E+00
0.430E+02	0.00000E+00
0.530E+02	0.00000E+00
0.630E+02	0.00000E+00
0.730E+02	0.00000E+00
0.830E+02	0.00000E+00
0.930E+02	0.00000E+00
0.103E+03	0.00000E+00
0.113E+03	0.00000E+00
0.123E+03	0.00000E+00
0.133E+03	0.47403E-01
0.143E+03	0.16979E+00
0.153E+03	0.34067E+00
0.163E+03	0.52027E+00
0.173E+03	0.67260E+00
0.183E+03	0.75404E+00
0.193E+03	0.75702E+00
0.203E+03	0.69620E+00
0.213E+03	0.59638E+00
0.223E+03	0.47304E+00
0.233E+03	0.35209E+00
0.243E+03	0.24326E+00

SWMU 23

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 23
Analyte : Barium

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	16764	[cm] (vadose zone thickness)
Tt	=	319	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	52	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	470.8	[-] (retardation coefficient)
Vc	=	3.05E-04	[cm/day] (contaminant travel velocity)
CTt	=	150412	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	25230.0	[m^2] (contaminated area of the land)
C init	=	5.54E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	5.54E+00	[ppm] (pore water contam. conc. at WT)
AL	=	1.2E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0018	[-] (avg hydraulic gradient)
Dv	=	89	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	1922	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	3.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.5299	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	90	[cm] (thickness of contamin. soil zone)
P. d.	=	808	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.60E+02	[ppm] (total soil concentration)
Slb	=	5.55E+00	[ppm] (chemical solubility)
Pwc	=	5.54E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	52	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	8500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	183.6	[-] (retardation coefficient)
T	=	17129	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 23
Analyte : CADMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	16764	[cm] (vadose zone thickness)
Tt	=	319	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.3	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	12.7	[-] (retardation coefficient)
Vc	=	1.13E-02	[cm/day] (contaminant travel velocity)
CTt	=	4072	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	25230.0	[m^2] (contaminated area of the land)
C init	=	4.13E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	4.13E+01	[ppm] (pore water contam. conc. at WT)
AL	=	9.1E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	89	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	1922	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	3.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	11.3900	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	90	[cm] (thickness of contamin. soil zone)
P. d.	=	22	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	5.24E+01	[ppm] (total soil concentration)
Slb	=	4.14E+01	[ppm] (chemical solubility)
Pwc	=	4.13E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.3	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.6	[-] (retardation coefficient)
T	=	519	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 23
Analyte : Chromium

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	16764	[cm] (vadose zone thickness)
Tt	=	319	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.2	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	11.8	[-] (retardation coefficient)
Vc	=	1.21E-02	[cm/day] (contaminant travel velocity)
CTt	=	3783	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	25230.0	[m^2] (contaminated area of the land)
C init	=	4.55E+02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	4.55E+02	[ppm] (pore water contam. conc. at WT)
AL	=	1.0E+03	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	89	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	1922	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	3.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	125.6314	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	90	[cm] (thickness of contamin. soil zone)
P. d.	=	20	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	5.37E+02	[ppm] (total soil concentration)
Slb	=	4.56E+02	[ppm] (chemical solubility)
Pwc	=	4.55E+02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.2	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.2	[-] (retardation coefficient)
T	=	486	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 23
Analyte : Copper

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	16764	[cm] (vadose zone thickness)
Tt	=	319	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.4	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	13.6	[-] (retardation coefficient)
Vc	=	1.05E-02	[cm/day] (contaminant travel velocity)
CTt	=	4360	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	25230.0	[m^2] (contaminated area of the land)
C init	=	1.25E+02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.25E+02	[ppm] (pore water contam. conc. at WT)
AL	=	2.8E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	89	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	1922	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	3.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	34.5062	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	90	[cm] (thickness of contamin. soil zone)
P. d.	=	23	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.70E+02	[ppm] (total soil concentration)
Slb	=	1.26E+02	[ppm] (chemical solubility)
Pwc	=	1.25E+02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.4	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.9	[-] (retardation coefficient)
T	=	552	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 23
Analyte : CYANIDE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	16764	[cm] (vadose zone thickness)
Tt	=	319	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	3206	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	25230.0	[m^2] (contaminated area of the land)
C init	=	4.11E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	4.11E+01	[ppm] (pore water contam. conc. at WT)
AL	=	9.1E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	89	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	1922	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	3.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	11.3468	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	90	[cm] (thickness of contamin. soil zone)
P. d.	=	17	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.11E+01	[ppm] (total soil concentration)
Slb	=	4.12E+01	[ppm] (chemical solubility)
Pwc	=	4.11E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

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EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 23
Analyte : LEAD

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	16764	[cm] (vadose zone thickness)
Tt	=	319	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	4.5	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	41.7	[-] (retardation coefficient)
Vc	=	3.45E-03	[cm/day] (contaminant travel velocity)
CTt	=	13308	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	25230.0	[m^2] (contaminated area of the land)
C init	=	2.07E+02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	2.07E+02	[ppm] (pore water contam. conc. at WT)
AL	=	4.8E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	89	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	1922	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	3.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	57.1944	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	90	[cm] (thickness of contamin. soil zone)
P. d.	=	71	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	8.60E+02	[ppm] (total soil concentration)
Slb	=	2.08E+02	[ppm] (chemical solubility)
Pwc	=	2.07E+02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	4.5	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	16.8	[-] (retardation coefficient)
T	=	1568	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
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EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 23
Analyte : NICKEL

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	16764	[cm] (vadose zone thickness)
Tt	=	319	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	150	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	1356.3	[-] (retardation coefficient)
Vc	=	1.06E-04	[cm/day] (contaminant travel velocity)
CTt	=	433278	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	25230.0	[m^2] (contaminated area of the land)
C init	=	2.22E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	2.22E-01	[ppm] (pore water contam. conc. at WT)
AL	=	4.9E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0018	[-] (avg hydraulic gradient)
Dv	=	89	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	1922	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	3.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0813	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	90	[cm] (thickness of contamin. soil zone)
P. d.	=	2326	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.00E+01	[ppm] (total soil concentration)
Slb	=	2.23E-01	[ppm] (chemical solubility)
Pwc	=	2.22E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	150	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	527.7	[-] (retardation coefficient)
T	=	49235	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assessment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 23
Analyte : VANADIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	16764	[cm] (vadose zone thickness)
Tt	=	319	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1000	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	9036.3	[-] (retardation coefficient)
Vc	=	1.59E-05	[cm/day] (contaminant travel velocity)
CTt	=	2886709	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	25230.0	[m^2] (contaminated area of the land)
C init	=	5.24E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	5.24E-02	[ppm] (pore water contam. conc. at WT)
AL	=	1.2E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	89	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	1922	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	3.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0145	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	90	[cm] (thickness of contamin. soil zone)
P. d.	=	15498	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.72E+01	[ppm] (total soil concentration)
Slb	=	5.25E-02	[ppm] (chemical solubility)
Pwc	=	5.24E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1000	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	3512.6	[-] (retardation coefficient)
T	=	327706	[years] (contam. travel time)

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Site Name : Tooele North - SWMU 23
Analyte : TOTAL PAH's

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	16764	[cm] (vadose zone thickness)
Tt	=	319	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	19	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	172.7	[-] (retardation coefficient)
Vc	=	8.33E-04	[cm/day] (contaminant travel velocity)
CTt	=	55161	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	25230.0	[m^2] (contaminated area of the land)
C init	=	3.72E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	3.72E-01	[ppm] (pore water contam. conc. at WT)
AL	=	8.2E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	89	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	1922	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	3.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.1027	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	90	[cm] (thickness of contamin. soil zone)
P. d.	=	296	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	6.40E+00	[ppm] (total soil concentration)
Slb	=	3.73E-01	[ppm] (chemical solubility)
Pwc	=	3.72E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	19	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	67.7	[-] (retardation coefficient)
T	=	6318	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
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EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 23
Analyte : TOTAL PCB's

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	16764	[cm] (vadose zone thickness)
Tt	=	319	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	204	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	1844.2	[-] (retardation coefficient)
Vc	=	7.80E-05	[cm/day] (contaminant travel velocity)
CTt	=	589143	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	465.0	[m^2] (contaminated area of the land)
C init	=	1.85E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.85E-01	[ppm] (pore water contam. conc. at WT)
AL	=	7.5E-03	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	12	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	265	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	265	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.07	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0262	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	90	[cm] (thickness of contamin. soil zone)
P. d.	=	3163	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.40E+01	[ppm] (total soil concentration)
Slb	=	1.86E-01	[ppm] (chemical solubility)
Pwc	=	1.85E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	204	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	8500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	717.4	[-] (retardation coefficient)
T	=	66926	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 23): Barium (mg/L)

TIME	CONCENTRATION
0.300E+05	0.00000E+00
0.320E+05	0.00000E+00
0.340E+05	0.00000E+00
0.360E+05	0.00000E+00
0.380E+05	0.00000E+00
0.400E+05	0.00000E+00
0.420E+05	0.00000E+00
0.440E+05	0.00000E+00
0.460E+05	0.00000E+00
0.480E+05	0.00000E+00
0.500E+05	0.00000E+00
0.520E+05	0.00000E+00
0.540E+05	0.00000E+00
0.560E+05	0.00000E+00
0.580E+05	0.00000E+00
0.600E+05	0.13232E-02
0.620E+05	0.13234E-01
0.640E+05	0.25145E-01
0.660E+05	0.36949E-01
0.680E+05	0.48545E-01
0.700E+05	0.60038E-01
0.720E+05	0.68732E-01
0.740E+05	0.77066E-01
0.760E+05	0.82520E-01
0.780E+05	0.86588E-01

MULTIMED OUTPUT (SWMU 23): Cadmium (mg/L)

TIME	CONCENTRATION
0.150E+04	0.00000E+00
0.155E+04	0.00000E+00
0.160E+04	0.15850E-01
0.165E+04	0.10118E+00
0.170E+04	0.18651E+00
0.175E+04	0.27131E+00
0.180E+04	0.35444E+00
0.185E+04	0.43757E+00
0.190E+04	0.50446E+00
0.195E+04	0.56677E+00
0.200E+04	0.61210E+00
0.205E+04	0.65065E+00
0.210E+04	0.67025E+00
0.215E+04	0.68065E+00
0.220E+04	0.68044E+00
0.225E+04	0.67029E+00
0.230E+04	0.65201E+00
0.235E+04	0.62627E+00
0.240E+04	0.59545E+00
0.245E+04	0.56033E+00
0.250E+04	0.52212E+00
0.255E+04	0.48228E+00
0.260E+04	0.44190E+00
0.265E+04	0.40128E+00
0.270E+04	0.36180E+00

MULTIMED OUTPUT (SWMU 23): Chromium (mg/L)

TIME	CONCENTRATION
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.00000E+00
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.00000E+00
0.850E+03	0.00000E+00
0.900E+03	0.00000E+00
0.950E+03	0.00000E+00
0.100E+04	0.00000E+00
0.105E+04	0.00000E+00
0.110E+04	0.00000E+00
0.115E+04	0.00000E+00
0.120E+04	0.00000E+00
0.125E+04	0.00000E+00
0.130E+04	0.00000E+00
0.135E+04	0.00000E+00
0.140E+04	0.00000E+00
0.145E+04	0.00000E+00
0.150E+04	0.47771E+00
0.155E+04	0.14707E+01
0.160E+04	0.24636E+01
0.165E+04	0.34371E+01
0.170E+04	0.44039E+01

MULTIMED OUTPUT (SWMU 23): Copper (mg/L)

TIME	CONCENTRATION
0.500E+03	0.00000E+00
0.600E+03	0.00000E+00
0.700E+03	0.00000E+00
0.800E+03	0.00000E+00
0.900E+03	0.00000E+00
0.100E+04	0.00000E+00
0.110E+04	0.00000E+00
0.120E+04	0.00000E+00
0.130E+04	0.00000E+00
0.140E+04	0.00000E+00
0.150E+04	0.00000E+00
0.160E+04	0.00000E+00
0.170E+04	0.00000E+00
0.180E+04	0.44418E+00
0.190E+04	0.90957E+00
0.200E+04	0.13616E+01
0.210E+04	0.17043E+01
0.220E+04	0.19220E+01
0.230E+04	0.20065E+01
0.240E+04	0.19868E+01
0.250E+04	0.18765E+01
0.260E+04	0.17038E+01
0.270E+04	0.14967E+01
0.280E+04	0.12746E+01
0.290E+04	0.10548E+01

MULTIMED OUTPUT (SWMU 23): Cyanide (mg/L)

TIME	CONCENTRATION
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.00000E+00
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.00000E+00
0.850E+03	0.00000E+00
0.900E+03	0.00000E+00
0.950E+03	0.00000E+00
0.100E+04	0.00000E+00
0.105E+04	0.00000E+00
0.110E+04	0.00000E+00
0.115E+04	0.00000E+00
0.120E+04	0.00000E+00
0.130E+04	0.11457E+00
0.135E+04	0.22147E+00
0.140E+04	0.32623E+00
0.145E+04	0.43033E+00
0.150E+04	0.51260E+00
0.155E+04	0.58387E+00
0.165E+04	0.65983E+00
0.170E+04	0.66949E+00

MULTIMED OUTPUT (SWMU 23): Lead (mg/L)

TIME	CONCENTRATION
0.350E+04	0.00000E+00
0.370E+04	0.00000E+00
0.390E+04	0.00000E+00
0.410E+04	0.00000E+00
0.430E+04	0.00000E+00
0.450E+04	0.00000E+00
0.470E+04	0.00000E+00
0.490E+04	0.00000E+00
0.510E+04	0.00000E+00
0.530E+04	0.92643E-01
0.550E+04	0.59711E+00
0.570E+04	0.11016E+01
0.590E+04	0.15961E+01
0.610E+04	0.20872E+01
0.630E+04	0.24905E+01
0.650E+04	0.28550E+01
0.670E+04	0.30868E+01
0.690E+04	0.32473E+01
0.710E+04	0.33239E+01
0.730E+04	0.33202E+01
0.750E+04	0.32504E+01
0.770E+04	0.31210E+01
0.790E+04	0.29465E+01
0.830E+04	0.25177E+01

MULTIMED OUTPUT (SWMU 23): Nickel (mg/L)

TIME	CONCENTRATION
0.200E+05	0.00000E+00
0.230E+05	0.00000E+00
0.260E+05	0.00000E+00
0.290E+05	0.00000E+00
0.320E+05	0.00000E+00
0.350E+05	0.00000E+00
0.380E+05	0.00000E+00
0.410E+05	0.00000E+00
0.440E+05	0.00000E+00
0.470E+05	0.00000E+00
0.500E+05	0.00000E+00
0.530E+05	0.00000E+00
0.560E+05	0.00000E+00
0.590E+05	0.00000E+00
0.620E+05	0.00000E+00
0.650E+05	0.00000E+00
0.680E+05	0.00000E+00
0.710E+05	0.00000E+00
0.740E+05	0.00000E+00
0.770E+05	0.00000E+00
0.800E+05	0.00000E+00
0.830E+05	0.00000E+00
0.860E+05	0.00000E+00
0.890E+05	0.00000E+00
0.920E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 23): Vanadium (mg/L)

TIME	CONCENTRATION
0.200E+05	0.00000E+00
0.230E+05	0.00000E+00
0.260E+05	0.00000E+00
0.290E+05	0.00000E+00
0.320E+05	0.00000E+00
0.350E+05	0.00000E+00
0.380E+05	0.00000E+00
0.410E+05	0.00000E+00
0.440E+05	0.00000E+00
0.470E+05	0.00000E+00
0.500E+05	0.00000E+00
0.530E+05	0.00000E+00
0.560E+05	0.00000E+00
0.590E+05	0.00000E+00
0.650E+05	0.00000E+00
0.680E+05	0.00000E+00
0.710E+05	0.00000E+00
0.740E+05	0.00000E+00
0.770E+05	0.00000E+00
0.800E+05	0.00000E+00
0.860E+05	0.00000E+00
0.890E+05	0.00000E+00
0.920E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 23): Total PAH's (mg/L)

TIME	CONCENTRATION
0.200E+05	0.00000E+00
0.230E+05	0.11952E-02
0.260E+05	0.43091E-02
0.290E+05	0.59167E-02
0.320E+05	0.56362E-02
0.350E+05	0.42981E-02
0.380E+05	0.27707E-02
0.410E+05	0.15211E-02
0.440E+05	0.11612E-02
0.500E+05	0.10159E-02
0.530E+05	0.94319E-03
0.560E+05	0.87052E-03
0.620E+05	0.72518E-03
0.650E+05	0.65251E-03
0.680E+05	0.57984E-03
0.710E+05	0.50717E-03
0.740E+05	0.43451E-03
0.770E+05	0.36184E-03
0.800E+05	0.28917E-03
0.830E+05	0.21650E-03
0.860E+05	0.14383E-03
0.890E+05	0.71159E-04
0.920E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 23) PCB's (mg/L)

TIME	CONCENTRATION
0.200E+05	0.00000E+00
0.230E+05	0.00000E+00
0.260E+05	0.00000E+00
0.290E+05	0.00000E+00
0.320E+05	0.00000E+00
0.350E+05	0.00000E+00
0.380E+05	0.00000E+00
0.410E+05	0.00000E+00
0.440E+05	0.00000E+00
0.470E+05	0.00000E+00
0.500E+05	0.00000E+00
0.530E+05	0.00000E+00
0.560E+05	0.00000E+00
0.590E+05	0.00000E+00
0.620E+05	0.00000E+00
0.650E+05	0.00000E+00
0.680E+05	0.00000E+00
0.710E+05	0.00000E+00
0.740E+05	0.00000E+00
0.770E+05	0.00000E+00
0.800E+05	0.00000E+00
0.830E+05	0.00000E+00
0.860E+05	0.00000E+00
0.890E+05	0.00000E+00
0.920E+05	0.00000E+00

SWMU 31

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 31
Analyte : LEAD

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	9906	[cm] (vadose zone thickness)
Tt	=	189	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	4.5	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	41.7	[-] (retardation coefficient)
Vc	=	3.45E-03	[cm/day] (contaminant travel velocity)
CTt	=	7864	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	16165.0	[m^2] (contaminated area of the land)
C init	=	9.73E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	9.73E+00	[ppm] (pore water contam. conc. at WT)
AL	=	1.4E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	71	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M L d.	=	1544	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	4.53	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.15E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.5	[cm] (thickness of contamin. soil zone)
P. d.	=	24	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.04E+01	[ppm] (total soil concentration)
Slb	=	9.74E+00	[ppm] (chemical solubility)
Pwc	=	9.73E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	4.5	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	16.8	[-] (retardation coefficient)
T	=	1568	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 31): Lead (mg/L)

TIME	CONCENTRATION
0.200E+04	0.00000E+00
0.210E+04	0.00000E+00
0.220E+04	0.00000E+00
0.230E+04	0.00000E+00
0.240E+04	0.00000E+00
0.250E+04	0.00000E+00
0.260E+04	0.00000E+00
0.270E+04	0.00000E+00
0.280E+04	0.00000E+00
0.290E+04	0.00000E+00
0.300E+04	0.00000E+00
0.310E+04	0.16228E-02
0.320E+04	0.91886E-02
0.330E+04	0.16754E-01
0.340E+04	0.24272E-01
0.350E+04	0.31283E-01
0.360E+04	0.38293E-01
0.370E+04	0.44310E-01
0.380E+04	0.49231E-01
0.390E+04	0.53500E-01
0.400E+04	0.56259E-01
0.410E+04	0.58158E-01
0.420E+04	0.59161E-01
0.440E+04	0.57910E-01

SWMU 32

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 32
Analyte : ARSENIC

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	9906	[cm] (vadose zone thickness)
Tt	=	189	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1894	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	232.0	[m^2] (contaminated area of the land)
C init	=	1.61E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.61E+01	[ppm] (pore water contam. conc. at WT)
AL	=	3.3E-01	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	1.61E+01	[ppm] (total soil concentration)
Slb	=	1.62E+01	[ppm] (chemical solubility)
Pwc	=	1.61E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	9	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	187	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	187	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.28E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 32
Analyte : CHROMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	9906	[cm] (vadose zone thickness)
Tt	=	189	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.2	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	11.8	[-] (retardation coefficient)
Vc	=	1.21E-02	[cm/day] (contaminant travel velocity)
CTt	=	2235	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	232.0	[m^2] (contaminated area of the land)
C init	=	4.58E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	4.58E+01	[ppm] (pore water contam. conc. at WT)
AL	=	9.3E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	9	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	187	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	187	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	6.47E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	69	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	5.40E+01	[ppm] (total soil concentration)
Slb	=	4.59E+01	[ppm] (chemical solubility)
Pwc	=	4.58E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.2	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.2	[-] (retardation coefficient)
T	=	486	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 32
Analyte : CADMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	9906	[cm] (vadose zone thickness)
Tt	=	189	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.3	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	12.7	[-] (retardation coefficient)
Vc	=	1.13E-02	[cm/day] (contaminant travel velocity)
CTt	=	2406	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	232.0	[m^2] (contaminated area of the land)
C init	=	3.16E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	3.16E+00	[ppm] (pore water contam. conc. at WT)
AL	=	6.4E-02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	9	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	187	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	187	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	4.46E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	74	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.01E+00	[ppm] (total soil concentration)
Slb	=	3.17E+00	[ppm] (chemical solubility)
Pwc	=	3.16E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.3	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.6	[-] (retardation coefficient)
T	=	519	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 32
Analyte : LEAD

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	9906	[cm] (vadose zone thickness)
Tt	=	189	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	4.5	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	41.7	[-] (retardation coefficient)
Vc	=	3.45E-03	[cm/day] (contaminant travel velocity)
CTt	=	7864	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	232.0	[m^2] (contaminated area of the land)
C init	=	1.70E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.70E+01	[ppm] (pore water contam. conc. at WT)
AL	=	3.5E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	9	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	187	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	187	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.40E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	242	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	7.06E+01	[ppm] (total soil concentration)
Slb	=	1.71E+01	[ppm] (chemical solubility)
Pwc	=	1.70E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	4.5	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	16.8	[-] (retardation coefficient)
T	=	1568	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 32): Arsenic (mg/L)

TIME	CONCENTRATION
0.500E+03	0.00000E+00
0.525E+03	0.00000E+00
0.550E+03	0.00000E+00
0.575E+03	0.00000E+00
0.600E+03	0.00000E+00
0.625E+03	0.00000E+00
0.650E+03	0.00000E+00
0.675E+03	0.00000E+00
0.700E+03	0.00000E+00
0.725E+03	0.00000E+00
0.750E+03	0.00000E+00
0.775E+03	0.00000E+00
0.800E+03	0.00000E+00
0.825E+03	0.73302E-01
0.850E+03	0.15986E+00
0.875E+03	0.23986E+00
0.900E+03	0.30888E+00
0.925E+03	0.37064E+00
0.950E+03	0.41648E+00
0.975E+03	0.44993E+00
0.100E+04	0.47226E+00
0.102E+04	0.48085E+00
0.105E+04	0.47750E+00
0.107E+04	0.46452E+00
0.110E+04	0.44268E+00

MULTIMED OUTPUT (SWMU 32): Chromium (mg/L)

TIME	CONCENTRATION
0.500E+03	0.00000E+00
0.525E+03	0.00000E+00
0.550E+03	0.00000E+00
0.575E+03	0.00000E+00
0.600E+03	0.00000E+00
0.625E+03	0.00000E+00
0.650E+03	0.00000E+00
0.675E+03	0.00000E+00
0.700E+03	0.00000E+00
0.725E+03	0.00000E+00
0.750E+03	0.00000E+00
0.775E+03	0.00000E+00
0.800E+03	0.00000E+00
0.825E+03	0.00000E+00
0.850E+03	0.00000E+00
0.875E+03	0.00000E+00
0.900E+03	0.00000E+00
0.925E+03	0.00000E+00
0.950E+03	0.00000E+00
0.975E+03	0.19478E+00
0.100E+04	0.40378E+00
0.102E+04	0.61197E+00
0.105E+04	0.77938E+00
0.107E+04	0.94615E+00
0.110E+04	0.10814E+01

MULTIMED OUTPUT SWMU 32: Cadmium (mg/L)

TIME	CONCENTRATION
0.500E+03	0.00000E+00
0.525E+03	0.00000E+00
0.550E+03	0.00000E+00
0.575E+03	0.00000E+00
0.600E+03	0.00000E+00
0.625E+03	0.00000E+00
0.650E+03	0.00000E+00
0.675E+03	0.00000E+00
0.700E+03	0.00000E+00
0.725E+03	0.00000E+00
0.750E+03	0.00000E+00
0.775E+03	0.00000E+00
0.800E+03	0.00000E+00
0.825E+03	0.00000E+00
0.850E+03	0.00000E+00
0.875E+03	0.00000E+00
0.900E+03	0.00000E+00
0.925E+03	0.00000E+00
0.950E+03	0.00000E+00
0.975E+03	0.00000E+00
0.100E+04	0.00000E+00
0.102E+04	0.00000E+00
0.105E+04	0.18805E+00
0.107E+04	0.38101E+00
0.110E+04	0.57397E+00

MULTIMED OUTPUT (SWMU 32): Lead (mg/L)

TIME	CONCENTRATION
0.500E+03	0.00000E+00
0.700E+03	0.00000E+00
0.900E+03	0.00000E+00
0.110E+04	0.00000E+00
0.130E+04	0.00000E+00
0.150E+04	0.00000E+00
0.170E+04	0.00000E+00
0.190E+04	0.00000E+00
0.210E+04	0.00000E+00
0.230E+04	0.00000E+00
0.250E+04	0.00000E+00
0.270E+04	0.00000E+00
0.290E+04	0.00000E+00
0.310E+04	0.00000E+00
0.330E+04	0.00000E+00
0.350E+04	0.92845E-01
0.370E+04	0.25584E+00
0.390E+04	0.38530E+00
0.410E+04	0.46562E+00
0.430E+04	0.49891E+00
0.450E+04	0.48867E+00
0.470E+04	0.44488E+00
0.490E+04	0.37758E+00
0.510E+04	0.29840E+00
0.530E+04	0.21473E+00

SWMU 35

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : ARSENIC

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2040	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	3.20E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	32.00	[ppm] (pore water contam. conc. at WT)
AL	=	4.0E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	21.0068	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	6	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	32.0000	[ppm] (total soil concentration)
Slb	=	3.21E+01	[ppm] (chemical solubility)
Pwc	=	3.20E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : CADMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.3	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	12.7	[-] (retardation coefficient)
Vc	=	1.13E-02	[cm/day] (contaminant travel velocity)
CTt	=	2591	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	1.13E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.13	[ppm] (pore water contam. conc. at WT)
AL	=	1.4E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.7391	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	7	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.4300	[ppm] (total soil concentration)
Slb	=	1.14E+00	[ppm] (chemical solubility)
Pwc	=	1.13E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.3	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	8500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	3.6	[-] (retardation coefficient)
T	=	519	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : LEAD

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	4.5	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	41.7	[-] (retardation coefficient)
Vc	=	3.45E-03	[cm/day] (contaminant travel velocity)
CTt	=	8469	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	3.13E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	31.32	[ppm] (pore water contam. conc. at WT)
AL	=	3.9E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	20.5577	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	24	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	130.0000	[ppm] (total soil concentration)
Slb	=	3.14E+01	[ppm] (chemical solubility)
Pwc	=	3.13E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	4.5	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	16.8	[-] (retardation coefficient)
T	=	1568	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : NITRATE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2040	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	2.30E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	23.00	[ppm] (pore water contam. conc. at WT)
AL	=	2.9E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	15.0986	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	6	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	23.0000	[ppm] (total soil concentration)
Slb	=	2.31E+01	[ppm] (chemical solubility)
Pwc	=	2.30E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : ALDRIN

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1600	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	14457.4	[-] (retardation coefficient)
Vc	=	9.94E-06	[cm/day] (contaminant travel velocity)
CTt	=	2939073	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	1.25E-05	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.00	[ppm] (pore water contam. conc. at WT)
AL	=	1.6E-04	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0000	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	8397	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	0.0180	[ppm] (total soil concentration)
Slb	=	1.26E-05	[ppm] (chemical solubility)
Pwc	=	1.25E-05	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1600	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5619.6	[-] (retardation coefficient)
T	=	524273	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : alpha-CHLORDANE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	500	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	4518.6	[-] (retardation coefficient)
Vc	=	3.18E-05	[cm/day] (contaminant travel velocity)
CTt	=	918600	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	2.22E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.02	[ppm] (pore water contam. conc. at WT)
AL	=	2.8E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0146	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	2625	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	10.0000	[ppm] (total soil concentration)
Slb	=	2.23E-02	[ppm] (chemical solubility)
Pwc	=	2.22E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	500	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	1756.8	[-] (retardation coefficient)
T	=	163899	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : alpha-ENDOSULFAN

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.02	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.2	[-] (retardation coefficient)
Vc	=	1.41E-02	[cm/day] (contaminant travel velocity)
CTt	=	2077	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	3.73E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.37	[ppm] (pore water contam. conc. at WT)
AL	=	4.7E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.2450	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	6	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	0.3800	[ppm] (total soil concentration)
Slb	=	3.74E-01	[ppm] (chemical solubility)
Pwc	=	3.73E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.02	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.6	[-] (retardation coefficient)
T	=	427	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : beta-BENZENEHEXACHLORIDE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.45	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	14.1	[-] (retardation coefficient)
Vc	=	1.02E-02	[cm/day] (contaminant travel velocity)
CTt	=	2867	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	1.14E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.11	[ppm] (pore water contam. conc. at WT)
AL	=	1.4E+00	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	0.1600	[ppm] (total soil concentration)
Slb	=	1.20E-01	[ppm] (chemical solubility)
Pwc	=	1.14E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0747	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	8	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	1.45	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	6.1	[-] (retardation coefficient)
T	=	568	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : p,p'-DDE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	500	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	4518.6	[-] (retardation coefficient)
Vc	=	3.18E-05	[cm/day] (contaminant travel velocity)
CTt	=	918600	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	2.89E-03	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.00	[ppm] (pore water contam. conc. at WT)
AL	=	3.6E-02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0019	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	2625	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.3000	[ppm] (total soil concentration)
Slb	=	2.90E-03	[ppm] (chemical solubility)
Pwc	=	2.89E-03	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	500	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	1756.8	[-] (retardation coefficient)
T	=	163899	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assessment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : delta-BENZENEHEXACHLORIDE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2040	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	2.40E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	2.40	[ppm] (pore water contam. conc. at WT)
AL	=	3.0E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.5755	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	6	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.4000	[ppm] (total soil concentration)
Slb	=	2.50E+00	[ppm] (chemical solubility)
Pwc	=	2.40E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : DIELDRIN

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetnc water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	17.5	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	159.1	[-] (retardation coefficient)
Vc	=	9.04E-04	[cm/day] (contaminant travel velocity)
CTt	=	32347	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	2.11E-03	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.00	[ppm] (pore water contam. conc. at WT)
AL	=	2.6E-02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0014	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	92	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	0.0335	[ppm] (total soil concentration)
Slb	=	2.12E-03	[ppm] (chemical solubility)
Pwc	=	2.11E-03	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	17.5	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	62.5	[-] (retardation coefficient)
T	=	5827	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
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EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : ENDRIN

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	4.16	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	38.6	[-] (retardation coefficient)
Vc	=	3.73E-03	[cm/day] (contaminant travel velocity)
CTt	=	7844	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	1.04E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.04	[ppm] (pore water contam. conc. at WT)
AL	=	1.3E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.6829	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	22	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.0000	[ppm] (total soil concentration)
Slb	=	1.05E+00	[ppm] (chemical solubility)
Pwc	=	1.04E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	4.16	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	15.6	[-] (retardation coefficient)
T	=	1456	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : gamma-CHLORDANE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	500	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	4518.6	[-] (retardation coefficient)
Vc	=	3.18E-05	[cm/day] (contaminant travel velocity)
CTt	=	918600	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	2.22E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.02	[ppm] (pore water contam. conc. at WT)
AL	=	2.8E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0145	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	2625	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	10.0000	[ppm] (total soil concentration)
Slb	=	2.23E-02	[ppm] (chemical solubility)
Pwc	=	2.22E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	500	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	1756.8	[-] (retardation coefficient)
T	=	163999	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : HEPTACHLOR

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	11	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	100.4	[-] (retardation coefficient)
Vc	=	1.43E-03	[cm/day] (contaminant travel velocity)
CTt	=	20408	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	1.50E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.01	[ppm] (pore water contam. conc. at WT)
AL	=	1.9E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0098	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	0.1500	[ppm] (total soil concentration)
Slb	=	1.05E+00	[ppm] (chemical solubility)
Pwc	=	1.50E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	11	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	39.6	[-] (retardation coefficient)
T	=	3697	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : HEPTACHLOR EPOXIDE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	10.5	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	95.9	[-] (retardation coefficient)
Vc	=	1.50E-03	[cm/day] (contaminant travel velocity)
CTt	=	19490	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	2.62E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.03	[ppm] (pore water contam. conc. at WT)
AL	=	3.3E-01	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	0.2500	[ppm] (total soil concentration)
Slb	=	2.63E-02	[ppm] (chemical solubility)
Pwc	=	2.62E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0172	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	56	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	10.5	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	37.9	[-] (retardation coefficient)
T	=	3533	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 35
Analyte : LINDANE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10668	[cm] (vadose zone thickness)
Tt	=	203	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2040	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	142650.0	[m^2] (contaminated area of the land)
C init	=	4.08E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.04	[ppm] (pore water contam. conc. at WT)
AL	=	5.1E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	212	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4478	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.52	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0268	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	30.48	[cm] (thickness of contamin. soil zone)
P. d.	=	6	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	0.0408	[ppm] (total soil concentration)
Slb	=	4.09E-02	[ppm] (chemical solubility)
Pwc	=	4.08E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 35): Arsenic (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.150E+03	0.00000E+00
0.200E+03	0.00000E+00
0.250E+03	0.00000E+00
0.300E+03	0.00000E+00
0.350E+03	0.00000E+00
0.400E+03	0.00000E+00
0.450E+03	0.00000E+00
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.00000E+00
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.00000E+00
0.850E+03	0.13908E+00
0.900E+03	0.27877E+00
0.950E+03	0.40372E+00
0.100E+04	0.49100E+00
0.105E+04	0.53509E+00
0.110E+04	0.54220E+00
0.115E+04	0.51844E+00
0.120E+04	0.47099E+00
0.125E+04	0.40940E+00
0.130E+04	0.34086E+00

MULTIMED OUTPUT (SWMU 35): Cadmium (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.150E+03	0.00000E+00
0.200E+03	0.00000E+00
0.250E+03	0.00000E+00
0.300E+03	0.00000E+00
0.350E+03	0.00000E+00
0.400E+03	0.00000E+00
0.450E+03	0.00000E+00
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.00000E+00
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.00000E+00
0.850E+03	0.00000E+00
0.900E+03	0.00000E+00
0.950E+03	0.00000E+00
0.100E+04	0.00000E+00
0.105E+04	0.19273E-02
0.110E+04	0.56332E-02
0.115E+04	0.91059E-02
0.120E+04	0.12389E-01
0.125E+04	0.14735E-01
0.130E+04	0.16436E-01

MULTIMED OUTPUT (SWMU 35): Lead (mg/L)

TIME	CONCENTRATION
0.100E+04	0.00000E+00
0.110E+04	0.00000E+00
0.120E+04	0.00000E+00
0.130E+04	0.00000E+00
0.140E+04	0.00000E+00
0.150E+04	0.00000E+00
0.160E+04	0.00000E+00
0.170E+04	0.00000E+00
0.180E+04	0.00000E+00
0.190E+04	0.00000E+00
0.200E+04	0.00000E+00
0.210E+04	0.00000E+00
0.220E+04	0.00000E+00
0.230E+04	0.00000E+00
0.240E+04	0.00000E+00
0.250E+04	0.00000E+00
0.260E+04	0.00000E+00
0.270E+04	0.00000E+00
0.280E+04	0.00000E+00
0.290E+04	0.00000E+00
0.300E+04	0.00000E+00
0.310E+04	0.00000E+00
0.320E+04	0.00000E+00
0.330E+04	0.00000E+00
0.340E+04	0.86625E-03

MULTIMED OUTPUT (SWMU 35): Nitrate (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.150E+03	0.00000E+00
0.200E+03	0.00000E+00
0.250E+03	0.00000E+00
0.300E+03	0.00000E+00
0.350E+03	0.00000E+00
0.400E+03	0.00000E+00
0.450E+03	0.00000E+00
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.00000E+00
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.00000E+00
0.850E+03	0.99964E-01
0.900E+03	0.20037E+00
0.950E+03	0.29017E+00
0.100E+04	0.35291E+00
0.105E+04	0.38460E+00
0.110E+04	0.38971E+00
0.115E+04	0.37263E+00
0.120E+04	0.33853E+00
0.125E+04	0.29425E+00
0.130E+04	0.24499E+00

MULTIMED OUTPUT (SWMU 35): Aldrin (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.390E+04	0.00000E+00
0.770E+04	0.00000E+00
0.115E+05	0.00000E+00
0.153E+05	0.00000E+00
0.191E+05	0.00000E+00
0.229E+05	0.00000E+00
0.267E+05	0.00000E+00
0.305E+05	0.00000E+00
0.343E+05	0.00000E+00
0.381E+05	0.00000E+00
0.419E+05	0.00000E+00
0.457E+05	0.00000E+00
0.495E+05	0.00000E+00
0.533E+05	0.00000E+00
0.571E+05	0.00000E+00
0.609E+05	0.00000E+00
0.647E+05	0.00000E+00
0.685E+05	0.00000E+00
0.723E+05	0.00000E+00
0.761E+05	0.00000E+00
0.799E+05	0.00000E+00
0.837E+05	0.00000E+00
0.875E+05	0.00000E+00
0.913E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 35): Alpha-chlordane (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.390E+04	0.00000E+00
0.770E+04	0.00000E+00
0.115E+05	0.00000E+00
0.153E+05	0.00000E+00
0.191E+05	0.00000E+00
0.229E+05	0.00000E+00
0.267E+05	0.00000E+00
0.305E+05	0.00000E+00
0.343E+05	0.00000E+00
0.381E+05	0.00000E+00
0.419E+05	0.00000E+00
0.457E+05	0.00000E+00
0.495E+05	0.00000E+00
0.533E+05	0.00000E+00
0.571E+05	0.00000E+00
0.609E+05	0.00000E+00
0.647E+05	0.00000E+00
0.685E+05	0.00000E+00
0.723E+05	0.00000E+00
0.761E+05	0.00000E+00
0.799E+05	0.00000E+00
0.837E+05	0.00000E+00
0.875E+05	0.00000E+00
0.913E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 35): alpha-Endosulfan (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.00000E+00
0.853E+03	0.11665E-02
0.903E+03	0.27724E-02
0.953E+03	0.42203E-02
0.100E+04	0.52905E-02
0.105E+04	0.59530E-02
0.110E+04	0.61806E-02
0.115E+04	0.60190E-02
0.120E+04	0.55737E-02

MULTIMED OUTPUT (SWMU 35): beta-Benzenehexachloride (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.00000E+00
0.853E+03	0.00000E+00
0.903E+03	0.00000E+00
0.953E+03	0.00000E+00
0.100E+04	0.00000E+00
0.105E+04	0.00000E+00
0.110E+04	0.00000E+00
0.115E+04	0.12706E-03
0.120E+04	0.47498E-03

MULTIMED OUTPUT (SWMU 35): p,p'-DDE (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.390E+04	0.00000E+00
0.770E+04	0.00000E+00
0.115E+05	0.00000E+00
0.153E+05	0.00000E+00
0.191E+05	0.00000E+00
0.229E+05	0.00000E+00
0.267E+05	0.00000E+00
0.305E+05	0.00000E+00
0.343E+05	0.00000E+00
0.381E+05	0.00000E+00
0.419E+05	0.00000E+00
0.457E+05	0.00000E+00
0.495E+05	0.00000E+00
0.533E+05	0.00000E+00
0.571E+05	0.00000E+00
0.609E+05	0.00000E+00
0.647E+05	0.00000E+00
0.685E+05	0.00000E+00
0.723E+05	0.00000E+00
0.761E+05	0.00000E+00
0.799E+05	0.00000E+00
0.837E+05	0.00000E+00
0.875E+05	0.00000E+00
0.913E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 35): delta-Benzenehexachloride

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.150E+03	0.00000E+00
0.200E+03	0.00000E+00
0.250E+03	0.00000E+00
0.300E+03	0.00000E+00
0.350E+03	0.00000E+00
0.400E+03	0.00000E+00
0.450E+03	0.00000E+00
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.00000E+00
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.00000E+00
0.850E+03	0.10431E-01
0.900E+03	0.20908E-01
0.950E+03	0.30279E-01
0.100E+04	0.36825E-01
0.105E+04	0.40132E-01
0.110E+04	0.40665E-01
0.115E+04	0.38883E-01
0.120E+04	0.35325E-01
0.125E+04	0.30705E-01
0.130E+04	0.25564E-01

MULTIMED OUTPUT (SWMU 35): Dieldrin (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.210E+04	0.00000E+00
0.410E+04	0.00000E+00
0.610E+04	0.00000E+00
0.810E+04	0.00000E+00
0.101E+05	0.00000E+00
0.121E+05	0.00000E+00
0.141E+05	0.12560E-04
0.161E+05	0.30104E-04
0.181E+05	0.33352E-04
0.201E+05	0.26091E-04
0.221E+05	0.15493E-04
0.241E+05	0.63540E-05
0.261E+05	0.00000E+00
0.281E+05	0.00000E+00
0.301E+05	0.00000E+00
0.321E+05	0.00000E+00
0.341E+05	0.00000E+00
0.361E+05	0.00000E+00
0.381E+05	0.00000E+00
0.401E+05	0.00000E+00
0.421E+05	0.00000E+00
0.441E+05	0.00000E+00
0.461E+05	0.00000E+00
0.481E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 35): Endrin (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.600E+03	0.00000E+00
0.110E+04	0.00000E+00
0.160E+04	0.00000E+00
0.210E+04	0.00000E+00
0.260E+04	0.00000E+00
0.310E+04	0.00000E+00
0.360E+04	0.10196E-01
0.410E+04	0.16301E-01
0.460E+04	0.15072E-01
0.510E+04	0.10147E-01
0.560E+04	0.49711E-02
0.610E+04	0.11553E-02
0.660E+04	0.00000E+00
0.710E+04	0.00000E+00
0.760E+04	0.00000E+00
0.810E+04	0.00000E+00
0.860E+04	0.00000E+00
0.910E+04	0.00000E+00
0.960E+04	0.00000E+00
0.101E+05	0.00000E+00
0.106E+05	0.00000E+00
0.111E+05	0.00000E+00
0.116E+05	0.00000E+00
0.121E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 35): gamma-chlordane (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.390E+04	0.00000E+00
0.770E+04	0.00000E+00
0.115E+05	0.00000E+00
0.153E+05	0.00000E+00
0.191E+05	0.00000E+00
0.229E+05	0.00000E+00
0.267E+05	0.00000E+00
0.305E+05	0.00000E+00
0.343E+05	0.00000E+00
0.381E+05	0.00000E+00
0.419E+05	0.00000E+00
0.457E+05	0.00000E+00
0.495E+05	0.00000E+00
0.533E+05	0.00000E+00
0.571E+05	0.00000E+00
0.609E+05	0.00000E+00
0.647E+05	0.00000E+00
0.685E+05	0.00000E+00
0.723E+05	0.00000E+00
0.761E+05	0.00000E+00
0.799E+05	0.00000E+00
0.837E+05	0.00000E+00
0.875E+05	0.00000E+00
0.913E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 35): Heptachlor

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.210E+04	0.00000E+00
0.410E+04	0.00000E+00
0.610E+04	0.00000E+00
0.810E+04	0.00000E+00
0.101E+05	0.21118E-03
0.121E+05	0.21572E-03
0.141E+05	0.10089E-03
0.161E+05	0.10844E-04
0.181E+05	0.00000E+00
0.201E+05	0.00000E+00
0.221E+05	0.00000E+00
0.241E+05	0.00000E+00
0.261E+05	0.00000E+00
0.281E+05	0.00000E+00
0.301E+05	0.00000E+00
0.321E+05	0.00000E+00
0.341E+05	0.00000E+00
0.361E+05	0.00000E+00
0.381E+05	0.00000E+00
0.401E+05	0.00000E+00
0.421E+05	0.00000E+00
0.441E+05	0.00000E+00
0.461E+05	0.00000E+00
0.481E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 35): Heptachlor epoxide (mg/L)

TIME	CONCENTRATION
0.100E+03	0.00000E+00
0.210E+04	0.00000E+00
0.410E+04	0.00000E+00
0.610E+04	0.00000E+00
0.810E+04	0.66422E-04
0.101E+05	0.41042E-03
0.121E+05	0.32674E-03
0.141E+05	0.11400E-03
0.161E+05	0.00000E+00
0.181E+05	0.00000E+00
0.201E+05	0.00000E+00
0.221E+05	0.00000E+00
0.241E+05	0.00000E+00
0.261E+05	0.00000E+00
0.301E+05	0.00000E+00
0.341E+05	0.00000E+00
0.361E+05	0.00000E+00
0.381E+05	0.00000E+00
0.401E+05	0.00000E+00
0.421E+05	0.00000E+00
0.441E+05	0.00000E+00
0.461E+05	0.00000E+00
0.481E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 35): Lindane (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.37782E-06
0.853E+03	0.18853E-03
0.903E+03	0.36537E-03
0.953E+03	0.52158E-03
0.100E+04	0.62979E-03
0.105E+04	0.68354E-03
0.110E+04	0.69079E-03
0.115E+04	0.65825E-03
0.120E+04	0.59629E-03

SWMU 36

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 36
Analyte : BARIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	52	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	470.8	[-] (retardation coefficient)
Vc	=	3.05E-04	[cm/day] (contaminant travel velocity)
CTt	=	73573	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	6505.0	[m^2] (contaminated area of the land)
C init	=	1.24E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.24E+01	[ppm] (pore water contam. conc. at WT)
AL	=	7.1E+00	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	5.80E+02	[ppm] (total soil concentration)
Slb	=	1.25E+01	[ppm] (chemical solubility)
Pwc	=	1.24E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	45	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	984	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	984	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.02	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.76E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	152	[cm] (thickness of contamin. soil zone)
P. d.	=	1364	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	52	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	183.6	[-] (retardation coefficient)
T	=	17129	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 36
Analyte : CADMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.3	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	12.7	[-] (retardation coefficient)
Vc	=	1.13E-02	[cm/day] (contaminant travel velocity)
CTt	=	1992	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	6505.0	[m^2] (contaminated area of the land)
C init	=	1.25E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.25E+00	[ppm] (pore water contam. conc. at WT)
AL	=	7.1E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	45	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	984	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	984	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.02	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.78E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	152	[cm] (thickness of contamin. soil zone)
P. d.	=	37	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.59E+00	[ppm] (total soil concentration)
Slb	=	1.26E+00	[ppm] (chemical solubility)
Pwc	=	1.25E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.3	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.6	[-] (retardation coefficient)
T	=	519	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 36
Analyte : CHROMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.2	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	11.8	[-] (retardation coefficient)
Vc	=	1.21E-02	[cm/day] (contaminant travel velocity)
CTt	=	1850	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	6505.0	[m^2] (contaminated area of the land)
C init	=	3.14E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	3.14E+01	[ppm] (pore water contam. conc. at WT)
AL	=	1.8E+01	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	3.71E+01	[ppm] (total soil concentration)
Slb	=	3.15E+01	[ppm] (chemical solubility)
Pwc	=	3.14E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	45	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	984	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	984	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.02	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	4.48E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	152	[cm] (thickness of contamin. soil zone)
P. d.	=	34	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	1.2	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.2	[-] (retardation coefficient)
T	=	486	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 36
Analyte : COPPER

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.4	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	13.6	[-] (retardation coefficient)
Vc	=	1.05E-02	[cm/day] (contaminant travel velocity)
CTt	=	2133	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	6505.0	[m^2] (contaminated area of the land)
C init	=	1.69E+03	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.69E+03	[ppm] (pore water contam. conc. at WT)
AL	=	9.6E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	45	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	984	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	984	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.02	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.41E+02	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	152	[cm] (thickness of contamin. soil zone)
P. d.	=	40	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.30E+03	[ppm] (total soil concentration)
Slb	=	1.70E+03	[ppm] (chemical solubility)
Pwc	=	1.69E+03	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.4	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.9	[-] (retardation coefficient)
T	=	552	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 36
Analyte : LEAD

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	4.5	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	41.7	[-] (retardation coefficient)
Vc	=	3.45E-03	[cm/day] (contaminant travel velocity)
CTt	=	6510	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	6505.0	[m^2] (contaminated area of the land)
C init	=	4.58E+02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	4.58E+02	[ppm] (pore water contam. conc. at WT)
AL	=	2.6E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	45	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	984	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	984	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.02	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	6.52E+01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	152	[cm] (thickness of contamin. soil zone)
P. d.	=	121	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.90E+03	[ppm] (total soil concentration)
Slb	=	4.59E+02	[ppm] (chemical solubility)
Pwc	=	4.58E+02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	4.5	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	16.8	[-] (retardation coefficient)
T	=	1568	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 36): Barium (mg/L)

TIME	CONCENTRATION
0.100E+05	0.00000E+00
0.120E+05	0.00000E+00
0.140E+05	0.00000E+00
0.160E+05	0.00000E+00
0.180E+05	0.00000E+00
0.200E+05	0.00000E+00
0.220E+05	0.00000E+00
0.240E+05	0.00000E+00
0.260E+05	0.00000E+00
0.280E+05	0.00000E+00
0.300E+05	0.00000E+00
0.320E+05	0.41116E-01
0.340E+05	0.10931E+00
0.360E+05	0.16400E+00
0.380E+05	0.19716E+00
0.400E+05	0.20947E+00
0.420E+05	0.20389E+00
0.440E+05	0.18403E+00
0.460E+05	0.15484E+00
0.480E+05	0.12087E+00
0.500E+05	0.85846E-01
0.520E+05	0.52216E-01
0.540E+05	0.21637E-01
0.560E+05	0.00000E+00
0.580E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 36): Cadmium (mg/L)

TIME	CONCENTRATION
0.300E+03	0.00000E+00
0.350E+03	0.00000E+00
0.400E+03	0.00000E+00
0.450E+03	0.00000E+00
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.00000E+00
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.00000E+00
0.850E+03	0.38773E-02
0.900E+03	0.10613E-01
0.950E+03	0.16113E-01
0.100E+04	0.19773E-01
0.105E+04	0.21499E-01
0.110E+04	0.21448E-01
0.115E+04	0.20049E-01
0.120E+04	0.17536E-01
0.125E+04	0.14448E-01
0.130E+04	0.11110E-01
0.135E+04	0.77267E-02
0.140E+04	0.45249E-02
0.145E+04	0.16753E-02
0.150E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 36): Chromium (mg/L)

TIME	CONCENTRATION
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.13811E+00
0.850E+03	0.30770E+00
0.900E+03	0.43894E+00
0.950E+03	0.51507E+00
0.100E+04	0.53912E+00
0.105E+04	0.51431E+00
0.110E+04	0.45527E+00
0.115E+04	0.37560E+00
0.120E+04	0.28648E+00
0.125E+04	0.19576E+00
0.130E+04	0.11013E+00
0.135E+04	0.34105E-01
0.140E+04	0.00000E+00
0.145E+04	0.00000E+00
0.150E+04	0.00000E+00
0.155E+04	0.00000E+00
0.160E+04	0.00000E+00
0.165E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 36): Copper (mg/L)

TIME	CONCENTRATION
0.300E+03	0.00000E+00
0.350E+03	0.00000E+00
0.400E+03	0.00000E+00
0.450E+03	0.00000E+00
0.500E+03	0.00000E+00
0.550E+03	0.00000E+00
0.600E+03	0.00000E+00
0.650E+03	0.00000E+00
0.700E+03	0.00000E+00
0.750E+03	0.00000E+00
0.800E+03	0.00000E+00
0.850E+03	0.00000E+00
0.900E+03	0.33094E+01
0.950E+03	0.12099E+02
0.100E+04	0.19393E+02
0.105E+04	0.24999E+02
0.110E+04	0.28384E+02
0.115E+04	0.29562E+02
0.120E+04	0.28721E+02
0.125E+04	0.26398E+02
0.130E+04	0.22940E+02
0.135E+04	0.18930E+02
0.140E+04	0.14623E+02
0.145E+04	0.10339E+02
0.150E+04	0.62843E+01

MULTIMED OUTPUT (SWMU 36): Lead (mg/L)

TIME	CONCENTRATION
0.250E+04	0.00000E+00
0.255E+04	0.00000E+00
0.260E+04	0.00000E+00
0.265E+04	0.00000E+00
0.270E+04	0.00000E+00
0.275E+04	0.49654E+00
0.280E+04	0.12541E+01
0.285E+04	0.20117E+01
0.290E+04	0.27693E+01
0.295E+04	0.35268E+01
0.300E+04	0.41438E+01
0.305E+04	0.47462E+01
0.310E+04	0.53487E+01
0.315E+04	0.59511E+01
0.320E+04	0.63468E+01
0.325E+04	0.67314E+01
0.330E+04	0.71160E+01
0.335E+04	0.73737E+01
0.340E+04	0.75633E+01
0.345E+04	0.77530E+01
0.350E+04	0.78062E+01
0.355E+04	0.78447E+01
0.360E+04	0.78155E+01
0.365E+04	0.77434E+01
0.370E+04	0.76157E+01

SWMU 40

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Sources: MULTIMED Model, EPA, August 1990;
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EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : ARSENIC

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2046	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	2.93E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	29.30	[ppm] (pore water contam. conc. at WT)
AL	=	3.6E+02	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	29.3000	[ppm] (total soil concentration)
Slb	=	2.94E+01	[ppm] (chemical solubility)
Pwc	=	2.93E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	19.0861	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	41	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

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EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : BARIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	52	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	470.8	[-] (retardation coefficient)
Vc	=	3.05E-04	[cm/day] (contaminant travel velocity)
CTt	=	96004	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	5.97E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	59.68	[ppm] (pore water contam. conc. at WT)
AL	=	7.4E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	38.8748	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	1911	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.80E+03	[ppm] (total soil concentration)
Slb	=	5.98E+01	[ppm] (chemical solubility)
Pwc	=	5.97E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	52	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	183.6	[-] (retardation coefficient)
T	=	17129	[years] (contam. travel time)

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Site Name : Tooele North - SWMU 40
Analyte : CADMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.3	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	12.7	[-] (retardation coefficient)
Vc	=	1.13E-02	[cm/day] (contaminant travel velocity)
CTt	=	2599	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	4.97E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	4.97	[ppm] (pore water contam. conc. at WT)
AL	=	6.1E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	3.2362	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	52	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	6.31E+00	[ppm] (total soil concentration)
Slb	=	4.98E+00	[ppm] (chemical solubility)
Pwc	=	4.97E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.3	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.6	[-] (retardation coefficient)
T	=	519	[years] (contam. travel time)

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Site Name : Tooele North - SWMU 40
Analyte : CHROMIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.2	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	11.8	[-] (retardation coefficient)
Vc	=	1.21E-02	[cm/day] (contaminant travel velocity)
CTt	=	2415	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	3.75E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	37.54	[ppm] (pore water contam. conc. at WT)
AL	=	4.6E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	24.4537	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	48	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.43E+01	[ppm] (total soil concentration)
Slb	=	3.76E+01	[ppm] (chemical solubility)
Pwc	=	3.75E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.2	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.2	[-] (retardation coefficient)
T	=	486	[years] (contam. travel time)

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Site Name : Tooele North - SWMU 40
Analyte : COPPER

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1.4	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	13.6	[-] (retardation coefficient)
Vc	=	1.05E-02	[cm/day] (contaminant travel velocity)
CTt	=	2783	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	1.65E+02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	164.69	[ppm] (pore water contam. conc. at WT)
AL	=	2.0E+03	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	107.2787	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	55	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.24E+02	[ppm] (total soil concentration)
Slb	=	1.66E+02	[ppm] (chemical solubility)
Pwc	=	1.65E+02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1.4	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	5.9	[-] (retardation coefficient)
T	=	552	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : LEAD

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	4.5	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	41.7	[-] (retardation coefficient)
Vc	=	3.45E-03	[cm/day] (contaminant travel velocity)
CTt	=	8494	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	3.85E+02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	385.43	[ppm] (pore water contam. conc. at WT)
AL	=	4.7E+03	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	1.60E+03	[ppm] (total soil concentration)
Slb	=	3.86E+02	[ppm] (chemical solubility)
Pwc	=	3.85E+02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	251.0688	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	169	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	4.5	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	16.8	[-] (retardation coefficient)
T	=	1568	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : MERCURY

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	10	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	91.4	[-] (retardation coefficient)
Vc	=	1.57E-03	[cm/day] (contaminant travel velocity)
CTt	=	18627	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	4.34E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half.life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.04	[ppm] (pore water contam. conc. at WT)
AL	=	5.3E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0283	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	371	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.95E-01	[ppm] (total soil concentration)
Slb	=	4.35E-02	[ppm] (chemical solubility)
Pwc	=	4.34E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	10	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	36.1	[-] (retardation coefficient)
T	=	3369	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : NICKEL

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	400	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	3615.1	[-] (retardation coefficient)
Vc	=	3.98E-05	[cm/day] (contaminant travel velocity)
CTt	=	737125	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	6.94E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.07	[ppm] (pore water contam. conc. at WT)
AL	=	8.5E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0452	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	14674	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.50E+01	[ppm] (total soil concentration)
Slb	=	6.95E-02	[ppm] (chemical solubility)
Pwc	=	6.94E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	400	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	1405.7	[-] (retardation coefficient)
T	=	131138	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
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Sources: MULTIMED Model, EPA, August 1990;
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Site Name : Tooele North - SWMU 40
Analyte : NITRATE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2046	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	1.15E+02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	115.00	[ppm] (pore water contam. conc. at WT)
AL	=	1.4E+03	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	74.9113	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	41	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.15E+02	[ppm] (total soil concentration)
Slb	=	1.16E+02	[ppm] (chemical solubility)
Pwc	=	1.15E+02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : VANADIUM

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1000	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	9038.3	[-] (retardation coefficient)
Vc	=	1.59E-05	[cm/day] (contaminant travel velocity)
CTt	=	1842507	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	5.51E-02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	0.06	[ppm] (pore water contam. conc. at WT)
AL	=	6.8E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.0359	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	36678	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.96E+01	[ppm] (total soil concentration)
Slb	=	6.95E-02	[ppm] (chemical solubility)
Pwc	=	5.51E-02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1000	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	3512.6	[-] (retardation coefficient)
T	=	327706	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
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Sources: MULTIMED Model, EPA, August 1990;
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EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : HMX

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2046	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	4.85E+02	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	485.00	[ppm] (pore water contam. conc. at WT)
AL	=	6.0E+03	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	315.9302	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	41	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.85E+02	[ppm] (total soil concentration)
Slb	=	4.86E+02	[ppm] (chemical solubility)
Pwc	=	4.85E+02	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : RDX

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2046	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	3.20E+03	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	3200.00	[ppm] (pore water contam. conc. at WT)
AL	=	3.9E+04	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	3.20E+03	[ppm] (total soil concentration)
Slb	=	3.21E+03	[ppm] (chemical solubility)
Pwc	=	3.20E+03	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2084.4878	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	41	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : 2,4-DINITROTOLUENE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2046	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	8.15E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	8.15	[ppm] (pore water contam. conc. at WT)
AL	=	1.0E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	5.3089	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	41	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	8.15E+00	[ppm] (total soil concentration)
Slb	=	8.16E+00	[ppm] (chemical solubility)
Pwc	=	8.15E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : 1,3,5-TRINITROBENZENE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2046	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	3.27E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	3.27	[ppm] (pore water contam. conc. at WT)
AL	=	4.0E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.1301	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	41	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.27E+00	[ppm] (total soil concentration)
Slb	=	3.28E+00	[ppm] (chemical solubility)
Pwc	=	3.27E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : 2,4,6-TRINITROTOLUENE

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2046	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	1.44E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	14.40	[ppm] (pore water contam. conc. at WT)
AL	=	1.8E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	9.3802	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	41	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.44E+01	[ppm] (total soil concentration)
Slb	=	1.45E+01	[ppm] (chemical solubility)
Pwc	=	1.44E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : TETRYL

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2046	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	1.80E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	18.00	[ppm] (pore water contam. conc. at WT)
AL	=	2.2E+02	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	1.80E+01	[ppm] (total soil concentration)
Slb	=	1.81E+01	[ppm] (chemical solubility)
Pwc	=	1.80E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	11.7252	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	41	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 40): ARSENIC (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.17767E-02
0.853E+03	0.88659E+00
0.903E+03	0.17529E+01
0.953E+03	0.25663E+01
0.100E+04	0.31750E+01
0.105E+04	0.35240E+01
0.110E+04	0.36384E+01
0.115E+04	0.35432E+01
0.120E+04	0.32872E+01

MULTIMED OUTPUT (SWMU 40): CADMIUM (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.00000E+00
0.853E+03	0.00000E+00
0.903E+03	0.00000E+00
0.953E+03	0.00000E+00
0.100E+04	0.00000E+00
0.105E+04	0.67315E-01
0.110E+04	0.18336E+00
0.115E+04	0.29735E+00
0.120E+04	0.41003E+00

MULTIMED OUTPUT (SWMU 40): Chromium (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.00000E+00
0.853E+03	0.00000E+00
0.903E+03	0.00000E+00
0.953E+03	0.53125E-01
0.100E+04	0.99222E+00
0.105E+04	0.19248E+01
0.110E+04	0.28361E+01
0.115E+04	0.35822E+01
0.120E+04	0.41465E+01

MULTIMED OUTPUT (SWMU 40): Copper (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.00000E+00
0.853E+03	0.00000E+00
0.903E+03	0.00000E+00
0.953E+03	0.00000E+00
0.100E+04	0.00000E+00
0.105E+04	0.00000E+00
0.110E+04	0.36713E+00
0.115E+04	0.39152E+01
0.120E+04	0.74632E+01

MULTIMED OUTPUT (SWMU 40): Lead (mg/L)

TIME	CONCENTRATION
0.100E+04	0.00000E+00
0.110E+04	0.00000E+00
0.120E+04	0.00000E+00
0.130E+04	0.00000E+00
0.140E+04	0.00000E+00
0.150E+04	0.00000E+00
0.160E+04	0.00000E+00
0.170E+04	0.00000E+00
0.180E+04	0.00000E+00
0.190E+04	0.00000E+00
0.200E+04	0.00000E+00
0.210E+04	0.00000E+00
0.220E+04	0.00000E+00
0.230E+04	0.00000E+00
0.240E+04	0.00000E+00
0.250E+04	0.00000E+00
0.260E+04	0.00000E+00
0.270E+04	0.00000E+00
0.280E+04	0.00000E+00
0.290E+04	0.00000E+00
0.300E+04	0.00000E+00
0.310E+04	0.00000E+00
0.320E+04	0.00000E+00
0.330E+04	0.00000E+00
0.340E+04	0.72174E-01

MULTIMED OUTPUT (SWMU 40): Mercury (mg/L)

TIME	CONCENTRATION
0.500E+04	0.00000E+00
0.550E+04	0.00000E+00
0.600E+04	0.00000E+00
0.650E+04	0.00000E+00
0.700E+04	0.00000E+00
0.750E+04	0.14496E-03
0.800E+04	0.15166E-02
0.850E+04	0.28594E-02
0.900E+04	0.40242E-02
0.950E+04	0.48278E-02
0.100E+05	0.52274E-02
0.105E+05	0.52103E-02
0.110E+05	0.48986E-02
0.115E+05	0.43747E-02
0.120E+05	0.37398E-02
0.125E+05	0.30686E-02
0.130E+05	0.24137E-02
0.135E+05	0.18159E-02
0.140E+05	0.12933E-02
0.145E+05	0.83348E-03
0.150E+05	0.47837E-03
0.155E+05	0.27279E-03
0.160E+05	0.23920E-03
0.165E+05	0.20561E-03
0.170E+05	0.17201E-03

MULTIMED OUTPUT (SWMU 22): Nickel (mg/L)

TIME	CONCENTRATION
0.200E+04	0.00000E+00
0.590E+04	0.00000E+00
0.980E+04	0.00000E+00
0.137E+05	0.00000E+00
0.176E+05	0.00000E+00
0.215E+05	0.00000E+00
0.254E+05	0.00000E+00
0.293E+05	0.00000E+00
0.332E+05	0.00000E+00
0.371E+05	0.00000E+00
0.410E+05	0.00000E+00
0.449E+05	0.00000E+00
0.488E+05	0.00000E+00
0.527E+05	0.00000E+00
0.566E+05	0.00000E+00
0.605E+05	0.00000E+00
0.644E+05	0.00000E+00
0.683E+05	0.00000E+00
0.722E+05	0.00000E+00
0.761E+05	0.00000E+00
0.800E+05	0.00000E+00
0.839E+05	0.00000E+00
0.878E+05	0.00000E+00
0.917E+05	0.00000E+00
0.956E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 40): Nitrate (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.00000E+00
0.153E+03	0.00000E+00
0.203E+03	0.00000E+00
0.253E+03	0.00000E+00
0.303E+03	0.00000E+00
0.353E+03	0.00000E+00
0.403E+03	0.00000E+00
0.453E+03	0.00000E+00
0.503E+03	0.00000E+00
0.553E+03	0.00000E+00
0.603E+03	0.00000E+00
0.653E+03	0.00000E+00
0.703E+03	0.00000E+00
0.753E+03	0.00000E+00
0.803E+03	0.69735E-02
0.853E+03	0.34798E+01
0.903E+03	0.68800E+01
0.953E+03	0.10073E+02
0.100E+04	0.12462E+02
0.105E+04	0.13831E+02
0.110E+04	0.14280E+02
0.115E+04	0.13907E+02
0.120E+04	0.12902E+02

MULTIMED OUTPUT (SWMU 40): Vanadium (mg/L)

TIME	CONCENTRATION
0.200E+04	0.00000E+00
0.590E+04	0.00000E+00
0.980E+04	0.00000E+00
0.137E+05	0.00000E+00
0.176E+05	0.00000E+00
0.215E+05	0.00000E+00
0.254E+05	0.00000E+00
0.293E+05	0.00000E+00
0.332E+05	0.00000E+00
0.371E+05	0.00000E+00
0.410E+05	0.00000E+00
0.449E+05	0.00000E+00
0.488E+05	0.00000E+00
0.527E+05	0.00000E+00
0.566E+05	0.00000E+00
0.605E+05	0.00000E+00
0.644E+05	0.00000E+00
0.683E+05	0.00000E+00
0.722E+05	0.00000E+00
0.761E+05	0.00000E+00
0.800E+05	0.00000E+00
0.839E+05	0.00000E+00
0.878E+05	0.00000E+00
0.917E+05	0.00000E+00
0.956E+05	0.00000E+00

MULTIMED OUTPUT (SWMU 40): HMX (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.800E+01	0.00000E+00
0.130E+02	0.00000E+00
0.180E+02	0.00000E+00
0.230E+02	0.00000E+00
0.280E+02	0.00000E+00
0.330E+02	0.00000E+00
0.380E+02	0.00000E+00
0.430E+02	0.00000E+00
0.480E+02	0.00000E+00
0.530E+02	0.00000E+00
0.580E+02	0.00000E+00
0.630E+02	0.00000E+00
0.680E+02	0.00000E+00
0.730E+02	0.33662E+02
0.780E+02	0.10512E+03
0.830E+02	0.18844E+03
0.880E+02	0.28220E+03
0.930E+02	0.37952E+03
0.980E+02	0.48642E+03
0.103E+03	0.56862E+03
0.108E+03	0.61387E+03
0.113E+03	0.61936E+03
0.118E+03	0.58793E+03
0.123E+03	0.52256E+03

MULTIMED OUTPUT (SWMU 40): RDX (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.800E+01	0.00000E+00
0.130E+02	0.00000E+00
0.180E+02	0.00000E+00
0.230E+02	0.00000E+00
0.280E+02	0.00000E+00
0.330E+02	0.00000E+00
0.380E+02	0.00000E+00
0.430E+02	0.00000E+00
0.480E+02	0.00000E+00
0.530E+02	0.00000E+00
0.580E+02	0.00000E+00
0.630E+02	0.00000E+00
0.680E+02	0.00000E+00
0.730E+02	0.22210E+03
0.780E+02	0.69356E+03
0.830E+02	0.12433E+04
0.880E+02	0.18619E+04
0.930E+02	0.25041E+04
0.980E+02	0.32094E+04
0.103E+03	0.37517E+04
0.108E+03	0.40503E+04
0.113E+03	0.40865E+04
0.118E+03	0.38791E+04
0.123E+03	0.34478E+04

MULTIMED OUTPUT (SWMU 40): 2,4-Dinitrotoluene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.800E+01	0.00000E+00
0.130E+02	0.00000E+00
0.180E+02	0.00000E+00
0.230E+02	0.00000E+00
0.280E+02	0.00000E+00
0.330E+02	0.00000E+00
0.380E+02	0.00000E+00
0.430E+02	0.00000E+00
0.480E+02	0.00000E+00
0.530E+02	0.00000E+00
0.580E+02	0.00000E+00
0.630E+02	0.00000E+00
0.680E+02	0.00000E+00
0.730E+02	0.00000E+00
0.780E+02	0.00000E+00
0.830E+02	0.00000E+00
0.880E+02	0.00000E+00
0.930E+02	0.00000E+00
0.980E+02	0.37522E+00
0.103E+03	0.10575E+01
0.108E+03	0.22119E+01
0.113E+03	0.35126E+01
0.118E+03	0.48626E+01
0.123E+03	0.62373E+01

MULTIMED OUTPUT (SWMU 40): 1,3,5-Trinitrobenzene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.800E+01	0.00000E+00
0.130E+02	0.00000E+00
0.180E+02	0.00000E+00
0.230E+02	0.00000E+00
0.280E+02	0.00000E+00
0.330E+02	0.00000E+00
0.380E+02	0.00000E+00
0.430E+02	0.00000E+00
0.480E+02	0.00000E+00
0.530E+02	0.00000E+00
0.580E+02	0.00000E+00
0.630E+02	0.00000E+00
0.680E+02	0.00000E+00
0.730E+02	0.00000E+00
0.780E+02	0.00000E+00
0.830E+02	0.00000E+00
0.880E+02	0.00000E+00
0.930E+02	0.00000E+00
0.980E+02	0.00000E+00
0.103E+03	0.00000E+00
0.108E+03	0.00000E+00
0.113E+03	0.79605E-02
0.118E+03	0.31041E+00
0.123E+03	0.64968E+00

MULTIMED OUTPUT (SWMU 40): 2,4,6-Trinitrotoluene (mg/L)

TIME	CONCENTRATION
0.100E+02	0.00000E+00
0.600E+02	0.00000E+00
0.110E+03	0.00000E+00
0.160E+03	0.00000E+00
0.210E+03	0.00000E+00
0.260E+03	0.00000E+00
0.310E+03	0.00000E+00
0.360E+03	0.00000E+00
0.410E+03	0.00000E+00
0.460E+03	0.00000E+00
0.510E+03	0.94539E+00
0.560E+03	0.21578E+01
0.610E+03	0.29868E+01
0.660E+03	0.32032E+01
0.710E+03	0.29494E+01
0.760E+03	0.24144E+01
0.810E+03	0.18026E+01
0.860E+03	0.12379E+01
0.910E+03	0.77305E+00
0.960E+03	0.42628E+00
0.101E+04	0.27823E+00
0.106E+04	0.25140E+00
0.111E+04	0.22458E+00
0.116E+04	0.19775E+00
0.121E+04	0.17092E+00

MULTIMED OUTPUT (SWMU 40): Tetryl (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.800E+01	0.00000E+00
0.130E+02	0.00000E+00
0.180E+02	0.00000E+00
0.230E+02	0.00000E+00
0.280E+02	0.00000E+00
0.330E+02	0.00000E+00
0.380E+02	0.00000E+00
0.430E+02	0.00000E+00
0.480E+02	0.00000E+00
0.530E+02	0.00000E+00
0.580E+02	0.00000E+00
0.630E+02	0.00000E+00
0.680E+02	0.00000E+00
0.730E+02	0.12502E+01
0.780E+02	0.39025E+01
0.830E+02	0.69950E+01
0.880E+02	0.10475E+02
0.930E+02	0.14086E+02
0.980E+02	0.18055E+02
0.103E+03	0.21104E+02
0.108E+03	0.22784E+02
0.113E+03	0.22987E+02
0.118E+03	0.21819E+02
0.123E+03	0.19393E+02

SECTION 2

Vadose Zone/Saturated Zone Modeling for COPCs
to On-Site an Off-Site Hypothetical Receptors

Note: All units in the MULTIMED output tables are in years for time and mg/l for concentrations.

SWMU 6

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : 2,4-Dinitrotoluene-avg

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	1.70E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.70E+00	[ppm] (pore water contam. conc. at WT)
AL	=	1.9E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0018	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.05E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.70E+00	[ppm] (total soil concentration)
Slb	=	1.71E+00	[ppm] (chemical solubility)
Pwc	=	1.70E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : 2,6-Dinitrotoluene-avg

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	3.00E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	3.00E-01	[ppm] (pore water contam. conc. at WT)
AL	=	3.3E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.82	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.65E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	3.00E-01	[ppm] (total soil concentration)
Slb	=	3.10E-01	[ppm] (chemical solubility)
Pwc	=	3.00E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : 1,3,5-Trinitrobenzene-avg

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	6.20E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	6.20E-01	[ppm] (pore water contam. conc. at WT)
AL	=	6.9E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	3.83E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	6.20E-01	[ppm] (total soil concentration)
Slb	=	6.30E-01	[ppm] (chemical solubility)
Pwc	=	6.20E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 6
Analyte : RDX-avg

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	126300.0	[m^2] (contaminated area of the land)
C init	=	7.00E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	7.00E-01	[ppm] (pore water contam. conc. at WT)
AL	=	7.8E+00	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	199	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4222	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.62	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	4.32E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	7.00E-01	[ppm] (total soil concentration)
Slb	=	7.10E-01	[ppm] (chemical solubility)
Pwc	=	7.00E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 6 - ON SITE RECEPTOR): 2,4-Dinitrotoluene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.130E+02	0.00000E+00
0.230E+02	0.00000E+00
0.330E+02	0.00000E+00
0.430E+02	0.00000E+00
0.530E+02	0.00000E+00
0.630E+02	0.00000E+00
0.730E+02	0.17484E-01
0.830E+02	0.10239E+00
0.930E+02	0.21314E+00
0.103E+03	0.32557E+00
0.113E+03	0.40313E+00
0.123E+03	0.45872E+00
0.133E+03	0.49697E+00
0.143E+03	0.48111E+00
0.153E+03	0.39756E+00
0.163E+03	0.27999E+00
0.173E+03	0.17048E+00
0.183E+03	0.87567E-01
0.193E+03	0.30363E-01
0.203E+03	0.13134E-02
0.213E+03	0.00000E+00
0.223E+03	0.00000E+00
0.233E+03	0.00000E+00
0.243E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 6 - OFF SITE RECEPTOR): 2,4-Dinitrotoluene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.430E+02	0.00000E+00
0.830E+02	0.10462E-16
0.123E+03	0.10462E-16
0.163E+03	0.10434E-11
0.203E+03	0.25249E-08
0.243E+03	0.14958E-06
0.283E+03	0.18839E-05
0.323E+03	0.10028E-04
0.363E+03	0.31021E-04
0.403E+03	0.66807E-04
0.443E+03	0.11234E-03
0.483E+03	0.15904E-03
0.523E+03	0.19918E-03
0.563E+03	0.22817E-03
0.603E+03	0.24458E-03
0.643E+03	0.24921E-03
0.683E+03	0.24416E-03
0.723E+03	0.23178E-03
0.763E+03	0.21444E-03
0.803E+03	0.19417E-03
0.843E+03	0.17259E-03
0.883E+03	0.15088E-03
0.923E+03	0.12987E-03
0.963E+03	0.11011E-03

MULTIMED OUTPUT (SWMU 6 - ON SITE RECEPTOR): 2,6-Dinitrotoluene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.130E+02	0.00000E+00
0.230E+02	0.00000E+00
0.330E+02	0.00000E+00
0.430E+02	0.00000E+00
0.530E+02	0.00000E+00
0.630E+02	0.00000E+00
0.730E+02	0.30854E-02
0.830E+02	0.18068E-01
0.930E+02	0.37613E-01
0.103E+03	0.57454E-01
0.113E+03	0.71141E-01
0.123E+03	0.80950E-01
0.133E+03	0.87701E-01
0.143E+03	0.84902E-01
0.153E+03	0.70158E-01
0.163E+03	0.49411E-01
0.173E+03	0.30085E-01
0.183E+03	0.15453E-01
0.193E+03	0.53582E-02
0.203E+03	0.23177E-03
0.213E+03	0.00000E+00
0.223E+03	0.00000E+00
0.233E+03	0.00000E+00
0.243E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 6 - OFF SITE RECEPTOR): 2,6-Dinitrotoluene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.430E+02	0.00000E+00
0.830E+02	0.18462E-17
0.123E+03	0.18462E-17
0.163E+03	0.18412E-12
0.203E+03	0.44557E-09
0.243E+03	0.26396E-07
0.283E+03	0.33245E-06
0.323E+03	0.17697E-05
0.363E+03	0.54743E-05
0.403E+03	0.11790E-04
0.443E+03	0.19825E-04
0.483E+03	0.28065E-04
0.523E+03	0.35150E-04
0.563E+03	0.40266E-04
0.603E+03	0.43162E-04
0.643E+03	0.43978E-04
0.683E+03	0.43086E-04
0.723E+03	0.40902E-04
0.763E+03	0.37841E-04
0.803E+03	0.34265E-04
0.843E+03	0.30457E-04
0.883E+03	0.26625E-04
0.923E+03	0.22918E-04
0.963E+03	0.19431E-04

MULTIMED OUTPUT (SWMU 6 - ON SITE RECEPTOR): 1,3,5-Trinitrobenzene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.130E+02	0.00000E+00
0.230E+02	0.00000E+00
0.330E+02	0.00000E+00
0.430E+02	0.00000E+00
0.530E+02	0.00000E+00
0.630E+02	0.00000E+00
0.730E+02	0.00000E+00
0.830E+02	0.67868E-06
0.930E+02	0.22871E-01
0.103E+03	0.55524E-01
0.113E+03	0.91327E-01
0.123E+03	0.12170E+00
0.133E+03	0.14783E+00
0.143E+03	0.16799E+00
0.153E+03	0.17271E+00
0.163E+03	0.15617E+00
0.173E+03	0.12556E+00
0.183E+03	0.90421E-01
0.193E+03	0.58365E-01
0.203E+03	0.33249E-01
0.213E+03	0.14969E-01
0.223E+03	0.34125E-02
0.233E+03	0.00000E+00
0.243E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 6 - OFF SITE RECEPTOR): 1,3,5-Trinitrobenzene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.430E+02	0.00000E+00
0.830E+02	0.11190E-18
0.123E+03	0.38176E-17
0.163E+03	0.77453E-14
0.203E+03	0.11997E-09
0.243E+03	0.15553E-07
0.283E+03	0.29455E-06
0.323E+03	0.20246E-05
0.363E+03	0.74702E-05
0.403E+03	0.18241E-04
0.443E+03	0.33569E-04
0.483E+03	0.50722E-04
0.523E+03	0.66636E-04
0.563E+03	0.79102E-04
0.603E+03	0.87113E-04
0.643E+03	0.90635E-04
0.683E+03	0.90264E-04
0.723E+03	0.86836E-04
0.763E+03	0.81229E-04
0.803E+03	0.74236E-04
0.843E+03	0.66521E-04
0.883E+03	0.58581E-04
0.923E+03	0.50775E-04
0.963E+03	0.43345E-04

MULTIMED OUTPUT (SWMU 6 - ON SITE RECEPTOR): RDX (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.230E+02	0.00000E+00
0.430E+02	0.00000E+00
0.630E+02	0.48150E-02
0.830E+02	0.22891E-01
0.103E+03	0.31716E-01
0.123E+03	0.33435E-01
0.143E+03	0.18316E-01
0.163E+03	0.33457E-02
0.183E+03	0.00000E+00
0.203E+03	0.00000E+00
0.223E+03	0.00000E+00
0.243E+03	0.00000E+00
0.263E+03	0.00000E+00
0.283E+03	0.00000E+00
0.303E+03	0.00000E+00
0.323E+03	0.00000E+00
0.343E+03	0.00000E+00
0.363E+03	0.00000E+00
0.383E+03	0.00000E+00
0.403E+03	0.00000E+00
0.423E+03	0.00000E+00
0.443E+03	0.00000E+00
0.463E+03	0.00000E+00
0.483E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 6 - OFF SITE RECEPTOR): RDX (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.430E+02	0.00000E+00
0.830E+02	0.43077E-17
0.123E+03	0.83842E-16
0.163E+03	0.24104E-10
0.203E+03	0.81624E-08
0.243E+03	0.22898E-06
0.283E+03	0.18907E-05
0.323E+03	0.76471E-05
0.363E+03	0.19561E-04
0.403E+03	0.36858E-04
0.443E+03	0.56381E-04
0.483E+03	0.74562E-04
0.523E+03	0.88841E-04
0.563E+03	0.98052E-04
0.603E+03	0.10215E-03
0.643E+03	0.10181E-03
0.683E+03	0.98009E-04
0.723E+03	0.91716E-04
0.763E+03	0.83843E-04
0.803E+03	0.75145E-04
0.843E+03	0.66899E-04
0.883E+03	0.57373E-04
0.923E+03	0.48983E-04
0.963E+03	0.41184E-04

SWMU 13

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 13
Analyte : CHLOROMETHANE-avg

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	119000.0	[m^2] (contaminated area of the land)
C init	=	4.90E-01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	4.90E-01	[ppm] (pore water contam. conc. at WT)
AL	=	5.1E+00	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	4.90E-01	[ppm] (total soil concentration)
Slb	=	4.91E-01	[ppm] (chemical solubility)
Pwc	=	4.90E-01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	193	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4102	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.67	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.94E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 13
Analyte : DIETHYL PHTHALATE-avg

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	8200	[cm] (vadose zone thickness)
Tt	=	156	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	1568	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	119000.0	[m^2] (contaminated area of the land)
C init	=	2.10E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	2.10E+00	[ppm] (pore water contam. conc. at WT)
AL	=	2.2E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	193	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4102	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.67	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	1.26E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	365	[cm] (thickness of contamin. soil zone)
P. d.	=	70	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	2.10E+00	[ppm] (total soil concentration)
Slb	=	2.11E+00	[ppm] (chemical solubility)
Pwc	=	2.10E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 13 - ON SITE RECEPTOR): Chloromethane (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.180E+02	0.00000E+00
0.330E+02	0.00000E+00
0.480E+02	0.00000E+00
0.630E+02	0.75676E-02
0.780E+02	0.58355E-01
0.930E+02	0.10674E+00
0.108E+03	0.13202E+00
0.123E+03	0.14348E+00
0.138E+03	0.11852E+00
0.153E+03	0.61461E-01
0.168E+03	0.18092E-01
0.183E+03	0.00000E+00
0.198E+03	0.00000E+00
0.213E+03	0.00000E+00
0.228E+03	0.00000E+00
0.243E+03	0.00000E+00
0.258E+03	0.00000E+00
0.273E+03	0.00000E+00
0.288E+03	0.00000E+00
0.303E+03	0.00000E+00
0.318E+03	0.00000E+00
0.333E+03	0.00000E+00
0.348E+03	0.00000E+00
0.363E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 13 - OFF SITE RECEPTOR): Chloromethane (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.430E+02	0.00000E+00
0.830E+02	0.30315E-17
0.123E+03	0.30315E-17
0.163E+03	0.18075E-11
0.203E+03	0.13411E-08
0.243E+03	0.55273E-07
0.283E+03	0.58137E-06
0.323E+03	0.27915E-05
0.363E+03	0.81216E-05
0.403E+03	0.16895E-04
0.443E+03	0.27923E-04
0.483E+03	0.39283E-04
0.523E+03	0.49239E-04
0.563E+03	0.56697E-04
0.603E+03	0.61256E-04
0.643E+03	0.63014E-04
0.683E+03	0.62391E-04
0.723E+03	0.59910E-04
0.763E+03	0.56092E-04
0.803E+03	0.51417E-04
0.843E+03	0.46285E-04
0.883E+03	0.40998E-04
0.923E+03	0.35779E-04
0.963E+03	0.30783E-04

MULTIMED OUTPUT (SWMU 13 - ON SITE RECEPTOR): Diethylphthalate (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.180E+02	0.00000E+00
0.330E+02	0.00000E+00
0.480E+02	0.00000E+00
0.630E+02	0.00000E+00
0.780E+02	0.41012E-01
0.930E+02	0.21702E+00
0.108E+03	0.40778E+00
0.123E+03	0.52856E+00
0.138E+03	0.59534E+00
0.153E+03	0.50950E+00
0.168E+03	0.30940E+00
0.183E+03	0.13528E+00
0.198E+03	0.33322E-01
0.213E+03	0.00000E+00
0.228E+03	0.00000E+00
0.243E+03	0.00000E+00
0.258E+03	0.00000E+00
0.273E+03	0.00000E+00
0.288E+03	0.00000E+00
0.303E+03	0.00000E+00
0.318E+03	0.00000E+00
0.333E+03	0.00000E+00
0.348E+03	0.00000E+00
0.363E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 13 - OFF SITE RECEPTOR): Diethylphthalate (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.430E+02	0.00000E+00
0.830E+02	0.12992E-16
0.123E+03	0.12992E-16
0.163E+03	0.17424E-12
0.203E+03	0.81368E-09
0.243E+03	0.68556E-07
0.283E+03	0.10699E-05
0.323E+03	0.66202E-05
0.363E+03	0.22962E-04
0.403E+03	0.54105E-04
0.443E+03	0.97757E-04
0.483E+03	0.14668E-03
0.523E+03	0.19278E-03
0.563E+03	0.22999E-03
0.603E+03	0.25530E-03
0.643E+03	0.26824E-03
0.683E+03	0.27003E-03
0.723E+03	0.26281E-03
0.763E+03	0.24883E-03
0.803E+03	0.23026E-03
0.843E+03	0.20898E-03
0.883E+03	0.18648E-03
0.923E+03	0.16387E-03
0.963E+03	0.14193E-03

SWMU 22

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 22
Analyte : HMX-avg

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	12000	[cm] (vadose zone thickness)
Tt	=	229	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2295	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	115.0	[m^2] (contaminated area of the land)
C init	=	4.38E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	4.38E+00	[ppm] (pore water contam. conc. at WT)
AL	=	4.4E-02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	6	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	132	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	132	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	6.19E-01	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	4.38E+00	[ppm] (total soil concentration)
Slb	=	4.39E+00	[ppm] (chemical solubility)
Pwc	=	4.38E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 22
Analyte : RDX-avg

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	12000	[cm] (vadose zone thickness)
Tt	=	229	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2295	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	115.0	[m^2] (contaminated area of the land)
C init	=	5.32E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	5.32E+01	[ppm] (pore water contam. conc. at WT)
AL	=	5.4E-01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	6	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	132	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	132	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	7.08	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	7.51E+00	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	305	[cm] (thickness of contamin. soil zone)
P. d.	=	58	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	5.32E+01	[ppm] (total soil concentration)
Slb	=	5.33E+01	[ppm] (chemical solubility)
Pwc	=	5.32E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 22 - ON SITE RECEPTOR): HMX (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.130E+02	0.00000E+00
0.230E+02	0.00000E+00
0.330E+02	0.00000E+00
0.430E+02	0.00000E+00
0.530E+02	0.00000E+00
0.630E+02	0.00000E+00
0.730E+02	0.00000E+00
0.830E+02	0.00000E+00
0.930E+02	0.49472E-02
0.103E+03	0.13118E-01
0.113E+03	0.20906E-01
0.123E+03	0.28585E-01
0.133E+03	0.34430E-01
0.143E+03	0.34966E-01
0.153E+03	0.30042E-01
0.163E+03	0.22599E-01
0.173E+03	0.14542E-01
0.183E+03	0.77866E-02
0.193E+03	0.28588E-02
0.203E+03	0.00000E+00
0.213E+03	0.00000E+00
0.223E+03	0.00000E+00
0.233E+03	0.00000E+00
0.243E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 22 - OFF SITE RECEPTOR): HMX (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.830E+02	0.00000E+00
0.163E+03	0.34550E-17
0.243E+03	0.34550E-17
0.323E+03	0.34550E-17
0.403E+03	0.12066E-14
0.483E+03	0.17072E-12
0.563E+03	0.23042E-10
0.643E+03	0.22525E-09
0.723E+03	0.11745E-08
0.803E+03	0.39130E-08
0.883E+03	0.95760E-08
0.963E+03	0.18802E-07
0.104E+04	0.31390E-07
0.112E+04	0.46354E-07
0.120E+04	0.62234E-07
0.128E+04	0.77489E-07
0.136E+04	0.90774E-07
0.144E+04	0.10111E-06
0.152E+04	0.10793E-06
0.160E+04	0.11103E-06
0.168E+04	0.11056E-06
0.176E+04	0.10678E-06
0.184E+04	0.10024E-06
0.192E+04	0.91428E-07

MULTIMED OUTPUT (SWMU 22 - ON SITE RECEPTOR): RDX (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.130E+02	0.00000E+00
0.230E+02	0.00000E+00
0.330E+02	0.00000E+00
0.430E+02	0.00000E+00
0.530E+02	0.00000E+00
0.630E+02	0.00000E+00
0.730E+02	0.00000E+00
0.830E+02	0.00000E+00
0.930E+02	0.60090E-01
0.103E+03	0.15933E+00
0.113E+03	0.25392E+00
0.123E+03	0.34720E+00
0.133E+03	0.41819E+00
0.143E+03	0.42470E+00
0.153E+03	0.36489E+00
0.163E+03	0.27449E+00
0.173E+03	0.17663E+00
0.183E+03	0.94577E-01
0.193E+03	0.34724E-01
0.203E+03	0.00000E+00
0.213E+03	0.00000E+00
0.223E+03	0.00000E+00
0.233E+03	0.00000E+00
0.243E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 22 - OFF SITE RECEPTOR): RDX (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.11576E-16
0.153E+03	0.11576E-16
0.203E+03	0.29166E-11
0.253E+03	0.16032E-08
0.303E+03	0.43306E-07
0.353E+03	0.30547E-06
0.403E+03	0.10098E-05
0.453E+03	0.21145E-05
0.503E+03	0.33243E-05
0.553E+03	0.43318E-05
0.603E+03	0.49646E-05
0.653E+03	0.51913E-05
0.703E+03	0.50704E-05
0.753E+03	0.46941E-05
0.803E+03	0.41547E-05
0.853E+03	0.35119E-05
0.903E+03	0.28855E-05
0.953E+03	0.22545E-05
0.100E+04	0.16638E-05
0.105E+04	0.11269E-05
0.110E+04	0.64884E-06
0.115E+04	0.22974E-06
0.120E+04	0.00000E+00

SWMU 40

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : HMX-avg

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2046	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	3.47E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	3.47	[ppm] (pore water contam. conc. at WT)
AL	=	4.3E+01	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	3.47E+00	[ppm] (total soil concentration)
Slb	=	3.48E+00	[ppm] (chemical solubility)
Pwc	=	3.47E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	2.2604	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	41	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : RDX-avg

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2046	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	1.70E+01	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	17.00	[ppm] (pore water contam. conc. at WT)
AL	=	2.1E+02	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Ov	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	11.0738	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	41	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.70E+01	[ppm] (total soil concentration)
Slb	=	1.71E+01	[ppm] (chemical solubility)
Pwc	=	1.70E+01	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : 2,4-DINITROTOLUENE-avg

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2046	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	1.20E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.20	[ppm] (pore water contam. conc. at WT)
AL	=	1.5E+01	[kg] (annual contaminant load entering GW)

Pore water concentration calculation based on soil total conc.

Tc	=	1.20E+00	[ppm] (total soil concentration)
Slb	=	1.21E+00	[ppm] (chemical solubility)
Pwc	=	1.20E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.7817	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	41	[years] (pulse duration - for MULTIMED simulation)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

VADOSE ZONE / SATURATED ZONE FLOW & CONTAMINANT TRANSPORT EQUATIONS
SCREENING MODEL - "GWM-1.WK3" spreadsheet by Peter Rzepecki, RUST E & I, 1994.

Sources: MULTIMED Model, EPA, August 1990;
Superfund Exposure Assesment Manual - Vadose Zone Equations
EPA/540/1-88/001, OSWER Directive 9285.5-1, April 1988

Site Name : Tooele North - SWMU 40
Analyte : TETRYL-avg

Volumetric water content in the vadose zone calculation

q	=	2.40E-02	[cm/day] (recharge)
Ks	=	864	[cm/day] (saturated K)
b	=	4.05	[-] (exponential parameter, page 71)
VWC sat.	=	0.43	[-] (volumetric water content - satur. cond.)
1/(2*b+3)	=	0.0901	
VWC	=	0.17	[-] (volumetric water content)

Interstitial ground water (pore water) velocity calculation

Vpw	=	0.14	[cm/day]
H	=	10700	[cm] (vadose zone thickness)
Tt	=	204	[years] (pore water travel time)

Contaminant velocity calculation

Kd	=	1	[ml/g] (distribution coeff. - soil/water)
Bd	=	1.51	[g/cm^3] (soil bulk density)
R v	=	10.0	[-] (retardation coefficient)
Vc	=	1.43E-02	[cm/day] (contaminant travel velocity)
CTt	=	2046	[years] (contaminant travel time)

GW contaminant load calculation

CA	=	140460.0	[m^2] (contaminated area of the land)
C init	=	1.13E+00	[ppm] (pore water contam. conc. at source boundary)
C t1/2	=	0	[days] (cont. half life t, if not degrad. set to 0)
k	=	#DIV/0!	[1/day]
C at GW	=	1.13	[ppm] (pore water contam. conc. at WT)
AL	=	1.4E+01	[kg] (annual contaminant load entering GW)

GW contaminant concentration estimate

K aq	=	864	[cm/day] (aquifer hydraulic conductivity)
Por aq	=	0.43	[-] (aquifer effective porosity)
Grad aq	=	0.0016	[-] (avg hydraulic gradient)
Dv	=	210	[cm] (vertical dispersivity)
H aq	=	1000	[cm] (aquifer thickness)
M t. d.	=	4445	[cm] (theoretical mixing depth at s. d. g.)
M depth	=	1000	[cm] (mixing depth at source downgrad. edge) (MULTIMED equation)
D factor	=	1.54	[-] (dilution factor - vadose zone/aquifer)
C aq edg	=	0.7361	[ppm] (gw contam. conc. at source downgr. edge) (assuming no lateral dispersivity)
H cont.	=	213	[cm] (thickness of contamin. soil zone)
P. d.	=	41	[years] (pulse duration - for MULTIMED simulation)

Pore water concentration calculation based on soil total conc.

Tc	=	1.13E+00	[ppm] (total soil concentration)
Slb	=	1.14E+00	[ppm] (chemical solubility)
Pwc	=	1.13E+00	[ppm] (pore water concentration) (assuming water density = 1 g/cc)

Contaminant travel time in aquifer calculation

R Kd	=	1	[ml/g] (aquifer distrib. coeff.)
R Grad aq	=	0.0095	[-] (avg regional hydraulic gradient)
D osr	=	6500	[m] (distance to "Off Site Receptor")
Bd a	=	1.51	[g/cm^3] (aquifer mat. bulk density)
R a	=	4.5	[-] (retardation coefficient)
T	=	421	[years] (contam. travel time)

MULTIMED OUTPUT (SWMU 40 - ON SITE RECEPTOR): HMX (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.130E+02	0.00000E+00
0.230E+02	0.00000E+00
0.330E+02	0.00000E+00
0.430E+02	0.00000E+00
0.530E+02	0.00000E+00
0.630E+02	0.00000E+00
0.730E+02	0.00000E+00
0.830E+02	0.14439E+00
0.930E+02	0.38408E+00
0.103E+03	0.66626E+00
0.113E+03	0.84420E+00
0.123E+03	0.81258E+00
0.133E+03	0.63522E+00
0.143E+03	0.41446E+00
0.153E+03	0.22113E+00
0.163E+03	0.84672E-01
0.173E+03	0.45130E-02
0.183E+03	0.00000E+00
0.193E+03	0.00000E+00
0.203E+03	0.00000E+00
0.213E+03	0.00000E+00
0.223E+03	0.00000E+00
0.233E+03	0.00000E+00
0.243E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 40 - OFF SITE RECEPTOR): HMX (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.18916E-16
0.153E+03	0.26992E-08
0.203E+03	0.32172E-05
0.253E+03	0.64608E-04
0.303E+03	0.26231E-03
0.353E+03	0.49575E-03
0.403E+03	0.63006E-03
0.453E+03	0.64020E-03
0.503E+03	0.56631E-03
0.553E+03	0.45455E-03
0.603E+03	0.33678E-03
0.653E+03	0.22952E-03
0.703E+03	0.13909E-03
0.753E+03	0.66179E-04
0.803E+03	0.88383E-05
0.853E+03	0.00000E+00
0.903E+03	0.00000E+00
0.953E+03	0.00000E+00
0.100E+04	0.00000E+00
0.105E+04	0.00000E+00
0.110E+04	0.00000E+00
0.115E+04	0.00000E+00
0.120E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 40 - ON SITE RECEPTOR): RDX (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.130E+02	0.00000E+00
0.230E+02	0.00000E+00
0.330E+02	0.00000E+00
0.430E+02	0.00000E+00
0.530E+02	0.00000E+00
0.630E+02	0.00000E+00
0.730E+02	0.00000E+00
0.830E+02	0.70740E+00
0.930E+02	0.18816E+01
0.103E+03	0.32641E+01
0.113E+03	0.41358E+01
0.123E+03	0.39809E+01
0.133E+03	0.31120E+01
0.143E+03	0.20305E+01
0.153E+03	0.10834E+01
0.163E+03	0.41482E+00
0.173E+03	0.22110E-01
0.183E+03	0.00000E+00
0.193E+03	0.00000E+00
0.203E+03	0.00000E+00
0.213E+03	0.00000E+00
0.223E+03	0.00000E+00
0.233E+03	0.00000E+00
0.243E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 40 - OFF SITE RECEPTOR): RDX (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.630E+02	0.00000E+00
0.123E+03	0.19554E-12
0.183E+03	0.20034E-05
0.243E+03	0.20568E-03
0.303E+03	0.12851E-02
0.363E+03	0.26108E-02
0.423E+03	0.31715E-02
0.483E+03	0.29532E-02
0.543E+03	0.23431E-02
0.603E+03	0.16515E-02
0.663E+03	0.10321E-02
0.723E+03	0.53422E-03
0.783E+03	0.15618E-03
0.843E+03	0.00000E+00
0.903E+03	0.00000E+00
0.963E+03	0.00000E+00
0.102E+04	0.00000E+00
0.108E+04	0.00000E+00
0.114E+04	0.00000E+00
0.120E+04	0.00000E+00
0.126E+04	0.00000E+00
0.132E+04	0.00000E+00
0.138E+04	0.00000E+00
0.144E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 40 - ON SITE RECEPTOR): 2,4-Dinitrotoluene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.130E+02	0.00000E+00
0.230E+02	0.00000E+00
0.330E+02	0.00000E+00
0.430E+02	0.00000E+00
0.530E+02	0.00000E+00
0.630E+02	0.00000E+00
0.730E+02	0.00000E+00
0.830E+02	0.00000E+00
0.930E+02	0.00000E+00
0.103E+03	0.31182E-01
0.113E+03	0.10358E+00
0.123E+03	0.18394E+00
0.133E+03	0.23767E+00
0.143E+03	0.24947E+00
0.153E+03	0.22472E+00
0.163E+03	0.17911E+00
0.173E+03	0.12599E+00
0.183E+03	0.79235E-01
0.193E+03	0.43141E-01
0.203E+03	0.17091E-01
0.213E+03	0.29146E-03
0.223E+03	0.00000E+00
0.233E+03	0.00000E+00
0.243E+03	0.00000E+00

MULTIMED OUTPUT (SWMU 40 - OFF SITE RECEPTOR): 2,4-Dinitrotoluene (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.60512E-17
0.153E+03	0.11089E-11
0.203E+03	0.10277E-06
0.253E+03	0.69743E-05
0.303E+03	0.50421E-04
0.353E+03	0.13113E-03
0.403E+03	0.20093E-03
0.453E+03	0.23017E-03
0.503E+03	0.22246E-03
0.553E+03	0.19324E-03
0.603E+03	0.15606E-03
0.653E+03	0.11916E-03
0.703E+03	0.86442E-04
0.753E+03	0.59124E-04
0.803E+03	0.37052E-04
0.853E+03	0.19496E-04
0.903E+03	0.56635E-05
0.953E+03	0.00000E+00
0.100E+04	0.00000E+00
0.105E+04	0.00000E+00
0.110E+04	0.00000E+00
0.115E+04	0.00000E+00
0.120E+04	0.00000E+00

MULTIMED OUTPUT (SWMU 40 - ON SITE RECEPTOR): Tetryl (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.800E+01	0.00000E+00
0.130E+02	0.00000E+00
0.180E+02	0.00000E+00
0.230E+02	0.00000E+00
0.280E+02	0.00000E+00
0.330E+02	0.00000E+00
0.380E+02	0.00000E+00
0.430E+02	0.00000E+00
0.480E+02	0.00000E+00
0.530E+02	0.00000E+00
0.580E+02	0.00000E+00
0.630E+02	0.00000E+00
0.680E+02	0.00000E+00
0.730E+02	0.15719E-01
0.780E+02	0.49066E-01
0.830E+02	0.87949E-01
0.880E+02	0.13171E+00
0.930E+02	0.17710E+00
0.980E+02	0.22701E+00
0.103E+03	0.26534E+00
0.108E+03	0.28646E+00
0.113E+03	0.28902E+00
0.118E+03	0.27434E+00
0.123E+03	0.24383E+00

MULTIMED OUTPUT (SWMU 40 - OFF SITE RECEPTOR): Tetryl (mg/L)

TIME	CONCENTRATION
0.300E+01	0.00000E+00
0.530E+02	0.00000E+00
0.103E+03	0.64322E-17
0.153E+03	0.26117E-08
0.203E+03	0.16467E-05
0.253E+03	0.26347E-04
0.303E+03	0.95547E-04
0.353E+03	0.17001E-03
0.403E+03	0.20886E-03
0.453E+03	0.20792E-03
0.503E+03	0.18141E-03
0.553E+03	0.14412E-03
0.603E+03	0.10581E-03
0.653E+03	0.71380E-04
0.703E+03	0.42614E-04
0.753E+03	0.19586E-04
0.803E+03	0.15866E-05
0.853E+03	0.00000E+00
0.903E+03	0.00000E+00
0.953E+03	0.00000E+00
0.100E+04	0.00000E+00
0.105E+04	0.00000E+00
0.110E+04	0.00000E+00
0.115E+04	0.00000E+00
0.120E+04	0.00000E+00

APPENDIX L

ESTIMATION OF EXPOSURE POINT CONCENTRATIONS; UPTAKE AND EXPOSURE MODELS AND PARAMETERS

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1.0 ESTIMATION OF EXPOSURE POINT CONCENTRATIONS; UPTAKE AND EXPOSURE MODELS AND PARAMETERS

1.1 ESTIMATION OF EXPOSURE POINT CONCENTRATIONS

The exposure point concentration (EPC) is defined as the concentration of a chemical of potential concern (COPC) in an exposure medium that will be contacted over a real or hypothetical exposure duration. Two exposure "cases" were evaluated: (1) reasonable maximum exposure (RME), which is defined by USEPA as the maximum exposure reasonably expected to occur at the site (USEPA 1989), and (2) central tendency exposure (CTE), which may be defined as the "more-likely-to-occur" scenario. EPCs were estimated based on the Phase I and Phase II data.

The arithmetic mean of the COPC data set formed the basis for estimating the RME EPCs. The RME EPC was represented as the 95th percentile upper confidence limit (UCL) on the arithmetic mean and was estimated following USEPA guidance (USEPA 1992) and statistical methods described by Gilbert (1987). To construct the UCL, a determination of the distribution type (i.e., normal, lognormal, or other) was made. One of two tests was used to determine the distribution type of site-related data. For $n \leq 50$, the W-test was employed. For $n > 50$, D'Agostino's test was used (Gilbert 1987). After determination of the distribution, the UCL was calculated based on the appropriate formula (Gilbert 1987; USEPA 1992). Data reported as less than (LT) or nondetect (ND) were assigned a value of one-half the certified reporting limit (CRL) for the purpose of calculating the UCL. Data sets having fewer than the 10 samples provide poor estimates of the mean concentration (i.e., the difference between the mean and the UCL is large). For these data sets, the highest measured concentration was used as the exposure point concentration (USEPA 1992).

The UCL is calculated for a normal distribution as follows:

$$UCL = (\bar{x} + t_{1-\alpha,\, n-1}) * \frac{s}{\sqrt{n}}$$

where

$\bar{x}$	=	Sample arithmetic mean
$t_{1-\alpha,n-1}$	=	Critical value for Student's t-Distribution (given in Table A2, Gilbert 1987)
α	=	0.05 (i.e., 1-α = 0.95 or 95% confidence limit for a one-tailed test)
n	=	Number of samples in the set
s	=	Sample standard deviation.

The UCL is calculated for a lognormal distribution as follows:

$$UCL = \exp[\ \bar{x} + 0.5s^2 + \frac{(s * H_{0.95})}{\sqrt{n - 1}}\ ]$$

where

$\bar{x}$	=	Mean of the transformed data
s	=	Standard deviation of the transformed data
$H_{0.95}$	=	H-statistic for computing the one-sided upper 95% confidence limit on a lognormal mean from standard statistical tables (Gilbert 1987)
n	=	Number of samples in the set

For future-use scenarios, EPCs for organics represent average medium-specific concentrations over time based on environmental half-lives from the scientific literature. This average is represented by the integral of the exponential decay equation over the assumed exposure interval. The basic equation is:

$$C_t = C_0 \exp(-kt)$$

where

C_t	=	soil concentration at time *t* (mg/kg)
C_0	=	initial soil concentration at time $t = 0$ (mg/kg)
t	=	time *t* (hrs, days, or yrs).
k	=	chemical-specific decay constant (hrs, days, or yrs)$^{-1}$, given by:

$$k = \frac{\ln 2}{t_{\frac{1}{2}_i}}$$

where

$t_{\frac{1}{2}_i}$ is the environmental half-life in soil for the i^{th} chemical

This equation yields an estimated soil concentration at some future time *t*. To obtain an average concentration for some exposure duration *ED* = T, the following integral must be evaluated.

$$C_{\bar{s}} = \int_0^T \frac{[C_0 \exp(-kt)]}{T}$$

Evaluating over the interval $t = 0$ to $t = T$, where T is the scenario-specific exposure duration (ED) yields:

$$C_{\bar{s}} = C_0[\frac{1 - \exp(-kT)}{kT}]$$

The above equation was used to estimate RME and CTE EPCs for organics over long-term exposure durations. The initial concentration was the 95th percentile UCL (RME) described above. Environmental half-lives were taken from Howard and others (1991) and are presented in Table 1. It was assumed that for explosives (HMX, RDX, 1,3,5-trinitrobenzene, and 2,4,6-trinitrotoluene) and PCBs (Arochlor 1248 and 1254) concentrations would remain at steady-state.

Table 1. Environmental Half-Lives in Soil for COPCs at TEAD-N

COPC	$t_{½_{max}}$ (days)	$t_{½_{min}}$ (days)
Anthracene	460	50
Benzenehexachloride, delta-	100	13.8
Benzo(a)pyrene	530	57
Chlordane	1,386	283
Chloromethane	28	7
Diethyl Phthalate	56	3
Endosulfan	9	0.1875
Endrin	592	21
Heptachlor	5	0.96
Heptachlor Epoxide	552	33
Phenanthrene	200	16
Pyrene	1,900	210

Precedent for this approach in USEPA Region VIII was established in the Record of Decision (ROD) for the Broderick Wood Products Superfund site.

1.2 MODELS FOR PLANT UPTAKE AND TRANSFER TO BEEF TISSUE

To evaluate potential health risks associated with the consumption of beef cattle grazed on TEAD-N, as well as produce that in the future may be grown on site, it was necessary to model COPC concentrations in plants grown in soils potentially affected by site conditions. Plant uptake will vary with plant species and on a chemical-by-chemical basis. Because efforts to conduct bioassays were unsuccessful at TEAD-N, plant concentrations were estimated using published plant-chemical uptake factors and beef biotransfer factors (Baes et al. 1984; USEPA 1989b; Stevens 1992). Where uptake and biotransfer factors were not available, estimates were made using published methodologies employing the octanol-water partition coefficient (K_{ow}) (Travis and Arms 1988; McKone 1994).

1.2.1 Homegrown Produce

Homegrown produce was divided into two categories: (1) leafy vegetables and (2) tubers and fruits. Because aerial deposition occurs primarily through wind erosion, a random, intermittent event that may affect soils that do not contain SWMU-related chemicals, only root uptake was modeled. The equation for the concentration of metals in leafy vegetables from root uptake is:

$$C_{pi} = B_{vi}[C_{si} f_{d\rightarrow w}]$$

where

C_{pi}	=	Wet weight concentration of the i^{th} chemical in leafy vegetables (mg/kg)
B_{vi}	=	Bioconcentration factor of the i^{th} chemical for leafy vegetables (unitless)
C_{si}	=	Concentration of the i^{th} chemical in root zone soils (mg/kg)
$f_{d\rightarrow w}$	=	Dry weight to wet weight conversion factor (unitless)

This model was also used to estimate concentrations in forage on grazing allotments at TEAD-N. An analogous equation is used to estimate the concentration of metals in tubers and fruits from root uptake.

$$C_{ti} = B_{ri}[C_{si} f_{d\rightarrow w}]$$

where

C_{ti}	=	Dry weight concentration of the i^{th} chemical in tubers and fruits (mg/kg)
B_{ri}	=	Bioconcentration factor of the i^{th} chemical for tubers and fruits (unitless)
C_{si}	=	Concentration of the i^{th} chemical in root zone soils (mg/kg)
$f_{d\rightarrow w}$	=	Dry weight to wet weight conversion factor (unitless)

Bioconcentration factors were taken from Baes and others (1984). Soil concentrations from the 0-to-0.5-foot stratum were used to approximate root zone concentrations for a semi-arid climate. Dry-to-wet weight conversion factors (Baes et al. 1984) are 0.07 for leafy vegetables and 0.22 for tubers and fruits.

A similar approach was used for estimation of the concentration of organic chemicals in edible produce. The appropriate equation for root uptake into leafy vegetables is:

$$C_{pi} = K_{ps} C_{si}$$

where

C_{pi}	=	Wet weight concentration of the i^{th} chemical in leafy vegetables (mg/kg)
K_{ps}	=	Bioconcentration factor of the i^{th} chemical for leafy vegetables (unitless)
C_{si}	=	Concentration of the i^{th} chemical in root zone soils (mg/kg)

An analogous equation is used to estimate the concentration of organic chemicals in tubers and fruits from root uptake.

$$C_{ti} = K_{ps(roots)} C_{si}$$

where

C_{ti}	=	Dry weight concentration of the i^{th} chemical in tubers and fruits (mg/kg)
$K_{ps(roots)}$	=	Bioconcentration factor of the i^{th} chemical for tubers and fruits (unitless)
C_{si}	=	Concentration of the i^{th} chemical in root zone soils (mg/kg)
$f_{d \to w}$	=	Dry weight to wet weight conversion factor (unitless)

Bioconcentration factors were taken from McKone (1994) and are adjusted for dry-to-wet weight conversion. Soil concentrations from the 0-to-0.5-foot stratum were used to approximate root zone concentrations for a semi-arid climate. Table 2 gives the appropriate bioconcentration factors for homegrown produce.

1.2.2 Beef

To model potential concentrations of COPCs in beef tissue, the first step is to model uptake in forage. The models described above for uptake into leafy vegetables were used to approximate forage uptake. The model for transfer to beef tissue is:

$$C_{bi} = B_{bi} [(C_{si} IR_s + C_{fi} IR_f) r_A]$$

where

C_{bi}	=	Concentration of the i^{th} chemical in beef tissue (mg/kg)
B_{bi}	=	Biotransfer factor of the i^{th} chemical for beef (unitless)
C_{si}	=	Concentration of the i^{th} chemical in root zone soils (mg/kg)
IR_s	=	Soil ingestion rate of grazing cattle (kg/day)
C_{fi}	=	Wet weight concentration of the i^{th} chemical in forage (mg/kg)
IR_f	=	Forage ingestion rate of grazing cattle (kg/day)
r_A	=	Ratio of land area of SWMU to land area of entire grazing allotment (unitless)

Table 2. Bioconcentration Factors for Homegrown Produce

Chemical	B_v[a]	B_r[b]	K_{ps}[c]	$K_{ps(roots)}$[d]
1,3,5-Trinitrobenzene	NA[e]	NA	0.381	13.3
2,4,6-Trinitrotoluene	NA	NA	0.381	13.3
Aluminum	0.004	0.00065	NA	NA
Anthracene	NA	NA	0.0179	0.627
Antimony	0.2	0.03	NA	NA
Arsenic	0.04	0.006	NA	NA
Barium	0.15	0.015	NA	NA
Benzenehexachlorde, delta-	NA	NA	0.0472	1.49
Benzo(a)pyrene	NA	NA	0.00131	0.0458
Beryllium	0.01	0.0015	NA	NA
Cadmium	0.55	0.15	NA	NA
Chlordane, gamma-	NA	NA	0.0000195	0.000682
Chlordane, alpha-	NA	NA	0.0028	0.098
Chloromethane	NA	NA	2.31	80.7
Chromium	0.0075	0.0045	NA	NA
Copper	0.4	0.25	NA	NA
Endosulfan	NA	NA	0.0672	2.35
Endrin	NA	NA	0.00616	0.216
Heptachlor	NA	NA	0.00497	0.174
Heptachlor epoxide	NA	NA	0.00568	0.199
HMX	NA	NA	1.70	59.6
Lead	0.045	0.009	NA	NA
Manganese	0.25	0.05	NA	NA
PCBs	NA	NA	0.00136	0.0476
Phenanthrene	NA	NA	0.0172	0.602
Pyrene	NA	NA	0.00484	0.169
RDX	NA	NA	2.41	84.3
Thallium	0.004	0.0004	NA	NA

[a]Bioconcentration factor for metals in leafy vegetables.
[b]Bioconcentration factor for metals in tubers and fruits.
[c]Bioconcentration fractor for organic chemicals in leafy vegetables.
[d]Bioconcentration factor for organic chemicals in tubers and fruits.
[e]Not applicable.

The biotransfer factors for metals are taken from Baes and co-workers (1984) and Stevens (1992). Biotransfer factors for organics are estimated using the chemical-specific K_{ow} and the method described by McKone (1994). These factors are given in Table 3.

Table 3. Biotransfer Factors for Beef

Chemical	B_b[(a)]	Source
1,3,5-Trinitrobenzene	0.0000045	after McKone, 1994
2,4,6-Trinitrotoluene	0.0000045	after McKone, 1994
Aluminum	0.0015	Baes et al., 1984
Anthracene	0.000871	after McKone, 1994
Antimony	0.001	Baes et al., 1984
Arsenic	0.0013	Stevens, 1992
Barium	0.00015	Baes et al., 1984
Benzo(a)pyrene	0.079	after McKone, 1994
Beryllium	0.001	Baes et al., 1984
Cadmium	0.00017	Stevens, 1992
Chloromethane	0.0000002	after McKone, 1994
Chromium	0.0019	Stevens, 1992
Copper	0.01	Stevens, 1992
Lead	0.000067	Stevens, 1992
HMX	0.00000034	after McKone, 1994
Manganese	0.0004	Baes et al., 1984
PCBs	0.0000000046	after McKone, 1994
Phenanthrene	0.000933	after McKone, 1994
Pyrene	0.00832	after McKone, 1994
RDX	0.000000187	after McKone, 1994
Thallium	0.04	Baes et al., 1984
Zinc	0.10	Baes et al., 1984

[a]Biotransfer factor

The soil ingestion rate is 0.3 kg/day (USEPA 1990a). The forage ingestion rate is based on calculations by the U.S. Department of Agriculture's Natural Resource Conservation Service office in Tooele, Utah. The calculations were undertaken to determine stocking rates for the grazing allotments on TEAD-N and assumed forage consumption of 800 pounds per month or 12.12 kg/day. Grazing allotment areas were obtained from the same source (Rust E&I, personal communication from W. Conrad, 1994). SWMU-specific areas were estimated from data collected during the field investigation.

1.3 EXPOSURE MODELS AND PARAMETERS

The models used for estimating RME and CTE through inhalation, ingestion, and dermal contact are described below. In addition, a brief description of the rationale for the exposure parameters used is given following each model.

1.3.1 Inhalation of Particulates

Potential exposure through direct inhalation of SWMU-related chemicals bound to resuspended particulates was evaluated for the hypothetical receptors defined by the exposure scenarios. Both on-site and off-site exposures were estimated based on the duration of exposure, the inhalation rate of the exposed individuals during the exposure, and the concentration of chemicals in the air breathed. The model used for estimating inhalation exposure is shown below:

$$\text{Daily Intake (mg/kg-day)} = \frac{Ca * BA * PPF * IR * ET * EF * ED}{BW * AT}$$

where

Ca	=	Chemical concentration in air (mg/m^3)
BA	=	Bioavailability (unitless)
PPF	=	Particulate penetration factor (unitless)
IR	=	Inhalation rate (m^3/hr)
ET	=	Exposure time (hrs/day)
EF	=	Exposure frequency (days/yr)
ED	=	Exposure duration (years)
BW	=	Body weight (kg)
AT	=	Noncarcinogenic or carcinogenic averaging time (days)

The exposure parameters are described in the following sections and are summarized in Table 4.

Table 4. Exposure Parameters for the Inhalation of Particulates Pathway

Parameters	RME	RME Reference	CTE	CTE Reference
Current/Future SWMU-Specific Laborer				
Ca = Chemical Concentration in Air (mg/m^3)	chemical-specific	NA	chemical-specific	NA
BA = Bioavailability (unitless)	0.5	Paustenbach et al., 1986	0.5	Paustenbach et al., 1986
PPF = Partic. Penetration Factor (unitless)	1.0 - outdoors 0.55 - indoors	Murphy & Yocum, 1986	1.0 - outdoors 0.55 - indoors	Murphy & Yocum, 1986
IR = Inhalation Rate (m^3/hr)	0.9	see text	0.6	see text
ET = Exposure Time (hrs/day)	10	assumed	2	assumed
EF = Exposure Frequency (days/yr)	192	assumed	50	assumed
ED = Exposure Duration (years)	25	AIHC, 1994	3	personal communication, 1994
BW = Body Weight (kg)	72	AIHC, 1994	72	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	9,125	AIHC, 1994	1,095	personal communication, 1994
Carcinogenic	27,375	USEPA, 1990	27,375	USEPA, 1990

Table 4. Exposure Parameters for the Inhalation of Particulates Pathway (continued)

Parameters	RME	RME Reference	CTE	CTE Reference
Future Construction Worker				
Ca = Chemical Concentration in Air (mg/m^3)	chemical-specific	NA	chemical-specific	NA
BA = Bioavailability (unitless)	0.5	Paustenbach et al., 1986	0.5	Paustenbach et al., 1986
PPF = Partic. Penetration Factor (unitless)	1.0	Murphy & Yocum,1986	1.0	Murphy & Yocum,1986
IR = Inhalation Rate (m^3/hr)	1.2	see text	0.8	see text
ET = Exposure Time (hrs/day)	10	assumed	8	assumed
EF = Exposure Frequency (days/yr)	140	assumed	60	assumed
ED = Exposure Duration (years)	3	assumed	1	assumed
BW = Body Weight (kg)	72	AIHC, 1994	72	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	1,095	assumed	365	assumed
Carcinogenic	27,375	USEPA, 1990	27,375	USEPA, 1990

Table 4. Exposure Parameters for the Inhalation of Particulates Pathway (continued)

Parameters	RME	RME Reference	CTE	CTE Reference
Current Off-Site Resident - Adult (Child)				
Ca = Chemical Concentration in Air (mg/m^3)	chemical-specific	NA	chemical-specific	NA
BA = Bioavailability (unitless)	0.5	Paustenbach et al., 1986	0.5	Paustenbach et al., 1986
PPF = Partic. Penetration Factor (unitless)	1.0 - outdoors 0.55 - indoors	Murphy & Yocum, 1986	1.0 - outdoors 0.55 - indoors	Murphy & Yocum, 1986
IR = Inhalation Rate (m^3/hr)	0.7 (1.0)	see text	0.6 (1.0)	see text
ET = Exposure Time (hrs/day)	19 (20) 17.1(17) - indoor 1.9 (3) - outdoor	see text	15 (20) 13.5 (17) - indoor 1.5 (3) - outdoor	see text
EF = Exposure Frequency (days/yr)	350	see text	335	see text
ED = Exposure Duration (years)	30 (18)	USEPA, 1989	8	AIHC, 1994
BW = Body Weight (kg)	72 (47)	AIHC, 1994	72 (32.5)	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	10,950 (6,570)	USEPA, 1989	2,920	AIHC, 1994
Carcinogenic	27,375	USEPA, 1990	27,375	USEPA, 1990

Table 4. Exposure Parameters for the Inhalation of Particulates Pathway (continued)

Parameters	RME	RME Reference	CTE	CTE Reference
Future On-Site Resident - Adult (Child)				
Ca = Chemical Concentration in Air (mg/m^3)	chemical-specific	NA	chemical-specific	NA
BA = Bioavailability (unitless)	0.5	Paustenbach et al., 1986	0.5	Paustenbach et al., 1986
PPF = Partic. Penetration Factor (unitless)	1.0 - outdoors 0.55 - indoors	Murphy & Yocum, 1986	1.0 - outdoors 0.55 - indoors	Murphy & Yocum, 1986
IR = Inhalation Rate (m^3/hr)	0.7 (1.0)	see text	0.6 (1.0)	see text
ET = Exposure Time (hrs/day)	19 (20) 17.1 (17) - indoor 1.9 (3) - outdoor	see text	15 (20) 13.5 (17) - indoor 1.5 (3) - outdoor	see text
EF = Exposure Frequency (days/yr)	350	see text	335	see text
ED = Exposure Duration (years)	30 (18)	USEPA, 1989	8	AIHC, 1994
BW = Body Weight (kg)	72 (47)	AIHC, 1994	72 (32.5)	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	10,950 (6,570)	USEPA, 1989	2,920	AIHC, 1994
Carcinogenic	27,375	USEPA, 1990	27,375	USEPA, 1990

Table 4. Exposure Parameters for the Inhalation of Particulates Pathway (continued)

Parameters	RME	RME Reference	CTE	CTE Reference
Future Recreational Visitor				
Ca = Chemical Concentration in Air (mg/m^3)	chemical-specific	NA	chemical-specific	NA
BA = Bioavailability (unitless)	0.5	Paustenbach et al. 1986	0.5	Paustenbach et al., 1986
PPF = Partic. Penetration Factor (unitless)	1.0 - outdoors	Murphy & Yocum, 1986	1.0 - outdoors	Murphy & Yocum, 1986
IR = Inhalation Rate (m^3/hr)	1.25	USEPA, 1989	0.75	AIHC, 1994
ET = Exposure Time (hrs/day)	4.5	assumed	2.5	assumed
EF = Exposure Frequency (days/yr)	52	assumed	26	assumed
ED = Exposure Duration (years)	30	USEPA, 1989	8	AIHC, 1994
BW = Body Weight (kg)	72	AIHC, 1994	72	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	10,950	USEPA, 1989	2,920	AIHC, 1994
Carcinogenic	27,375	USEPA, 1989	27,375	USEPA, 1990

1.3.1.1 *Chemical Concentration in Air (Ca)*

Chemical concentrations in air were based on the RME and CTE concentrations in soils and the maximum particulate concentration as predicted by the air dispersion modeling (Section 1.1). Receptors were assumed to be located at the estimated maximum ground level concentration. Only respirable particulates (i.e., particulates less than 10μm in aerodynamic diameter (PM_{10})) were modeled. The results of the air dispersion modeling and the apportionment among the COPCs is described in Appendix N.

Studies have shown that there can be marked differences in indoor and outdoor concentrations of particulates or among microenvironments in the same area. Indoor concentrations of particulates are expected to be less than those found outdoors. Two factors contribute to the reduction of long-term indoor concentrations of SWMU-related particulates relative to outdoor concentrations. The first is due to the fact that larger particles will become lodged in the exterior of buildings without transport into the interior. The second is due to deposition on indoor surfaces such as rugs, draperies and furniture. To compensate for the uncertainty involved in estimating the magnitude of these two phenomena and maintain a bias toward health protection in the risk assessment, particulate deposition was assumed to be negligible. Therefore, indoor concentrations of SWMU-related particulates was assumed to depend only upon outdoor concentrations and the size-dependent particulate penetration factor.

The particle penetration factor varies with particle size. It is also dependent on whether windows are open or closed. Because no data were available relative to particle size distribution at TEAD-N, it was assumed that the volume of particulates with diameter less than 1 μm was equal to the volume greater than 1μm. It was also assumed that windows were open 6 months out of the year. Given the severity of Utah winters, the latter assumption is likely conservative. Using the above assumptions and the data of Murphy and Yocum (1986), an average particle penetration factor of 0.55 was derived.

1.3.1.2 *Bioavailability (BA)*

For volatile emissions, the entire quantity inhaled is potentially available for absorption through the lungs. However, for particulate emissions, less than the entire quantity inhaled actually contacts the lung and is, therefore, available for absorption.

A bioavailability factor of 0.5 was assumed based on experimental work with dioxin (Paustenbach et al. 1986). This work showed that approximately 50 percent of respirable particles are available for absorption. The rest are deposited in the upper airways, where absorption does not occur, or expired.

1.3.1.3 *Exposure Time (ET)*

The exposure time is defined as the time a receptor is potentially exposed to chemicals and is measured in unit of hours per day. Specific exposure times were developed for each receptor and are described below.

1.3.1.3.1 ***Current/Future SWMU-Specific Laborer***. Because the majority of laborers at TEAD-N work 10-hour-days, the assumed exposure time is 10 hrs/day for the RME scenario. For all SWMUs except the RME scenario for SWMU 23, it is assumed that all of the exposure is from outdoor activities. For SWMU 23, the RME exposure time is comprised of 1 hour spent outdoors and the remaining hours spent indoors. For the CTE scenarios, it is assumed that the laborer, either military or civilian, has duties that require 2 hrs/day at the site, all of which is spent outdoors.

1.3.1.3.2 ***Future Construction Worker***. Under the CTE scenario it is assumed that the construction worker is at the site for a total of 8 hours/day, all of which is spent outdoors. The construction worker is assumed to work 10 hour-days, with minimal breaks, for the RME scenario (all outdoors).

1.3.1.3.3 ***Current Off-Site Resident***. The time spent at home or away from home by adults varies by age and sex. Part of this variation is attributed to the difference in time spent at work. The time-activity data collected by the University of Michigan (Robinson 1977) were arranged into ten broad categories including: market work, house/yard work, child care, services/shopping, personal care, education, organizations, social entertainment, active leisure, and passive leisure. Based on these activities, the weighted mean hours per week at home/away from home were estimated at 108/64 for men and women combined (AIHC 1994). This value is used to represent the CTE scenario.

If the adult works at another location, a time at work adjustment of 23/168 (hours at work/total hours a week) is also included (AIHC 1994). Therefore, if the RME scenario is represented by an at-home worker, the time at home is increased by 23 hours/week to 131 hours/week.

Activity pattern data for children are presented in the USEPA (1990b). While such data are limited, they suggest that on the average, children spend approximately 30 hours/week away from home, engaged in activities such as shopping, church, school, and visiting (AIHC 1994).

A number of investigators have attempted to determine the amount of time spent indoors versus the amount of time spent outdoors. Using the time-activity data from the studies conducted by Chapin (1974) and Szalai (1972), the activities are classified as outdoor, transit, or indoors (AIHC 1994). Mean time values were added and percent of daily time in each location was estimated for the amount of time spent outdoor and in transit. All remaining time was assumed to be spent

indoors. The percent of daily time spent outdoors, in transit, or indoors was 3, 7, and 90 for adults, 5, 6, and 89 for adolescents and 9, 5, and 86 for children (AIHC 1994).

Based on this information, the exposure time for the CTE is estimated assuming the adult (both parents) work outside the home. For the RME scenario, it is assumed that the one parent does not work and stays at home conducting normal living activities. The exposure times for both the CTE and RME scenario are estimated as follows:

$$ET_{CTE} = \left(\frac{168 \text{ hrs total}}{\text{week}} - \frac{60 \text{ hour}}{\text{week}}\right) * \frac{\text{week}}{7 \text{ days}} \cong 15 \text{ hrs/day}$$

$ET_{CTE\text{-}indoors}$ = 15 hrs/day x 0.90 ≅ 13.5 hrs/day
$ET_{CTE\text{-}outdoors}$ = 15 hrs/day x 0.10 ≅ 1.5 hr/day

$$ET_{RME} = \left(\frac{168 \text{ hrs total}}{\text{week}} - \frac{37 \text{ hour}}{\text{week}}\right) * \frac{\text{week}}{7 \text{ days}} \cong 19 \text{ hrs/day}$$

$ET_{RME\text{-}indoors}$ = 19 hrs/day x 0.90 ≅ 17.1 hrs/day
$ET_{RME\text{-}outdoors}$ = 19 hrs/day x 0.10 ≅ 1.9 hr/day

For the child, age 0 to 18, the exposure time for the CTE and RME scenario is the same because it is assumed that the child will be required to attend a school or day care at a location other than their home. The indoor/outdoor break out is estimated below.

$$ET_{CTE/RME} = \left(\frac{168 \text{ hrs total}}{\text{week}} - \frac{30 \text{ hour}}{\text{week}}\right) * \frac{\text{week}}{7 \text{ days}} \cong 20 \text{ hrs/day}$$

$ET_{CTE/RME\text{-}indoors}$ = 20 hrs/day x 0.86 ≅ 17 hrs/day
$ET_{CTE/RME\text{-}outdoors}$ = 20 hrs/day x 0.14 ≅ 3 hr/day

1.3.1.3.4 ***Future On-Site Resident***. The exposure times for all future on-site residential receptors are assumed to be identical to those assumed for current off-site residents.

1.3.1.3.5 ***Future Recreational Visitor***. Under the CTE scenario it is assumed that the recreational visitor or golfer will play a 9-hole game of golf. Playing a 9-hole golf game is estimated to require 2.5 hours. For the RME scenario, the golfer is assumed to play a 18-hole golf game estimated to last approximately 4.5 hours.

1.3.1.4 *Inhalation Rate (IR)*

The inhalation rate is dependent upon age, sex, and activity level. Specific rates were developed for each receptor and are described below.

1.3.1.4.1 ***Current/Future SWMU-Specific Laborer.*** The SWMU-specific laborer is exposed to surface soil during work activities. The inhalation rate under the CTE scenario was estimated assuming that the laborer is exposed for a total of 2 hours/day conducting "light effort", such as minor repairs. The inhalation rate is 0.6 m^3/hr (USEPA 1990b).

The inhalation rate under the RME scenario was estimated assuming that the laborer is exposed for a total of 10 hours/day. Two hours are spent outdoors engaged in "moderate effort" and 6 hours are spent either indoors or outdoors conducting "light effort" for a total exposure time of 8 hours (USEPA 1990b).

$$IR_{RME} = (\frac{2}{10})(2.1\ m^3/hr) + (\frac{6}{10})(0.6\ m^3/hr) \cong 0.9\ m^3/hr$$

1.3.1.4.2 ***Future Construction Worker.*** The construction worker is exposed to subsurface soil during construction activities. The inhalation rate under the CTE scenario was estimated assuming that the worker is exposed for a total 8 hours/day of which 1 hour is spent conducting "moderate effort", such as major repairs or alterations and 7 hours are spent conducting "light effort", such as cleanup or minor repairs (USEPA 1990b). The inhalation rate is estimated as follows:

$$IR_{CTE} = (\frac{1}{8})(2.1\ m^3/hr) + (\frac{7}{8})(0.6\ m^3/hr) \cong 0.8\ m^3/hr$$

The inhalation rate under the RME scenario is estimated the same as above except that 1 hour is spent conducting "heavy" effort, 2 hours are spent conducting "moderate effort", and 7 hours are spent conducting "light effort" for a total exposure time of 10 hours (USEPA 1990b).

$$IR_{RME} = (\frac{1}{10})(3.9\ m^3/hr) + (\frac{2}{10})(2.1\ m^3/hr) + (\frac{7}{10})(0.6\ m^3/hr) \cong 1.2\ m^3/hr$$

1.3.1.4.3 ***Current Off-Site Resident.*** The off-site adult resident is exposed to particulates generated from surface soil while residing at home. The inhalation rate under the CTE scenario was estimated assuming that the adult resident is at home for a total of 15 hours/day of which 1 hour is spent outdoors conducting "light to moderate" effort, such as vigorous gardening or snow

shovelling, 6 hours indoors conducting "resting to light" effort, such as cooking or watching television, and 8 hours sleeping (USEPA 1990b). The inhalation rate is estimated as follows:

$$IR_{CTE} = (\frac{1}{15})(1.4\ m^3/hr) + (\frac{6}{15})(0.6\ m^3/hr) + (\frac{8}{15})(0.5 m^3/hr) \cong 0.6\ m^3/hr$$

The inhalation rate under the RME scenario is estimated the same as above except that 0.5 hours are spent conducting "heavy" effort, 1 hour is spent conducting "moderate" effort, 9.5 hours are spent conducting "light" effort, and 8 hours sleeping for a total exposure time of 19 hours (USEPA 1990b).

$$IR_{RME} = (\frac{0.5}{19})(3.9\ m^3/hr) + (\frac{1}{19})(2.1\ m^3/hr) +$$

$$(\frac{9.5}{19})(0.6\ m^3/hr) + (\frac{8}{19})(0.5\ m^3/hr) \cong 0.7\ m^3/hr$$

For the child resident, the inhalation rate under the CTE scenario was estimated assuming that the child resident is at home for a total of 20 hours/day of which 3 hours are spent outdoors conducting "moderate" effort, such as sports, 9 hours are spent indoors conducting "resting to light" effort, such as playing a game or watching television, and 8 hours sleeping (USEPA 1990b). The inhalation rate is estimated as follows:

$$IR_{CTE} = (\frac{3}{20})(2.6\ m^3/hr) + (\frac{9}{20})(0.9\ m^3/hr) + (\frac{8}{20})(0.4\ m^3/hr) \cong 1.0\ m^3/hr$$

The inhalation rate under the RME scenario is estimated the same as above except that 1 hour is spent conducting "heavy" effort, 2 hours is spent conducting "moderate" effort, 5.5 hours are spent conducting "light" effort, and 8 hours sleeping for a total exposure time of 20 hours (USEPA 1990b).

$$IR_{RME} = (\frac{1}{20})(3.3\ m^3/hr) + (\frac{2}{20})(2.6\ m^3/hr) + (\frac{9}{20})(0.9\ m^3/hr) +$$

$$(\frac{8}{20})(0.4\ m^3/hr) \cong 1.0\ m^3/hr$$

1.3.1.4.4 ***Future On-Site Resident***. The on-site resident is exposed to particulates generated from surface soil while residing at home. The inhalation rates for all receptors are assumed to be identical to those assumed for current off-site residents.

1.3.1.4.5 ***Future Recreational Visitor***. The recreational visitor is exposed to particulates generated from surface soil while golfing at the site. Under the CTE scenario, an inhalation rate of 18 m^3/day is assumed (AIHC 1994). This value was estimated based on the combined average adult inhalation rates for men and women listed in USEPA (1990) assuming a normal daily activity pattern. The inhalation rate under the RME scenario was estimated assuming the upper-bound inhalation rate of 30 m^3/day (USEPA 1989). These rates were then converted to 0.75 and 1.25 m^3/hr, respectively.

1.3.1.5 ***Exposure Frequency (EF)***

The exposure frequency is defined as the time a receptor is potentially exposed to contaminants and is measured in unit of days or events per year. Specific exposure frequencies were developed for each receptor and are described below.

1.3.1.5.1 ***Current/Future SWMU-Specific Laborer***. Most laborers at TEAD-N currently work 4-day-weeks. For the RME, as a worst-case estimate, it is could be assumed that the laborer is working at one particular SWMU the entire working year or 192 days/yr. This exposure frequency is developed assuming that the laborer spends approximately 4 weeks on vacation, sick leave, and holidays away from the site.

For the CTE, it is assumed that the laborer is working on various assignments at various SWMUs through out the year. Therefore, the time that the laborer will spend at any given SWMU is estimated to be 50 days/year.

1.3.1.5.2 ***Future Construction Worker***. For the CTE, it is assumed that the worker is working at the site for an exposure frequency of 60 days, equivalent to a 3-month construction project. For the RME, it is assumed that the worker is working at the site for an exposure frequency of 140 days or a 6-month construction project.

1.3.1.5.3 ***Current Off-Site Resident*** . The exposure frequency for the adult/child resident is estimated based on the exposure time, as discussed above, and vacation/holiday time spent away from home. It is assumed that the adult resident spends approximately 4 weeks away from home on vacation and long holiday weekends. Therefore, the exposure frequency is approximately 335 days/year.

For the RME scenario, the exposure frequency for the adult/child resident is estimated the same way as the CTE, however the vacation/holiday time spent away from home is reduced to 2 weeks/yr. The exposure frequency for the RME scenario is approximately 350 days/year.

1.3.1.5.4 ***Future On-Site Resident***. The exposure frequency for the future on-site resident is assumed to equal that of the current off-site resident scenario.

1.3.1.5.5 ***Future Recreational Visitor***. It is assumed the recreational visitor or golfer will play a game of golf every week or at least once every two weeks on a course at the site for the RME and CTE scenario, respectively.

1.3.1.6 ***Exposure Duration (ED)***

Exposure duration is defined as the total length of time a receptor may be exposed to the emissions. Specific exposure times were developed for each receptor and are described below.

1.3.1.6.1 ***Current/Future SWMU-Specific Laborer***. Military personal are rotated on assignment an average of every 3 years (personal communication, 1994). Therefore, for the CTE scenario, it is assumed that the worker tenure for military staff will not exceed three years. Under the RME scenario, it is proposed that the worker is a civilian and the exposure duration is 25 years (AIHC 1994). According to data from the Bureau of Labor Statistics (1987), 25 years is the upper 95th percentile of the distribution for number of years spent at a specific job.

1.3.1.6.2 ***Future Construction Worker***. It is assumed that a single construction company could obtain a contract for construction at the same location or SWMU for an extended period of time. For the CTE scenario, it is assumed that a worker would spent a maximum of 1 year at the work site or SWMU. For a large construction project, it is conceivable that a construction worker could spent up to 3 years at the same location (RME scenario).

1.3.1.6.3 ***Current Off-Site Resident***. For the CTE scenario, years spent at one residence for the adult/child is assumed to be 8 years (AIHC 1994). The upper-bound 95th percentile of 30 years is used for the RME scenario (USEPA 1989). For the child, the exposure duration is 18 years which is based on a child growing up in the same residence and then leaving for further education or work opportunities.

1.3.1.6.4 ***Future On-Site Resident***. For the CTE scenario, years spent at one residence for the adult/child is assumed to be 8 years (AIHC 1994). The upper-bound 95th percentile of 30 years is used for the RME scenario (USEPA 1989). For the child, the exposure duration is 18 years which is based on a child growing up in the same residence and then leaving for further education or work opportunities.

1.3.1.6.5 ***Future Recreational Visitor***. For the CTE scenario, years spent at one residence for the adult is assumed to be 8 years (AIHC 1994). The upper-bound 95th percentile of 30 years is used for the RME scenario (USEPA 1989).

1.3.1.7 *Body Weight (BW)*

Estimated average body weights of adults is 72 kg (AIHC 1994). This value is the average of the mean 50th percentile values for adults (men and women) across the age spectrum of 18 to 75 years listed in USEPA (1990b). The estimated average body weight of children both male and female, ages 0 to 18, based on the 50th and 95th percentile is 32.5 kg and 47 kg, respectively for the CTE and RME scenario (USEPA 1990b). The estimated average body weight of juveniles both male and female, ages 6 to 12, based on the 50th and 95th percentile is 29 kg and 45 kg, for the CTE and RME, respectively (USEPA 1990b).

1.3.1.8 *Averaging Time (AT)*

The averaging time used is dependent on the toxicological endpoint of the chemical of concern. For chemicals that are thought to be potential carcinogens, intakes were averaged over the estimated lifetime of each receptor, which is 27,375 days (75 years x 365 days/year). For systemic toxicants, intakes were averaged over the estimated duration of exposure specific to each receptor exposure scenario (i.e., ED (in years) • 365 days).

The distinction between carcinogens and systemic toxicants is based on the theory that the mechanism of action for these two categories is different. It is assumed that a high dose of a carcinogen received over a short period is equivalent to a low dose spread over a lifetime. Systemic toxicants are assumed to have a threshold below which no toxic effect is observed. Therefore, intakes are averaged over the actual exposure duration.

1.3.2 Ingestion of Soil

Exposure to soil through ingestion was estimated based on the duration of exposure, the ingestion rate during exposure, and the concentration of chemicals in the material ingested. The model used for estimating ingestion exposure is shown below:

$$\text{Daily Intake (mg/kg)} = \frac{Cs * IR * FI * EF * ED * 1E\text{-}06\ (kg/mg)}{BW * AT}$$

where

Cs	=	Chemical concentration in soil (mg/kg)
IR	=	Ingestion rate (mg/day)
FI	=	Fraction ingested from source medium (unitless)
EF	=	Exposure frequency (days/yr)
ED	=	Exposure duration (years)
BW	=	Body weight (kg)
AT	=	Carcinogenic or noncarcinogenic averaging time (days)

The exposure parameters are described in the following sections and summarized in Table 5.

1.3.2.1 *Chemical Concentration in Soil (Cs)*

The chemical concentration for each constituent in soil was derived by evaluating the Phase I and II Remedial Investigation data. The CTE and RME concentrations are based on the upper 95th percentile confidence limit on the arithmetic mean (USEPA 1989, 1991). For a more detailed description of the rationale used to develop these concentrations, the reader is referred to the exposure point concentrations sections for each SWMU.

1.3.2.2 *Ingestion Rate (IR)*

The ingestion rate is dependent upon age, sex, and activity level. Specific rates were developed for each receptor and are described below.

1.3.2.2.1 ***Current/Future SWMU-Specific Laborer***. Under the CTE and RME scenarios, the ingestion rate of soil is 10 and 50 mg/day, respectively (AIHC 1994; Finely et al. 1994).

1.3.2.2.2 ***Future Construction Worker***. Under the CTE and RME scenarios, the ingestion rate of soil is 240 and 480 mg/day, respectively (USEPA 1990b). For both scenarios, the adult is assumed to engage in outdoor physical activity. The estimate is based on ingesting a 50 μm-thick layer of soil from the inside surfaces of the fingers and thumb of one hand twice daily for the RME scenario and once for the CTE scenario (USEPA 1990b).

1.3.2.2.3 ***Future On-Site Resident***. The ingestion rate of soil for children is 16 and 110 mg/day for the CTE and RME scenarios, respectively (Finley et al. 1994). These rate are based on the 50th and 95th percentile for children. For the adults, the soil ingestion rate is 10 and 50 mg/day for the CTE and RME scenarios, respectively (AIHC 1994; Finley et al. 1994).

1.3.2.2.4 ***Future Recreational Visitor***. The CTE and RME ingestion rate for the recreational visitor or golfer is assumed to be 10 mg/day (AIHC 1994).

1.3.2.3 *Fraction Ingested from Source Medium*

The fraction ingested (FI) is defined as the percentage of soil ingested from the source medium over the total waking exposure period (USEPA 1989). Specific exposure fractions were developed for each receptor and are described below.

Table 5. Exposure Parameters for the Ingestion of Soil Pathway

Parameters	RME	RME Reference	CTE	CTE Reference
Current/Future SWMU-Specific Laborer				
Cs = Chemical Concentration in Soil (mg/kg)	chemical-specific	NA	chemical-specific	NA
IR = Ingestion Rate (mg/day)	50	Finley et al., 1994	10	AIHC, 1994
FI = Fraction Ingested from Source Medium (unitless)	0.625	assumed	0.125	assumed
EF = Exposure Frequency (days/yr)	192	assumed	50	assumed
ED = Exposure Duration (years)	25	AIHC, 1994	3	personal communication, 1994
BW = Body Weight (kg)	72	AIHC, 1994	72	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	9,125	AIHC, 1994	1,095	personal communication, 1994
Carcinogenic	27,375	USEPA, 1990	27,375	USEPA, 1990
Future Construction Worker				
Cs = Chemical Concentration in Soil (mg/kg)	chemical-specific	NA	chemical-specific	NA
IR = Ingestion Rate (mg/day)	480	USEPA, 1990	240	USEPA, 1990
FI = Fraction Ingested from Source Medium (unitless)	1.0	assumed	1.0	assumed

Table 5. Exposure Parameters for the Ingestion of Soil Pathway (continued)

Parameters	RME	RME Reference	CTE	CTE Reference
EF = Exposure Frequency (days/yr)	140	assumed	60	assumed
ED = Exposure Duration (years)	3	assumed	1	assumed
BW = Body Weight (kg)	72	AIHC, 1994	72	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	1,095	assumed	365	assumed
Carcinogenic	27,375	USEPA, 1990	27,375	USEPA, 1990
Future On-Site Resident - Adult (Child)				
Cs = Chemical Concentration in Soil (mg/kg)	chemical-specific	NA	chemical-specific	NA
IR = Ingestion Rate (mg/day)	50 (110)	Finley et al., 1994	10 (16)	AIHC, 1994 (Finley et al., 1994)
FI = Fraction Ingested from Source Medium (unitless)	1.0	assumed	1.0	assumed
EF = Exposure Frequency (days/yr)	273 (288)	see text	216 (276)	see text
ED = Exposure Duration (years)	30 (18)	USEPA, 1989	8	AIHC, 1994
BW = Body Weight (kg)	72 (47)	AIHC, 1994	72 (32.5)	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	10,950 (6,570)	USEPA, 1989	2,920	AIHC, 1994
Carcinogenic	27,375	USEPA, 1990	27,375	USEPA, 1990

Table 5. Exposure Parameters for the Ingestion of Soil Pathway (continued)

Parameters	RME	RME Reference	CTE	CTE Reference
Future Recreational Visitor				
Cs = Chemical Concentration in Soil (mg/kg)	chemical-specific	NA	chemical-specific	NA
IR = Ingestion Rate (mg/day)	10	AIHC, 1994	10	AIHC, 1994
FI = Fraction Ingested from Source Medium (unitless)	1.0	assumed	1.0	assumed
EF = Exposure Frequency (days/yr)	52	assumed	26	assumed
ED = Exposure Duration (years)	30	USEPA, 1989	8	AIHC, 1994
BW = Body Weight (kg)	72	AIHC, 1994	72	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	10,950	USEPA, 1989	2,920	AIHC, 1994
Carcinogenic	27,375	USEPA, 1989	27,375	USEPA, 1990

1.3.2.3.1 ***Current/Future SWMU-Specific Laborer***. It is assumed that the majority of laborers at TEAD-N work 2- to 10-hour-days at one particular SWMU. In addition, the soil ingestion rate is based on the total waking hours in a day and is not specific to laborer activities. Therefore, assuming 16 hours per day as the total waking hours in a day which a person is active, the RME and CTE ingestion fraction is 0.625 and 0.125, respectively.

1.3.2.3.2 ***Future Construction Worker***. The soil ingestion rate for the construction worker scenario is based on specific outdoor activities and does not include incidental soil ingestion while conducting other activities, such as working in the garden, home maintenance, or food preparation, during the total waking hour exposure period. Therefore, an ingestion fraction was not applied to this receptor.

1.3.2.3.3 ***Future On-Site Resident***. The soil ingestion rate for the on-site resident is based on incidental soil ingestion while conducting activities, such as working in the garden, home maintenance, or food preparation, during the total waking hour exposure period. Therefore, an ingestion fraction was not applied to this receptor.

1.3.2.3.4 ***Future Recreational Visitor***. The soil ingestion rate for the recreational visitor is based on incidental soil ingestion while conducting activities, such as golfing. Therefore, an ingestion fraction was not applied to this receptor.

1.3.2.4 ***Exposure Frequency (EF)***

The exposure frequency is defined as the time a receptor is potentially exposed to chemicals and is measured in unit of days or events per year. Specific exposure frequencies were developed for each receptor and are described below.

1.3.2.4.1 ***Current/Future SWMU-Specific Laborer***. Most laborers at TEAD-N currently work 4-day-weeks. For the RME, as a worst-case estimate, it is could be assumed that the laborer is working at one particular SWMU the entire working year or 192 days/yr. This exposure frequency is developed assuming that the laborer spends approximately 4 weeks on vacation, sick leave, and holidays away from the site.

For the CTE, it is assumed that the laborer is working on various assignments at various SWMUs throughout the year. Therefore, the time that the laborer will spending at any given SWMU is estimated to be 50 days/year.

1.3.2.4.2 ***Future Construction Worker***. For the CTE, it is assumed that the worker is working at the site for an exposure frequency of 60 days, equivalent to a 3-month construction project.

For the RME, it is assumed that the worker is working at the site for an exposure frequency of 140 days or a 6-month construction project.

1.3.2.4.3 ***Future On-Site Resident***. The exposure frequency for the adult/child resident is estimated based on the exposure time, as discussed above, and vacation/holiday time spent away from home. Because the ingestion rate for soil is in units of mass per day, the exposure time is pro-rated to 24-hour day equivalents.

It is assumed that the adult resident spends approximately 4 weeks away from home on vacation and long holiday weekends. The exposure frequency for the CTE scenario is estimated as follows:

$$EF_{adult\text{-}CTE} = \left(\frac{108\ hrs}{week}\right)\left(\frac{52\ weeks - 4\ weeks}{yr}\right)\left(\frac{day}{24\ hr}\right) \cong 216\ days/yr$$

$$EF_{child\text{-}CTE} = \left(\frac{138\ hrs}{week}\right)\left(\frac{52\ weeks - 4\ weeks}{yr}\right)\left(\frac{day}{24\ hr}\right) \cong 276\ days/yr$$

For the RME scenario, the exposure frequency for the adult/child resident is estimated the same way as the CTE, however the vacation/holiday time spent away from home is reduced to 2 weeks/yr. The exposure frequency for the RME scenario is estimated as follows:

$$EF_{adult\text{-}RME} = \left(\frac{131\ hrs}{week}\right)\left(\frac{52\ weeks - 2\ weeks}{yr}\right)\left(\frac{day}{24\ hr}\right) \cong 273\ days/yr$$

$$EF_{child\text{-}RME} = \left(\frac{138\ hrs}{week}\right)\left(\frac{52\ weeks - 2\ weeks}{yr}\right)\left(\frac{day}{24\ hr}\right) \cong 288\ days/yr$$

1.3.2.4.4 ***Future Recreational Visitor***. It is assumed the recreational visitor or golfer will play golf every week or at least once every two weeks on a course at the site for the RME and CTE scenario, respectively.

1.3.2.5 *Exposure Duration (ED)*

Exposure duration is defined as the total length of time a receptor may be exposed to the soil. Specific exposure times were developed for each receptor and are described below.

1.3.2.5.1 ***Current/Future SWMU-Specific Laborer***. Military personal are rotated on assignment an average of every 3 years (personal communication, 1994). Therefore, for the CTE scenario, it is assumed that the worker tenure for military staff will not exceed three years. Under the RME scenario, it is proposed that the worker is a civilian and the exposure duration is 25 years (AIHC 1994). According to data from the U.S. Department of Labor (1987), 25 years is the upper 95th percentile of the distribution for number of years spent at a specific job.

1.3.2.5.2 ***Future Construction Worker***. It is assumed that a single construction company could obtain a contract for construction at the same location or SWMU for an extended period of time. For the CTE scenario, it is assumed that a worker would spent a maximum of 1 year at the work site or SWMU. For a large construction project, it is conceivable that a construction worker could spent up to 3 years at the same location (RME scenario).

1.3.2.5.3 ***Future On-Site Resident***. For the CTE scenario, years spent at one residence for the adult/child is assumed to be 8 years (AIHC 1994). The upper-bound 95th percentile of 30 years is used for the RME scenario (USEPA 1989). For the child, the exposure duration is 18 years which is based on a child growing up in the same residence and then leaving for further education or work opportunities.

1.3.2.5.4 ***Future Recreational Visitor***. For the CTE scenario, years spent at one residence for the adult is assumed to be 8 years (AIHC 1994). The upper-bound 95th percentile of 30 years is used for the RME scenario (USEPA 1989).

1.3.2.6 *Body Weight (BW)*

Estimated average body weights of adults is 72 kg (AIHC 1994). This value is the average of the mean 50th percentile values for adults (men and women) across the age spectrum of 18 to 75 years listed in USEPA (1990). The estimated average body weight of children both male and female, ages 0 to 18, based on the 50th and 95th percentile is 32.5 kg and 47 kg, respectively for the CTE and RME scenario (USEPA 1990b). The estimated average body weight of juveniles both male and female, ages 6 to 12, based on the 50th and 95th percentile is 29 kg and 45 kg, for the CTE and RME, respectively (USEPA 1990b).

1.3.2.7 *Averaging Time (AT)*

The averaging time used is dependent on the toxicological endpoint of the chemical of concern. For chemicals that are thought to be potential carcinogens, intakes were averaged over the estimated lifetime of each receptor, which is 27,375 days (75 years x 365 days/year). For systemic toxicants, intakes were averaged over the estimated duration of exposure specific to each receptor exposure scenario (i.e., ED • 365 days).

The distinction between carcinogens and systemic toxicants is based on the theory that the mechanism of action for these two categories is different. It is assumed that a high dose of a carcinogen received over a short period is equivalent to a low dose spread over a lifetime. Systemic toxicants are assumed to have a threshold below which no toxic effect is observed. Therefore, intakes are averaged over the actual exposure duration.

1.3.3 Dermal Contact with Soil Pathway

Exposure to soil through dermal contact was estimated based on the duration and frequency of exposure, the skin surface area exposed, and the concentration of chemicals in the material absorbed. The model used for estimating dermal exposure is shown below:

$$\text{Daily Dose (mg/kg-day)} = \frac{Cs * FI * SA * AF * ABS * EF * ED * 1E\text{-}06\ (kg/mg)}{BW * AT}$$

where

Cs	=	Chemical concentration in soil (mg/kg)
FI	=	Fraction from source medium (unitless)
SA	=	Surface area (cm^2/day)
AF	=	Adherence factor (mg/cm^2)
ABS	=	Absorption factor (unitless)
EF	=	Exposure factor (days/yr)
ED	=	Exposure duration (years)
BW	=	Body weight (kg)
AT	=	Noncarcinogenic or carcinogenic averaging time (days)

The exposure parameters are described in the following sections and summarized in Table 6.

1.3.3.1 *Chemical Concentration in Soil (Cs)*

The chemical concentration for each constituent in soil was derived by evaluating the Phase I and II Remedial Investigation data. The CTE and RME concentrations are based on the upper 95th percentile confidence limit on the arithmetic mean (USEPA 1989, 1991). For a more detailed description of the rationale used to develop these concentrations, the reader is referred to the exposure point concentrations sections for each SWMU.

Table 6. Exposure Parameters for the Dermal Contact with Soil Pathway

Parameters	RME	RME Reference	CTE	CTE Reference
Current/Future SWMU-Specific Laborer				
Cs = Chemical Concentration in Soil (mg/kg)	chemical-specific	NA	chemical-specific	NA
FI = Fraction Ingested from Source Medium (unitless)	0.625	assumed	0.125	assumed
SA = Exposed Surface Area (cm^2/event)	3,420	AIHC, 1994	2,000	USEPA, 1990
AF = Adherence Factor (mg/cm^2)	1.7	Finley et al., 1994	0.25	Finley et al., 1994
ABS = Absorption Factor (unitless)	chemical-specific	NA	chemical-specific	NA
EF = Exposure Frequency (days/yr)	192	assumed	50	assumed
ED = Exposure Duration (years)	25	AIHC, 1994	3	personal communication, 1994
BW = Body Weight (kg)	72	AIHC, 1994	72	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	9,125	AIHC, 1994	1,095	personal communication, 1994
Carcinogenic	27,375	USEPA, 1990	27,375	USEPA, 1990

Table 6. Exposure Parameters for the Dermal Contact with Soil Pathway (continued)

Parameters	RME	RME Reference	CTE	CTE Reference
Future Construction Worker				
Cs = Chemical Concentration in Soil (mg/kg)	chemical-specific	NA	chemical-specific	NA
FI = Fraction Ingested from Source Medium (unitless)	1.0	assumed	1.0	assumed
SA = Exposed Surface Area (cm^2/event)	5,000	AIHC 1994	3,420	AIHC, 1994
AF = Adherence Factor (mg/cm^2)	1.7	Finley et al., 1994	0.25	Finley et al., 1994
ABS = Absorption Factor (unitless)	chemical-specific	NA	chemical-specific	NA
EF = Exposure Frequency (days/yr)	140	assumed	60	assumed
ED = Exposure Duration (years)	3	assumed	1	assumed
BW = Body Weight (kg)	72	AIHC, 1994	72	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	1,095	assumed	365	assumed
Carcinogenic	27,375	USEPA, 1990	27,375	USEPA, 1990
Future On-Site Resident - Adult (Child)				
Cs = Chemical Concentration in Soil (mg/kg)	chemical-specific	NA	chemical-specific	NA
FI = Fraction Ingested from Source Medium (unitless)	1.0	assumed	1.0	assumed

Table 6. Exposure Parameters for the Dermal Contact with Soil Pathway (continued)

Parameters	RME	RME Reference	CTE	CTE Reference
SA = Exposed Surface Area (cm^2/event)	3,420 (1,480)	AIHC, 1994	2,000 (1,190)	USEPA, 1990 (AIHC, 1994)
AF = Adherence Factor (mg/cm^2)	1.7	Finley et al., 1994	0.25	Finley et al., 1994
ABS = Absorption Factor (unitless)	chemical-specific	NA	chemical-specific	NA
EF = Exposure Frequency (days/yr)	273 (288)	see text	216 (276)	see text
ED = Exposure Duration (years)	30 (18)	USEPA, 1989	8	AIHC, 1994
BW = Body Weight (kg)	72 (47)	AIHC, 1994	72 (32.5)	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	10,950 (6,570)	USEPA, 1989	2,920	AIHC, 1994
Carcinogenic	27,375	USEPA, 1990	27,375	USEPA, 1990
Future Recreational Visitor				
Cs = Chemical Concentration in Soil (mg/kg)	chemical-specific	NA	chemical-specific	NA
FI = Fraction Ingested from Source Medium (unitless)	1.0	assumed	1.0	assumed
SA = Exposed Surface Area (cm^2/event)	3,420	AIHC, 1994	3,420	AIHC, 1994
AF = Adherence Factor (mg/cm^2)	1.7	Finley et al., 1994	0.25	Finley et al., 1994
ABS = Absorption Factor (unitless)	chemical-specific	NA	chemical-specific	NA
EF = Exposure Frequency (days/yr)	52	assumed	26	assumed

Table 6. Exposure Parameters for the Dermal Contact with Soil Pathway (continued)

Parameters	RME	RME Reference	CTE	CTE Reference
ED = Exposure Duration (years)	30	USEPA, 1989	8	AIHC, 1994
BW = Body Weight (kg)	72	AIHC, 1994	72	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	10,950	USEPA, 1989	2,920	AIHC, 1994
Carcinogenic	27,375	USEPA, 1989	27,375	USEPA, 1990

1.3.3.2 *Fraction Ingested from Source Medium*

The fraction ingested (FI) is defined as the percentage of soil ingested based on dermal exposure from the source medium over the total waking exposure period (USEPA 1989). Specific exposure fractions were developed for each receptor and are described below.

1.3.3.2.1 ***Current/Future SWMU-Specific Laborer***. It is assumed that the majority of laborers at TEAD-N work 2- to 10-hour-days at one particular SWMU. In addition, the soil ingestion rate is based on the total waking hours in a day and is not specific to laborer activities. Therefore, assuming 16 hours per day as the total waking hours in a day which a person is active, the RME and CTE ingestion fraction is 0.625 and 0.125, respectively.

1.3.3.2.2 ***Future Construction Worker***. The soil ingestion rate for the construction worker scenario is based on specific outdoor activities and does not include incidental soil ingestion while conducting other activities, such as working in the garden, home maintenance, or food preparation, during the total waking hour exposure period. Therefore, an ingestion fraction was not applied to this receptor.

1.3.3.2.3 ***Future On-Site Resident***. The soil ingestion rate for the on-site resident is based on incidental soil ingestion while conducting activities, such as working in the garden, home maintenance, or food preparation, during the total waking hour exposure period. Therefore, an ingestion fraction was not applied to this receptor.

1.3.3.2.4 ***Future Recreational Visitor***. The soil ingestion rate for the recreational visitor is based on incidental soil ingestion while conducting activities, such as golfing. Therefore, an ingestion fraction was not applied to this receptor.

1.3.3.3 *Surface Area (SA)*

The exposed surface area is defined as the skin surface area available for contact with soil and is measured in units of cm^2/day or event. Specific skin surface areas were developed for each receptor and are described below.

1.3.3.3.1 ***Current/Future SWMU-Specific Laborer***. The exposed skin surface area for the CTE and RME scenarios, is assumed to be 2,000 cm^2 which includes the hands, head, and neck (USEPA 1990b) and 3,420 cm^2 which includes the hands, head, neck, and forearms (AIHC 1994).

1.3.3.3.2 ***Future Construction Worker***. The exposed skin surface area for the CTE and RME scenarios, is assumed to be 3,420 cm^2 which includes the hands, head, neck, and forearms (AIHC

1994) and 5,000 cm^2 which is estimated assuming that 30 percent of the body is exposed (AIHC 1994).

1.3.3.3.3 ***Future On-Site Resident***. The exposed skin surface area of the adult for the CTE and RME scenarios, is assumed to be 2,000 cm^2 which includes the hands, head, and neck (USEPA 1990b) and 3,420 cm^2 which includes the hands, head, neck, and forearms (AIHC 1994). For the children, ages 0 to 18, the average skin surface area of males and females for the 50th and 95th percentile are 1,190 and 1,480 cm^2 which corresponds to the CTE and RME scenarios (AIHC 1994).

1.3.3.3.4 ***Future Recreational Visitor***. The exposed skin surface area for the recreational visitor or golfer is assumed to include the hands, head, neck, and forearms, 3,420 cm^2 (AIHC 1994).

1.3.3.4 *Adherence Factor (AF)*

Chemical uptake via dermal contact with contaminated soils occurs primarily as a result of direct soil contact. The degree to which soil adheres to skin is a key factor governing dermal uptake of chemicals from soil. The 50th and 95th percentile for the soil adherence is 0.25 and 1.7 mg/cm^2 (Finley et al. 1994).

1.3.3.5 *Absorption Factor (ABS)*

Absorption factors are used to reflect the desorption of the chemical from soil and the absorption of the chemical across the skin and into the blood stream. Unless specific values are available for the chemicals being evaluated, the USEPA has recommended for inorganics and organics are 0.001 and 0.01 (USEPA Region X 1990).

1.3.3.6 *Exposure Frequency (EF)*

The exposure frequency is defined as the time a receptor is potentially exposed to contaminants and is measured in unit of days or events per year. Specific exposure frequencies were developed for each receptor and are described below.

1.3.3.6.1 ***Current/Future SWMU-Specific Laborer***. Most laborers at TEAD-N currently work 4-day-weeks. For the RME, as a worst-case estimate, it is could be assumed that the laborer is working at one particular SWMU the entire working year or 192 days/yr. This exposure frequency is developed assuming that the laborer spends approximately 4 weeks on vacation, sick leave, and holidays away from the site.

For the CTE, it is assumed that the laborer is working on various assignments at various SWMUs throughout the year. Therefore, the time that the laborer will spending at any given SWMU is estimated to be 50 days/year.

1.3.3.6.2 ***Future Construction Worker***. For the CTE, it is assumed that the worker is working at the site for an exposure frequency of 60 days, equivalent to a 3-month construction project. For the RME, it is assumed that the worker is working at the site for an exposure frequency of 140 days or a 6-month construction project.

1.3.3.6.3 ***Future On-Site Resident***. The exposure frequency for the adult/child resident is estimated based on the exposure time, as discussed above, and vacation/holiday time spent away from home. Because the contact rate for soil is in units of skin area per day, the exposure time is pro-rated to 24-hour day equivalents.

It is assumed that the adult resident spends approximately 4 weeks away from home on vacation and long holiday weekends. The exposure frequency for the CTE scenario is estimated as follows:

$$EF_{adult\text{-}CTE} = \left(\frac{108\ hrs}{week}\right)\left(\frac{52\ weeks - 4\ weeks}{yr}\right)\left(\frac{day}{24\ hr}\right) \cong 216\ days/yr$$

$$EF_{child\text{-}CTE} = \left(\frac{138\ hrs}{week}\right)\left(\frac{52\ weeks - 4\ weeks}{yr}\right)\left(\frac{day}{24\ hr}\right) \cong 276\ days/yr$$

For the RME scenario, the exposure frequency for the adult/child resident is estimated the same way as the CTE, however the vacation/holiday time spent away from home is reduced to 2 weeks/yr. The exposure frequency for the RME scenario is estimated as follows:

$$EF_{adult\text{-}RME} = \left(\frac{131\ hrs}{week}\right)\left(\frac{52\ weeks - 2\ weeks}{yr}\right)\left(\frac{day}{24\ hr}\right) \cong 273\ days/yr$$

$$EF_{child\text{-}RME} = \left(\frac{138\ hrs}{week}\right)\left(\frac{52\ weeks - 2\ weeks}{yr}\right)\left(\frac{day}{24\ hr}\right) \cong 288\ days/yr$$

1.3.3.6.4 ***Future Recreational Visitor***. It is assumed the recreational visitor or golfer will play a game of golf every week or at least once every two weeks on a course at the site for the RME and CTE scenario, respectively.

1.3.3.7 *Exposure Duration (ED)*

Exposure duration is defined as the total length of time a receptor may be exposed to the soil. Specific exposure times were developed for each receptor and are described below.

1.3.3.7.1 ***Current/Future SWMU-Specific Laborer***. Military personnel are rotated on assignment an average of every 3 years (personal communication, 1994). Therefore, for the CTE scenario, it is assumed that the worker tenure for military staff will not exceed three years. Under the RME scenario, it is proposed that the worker is a civilian and the exposure duration is 25 years (AIHC 1994). According to data from the Bureau of Labor Statistics (1987), 25 years is the upper 95th percentile of the distribution for number of years spent at a specific job.

1.3.3.7.2 ***Future Construction Worker***. It is assumed that a single construction company could obtain a contract for construction at the same location or SWMU for an extended period of time. For the CTE scenario, it is assumed that a worker would spent a maximum of 1 year at the work site or SWMU. For a large construction project, it is conceivable that a construction worker could spent up to 3 years at the same location (RME scenario).

1.3.3.7.3 ***Future On-Site Resident***. For the CTE scenario, years spent at one residence for the adult/child is assumed to be 8 years (AIHC 1994). The upper-bound 95th percentile of 30 years is used for the RME scenario (USEPA 1989). For the child, the exposure duration is 18 years which is based on a child growing up in the same residence and then leaving for further education or work opportunities.

1.3.3.7.4 ***Future Recreational Visitor***. For the CTE scenario, years spent at one residence for the adult is assumed to be 8 years (AIHC 1994). The upper-bound 95th percentile of 30 years is used for the RME scenario (USEPA 1989).

1.3.3.8 *Body Weight (BW)*

Estimated average body weights of adults is 72 kg (AIHC 1994). This value is the average of the mean 50th percentile values for adults (men and women) across the age spectrum of 18 to 75 years listed in USEPA (1990). The estimated average body weight of children both male and female, ages 0 to 18, based on the 50th and 95th percentile is 32.5 kg and 47 kg, respectively for the CTE and RME scenario (USEPA 1990b). The estimated average body weight of juveniles both male and female, ages 6 to 12, based on the 50th and 95th percentile is 29 kg and 45 kg, for the CTE and RME, respectively (USEPA 1990b).

1.3.3.9 *Averaging Time (AT)*

The averaging time used is dependent on the toxicological endpoint of the chemical of concern. For chemicals that are thought to be potential carcinogens, intakes were averaged over the estimated lifetime of each receptor, which is 27,375 days (75 years x 365 days/year). For systemic toxicants, intakes were averaged over the estimated duration of exposure specific to each receptor exposure scenario (i.e., ED • 365 days).

The distinction between carcinogens and systemic toxicants is based on the theory that the mechanism of action for these two categories is different. It is assumed that a high dose of a carcinogen received over a short period is equivalent to a low dose spread over a lifetime. Systemic toxicants are assumed to have a threshold below which no toxic effect is observed. Therefore, intakes are averaged over the actual exposure duration.

1.3.4 Produce Ingestion Pathway

The intake of contaminants via fruits and vegetables was estimated based on the duration and frequency of exposure, the ingestion rate, and the concentration of chemicals in the material ingested. The model used for estimating ingestion exposure is shown below:

$$\text{Daily Intake (mg/kg-day)} = \frac{Cp * IR * FI * EF * ED}{BW * AT}$$

where

Cp	=	Chemical concentration in produce (mg/kg)
IR	=	Ingestion rate (mg/kg)
FI	=	Fraction ingested from source (unitless)
EF	=	Exposure frequency (days/yr)
ED	=	Exposure duration (years)
BW	=	Body weight (kg)
AT	=	Noncarcinogenic or carcinogenic averaging time (days)

The exposure parameters are described in the following sections and summarized in Table 7.

1.3.4.1 *Chemical Concentration in Produce (Cp)*

The concentration of contaminants present in crops is dependent on the amount of contaminants in the soil, length of the growing season, and type of crop grown. The model used for estimating potential chemical concentrations in homegrown vegetables is described in Section 1.2. Leafy and root vegetables were modelled separately.

Table 7. Exposure Parameters for the Ingestion of Homegrown Produce Pathway

Parameters	RME	RME Reference	CTE	CTE Reference
Future On-Site Resident - Adult (Child)				
Cp = Chemical Concentration in Produce (mg/kg)	chemical-specific	NA	chemical-specific	NA
IR = Ingestion Rate (g/day)	420 total (300 total)	McKone, 1994	300 total (220 total)	McKone, 1994
	47% Fruits/Tubers	NFCS, 1993	47% Fruits/Tubers	NFCS, 1993
	14% Leafy Vegs.	NFCS, 1993	14% Leafy Vegs.	NFCS, 1993
FI = Fraction Ingested from Source (unitless)	0.6	McKone, 1994	0.24	McKone, 1994
EF = Exposure Frequency (days/yr)	365	USEPA, 1989	365	USEPA, 1989
ED = Exposure Duration (years)	30 (18)	USEPA, 1989	8	AIHC, 1994
BW = Body Weight (kg)	72 (47)	AIHC, 1994	72 (32.5)	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	10,950 (6,570)	USEPA, 1989	2,920	AIHC, 1994
Carcinogenic	27,375	USEPA, 1990	27,375	USEPA, 1990

Uptake of contaminants by leafy vegetable may occur through both direct deposition of particulates onto the edible portion of the plant and uptake by the roots from the soil. Root vegetables are protected from direct deposition. It was assumed that the vegetables were eaten raw and unwashed. This assumption eliminates two significant removal processes from the concentration estimates.

1.3.4.2 *Ingestion Rate (IR)*

Under the CTE scenario, average produce consumption rates of fruits and vegetables for adults and children were used to calculate the overall daily intake (McKone 1994). To apportion this between leafy vegetables, tubers, and fruits, U.S. Department of Agriculture (USDA) data (1993) on produce consumption in the west was used. This yielded consumption rates of 300 g/day for adults and 220 g/day for children.

The RME adult and child consumption rates are based on the upper 95th percentile ($\mu + 2\sigma$) of the average consumption rate used for the CTE scenario. This yielded consumption rates of 420 g/day for adults and 300 g/day for children.

1.3.4.3 *Fraction Ingested from Source (FI)*

Homegrown foods are those foods produced on the land associated with a household and, for the most part, consumed within that household. The USEPA has compiled data for households on the fractions of consumed foods that are homegrown. In farm households, the annual fraction of consumed vegetables that is homegrown is 24 percent (McKone 1994). The RME fraction is based on the upper 95th percentile ($\mu + 2\sigma$) of the average fraction used for the CTE scenario or 60 percent (McKone 1994).

1.3.4.4 *Exposure Frequency (EF)*

The ingestion rates described above are based on annual averages, therefore, ingestion of homegrown beef was assumed to occur daily (365 days/yr) for both adults and children.

1.3.4.5 *Exposure Duration (ED)*

Exposure duration is defined as the total length of time a receptor may be exposed to the media. For the CTE scenario, years spent at one residence for the future on-site adult/child resident is assumed to be 8 years (AIHC 1994). The upper-bound 95th percentile of 30 years is used for the RME scenario (USEPA 1989). For the child, the exposure duration is 18 years which is based on a child growing up in the same residence and then leaving for further education or work opportunities.

1.3.4.6 *Body Weight (BW)*

Estimated average body weights of adults is 72 kg (AIHC 1994). This value is the average of the mean 50th percentile values for adults (men and women) across the age spectrum of 18 to 75 years listed in USEPA (1990). The estimated average body weight of children both male and female, ages 0 to 18, based on the 50th and 95th percentile is 32.5 kg and 47 kg, respectively for the CTE and RME scenario (USEPA 1990b).

1.3.4.7 *Averaging Time (AT)*

The averaging time used is dependent on the toxicological endpoint of the chemical of concern. For chemicals that are thought to be potential carcinogens, intakes were averaged over the estimated lifetime of each receptor, which is 27,375 days (75 years x 365 days/year). For systemic toxicants, intakes were averaged over the estimated duration of exposure specific to each receptor exposure scenario (i.e., ED • 365 days).

The distinction between carcinogens and systemic toxicants is based on the theory that the mechanism of action for these two categories is different. It is assumed that a high dose of a carcinogen received over a short period is equivalent to a low dose spread over a lifetime. Systemic toxicants are assumed to have a threshold below which no toxic effect is observed. Therefore, intakes are averaged over the actual exposure duration.

1.3.5 Beef Ingestion Pathway

The intake of contaminants via ingestion of beef was estimated based on the duration and frequency of exposure, the ingestion rate, and the concentration of chemicals in the material ingested. The model used for estimating ingestion exposure is shown below:

$$\text{Daily Intake (mg/kg-day)} = \frac{Cb * IR * FI * EF * ED}{BW * AT}$$

where

Cb	=	Chemical concentration in beef (mg/kg)
IR	=	Ingestion rate (mg/day)
FI	=	Fraction ingested from source (unitless)
EF	=	Exposure frequency (days/yr)
ED	=	Exposure duration (years)
BW	=	Body weight (kg)
AT	=	Noncarcinogenic or carcinogenic averaging time (days)

The exposure parameters are described in the following sections and summarized in Table 8.

Table 8. Exposure Parameters for the Ingestion of Beef Pathway

Parameters	RME	RME Reference	CTE	CTE Reference
Beef Consumer - Adult (Child)				
Cb = Chemical Concentration in Beef (mg/kg)	chemical-specific	NA	chemical-specific	NA
IR = Ingestion Rate (kg/day)	82,000	AIHC, 1994	40,000	AIHC, 1994
FI = Fraction Ingested from Source (unitless)	0.88	McKone, 1994	0.44	McKone, 1994
EF = Exposure Frequency (days/yr)	365	USEPA, 1989	365	USEPA, 1989
ED = Exposure Duration (years)	30 (18)	USEPA, 1989	8	AIHC, 1994
BW = Body Weight (kg)	72 (47)	AIHC, 1994	72 (32.5)	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	10,950 (6,570)	USEPA, 1989	2,920	AIHC, 1994
Carcinogenic	27,375	USEPA, 1990	27,375	USEPA, 1990

1.3.5.1 *Chemical Concentration in Beef (Cb)*

The concentration of chemicals in beef is the product of the concentration of chemicals in feed, the amount of feed ingested by cattle, and a chemical-specific biotransfer factor. It was conservatively assumed that all cattle feed is grown in the SWMU-specific area. A detailed description of the model used for estimating potential concentrations in beef is described in Section 1.2.

1.3.5.2 *Ingestion Rate (IR)*

Under the CTE scenario, a beef consumption rate of 40,000 mg/day was used to calculate the daily intake (AIHC 1994). The RME beef consumption rate is based on the upper 95th percentile ($\mu + 2\sigma$) of the average consumption rate used for the CTE scenario or 82,000 mg/day (AIHC 1994). For children, it was conservatively assumed to be the same as for the adult for both scenarios.

1.3.5.3 *Fraction Ingested from Source (FI)*

Homegrown foods are those foods produced on the land associated with a household and, for the most part, consumed within that household. The USEPA has compiled data for households on the fractions of consumed foods that are homegrown. In farm households that consume beef, the annual fraction of consumed beef that is homegrown is 44 percent (McKone 1994). The RME fraction is based on the upper 95th percentile ($\mu + 2\sigma$) of the average fraction used for the CTE scenario or 88 percent (McKone 1994).

1.3.5.4 *Exposure Frequency (EF)*

The ingestion rates described above are based on annual averages, therefore, ingestion of homegrown beef was assumed to occur daily (365 days/yr) for both adults and children.

1.3.5.5 *Exposure Duration (ED)*

Exposure duration is defined as the total length of time a receptor may be exposed to the media. For the CTE scenario, years spent at one residence for the future on-site adult/child resident is assumed to be used for the RME scenario (USEPA 1989). For the child, the exposure duration is 18 years which is based on a child growing up in the same residence and then leaving for further education or work opportunities.

1.3.5.6 *Body Weight (BW)*

Estimated average body weights of adults is 72 kg (AIHC 1994). This value is the average of the mean 50th percentile values for adults (men and women) across the age spectrum of 18 to 75 years listed in USEPA (1990). The estimated average body weight of children both male and female, ages 0 to 18, based on the 50th and 95th percentile is 32.5 kg and 47 kg, respectively for the CTE and RME scenario (USEPA 1990b).

1.3.5.7 *Averaging Time (AT)*

The averaging time used is dependent on the toxicological endpoint of the chemical of concern. For chemicals that are thought to be potential carcinogens, intakes were averaged over the estimated lifetime of each receptor, which is 27,375 days (75 years x 365 days/year). For systemic toxicants, intakes were averaged over the estimated duration of exposure specific to each receptor exposure scenario (i.e., ED • 365 days).

The distinction between carcinogens and systemic toxicants is based on the theory that the mechanism of action for these two categories is different. It is assumed that a high dose of a carcinogen received over a short period is equivalent to a low dose spread over a lifetime. Systemic toxicants are assumed to have a threshold below which no toxic effect. Therefore, intakes are averaged over the actual exposure duration.

1.3.6 Ingestion of Groundwater

Exposure to groundwater through ingestion was estimated based on the duration of exposure, the ingestion rate during exposure, and the concentration of chemicals in the media ingested. The model used for estimating ingestion exposure is shown below:

$$\textbf{Daily Intake (mg/kg)} = \frac{Cgw * IR * EF * ED}{BW * AT}$$

where

Cgw	=	Chemical concentration in groundwater (mg/L)
IR	=	Ingestion rate (L/day)
EF	=	Exposure frequency (days/yr)
ED	=	Exposure duration (years)
BW	=	Body weight (kg)
AT	=	Carcinogenic or noncarcinogenic averaging time (days)

The exposure parameters are described in the following sections and summarized in Table 9.

Table 9. Exposure Parameters for the Ingestion of Groundwater Pathway

Parameters	RME	RME Reference	CTE	CTE Reference
Future On-site Resident - Adult				
Cgw = Chemical Concentration in Groundwater (mg/L)	chemical-specific	NA	chemical-specific	NA
IR = Ingestion Rate (L/day)	2.0	USEPA, 1996	1.4	USEPA 1996
EF = Exposure Frequency (days/yr)	273	see text	216	see text
ED = Exposure Duration (years)	30	USEPA, 1989	8	AIHC, 1994
BW = Body Weight (kg)	72	AIHC, 1994	72	AIHC, 1994
AT = Averaging Time (days)				
Noncarcinogenic	10,950	USEPA, 1989	2,920	AIHC, 1994
Carcinogenic	27,375	USEPA, 1990	27,375	USEPA, 1990

1.3.6.1 *Chemical Concentration in Groundwater (Cgw)*

The chemical concentration for each constituent in groundwater was derived by evaluating the existing monitoring well data. The CTE and RME concentrations are based on the upper 95th percentile confidence limit on the arithmetic mean (USEPA 1989, 1991). For a more detailed description of the rationale used to develop these concentrations, the reader is referred to the exposure point concentrations sections for each SWMU.

1.3.6.2 *Ingestion Rate (IR)*

The ingestion rate is dependent upon age, sex, and activity level. The ingestion rate of groundwater for future on-site adult residents is 1.4 L/day and 2.0 L/day for the CTE and RME scenarios, respectively (USEPA 1996). These rates are based on the average and 80th-90th percentile of intake rates among the adult population (USEPA 1996)

1.3.6.3 *Exposure Frequency (EF)*

The exposure frequency is defined as the time a receptor is potentially exposed to chemicals in groundwater and is measured in unit of days or events per year. The exposure frequency for the future on-site adult resident is estimated based on the exposure time, as discussed above, and vacation/holiday time spent away from home. Because the ingestion rate for groundwater is in units of volume per day, the exposure time is pro-rated to 24-hour day equivalents.

It is assumed that the adult resident spends approximately 4 weeks away from home on vacation and long holiday weekends. The exposure frequency for the CTE scenario is estimated as

$$EF_{adult\text{-}CTE} = \left(\frac{108\ hrs}{week}\right)\left(\frac{52\ weeks\ -\ 4\ weeks}{yr}\right)\left(\frac{day}{24\ hr}\right) \cong 216\ days/yr$$

For the RME scenario, the exposure frequency for the adult resident is estimated the same way as the CTE, however the vacation/holiday time spent away from home is reduced to 2 weeks/yr. The exposure frequency for the RME scenario is estimated as follows:

$$EF_{adult\text{-}RME} = \left(\frac{131\ hrs}{week}\right)\left(\frac{52\ weeks\ -\ 2\ weeks}{yr}\right)\left(\frac{day}{24\ hr}\right) \cong 273\ days/yr$$

1.3.6.4 *Exposure Duration (ED)*

Exposure duration is defined as the total length of time a receptor may be exposed to the groundwater. For the CTE scenario, years spent at one residence for the adult is assumed to be 8

years (AIHC 1994). The upper-bound 95th percentile of 30 years is used for the RME scenario (USEPA 1989).

1.3.6.5 *Body Weight (BW)*

Estimated average body weights of adults is 72 kg (AIHC 1994). This value is the average of the mean 50th percentile values for adults (men and women) across the age spectrum of 18 to 75 years listed in USEPA (1990).

1.3.6.6 *Averaging Time (AT)*

The averaging time used is dependent on the toxicological endpoint of the chemical of concern. For chemicals that are thought to be potential carcinogens, intakes were averaged over the estimated lifetime of each receptor, which is 27,375 days (75 years x 365 days/year). For systemic toxicants, intakes were averaged over the estimated duration of exposure specific to each receptor exposure scenario (i.e., ED • 365 days).

The distinction between carcinogens and systemic toxicants is based on the theory that the mechanism of action for these two categories is different. It is assumed that a high dose of a carcinogen received over a short period is equivalent to a low dose spread over a lifetime. Systemic toxicants are assumed to have a threshold below which no toxic effect is observed. Therefore, intakes are averaged over the actual exposure duration.

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APPENDIX M

TOXICITY ASSESSMENT

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1.0 TOXICITY ASSESSMENT

The purpose of the toxicity assessment is to review the inherent toxicity of a chemical and to examine the potential for exposure to the chemical to be associated with adverse effects in humans. Adverse effects include both systemic and carcinogenic health effects in humans. Reactions to chemical agents by living organisms are generally dependent on the route of exposure, the duration and frequency of exposure, the concentration to which the organisms are exposed (dose), and the sensitivity of the organism exposed.

Sources of information for this toxicity review include the U.S. Environmental Protection Agency's (USEPA) Integrated Risk Information System (IRIS) (USEPA 1995), Health Effects Assessment Summary Tables (HEAST) (USEPA 1994a), USEPA criteria documents, and Agency for Toxic Substances and Disease Registry (ATSDR) toxicological profiles. The hierarchy of toxicological information sources used in this risk assessment is based on USEPA guidance (USEPA 1989).

Toxicity profiles are included for each chemical of potential concern (COPC) with USEPA numeric toxicity criteria based on information in the documents cited above. Major effects are presented in the included profiles as well as descriptions of important toxicokinetic findings, such as absorption into, distribution in, metabolism by, and excretion from the body. Uncertainties and important data gaps are reviewed, and important studies used in the derivation of critical toxicity criteria are summarized (USEPA 1995).

Criteria for carcinogens are provided as carcinogenic slope factors (CSFs) in units of risk per milligram of chemical exposure per kilogram body weight per day. These factors are based on the assumption that no threshold for carcinogenic effects exists and any dose is associated with some finite cancer risk.

Systemic effects may be associated with exposure periods between 2 weeks and 7 years (subchronic), 7 years and a lifetime (chronic), or from a single exposure event (acute) (USEPA 1989). For each specific reaction caused by a chemical, there is a correlation relationship referred to as the dose-response relationship (USEPA 1989). Generally, the dose required for expression of an acute effect is a larger dose than the dose required for the expression of a subchronic or chronic effect. Criteria for systemic toxicants, or for significant systemic effects caused by carcinogens, are provided as reference doses (RfDs) in units of milligrams of chemical exposure per kilogram body weight per day. RfDs may be interpreted as thresholds below which any effects are not expected to occur even in the most sensitive populations.

Quantitative chemical dose-response information, in the form of critical toxicity criteria is presented in Section 2.0. Uncertainties associated with toxicity criteria estimates are discussed in Section 3.0. Individual chemical profiles in support of toxicity criteria and a discussion of the uncertainty associated with the criteria are included as Attachment A.

2.0 TOXICITY CRITERIA

2.1 KNOWN OR SUSPECTED CARCINOGENS

2.1.1 Evidence of Carcinogenicity

The USEPA has developed a system for stratifying the weight of evidence supporting classification of a chemical as a carcinogen. This classification system characterizes the overall weight of evidence of carcinogenicity based on the availability of human, animal, and other supportive data (USEPA 1995, 1994a). Three major factors are considered in characterizing the overall weight of evidence of carcinogenicity: (1) the quality of evidence from human studies; (2) the quality of evidence from animal studies, which are combined into a characterization of the overall weight of evidence for human carcinogenicity; and (3) other supportive data which are assessed to determine whether the overall weight of evidence should be modified. While the USEPA classification system is scheduled to be modified, the system presently in use for the characterization of the overall weight of carcinogenicity for the chemicals of potential concern at TEAD-N has the following five categories:

- Group A-Human Carcinogen—Indicates that there is sufficient evidence from epidemiological studies to support a causal association between an agent and cancer.

- Group B-Probable Human Carcinogen—Indicates that there is at least limited evidence from epidemiological studies of carcinogenicity to humans (Group B1) or that, in the absence of adequate data on humans, there is sufficient evidence of carcinogenicity in animals (Group B2).

- Group C-Possible Human Carcinogen—Indicates that there is limited evidence of carcinogenicity in animals in the absence of adequate data on humans.

- Group D-Not Classified—Indicates that the evidence for carcinogenicity in animals is inadequate.

- Group E-Evidence of Noncarcinogenicity to Humans—Indicates that there is evidence for noncarcinogenicity in at least two adequate animal tests in different species or in both epidemiological and animal studies.

2.1.2 Cancer Slope Factors

The USEPA Cancer Review Validation Effort (CRAVE) Committee has used a variety of specialized models to estimate the upper-bound risk of carcinogenesis for over 50 compounds. Estimations of risk rely on data derived from the results of human epidemiological studies or chronic animal bioassays. The likelihood that a chemical is a human carcinogen is a function of the weight of evidence of animal and/or human studies relating to the following:

- Increase in the number of tissues affected by the chemical;
- Increases in the number of animal species, strains, sexes, and number of experiments and doses showing a carcinogenic response;
- Occurrence of clear dose-response relationships and high levels of statistical significance of the increased tumor incidence in treated compared to control groups;
- A dose-related shortening of time-to-tumor occurrence or time-to-death with tumor; and
- A dose-related increase in the proportions of tumors that are malignant.

Animal studies are usually conducted using relatively high doses in order to observe possible adverse effects. Because humans are expected to be exposed at lower doses, the data are adjusted by using a mathematical model. The data from animal studies that are fitted to the linearized multi-stage scaling factors are often applied to derive CSFs for humans. Dose-response data derived from human epidemiological studies are fitted to dose-time-response curves on an individual basis. These models provide rough, but plausible, estimates of the upper limits on cancer risk. Although the actual risk is unlikely to be higher than the estimated risk, it could be considerably lower.

The unit of exposure is expressed as milligrams of chemical per kilogram of body weight per day (mg/kg-day). The CSF, which represents the upper 95th percentile confidence limit on the dose-response curve, is in units of (mg/kg-day)$^{-1}$. The product of the CSF and estimated exposure is an unitless probability estimate of the incremental increase in the probability of an individual's risk of developing cancer over an "average" lifetime per unit of exposure. For example, if the product of the CSF and the average daily dose is 1×10^{-6}, the predicted upper-bound incremental lifetime cancer risk (ILCR) for the exposed individual is one in one million (1:1,000,000). This estimated risk would be in addition to any "background" risk of cancer not related to the chemical exposure. CSFs for all known or suspected carcinogenic chemicals of potential concern are listed in Table 1. The data used to develop each CSF are found in the corresponding USEPA health assessment literature for each chemical and are summarized in the toxicity profiles (Attachment A).

It is assumed that all of the chemicals that are carcinogenic through the oral route are potentially carcinogenic through the dermal route, although few data are available concerning the carcinogenic activity of chemicals that are systemically absorbed through the skin. In the absence of dermal slope factors for all of the carcinogenics, an estimated dermal slope factor was derived for each chemical in accordance with USEPA guidance by dividing its respective oral slope factor by an appropriate gastrointestinal absorption factor (USEPA 1989). These derived dermal toxicity values are presented in Table 2. This approach is intended to adjust the dermal slope factor to represent the potency of the absorbed dermal dose.

Table 1. Chemicals of Potential Concern: Toxicity Values for Potential Carcinogenic Effects

Chemical of Potential Concern	Slope Factor (SF)				Weight of Evidence Classification	Tumor Site	
	Oral $(mg/kg\text{-}day)^{-1}$	Ref	Inhalation $(mg/kg\text{-}day)^{-1}$	Ref		Oral	Inhalation
			Metals				
Aluminum	not available		not available				
Antimony	not available		not available				
Arsenic	1.5E+00	I	1.5E+01	I	A	Skin	Lungs
Barium	not available		not available				
Beryllium	4.3E+00	I	8.4E+00	I	B2	All sites	Lungs
Cadmium	not available		6.3E+00	I	B1		Lungs
Chromium (VI)	not available		4.2E+01	I	A		Lungs
Copper	not available		not available		D		
Iron	not available		not available				
Lead	not available		not available		B2		
Manganese	not available		not available		D		
Thallium	not available		not available		D		
Zinc	not available		not available		D		
			Explosives				
2,4,6-Trinitrotoluene	3E-02	I	not available		C	Urinary bladder	
1,3,5-Trinitrobenzene	not available		not available				
HMX	not available		not available		D		
RDX	1.1E-01	I	not available		C	Liver	

Table 1. Chemicals of Potential Concern: Toxicity Values for Potential Carcinogenic Effects (continued)

Chemical of Potential Concern	Slope Factor (SF) Oral (mg/kg-day)$^{-1}$	Ref	Inhalation (mg/kg-day)$^{-1}$	Ref	Weight of Evidence Classification	Tumor Site Oral	Tumor Site Inhalation
Semivolatile Compounds							
Anthracene	not available		not available		D		
Benzenehexachloride (delta-)[a]	1.8E+00	I	1.8E+00	I	D	Liver	Liver
Benzo(a)pyrene	7.3E+00	I	not available		B2	Forestomach	
Chlordane (alpha-)[b]	1.3E+00	I	1.3E+00	I	B2	Liver	Liver
Chlordane (gamma-)[b]	1.3E+00	I	1.3E+00	I	B2	Liver	Liver
Chloromethane	1.3E-02	H	6.3E-03	H	C	Kidney	Kidney
Diethyl Phthalate	not available		not available				
Endosulfan (alpha-)	not available		not available				
Endrin	not available		not available		D		
Heptachlor	4.5E+00	I	4.55E+00	I	B2	Liver	Liver
Heptachlor Epoxide	9.1E+00	I	9.1E+00	I	B2	Liver	Liver
PCB-1248[c]	7.7E+00	I	not available		B2	Liver	
PCB-1254[c]	7.7E+00	I	not available		B2	Liver	
Phenanthrene	not available		not available		D		
Pyrene	not available		not available		D		

Table 1. Chemicals of Potential Concern: Toxicity Values for Potential Carcinogenic Effects (continued)

Chemical of Potential Concern	Slope Factor (SF) Oral (mg/kg-day)$^{-1}$	Ref	Slope Factor (SF) Inhalation (mg/kg-day)$^{-1}$	Ref	Weight of Evidence Classification	Tumor Site Oral	Tumor Site Inhalation

[a]Toxicity value for technical-benzenehexachloride used in lieu of data for delta-benzenehexachloride.
[b]Toxicity value for chlordane used in lieu of data for specific isomers.
[c]Toxicity value for total polychlorinated biphenyls used in lieu of data for specific isomers.

Abbreviations: I - IRIS (USEPA 1995)
H - HEAST (USEPA 1994a)
A - Human Carcinogen
B1/B2 - Probable Human Carcinogen
C - Possible Human Carcinogen
D - Not Classified

Table 2. Derived Dermal Toxicity Values

Chemical	Oral Absorption Coefficient (unitless)	Dermal Absorption Coefficient (unitless)	Chronic Oral Reference Dose (mg/kg-day)	Subchronic Oral Reference Dose (mg/kg-day)	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$	Chronic Dermal Reference Dose (mg/kg-day)	Subchronic Dermal Reference Dose (mg/kg-day)	Dermal Slope Factor $(mg/kg\text{-}day)^{-1}$
				Metals				
Aluminum	0.2[4]	0.001[2]	1.0E+00	1.0E+00	NTV	2.0E-01	2.0E-01	NC
Antimony	0.2[4]	0.001[2]	4.0E-04	4.0E-04	NTV	8.0E-05	8.0E-05	NC
Arsenic	0.98[1]	0.001[2]	3.0E-04	3.0E-04	1.5E+00	2.9E-04	2.9E-04	1.5E+00
Barium	0.10[1]	0.001[2]	7.0E-02	7.0E-02	NTV	7.0E-03	7.0E-03	NC
Beryllium	0.001[1]	0.001[2]	5.0E-03	5.0E-03	4.3E+00	5.0E-06	5.0E-06	4.3E+03
Cadmium	0.06[1]	0.001[2]	5.0E-04 (water) 1.0E-03 (food)	5.0E-04 (water) 1.0E-03 (food)	NTV	3.0E-05 (water) 6.0E-05 (food)	3.0E-05 (water) 6.0E-05 (food)	NC
Chromium (VI)	0.05[1]	0.001[2]	5.0E-03	2.0E-02	NTV	2.5E-04	1.0E-03	NC
Copper[a]	NA	NA	NA	NA	NA	NC	NC	NC
Iron	NA	NA	NTV	NTV	NTV	NC	NC	NC
Lead	NA	NA	NTV	NTV	NTV	NC	NC	NC
Manganese	0.03[5]	0.001[2]	5.0E-03 (water) 1.4E-01 (food)	5.0E-03 (water) 1.4E-01 (food)	NTV	1.5E-04 4.2E-03	1.5E-04 4.2E-03	NC
Thallium	0.2[4]	0.001[2]	8.0E-05	8.0E-04	NTV	1.6E-05	1.6E-05	NC
Zinc	0.50[1]	0.001[2]	3.0E-01	3.0E-01	NTV	1.5E-01	1.5E-01	NC

Table 2. Derived Dermal Toxicity Values (continued)

Chemical	Oral Absorption Coefficient (unitless)	Dermal Absorption Coefficient (unitless)	Chronic Oral Reference Dose (mg/kg-day)	Subchronic Oral Reference Dose (mg/kg-day)	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$	Chronic Dermal Reference Dose (mg/kg-day)	Subchronic Dermal Reference Dose (mg/kg-day)	Dermal Slope Factor $(mg/kg\text{-}day)^{-1}$
Explosives								
2,4,6-Trinitrotoluene	0.50[3]	0.01[2]	5.0E-04	5.0E-04	3.0E-02	2.5E-04	2.5E-04	6.0E-02
1,3,5-Trinitrobenzene	0.50[3][b]	0.01[2]	5.0E-05	5.0E-04	NTV	2.5E-05	2.5E-04	NC
HMX	0.30[3]	0.01[2]	5.0E-02	5.0E-02	NTV	1.5E-02	1.5E-02	NC
RDX	1.00[3]	0.01[2]	3.0E-03	3.0E-03	1.1E-01	3.0E-03	3.0E-03	1.1E-01
Pesticides								
Anthracene	0.708[4]	0.01[2]	3.0E-01	3.0E+00	NTV	2.1E-01	2.1E+00	NC
Benzenehexachloride (delta-)	0.919[5]	0.01[2]	NTV	NTV	1.8E+00	NC	NC	2.0E+00
Benzo(a)pyrene	0.5[1]	0.01[2]	NTV	NTV	7.3E+00	NC	NC	1.5E+01
Chlordane (alpha-)	0.8[6][e]	0.01[2]	6.0E-05	6.0E-05	1.3E+00	4.8E-05	4.8E-05	1.6E+00
Chlordane (gamma-)	0.8[6][e]	0.01[2]	6.0E-05	6.0E-05	1.3E+00	4.8E-05	4.8E-05	1.6E+00
Chloromethane	0.8[4]	0.01[2]	4.0E-03	4.0E-03	1.3E-02	3.2E-03	3.2E-03	1.6E-02
Diethyl Phthalate	NA	0.01[2]	8.0E-01	8.0E+00	NTV	NA	NA	NC
Endosulfan (alpha-)	0.5[4]	0.01[2]	6.0E-03	6.0E-03	NTV	3.0E-03	3.0E-03	NC
Endrin	NA	0.01[2]	3.0E-04	3.0E-04	NTV	NC	NC	NC
Heptachlor	NA	0.01[2]	5.0E-04	5.0E-04	4.55E+00	NC	NC	NC
Heptachlor Epoxide	NA	0.01[2]	1.3E-05	1.3E-05	9.1E+00	NC	NC	NC
PCB-1248	0.95[1][d]	0.01[2]	2.0E-05	2.0E-05	7.7E+00	1.9E-05	1.9E-05	8.1E+00

Table 2. Derived Dermal Toxicity Values (continued)

Chemical	Oral Absorption Coefficient (unitless)	Dermal Absorption Coefficient (unitless)	Chronic Oral Reference Dose (mg/kg-day)	Subchronic Oral Reference Dose (mg/kg-day)	Oral Slope Factor $(mg/kg\text{-}day)^{-1}$	Chronic Dermal Reference Dose (mg/kg-day)	Subchronic Dermal Reference Dose (mg/kg-day)	Dermal Slope Factor $(mg/kg\text{-}day)^{-1}$
PCB-1254	0.95[(1)(d)]	0.01[(2)]	2.0E-05	2.0E-05	7.7E+00	1.9E-05	1.9E-05	8.1E+00
Phenanthrene	NA	NA	NTV	NTV	NTV	NC	NC	NC
Pyrene	0.5[(1)(e)]	0.01[(2)]	3.0E-02	3.0E-01	NTV	1.5E-02	1.5E-01	NC

[1]Owen, 1990
[2]USEPA, 1992a
[3]USEPA, 1992b
[4]USEPA, 1994b
[5]USEPA, 1984
[a]Dermal toxicity values for copper were not estimated because the oral reference dose is based on drinking water standards.
[b]Value for 2,4,6-trinitrotoluene used in lieu of specific data for 1,3,5-trinitrobenzene.
[c]Value for chlordane used in lieu of specific data for chlordane isomers.
[d]Value for total polychlorinated biphenyls used in lieu of specific data.
[e]Value for benzo(a)pyrene used in lieu of specific data.

Abbreviations:
NTV - no toxicity value
NC - not calculated
NA - not applicable; cancer and/or noncancer effects are direct effects on the gastrointestinal system, therefore it is inappropriate to convert the oral toxicity values to dermal toxicity values.

2.2 SYSTEMIC TOXICANTS

RfDs are toxicity values developed by EPA for chemicals exhibiting systemic effects after oral exposure. RfDs are usually derived from no-observable-adverse-effect levels (NOAELs) or the lowest-observed-adverse-effect levels (LOAELs) taken either from human studies (often involving workplace exposures) or from animal studies. These levels are adjusted downward using appropriate uncertainty or safety factors.

Uncertainty factors are applied to correct for the possibilities that humans are more sensitive than experimental animals. In addition, uncertainty factors may be applied to account for sensitive subpopulations of humans (e.g., children, pregnant women, individuals with hay fever or asthma). Depending upon the information available, other factors may also be applied.

The RfD is an estimate of a level of daily exposure to a chemical which could be without adverse effects even if the exposure occurs continuously over a specified period of time (USEPA 1989). An RfD is probably associated with an uncertainty spanning an order of magnitude or more (USEPA 1989). RfDs are presented in units of milligram of chemical per kilogram of body weight per day for comparison with estimates of intake into the body. Intakes that are less than the RfD are not likely to be of concern. Chronic intakes that are greater than the chronic RfD indicate a possibility for adverse effects, at least in sensitive populations. Whether such exposures actually lead to adverse effects will (depending on the chemical) be a function of a number of factors. The accuracy of uncertainty factors applied to the NOAEL, the appropriateness of animal models used in studies extrapolated to humans, and the potential for the chemical to cause effects in organisms or systems (e.g., reproductive and immune systems) that have not been adequately studied all contribute uncertainty to the RfD estimate. It is generally accepted that the health-protective assumptions made by the USEPA in deriving RfDs will, in most cases, mean there is little concern for potential noncarcinogenic health effects for exposures slightly in excess of the RfDs, with the probability of adverse effects increasing with increasing exposure levels. The RfDs for systemic effects of potential COPCs for chronic exposure are presented in Table 3. In addition, RfDs for subchronic exposure are presented in Table 4.

No RfDs have been developed by the USEPA for the dermal route. Therefore, dermal RfDs were derived for the chemicals of concern in accordance with USEPA guidelines (USEPA 1989). Chronic dermal RfDs were derived by multiplying the values used as the chronic oral RfDs by appropriate gastrointestinal absorption factors (see Table 2). The absorption factors that were used in deriving the dermal RfDs were the same as those used in deriving the dermal slope factors.

3.0 UNCERTAINTIES ASSOCIATED WITH TOXICITY ASSESSMENT

There are many uncertainties associated with the use of toxicological information in health risk assessments which are related to uncertainties intrinsic to toxicology. Chief among these

Table 3. Chemicals of Potential Concern: Toxicity Values for Potential Systemic Effects from Chronic Exposure

Chemical of Potential Concern	Chronic Reference Dose (RfD) (mg/kg-day)				RfD Confidence and Uncertainty				Critical Effects
					Oral		Inhalation		
	Oral	Ref	Inhalation	Ref	CL	UF	CL	UF	
Metals									
Aluminum	1E+00	(a)	1.4E-03	(a)	L	100	M	300	Neurotoxicity; Psychomotor impairment for an occupational exposure
Antimony	4E-04	I	not available		L	1,000			Longevity, blood glucose, and cholesterol
Arsenic	3E-04	I	not available		M	3			Hyperpigmentation, keratosis and possible vascular complications
Barium	7E-02	I	1.43E-04	H	M	3	NP	1,000	Increased blood pressure; Fetus toxicity
Beryllium	5E-03	I	not available		L	100			No adverse effects
Cadmium	5E-04 (water) 1E-03 (food)	I	not available		H	10			Significant proteinuria/Human studies involving chronic exposure
Chromium (VI)	5E-03	I	not available		L	500			No effects reported
Copper[b]	4E-02	H	not available		NA	NA			Gastrointestinal system irritation
Iron	not available		not available						
Lead	not available		not available						
Manganese	5E-03 (water) 1.4E-01 (food)	I	1.4E-05	I	NA	1	M	1,000	CNS effects; Impairment of neuro behavioral function
Thallium[c]	8E-05	I	not available		L	1			No adverse effects

Table 3. Chemicals of Potential Concern: Toxicity Values for Potential Systemic Effects from Chronic Exposure (continued)

Chemical of Potential Concern	Chronic Reference Dose (RfD) (mg/kg-day)			RfD Confidence and Uncertainty					Critical Effects
				Oral			Inhalation		
	Oral	Ref	Inhalation	Ref	CL	UF	CL	UF	
Zinc	3E-01	I	not available		M	3			47% decrease in erythrocyte superoxide dismutase concentration in adult females after 10 weeks of zinc exposure
Explosives									
2,4,6-Trinitrotoluene	**5E-04**	**I**	not available		M	1,000			Liver effects
1,3,5-Trinitrobenzene	5E-05	I	not available		L	10,000			Increased splenic weight
HMX	5E-02	I	not available		L	1,000			Hepatic lesions
RDX	3E-03	I	not available		H	100			Inflammation of the prostate
Semivolatile Compounds									
Anthracene	3E-01	I	not available		L	3,000			No observed effects
Benzenehexachloride (delta-)	not available		not available						
Benzo(a)pyrene	not available		not available						
Chlordane (alpha-)[d]	6E-05	I	not available		L	1,000			Regional liver hypertrophy in females
Chlordane (gamma-)[d]	6E-05	I	not available		L	1,000			Regional liver hypertrophy in females
Chloromethane	4E-03	(e)	not available		L	1,000			Neurobehavioral effects
Diethyl Phthalate	8E-01	I	not available		L	1,000			Decreased growth rate, food consumption and altered organ weights

Table 3. Chemicals of Potential Concern: Toxicity Values for Potential Systemic Effects from Chronic Exposure (continued)

Chemical of Potential Concern	Chronic Reference Dose (RfD) (mg/kg-day)				RfD Confidence and Uncertainty				Critical Effects
					Oral		Inhalation		
	Oral	Ref	Inhalation	Ref	CL	UF	CL	UF	
Endosulfan (alpha-)[f]	6E-03	I	not available		M	100			Reduced body weight gain in males and females, increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males
Endrin	3E-04	I	not available		M	100			Mild histological lesions in liver, occasional convulsions
Heptachlor	5E-04	I	not available		L	300			Liver weight increases in males
Heptachlor Epoxide	1.3E-05	I	not available		L	1,000			Increased liver-to-body weight ratio in both males and females
PCB-1248[g]	2E-05	I	not available		M	300			Ocular exudate, inflamed and prominant Miebomain glands, distorted growth of finger and toe nails, decreased antibody response to sheep erythrocytes
PCB-1254	2E-05	I	not available		M	300			Ocular exudate, inflamed and prominant Miebomain glands, distorted growth of finger and toe nails, decreased antibody response to sheep erythrocytes
Phenanthrene	not available		not available						

Table 3. Chemicals of Potential Concern: Toxicity Values for Potential Systemic Effects from Chronic Exposure (continued)

Chemical of Potential Concern	Chronic Reference Dose (RfD) (mg/kg-day)				RfD Confidence and Uncertainty				Critical Effects
					Oral		Inhalation		
	Oral	Ref	Inhalation	Ref	CL	UF	CL	UF	
Pyrene	3E-02	I	not available		L	3,000			Kidney effects

[a]Provisional value provided by EPA-ECAO Regional Support (94-001b/6-20-94).
[b]Chronic reference dose for copper estimated based on current drinking water standard of 1.3 mg/L.
[c]Value for thallium sulfate used in lieu of specific data for thallium.
[d]Value for chlordane used in lieu of specific data for chlordane isomers.
[e]Provisional value provided by EPA-ECAO Regional Support (94-009a/3-21-94).
[f]Value for endosulfan used in lieu of specific data for endosulfan isomers.
[g]Value for PCB-1254 used in lieu of specific data for PCB-1248.

Abbreviations:
I - IRIS (USEPA 1995)
H - HEAST (USEPA 1994a)
L - Low
M - Medium
H - High
CL - Confidence Limit
UF - Uncertainty Factor
NA - Not Applicable
NP - Not Provided

Table 4. Chemicals of Potential Concern: Toxicity Values for Potential Systemic Effects from Subchronic Exposure

Chemical of Potential Concern	Subchronic Reference Dose (RfD_{sc}) (mg/kg-day)				RfD_{sc} Confidence and Uncertainty				Critical Effects
					Oral		Inhalation		
	Oral	Ref	Inhalation	Ref	CL	UF	CL	UF	
Metals									
Aluminum	1E+00[a]	(b)	1.4E-03	(b)	L	100	M	300	Neurotoxicity; Psychomotor impairment for an occupational exposure
Antimony	4E-04	H	not available		NP	1,000			Increased mortality, altered blood chemistries
Arsenic	3E-04[a]	H	not available		M	3			Hyperpigmentation, keratosis, and possible vascular complications
Barium	7E-02[a]	H	1.43E-03	H	M	3	NP	100	Increased blood pressure; Fetus toxicity
Beryllium	5E-03[a]	H	not available		L	100			None observed
Cadmium	5E-04[a] (water) 1E-03[a] (food)	I	not available		H	10			Significant proteinuria/Human studies involving chronic exposure
Chromium (VI)	2E-02	H	not available		NP	100			None observed
Copper[c]	4E-02	H	not available		NA	NA			Gastrointestinal system irritation
Iron	not available		not available						
Lead	not available		not available						
Manganese	5E-03[a] (water) 1.4E-01[a] (food)	H	1.4E-05[c]		NA	1	M	1,000	CNS effects; Impairment of neuro behavioral function

Table 4. Chemicals of Potential Concern: Toxicity Values for Potential Systemic Effects from Subchronic Exposure (continued)

Chemical of Potential Concern	Subchronic Reference Dose (RfD_{sc}) (mg/kg-day)				RfD_{sc} Confidence and Uncertainty				Critical Effects
					Oral		Inhalation		
	Oral	Ref	Inhalation	Ref	CL	UF	CL	UF	
Thallium[d]	8E-04	H	not available		NP	300			No adverse effects
Zinc	3E-01[a]	H	not available		M	3			Decreased blood enzyme
Explosives									
2,4,6-Trinitrotoluene	5E-04[a]	I	not available		M	1,000			Liver effects
1,3,5-Trinitrobenzene	5E-04	H	not available		NP	1,000			Increased splenic weight
HMX	5E-02[a]	I	not available		L	1,000			Hepatic lesions
RDX	3E-03[a]	H	not available		H	100			Inflammation of the prostate
Semivolatile Compounds									
Anthracene	3E+00	H	not available		NP	300			None observed
Benzenehexachloride (delta-)	not available		not available						
Benzo(a)pyrene	not available		not available						
Chlordane (alpha-)[e]	6E-05[a]	H	not available		L	1,000			Regional liver hypertrophy in females
Chlordane (gamma-)[e]	6E-05[a]	H	not available		L	1,000			Regional liver hypertrophy in females
Chloromethane	4E-03	(f)	not available		L	1,000			Neurobehavioral effects
Diethyl Phthalate	8E+00	H	not available		NP	100			Decreased body and organ weight
Endosulfan (alpha-)[g]	6E-03[a]	H	not available		M	100			Reduced body weight gain in males and females, increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males

Table 4. Chemicals of Potential Concern: Toxicity Values for Potential Systemic Effects from Subchronic Exposure (continued)

Chemical of Potential Concern	Subchronic Reference Dose (RfD_{sc}) (mg/kg-day)				RfD_{sc} Confidence and Uncertainty				Critical Effects
					Oral		Inhalation		
	Oral	Ref	Inhalation	Ref	CL	UF	CL	UF	
Endrin	3E-04[a]	H	not available		M	100			Mild histological lesions in liver, occasional convulsions
Heptachlor	5E-04[a]	H	not available		L	300			Liver weight increases in males
Heptachlor Epoxide	1.3E-05[a]	H	not available		L	1,000			Increased liver-to-body weight ratio in both males and females
PCB-1248[h]	2E-05[a]	I	not available		M	300			Ocular exudate, inflamed and prominent Miebomain glands, distorted growth of finger and toe nails, decreased antibody response to sheep erythrocytes
PCB-1254	2E-05[a]	I	not available		M	300			Ocular exudate, inflamed and prominent Miebomain glands, distorted growth of finger and toe nails, decreased antibody response to sheep erythrocytes
Phenanthrene	not available		not available						

Table 4. Chemicals of Potential Concern: Toxicity Values for Potential Systemic Effects from Subchronic Exposure (continued)

Chemical of Potential Concern	Subchronic Reference Dose (RfD_{sc}) (mg/kg-day)				RfD_{sc} Confidence and Uncertainty				Critical Effects
					Oral		Inhalation		
	Oral	Ref	Inhalation	Ref	CL	UF	CL	UF	
Pyrene	3E-01	H	not available		NP	300			Kidney effects

[a]Chronic RfD adopted as the subchronic RfD.

[b]Provisional value provided by EPA-ECAP Regional support (94-001b/6-20-94).
[c]Chronic reference dose for copper estimated based on current drinking water standard of 1.3 mg/L.
[d]Value for thallium sulfate used in lieu of specific data for thallium.
[e]Toxicity value for chlordane used in lieu of specific data for chlordane isomers.
[f]Provisional value provided by EPA-ECAP Regional support (94-009a/3-21-94).
[g]Toxicity value endosulfan in lieu of data for specific isomers.
[h]Toxicity value for PCB-1254 used in lieu of data for PCB-1248.

Abbreviations:
I - IRIS (USEPA 1995)
H - HEAST (USEPA 1994a)
L - Low
M - Medium
H - High
CL - Confidence Limit
UF - Uncertainty Factor
NP - Not Provided

uncertainties are: (1) the use of dose-response information from high-dose studies to predict adverse health effects at low doses; (2) the applicability of experimental animal studies to predict accurate health effects in humans; (3) the use of dose-response information from short-term exposure studies to predict adverse health effects of long-term exposures; (4) the use of toxicity values derived from homogenous animal populations which are likely to contain sensitive individuals; (5) quality of the study (i.e., design and conduct of the study); and (6) the selection criteria for the appropriate study used in the development of toxicity values.

These and other uncertainties are limitations to the risk assessment process which cannot be resolved quantitatively given the current understanding of toxicology and human health and using current risk assessment methodology. These uncertainties are addressed in part by consistent application of conservative assumptions regarding the toxic effects of chemicals, such as uncertainty factors for RfDs and upper bound estimates for CSFs. Such procedures are intended to protect public health and are expected, in many cases, to overstate potential impacts on human health.

4.0 REFERENCES

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ATTACHMENT A

TOXICOLOGICAL PROFILE

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M.2 ANTHRACENE

Status of Data	*Last Revised*
Oral RfD Assessment	07/01/93
Inhalation RfC Assessment	Pending
Carcinogenicity Assessment	01/01/91

M.2.1 NONCARCINOGENIC ASSESSMENT

M.2.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses	UF	MF	RfD
No observed effects	NOEL: 1000 mg/kg/day	3000	1	3E-1 mg/kg/day

Subchronic Toxicity Study in Mice — LOAEL: none

U.S. EPA, 1989

Principal and Supporting Studies

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

Anthracene was administered to groups of 20 male and female CD-1 (ICR)BR mice by oral gavage at doses of 0, 250, 500, and 1000 mg/kg/day for at least 90 days. Mortality, clinical signs, body weights, food consumption, opthalmology findings, hematology and clinical chemistry results, organ weights, organ-to-body weight ratios, gross pathology, and histopathology findings were evaluated. No treatment-related effects were noted. The no-observed-effect level (NOEL) is the highest dose tested (1000 mg/kg/day).

Uncertainty and Modifying Factors

UF -- An uncertainty factor of 3000 was used: 10 to account for interspecies extrapolation, 10 for intraspecies variability and 30 for both the use of a subchronic study for chronic RfD derivation and for lack of reproductive/developmental data and adequate toxicity data in a second species.

MF -- None

Additional Comments

In a chronic bioassay (Schmahl, 1955), a group of 28 BD I and BD III rats received anthracene in the diet, starting when the rats were approximately 100 days old. The daily dosage was 5 to 15 mg/rat, and the experiment was terminated when a total dose of 4.5 g/rat was achieved, on the 550th experimental day. The rats were observed until they died, with some living more than 1000 days. No treatment-related effects on lifespan or gross and histological appearance of tissues were observed. Body weights were not mentioned, and

hematological parameters were not measured. No chronic LOAEL could be determined from this study.

Confidence in the Oral RfD
Study -- Low
Data Base -- Low
RfD -- Low

Confidence in the study is low. It was a well-designed experiment examining a variety of toxicological endpoints; however, failure to identify a LOAEL precludes a higher level of confidence. Confidence in the data base is low, because of the lack of adequate toxicity data in a second species and developmental/reproductive studies. Low confidence in the RfD follows.

M.2.1.2 Reference Concentration for Chronic Inhalation Exposure

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

M.2.1.3 Noncarcinogenic Assessment References

Schmahl, D. 1955. Testing of naphthalene and anthracene as carcinogenic agents in the rat. Krebsforsch. 60: 697-710. (Ger.)

U.S. EPA. 1987. Health and Environmental Effects Profile for Anthracene. 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. 1989. Subchronic toxicity in mice with anthracene. Final Report. Hazelton Laboratories America, Inc. Prepared for the Office of Solid Waste, Washington, DC.

M.2.2 CARCINOGENIC ASSESSMENT

Weight-of-Evidence Classification
Classification -- D, not classifiable as to human carcinogencity

Basis -- Based on no human data and inadequate data from animal bioassays.

Human Carcinogenicity Data
None.

Animal Carcinogenicity Data

Inadequate. A group of 28 BDI or BDIII rats (sex not specified) were fed a diet containing anthracene in oil, 6 days/week for 78 weeks, and observed until natural death, approximately 700 days (Schmahl, 1955). The total dose was 4.5 g anthracene/rat (approximately 28 mg/kg/day). No concurrent controls were used. No tumors were observed.

Groups of 60 female 3-to 6-month-old Osborne-Mendel rats were observed for 55-81 weeks after receiving a single lung implant of anthracene (0.5 mg/rat, approximately 2 mg/kg) dissolved in a 1:1 (v:v) mixture of beeswax and trioctanoin (0.1 mL) (Stanton et al., 1972). Controls received an implant of the vehicle. No tumors were observed.

Tests for complete carcinogenicity and initiating activity in mouse skin-painting assays have not shown positive results. No tumors were observed in an assay of initiating activity in which Crl:CD/1 (ICR)BR female albino mice were exposed to 1 mg anthracene in acetone, and then treated with 12-o-tetradecanoyl-phorbol-13 acetate as the promoting agent 3 times/week for 20 weeks (LaVoie et al., 1985).

A single dermal application of 10 um anthracene (purity not stated) in benzene was administered to 30 female CD-1 mice; this initial application was followed 7 days later by twice-weekly applications of 5 um 12-0-tetradecanoyl phorbol-13-acetate (TPA) for 35 weeks. Survival in the group was 93% after 35 weeks. By week 20 of the test, 2/28 mice had developed skin tumors; this increased to 4/28 by week 35. In the control group, in which 30 mice received only the TPA applications, a mouse developed a skin tumor at week 25 (Scribner, 1973).

Kennaway (1924) administered 40% anthracene (purity unknown) either in lanolin or as an ether-extract to two groups of 100 mice each (sex and strain not stated). In the lanolin-group, 44% of the mice survived 131 days and in the ether-extract group only 6% survived until day 160. In the lanolin-group 1/44 surviving mice had developed a papilloma by day 131; no mice had developed tumors in the ether-extract by day 160. No information pertaining to the use of a control group was given.

Druckrey and Schmahl (1955) administered a diet containing anthracene in oil 6 days/week to 28 BDI or BDIII rats (sex not stated) for 78 weeks. The total dose was 4.5 g anthracene/rat. No treatment-related tumors were found, and no control groups appear to have been utilized.

Supporting Data for Carcinogenicity

Tests for DNA damage and gene mutations in prokaryotes have generally shown negative results. Negative results were observed in tests for DNA damage in Escherichia coli at concentrations up to 250 ug/mL and Bacillus subtilis at 62 ug/mL (Rosenkrantz and Poirier, 1979; McCarroll et al., 1981; DeFlora et al., 1984). Negative results were obtained in tests for reverse mutation in six strains of Salmonella typhimurium, at concentrations up to 1000 ug/plate (McCann et al., 1975; Simmon, 1979a; LaVoie et al., 1979; Salamone et al., 1979; Ho et al., 1981; DeFlora et al., 1984; Bos et al., 1988). Tests for forward mutation at 40 ug/mL were negative (Kaden et al., 1979). Positive results for reverse mutation in

Salmonella typhimurium (TA97) at 10 ug/plate were reported (Sakai et al., 1985). Anthracene was tested in bacterial assays in 20 laboratories as part of an international collaborative study. One lab reported a positive in TA100 without activation, one lab reported a positive in TA98 and TA100 but only with S9 and all other labs reported negative results (Bridges et al., 1981).

Anthracene has consistently been negative in yeast test systems measuring mitotic recombination (Simmon, 1979b; de Serres and Hoffman, 1981), gene conversion, mutation and chromosome loss (de Serres and Hoffman, 1981).

Tests for DNA damage, mutation, chromosome effects and cell transformation in a variety of eukaryotic cell preparations have shown negative results. Anthracene showed negative results in tests for DNA damage (DNA synthesis) in primary rat hepatocytes (1 ug/mL), Chinese hamster ovary cells (1000 ug/mL), or HeLa cells (100 ug/mL) (Williams, 1977; Probst et al., 1981; Garrett and Lewtas, 1983; Martin et al., 1978; Martin and McDermid, 1981). It yielded negative results in tests for forward mutation in Chinese hamster V79 cells (125 ug/mL), mouse lymphoma L5178Y cells (18 ug/mL) and human lymphoblastoid cells (36 ug/mL) (Knapp et al., 1981; Amacher and Turner, 1980; Amacher et al., 1980; Barfknecht et al., 1981). Results obtained in tests for sister-chromatid exchange and chromosome breaks in Chinese hamster D6 cells and rat liver epithelial ARL-18 cells at 178 ug/mL were negative (Abe and Sasaki, 1977; Tong et al., 1981). Results reported in tests for cell transformation (morphological changes) at concentrations up to 30 ug/mL in mouse BALB/3T3 cells, guinea pig fetal cells, Syrian hamster embryo cells and mouse embryo C3H10T1/2 cells (DiPaolo et al., 1972; Evans and DiPaolo, 1975; Pienta et al., 1977; Lubet et al., 1983) were negative. In the international collaborative study negative results were reported with in vitro assays measuring unscheduled DNA synthesis, sister chromatid exchange, chromosome aberrations, and gene mutations (Brookes and Preston, 1981).

M.2.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

None.

M.2.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

None.

M.2.2.3 Carcinogenic Assessment References

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M.3 ANTIMONY

Status of Data	*Last Revised*
Oral RfD Assessment	02/01/91
Inhalation RfC Assessment	no data
Carcinogenicity Assessment	no data

M.3.1 NONCARCINOGENIC ASSESSMENT

M.3.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD
Longevity, blood glucose, and cholesterol	NOEL: none	1000	1	4E-4 mg/kg/day
Rat Chronic Oral Bioassay	LOAEL: 0.35 mg/kg bw/day			
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.

Principal and Supporting Studies

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

An experimental group of 50 male and 50 female rats was administered 5 ppm potassium antimony tartrate in water. Over the period of study, growth rates of treated animals were not affected, but male rats survived 106 and females 107 fewer days than did controls at median lifespans. Nonfasting blood glucose levels were decreased in treated males, and cholesterol levels were altered in both sexes. Since there was only one level of antimony administered, a NOEL was not established in this study. A decrease in mean heart weight for the males was noted. No increase in tumors was seen as a result of treatment. Although not precisely stated, the concentration of 5 ppm antimony was expressed as an exposure of 0.35 mg/kg/day by the authors.

Uncertainty and Modifying Factors

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

Additional Comments

In a similar study (Kanisawa and Schroeder, 1969), groups of CD-1 mice (54/sex) were given potassium antimony tartrate in drinking water at 0 or 5 mg/L (5 ppm) for 540 days (18 months). Lifespans were significantly reduced in both males and females, but the degree of antimony toxicity was less severe in mice than rats. Bradley and Fredrick (1941) and Browning (1969) reported disturbances in glucose and cholesterol metabolism in rats ingesting 5 mg/L antimony, but no signs of injury to the heart were observed in rats receiving doses up to 100 mg/kg/day. Substantially higher doses of antimony trioxide were tolerated by rats in studies by Sunagawa (1981) and Gross et al. (1955a,b), suggesting a NOAEL of 500 mg/kg, but these studies are of inadequate duration to assess adverse effects on toxicity.

Seventy people became acutely ill after drinking lemonade containing 0.013% antimony (Dunn, 1928 and Monier-Williams, 1934). The lemonade had been prepared and left overnight in buckets coated with an enamel containing 2.88% antimony trioxide. Fifty-six people were taken to the hospital with burning stomach pains, colic, nausea and vomiting. Most recovered within 3 hours, but in some cases recovery was not complete for several days. It is estimated that a person consuming 300 mL of lemonade would have received a dose of approximately 36 mg antimony, or approximately 0.5 mg/kg for a 70-kg adult.

According to U.S. EPA (1980), multimedia antimony exposures are essentially negligible by comparison to occupational exposures at which discrete clinical health effects have been observed. Myocardial effects are among the best-characterized human health effects associated with antimony exposure. Studies by Brieger et al. (1954) suggest an inhalation NOEL for myocardial damage to be approximately 0.5 mg/cu.m. This exposure is approximately equivalent to an oral reference dose of 0.003 mg/kg bw/day (i.e., 0.5 mg/cu.m x 10 cu.m/day x 0.5 / 1.0 x 5 days/7 days / 70 kg / 10). Parallel studies in rats and rabbits resulted in observation of EKG alterations following exposure to 3.1-5.6 mg/cu.m. There are, however, no adequate data on oral exposure to antimony which permit reasonable estimate of no effect levels regarding heart damage.

One study (Belyaeva, 1967) indicated that women workers exposed in an antimony plant experienced a greater incidence of spontaneous abortions than did a control group of nonexposed working women. A high rate of premature deliveries among women workers in antimony smelting and processing was also observed (Aiello, 1955).

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.

M.3.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.3.1.3 Noncarcinogenic Reference Dose References

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M.3.2 CARCINOGENIC ASSESSMENT

This substance/agent has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential.

M.4 ARSENIC

Status of Data	*Last Revised*
Oral RfD Assessment	02/01/93
Inhalation RfC Assessment	No data
Carcinogenicity Assessment	07/01/95

M.4.1 NONCARCINOGENIC ASSESSMENT

M.4.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD
Hyperpigmentation, mg/kg/day keratosis and possible vascular complications	NOAEL: 0.009 mg/L converted to 0.0008 mg/kg-day	3	1	3E-4
Human chronic oral exposure	LOAEL: 0.17 mg/L converted to 0.014 mg/kg-day			

Tseng, 1977;
Tseng et al., 1968

*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.

Principal and Supporting Studies

Tseng, W.P. 1977. Effects and dose-response relationships of skin cancer and blackfoot disease with arsenic. Environ. Health Perspect. 19: 109-119.

Tseng, W.P., H.M. Chu, S.W. How, J.M. Fong, C.S. Lin and S. Yeh. 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. J. Natl. Cancer Inst. 40: 453-463.

The data reported in Tseng (1977) show an increased incidence of blackfoot disease that increases with age and dose. Blackfoot disease is a significant adverse effect. The prevalences (males and females combined) at the low dose are 4.6 per 1000 for the 20-39 year group, 10.5

per 1000 for the 40-59 year group, and 20.3 per 1000 for the >60 year group. Moreover, the prevalence of blackfoot disease in each age group increases with increasing dose. However, a recent report indicates that it may not be strictly due to arsenic exposure (Lu, 1990). The data in Tseng et al. (1968) also show increased incidences of hyperpigmentation and keratosis with age. The overall prevalences of hyperpigmentation and keratosis in the exposed groups are 184 and 71 per 1000, respectively. The text states that the incidence increases with dose, but data for the individual doses are not shown. These data show that the skin lesions are the more sensitive endpoint. The low dose in the Tseng (1977) study is considered a LOAEL.

The control group described in Tseng et al. (1968; Table 3) shows no evidence of skin lesions and presumably blackfoot disease, although this latter point is not explicitly stated. This group is considered a NOAEL.

The arithmetic mean of the arsenic concentration in the wells used by the individuals in the NOAEL group is 9 ug/L (range: 1-17 ug/L) (Abernathy et al., 1989). The arithmetic mean of the arsenic concentration in the wells used by the individuals in the LOAEL group is 170 ug/L (Tseng, 1977; Figure 4). Using estimates provided by Abernathy et al. (1989), the NOAEL and LOAEL doses for both food and water are as follows: LOAEL - [170 ug/L x 4.5 L/day + 2 ug/day (contribution of food)] x (1/55 kg) = 14 ug/kg/day; NOAEL - [9 ug/L x 4.5 L/day + 2 ug/day (contribution of food)] x (1/55 kg) = 0.8 ug/kg/day.

Although the control group contained 2552 individuals, only 957 (approximately 38%) were older than 20, and only 431 (approximately 17%) were older than 40. The incidence of skin lesions increases sharply in individuals above 20; the incidence of blackfoot disease increases sharply in individuals above 40 (Tseng, 1968; Figures 5, 6 and 7). This study is less powerful than it appears at first glance. However, it is certainly the most powerful study available on arsenic exposure to people.

This study shows an increase in skin lesions, 22% (64/296) at the high dose vs. 2.2% (7/318) at the low dose. The average arsenic concentration in the wells at the high dose is 410 ug/L and at the low dose is 5 ug/L (Cebrian et al., 1983; Figure 2 and Table 1) or 7 ug/L (cited in the abstract). The average water consumption is 3.5 L/day for males and 2.5 L/day for females.

There were about an equal number of males and females in the study. For the dose estimates given below we therefore assume an average of 3 L/day. No data are given on the arsenic exposure from food or the body weight of the participants (we therefore assume 55 kg). The paper states that exposure times are directly related to chronological age in 75% of the cases. Approximately 35% of the participants in the study are more than 20 years old (Figure 1).

Exposure estimates (water only) are: high dose - 410 ug/L x 3 L/day x (1/55 kg) = 22 ug/kg/day; low dose - 5-7 ug/L x 3 L/day x (1/55 kg) = 0.3-0.4 ug/kg/day.

The high-dose group shows a clear increase in skin lesions and is therefore designated a LOAEL. There is some question whether the low dose is a NOAEL or a LOAEL since there is no way of knowing what the incidence of skin lesions would be in a group where the exposure to arsenic is zero. The 2.2% incidence of skin lesions in the low-dose group is higher than that reported in the Tseng et al. (1968) control group, but the dose is lower (0.4 vs. 0.8 ug/kg/day).

The Southwick et al. (1983) study shows a marginally increased incidence of a variety of skin lesions (palmar and plantar keratosis, diffuse palmar or plantar hyperkeratosis, diffuse pigmentation, and arterial insufficiency) in the individuals exposed to arsenic. The incidences are 2.9% (3/105) in the control group and 6.3% (9/144) in the exposed group. There is a slight, but not statistically significant increase in the percent of exposed individuals that have abnormal nerve conduction (8/67 vs. 13/83, or 12% vs. 16% (Southwick et al., 1983; Table 8). The investigators excluded all individuals older than 47 from the nerve conduction portion of the study. These are the individuals most likely to have the longest exposure to arsenic.

Although neither the increased incidence of skin lesions nor the increase in abnormal nerve conduction is statistically significant, these effects may be biologically significant because the same abnormalities occur at higher doses in other studies. The number of subjects in this study was insufficient to establish statistical significance.

Table 3 (Southwick et al., 1983) shows the annual arsenic exposure from drinking water. No data are given on arsenic exposure from food or the body weight (assume 70 kg). Exposure times are not clearly defined, but are >5 years, and dose groups are ranges of exposure.

Exposure estimates (water only) are: dosed group - 152.4 mg/year x 1 year/365 days x (1/70) kg = 6 ug/kg/day; control group - 24.2 mg/year x year/365 days x (1/70) kg = 0.9 ug/kg/day.

Again because there are no data for a group not exposed to arsenic, there is some question if the control group is a NOAEL or a LOAEL. The incidence of skin lesions in this group is about the same as in the low-dose group from the Cebrian et al. (1983) study; the incidence of abnormal nerve conduction in the control group is higher than that from the low-dose group in the Hindmarsh et al. (1977) study described below. The control dose is comparable to the dose to the control group in the Tseng et al. (1968) and Hindmarsh et al. (1977) studies. The dosed group may or may not be a LOAEL, since it is does not report statisically significant effects when compared to the control.

This study shows an increased incidence of abnormal clinical findings and abnormal electromyographic findings with increasing dose of arsenic (Hindmarsh et al., 1977; Tables III and VI). However, the sample size is extremely small. Percentages of abnormal clinical signs possibly attributed to As were 10, 16, and 40% at the low, mid and high doses, respectively. Abnormal EMG were 0, 17 and 53% in the same three groups.

The exact doses are not given in the Hindmarsh et al. (1977) paper; however, some well data are reported in Table V. The arithmetic mean of the arsenic concentration in the high-dose and mid-dose wells is 680 and 70 ug/L, respectively. Figure 1 (Hindmarsh et al., 1977) shows that the average arsenic concentration of the low-dose wells is about 25 ug/L. No data are given on arsenic exposure from food. We assume daily water consumption of 2 liters and body weight of 70 kg. Exposure times are not clearly stated.

Exposure estimates (water only) are: low - 25 ug/L x 2 L/day x (1/70) kg = 0.7 ug/kg/day; mid - 70 ug/L x 2 L/day x (1/70) kg = 2 ug/kg/day; high - 680 ug/L x 2 L/day x (1/70) kg = 19 ug/kg/day.

The low dose is a no-effect level for abnormal EMG findings. However, because there is no information on the background incidence of abnormal clinical findings in a population with zero exposure to arsenic, there is no way of knowing if the low dose is a no-effect level or another marginal effect level for abnormal clinical findings. The low dose is comparable to the dose received by the control group in the Tseng (1977) and Southwick et al. (1983) studies.

The responses at the mid dose do not show a statistically significant increase but are part of a statistically significant trend and are biologically significant. This dose is an equivocal NOAEL/LOAEL. The high dose is a clear LOAEL for both responses.

As discussed previously there is no way of knowing whether the low doses in the Cebrian et al. (1983), Southwick et al. (1983) and Hindmarsh et al. (1977) studies are NOAELs for skin lesions and/or abnormal nerve conduction. However, because the next higher dose in the Southwick and Hindmarsh studies only shows marginal effects at doses 3-7 times higher, the Agency feels comfortable in assigning the low doses in these studies as NOAELs.

The Tseng (1977) and Tseng et al. (1968) studies are therefore considered superior for the purposes of developing an RfD and show a NOAEL for a sensitive endpoint. Even discounting the people <20 years of age, the control group consisted of 957 people that had a lengthy exposure to arsenic with no evidence of skin lesions.

The following is a summary of the defined doses in mg/kg-day from the principal and supporting studies:

1) Tseng (1977): NOAEL = 8E-4; LOAEL = 1.4E-2
2) Cebrian et al. (1983): NOAEL = 4E-4; LOAEL = 2.2E-2
3) Southwick et al. (1983): NOAEL = 9E-4; LOAEL = none (equivocal effects at 6E-3)
4) Hindmarsh et al., 1977: NOAEL = 7E-4; LOAEL = 1.9E-2 (equivocal effects at 2E-3)

Uncertainty and Modifying Factors

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

Additional Studies/Comments

Ferm and Carpenter (1968) produced malformations in 15-day hamster fetuses via intravenous injections of sodium arsenate into pregnant dams on day 8 of gestation at dose levels of 15, 17.5, or 20 mg/kg bw. Exencephaly, encephaloceles, skeletal defects and genitourinary systems defects were produced. These and other terata were produced in mice and rats all at levels around 20 mg/kg bw. Minimal effects or no effects on fetal development have been observed in studies on chronic oral exposure of pregnant rats or mice to relatively low levels of arsenic via drinking water (Schroeder and Mitchner, 1971). Nadeenko et al. (1978) reported that intubation of rats with arsenic solution at a dose level of 25 ug/kg/day for a period of 7 months, including pregnancy, produced no significant embryotoxic effects and only infrequent slight expansion of ventricles of the cerebrum, renal pelves and urinary bladder. Hood et al. (1977) reported that very high single oral doses of arsenate solutions (120 mg/kg) to pregnant mice were necessary to cause prenatal fetal toxicity, while multiple doses of 60 mg/kg on 3 days had little effect.

Extensive human pharmacokinetic, metabolic, enzymic and long-term information is known about arsenic and its metabolism. Valentine et al. (1987) established that human blood arsenic levels did not increase until daily water ingestion of arsenic exceeded approximately 250 ug/day (approximately 120 ug of arsenic/L. Methylated species of arsenic are successively 1 order of magnitude less toxic and less teratogenic (Marcus and Rispin, 1988). Some evidence suggests that inorganic arsenic is an essential nutrient in goats, chicks, minipigs and rats (NRC, 1989). No comparable data are available for humans.

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.

M.4.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

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M.4.2 CARCINOGENICTY ASSESSMENT

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.

Human Carcinogenity 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 α-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.

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).

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 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).

M.4.2 CARCINOGENIC ASSESSMENT

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

Dose-Response Data
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.

Additional Comments
None.

Discussion of Confidence
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.

M.4.2.1 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Summary of Risk Estimate
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)

Dose Response Data for Carcinogenicity
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

Additional Comments
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.

Discussion of Confidence

Overall a large study population was observed. Exposure assessments included air measurements for the Anaconda smelter and both air measurements and urinary arsenic for the ASARCO smelter. Observed lung cancer incidence was significantly increased over expected values. The range of the estimates derived from data from two different exposure areas was within a factor of 6.

M.4.2.3 Carcinogenic Assessment References

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M.5 BARIUM

Status of Data	*Last Revised*
Oral RfD Assessment	08/01/90
Inhalation RfC Assessment	12/01/91
Carcinogenicity Assessment	no data

M.5.1 NONCARCINOGENIC ASSESSMENT

M.5.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD
Increased blood pressure	NOAEL: 10 mg/L (0.21 mg/kg/day)	3	1	7E-2 mg/kg/day

Subchronic to Chronic LOAEL: None
Human Drinking Water Studies

Wones et al., 1990;
Brenniman and Levy, 1984

*Conversion Factors: 10 mg/L x 1.5 L/day/70 kg = 0.21 mg/kg/day

Principal and Supporting Studies

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

Brenniman, G.R. and P.S. Levy. 1984. High barium levels in public drinking water and its association with elevated blood pressure. In: Advances in Modern Toxicology IX, E.J. Calabrese, Ed. Princeton Scientific Publications, Princeton NJ. p. 231-249.

No single study considered alone is appropriate to calculate a lifetime RfD for barium. The RfD must be based rather on a weight of evidence approach which takes into account recent findings of the Wones et al. (1990) and Brenniman and Levy (1984) epidemiologic studies as well as the various rodent studies that have been conducted (Perry et al., 1983; McCauley et al., 1985; Schroeder and Mitchener, 1975a,b; Tardiff et al., 1980). Because of the number of studies involved, the complete reference citations are given in the Section VI.

Wones et al. (1990) administered barium (as barium chloride) in the drinking water of 11 healthy male volunteers. Subjects ranged in age from 27 to 61 years and had no previous history of diabetes, hypertension, or cardiovascular disease. Diets were strictly controlled throughout the 10-week study. Subjects were given 1.5 L/day of distilled and charcoal-filtered water containing 0 mg/L barium for weeks 0 to 2; 5 mg/L for weeks 3 to 6, and 10 mg/L for weeks 7 to 10. Blood and urine samples, as well as morning and evening blood pressures,

were taken. Electrocardiograms and 24-hour continuous electrocardiographic monitoring were also performed.

There were no changes in systolic or diastolic blood pressures, or serum chemistry, especially total cholesterol, HDL, LDL, triglycerides, potassium or glucose levels. There was an increase in serum calcium levels that was attributed to a decrease in serum albumin levels. This increase, although statistically significant, was considered borderline and not clinically significant. There were also no changes in cardiac cycle as noted by electrocardiograms and no significant arrhythmias. A NOAEL of 10 mg/L was identified in this study which corresponds to 0.21 mg/kg/day, based on an actual consumption rate of 1.5 L/day and a 70-kg body weight.

Brenniman and Levy (1984) conducted a retrospective epidemiology study which compared human mortality and morbidity rates in populations ingesting elevated barium levels (2 to 10 mg/L) in their drinking water to populations ingesting very little or no barium (less than or equal to 0.2 mg/L). Mortality rates for cardiovascular diseases were determined for the years 1971-1975 and were age-adjusted. For the morbidity study, 1175 adult males and 1203 adult females were selected from communities in which the average drinking water concentration was 7.3 mg/L. Differences in mortality rates from all cardiovascular diseases were significantly higher ($p < 0.05$) in the communities with elevated barium. However, these differences were largely in the 65 and over age group and did not account for confounding variables such as population mobility, or use of water softeners or medication.

Differences in blood pressure, prevalance of hypertension, stroke, and heart and renal disease were also measured between the individuals in the two communities. Data were analyzed using signed ranked test for age-specific rates, the weighted Z test for prevalence rates, and analysis of variance for blood pressures. No significant differences were found in mean systolic and diastolic pressures between the two communities. No significant differences were found when the total populations were broken down by duration (10 years or more), medication, or use of water softeners. Also, the prevalence rates for hypertension, stroke, and heart and kidney disease were not significantly different between the communities.

A concentration of 7.3 mg/L corresponds to a dose of 0.20 mg/kg/day (assuming a 70-kg adult drinks 2 L/day).

Uncertainty and Modifying Factors

UF -- According to U.S. EPA guidelines, an uncertainty factor of 10 is applied when a NOAEL from a subchronic human study is employed. However, data are available from chronic human studies which support this NOAEL, as well as several oral chronic animal studies. Therefore, this UF is not considered necessary. In addition, another factor of 10 is used with a human study to protect sensitive individuals. However, the data base supports the finding that the critical effect is hypertension which results from long exposure durations, and that the population most at risk is the adult male. Furthermore, the chosen study is a careful observation of this critical effect in adult males. Because of both the critical study's unique

focus and the supporting studies, a 3-fold UF, instead of a 10-fold UF, was chosen as most appropriate to protect for sensitive individuals within that population.

MF -- None

Additional Comments

Occupational studies of workers exposed to barium dust have shown that workers develop "baritosis." Affected workers showed no symptoms, no abnormal physical signs, no loss of vital capacity or interference with function, although they had a significantly higher incidence of hypertension.

McCauley et al. (1985) studied the histologic and cardiovascular effects of drinking water containing 0, 10, 100, or 250 mg/L barium for 36 weeks; 0, 1, 10, 100, or 1000 mg/L barium for 16 weeks, or 0, 10, 100, or 250 mg/L (0, 1.4, 14, 35, or 140 mg/kg Ba) barium for 68 weeks on male Sprague-Dawley rats (6/group). Females were exposed to 0 or 250 mg/L for 46 weeks. No significant histologic, carcinogenic, or cardiovascular (including hypertension) effects were observed. No changes were reported in body weight, or food and water consumption in any of the treated animals. Animals treated at the highest dose (1000 mg/L) did exhibit ultrastructural changes in the kidney glomeruli and the presence of myelin figures. No other effects were reported at any dose level for males or females.

Perry et al. (1983) exposed weanling rats to barium at 1, 10, or 100 ppm in drinking water for up to 16 months (average daily barium doses of 0.051, 0.51, and 5.1 mg/kg, respectively). There were no signs of toxicity at any barium dose level. Systolic blood pressure measurements revealed no increase in animals exposed to 1 ppm for 16 months, an increase of 4 mm Hg ($p < 0.01$) in animals exposed to 10 ppm barium for 16 months, and an increase of 16 mm Hg ($p < 0.001$) in animals exposed to 100 ppm barium for 16 months. The animals in this study were maintained in a special contaminant-free environment and fed a diet designed to reduce exposure to trace metals. It is possible that the restricted intake of certain beneficial metals (e.g., calcium and potassium) may have predisposed the test animals to the hypertensive effects of barium (U.S. EPA, 1985).

Schroeder and Mitchener (1975a,b) exposed rats and mice to 5 mg/L barium in drinking water for a lifetime (approximately 0.25 mg/kg/day for rats and 0.825 mg/kg/day for mice). No adverse effects were observed; however, blood pressure was not measured.

Tardiff et al. (1980) exposed rats to barium at 0, 10, 50, or 250 ppm in drinking water for 4, 8, and 13 weeks. The barium concentrations were approximately 0, 2.75, 13.7, and 66.25 mg/kg/day at the beginning of the study and 0, 1.7, 6.6, and 31.5 mg/kg/day at the end of the study. Although the barium body burden increased with increasing barium dosage, no conclusive signs of barium toxicity were observed in these animals. Blood pressure was not measured.

Confidence in the Oral RfD
Study -- Medium
Data Base -- Medium
RfD -- Medium

As previously stated, EPA does not believe that any single study, considered alone, is adequate to calculate an RfD for barium. However, EPA believes that medium confidence can be placed in the total data base used to determine the RfD.

M.5.1.2 Reference Concentration for Chronic Inhalation Exposure

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

M.5.1.3 Noncarcinogenic Reference Dose References

Brenniman, G.R. and P.S. Levy. 1984. Epidemiological study of barium in Illinois drinking water supplies. In: Advances in Modern Environmental Toxicology IX, E.J. Calabrese, R.W. Tuthill and L. Condie, Ed. Princeton Scientific Publications, Princeton NJ. p. 231-240.

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Schroeder, H.A. and M. Mitchener. 1975a. Life-term effects of mercury, methyl mercury and nine other trace metals on mice. J. Nutr. 105: 452-458.

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

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M.5.2 CARCINOGENIC ASSESSMENT

This substance/agent has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential.

M.6 HEXACHLOROCYCLOHEXANE, TECHNICAL

Status of Data	*Last Revised*
Oral RfD Assessment	No data
Inhalation RfC Assessment	No data
Carcinogenicity Assessment	07/01/93

M.6.1 NONCARCINOGENIC ASSESSMENT

M.6.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Not available at this time.

M.6.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.6.2 CARCINOGENIC ASSESSMENT

Weight-of-Evidence Classification
Classification -- B2; probable human carcinogen

Basis -- Assays in four strains of mice have yielded positive carcinogenicity results for t-HCH administered in the diet.

Human Carcinogenicity Data
Inadequate. One case report of a Japanese sanitation employee with acute leukemia was associated with occupational exposure to HCH and DDT (Hoshizaki et al., 1969).

Animal Carcinogencity Data
t-HCH has been reported to increase the incidences of liver neoplasms in four strains of mice (Hanada et al., 1973; Goto et al., 1972; Kashyap et al., 1979; Nigam et al., 1984; Bhatt et al., 1981; Munir et al., 1983; Nagasaki et al., 1972a,b; Munir and Bhide, 1984).

Munir et al. (1983) gave dietary t-HCH at 0, 125, 250 or 500 ppm to male Swiss mice from age 8-10 weeks. Animals were killed at 8-11, 12-14, 15-17 or 18-22 months of age. Both treatment dose- and duration-related increases in incidence of benign hepatic nodules and hepatocellular carcinomas were observed. Hanada et al. (1973) fed groups of 10-11 each male and female dd mice diets containing 0, 100, 300 or 600 ppm t-HCH for a period of 32 weeks. Mice were maintained on basal diet an additional 6 weeks. At the end of this time incidence of liver nodules and hepatomas was increased in both males and females of the two upper dose groups. Nagasaki also administered dd mice dietary t-HCH. Males only received basal diet

(14 mice) or 6.6 ppm (20), 66.0 ppm (20) or 660.0 ppm (20) t-HCH. Treatment was for 24 weeks, at which time the animals were killed. Cellular hyperplasia of the liver was treatment-related. Nodules and hepatomas were seen in 100% of the highest dose animals, but not in the two lower dose groups. Goto et al. (1972) observed increases in liver weight of ICR-JCL mice fed 600 ppm t-HCH in the diet. All t-HCH-treated mice developed hepatomas. Dietary t-HCH at levels up to 500 ppm has not been shown to produce tumors in Wistar rats or Syrian golden hamsters after 30 months of treatment (Munir et al., 1983).

Supporting Data for Carcinogenicity
No data on genetic toxicology of t-HCH are available. Alpha-HCH, which comprises approximately 65% of t-HCH, has been observed to produce similar results to t-HCH in carcinogen bioassays.

M.6.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Summary of Risk Estimates
Oral Slope Factor -- 1.8E+0 per (mg/kg)/day
Drinking Water Unit Risk -- 5.1E-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)	2E+0 ug/L
E-5 (1 in 100,000)	2E-1 ug/L
E-6 (1 in 1,000,000)	2E-2 ug/L

Dose-Response Data
Tumor Type -- liver nodules and hepatocellular carcinomas
Test Animals -- mouse/Swiss, male
Route -- diet
Reference -- Munir et al., 1983

Additional Comments
Animal doses were converted from ppm to mg/kg/day by multiplying by a food factor of 0.13. Control data are from a similar experiment in male Swiss mice reported in the same paper. Unlike the treated animals, which were killed at 15-17 months, the controls were killed at 15-20 months. The data set chosen was for those mice treated over the greatest proportion of their lifespan. The slope factor calculation included an adjustment for the short duration of the experiment. The human equivalent dose was calculated by multiplying the transformed dose by (0.03/70)**1/3 for body weight adjustment and (15/24)**3 to adjust the length of the experiment to the lifespan of the animal.

The unit risk should not be used if the water concentration exceeds 200 ug/L, since above this concentration the unit risk may not be appropriate.

Discussion of Confidence
The number of animals treated was relatively small, and the dose range was limited. Slope factors using data of Nagasaki et al. (1972a,b) and data from Munir et al. (1983) for mice killed at 12-14 months were 7.4 and 1.2 per (mg/kg)/day, respectively. These values are generally supportive of the risk estimate.

M.6.2.1 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Summary of Risk Estimates
Inhalation Unit Risk -- 5.1E-4 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-1 ug/cu.m
E-5 (1 in 100,000)	2E-2 ug/cu.m
E-6 (1 in 1,000,000)	2E-3 ug/cu.m

Dose-Response Data for Carcinogenicity
The inhalation risk estimates were calculated from the oral exposure data.

Additional Comments
The unit risk should not be used if the air concentration exceeds 20 ug/cu.m, since above this concentration the unit risk may not be appropriate.

Discussion of Confidence
See oral discussion.

M.6.2.3 Carcinogenic Assessment References

Bhatt, D.K., S.K. Nigam, B.C. Lakkad, et al. 1981. Sequential histochemical changes in the distribution of alkaline phosphatase in hexachlorocyclohexane (HCCH) induced liver tumours in pure inbred Swiss mice. Cell Mol. Biol. 27(2-3): 231-238.

Goto, M., M. Hattori, T. Miyagawa and M. Enomoto. 1972. Ecological contribution on chemistry. II. Formation of hepatoma in mice after ingestion of HCH isomers in high doses. Chemosphere. 1(6): 279-282.

Hanada, M., C. Yutani and T. Miyaji. 1973. Induction of hepatoma in mice by benzene hexachloride. Gann. 64: 511-513.

Hoshizaki, H., Y. Niki, H. Tajima, Y. Terada and A. Kasahara. 1969. A case of leukemia following exposure to insecticide. Acta. Haematol. Japan. 32(4): 672-677.

Kashyap, S.K., S.K. Nigam, R.C. Gupta, A.B. Karnik and S.K. Chatterjee. 1979.Carcinogenicity of hexachlorocyclohexane (BHC) in purebred Swiss mice. J.Environ. Sci. Health. B14(3): 305-318.

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Nagasaki, H., S. Tomii, T. Mega, M. Marugami and N. Ito. 1972a. Hepatocarcinogenic effect of alpha, beta, gamma and delta isomers of benzene hexachloride in mice. Gann. 63(3): 393.

Nagasaki, H., S. Tomii, T. Mega, M. Marugami and N. Ito. 1972b. Carcinogenicity of benzene hexachloride (BHC). In: Topics in Chemical Carcinogenesis, W. Nakahara, S. Takayama, T. Sugimura and S. Odashima, Ed. Univ. of Tokyo Press, Tokyo. p. 343-353.

Nigam, S.K., B.C. Lakkad, A.B. Karnik and K.M. Thakore. 1984. Ultrastructural changes in liver of mice exposed to hexachlorocyclohexane. Ind. J. Exp. Biol. 22(4): 199-204.

U.S. EPA. 1986. Health and Environmental Effects Profile for Hexachlorocyclohexanes. 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.

M.7 BENZO(A)PYRENE

Status of Data	*Last Revised*
Oral RfD Assessment	No data
Inhalation RfC Assessment	No data
Carcinogenicity Assessment	11/01/94

M.7.1 NONCARCINOGENIC ASSESSMENT

M.7.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Not available at this time.

M.7.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.7.2 CARCINOGENIC ASSESSMENT

Weight-og-Evidence Classification
Classification -- B2; probable human carcinogen

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

Human Carcinogenicity Data
Inadequate. Lung cancer has been shown to be induced in humans by various mixtures of polycyclic aromatic hydrocarbons known to contain BAP including cigarette smoke, roofing tar and coke oven emissions. It is not possible, however, to conclude from this information that BAP is the responsible agent.

Animal Carcinogenicity Data
Sufficient. The animal data consist of dietary, gavage, inhalation, intratracheal instillation, dermal and subcutaneous studies in numerous strains of at least four species of rodents and several primates. Repeated BAP administration has been associated with increased incidences of total tumors and of tumors at the site of exposure. Distant site tumors have also been observed after BAP administration by various routes. BAP is frequently used as a positive control in carcinogenicity bioassays.

BAP administered in the diet or by gavage to mice, rats and hamsters has produced increased incidences of stomach tumors. Neal and Rigdon (1967) fed BAP (purity not reported) at concentrations of 0, 1, 10, 20, 30, 40, 45, 50, 100 and 250 ppm in the diets of male and female CFW-Swiss mice. The age of the mice ranged from 17-180 days old and the treatment time from 1-197 days; the size of the treated groups ranged from 9 to 73. There were 289 mice (number of mice/sex not stated) in the control group. No forestomach tumors were reported in the 0-, 1- and 10-ppm dose groups. The incidence of forestomach tumors in the 20-, 30-, 40-, 45-, 50-, 100- and 250-ppm dose groups were 1/23, 0/37, 1/40, 4/40, 23/34, 19/23 and 66/73, respectively. The authors felt that the increasing tumor incidences were related to both the concentration and the number of doses administered. Historical control forestomach tumor data are not available for CFW-Swiss strain mice. In historical control data from a related mouse strain, SWR/J Swill, the forestomach tumor incidence rate was 2/268 and 1/402 for males and females, respectively (Rabstein et al., 1973).

Brune et al., (1981) fed 0.15 mg/kg BAP (reported to be "highly pure") in the diet of 32 Sprague-Dawley rats/sex/group either every 9th day or 5 times/week. These treatments resulted in annual average doses of 6 or 39 mg/kg, respectively. An untreated group of 32 rats/sex served as the control. Rats were treated until moribund or dead; survival was similar in all groups. Histologic examinations were performed on each rat. The combined incidence of tumors of the forestomach, esophagus and larynx was 3/64, 3/64 and 10/64 in the control group, the group fed BAP every 9th day and the group fed BAP 5 times/week, respectively. A trend analysis showed a statistically significant tendancy for the proportion of animals with tumors of the forestomach, esophagus or larynx to increase steadily with dose (Knauf and Rice, 1992).

As part of the same study, Brune et al. (1981) administered BAP ("highly pure") orally to Sprague-Dawley rats by caffeine gavage. The rats were treated until moribund or dead; all rats were subjected to terminal histopathologic examination. Gavaged rats were divided into 3 dose groups of 32 rats/sex/group; the groups received 0.15 mg/kg per gavage either every 9th day (Group A), every 3rd day (Group B) or 5 times per week (Group C); these treatments resulted in annual average doses of 6, 18 or 39 mg/kg, respectively. Untreated and gavage (5 times/week) controls (32 rats/sex/group) were included. The median survival times for the untreated control group; the gavage control group; and groups A, B and C were 129, 102, 112, 113 and 87 weeks, respectively. The survival time of Group C was short compared with controls and may have precluded tumor formation (Knauf and Rice, 1992). The combined tumor incidence in the forestomach, esophagus and larynx was 3/64, 6/64, 13/64, 26/64 and 14/64 for the untreated control group, gavage control group, group A, group B and group C, respectively. There was a statistically significant association between the dose and the proportions of rats with tumors of the forestomach, esophagus or larynx. This association is not characterized by a linear trend. The linearity was affected by the apparently reduced tumor incidence that is seen in the high-dose group (Knauf and Rice, 1992).

Intratracheal instillation and inhalation studies in guinea pigs, hamsters and rats have resulted in elevated incidences of respiratory tract and upper digestive tract tumors (U.S. EPA, 1991a). Male Syrian golden hamsters (24/group) were exposed by inhalation to 0, 2.2, 9.5 or 46.5 mg

BAP/cu.m in a sodium chloride aerosol (Thyssen et al., 1981). (Greater than 99% of the particles had diameters between 0.2 and 0.5 um.) For the first 10 weeks of the study, the hamsters were exposed to BAP daily for 4.5 hours/day; thereafter, daily for 3 hours/day. Animals dying within the first year of the study were replaced; the effective number of hamsters in the control, low-, mid- and high-dose groups was 27, 27, 26 and 25, respectively. (The total time of treatment, although over 60 weeks, was not stated.) During the first 10 weeks, animals in the 3 dose groups reportedly lost weight. After week 10, however, the body weights in all groups were similar until week 60 when the body weights of hamsters in the high-dose group decreased and the mortality increased significantly. The incidence of respiratory tract tumors (including tumors of the nasal cavity, larynx and trachea) in the control, low-, mid- and high-dose groups was 0/27, 0/27, 9/26 and 13/25, respectively; the incidences of upper digestive tract tumors (including tumors of the pharynx, esophagus and forestomach) were 0/27, 0/27, 7/26 and 14/25, respectively. Trend analysis for incidences of both respiratory tract tumors and upper gastrointestinal tract tumors showed a statistically significant tendancy for the proportion of animals with either tumor type to increase steadily with increased dose (Knauf and Rice, 1992).

Intraperitoneal BAP injections have caused increases in the number of injection site tumors in mice and rats (reviewed in U.S. EPA, 1991a). Subcutaneous BAP injections have caused increases in the number of injection site tumors in mice, rats, guinea pigs, hamsters and some primates (IARC, 1983; U.S. EPA, 1991a). BAP is commonly used as a positive control in many dermal application bioassays and has been shown to cause skin tumors in mice, rats, rabbits and guinea pigs. BAP is both an initiator and a complete carcinogen in mouse skin (IARC, 1983). Increased incidences of distant site tumors have also been reported in animals as a consequence of dermal BAP exposure (reviewed in U.S. EPA, 1991a).

BAP has also been reported to be carcinogenic in animals when administered by the following routes: i.v.; transplacentally; implantation in the stomach wall, lung, renal parenchyma and brain; injection into the renal pelvis; and vaginal painting (U.S. EPA, 1991a).

Supporting Data for Carcinogenicity

Benzo[a]pyrene has been shown to cause genotoxic effects in a broad range of prokaryotic and mammalian cell assay systems (U.S. EPA, 1991a). In prokaryotes, BAP tested positive in DNA damage assays and in both reverse and forward mutation assays. In mammalian cell culture assays, BAP tested positive in DNA damage assays, forward mutation assays, chromosomal effects assays and cell transformation assays.

M.7.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Summary of Risk Estimates

Oral Slope Factor -- 7.3E+0 per (mg/kg)/day
Drinking Water Unit Risk -- 2.1E-4 per (ug/L)
Extrapolation Method -- Risk estimate based on a geometric mean of four slope factors obtained by differing modeling procedures. Derived from the combination of

multiple data sets from two different reports using more than one sex and species.
Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	5E-1 ug/L
E-5 (1 in 100,000)	5E-2 ug/L
E-6 (1 in 1,000,000)	5E-3 ug/L

Dose-Response Data
Tumor Type -- forestomach, squamous cell papillomas and carcinomas
Test Animals -- CFW mice, sex unknown
Route -- oral, diet
Reference -- Neal and Rigdon, 1967

Tumor Type -- squamous cell carcinoma of the forestomach
Test Animals -- SWR/J Swill mice
Route -- oral, diet
Reference -- Rabstein et al., 1973

Test Animals -- Sprague-Dawley rats, males and females
Route -- oral, diet
Reference -- Brune et al., 1981

Additional Comments
At the June 1992 CRAVE Work Group meeting, it was noted that an error had been made in the 1991 document "Dose-Response Analysis of Ingested Benzo[a]pyrene" which is quoted in the Drinking Water Criteria Document for PAH. In the calculation of the doses in the Brune et al. (1981) study it was erroneously concluded that doses were given in units of mg/year, whereas it was in fact mg/kg/year. When the doses are corrected the slope factor is correctly calculated as 11.7 per (mg/kg)/day, as opposed to 4.7 per (mg/kg)/day as reported in the Drinking Water Criteria Document. The correct range of slope factors is 4.5 to 11.7 per (mg/kg)/day, with a geometric mean of 7.3 per (mg/kg)/day. A drinking water unit risk based on the revised slope factor is 2.1E-4 per (ug/L). Therefore, these values have been changed on IRIS and an Erratum to the Drinking Water Criteria Document is being prepared.

Risk estimates were calculated from two different studies in two species of outbred rodents (Neal and Rigdon, 1967; Brune et al., 1981). These studies have several commonalities including mode of administration, tumor sites, tumor types and the presumed mechanisms of action. The data sets were not combined prior to modeling (the preferred approach) because they employed significantly dissimilar protocols.

The geometric mean from several slope factors, each considered to be of equal merit, was used to calculate a single unit risk. These four slope factor estimates span less than a factor of three and each is based on an acceptable, but less-than-optimal, data set. Each estimate is based on

a low-dose extrapolation procedure which entails the use of multiple assumptions and default procedures.

Clement Associates (1990) fit the Neal and Rigdon (1967) data to a two-stage dose response model. In this model the transition rates and the growth rate of preneoplastic cells were both considered to be exposure-dependent. (The functional form for the dose-dependence of preneoplastic cell growth rate was simple saturation.) A term to permit the modeling of BAP as its own promoter was also included. Historical control stomach tumor data from a related, but not identical, mouse strain, SWR/J Swill (Rabstein et al., 1973) and the CFW Texas colony (Neal and Rigdon, 1967) were used in the modeling. In calculating the lifetime unit risk for humans several standard assumptions were made: mouse food consumption was 13% of its body weight/day; human body weight was assumed to be 70 kg and the assumed body weight of the mouse 0.034 kg. The standard assumption of surface area equivalence between mice and humans was the cube root of 70/0.034. A conditional upper bound estimate was calculated to be 5.9 per (mg/kg)/day (U.S. EPA, 1991a).

A U.S. EPA report (1991b) argued that the upper-bound estimate calculated in Clement Associates (1990) involved the use of unrealistic conditions placed on certain parameters of the equation. Other objections to this slope factor were also raised. The authors of this report used the Neal and Rigdon (1967) data to generate an upper-bound estimate extrapolated linearly from the 10% response point to the background of an empirically fitted dose-response curve (Clement Associates, 1990). Other results, from similar concepts and approaches used for other compounds, suggest that the potency slopes calculated in this manner are comparable to those obtained from a linearized multistage procedure for the majority of the other compounds. The upper bound estimate calculated in U.S. EPA (1991b) is 9.0 per (mg/kg)/day.

The authors of U.S. EPA (1991b) selected a model to reflect the partial lifetime exposure pattern over different parts of the animals' lifetimes. The authors thought that this approach more closely reflected the Neal and Rigdon (1967) regimen. A Weibull-type dose-response model was selected to accommodate the partial lifetime exposure; the upper-bound slope factor calculated from this method was 4.5 per (mg/kg)/day.

Using the dietary portion of the Brune et al. (1981) rat data, a linearized multistage procedure was used to calculate an upper bound slope factor for humans. In the interspecies conversion the assumed human body weight was 70 kg and the rat 0.4 kg. The slope factor calculated by this method was 11.7 per (mg/kg)/day.

Discussion of Confidence

The data are considered to be less than optimal, but acceptable. There are precedents for using multiple data sets from different studies using more than one sex, strain and species; the use of the geometric mean of four slope factors is preferred because it makes use of more of the available data. The use of the geometric means was based on arguments presented in a personal communication (Stiteler, 1991).

M.7.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Not available.

M.7.2.3 Carcinogenic Assessment References

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M.8 BERYLLIUM

Status of Data	*Last Revised*
Oral RfD Assessment	02/01/93
Inhalation RfC Assessment	no data
Carcinogenicity Assessment	09/01/92

M.8.1 NONCARCINOGENIC ASSESSMENT

M.8.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD
No adverse effects Rat, Chronic Oral Bioassay	NOAEL: 5 ppm in drinking water (0.54 mg/kg bw/day)	100	1	5E-3 mg/kg/day
Schroeder and Mitchner, 1975	LOAEL: none			

*Conversion Factors: 5 ppm (5 mg/L) x 0.035 L/day / 0.325 kg bw = 0.54 mg/kg bw/day

Principal and Supporting Studies

Schroeder, H.A. and M. Mitchner. 1975. Life-term studies in rats: Effects of aluminum, barium, beryllium and tungsten. J. Nutr. 105: 421-427.

Fifty-two weanling Long-Evans rats of each sex received 0 or 5 ppm beryllium (as BeSO4, beryllium sulfate) in drinking water. Exposure was for the lifetime of the animals. At natural death the rats were dissected and gross and microscopic changes were noted in heart, kidney, liver, and spleen. There were no effects of treatment on these organs or on lifespan, urinalysis, serum glucose, cholesterol, and uric acid, or on numbers of tumors. Male rats experienced decreased growth rates from 2 to 6 months of age.

Similar studies were carried out on Swiss (CD strain) mice in groups of 54/sex at doses of approximately 0.95 mg/kg/day (Schroeder and Mitchner, 1975). Female animals showed decreased body weight compared with untreated mice at 6 of 8 intervals. Male mice exhibited slight increases in body weight. These effects were not considered adverse, therefore, 0.95 mg/kg/day is considered a NOAEL.

An unpublished investigation by Cox et al. (1975) indicates a much higher dose level (approximately 25 mg/kg/day) in the diet may be a NOEL.

Uncertainty and Modifying Factors

UF -- The uncertainty factor of 100 reflects a factor of 10 each for interspecies conversion and for the protection of sensitive human subpopulations.

MF -- None

Additional Comments
This RfD is limited to soluble beryllium salts. Data on the teratogenicity or reproductive effects of beryllium are limited. It has been reported to produce embryolethality and terata in chick embryos (Puzanova et al., 1978).

Confidence in the Oral RfD
Study -- Low
Data Base -- Low
RfD -- Low

Confidence in the study is rated as low because only one dose level was administered. Although numerous inhalation investigations and a supporting chronic oral bioassay in mice exist, along with the work by Cox et al. (1975) which indicates that a higher dose level might be a NOEL, these studies are considered as low to medium quality; thus, the data base is given a low confidence rating. The overall confidence in the RfD is low, reflecting the need for more toxicity data by the oral route.

M.8.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.8.2 CARCINOGENIC ASSESSMENT

Weight-of-Evidence Classification
Classification -- B2; probable human carcinogen.

Basis -- Beryllium has been shown to induce lung cancer via inhalation in rats and monkeys and to induce osteosarcomas in rabbits via intravenous or intramedullary injection. Human epidemiology studies are considered to be inadequate.

Human Carcinogenicity Data
Inadequate. Reported increases, while apparently associated with exposure, did not take a variety of possible confounding factors into account. Wagoner et al. (1980) observed 47 deaths from cancer among 3055 white males employed in beryllium-processing with a median duration of employment of 7.2 months. Among the 2068 followed for 25 years or more, 20 lung cancer deaths were observed. These increased incidences were statistically significant. When lung cancer mortality data became available for 1968-1975, the number of expected deaths was recalculated and the increased incidence was statistically significant only among workers followed 25 years or more (Bayliss, 1980; MacMahon, 1977, 1978). When the number of expected deaths was adjusted for smoking, the increased incidence was no longer significant (U.S. EPA, 1986).

An earlier study of workers from this same beryllium processing plant, and several studies of workers from this plant combined with workers from other beryllium plants, have reported a statistically significant increased incidence of lung cancer (Bayliss and Wagoner, 1977; Mancuso, 1970, 1979, 1980). No adjustment was made for smoking in these studies, and all were limited in their ability to detect a possible increased incidence of lung cancer because of methodological constraints and deficiencies.

Animal Carcinogenicity Data

Sufficient. Based on the evidence for induction of tumors by a variety of beryllium compounds in male and female monkeys and in several strains of rats of both sexes, via inhalation and intratracheal instillation, and the induction of osteosarcomas in rabbits by intravenous or intramedullary injection in multiple studies.

Slight increases in cancer incidence (not statistically significant in comparison with controls) were reported in Long-Evans rats (52/sex/group) administered 5 ppm beryllium sulfate in the drinking water for a lifetime. The authors reported a slight excess of grossly observed tumors in the 5 ppm group (9/33) over controls (4/26) in the male rats. The power of this test to detect a carcinogenic effect was reduced by high mortality (approximately 60% survived a pneumonia epidemic at 20 months) (Schroeder and Mitchener, 1975a). Schroeder and Mitchener (1975b) administered 5 ppm beryllium sulfate in drinking water to Swiss mice (54/sex/group) over a lifetime. A non-statistically significant increase in incidence of lymphoma leukemias were reported in the females (9/52) relative to controls (3/47).

An increase in reticulum cell sarcomas of the lungs was seen in male, but not female Wistar-derived rats administered beryllium sulfate in the diet at 5 and 50 ppm, but not at 500 ppm (Morgareidge et al., 1977). The incidence in males equaled 10/49, 17/35, 16/40 and 12/39 for the control, low, intermediate and high dose groups, respectively. Since the results were published only as an abstract, and since no response was seen at the highest dose, these results are considered to be only suggestive for the induction of cancer via this route.

Osteogenic sarcomas were induced in rabbits by intravenous injection of beryllium compounds in at least 12 different studies and by intramedullary injection in at least four studies (U.S. EPA, 1991). Bone tumors were induced by beryllium oxide, zinc beryllium silicate, beryllium phosphate, beryllium silicate and beryllium metal. No bone tumors were reported to be induced by intravenous injection of beryllium oxide or zinc beryllium silicate in rats or guinea pigs (Gardner and Heslington, 1946). Positive results, however, were reported in mice injected with zinc beryllium silicate, although the numbers were not listed (Cloudman et al., 1949). The sarcomas were generally reported to be quite malignant and metastasized to other organs.

Lung tumors, primarily adenomas and adenocarcinomas, have been induced via the inhalation route in both male and female Sprague-Dawley rats during exposure periods of up to 72 weeks by beryllium sulfate (Reeves et al., 1967), in both male and female Sherman and Wistar rats by beryllium phosphate, beryllium fluoride and zinc beryllium silicate (Schepers, 1961), in male

Charles River CR-CD rats by beryl ore (Wagner et al., 1969) and in both male and female rhesus monkeys by beryllium sulfate (Vorwald, 1968). Positive results were seen in rats exposed to beryllium sulfate at concentrations as low as 2 ug/cu.m (Vorwald, 1968).

Tumors were also induced by intratracheal instillation of metallic beryllium, beryllium-aluminum alloys and beryllium oxide in both Wistar rats and rhesus monkeys. Adenomas, adenocarcinomas and malignant lymphomas were seen in the lungs, with lymphosarcomas and fibrosarcomas present at extrapulmonary sites (Groth et al., 1980; Ishinishi et al., 1980).

Supporting Data for Carcinogenicity
Beryllium sufate and beryllium chloride have been shown to be nonmutagenic in bacterial and yeast gene mutation assays (Simmon et al., 1979). In contrast, gene mutation studies in Chinese hamster V79 and CHO cells were positive (Miyaki et al., 1979; Hsie et al., 1979). Chromosomal aberrations and sister chromatid exchange were also induced by beryllium in cultured human lymphocytes and Syrain hamster embryo cells (Larramendy et al., 1981).

M.8.1.3 Noncarcinogenic Reference Dose References

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M.8.2 CARCINOGENIC ASSESSMENT

M.8.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Summary of Risk Estimates
Oral Slope Factor -- 4.3 per(mg/kg)/day
Drinking Water Unit Risk -- 1.2E-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)	8.3E-1 ug/L
E-5 (1 in 100,000)	8.3E-2 ug/L
E-6 (1 in 1,000,000)	8.3E-3 ug/L

Dose-Response Data
Tumor Type -- gross tumors, all sites combined
Test Animals -- rat/Long-Evans, male
Route -- drinking water
Reference -- Schroeder and Mitchener, 1975a

Additional Comments
The solubility and speciation of beryllium in air and water media vary, with ambient air characterized by relatively insoluble beryllium compounds such as beryllium oxide and metallic beryllium, and water characterized by more soluble forms. Carcinogenic potency varies according to the form of beryllium present.

Human equivalent doses were calculated using a human body weight of 70 kg, an animal weight of 0.325 kg and length of exposure, experiment and lifespan of 1126 days for treated and control animals.

The unit risk should not be used if the water concentration exceeds 8.3E+1 ug/L, since above this concentration the unit risk may not be appropriate.

Discussion of Confidence
The estimate is derived from a study which did not show a significant increase in tumorigenic response. While this study is limited by use of only one non-zero dose group, the occurrence of high mortality and unspecified type and site of the tumors, it was used as the basis of the quantitative estimate because exposure occurred via the most relevant route. Oral risk estimates derived by extrapolation from studies in other species/strains for the intravenous and inhalation routes (also highly uncertain) are within an order of magnitude.

M.8.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Summary of Risk Estimates
Inhalation Unit Risk -- 2.4E-3 per (ug/cu.m)
Extrapolation Method -- Relative risk
Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	4E-2 ug/cu.m
E-5 (1 in 100,000)	4E-3 ug/cu.m
E-6 (1 in 1,000,000)	4E-4 ug/cu.m

Dose-Response Data
Tumor Type --
Test Animals -- humans
Route -- inhalation, occupational exposure
Reference --

Additional Comments
Human data were used for the inhalation exposure quantitation despite limitations in the study. Humans are most likely to be exposed by inhalation to beryllium oxide, rather than other beryllium salts. Animal studies by inhalation of beryllium oxide have utilized intratracheal instillation, rather than general inhalation exposure.

Effective dose was determined by adjusting for duration of daily(8/24 hours) and annual (240/365 days) exposure, and the fraction of thelifetime at risk (i.e., time from onset of employment to termination of follow-up). The risk estimates were based on the data of Wagoner et al. (1980) in which the smoking adjusted, expected lung cancer deaths were found to range from 13.91 to 14.67, in comparison to 20 observed. Relative risk estimates of 1.36 and 1.44 were derived and the 95% confidence limits of these estimates, 1.98 and 2.09, respectively, were used to estimate the lifetime cancer risk. Note that all of the above estimates are based on one data set using a range of estimated exposure and exposure times. Because of uncertainties regarding workplace beryllium concentration and exposure duration, unit risks were derived using two estimates each of concentration, fraction of lifetime exposed and relative risk. The recommended value is the arithmetic mean of the 8 derived unit risks.

The unit risk should not be used if the air concentration exceeds 4 ug/cu.m, since above this concentration the unit risk may not be appropriate.

Discussion of Confidence
The estimate of risk for inhalation exposure was based upon an epidemiologic study having several confounding variables. The estimates of exposure levels and duration were also somewhat uncertain. While a quantitative assessment based on several animal studies resulted in a similar estimate of risk (which increases the confidence somewhat), the quality of the available studies was poor (that is, they were conducted at single dose levels or lacked control groups).

M.8.2.3 Carcinogenic Assessment Reference

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M.9 CADMIUM

Status of Data	*Last Revised*
Oral RfD Assessment	02/01/94
Inhalation RfC Assessment	pending
Carcinogenicity Assessment	06/01/62

M.9.1 NONCARCINOGENIC ASSESSMENT

M.9.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD
Significant proteinuria	NOAEL (water): 0.005 mg/kg/day (water)	10	1	5E-4 mg/kg/day
Human studies involving chronic exposures	NOAEL (food): 0.01 mg/kg/day (food)	10	1	1E-3 mg/kg/day

U.S. EPA, 1985

*Conversion Factors: See text for discussion

Principal and Supporting Sudies

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).

Uncertainty and Modifying Factors

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

Additiona Comments

Cd is unusual in relation to most, if not all, of the substances for which an oral RfD has been determined in that a vast quantity of both human and animal toxicity data are available. The RfD is based on the highest level of Cd in the human renal cortex (i.e., the critical level) not associated with significant proteinuria (i.e., the critical effect). A toxicokinetic model has been used to determine the highest level of exposure associated with the lack of a critical effect. Since the fraction of ingested Cd that is absorbed appears to vary with the source (e.g., food vs. drinking water), it is necessary to allow for this difference in absorption when using the toxicokinetic model to determine an RfD.

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.

M.9.1.2 Reference Concentration for Chronic Inhalation Exposure

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

M.9.1.3 Noncarcinogenic Reference Dose References

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contaminants mercury, lead, and cadmium. Sixteenth Report of the Joint FAO/WHO Expert Committee on Food Additives. WHO Technical Report Series No. 505, FAO Nutrition Meetings Report Series No. 51. Geneva, Switzerland.

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M.9.2 CARCINOGENIC ASSESSMENT

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.

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.

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.

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).

M.9.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Not available. There are no positive studies of orally ingested cadmium suitable for quantitation.

M.9.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

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

Dose-Response Data
Tumor Type -- lung, trachea, bronchus cancer deaths
Test Animals -- human/white male
Route -- inhalation, exposure in the workplace
Reference -- Thun et al., 1985

Additional Comments
The unit risk should not be used if the air concentration exceeds 6 ug/cu.m, since above this concentration the unit risk may not be appropriate.

Discussion of Confidence
The data were derived from a relatively large cohort. Effects of arsenic and smoking were accounted for in the quantitative analysis for cadmium effects.

An inhalation unit risk for cadmium based on the Takenaka et al. (1983) analysis is 9.2E-2 per (ug/cu.m). While this estimate is higher than that derived from human data [1.8E-3 per (ug/cu.m)] and thus more conservative, it was felt that the use of available human data was more reliable because of species variations in response and the type of exposure (cadmium salt vs. cadmium fume and cadmium oxide).

M.9.2.3 Carcinogenic Assessment References

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Watanabe, T., T. Shimada and A. Endo. 1979. Mutagenic effects of cadmium on mammalian oocyte chromosomes. Mutat. Res. 67: 349-356.

M.10 CHLORDANE

Status of Data	*Last Revised*
Oral RfD Assessment	07/01/89
Inhalation RfC Assessment	Pending
Carcinogenicity Assessment	07/01/93

M.10.1 NONCARCINOGENIC ASSESSMENT

M.10.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD	
Regional liver hypertrophy in females	NOEL: 1 ppm (0.055 mg/kg/day)		1000	1 mg/kg/day	6E-5
30-Month Rat Feeding Study	LEL: 5 ppm (0.273 mg/kg/day)				

Velsicol Chemical Co.,1983a

*Conversion Factors: Actual dose tested

Principal an Supporting Studies

Velsicol Chemical Company. 1983a. MRID No. 00138591, 00144313. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Charles River Fischer 344 rats (80/sex/dose) were fed technical chlordane at dietary levels of 0, 1, 5, and 25 ppm for 130 weeks. Body weight, food consumption, and water uptake were monitored at regular intervals. Clinical laboratory studies were performed and organ weights measured on eight animals/sex/group at weeks 26 and 52, and on all survivors at week 130. Gross and microscopic pathology were performed on all tissues. Daily dose level of 0.045, 0.229, and 1.175 mg/kg/day for males and 0.055, 0.273, and 1.409 mg/kg/day for females for the 1, 5, and 25 ppm treatment groups, respectively, were calculated from food consumption and body weight data.

Following the submission of a 30-month chronic feeding/oncogenicity study in Fischer 344 rats, the Agency reviews by the Office of Pesticides Programs and the Cancer Assessment Group of these data indicated that male rats at the highest dosage exhibited an increase in liver tumors (ICF Clement, 1987). The registrant, Velsicol Chemical Company, subsequently convened the Pathology Working Group to reevaluate the slides of livers of the chlordane-treated rats reported in MRID No. 00138591. It was concluded that liver lesions had not occurred in male rats and that 25 ppm (0.1175 mg/kg/day) was the NOEL for males. Liver lesions (hypertrophy), however, had occurred in female rats at 5 ppm (0.273

mg/kg/day), which was considered an LEL. Therefore an NOEL of 1 ppm (0.055 mg/kg/day) (LDT) was established for female rats.

Uncertainty and Modifying Factors

UF -- An uncertainty factor of 100 was used to account for the inter- and intraspecies differences. An additional UF of 10 was used to account for the lack of an adequate reproduction study and adequate chronic study in a second mammalian species, and the generally inadequate sensitive endpoints studied in existing studies, particularly since chlordane is known to bioaccumulate over a chronic duration.

MF -- None

Additional Comments

Data Considered for Establishing the RfD

1) 30-Month Feeding (oncogenic) - rat: Principal study - see previous description; core grade minimum

2) 24-Month Chronic Toxicity - mouse: NOEL=1 ppm (0.15 mg/kg/day); LEL=5 ppm (0.75 mg/kg/day) (hepatocellular swelling and necrosis in males; hepatocyte swelling in males, and increased live weight in males and females); At 12.5 ppm (1.875 mg/kg/day) (HDT); core grade minimum (Velsicol Chemical Co., 1983b)

Data Gap(s): Chronic Dog Feeding Study, Rat Reproduction Study, Rat Teratology Study, Rabbit Teratology Study

Confidence in the Oral RfD

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

The critical study is of adequate quality and is given a medium rating. The data base is given a low confidence rating because of 1) the lack of an adequate reproduction study and adequate chronic study in a second mammalian species and 2) inadequate sensitive endpoints studied in existing studies, particularly since chlordane is known to bioaccumulate over a chronic duration. Low confidence in the RfD follows.

M.10.1.2 Reference Concentration for Chronic Inhalation Exposure

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

M.10.1.3 Noncarcinogenic Assessment References

ICF-Clement. 1987. MRID No. 40433701. Available from EPA. Write to FOI, EPA, Washington DC 20460.

Velsicol Chemical Co. 1983a. MRID No. 00138591, 00144313. Available from EPA. Write to FOI, EPA, Washington DC 20460.

Velsicol Chemical Co. 1983b. MRID No. 00144312. Available from EPA. Write to FOI, EPA, Washington DC 20460.

M.10.2 CARCINOGENIC ASSESSMENT

Weight-of-Evidence Classification
Classification -- B2; probable human carcinogen

Basis -- Sufficient evidence in studies in which benign and malignant liver tumors were induced in four strains of mice of both sexes and in F344 male rats; structurally related to other liver carcinogens.

Human Carcinogenicity Data
Inadequate. There were 11 case reports involving central nervous system effects, blood dyscrasias and neuroblastomas in children with pre-/postnatal exposure to chlordane and heptachlor (Infante et al., 1978). As no other information was available, no conclusions can be drawn.

There were three epidemiologic studies of workers exposed to chlordane and/or heptachlor. One study of pesticide applicators was considered inadequate in sample size and duration of follow-up. This study showed marginal statistically significant increased mortality from bladder cancer (3 observed) (Wang and McMahon, 1979a). The other two studies were of pesticide manufacturing workers. Neither of them showed any statistically significantly increased cancer mortality (Wang and McMahon, 1979b; Ditraglia et al., 1981). Both these populations also had confounding exposures from other chemicals.

Animal Carcinogenicity Data
Sufficient. Chlordane has been studied in four mouse and four rat long-term carcinogenesis bioassays. Dose-related incidences of liver carcinoma constitute the major finding in mice. Becker and Sell (1979) tested chlordane (90:10 mixture of chlordane to heptachlor) in C57B1/6N mice, a strain historically known not to develop spontaneous liver tumors. An unspecified number of mice were fed chlordane at 0, 25 and 50 ppm (0, 3.57, 7.14 mg/kg bw) for 18 months. None of the controls developed tumors or nodular lesions of the liver. Twenty-seven percent (16 mice) of the surviving treated mice developed primary hepatocellular carcinomas. Velsicol (1973) fed groups of 100 male and 100 female CD-1 mice diets with 0, 5, 25 or 50 ppm analytical grade chlordane for 18 months. A significant

($p < 0.01$) dose-related increase in nodular hyperplasias in the liver of male and female mice was reported at the the two highest dose levels. A histological review by Reuber (U.S. EPA, 1985) reported a high incidence ($p < 0.01$) of hepatic carcinomas instead of hyperplastic nodules at 25 and 50 ppm.

A dose-related increase ($p < 0.001$ after lifetable adjustment) of hepatocellular carcinomas was also observed in both sexes of B6C3F1 mice (NCI, 1977). Male and female mice were fed technical-grade chlordane (purity\= 94.8%) at TWA concentrations (TWAC) of 29.9 and 56.2 ppm and 30.1 and 63.8 ppm, respectively, for 80 weeks. In this study there were individual matched controls for the low and high dose groups. ICR male mice developed hepatocellular adenomas and hemangiomas when fed 12.5 ppm chlordane for 24 months. No tumors were observed in the female mice when tested at the same concentrations: 0, 1, 5, and 12.5 ppm (Velsicol, 1983a).

Velsicol (1983b) reported a long-term (130 weeks) carcinogenesis bioassay on 80 male and 80 female F344 rats fed concentrations of 0, 1, 5, and 25 ppm chlordane. A significant increase in adenomas of the liver was observed in male rats receiving 25 ppm. Although no tumors were observed in female rats, hepatocellular swelling was significantly increased at 25 ppm. The NCI (1977) reported a significant increase ($p < 0.05$) of neoplastic nodules of the liver in low-dose Osborne-Mendel female rats (TWAC of 120.8 ppm) but not in the high-dose group (TWAC of 241.5 ppm). No tumor incidence was reported for the males fed TWAC of 203.5 and 407 ppm. Loss of body weight and a dose-related increase in mortality was observed in all treated groups. High mortality and reduced growth rates in Osborne-Mendel rats was also observed by Ingle (1952) when the rats were exposed to 150 and 300 ppm chlordane but not at 5, 10, and 30 ppm. No treatment-related incidence of tumors was reported. Significantly enlarged livers and liver lesions were found in male and female albino rats fed chlordane at greater than or equal to 80 ppm (Ambrose et al., 1953a,b). No treatment-related increase in tumors was found, but the study duration (400 days) was short.

Supporting Data for Carcinogenicity

Gene mutation assays indicate that chlordane is not mutagenic in bacteria (Wildeman and Nazar, 1982; Probst et al., 1981; Gentile et al., 1982). Positive results have been reported in Chinese hamster lung V79 cells and mouse lymphoma L5178Y cells with and without exogenous metabolism, as well as in plant assays. Chlordane did not induce DNA repair in bacteria, rodent hepatocytes (Maslansky and Williams, 1981), or human lymphoid cells (Sobti et al., 1983). It is a genotoxicant in yeast (Gentile et al., 1982; Chambers and Dutta, 1976), human fibroblasts (Ahmed et al., 1977), and fish (Vigfusson et al., 1983).

Five compounds structurally related to chlordane (aldrin, dieldrin, heptachlor, heptachlor epoxide, and chlorendic acid) have produced liver tumors in mice. Chlorendic acid has also produced liver tumors in rats.

M.10.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Summary of Risk Estimates
Oral Slope Factor -- 1.3E+0 per (mg/kg)/day
Drinking Water Unit Risk -- 3.7E-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)	3E+0 ug/L
E-5 (1 in 100,000)	3E-1 ug/L
E-6 (1 in 1,000,000)	3E-2 ug/L

Dose-Response Data
Tumor Type -- hepatocellular carcinoma
Test Animals -- mouse/CD-1 (Velsicol); mouse/B6C3F1 (NCI)
Route -- diet
Reference -- Velsicol, 1973; NCI, 1977

Additional Comments
Four data sets for mice and one data set for rats showed a significant increase in liver tumors; namely hepatocellular carcinomas in mice (NCI, 1977; Velsicol, 1973) and hepatocellular adenomas in rats (Velsicol, 1983a). The quantitative estimate is based on the geometric mean from the four mouse data sets as mice were the more sensitive species tested and as risk estimates for a similar compound (heptachlor) were similarly derived from mouse tumor data. The slope factors for the data sets are these: 2.98 per (mg/kg)/day for CD-1 female mice, 4.74 per (mg/kg)/day for CD-1 male mice, 0.76 per (mg/kg)/day for B6C3F1 male mice, and 0.25 per (mg/kg)/day for B6C3F1 female mice. Low and high dose groups in the NCI (1977) study had individual matched controls.

The unit risk should not be used if the water concentration exceeds 300 ug/L, since above this concentration the unit risk may not be appropriate.

Discussion of Confidence
Liver carcinomas were induced in mice of both sexes in two studies. An adequate number of animals was observed, and dose-response effects were reported in all studies. The geometric mean of slope factors (0.25 to 4.74 per (mg/kg)/day for the most sensitive species is consistent with that derived from rat data (1.11/mg/kg/day).

M.10.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation

Summary of Risk Estimates
Inhalation Unit Risk -- 3.7E-4 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)	3E-1 ug/cu.m
E-5 (1 in 100,000)	3E-2 ug/cu.m
E-6 (1 in 1,000,000)	3E-3 ug/cu.m

Dose-Response Data for Carcinogenicity
The inhalation risk estimates were calculated from the oral data.

Additional Comments
The unit risk should not be used if the air concentration exceeds 30 ug/cu.m, above this concentration the unit risk may not be appropriate.

Discussion of Confidence
See oral exposure.

M.10.2.3 Carcinogenic Assessment References

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M.12 CHROMIUM (VI)

Status of Data	*Last Revised*
Oral RfD Assessment	02/01/95
Inhalation RfC Assessment	Pending
Carcinogenicity Assessment	03/01/91

M.12.1 NONCARCINOGENIC ASSESSMENT

M.12.1.1 Noncarcinogenic Reference Dose for Chronic Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD
No effects reported mg/kg/ay	NOAEL: 25 mg/L of chromium as K2CrO4	500	1	5E-3
Rat, 1-Year Drinking Study	(converted to 2.4 mg of chromium(VI)/kg/day)			
MacKenzie et al., 1958	LOAEL: none			

*Conversion Factors: Drinking water consumption = 0.097 L/kg/day (reported)

Principal and Supporting Studies

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

Groups of eight male and eight female Sprague-Dawley rats were supplied with drinking water containing 0-11 ppm (0-11 mg/L) hexavalent chromium (as K2CrO4) for 1 year. The control group (10/sex) received distilled water. A second experiment involved three groups of 12 males and 9 female rats. One group was given 25 ppm (25 mg/L) chromium (as K2CrO4); 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 on appearance, weight gain, or food consumption, and there were no pathologic changes in the blood or other tissues in any treatment group. The rats receiving 25 ppm of chromium (as K2CrO4) showed an approximate 20% reduction in water consumption. This dose corresponds to 2.4 mg chromium(VI)/kg/day based on actual body weight and water consumption data.

For rats treated with 0-11 ppm (in the diet), blood was examined monthly, and tissues (livers, kidneys and femurs) were examined at 6 months 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 months. An abrupt rise in tissue chromium concentrations was noted in rats treated with greater 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 and humans. Anwar et al. (1961) observed no significant effects in female dogs (2/dose group) given up to 11.2 ppm chromium(VI) (as K2CrO4) in drinking water for 4 years. The calculated doses were 0.012-0.30 mg/kg of chromium(VI). In humans, no adverse health effects were detected (by physical examination) in a family of four persons who drank for 3 years from a private well containing chromium(VI) at approximately 1 mg/L (0.03 mg/kg/day for a 70-kg human).

Uncertainty and Modifying Factors

UF -- The uncertainty factor of 500 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 5 to compensate for the less-than-lifetime exposure duration of the principal study.

MF -- None

Additional Comments

This RfD is limited to metallic chromium(VI) of soluble salts. Examples of soluble salts include potassium dichromate (K2CR2O7), sodium dichromate (Na2Cr2O7), potassium chromate (K2CrO4) and sodium chromate (Na2CrO4).

Trivalent chromium is an essential nutrient. There is some evidence to indicate that hexavalent chromium is reduced in part to trivalent chromium in vivo (Petrilli and DeFlora, 1977, 1978; Gruber and Jennette, 1978).

The literature available on possible fetal damage caused by chromium compounds is limited. No studies were located on teratogenic effects resulting from ingestion of chromium.

Confidence in the Oral RfD

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

Confidence in the chosen study is low because of the small number of animals tested, the small number of parameters measured and the lack of toxic effect at the highest dose tested. Confidence in the data base is low because the supporting studies are of equally low quality, and teratogenic and reproductive endpoints are not well studied. Low confidence in the RfD follows.

M.12.1.2 Reference Concentration for Chronic Inhalation Exposure

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

M.12.1.3 Noncarcinogenic Reference Dose References

Anwar, R.A., F.F. Langham, C.A. Hoppert, B.V. Alfredson and R.U. Byerrum. 1961. Chronic toxicity studies. III. Chronic toxicity of cadmium and chromium in dogs. Arch. Environ. 3: 456-460.

Gruber, J.E. and K.W. Jennette. 1978. Metabolism of the carcinogen chromate by rat liver microsomes. Biochem. Biophys. Res. Commun. 82(2): 700-706.

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Petrilli, F.L. and S. DeFlora. 1977. Toxicity and mutagencity of hexavalent chromium on Salmonella typhimurium. Appl. Environ. Microbiol. 33(4): 805-809.

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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, DC.

M.12.2 CARCINOGENIC ASSESSMENT

Weight-of-Evidence Classification
Classification -- A; human carcinogen

Basis -- Results of occupational epidemiologic studies of chromium-exposed workers are consistent across investigators and study populations. Dose-response relationships have been established for chromium exposure and lung cancer. Chromium-exposed workers are exposed to both chromium III and chromium VI compounds. Because only chromium VI has been found to be carcinogenic in animal studies, however, it was concluded that only chromium VI should be classified as a human carcinogen.

Human Carcinogenicity Data

Sufficient. Epidemiologic studies of chromate production facilities in the United States (Machle and Gregorius, 1948; Brinton et al., 1952; Mancuso and Hueper, 1951, Mancuso, 1975; Baetjer, 1950; Taylor, 1966; Enterline, 1974; Hayes et al., 1979; Hill and Ferguson, 1979), Great Britain (Bidstrup, 1951; Bidstrup and Case, 1956; Alderson et al., 1981), Japan (Watanabe and Fukuchi, 1975; Ohsaki et al., 1978; Sano and Mitohara, 1978; Satoh et al., 1981) and West Germany (Korallus et al., 1982; Bittersohl, 1971) have established an association between chromium (Cr) exposure and lung cancer. Most of these studies did not attempt to determine whether Cr III or Cr VI compounds were the etiologic agents.

Three studies of the chrome pigment industry, one in Norway (Langard and Norseth, 1975), one in England (Davies, 1978, 1979), and the third in the Netherlands and Germany (Frentzel-Beyme, 1983) also found an association between occupational chromium exposure (predominantly to Cr VI) and lung cancer.

Results of two studies of the chromium plating industry (Royle, 1975; Silverstein et al., 1981) were inconclusive, while the findings of a Japanese study of chrome platers were negative (Okubo and Tsuchiya, 1979). The results of studies of ferrochromium workers (Pokrovskaya and Shabynina, 1973; Langard et al., 1980; Axelsson et al., 1980) were inconclusive as to lung cancer risk.

Animal Carcinogenicity Data

Sufficient. Hexavalent chromium compounds were carcinogenic in animal assays producing the following tumor types: intramuscular injection site tumors in Fischer 344 and Bethesda Black rats and in C57BL mice (Furst et al., 1976; Maltoni, 1974, 1976; Payne, 1960; Heuper and Payne, 1959); intra- plural implant site tumors for various chromium VI compounds in Sprague-Dawley and Bethesda Black rats (Payne, 1960; Heuper 1961; Heuper 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).

Supporting Data for Carcinogenicity

A large number of chromium compounds have been assayed in in vitro genetic toxicology assays. In general, hexavalent chromium is mutagenic in bacterial assays whereas trivalent chromium is not (Lofroth, 1978; Petrellie and Flora, 1977, 1978). Likewise Cr VI but not Cr III was mutagenic in yeasts (Bonatti et al., 1976) and in V79 cells (Newbold et al., 1979). Chromium 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., 1978) 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).

There are no long-term studies of ingested Cr VI. There appears to be significant in vivo conversion of Cr VI to Cr III and III to VI; Cr III is an essential trace element.

M.12.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Not available.

M.12.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Summary of Risk Estimates
Inhalation Unit Risk -- 1.2E-2 per (ug/cu.m)
Extrapolation Method -- Multistage, extra risk
Air Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	8E-3 ug/cu.m
E-5 (1 in 100,000)	8E-4 ug/cu.m
E-6 (1 in 1,000,000)	8E-5 ug/cu.m

Dose-Response Data for Carcinogenicity

human Route: Occupational exposure (inhalation)

Age (years)	Midrange (ug/cu.m)	Deaths from Lung Cancer	Person	Reference
50	5.66	3	1345	Mancuso, 1975
	25.27	6	931	
	46.83	6	299	
60	4.68	4	1063	
	20.79	5	712	
	39.08	5	211	
Age (years)	Midrange (ug/cu.m)	Deaths from Lung Cancer	Person	Reference
70	4.41	2	401	
	21.29	4	345	

Additional Comments
The cancer mortality in Mancuso (1975) was assumed to be due to Cr VI, which was further assumed to be no less than one-seventh of total chromium. It was also assumed that the smoking habits of chromate workers were similar to those of the U.S. white male population. The unit risks of Langard et al. (1980), Axelsson et al. (1980), and Pokrovskaya and Shabynina (1973) are 1.3E-1, 3.5E-2 and 9.2E-2 per (ug/cu.m), respectively.

Hexavalent chromium compounds have not produced lung tumors in animals by inhalation. 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 ug/cu.m, since above this concentration the unit risk may not be appropriate.

Discussion of Confidence

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 7-fold underestimation of risk. The use of 1949 hygiene data, 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 male population, since it is generally accepted that the proportion of smokers is higher for industrial workers than for the general population.

M.12.2.3 Carcinogenic Assessment References

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M.13 COPPER

Status of Data	*Last Revised*
Oral RfD Assessment	No data
Inhalation RfC Assessment	No data
Carcinogenicity Assessment	08/01/91

M.13.1 NONCARCINOGENIC ASSESSMENT

M.13.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Not available at this time.

M.13.1.2 Noncarcinogenic Reference Dose for Chronic Inhalation Exposure

Not available at this time.

M.13.2 CARCINOGENIC ASSESSMENT

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.

Human Carcinogenicity Data
None.

Animal Carcinogenity 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.

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.

M.13.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Not available.

M.13.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Not available.

M.13.2.3 Carcinogenic Assessment References

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M.14 DIETHYL PHTHALATE

Status of Data	*Last Revised*
Oral RfD Assessment	02/01/93
Inhalation RfC Assessment	no data
Carcinogenicity Assessment	02/01/93

M.14.1 NONCARCINOGENIC ASSESSMENT

M.14.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD
Decreased growth rate, food consumption and altered organ weights	NOAEL: 1% of diet (750 mg/kg bw/day)	1000	1	8E-1 mg/kg/day
Rat, Subchronic Oral Feeding Study	LOAEL: 5% of diet (3160 mg/kg bw/day)			
Brown et al., 1978				

*Conversion Factors: Converted doses estimated by principal study authors, based on food consumption and body weight data.

Principal and Supporting Studies

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

Groups of CD rats (15/sex) were fed diets containing 0, 0.2, 1.0, or 5.0% DEP for 16 weeks. The authors estimated the mean intakes to be 0, 150, 770, and 3160 mg/kg/day for the males and 0, 150, 750, and 3710 mg/kg/day for the females. Additional groups of five rats/sex were fed similar diets for 2 or 6 weeks. Hematological examinations (red blood cell count, hematocrit, hemoglobin) were performed on animals fed diets for 2, 6, and 16 weeks. Differential white blood cell counts were also conducted on 0 and 5% dose groups at 16 weeks. Food and water intake and body weight were measured for all groups weekly. Urinalyses were conducted during weeks 2, 6, and 15 on 5 to 15 rats/sex/dose group. After 16 weeks of treatment, autopsy, hematologic and histologic examinations were conducted on all animals.

No changes in behavior or other clinical signs of toxicity were observed. The authors reported significantly less weight gain throughout the duration of the experiment in both sexes given 5% DEP (15 to 25% decrease) and in females (5 to 8% decrease) fed 1% DEP. Mean food consumption of the previous groups was also decreased (by 11 to 23%) relative to controls.

No significant dose- or time-related trends in urinalysis or hematology results were found. Absolute weights of brain, heart, spleen, and kidneys were decreased in both sexes fed 5% DEP. Relative weights of the brain, liver, kidneys, stomach, small intestines, and full caecum were significantly greater in both sexes after 16 weeks at the 5% dietary level when compared with controls. No histologic changes because of treatment were reported.

In another experiment summarized by Brown et al. (1978), groups of six rats/sex were pair-fed diets containing either 0 or 5% DEP for 16 weeks. Body weights were measured weekly. The authors reported that rats fed 5% DEP consumed more food and gained less weight than controls. The differences in food consumption (1 to 5%) were not statistically significant, and mean weight differences were 7 to 10%, which the authors reported as statistically significant.

The RfD receives support from the results of a 2-year feeding study using rats (Food Research Laboratories, Inc., 1955). Albino weanling rats (strain not specified) (15/sex) were fed 0, 0.5, 2.5, and 5.0% diethyl phthalate in the diet. Animals were maintained for a 2-year period during which two males and two females/group were examined at 12-week intervals for the following: red and white blood cell counts, differential white count, hemoglobin, blood sugar and nitrogen, and urinalysis. Growth of animals in the 5% treatment group was retarded throughout the study, with no depression of food intake. There was a significant decrease in efficiency of food utilization in this group compared with controls. There were no other treatment-related effects either on the parameters listed above or on gross organ appearance or histopathology.

Uncertainty and Modifying Factors

UF -- A factor of 10 for extrapolation from subchronic to chronic exposure, 10 for interspecies variation, and an additional 10-fold factor to protect sensitive human subpopulations were used in determining the RfD.

MF -- None

Additional Comments

Data regarding developmental and reproductive effects is extremely limited. Singh et al. (1972) observed skeletal malformations in Sprague-Dawley rats after i.p. administration (0.506, 1.012, and 1.686 mL/kg) on days 5, 10, and 15 of gestation. In addition, fetuses were significantly smaller than untreated controls. Exposure to DEP does not appear to affect the reproductive performance of mice after oral administration of 0.25, 1.25, and 2.5% DEP for 18 weeks (NTP, 1984). Second-generation breeding pairs exposed to 2.5% DEP exhibited increased right epididymis and prostate weights in males and decreased pituitary weight in females (NTP, 1984).

Confidence in the Oral RfD

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

Sufficient numbers of rats of both sexes were employed and multiple endpoints, including histopathology, were studied; confidence in the study is rated medium. Since only limited supporting data are available and the chosen study was of less than lifetime duration, confidence in the data base is rated low. Low confidence in the RfD follows.

M.14.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.14.1.3 Noncarcinogenic Assessment References

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

Food Research Laboratoes, Inc. 1955. Toxicological studies of diethyl phthalate. Laboratory No. 67567. Celanese Corp. of America. Summit Research Laborates, Summit, NJ.

NTP (National Toxicology Program). 1984. Diethyl Phthalate: Reproduction and fertility assessment in CD-1 mice when administered in the feed. Final report. NTP, Research Triangle Park, NC.

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M.14.2 CARCINOGENIC ASSESSMENT

Weight-of-Evidence Classification
Classification -- D; not classifiable as a human carcinogen.

Basis -- Pertinent data regarding carcinogenicity were not located in the available literature.

Human Carcinogenicity Data
None.

Animal Carcinogenicity Data
Inadequate. Dietary studies in rats with exposure durations of 2 years (Food Research Laboratories, Inc., 1955) and 16 weeks (Brown et al., 1978) were not designed to measure carcinogenic effects.

Supporting Data for Carcinogenicity
DEP was found to be a weak direct-acting mutagen in forward and reverse mutation assays in Salmonella typhimurium (Seed, 1982; Rubin et al., 1979; Kozumbo et al., 1982). DEP was

negative in mammalian cell chromosomal aberration assays (Ishidate and Odashima, 1977; Tsuchiya and Hattori, 1977). Research indicates that DEP is hydrolyzed to monoesters (Rowland et al., 1977). There is limited evidence that DEP is a weak inducer of peroxisome proliferation (U.S. EPA, 1987).

M.14.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Not available.

M.14.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Not available.

M.14.2.3 Carcinogenic Assessment References

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

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Kozumbo, W.J., R. Kroll and R.J. Rubin. 1982. Assessment of the mutagenicity of phthalate esters. Environ. Health Perspect. 45: 103-109.

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Rubin, R.J., W. Kozumbo and R. Kroll. 1979. Ames mutagenic assay of a series of phthalic acid esters: Positive response of the dimethyl and diethyl esters in TA100. Soc. Toxicol. Ann. Meet., New Orleans, March 11-15. p. 11. (Abstract)

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.

M.15 2,4-DINITROTOLUENE

Status of Data	*Last Revised*
Oral RfD Assessment	No data
Inhalation RfC Assessment	No data
Carcinogenicity Assessment	09/01/90

M.15.1 NONCARCINOGENIC ASSESSMENT

M.15.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Not available at this time.

M.15.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.15.2 CARCINOGENIC ASSESSMENT

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.

Human Carcinogenicity Data
None.

Animal Carcinogencity 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.

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).

M.15.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

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

Dose-Response Data
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

Additional Comments
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.

Discussion of Confidence
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.

M.15.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Not available.

M.15.2.3 Carcinogenic 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.

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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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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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M.16 2,6-DINITROTOLUENE

Status of Data	*Last Revised*
Oral RfD Assessment	02/01/93
Inhalation RfC Assessment	No data
Carcinogenicity Assessment	No data

M.16.1 NONCARCINOGENIC ASSESSMENT

M.16.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses	UF	MF	RfD
Neurotoxicity, Heinz bodies and biliary tract hyperplasia	NOAEL: 0.2 mg/kg/day	100	1	2E-3 mg/kg/day
Dog Feeding Study 2-Year	LOAEL: 1.5 mg/kg/day			
Ellis et al., 1985				

Principal Supporting Studies

Ellis, H.V., C.B. Hong, C.C. Lee, J.C. Dacre and J.P. Glennon. 1985. Subchronic and chronic toxicity studies of 2,4-dinitrotoluene. Part I. Beagle dogs. J. Am. College Toxicol. 4(4): 233-242.

Ellis et al. (1985) reported the results of a chronic toxicity study commissioned by the U.S. Army (Ellis et al., 1979) of dogs fed 98% pure 2,4-dinitrotoluene (2,4-DNT) for up to 24 months. Groups of beagle dogs (6/sex/dose) were fed 2,4-DNT in gelatin capsules at 0, 0.2, 1.5, or 10 mg/kg/day. In male dogs fed 10 mg/kg/day, 4 of the 6 males were sacrificed moribund by study week 19 after exhibiting progressive paralysis. Neurotoxic effects, characterized by incoordination and paralysis, were exhibited by all dogs at this dose level within 6 months of study initiation and during month 16 in one dog receiving 1.5 mg/kg/day. CNS lesions included vacuolization, endothelial proliferation, and gliosis of the cerebellum. In dogs fed 1.5 and 10 mg/kg/day, there was methemoglobinemia with associated reticulocytosis and Heinz bodies; biliary tract hyperplasia; and pigmentation of the gallbladder, kidneys, and spleen. The hematologic effects were minimal during year 2, presumably due to an adaptive response. No males had testicular effects. The LOAEL in this study is 1.5 mg/kg/day based on neurotoxicity and the presence of Heinz bodies and biliary tract hyperplasia. The NOAEL is 0.2 mg/kg/day.

In a separate study (reported in Lee et al., 1978), groups of dogs (2/sex/dose) were given 2,4-DNT in capsules at doses of 0, 1, 5, or 25 mg/kg/day for 13 weeks. There was no apparent toxicity in the low- and mid-dose groups. In the high-dose group 2,4-DNT was toxic

after 12-22 days and was lethal after 22 or more days. There was great variation in individual susceptibility. All affected dogs exhibited decreased food consumption, weight loss, urine stains on the fur, pale gums, neuromuscular incoordination, and paralysis. Hematological indices showed methemoglobinemia, anemia, and Heinz bodies. The dogs were in fair to poor nutritional condition with little or no body fat. Histologically, there was hemosiderosis in the liver and spleen, cloudy swelling of the kidneys in males and females, and aspermatogenesis in males. Dogs sacrificed during weeks 6 and 7 had brain lesions characterized by gliosis, edema, and demyelination of the cerebellum, spinal cord, and brain stem. After 4 weeks, dogs partially recovered from the various effects. The LOAEL is 25 mg/kg/day based on body weight loss, hematological abnormalities, neurological signs, and histopathology. The NOAEL is 5 mg/kg/day because no DNT-related effects were observed at this and lower doses.

Lee et al. (1985) reported the results of a chronic toxicity study commissioned by the U.S. Army (Ellis et al., 1979) of rats fed 98% pure 2,4-DNT in the diet for up to 24 months. Groups of CD (Sprague-Dawley) rats (38/sex) were provided an average 2,4-DNT intake of 0, 0.57, 3.9, or 34 mg/kg/day for males, and 0, 0.71, 5.1, or 45 mg/kg/day for females. After 12 months, 8 animals/sex/group were killed for necropsy; the remaining rats were sacrificed after 24 months. Four animals/sex/group were sacrificed at 13 and 25 months after being returned to normal diets for 1 month.

Cumulative deaths in high-dose males and females were significantly higher than in controls; 50% mortality occurred in high-dose rats by month 20 and in controls by month 23. Weight gains were reduced in high-dose animals (approximately 30-40%) and mid-dose (approximately 6-7%) animals compared with controls. Low-dose rats exhibited growth rates comparable to those of controls. Anemia and reticulocytosis occurred in mid- and high-dose males and in high-dose females after 12 months. The incidence of hyperplastic liver foci was increased in high-dose males (16/29) and mid-dose females (19/27). At 12 months, 6/7 high-dose males had marked atrophy of the testes with severe atrophy of the seminiferous tubules and almost complete lack of spermatogenesis. This lesion is common in geriatric rats, but is not normally seen in rats of this age. Beyond 12 months, severe atrophy of the seminiferous tubules occurred in 16% (4/25) of the controls, 26% (7/27) of the low-dose males, 33% (6/19) of the mid-dose males, and 81% (22/27) of the high-dose males. The authors did not report the statistical significance of these effects. However, only the highest dose effect is significant by Chi square ($p = 0.01$) and Fischer's Exact Test ($p = 0.004$). The LOAEL is 34 mg/kg/day based on the incidence of changes in the seminiferous tubules of male rats. The NOAEL is 3.9 mg/kg/day.

In a separate study (Lee et al., 1978), groups of CD rats (16/sex/dose) were fed diets containing 0, 0.07, 0.20, or 0.7% 2,4-DNT (98% pure) for up to 13 weeks. The corresponding daily intakes were 0, 34, 93, or 266 mg/kg/day for males, and 0, 38, 108, or 145 mg/kg/day for females. Four animals/sex/group were sacrificed at 4 and 13 weeks after being returned to normal diets for 1 month. All high-dose females died within 3 weeks. One male in the mid-dose group and 6 in the high-dose group died between weeks 4 and 13. All surviving animals exhibited dose-dependent decreases in body weight gain, which ranged from

approximately 9-55% when compared with controls. Food consumption was decreased in all dose groups. Orange to yellowish urine stains were observed on the fur of high-dose rats, and one male had widespread and stiff hind legs. Mid- and high-dose animals of both sexes were anemic, characterized by decreases in erythrocyte count, hematocrit, and hemoglobin, and concurrent reticulocytosis. Absolute liver and kidney weights were slightly increased in mid-dose males, and relative weights of these organs were significantly increased. There was splenic hemosiderosis in mid- and high-dose males and females. Spermatogenesis was decreased in mid-dose males and completely arrested in high-dose males. One high-dose male showed some signs of neuromuscular effects with demyelination in the cerebellum and brain stem. The LOAEL was 34 mg/kg/day based on decreased body weight gain and food consumption in male rats. There was no NOAEL because effects occurred at all doses tested.

Hong et al. (1985) reported the results of a chronic toxicity study commissioned by the U.S. Army (Ellis et al., 1979) of mice fed 98% pure 2,4-dinitrotoluene (2,4-DNT) in the diet for up to 24 months. Groups of 38 male and 38 female CD-1 mice were administered 2,4-DNT in their diets at average doses of 0, 14, 95, or 898 mg/kg/day. Both sexes of the high-dose animals and the males of the mid-dose groups had decreased weight gain that was approximately 10-22% lower than that of controls. High-dose males and females exhibited toxic anemia, reticulocytosis, and significant ($p < 0.05$) increases in spleen and liver weights. All treated mice had an increased dose-related pigment in many tissues and organs including the liver, spleen, lungs, and kidney. High-dose females demonstrated ovarian atrophy. Mid- and high-dose males exhibited testicular atrophy.

In a separate study (Lee et al., 1978), groups of 16 male and 16 female CD-1 mice were fed diets containing 0, 0.07, 0.20, or 0.7% 2,4-DNT (98% pure) for 13 weeks. The corresponding daily intakes were 0, 47, 137, or 413 mg/kg/day for males, and 0, 52, 147, or 468 mg/kg/day for females. Five mice died during the study. Compared with controls, treated males exhibited a dose-dependent decrease in body weight (3, 11, and 19% from low to high dose) and, in the high-dose group only, there was decreased food consumption. The high-dose group of both sexes were anemic (decreased erythrocyte count, hematocrit, and hemoglobin) with concurrent reticulocytosis, mild hepatocellular dysplasia, and Kupffer cell dysplasia. High- and mid-dose males had mild degeneration of the seminiferous tubules or testicular degeneration. After 4 weeks off treatment, mice recovered completely. The LOAEL was 47 mg/kg/day, based on body weight loss in males. There was no NOAEL because effects occurred at all doses tested.

Groups of 10 male Sprague-Dawley rats were administered 2,4-DNT (purity not reported) in corn oil by oral gavage at 0, 60, 180, or 240 mg/kg/day for 5 days (Lane et al., 1985). Significant reductions in the mating index and a sharp decrease in sperm-positive and pregnant females were observed in the 240-mg/kg/day dose group. Because of this finding, statistical evaluation of the reproductive results was difficult. No dominant lethal effects, characterized by early fetal deaths, were observed. Dose levels at or below 180 mg/kg/day did not result in changes in fertility or fetal death.

Bloch et al. (1988) fed groups of 9-10 Sprague-Dawley rats 2,4-DNT (97% pure) at dietary levels of 0, 0.1, or 0.2% (0, 1000, or 2000 ppm, respectively; or 0, 100, or 200 mg/kg/day, respectively). Effects observed in the highest dose group included significant body weight reduction ($p < 0.05$), significant increases in serum follicle stimulating hormone and luteinizing hormone ($p < 0.05$), significantly reduced sperm count ($p < 0.01$), disruption of spermatogenesis, and histological alterations or degeneration in Sertoli cells, spermatocytes, and spermatids. No significant effects were observed in the low-dose rats.

In a 3-generation study conducted by Ellis et al. (1979), groups of 10-24 Sprague-Dawley rats/sex were fed diets containing 0, 15, 100, or 700 ppm (approximately 0, 0.75, 5, or 35 mg/kg/day, respectively) 2,4-DNT (98% pure) for up to 6 months prior to mating. Each parental generation produced two sets of offspring (Fa and Fb litters). The study was terminated during the third generation after weaning of the second litter (Fb). The highest dose was associated with reduced parental body weight, reduced pup survival, reduced fertility in F1 animals, and slightly lower mean litter size and pup weight. At mid- and low-dose levels there were slight reductions in body weight for first and third generation pups; however, parental fertility and offspring viability were not affected. The LOAEL is 700 ppm, based on severe reductions in fertility. The NOAEL is 100 ppm.

Technical grade DNT (76% 2,4-DNT; 19% 2,6-DNT; 5% other isomers) wasadministered in corn oil by gavage to groups of 5-20 time-mated female Fischer 344 rats on gestation days 7-20 (Price et al., 1985). The doses were 0, 14, 35, 37.5, 75, 100, or 150 mg/kg/day. In the 150 mg/kg/day group there was 46% mortality and clinical signs of toxicity began on gestation day 11. Mortality for the other treatment groups was similar to that of the control group. Corrected body weight gain (minus gravid uterine weight) was significantly reduced in dams receiving 14, 100, or 150 mg/kg/day. Relative liver weight was increased significantly in the 75- and 100-mg/kg/day groups. Relative spleen weight was significantly increased at all doses except 14 mg/kg/day. There were no treatment-related effects on the number of corpora lutea, implantations, live and dead fetuses, litter size, sex ratio, fetal weight, crown rump length, placental weight, or incidences of malformations and variations. There was a statistically insignificant increase in the percent resorptions in the 150-mg/kg/day group, which was considered to be indicative of a compound-related effect. Developmental effects noted in the fetuses were reduced liver weight at 14 mg/kg/day, and increased spleen weight at 35 and 75 mg/kg/day.

Uncertainty and Modifying Factors

UF -- This uncertainty factor includes a factor of 10 for interspecies variability and a factor of 10 for intraspecies variability.

MF -- None

Additional Studies/Comments

Reported human health effects from DNT exposure are from occupational exposure studies in which workers were exposed primarily by inhalation with some contribution assumed from dermal absorption and ingestion (Etnier, 1987; Turner, 1986; Turner et al., 1985; Woollen et

al., 1985). Major effects from chronic exposure include methemoglobinemia, characterized by Heinz body formation and compensatory reticulocytosis; cyanosis; neurotoxicity; and possible excess mortality from ischemic heart disease and residual circulatory system effects. Neurotoxicity is characterized by vertigo, paresthesia, tremors, unconsciousness, and paralysis. Humans appear to metabolize DNT qualitatively similar to animals with rapid absorption and urinary excretion of metabolites.

Heinz body formation has been observed in humans, dogs, and rodents that were exposed to DNT. Heinz bodies are thought to consist of denatured hemoglobin, possibly sulfhemoglobin, that may form disulfide bonds with red blood cell membranes and thus lead to impaired ion transport resulting in hyperpermeability and hemolysis (Smith, 1986). Cat, mouse, dog, and human erythrocytes are thought to be particularly susceptible to Heinz body formation.

Monitoring and production data indicate that the occurrence of 2,6-DNT is usually found in the presence of 2,4-DNT with the latter more significant by volume. Subchronic (13 week) studies in dogs, rats, and mice indicate that 2,4- and 2,6-DNT systemic toxicity may be qualitatively and quantitatively similar. Oral dosing studies with technical grade DNT (tg-DNT; approximately 75% 2,4-DNT, 20% 2,6-DNT, and 5% other isomers) do not elucidate the relative contribution of the various isomers to toxic effects.

Dinitrotoluene isomers are metabolized initially by liver oxidation (Rickert et al., 1984). Some metabolites are conjugated with sulfate or glucuronate and subsequently excreted in the urine or bile. The bile metabolites are hydrolyzed and reduced further by intestinal microflora. The bacterial metabolites are reabsorbed from the gut into the systemic circulation, oxidized in the liver, and excreted either in the urine or the bile for additional reduction by intestinal bacteria. There are species qualitative and quantitative differences; however, typical urinary metabolites of orally administered 2,4-DNT[ring-14C] in female CD rats, CD-1 mice, New Zealand white rabbits, beagle dogs, and rhesus monkeys were the glucuronide conjugates of 2,4-dinitrobenzyl alcohol and 2-amino-4-nitrobenzyl alcohol. Smaller amounts of 2,4-diaminotoluene, 2,4-diaminobenzyl alcohol, 2-amino-4-nitrotoluene, 4-amino-2-nitrotoluene, and 2,4-dinitrobenzoic acid were also recovered from each species. Several studies demonstrated similar urinary metabolites in male rats and mice. Humans exposed occupationally (via inhalation and assumed dermal routes) to tg-DNT excreted some of the same urinary metabolites demonstrated in animals (e.g., the unchanged parent compound, 2,4-dinitrobenzyl alcohol, 2,4-dinitrobenzyl alcohol glucuronide, and 2,4-dinitrobenzoic acid) (Levine et al., 1985; Turner, 1986; Turner et al., 1985; Woolen et al., 1985). Other 2,4-DNT metabolites detected in the workers include 2-amino-4-nitrobenzoic acid, 4-amino-2-nitrobenzoic acid, 2-acetylamino-4-nitrobenzoic acid, and 4-acetylamino-2-nitrobenzoic acid.

Confidence in the Oral RfD
Study -- High
Data Base -- High
RfD -- High

The toxic effects observed in the 2-year dog study are based on an adequate number of animals of both sexes. In addition, a variety of gross, histological, hematologic, and clinical endpoints were evaluated. These effects are consistent with those reported to occur in exposed humans. The data base is rated high to medium because there are numerous acute, subchronic, chronic, and lifetime studies in several mammalian species. However, developmental toxicity studies with 2,4-DNT are lacking. Several rodent strains have been tested, and both sexes have been tested in all species. Pharmacokinetics and toxic effects demonstrated in laboratory animal species are consistent with observations from human exposure studies. The ratings for both the study and the data base result in a high to medium level of confidence in the RfD.

M.15.1.2 Reference Concentration for Chronic Inhalation Exposure

The health effects data for 2,4-dinitrotoluene 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. 1980. Ambient Water Quality Criteria for Dinitrotoluenes. 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 440/5-80-045. NTIS PB 81-117566/AS.

U.S. EPA. 1986. Health and Environmental Effects Profile for Dinitrotoluenes. 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. ECAO-CIN-P183. (Final Draft)

U.S. EPA. 1989. Ambient Water Quality Criteria Document Addendum for Dinitrotoluenes. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. ECAO-CIN-643. (Draft)

M.15.1.3 Noncarcinogenic Assessment References

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M.15.2 CARCINOGENIC ASSESSMENT

This substance/agent has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential.

M.17 ENDOSULFAN

Status of Data	*Last Revised*
Oral RfD Assessment	0/01/94
Inhalation RfC Assessment	No data
Carcinogenicity Assessment	07/01/94

M.17.1 NONCARCINOGENIC ASSESSMENT

M.17.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD
Reduced body weight gain in males and females; increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males	NOAEL: 15 ppm [0.6 mg/kg-day (male); 0.7 mg/kg-day (female)]	100	1	6E-3 mg/kg/day
2-Year Rat Feeding Study	LOAEL: 75 ppm [2.9 mg/kg-day (male); 3. mg/kg-day (female)]			
Hoechst Celanese Corp., 1989a				
Decreased weight gain in males and neurologic findings in both sexes	NOAEL: 10 ppm 0.57 mg/kg-day (female)			
1-Year Dog Feeding Study	LOAEL: 30 ppm [1.9 mg/kg-day (female); 2.1 mg/kg-day (male)]			
Hoechst Celanese Corp., 1989b				

*Conversion Factors and Assumptions -- Actual dose tested

Principal and Supporting Studies

Hoechst Celanese Corporation. 1989a. MRID No. 40256502, 41099502. HED Doc. No. 007937. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Hoechst Celanese Corporation. 1989b. MRID No. 41099501. HED Doc. No. 007937. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Groups of Sprague-Dawley rats (50/sex/dose) were administered endosulfan in the diet for 2 years at dietary concentrations of 0, 3, 7.5, 15 and 75 ppm (Male: 0, 0.1, 0.3, 0.6 and 2.9 mg/kg-day; Female: 0, 0.1, 0.4, 0.7 and 3.8 mg/kg-day) (Hoechst Celanese Corp., 1989a). A satellite group of 20 animals/sex/dose were used for toxicity evaluation and sampled at intervals for hematology and clinic chemistry; survivors were sacrificed after 104 weeks. An additional group of 10 rats/sex were used for pretest hematology and health check. Animals received food and water ad libitum.

No effects of dosing on clinical signs, mortality, food and water consumption, ophthalmological examinations and urinalysis were observed. Mean body weight gains tended to be decreased in both males and females receiving 15 and 75 ppm. Weight gains were significantly depressed ($p < 0.05$) during weeks 6-18 in males receiving 15 and 75 ppm when compared with controls; decreases did not achieve a level of significance in males or females receiving 15 ppm at other intervals. Between weeks 0-64, body weight gains were 9 and 13% lower in males and females of the 75 ppm group, respectively, than those of the controls. Overall weight gains (weeks 0-104) were 17% lower than the controls in both males and females receiving 75 ppm and 9% lower in rats receiving 15 ppm. The gains were significantly lower ($p < 0.01$) in both sexes at the 75 ppm dose. No weight gain effects were seen in males and females receiving 3 or 7.5 ppm when compared with controls.

No toxicologically important changes in hematology and clinical chemistry parameters were observed. The incidence of bilaterally enlarged kidneys was increased in females of both the satellite (2 and 8 for the control and 75 ppm dose groups, respectively) and main (8 and 18 for the control and 75 ppm dose groups, respectively) groups receiving 75 ppm when compared with controls. Other findings in the kidneys (paleness, irregular or uniform cortical scarring and cysts) occurred at similar frequencies in control and dosed groups. The incidence of progressive glomerulonephrosis was high in all dose groups including controls which is not an uncommon finding in studies with chlorinated hydrocarbon pesticides. The severity appeared to be dose-related. The incidence of severe (marked) glomerulonephrosis was increased in both males and females receiving 75 ppm. In males, the increased incidence at 75 ppm was accounted for by rats that died. In decendents, the incidence combining both males in the main and satellite groups was 10/41, 10/43, 17/46 and 14/46 and 20/46 at 0, 3, 7.5, 15 and 75 ppm. The incidence in high-dose males (30/70, 43%) was reported to be higher than normally observed for historical controls. The laboratory control incidence in six studies was 70/300 (19.7%) with a range of 10 to 38%.

The incidence of aneurysms of the blood vessels was increased in the high-dose males of both the satellite (6/20) and the main study (13/50) when compared with controls (1/20 and 9/50). The percent incidence (27%) in the combined high-dose males was higher than normally found in historical controls (10%, range in five studies 4-18%). Other nonneoplastic findings were considered within the normal range of background.

Based on reduced body weight gain in males and females, and increased incidence of marked progressive glomerulonephrosis and blood vessel aneurysms in males, the LEL for systemic toxicity is 75 ppm (Male: 2.9 mg/kg-day; Female: 3.8 mg/kg-day). The NOEL for systemic toxicity is 15 ppm (Male: 0.6 mg/kg-day; Female: 0.7 mg/kg-day).

Endosulfan was fed in the diet to beagle dogs (6/sex/dose) for 1 year at dietary levels of 0, 3, 10 and 30 ppm (Male: 0, 0.2, 0.65 and 2.1 mg/kg-day; Female: 0, 0.18, 0.57 and 1.9 mg/kg-day) (Hoechst Celanese Corp., 1989b). An additional group of 6 dogs/sex received 30 ppm for 54 days, after which the dose was increased to 45 ppm (Male: 3.2 mg/kg-day; Female: 2.9 mg/kg-day) and continued at that level until a final increase to 60 ppm (Male: 4.1 mg/kg-day; Female: 3.8 mg/kg-day) was administered at 106 days. Animals received food and water ad libitum.

At the highest dose tested, severe nervous symptoms developed. A loss or weakening of placing and righting reactions was observed and substantial weight loss resulted (0.36 and 0.45 kg for males and females, respectively, between weeks 15 and 21). This group was sacrificed at 146-147 days owing to poor overall condition; one male had been sacrificed at 126 days. The overall weight gain in males receiving 30 ppm (to week 54) was 30% lower than the controls. Tonic contractions of the muscles of the abdomen and chaps a few hours after feeding was noted in both sexes receiving 30 ppm; one male was sacrificed at 39 weeks. The sacrificed dog at 30 ppm had gross and histologic changes in the lungs. The sacrificed dog receiving 60 ppm had pulmonary edema. The histologic findings in dosed and control groups for dogs sacrificed by design were generally unremarkable and incidental.

Based on decreased weight gain in males and neurologic findings in both sexes, the LEL for systemic toxicity is 30 ppm (Male: 2.1 mg/kg-day; Female: 1.9 mg/kg-day). The NOEL for systemic toxicity is 10 ppm (Male: 0.65 mg/kg-day; Female: 0.57 mg/kg-day).

Uncertainty and Modifying Factors

UF -- The uncertainty factor of 100 reflects 10 for intraspecies variability and 10 for interspecies extrapolation.

MF -- None

Additional Studies/Comments

The observation of a yellowish discoloration of the kidneys was found in a 30-day rat feeding study (Hoechst Celanese Corp., 1985), a 90-day rat feeding study (Hoechst Aktiengesellschaft, 1985), and a 2-generation reproduction study in rats (Hoechst Aktiengesellschaft, 1984a). This observation was not found in the rat chronic feeding/oncogenicity study (Hoechst Celanese Corp., 1989a), the 1-year dog feeding study (Hoechst Celanese Corp., 1989b), or the mouse carcinogenicity study (Hoechst Celanese Corp., 1988). Hoechst Celanese Corporation argues that data from chronic toxicity studies, metabolism, and special studies indicate that this is not an adverse hematopoietic effect, but is indicative of the physical presence and harmless process of elimination of endosulfan and its metabolites via the kidney. Electron microscopy and tissue residue analysis of the kidneys from the 30-day study indicated that the

alpha-endosulfan and to a lesser extent beta-endosulfan, endosulfan sulfate, and endosulfan lactone were stored temporarily in the kidneys. Additionally, negative results were obtained from the staining of the kidneys with Prussian Blue to detect the presence of ferritin (evidence of hemosiderosis).

Data Considered for Establishing the RfD

1) 2-Year Feeding - rat: Principal study -- see previous description; Core grade minimum (Hoechst Celanese Corp., 1989a).

2) 1-Year Feeding - dog: Co-principal study -- see previous description; Core grade minimum (Hoechst Celanese Corp., 1989b).

3) 2-Generation Reproduction - rat: Core grade minimum (Hoechst Aktiengesellschaft, 1984a).

Four week old male and female rats of Crl:COBS CD(SD)BR strain were allowed to acclimate for 7 days and were then distributed randomly to groups of 32 of each sex for the F0 generation. F1b animals were distributed randomly to groups of 28 of each sex for the second generation. Animals were administered endosulfan in the diet at dose levels of 0, 3, 15 or 75 ppm (Male: 0, 0.2, 1.1 and 5.4 mg/kg-day; Female: 0, 0.25, 2.6 and 6.6 mg/kg-day).

Mortality, food/water consumption, and body weight gain were not affected in either generation, but a decrease in body weight gain ($p < 0.05$) was observed in the F0 females following the start of dosing. Pregnancy rate, gestation times, the ability to rear young to weaning, and precoital time were comparable among the groups at both matings in both generations. F0 males displayed increased heart weight at the mid- and high-dose levels (dose-related) and increased liver and kidney weights at the high-dose level. F0 females displayed increased brain and liver weights at the high-dose level. In the F1b adults, the high-dose males displayed increased kidney weights compared with the controls and females displayed increased liver weights at the mid- and high-dose levels. Although the changes in the heart and liver weights reported in the mid-dose F0 males and females were statistically significant, the U.S. EPA noted that these effects were slight and limited to one sex of one litter and occurred only in one generation. In view of this and in the absence of histopathological changes in the liver and heart, the U.S. EPA concluded that these statistically significant changes in organ weights should not be considered biologically significant. Therefore, based on a decrease in body weight gain in F0 females, the LEL for systemic toxicity is 75 ppm (Male: 5.4 mg/kg-day; Female: 6.6 mg/kg-day). The NOEL for systemic toxicity is 15 ppm (Male: 1.1 mg/kg-day; Female: 2.6 mg/kg-day).

No effect of treatment on litter size was observed throughout both matings of both generations. In the first mating of the F0 generation, an increase was noted in the cumulative litter loss (%) at the high-dose level. Litter and pup weights were comparable at birth among the groups in both generations, but a decrease in litter weight was observed during the lactation to weaning period in both matings in the F0 generation, which was significant at the high-dose level in the

first mating and at the mid- and high-dose levels in the second mating (dose-related). Because there was no corroborative finding of a decrease in the number of pups per litter or in pup weight, this decrease in litter weight is not considered to be treatment-related. Increased pituitary weights (high-dose female pups of 1st mate of F0 generation) and increased uterine weights (high-dose female pup of 1st mate of F1b generation) were observed in the offspring. There were no histopathological findings observed in either the F1b adults of the selected pups from the second mate or the F1b generation that could be attributed to treatment. Based on increased pituitary and uterine weights, the LEL for offspring toxicity is 75 ppm (Male: 5.4 mg/kg-day; Female: 6.6 mg/kg-day). The NOEL for offspring toxicity is 15 ppm (Male: 1.1 mg/kg-day; Female: 2.6 mg/kg-day).

No evidence of reproductive toxicity was found at any of the dose levels tested. Therefore, the NOEL for reproductive toxicity is equal to or greater than 75 ppm (Male: 5.4 mg/kg-day; Female: 6.6 mg/kg-day).

4) Developmental toxicity - rat: Core grade supplementary (FMC Corp., 1980).

Groups of pregnant CD Sprague-Dawley rats were administered endosulfan by daily oral gavage on days 6 through 19 of gestation at dose levels of 0, 0.66, 2.0 or 6.0 mg/kg-day. Although the original protocol specified 25 animals per treatment group, 10 additional animals were added to the high-dose group (due to mortality among the original animals) and five additional animals were added to the control group (due to a loss of some tissues during the processing).

Maternal toxicity was apparent in the high-dose group in the form of significantly reduced body weights and body weight gain during gestation ($p < 0.01$). Toxic signs observed in the high-dose group included face rubbing (20/35 animals), brown exudate (4/35), rough coat (5/35), flaccidity (8/35) and hyperactivity (11/35). Face rubbing was reported in 6/25 mid-dose animals and alopecia was reported in 2/25. No face rubbing was reported in low dose animals or controls. Mean fetal weight and crown-rump length were significantly reduced ($p < 0.05$ and $p < 0.01$, respectively) in the 6 mg/kg-day group. An increase in misaligned sternebrae was observed in all treated groups compared with concurrent controls. The increase in litters and fetuses affected at each dose level was above that reported in the historical control data base (18% of litters and 1.85% of fetuses based upon the examination of 65 litters and 863 fetuses). However, the variability between studies was not reported. An increased incidence of litters with extra ribs and poorly ossified and unossified sternebrae was observed at the high-dose level. More detailed historical control data may be useful in determining whether the apparent increase in misaligned sternebrae is due to an unusually low incidence in the concurrent control group and within the variability observed between studies. In the absence of such information, it is recommended that this finding be considered to be compound-related. An additional review of this study by the U.S EPA concluded that replacement of animals during or after the study made it difficult to interpret the data and derive a NOEL and LEL for this study. The U.S. EPA has recommended a repeat of this study.

5) Developmental toxicity - rabbit: Core grade minimum (FMC Corp., 1981).

Groups of 20 pregnant New Zealand white rabbits were administered endosulfan by oral gavage on days 6 through 28 of gestation at dose levels of 0, 0.3, 0.7 or 1.8 mg/kg-day. When mortality was observed at the highest dose level, six more mated rabbits were added to this group.

Two animals in the control and one in the middle dose level showed nasal congestion. In the highest dose level, four animals showed a noisy and rapid breathing, hyperactivity and convulsions. Body weight gains during the days 19-29 and corrected for gravid uterine weights at sacrifice were less in the high-dose group than in controls. The former was also less than the control for the mid-dose group. However, these differences were not statistically significant. Based on these effects, the NOEL and LEL for maternal toxicity are 0.7 and 1.8 mg/kg-day, respectively. No developmental effects were observed at any dose tested. Therefore, the NOEL for developmental toxicity is equal to or greater than 1.8 mg/kg-day, the highest dose tested.

Other Data Reviewed:

6) 2-Year Feeding - mouse: Core grade minimum (Hoechst Celanese Corp., 1988).

Groups of Hoe:NMRKf mice (60/sex/dose) were fed endosulfan in the diet at dose levels of 0, 2, 6, or 18 ppm (Male: 0, 0.28, 0.84, and 2.51 mg/kg-day; Female: 0, 0.32, 0.97, and 2.86 mg/kg-day) for 2 years. A satellite group of 20 mice/sex/group was used for interim sacrifices at 12 and 18 months. Animals were individually housed and received food and water ad libitum.

No overt signs of toxicity or dose-related effects were noted on clinical observations, food consumption, hematology, clinical chemistry, urinalysis, organ weights, macroscopic pathology, or microscopic pathology. Decreased survival ($p < 0.05$) in high-dose females and body weight reduction ($p < 0.05$) in high-dose males throughout the study were considered to be compound-related effects. Based on these findings, the NOEL and LEL for systemic toxicity are 6 ppm (Male: 0.84 mg/kg-day; Female: 0.97 mg/kg-day) and 18 ppm (Male: 2.51 mg/kg-day; Female: 2.86 mg/kg-day), respectively.

7) 90-Day Feeding - rat: Core grade minimum (Hoechst Aktiengesellschaft, 1985).

Groups of CD Sprague-Dawley rats (25/sex/dose) were fed endosulfan in the diet at dose levels of 0, 10, 30, 60 or 360 ppm (0, 0.5, 1.5, 3 and 18 mg/kg-day) for 13 weeks. Twenty of each group were sacrificed at 13 weeks and five were sacrificed after an additional 4-week recovery period.

No significant mortality during the test period was reported. Body weight was marginally lowered in males and females at 360 ppm. Depressed RBC parameters were observed in the 60 and 360 ppm groups (6 and 13 weeks, both sexes) and in males at 30 ppm (6 weeks).

Several other statistically significant decreases from control were observed but these were not dose-related. In general, the magnitude of the differences observed at each time point is small (<10%). Kidney weight (relative) was increased in both sexes at the high-dose level (360 ppm) and in males at 60 ppm. Two types of histopathological findings in the kidney were noted at 13 weeks: (1) occasional cells o proximal tubules showing yellowish discoloration of the cytoplasm (all dose levels), and (2) darker and more particulate granular and/or clumped pigmentation, predominantly in cells of the straight portions and, to a lesser extent in the proximal convoluted tubules (both sexes at 360 ppm/males at 60 ppm). From the data following the 4-week recovery period, the discoloration and/or pigmentation was not persistent after withdrawal of treatment, which Hoechst Aktiengesellschaft states is indicative of the ongoing process of excretion of test material via the kidneys. Hoechst Aktiengesellschaft concludes that, although this process can be observed (due to the coloration of the kidney tissues and the observation of "dark urine"), it is not indicative of an adverse effect on the kidney, as confirmed by the histopathological findings.

8) 13-Week Feeding - mouse: Core grade minimum (Hoechst Aktiengesellschaft, 1984b).

Groups of CD-1 mice (20/sex/group) were fed endosulfan in the diet at dose levels of 0, 2, 6, 18 or 54 ppm (Male: 0, 0.24, 0.74, 2.13 and 7.3 mg/kg-day; Female: 0, 0.27, 0.8, 2.39 and 7.52 mg/kg-day) for 13 weeks. Ten animals of each sex were sacrificed after an approximately 20 day observation period prior to the test period and examined microscopically.

Increased mortality (12/20 males and 10/20 females) was observed at 54 ppm. Glucose levels in females were significantly ($p<0.01$) lowered at 6, 18 and 54 ppm. Hemoglobin levels were significantly ($p<0.05$) elevated at 2, 6 and 18 ppm in females and appeared elevated at 54 ppm; however, this value was not analyzed due to the few survivors. Mean corpuscular hemoglobin concentration was significantly ($p<0.05$) lowered at 2, 6, and 18 ppm in females and appeared lowered at 54 ppm, however this value was not analyzed for the reason cited above. Based on the effects observed in females at the lowest dose tested, the LEL for systemic toxicity was 2 ppm (0.27 mg/kg-day). A NOEL for systemic toxicity was not established.

9) 30-Day Feeding - rat: Core grade supplementary (Hoechst Celanese Corp., 1985).

Groups of male Wistar rats (10 animals for control, 50 animals for each test dose) were fed endosulfan in the diet at dose levels of 0, 360 and 720 ppm (0, 34, and 67.8 mg/kg-day) for 30 days.

No overt signs of toxicity or dose-related effects were observed on body weight, food or water consumption, clinical observations, or ophthalmology. Two dosed animals died during the study with no discernible signs of toxicity. Absolute and relative liver weights of males receiving doses of 360 and 760 ppm and kidney weights of males receiving 720 ppm were increased ($p<0.05$) following the dosing period; organ weights of dosed males were similar to controls following a 30-day recovery period. Macroscopic examination revealed discoloration

of the kidneys following the dosing period; histopathologically, the number and size of the lysosomes of the proximal convoluted tubules of the kidneys were increased following the dosing period with this finding exhibited to a greater extent in high-dose males. The renal changes were found to be reversible following the recovery period without evidence of renal lesions. No evidence of comparable lysosmal activity in the brain or liver was reported. Electron microscopy and tissue analysis confirmed that alpha-endosulfan and to a lesser extent beta-endosulfan, endosulfan sulfate and endosulfan-lactone were stored temporarily in the kidneys during the dosing period; only negligible amounts of endosulfan metabolites were found in the liver. Based on kidney changes during the dosing period, the LEL for systemic toxicity was 360 ppm (34 mg/kg-day), the lowest dose tested. A NOEL for systemic toxicity was not established.

Data Gap(s): Rat Developmental Toxicity Study

Confidence in the Oral RfD
Study -- Medium
Data Base -- Medium
RfD -- Medium

The principal studies are of adequate quality and therefore the given a medium confidence rating. Additional studies are supportive of the principal studies. However, due to the replacement of test animals during the available developmental toxicity study (FMC Corp., 1980), the data are considered inadequate to address the requirement for testing in a second species. Due to the lack of these developmental data in a second species, the data base is given a medium-to-high confidence rating. Medium-to-high confidence in the RfD follows.

M.17.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.17.1.3 Noncarcinogenic Assessment References

FMC Corporation. 1980. MRID No. 00055544. HED Doc. No. 000416. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

FMC Corporation. 1981. MRID No. 00094837. HED Doc. No. 001488. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Hoechst Aktiengesellschaft. 1984a. MRID No. 00148264. HED Doc. No. 004881, 008868, 009552. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Hoechst Aktiengesellschaft. 1984b. MRID No. 00147182. HED Doc. No. 004733. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Hoechst Aktiengesellschaft. 1985. MRID No. 00145668. HED Doc. No. 005115, 008868. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Hoechst Celanese Corporation. 1985. MRID No. 00147299, 40767601. HED Doc. No. 007163. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Hoechst Celanese Corporation. 1988. MRID No. 00162996, 40256501, 40792401. HED Doc. No. 007155. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Hoechst Celanese Corporation. 1989a. MRID No. 40256502, 41099502. HED Doc. No. 007937. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Hoechst Celanese Corporation. 1989b. MRID No. 41099501. HED Doc. No. 007937. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

M.17.2 CARCINOGENIC ASSESSMENT

This substance/agent has been evaluated by the U.S. EPA for evidence of human carcinogenic potential. This does not imply that this agent is necessarily a carcinogen. The evaluation for this chemical is under review by an inter-office Agency work group. A risk assessment summary will be included on IRIS when the review has been completed.

M.18 ENDRIN

Status of Data	*Last Revised*
Oral RfD Assessment	04/01/91
Inhalation RfC Assessment	No data
Carcinogenicity Assessment	07/01/93

M.18.1 NONCARCINOGENIC ASSESSMENT

M.18.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD
Mild histological lesions in liver, occasional convulsions mg/kg/day	NOEL: 1 ppm in diet (0.025 mg/kg/day)	100	1	3E-4
Dog Chronic Oral Bioassay	LOAEL: 2 ppm in diet (0.05 mg/kg/day)			

Velsicol Chemical Corporation, 1969

*Conversion Factors: 1 ppm = 0.025 mg/kg/day (assumed dog food consumption)

Prinicipal and Supporting Studies

Velsicol Chemical Corporation. 1969. MRID. No. 00030198. Available from EPA. Write FOI, EPA, Washington, DC. 20460.

Groups of 3 to 7 dogs/sex were fed diets containing 0.1, 0.5, 1.0, 2.0 or 4.0 ppm endrin for 2 years. Dogs receiving 2 or 4 ppm experienced occasional convulsions, slightly increased relative liver weights, and mild histopathological effects in the liver (slight vacuolization of hepatic cells). No adverse effects on these parameters or on growth, food consumption, behavior, serum chemistry, urine chemistry or histological appearance of major organs occurred at 1 ppm (NOEL) or less. The 2 ppm level is the LOAEL. The authors provided data concerning actual endrin consumptions as weekly averages, but no overall averages were calculated. Visual inspection of these data indicated that application of the standard food factor of 2.5% bw/day would closely approximate actual consumption. Therefore, the 1 ppm NOEL was equivalent to an endrin intake of 0.025 mg/kg/day.

An earlier study (Treon et al., 1955) established a dietary NOEL of 1 ppm for both dogs and rats for long-term feeding (18 months - 2 years). LOAELs of 3 ppm and 5 ppm were reported for dogs and rats, respectively. The primary target organs were the kidney and the liver. Dogs are judged to be more sensitive than rats to long-term exposure to endrin because of the lower food consumption of dogs (than rats) and because of the much shorter duration of exposure (in this study) relative to lifetime for dogs as compared to rats.

Uncertainty and Modifying Factors

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

Additional Comments

Acute lethality data suggest that rabbits and monkeys are much more sensitive to endrin than rats (Treon et al., 1955). Long-term studies have not been conducted with species other than rats or dogs. Conflicting evidence exists as to the developmental toxicity of endrin. Developmental effects have been observed to occur at dose levels much greater than those associated with chronic toxicity; these studies are discussed in U.S. EPA (1987).

Confidence in the Oral RfD

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

The principal study was of average quality and is given medium confidence. The data base is assigned medium confidence because, although the chronic data is supportive, information on reproductive effects is lacking. Medium confidence in the RfD follows.

M.18.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.18.1.3 Noncarcinogenic Assessment References

Treon, J.F., F.P. Cleveland and J. Cappel. 1955. Toxicity of endrin for laboratory animals. Agric. Food Chem. 3(10): 842-848.

U.S. EPA. 1987. Health Effects Assessment for Endrin. Final Draft. Environmental Criteria and Assessment Office, Cincinnati, OH. ECAO-CIN-H089.

U.S. EPA. 1985. Drinking Water Criteria Document for Endrin. Final Draft. Environmental Criteria and Assessment Office, Cincinnati, OH. NTIS PB86-117967.

Velsicol Chemical Corporation. 1969. MRID. No. 00030198. Available from EPA. Write FOI, EPA, Washington, DC. 20460.

M.18.2 CARCINOGENIC ASSESSMENT

Weight-of-Evidence Classifcation
Classification -- D; not classifiable as to carcinogenicity for humans

Basis -- Oral administration of endrin did not produce carcinogenic effects in either sex of two strains of rats and three strains of mice. An NCI bioassay was suggestive of responses in male and female rats although NCI reported a no evidence conclusion. The inadequacies of several of the bioassays call into question the strength of the reported negative findings. These inadequacies and the suggestive responses in the NCI bioassay do not support a Group E classification; rather a Group D classification best reflects the equivocal data.

Human Carcinogenicity Data
Inadequate. Ditraglia et al. (1981) conducted a retrospective cohort study to examine the mortality of workers employed in the manufacture of organochlorine pesticides including endrin. No statistically significant excesses or deficits in mortality for any specific cancer site were noted. Limited follow-up time (12 years), lack of exposure data, and few deaths give this study low power.

Animal Carcinogenicity Data
Inadequate. The potential carcinogenic effects of endrin have been evaluated following oral exposure to 1-100 ppm endrin in the diet of Carworth Farm rats, (Treon et al., 1955), Osborne-Mendel rats (Deichmann et al, 1970; NCI, 1979), C57Bl/6J mice (Witherup et al., 1970), C3D2F1/J mice (Witherup et al., 1970) and B6C3F1 mice (NCI, 1979). There was no evidence of carcinogenicity in any of these studies. Treon et al., (1955) also failed to note any increase in tumorigenesis in dogs exposed up to 18.7 months at the maximum tolerated dose. The length of this study was insufficient to provide for the expected latency period in dogs.

The NCI (1979) bioassay was done in Osborne-Mendel rats (50/sex/group) and B6C3F1 mice (50/sex/group); matched control groups included 10 animals/sex/ species. Since the number of animals in the matched-control groups was small, pooled-control groups from concurrent pesticide bioassays were used for statistical evaluation.

Endrin was administered daily in the diet for 80 weeks. Rats were observed for an additional 31 to 34 weeks and mice were observed for an additional 11 weeks. The initial doses for male rats and all mice were 2.5 or 5 ppm and for female rats were 5 or 10 ppm. Because of subsequent toxic effects, the doses for the female rats and male mice were reduced during the course of the studies. High-dose male mice were fed treatment and control diets on alternate weeks for 10 weeks. The resulting time-weighted average dose fed in the diets of treated animals was reported as follows: 2.5 or 5 ppm for male rats, 3 or 6 ppm for female rats, 1.6 or 3.2 ppm for male mice, and 2.5 or 5 ppm for female mice.

When compared with pooled controls, a statistically significant increase in hemangioma was observed in low-dose male rats (0/49, 5/46, 3/47), and a significant increase in adrenal adenoma or carcinoma was seen in high-dose male rats (2/44, 4/46, 8/44). Islet-cell

carcinoma incidence in male rats showed a significant positive trend but the pairwise comparisons were not significant. A statistically significant increase in pituitary adenoma was observed in the high-dose female rats (4/44, 11/47, 13/45) and a significant increase in adrenal adenoma or carcinoma was observed in the low-dose female rats (4/46, 14/49, 7/47).

Although NCI concluded from the bioassays that endrin was not carcinogenic, the responses noted above cannot be totally ignored. A primary reviewer for NCI noted that the negative findings could be a reflection of the high toxicity of endrin, which only permitted the administration of relatively low chronic doses. Furthermore, the reviewer observed that an accidental overdose among low-dose male mice resulted in the early death of several animals in this treatment group. The study was marred by a small number (10) of matched controls; however, this deficiency was compensated by the use of pooled controls.

Reuber (1978) reported positive carcinogenic effects of endrin had been observed in a FDA bioassay (Bierbower, 1965). Male and female Osborne-Mendel rats were exposed to 0.1 to 25 ppm endrin in the diet. At the 0.1 ppm dose, incidence of hyperplastic nodules and malignant tumors of the liver was significantly increased in female rats and in male and female rats combined. A variety of other tumors were observed including mammary gland, uterine, and thyroid tumors in females and thyroid and adrenal cortex tumors in males.

Reuber (1979) independently reevaluated several endrin carcinogenicity studies including the NCI (1979) study and the FDA (Bierbower, 1965) study and determined that a significant increase in tumor incidence was present. It is difficult to draw conclusions from Reuber's findings, however, since his criteria for classifying lesions as tumorigenic appear to differ from those of other investigators. Reuber did not provide slide by slide tabulation of his findings nor did he distinguish between primary and/or metastatic tumors in the liver (Albert, 1977).

Supporting Data for Carcinogenicity

Maslansky and Williams (1981) showed that endrin (10-3 and 10-4 M) was not genotoxic in the hepatocyte primary culture (HPC)/DNA repair assay using hepatocytes from male Fischer F344 rats, male CD-1 mice, and mal Syrian hamsters. DNA repair was observed in response to a positive control in all three systems. Endrin was not mutagenic in microbial systems with or without metabolic activation (Moriya et al., 1983; Probst et al., 1981; Glatt et al., 1983), and endrin exposure did not significantly affect sister-chromatid exchange frequencies in a human lymphoid cell line (Sobti et al., 1983). Endrin is also structurally related to aldrin, dieldrin, chlordane, chlorendic acid, and heptachlor which are known to be carcinogenic in animals.

M.18.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Not available.

M.18.2.2 Quantiative Estimate of Carcinogenic Risk from Inhalation Exposure

Not available.

M.18.2.3 Carcinogenic Assessment References

Albert, R. 1977. Memorandum to Kyle Barbehenn, Endrin Project Manager, Office of Special Pesticide Review. Carcinogen Assessment Group, Washington, DC. June 20.

Bierbower, G.W. 1965. Final report on pathological study of rats fed endrin or dieldrin. Prepared as a memo to A.J. Lehman. Food and Drug Administration. (Cited in: Reuber, 1978)

Deichmann, W.B., W.E. MacDonald, E. Blum, et al. 1970. Tumorigenicity of aldrin, dieldrin and endrin in the albino rat. Ind. Med. 39: 426-434.

Ditraglia, D., D.P. Brown, T. Namekata and N. Iverson. 1981. Motality study of workers employed at organochlorine pesticide manufacturing plants. Scand. J. Work Environ. Health. 7: 140-146.

Glatt, H., R. Jung and F. Oesch. 1983. Bacterial mutagenicity investigation of epoxides: Drugs, drug metabolites, steroids and pesticides. Mutat. Res. 11: 99-118.

Maslansky, C.J. and G.M. Williams. 1981. Evidence for an epigenetic mode of action in organochlorine pesticide hepatocarcinogenicity: A lack of genotoxicity in rat, mouse, and hamster hepatocytes. J. Toxicol. Environ. Health. 8: 121-130.

Moriya, M., T. Ohta, K. Watanabe, T. Miyazawa, K. Kato and Y. Shirasu. 1983. Further mutagenicity studies on pesticides in bacterial reversion assay systems. Mutat. Res. 116: 185-216.

NCI (National Cancer Institute). 1979. Bioassay of endrin for possible carcinogenicity. Carcinogenesis Technical Report Series 12, NCR-CG-TR-12. Publ. No. (NIH) 79-812.

Probst, G.S., K.E. McMahon, L.E. Hill, et al. 1981. Chemically-induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 compounds. Environ. Mutagen. 3: 11-32.

Reuber, M.D. 1978. Carcinomas, sarcomas and other lesions in Osborne-Mendel rats ingesting endrin. Exp. Cell. Biol. 46: 129-145.

Reuber, M.D. 1979. Carcinogenicity of endrin. Sci. Total Environ. 12:101-135.

Sobti, R.C., A. Krishan and J. Davies. 1983. Cytokinetic and cytogenetic effect of agricultural chemicals on human lymphoid cells in vitro. II. Organochlorine pesticides. Arch. Toxicol. 52: 221-231.

Treon, J.F., F.P. Cleveland and J. Cappel. 1955. Toxicity of endrin for laboratory animals. J. Agric. Food Chem. 3: 842-848.

U.S. EPA. 1987a. Drinking Water Criteria Document for Endrin. 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)

U.S. EPA. 1987b. Health Effects Assessment for Endrin. 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. (Final Draft)

Witherup, S., K.L. Stemmer, P. Taylor and P. Bietsch. 1970. The incidence of neoplasms in two strains of mice sustained on diets containing endrin. Kettering Lab., Univ. Cincinnati, Cincinnati, OH.

M.19 HEPTACHLOR

Status of Data	*Last Revised*
Oral RfD Assessment	03/01/91
Inhalation RfC Assessment	No data
Carcinogenicity Assessment	07/01/93

M.19.1 NONCARCINOGENIC ASSESSMENT

M.19.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD
Liver weight increases increases in males	NOEL: 3 ppm diet (0.15 mg/kg/day)	300	1	5E-4 mg/kg/day
2-Year Rat Feeding Study	LEL: 5 ppm diet (0.25 mg/kg/day)			

Velsicol Chemical, 1955a

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

Principal and Supporting Studies

Velsicol Chemical Corporation. 1955a. MRID No. 00062599. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Six groups of CF strain white rats containing 20/sex were fed for 2 years with diets of 0, 1.5, 3, 5, 7, or 10 ppm of heptachlor. Lesions in the liver were limited to 7 ppm and above and were characteristic of chlorinated hydrocarbons (that is, hepatocellular swelling and peripheral arrangements of the cytoplasmic granules of cells of the central zone of the liver lobules). The NOEL for the lesions was 5 ppm and the LEL was 7 ppm. The NOEL for increased liver-to-body weight for males only was 3 ppm and the LEL was 5 ppm.

Uncertainty and Modifying Factors

UF -- Based on a chronic exposure study, an uncertainty factor of 100 was used to account for inter- and intraspecies differences. An additional factor of 3 was considered appropriate because of the lack of chronic toxicity data in a second species, for a total uncertainty factor of 300. The serious deficiencies in the toxicologic data base would normally warrant a 10-fold factor for this area of uncertainty. However, toxicity data for other cyclodiene insecticides (aldrin, dieldrin, chlordane, and heptachlor epoxide) suggest that dogs and rats do not differ greatly in sensitivity to the effects of this class of compounds. Furthermore, liver toxicity has been fairly well established as the most sensitive endpoint for this class of compounds, which reduces the uncertainty attributable to the lack of information on other toxic effects.

MF -- None

Additional Comments
Data Considered for Establishing the RfD:

1) 2-Year Feeding - rat: Principal study - see previous description; no core grade

2) 8-Month Feeding - rat: NOEL=none; LEL=5 ppm (0.25 mg/kg/day) (LDT) (swelling of cells); no core grade (Velsicol Chemical, 1964)

3) 1-Generation Reproduction - rat: NOEL=5 ppm (0.25 mg/kg/day); LEL=7 ppm (0.35 mg/kg/day) (increased pup death); no core grade (Velsicol Chemical, 1955b)

4) 3-Generation Reproduction - rat: NOEL=10 ppm (0.5 mg/kg/day) (HDT) (no adverse effects); no core grade (Velsicol Chemical, 1967)

Data Gap(s): Chronic Dog Feeding Study; Rat Teratology Study; Rabbit Teratology Study

Confidence in the Oral RfD
Study -- Low
Data Base -- Low
RfD -- Low

The principal study is of low quality and is given a low confidence rating. Since ths data base on chronic toxicity is incomplete, the data base is given a low confidence rating. Low confidence in the RfD follows.

M.18.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.18.1.3 Noncarcinogenic Assessment References

Velsicol Chemical Corporation. 1955a. MRID No. 00062599. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Velsicol Chemical Corporation. 1955b. MRID No. 00062599. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Velsicol Chemical Corporation. 1964. MRID No. 00086210. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Velsicol Chemical Corporation. 1967. MRID No. 00147058. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

M.18.2 CARCINOGENIC ASSESSMENT

Weight-of-Evidence Classification
Classification -- B2; probable human carcinogen

Basis -- Inadequate human data, but sufficient evidence exist from studies in which benign and malignant liver tumors were induced in three strains of mice of both sexes. Several structurally related compounds are liver carcinogens.

Human Carcinogenicity Data
Inadequate. There were 11 case reports involving central nervous system effects, blood dyscrasias, and neuroblastomas in children with pre- or postnatal exposure to chlordane and heptachlor (Infante et al., 1978). Since no other information was available, no conclusions can be drawn.

There were three epidemiologic studies of workers exposed to chlordane and/or heptachlor. One retrospective cohort study of pesticide applicators was considered inadequate in sample size and duration of follow-up. This study showed marginal statistically significant increased mortality from bladder cancer (3 observed) (Wang and McMahon, 1979a). The other two studies were retrospective cohort studies of pesticide manufacturing workers. Neither of them showed any statistically significant increased cancer mortality (Wang and McMahon, 1979b; Ditraglia et al., 1981). Both these populations also had confounding exposures from other chemicals.

Animal Carcinogenicity Data
Sufficient. Long-term carcinogenicity bioassays with heptachlor have been performed in rats and mice, with the latter showing a carcinogenic response. Davis (1965) fed groups of 100 male and 100 female C3H mice diets with 0 or 10 ppm heptachlor (purity not specified) for 2 years. Survival was low, with 50% of the controls and 30% of the treated mice surviving until the end of the experiment. A 2-fold increase in benign liver lesions over the controls was reported. After a histologic reevaluation, Reuber (as cited in Epstein, 1976), as well as four other pathologists, remarked a statistically significant increase in liver carcinomas in the treated male (64/87) and female (57/78) groups by comparison to controls (22/73 and 2/53 for males and females, respectively).

The NCI (1977) reported a significant dose-related increase of hepatocellular carcinomas in male and female B6C3F1 mice. Fifty male and 50 female mice were fed diets delivering technical-grade heptachlor at TWA concentrations of 6.1 and 13.8 ppm and 9 and 18 ppm, respectively. Treatment was for 80 weeks, followed by 10 weeks of observation. The authors also reported a statistically significant increase of hepatocellular carcinomas in high-dose males and females over the controls.

No indication of treatment-related increase of tumors has been reported in chronic studies with rats. In an early experiment, Witherup et al. (1955) fed 20 male and 20 female CFN rats each at 1.5, 3.5, 7.0, and 10.0 ppm in the diet for 110 weeks. Although no increase in tumors was

found, liver lesions, described as the "chlorinated hydrocarbon" type, were observed at 7 and 10 ppm. Using 25 female CD rats, Jolley et al. (1966) also observed no malignant lesions of the liver but did find hepatocytomegaly when the rats were fed 7.5, 10, and 12.5 ppm heptachlor:heptachlor epoxide (mixture of 75:25). Over the 2 years of the experiment, a dose-related increase in mortality was observed. Two additional experiments, Cabral et al. (1972) and NCI (1977), found no increased incidence of hepatocellular carcinomas when the mixture was administered to Wistar rats by gavage or to Osborne-Mendel rats by diet.

Supporting Data for Carcinogencity
Gene mutation assays indicate that heptachlor is not mutagenic in bacteria (Probst et al., 1981; Shirasu et al., 1976; Moriya et al., 1983) or mammalian liver cells (Telang et al., 1982). Negative results were reported in two dominant lethal assays using male germinal cells (Epstein et al., 1972; Arnold et al., 1977). DNA repair assays indicate that heptachlor is not genotoxic in rodent hepatocytes (Maslansky and Williams, 1981; Probst et al., 1981) but showed qualitative evidence of unscheduled DNA synthesis in human fibroblasts (Ahmed et al., 1977).

Five compounds structurally related to heptachlor (heptachlor epoxide, chlordane, aldrin, dieldrin, and chlorendic acid) have produced liver tumors in mice. Chlorendic acid has also produced liver tumors in rats.

M.18.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Summary of Risk Estimates
Oral Slope Factor -- 4.5E+0 per (mg/kg)/day
Drinking Water Unit Risk -- 1.3E-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)	8E-1 ug/L
E-5 (1 in 100,000)	8E-2 ug/L
E-6 (1 in 1,000,000)	8E-3 ug/L

Dose-Response Data
Tumor Type -- hepatocellular carcinomas
Test Animals -- mouse/C3H; mouse/B6C3F1
Route -- diet
Reference -- Davis, 1965; NCI, 1977

Additional Comments
Four data sets showed a significant increase in hepatocellular carcinomas in treatment groups compared with controls in mice. The quantitative estimate is the geometric mean of the slope factors from the four mouse data sets. The slope factors for each set are: 12.4 per

(mg/kg)/day for C3H male mice, 14.9 per (mg/kg)/day for C3H female mice, 2.79 per (mg/kg)/day for B6C3F1 male mice, and 0.83 per (mg/kg)/day for B6C3F1 female mice. Although the magnitude of the responses differed somewhat, a combined risk estimate was chosen because the two strains are related and so that relevant data will not be discarded.

The above unit risk should not be used if the water concentration exceeds 80 ug/L, since above this concentration the unit risk may not be appropriate.

Discusssion of Confidence
Adequate numbers of animals were treated and observed for the majority of their expected lifetime. The incidences of malignant lesions were significantly increased in all four data sets, and dose-response effects were observed in the NCI (1977) study.

M.18.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Summary of Risk Estimates
Inhalation Unit Risk -- 1.3E-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)	8E-2 ug/cu.m
E-5 (1 in 100,000)	8E-3 ug/cu.m
E-6 (1 in 1,000,000)	8E-4 ug/cu.m

Dose-Response Data for Carcinogenicity
The risk estimates were calculated from the oral data presented in II.B.2.

Additional Comments
The above unit risk should not be used if the air concentration exceeds 8 ug/cu.m, since above this concentration the unit risk may not be appropriate.

Discussion of Confidence
See oral discussion.

M.18.2.3 Carcinogenic Assessment References

Davis, K. 1965. Pathology Report on Mice Fed Aldrin, Dieldrin, Heptachlor and Heptachlor Epoxide for Two Years. Internal FDA memorandum to Dr. A.J. Lehman, July 19.

Epstein, S.S. 1976. Carcinogenicity of heptachlor and chlordane. Sci. Total Environ. 6: 103-154.

NCI (National Cancer Institute). 1977. Bioassay of Heptachlor for Possible Carcinogenicity. NCI Carcinogenesis Tech. Rep. Ser. No. 9. (Also published as DHEW Publication No. [NIH] 77-809).

Reuber, M.D. 1977. Histopathology of Carcinomas of the Liver in Mice Ingesting Heptachlor or Heptachlor Epoxide. Exp. Cell Biol. 45: 147-157.

U.S. EPA. 1986. Carcinogenicity Assessment of Chlordane and Heptachlor/Heptachlor Epoxide. Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC. OHEA-C-204.

M.20 HEPTACHLOR EPOXIDE

Status of Data	*Last Revised*
Oral RfD Assessment	03/01/91
Inhalation RfC Assessment	No data
Carcinogenicity Assessment	07/01/93

M.20.1 NONCARCINOGENIC ASSESSMENT

M.20.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD
Increased liver-to-body weight ratio in both males and females	NOEL: none	1000	1	1.3E-5 mg/kg/day

60-Week Dog Feeding Study — LEL: 0.5 ppm (diet) (0.0125 mg/kg/day)

Dow Chemical Co., 1958

*Conversion Factors: 1 ppm = 0.025 mg/kg/day (assumed dog food consumption)

Principal and Supporting Studies

Dow Chemical Company. 1958. MRID No. 00061912. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Beagle dogs from 23 to 27 weeks of age were divided into five groups (3 females and 2 males) and given diets containing 0, 0.5, 2.5, 5 or 7.5 ppm of heptachlor epoxide for 60 weeks. Liver-to-body weight ratios were significantly increased in a treatment-related fashion. Effects were noted for both males and females at the LEL of 0.5 ppm. A NOEL was not established.

Uncertainty and Modifying Factors

UF -- Based on a chronic exposure study, an uncertainty factor of 1000 was used to account for inter- and intraspecies differences and to account for the fact that a NOEL was not attained.

MF -- None

Additional Comments

None.

Confidence in the Oral RfD
Study -- Low
Data Base -- Medium
RfD -- Low

The principal study is of low quality and is given a low confidence rating. Since the data base on chronic toxicity is complete but consists of low-quality studies, the data base is given a medium to low confidence rating. Low confidence in the RfD follows.

M.20.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.20.1.3 Noncarcinogenic Assessment References

Dow Chemical Company. 1958. MRID No. 00061912. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Dow Chemical Company. 1959a. MRID No. 00062676. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Dow Chemical Company. 1959b. MRID No. 00061911. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Dow Chemical Company. 1966. MRID No. 00086208. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Dow Chemical Company. 1967. MRID No. 00147057. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Dow Chemical Company. 1973a. MRID No. 00050058. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

Dow Chemical Company. 1973b. MRID No. 000523262, 00062678, 00064943. Available from EPA. Write to FOI, EPA, Washington, DC 20460.

M.20.2 CARCINOGENIC ASSESSMENT

Weight-of-Evidence Classification
Classification -- B2; probable human carcinogen

Basis -- Sufficient evidence exists from rodent studies in which liver carcinomas were induced in two strains of mice of both sexes and in CFN female rats. Several structurally related compounds are liver carcinogens.

Human Carcinogenicity Data

Inadequate. There are no published epidemiologic evaluations of heptachlor epoxide. It is not commercially available in the United States,but is a product of heptachlor oxidation.

There were 11 case reports involving central nervous system effects, blood dyscrasias and neuroblastomas in children with pre-/postnatal exposure to chlordane and heptachlor (Infante et al., 1978). Since no other information was available, no conclusions can be drawn.

There were three epidemiologic studies of workers exposed to chlordane and/or heptachlor. One retrospective cohort study of pesticide applicators was considered inadequate in sample size and duration of follow-up. This study showed marginal statistically significant increased mortality from bladder cancer (3 observed) (Wang and McMahon, 1979a). Two other retrospective cohort studies were of pesticide manufacturing workers. Neither of them showed any statistically significant increased cancer mortality (Wang and McMahon, 1979b; Ditraglia et al., 1981). Both these populations also had confounding exposures from other chemicals.

Animal Carcinogenicity Data

Sufficient. Four long-term carcinogenesis bioassays of heptachlor epoxide have been reported. The major finding in mice has been an increased incidence of liver carcinomas. Davis (1965) fed groups of 100 male and 100 female C3H mice 0 or 10 ppm heptachlor epoxide for 2 years. Survival was generally low, with 50% of controls and 9.5% of treated mice living 2 years. A 2-fold increase in benign liver lesions (hepatic hyperplasia and benign tumors) over the controls was reported. Reevaluation by Reuber (1977b) revealed a significant increase in liver carcinomas in the dosed group (77/81 in females and 73/79 in males) over the controls (2/53 in females and 22/73 in males). The Velsicol Chemical Co. (1973) tested a 75:25 mixture of heptachlor epoxide:heptachlor in groups of 100 male and 100 female CD-1 mice. The mice were fed 0, 1, 5, and 10 ppm for 18 months. A statistically significant increase of hyperplasia was observed in the 5, and 10 ppm dose groups in both sexes; Reuber's reevaluation (U.S. EPA, 1985) resulted in a change in diagnosis for benign to liver carcinomas, thereby increasing the incidence of hepatic carcinomas ($p < 0.01$). Four independent pathologists concurred with Reuber's reevaluation.

The earliest bioassay with rats (Witherup et al., 1959) tested 25 male and 25 female CFN rats each at 0.5, 2.5, 5.0, 7.5, and 10 ppm for 108 weeks. The authors observed malignant and benign tumors randomly among test groups and controls. Reuber's reevaluation (1985) reported a significant increase of hepatic carcinomas above the controls at 5 and 10 ppm in the female rats. A reevaluation by Williams (1985) reported a significant increase of hepatic nodules at the 10 ppm level in the males over the controls. The Kettering Laboratory (Jolley et al., 1966) tested a mixture of 75:25 heptachlor:heptachlor epoxide in the diet of 25 female CD rats at 5, 7.5, 10, and 12.5 ppm for 2 years. Although no malignant lesions of the liver were observed, hepatocytomegaly was increased at 7.5, 10, and 12.5 ppm.

Supporting Data for Carcinogenicity

Gene mutation assays indicate that heptachlor epoxide is not mutagenic in bacteria (Moriya et al., 1983). In two mouse dominant lethal assays, heptachlor epoxide did not induce major chromosomal aberrations in male germinal cells (Arnold et al., 1977; Epstein et al., 1972). Ahmed et al. (1977) reported qualitative evidence of uuncheduled DNA synthesis response in SV40 transformed human fibroblasts in the presence of hepatic homogenates and heptachlor epoxide.

Five compounds structurally related to heptachlor epoxide (chlordane, aldrin, dieldrin, heptachlor and chlorendic acid) have produced liver tumors in mice. Chlorendic acid has also produced liver tumors in rats.

M.20.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Summary of Risk Estimates

Oral Slope Factor -- 9.1E+0 per (mg/kg)/day
Drinking Water Unit Risk -- 2.6E-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)	4E-1 ug/L
E-5 (1 in 100,000)	4E-2 ug/L
E-6 (1 in 1,000,000)	4E-3 ug/L

Dose-Response Data

Tumor Type -- hepatocellular carcinomas
Test Animals -- mouse/C3H (Davis); mouse/CD1 (Velsicol)
Route -- diet
Reference -- Davis, 1965; Velsicol, 1973

Additional Comments

The Davis (1965) study was designed to be for lifetime exposure. Thus, although survival was low, no correction for duration of experiment was made. Five data sets (four in mice and one in rats) show an increased incidence of hepatocellular carcinomas in treated groups compared with controls. There are four slope factors, 27.7 per (mg/kg)/day for C3H male mice, 36.2 per (mg/kg)/day for C3H female mice, 1.04 per (mg/kg)/day for CD-1 female mice, and 6.48 per (mg/kg)/day for CD-1 male mice. Since mice were the more sensitive species tested and to avoid discarding relevant data, the quantitative estimate is based on the geometric mean of 9.1 per (mg/kg)/day. This geometric mean is consistent with the potency estimate from rats of 5.8 per (mg/kg)/day (CFN females).

The above unit risk should not be used if the water concentration exceeds 40 ug/L, since above this concentration the unit risk may not be appropriate.

Discussion of Confidence
Adequate numbers of animals were treated in both studies, but survival in the Davis (1985) study was low. A dose-related increase in tumor incidence was observed in CD-1 mice. Slope factors were consistent in two species of rodents.

M.20.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Summary of Risk Estimates
Inhalation Unit Risk -- 2.6E-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)	4E-2 ug/cu.m
E-5 (1 in 100,000)	4E-3 ug/cu.m
E-6 (1 in 1,000,000)	4E-4 ug/cu.m

Dose-Response Data for Carcinogenicity
The inhalation risk estimates were calculated from the oral data.

Additional Comments
The above unit risk should not be used if the air concentration exceeds 4 ug/cu.m, since above this concentration the unit risk may not be appropriate.

Discussion of Confidence
See oral discussion.

M.20.2.3 Carcinogenic Assessment References

Davis, K.J. 1965. Pathology Report on Mice Fed Aldrin, Dieldrin, Heptachlor and Heptachlor Epoxide for Two Years. Internal FDA memorandum to Dr. A.J. Lehman, July 19.

Epstein, S.S. 1976. Carcinogenicity of heptachlor and chlordane. Sci. Total Environ. 6: 103-154.

Reuber, M.D. 1977. Histopathology of carcinomas of the liver in mice ingesting heptachlor or heptachlor epoxide. Exp. Cell Biol. 45: 147-157.

U.S. EPA. 1985. Hearing Files on Chlordane, Heptachlor Suspension (unpublished draft). Available for inspection at: U.S. EPA, Washington, DC.

U.S. EPA. 1986. Carcinogenicity Assessment of Chlordane and Heptachlor/Heptachlor Epoxide. Prepared by the Office of Health and Environmental Assessment, Carcinogen Assessment Group, Washington, DC. OHEA-C-204.

Velsicol Chemical Corporation. 1973. MRID No. 00062678. Available from EPA. Write to FOI, EPA, Washington, D.C. 20460.

M.21 HMX

Status of Data	*Last Revised*
Oral RfD Assessment	02/01/93
Inhalation RfC Assessment	No data
Carcinogenicity Assessment	02/01/93

M.21.1 NONCARCINOGENIC ASSESSMENT

M.21.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

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				

Principal and Supporting Studies

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 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.

Uncertainty and Modifying Factors

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

Additional Comments

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).

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.

M.21.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.21.1.3 Noncarcinogenic Assessment References

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.

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.

M.21.2 CARCINOGENIC ASSESSMENT

Weight-of-Evidence Classification
Classification -- D; not classifiable as to human carcinogenicity

Basis -- No cancer bioassays or epidemiological studies are available.

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.

Animal Carcinogenicity Data
There are no lifetime (chronic) bioassays that evaluate carcinogenicity.

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.

M.21.2.2 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Not available.

M.21.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Not available.

M.21.2.3 Carcinogenic 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.

M.23 LEAD

Status of Data	*Last Revised*
Oral RfD Assessment	02/01/91
Inhalation RfC Assessment	no data
Carcinogenicity Assessment	11/01/91

M.23.1 NONCARCINOGENIC ASSESSMENT

M.23.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

A great deal of information on the health effects of lead has been obtained through decades of medical observation and scientific research. This information has been assessed in the development of air and water quality criteria by the Agency's Office of Health and Environmental Assessment (OHEA) in support of regulatory decision-making by the Office of Air Quality Planning and Standards (OAQPS) and by the Office of Drinking Water (ODW). By comparison to most other environmental toxicants, the degree of uncertainty about the health effects of lead is quite low. It appears that some of these effects, particularly changes in the levels of certain blood enzymes and in aspects of children's neurobehavioral development, may occur at blood lead levels so low as to be essentially without a threshold. The Agency's RfD Work Group discussed inorganic lead (and lead compounds) at two meetings (07/08/85 and 07/22/85) and considered it inappropriate to develop an RfD for inorganic lead. For additional information, interested parties are referred to the 1986 Air Quality Criteria for Lead (EPA-600/8-83/028a-dF) and its 1990 Supplement (EPA/600/8-89/049F) or the following Agency scientists:

Harlal Choudhury / NCEA -- (513)569-7536
J. Michael Davis / NCEA -- (919)541-4162
Jeff Cohen / OST -- (202)260-5456
John Haines / OAQPS -- (919)541-5533

M.23.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.23.2 CARCINOGENIC ASSESSMENT

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.

Human Carcinogenicity Data

Inadequate. There are four epidemiologic studies of occupational cohorts exposed to lead and lead compounds. Two studies (Dingwall-Fordyce and Lane, 1963; Nelson et al., 1982) did not find any association between exposure and cancer mortality. Selevan et al. (1985), in their retrospective cohort mortality study of primary lead smelter workers, found a slight decrease in the total cancer mortality (SMR=95). Apparent excesses were observed for respiratory cancer (SMR=111, obs=41, $p>0.05$) and kidney cancer (SMR=204, obs=6, $p>0.05$). Cooper and Gaffey (1975) and Cooper (1985 update) performed a cohort mortality study of battery plant workers and lead smelter workers. They found statistically significant excesses for total cancer mortality (SMR=113, obs=344), stomach cancer (SMR=168, obs=34), and lung cancer (SMR=124, obs=109) in the battery plant workers. Although similar excesses were observed in the smelter workers, they were not statistically significant. Cooper and Gaffey (1975) felt it was possible that individual subjects were monitored primarily on the basis of obvious signs of lead exposure, while others who showed no symptoms of lead poisoning were not monitored.

All of the available studies lacked quantitative exposure information, as well as information on the possible contribution from smoking. All studies also included exposures to other metals such as arsenic, cadmium, and zinc for which no adjustment was done. The cancer excesses observed in the lung and stomach were relatively small (<200). There was no consistency of site among the various studies, and no study showed any dose-response relationship. Thus, the available human evidence is considered to be inadequate to refute or demonstrate any potential carcinogenicity for humans from lead exposure.

Animal Carcinogenicity Data

Sufficient. The carcinogenic potential of lead salts (primarily phosphates and acetates) administered via the oral route or by injection has been demonstrated in rats and mice by more than 10 investigators. The most characteristic cancer response is bilateral renal carcinoma. Rats given lead acetate or subacetate orally have developed gliomas, and lead subacetate also produced lung adenomas in mice after i.p. adminstration. Most of these investigations found a carcinogenic response only at the highest dose. The lead compounds tested in animals are almost all soluble salts. Metallic lead, lead oxide and lead tetralkyls have not been tested adequately. Studies of inhalation exposure have not been located in the literature.

Azar et al. (1973) administerd 10, 50, 100, and 500 ppm lead as lead acetate in dietary concentrations to 50 rats/sex/group for 2 years. Control rats (100/sex) received the basal laboratory diet. In a second 2-year feeding study, 20 rats/group were given diets containing 0, 1000, and 2000 ppm lead as lead acetate. No renal tumors were reported in the control groups or in treated animals of either sex receiving 10 to 100 ppm. Male rats fed 500, 1000, and 2000 ppm lead acetate had an increased renal tumor incidence of 5/50, 10/20, and 16/20, while 7/20 females in the 2000-ppm group developed renal tumors.

The Azar et al. (1973) study is limited by the lack of experimental detail. The possibility of environmental contamination from lead in the air or drinking water was not mentioned. The strains of rats used were not specified in the study, but the Health Effects Assessment for Lead (U.S. EPA, 1984) indicates the rats were Wistar strain. The weight gain at 1000 and 2000 ppm was reported to be depressed, but details were not given.

Kasprzak et al. (1985), in investigating the interaction of dietary calcium on lead carcinogenicity, fed 1% lead subacetate (8500 ppm Pb) to male Sprague-Dawley rats in the diet for 79 weeks. Of the rats surviving (29/30) in this treatment group beyond 58 weeks, 44.8% had renal tumors. Four rats had adenocarcinomas; the remainaing nine had adenomas. Bilateral tumors were noted. No renal tumors were noted among the controls.

As part of a study to determine interactions between sodium nitrite, ethyl urea and lead, male Sprague-Dawley rats were given lead acetate in their drinking water for 76 weeks (Koller et al., 1986). The concentration of lead was 2600 ppm. No kidney tumors were detected among the 10 control rats. Thirteen of 16 (81%) lead-treated rats had renal tubular carcinoma; three tumors were detected at 72 weeks and the remainder detected at the termination of the study.

Van Esch and Kroes (1969) fed basic lead acetate at 0, 0.1%, and 1.0% in the diet to 25 Swiss mice/sex/group for 2 years. No renal tumors developed in the control group, but 6/25 male mice of 0.1% basic lead acetate group had renal tumors (adenomas and carcinomas combined). In the 1.0% group, one female had a renal tumor. The authors thought that the low incidence in the 1.0% group was due to early mortality.

Hamsters given lead subacetate at 0.5% and 1% in the diet had no significant renal tumor response (Van Esch and Kroes, 1969).

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).

M.23.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

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.

M.23.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Not available.

M.23.2.3 Carcinogenic 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.

Casto, B.C., J. Meyers and J.A. DiPaolo. 1979. Enhancement of viral transformation for evaluation of the carcinogenic or mutagenic potential of inorganic metal salts. Cancer Res. 39: 193-198.

Cooper, W.C. 1985. Mortality among employees of lead battery plants and lead producing plants, 1947-1980. Scand. J. Work Environ. Health. 11: 331-345.

Cooper, W.C. and W.R. Gaffey. 1975. Mortality of lead workers. In: Proceedings of the 1974 Conference on Standards of Occupational Lead Exposure, J.F. Cole, Ed., February, 1974. Washington, DC. J. Occup. Med. 17: 100-107.

Dingwall-Fordyce, I. and R.E. Lane. 1963. A follow-up study of lead workers. Br. J. Ind. Med. 20: 313-315.

DiPaolo, J.A., R.L. Nelson and B.C. Casto. 1978. In vitro neoplastic transformation of Syrian hamster cells by lead acetate and its relevance to environmental carcinogenesis. Br. J. Cancer. 38: 452-455.

Grandjean, P., H.C. Wulf and E. Niebuhr. 1983. Sister chromatid exchange in response to variations in occupational lead exposure. Environ. Res. 32:199-204.

Kasprzak, K.S., K.L. Hoover and L.A. Poirier. 1985. Effects of dietary calcium acetate on lead subacetate carcinogenicity in kidneys of male Sprague-Dawley rats. Carcinogenesis. 6(2): 279-282.

Koller, L.D., N.I. Kerkvliet and J.H. Exon. 1986. Neoplasia induced in male rats fed lead acetate, ethyl urea and sodium nitrate. Toxicol. Pathol. 13: 50-57.

Nelson, D.J., L. Kiremidjian-Schumacher and G. Stotzky. 1982. Effects of cadmium, lead, and zinc on macrophage-mediated cytotoxicity toward tumor cells. Environ. Res. 28: 154-163.

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M.24 MANGANESE

Status of Data	*Last Revised*
Oral RfD Assessment	11/01/95
Inhalation RfC Assessment	12/01/93
Carcinogenicity Assessment	03/01/94

M.24.1 NONCARCINOGENIC ASSESSMENT

M.24.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD
CNS effects mg/kg/day	NOAEL (food): 0.14 mg/kg-day	1	1	1.4E-1
Human Chronic Ingestion Data	LOAEL: None			
NRC, 1989; Freeland-Graves et al., 1987; WHO, 1973;				

*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.

Prinicipal and Supporting Studies

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.

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.

Uncertainty and Modifying Factors

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.

Additional Studies/Comments

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 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 this consumption of "large doses of vitamins and minerals for 4 to 5 years." Unfortunately, no quantitative data were reported.

Another case study of manganese intoxication involved a 62-year-old male who had been receiving total parenteral nutrition that provided 2.2 mg of manganese (form not stated) daily for 23 months (Ejima et al., 1992). The patient's whole blood manganese was found to be elevated, and he was diagnosed as having parkinsonism, with dysarthria, mild rigidity, hypokinesia with masked face, a halting gait and severely impaired postural reflexes. To be able to compare the manganese load in this individual with that corresponding to an oral intake, the difference between the direct intravenous exposure and the relatively low level of absorption of manganese from the 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 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 ormula, 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).

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 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.

M.24.1.2 Reference Concentration for Chronic Inhalation Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD
Impairment of neuro-behavioral function	NOAEL: None LOAEL: 0.15 mg/cu.m	1000	1	5E-5 mg/cu.m

Occupational exposure to manganese dioxide
LOAEL(ADJ): 0.05 mg/cu.m
LOAEL(HEC): 0.05 mg/cu.m

Roels et al., 1992

Critical Effect	Experimental Doses*	UF	MF	RfD
Impairment of neuro-behavioral function	NOAEL: None LOAEL: 0.97 mg/cu.m			

Occupational exposure to manganese oxides and salts
LOAEL(ADJ): 0.34 mg/cu.m
LOAEL(HEC): 0.34 mg/cu.m

Roels et al., 1987

*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.

Principal and Supporting Studies

Roels, H., R. Lauwerys, J.-P. Buchet et al. 1987. Epidemiological survey among workers exposed to manganese: Effects on lung, central nervous system, and some biological indices. Am. J. Ind. Med. 11: 307-327.

Roels, H.A., P. Ghyselen, J.P. Buchet, E. Ceulemans, and R.R. Lauwerys. 1992. Assessment of the permissible exposure level to manganese in workers exposed to manganese dioxide dust. Br. J. Ind. Med. 49: 25-34.

Roels et al. (1992) conducted a cross-sectional study of 92 male workers exposed to manganese dioxide (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. Alhough it is not clear that such was the case, the possibility of confounding suggests that the LOAEL should not be based on the results of the exposure-response analysis until these results can be confirmed by other studies. Also, statistical correction for multiple comparisons should be included in the exposure response analysis.

A LOAEL may be derived from the Roels et al. (1992) study by using the IRD concentration of MnO2, expressed as mg Mn/cu.m x years (based on 8-hour TWA occupational exposures for various job classifications, multiplied by individual work histories in years). Dividing the geometric mean IRD concentration (0.793 mg/cu.m x years) by the average duration of the workers' exposure to MnO2 (5.3 years) yields a LOAEL of 0.15 mg/cu.m. The LOAEL(HEC) is 0.05 mg/cu.m.

Roels et al. (1987) conducted a cross-sectional study in 141 male workers exposed to MnO2, manganese tetroxide (Mn3O4), and various Mn salts (sulfate, carbonate, and nitrate). A matched group of 104 male workers was selected as a control group. The two groups were matched for socioeconomic status and background environmental factors; in addition, both groups had comparable work-load and work-shift characteristics.

The TWA of total airborne Mn dust ranged from 0.07 to 8.61 mg/cu.m, with an overall arithmetic mean of 1.33 mg/cu.m, a median of 0.97 mg/cu.m, and a geometric mean of 0.94 mg/cu.m. The duration of employment ranged from 1 to 19 years, with a mean of 7.1 years. The particle size and purity of the dust were not reported. Neurological examination, neurobehavioral function tests (simple reaction time, short-term memory, eye-hand coordination, and hand tremor), spirographic measurements, blood and urine tests, and a self-administered questionnaire were used to assess possible toxic effects of Mn exposure. The questionnaire was designed to detect CNS and respiratory symptoms.

Significant differences in mean scores between Mn-exposed and reference subjects were found for objective measures of visual reaction time, eye-hand coordination, hand steadiness, and audio-verbal short-term memory. The prevalence of abnormal scores on eye-hand coordination and hand steadiness tests showed a dose-response relationship with blood Mn levels; short-term memory scores were related to years of Mn exposure but not to blood Mn levels. The prevalence of subjective symptoms was greater in the exposed group than in controls for 20 of 25 items on the questionnaire, with four items being statistically significant: fatigue, tinnitus, trembling of fingers, and irritability.

A significantly greater prevalence of coughs during the cold season, dyspnea during exercise, and recent episodes of acute bronchitis was self-reported in the exposed group. Lung function parameters were only slightly (<10%) lower in the Mn-exposed workers, with the only significant alterations evident in Mn-exposed smokers. These mild changes in Mn-exposed workers (apart from the effects of smoking) and the absence of respiratory effects in the more recent study by Roels et al. (1992) suggest that the nervous system is a more sensitive target for Mn toxicity.

Based upon the findings of impaired neurobehavioral function in workers whose average Mn exposure was estimated by the geometric mean TWA of total airborne Mn dust at the time of the study, a LOAEL of 0.97 mg/cu.m was identified, with a LOAEL(HEC) of 0.34 mg/cu.m. Note that this LOAEL(HEC) is based on total Mn dust of mixed forms, whereas the LOAEL(HEC) from the more recent Roels et al. (1992) study is based on the measured respirable dust fraction of MnO2 only. However, the geometric mean total dust concentrations n the 1987 and 1992 studies by Roels et al. were approximately the same (0.94 and 0.95 mg/cu.m, respectively).

The findings of Roels et al. (1987, 1992) are supported by other recent reports that provide comparable and consistent indications of neurobehavioral dysfunction in Mn-exposed workers (Mergler et al., 1993; Iregren, 1990; Wennberg et al., 1991, 1992).

Mergler et al. (1993) conducted a cross-sectional study of 115 male ferromanganese and silicomanganese alloy workers in southwest Quebec. A matched-pair design was employed because of presumptively high environmental pollutant levels; 74 pairs of workers and referents were matched on age, educational level, smoking status, number of children, and length of residency in the region.

Air concentrations of respirable and total dust were sampled by stationary monitors during silicomanganese production. The geometric mean of a series of 8-hour TWAs was 0.035 mg Mn/cu.m (range: 0.001-1.273 mg Mn/cu.m) for respirable dust and 0.225 mg Mn/cu.m (range: 0.014-11.480 mg Mn/cu.m) for total dust. The authors noted that past dust levels at certain job sites had been considerably higher. The mean duration of the workers' Mn exposure was 16.7 years and included Mn fumes as well as mixed oxides and salts of Mn. Geometric mean MnB was significantly higher in the Mn alloy workers, but MnU did not differ significantly between exposed workers and controls.

The number of discordant pairs, in which workers reported undesirable symptoms on a self-administered questionnaire but their matched pairs did not, was statistically significant for 33 of 46 items, including the following: fatigue; emotional state; memory, attention, and concentration difficulties; nightmares; sweating in the absence of physical exertion; sexual dysfunction; lower back pain; joint pain; and tinnitus. Workers did not report symptoms typical of advanced Mn poisoning (e.g., hand tremor, changes in handwriting, loss of balance when turning, difficulty in reaching a fixed point) significantly more than referents, which suggests that the other reported symptoms were probably not due to bias on the part of the workers.

The greatest differences in neurobehavioral function were evident in tests of motor function, especially tests requiring coordinated alternating and/or rapid movements. Workers performed significantly worse on the motor scale of a neuropsychological test battery both in overall score and in eight subscales of rapid sequential or alternating movements. Worker performance also was significantly worse on tests of hand steadiness, parallel-line drawing performance, and ability to rapidly identify and mark specified alphabetic characters within strings of letters. Performance on a variety of other tests of psychomotor function, including simple reaction time, was worse in Mn-exposed workers but marginally significant ($0.05 < p < 0.10$). In addition, Mn alloy workers differed significantly from referents on measures of cognitive flexibility and emotional state. Olfactory perception also was significantly enhanced in the Mn-alloy workers.

The matched-pair design of Mergler et al. (1993) helped reduce differences between exposed and referent subjects that might otherwise have confounded the study. However, to the extent that the referents also may have had significant exposure to Mn in the ambient atmosphere, such exposure may have reduced the differences in neurobehavioral performance between workers and referents. This possibility is supported by the fact that the finger-tapping speed of both workers and referents on a computerized test was slower than that of Mn-exposed workers assessed on the same test by Iregren (1990) in Sweden. In the absence of a NOAEL, the LOAEL from the study of Mergler et al. (1993) is based on the geometric mean respirable dust level (0.035 mg Mn/cu.m), with a LOAEL(HEC) of approximately 0.01 mg/cu.m, which is about five-fold lower than the LOAEL(HEC) identified in the study by Roels et al. (1992).

Workers exposed to Mn in two Swedish foundries (15 from each plant) were evaluated in a study first reported by Iregren (1990). The exposure to Mn varied from 0.02 to 1.40 mg/cu.m (mean = 0.25 mg/cu.m; median = 0.14 mg/cu.m) for 1-35 years (mean = 9.9 years). Earlier monitoring measurements made in both factories suggested that essentially no changes in exposure had occurred in either factory for the preceding 18 years. Each exposed worker was matched for age, geographical area, and type of work to two workers not exposed to Mn in other industries. Neurobehavioral function was assessed by eight computerized tests and two manual dexterity tests. There were significant differences between exposed and control groups for simple reaction time, the standard deviation of reaction time, and finger-tapping speed of the dominant hand. In addition, digit-span short-term memory, speed of mental addition, and verbal (vocabulary) understanding differed significantly between exposed and control groups. The difference in verbal understanding suggested that the two groups were not well matched for general cognitive abilities. With verbal performance used as an additional matching criterion, differences between the groups in simple reaction time, the standard deviation of reaction time, and finger-tapping speed remained statistically significant, despite a decrease in statistical power due to reducing the size of the reference group to 30 workers. Further analyses using verbal test scores as a covariate also indicated that these same three measures of neurobehavioral function were statistically different in exposed and control workers. No significant correlation was found within the exposed group to establish a concentration-response relationship.

Additional reports of neurobehavioral and electrophysiological evaluations of these same workers have been published by Wennberg et al. (1991, 1992). Although none of the latter results achieved statistical significance at $p = 0.05$, increased frequency of self-reported health symptoms, increased prevalence of abnormal electroencephalograms, slower brain-stem auditory-evoked potential latencies, and slower diadochokinesometric performance were found in the exposed workers. Diadochokinesis refers to the ability to perform rapidly alternating movements such as supination and pronation of the forearm, and is an indicator of extrapyramidal function (see Additonal Comments/Studies). Also, an increase in P-300 latency, as suggested by these results, has been observed in patients with parkinsonism according to Wennberg et al. (1991), who viewed these results in Mn-exposed workers as early (preclinical) signs of disturbances similar to parkinsonism. Based on the impairments in reaction time and finger-tapping speed reported in the Swedish studies (Iregren, 1990; Wennberg et al., 1991, 1992), the LOAEL(HEC) is calculated to be 0.05 mg/cu.m. Although numerically the same value as that derived from Roels et al. (1992), the Swedish study measured total dust.

However, Wennberg et al. (1991) stated that the respirable dust level was 20-80% of total dust, which implies that the LOAEL(HEC) from the Swedish studies could be somewhat lower than that from Roels et al. (1992).

All of the above studies taken together provide a consistent pattern of evidence indicating that neurotoxicity is associated with low-level occupational Mn exposure. The fact that the speed and coordination of motor function are especially impaired is consistent with other epidemiological, clinical, and experimental animal evidence of Mn intoxication (see Additional Comments/Studies). Moreover, the LOAEL(HEC)s obtained from these studies are not appreciably different. Nevertheless, some differences between the studies are evident in the durations of exposure and forms of Mn to which workers were exposed. In the Roels et al. (1992) study, the mean period of exposure was 5.3 years (range: 0.2-17.7 years), and exposure was limited to MnO2. In the other studies, mixed forms of Mn were present, and the mean durations of exposure were longer: 7.1 years in Roels et al. (1987), 9.9 years in Iregren (1990), and 16.7 years in Mergler et al. (1993). The findings of Mergler et al. (1993) suggest that the LOAEL(HEC) could be at least as low as approximately 0.01 mg/cu.m. However, the variable concentrations and mixed compounds of Mn to which workers were exposed make it difficult to rely primarily upon the findings of Mergler et al. (1993) in deriving the RfC.

Nevertheless, their results provide support for the findings of Roels et al. (1992) and suggest that the longer period of exposure (16.7 years in Mergler et al. (1993) vs. 5.3 years in Roels et al., 1992) may have contributed to the apparent differences in sensitivity. Although analyses by Roels et al. (1987, 1992) and Iregren (1990) generally did not indicate that duration of exposure correlated significantly with neurobehavioral outcomes, none of these studies involved average exposures as long as those in the Mergler et al. (1993) study. Also, the oldest worker in the Roels et al. (1992) study was less than 50 years old, and the average age in that study was only 31.3 years vs. 34.3 years in Roels et al. (1987), 43.4 years in Mergler et al. (1993), and 46.4 in Iregren (1990). These points suggest that chronic exposure to Mn

and/or interactions with aging could result in effects at lower concentrations than would be detected after shorter periods of exposure and/or in younger workers.

Based on the findings of neurobehavioral impairment by Roels et al. (1987, 1992), with supporting evidence from Mergler et al. (1993) and the Swedish reports (Iregren, 1990; Wennberg et al., 1991, 1992), the LOAEL for derivation of the RfC is 0.15 mg/cu.m, and the LOAEL(HEC) is 0.05 mg/cu.m.

Uncertainty and Modifying Factors

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.

MF -- None

Additional Studies/Comments

Manganese toxicity varies depending upon the route of exposure. When ingested, Mn is considered to be among the least toxic of the trace elements. In the normal adult, between 3 and 10% of dietary Mn is absorbed. Total body stores normally are controlled by a complex homeostatic mechanism regulating absorption and excretion. As detailed in the Uncertainty Factor Text and the Additonal Comments/Studies for the oral RfD, toxicity from ingested Mn is rarely observed. However, deficiencies of calcium and iron have been noted to increase Mn absorption (Mena et al., 1969; Murphy et al., 1991). Also, Mena et al. (1969) found that anemic subjects absorbed 7.5% of ingested Mn, whereas normal subjects absorbed 3%. Interestingly, manganism patients absorbed 4%, and apparently healthy Mn miners absorbed only 3%. These differences suggest that certain subpopulations, such as children, pregnant women, elderly persons, iron- or calcium-deficient individuals, and individuals with liver impairment, may have an increased potential for excessive Mn body burdens due to increased absorption or altered clearance mechanisms, which may be of particular importance for those exposed to Mn by multiple routes.

As a route of Mn exposure, the respiratory tract is the most important portal of entry. The inhalation toxicity of Mn is in part a function of particle dosimetry and subsequent pharmacokinetic events. Particle size determines the site of deposition in the respiratory tract. Generally, in humans, fine mode particles (<2.5 um) preferentially deposit in the pulmonary region, and coarse mode particles (>2.5 um) deposit in the tracheobronchial and extrathoracic regions. Those particles depositing in the extrathoracic and tracheobronchial regions are predominantly cleared by the mucociliary escalator into the gastrointestinal tract where absorption is quite low (about 3%). Particles deposited in the pulmonary region are cleared predominantly to the systemic compartment by absorption into the blood and lymph circulation.

Disregarding the possibility of counteracting mechanisms, 100% absorption of particles deposited in the pulmonary region is assumed. Another possible route of exposure may exist.

Studies such as those of Perl and Good (1987) and Evans and Hastings (1992) have indicated that neurotoxic metals such as aluminum and cadmium can be directly transported to the brain olfactory bulbs via nasal olfactory pathways (i.e., from extrathoracic deposition). The alteration in olfactory perception that Mergler et al. (1993) found in Mn-exposed workers lends support to the speculation that this pathway may also operate for Mn, which would further complicate understanding of target-site dosimetry.

The human health effects data base on Mn does not include quantitative inhalation pharmacokinetics information on the major oxides of Mn. Two of the studies described in the Principal and Support Studies (Roels et al., 1992; Mergler et al., 1993) measured respirable as well as total Mn dust, and one study (Roels et al., 1992) dealt with workers exposed to only one form of Mn, namely MnO2. However, this information does not allow quantitative determinations of the dose delivered to the respiratory tract or estimates of target-site doses. After absorption via the respiratory tract, Mn is transported through the blood stream directly to the brain, bypassing the liver and the opportunity for first-pass hepatic clearance. This direct path from the respiratory tract to the brain is the primary reason for the differential toxicity of inhaled and ingested Mn. Pharmacokinetic analyses based on inhalation of manganese chloride (MnCl2) by macaque monkeys (Newland et al., 1987) indicated that clearance from the brain was slower than from the respiratory tract and that the rate of clearance depended on the route of exposure. Brain half-times were 223-267 days after inhalation vs. 53 days following subcutaneous administration (Newland et al., 1987) or 54 days in humans given Mn intravenously (Cotzias et al., 1968). These long half-times were thought to reflect both slower clearance of brain stores and replenishment from other organs, particularly the respiratory tract. In rats, Drown et al. (1986) also observed slower clearance of labeled Mn from brain than from the respiratory tract. Several occupational physicians have reported large individual differences in workers' susceptibility to Mn intoxication, which Rodier (1955) speculated might be due in part to differences in the ability to clear particulate Mn from the lung.

The bioavailability of different forms of Mn is another matter for consideration. Roels et al. (1992) noted that geometric mean blood and urinary Mn levels of workers exposed only to MnO2 in their 1992 report were lower (MnB: 0.81 ug/dL; MnU: 0.84 ug/g creatinine) than those of workers exposed to mixed oxides and salts in their 1987 report (MnB: 1.22 ug/dL; MnU: 1.59 ug/g creatine), even though airborne total dust levels were approximately the same (geometric means of 0.94 and 0.95 mg/cu.m, respectively). Mena et al. (1969) observed no difference between the absorption of 1 um particles of MnCl2 and manganese sesquioxide (Mn2O3) in healthy adults. Drown et al. (1986) found that following intratracheal instillation of MnCl2 and Mn3O4 in rats, the soluble chloride cleared four times faster than the insoluble oxide from the respiratory tract. However, despite this initial difference, after 2 weeks the amounts of labeled Mn in the respiratory tract were similar for the two compounds. Recent work by Komura and Sakamoto (1993) comparing different forms of Mn in mouse diet suggested that less soluble forms such as MnO2 were taken up to a significantly greater degree in cerebral cortex than the more soluble forms of MnCl2 and manganese acetate [Mn(CH3COO)2]; however, the corpus striatal binding characteristics of the +4 valence state of Mn in MnO2 were not substantially different from those of the divalent forms in MnCl2,

Mn(CH3COO)2, and manganese carbonate. Different oxidation states of certain metals (e.g., chromium, nickel, mercury) are known to have different toxicities, and some researchers have suggested that endogenous Mn can have quite different roles in Mn neurotoxicity depending on its oxidation state (e.g., Archibald and Tyree, 1987; Donaldson et al., 1982). There have been unsubstantiated claims that the higher valence states of Mn (Mn+3, Mn+4) and the higher oxides in ores (Mn2O3 and Mn3O4) are more toxic (Oberdoerster and Cherian, 1988). At present, however, insufficient information exists by which to determine the relative toxicities of different forms of Mn, and thus, for the purpose of deriving an RfC for Mn, no distinction is made among various compounds of Mn.

Because Mn is an essential element and is commonly ingested in diet, total Mn exposure is an issue. It would be desirable to know the effective target-site doses and apportion the dose to both the inhalation and oral routes of exposure. However, given the lack of data regarding oral and inhalation pharmacokinetics under environmental conditions, such quantitative apportionment is not possible at present.

Among the primary effects associated with Mn toxicity from inhalation exposure in humans are signs and symptoms of CNS toxicity. The first medical description of chronic Mn neurotoxicity (manganism) in workers is generally credited to Couper in the 1830s (NAS, 1973). Although the course and degree of Mn intoxication can vary greatly among individuals, manganism is generally considered to consist of two or three phases (Rodier, 1955). The first is the psychiatric aspect, which includes disturbances such as excessive weeping and laughing, sleep disturbance, irritability, apathy, and anorexia. These symptoms can occur independently of the second phase, neurological signs. The latter may include gait disturbances, dysarthria, clumsiness, muscle cramps, tremor, and mask-like facial expression. In addition, there may be a final stage of Mn intoxication involving symptoms of irreversible dystonia and hyperflexion of muscles that may not appear until many years after the onset of exposure (Cotzias et al., 1968). Cotzias et al. (1976) noted a parallel between these stages of symptoms and the biphasic pattern of dopamine levels over time in the Mn-exposed individuals noted above. Indeed, various specific features of Mn toxicity show biphasic patterns in which there is generally first an increase then a decrease in performance (e.g., a notable increase in libido followed by impotence, or excitement followed by somnolence) (Rodier, 1955).

In addition to studies described in the Principal and Supporting Studies, numerous investigators have reported CNS effects in workers exposed to Mn dust or fumes (Sjoegren et al., 1990; Huang et al., 1989; Wang et al., 1989; Badawy and Shakour, 1984; Siegl and Bergert, 1982; Chandra et al., 1981; Saric et al., 1977; Cook et al., 1974; Smyth et al., 1973; Emara et al., 1971; Tanaka and Lieben, 1969; Schuler et al., 1957; Rodier, 1955; Flinn et al., 1941). Limitations in these studies generally preclude describing a quantitative concentration-response relationship. Exposure information is often quite limited, with inadequate information on the historical pattern of Mn concentrations or on the chemical composition and particle size distribution of the dust. In addition, exposure to other chemicals in the workplace is often not adequately characterized. Despite these limitations, such studies collectively point to neurobehavioral dysfunction as a primary endpoint for Mn toxicity.

The neuropathological bases for manganism have been investigated by many researchers and have indicated the involvement of the corpus striatum and the extrapyramidal motor system (e.g., Archibald and Tyree, 1987; Donaldson and Barbeau, 1985; Donaldson et al., 1982; Eriksson et al., 1987, 1992). Neuropathological lesions have generally been associated with the basal ganglia, specifically involving neuronal degeneration in the putamen and globus pallidus (e.g., Newland et al., 1987). Brain imaging studies (e.g., Wolters et al., 1989; Nelson et al., 1993) more recently have begun to provide additional insight into the brain structures involved in Mn toxicity.

In terms of the neurochemistry of Mn toxicity, several studies have shown that dopamine levels are affected by Mn exposure in humans, monkeys, and rodents, with various indications of an initial increase in dopamine followed by a longer term decrease (e.g., Cotzias et al., 1976; Bird et al., 1984; Barbeau, 1984; Brouillet et al., 1993). Some theories of Mn neurotoxicity have focused on the role of excessive Mn in the oxidation of dopamine resulting in free radicals and cytotoxicity (e.g., Donaldson et al., 1982; Barbeau, 1984). In addition, the fundamental role of mitochondrial energy metabolism in Mn toxicity has been indicated by the studies of Aschner and Aschner (1991), Gavin et al. (1992), and others. Brouillet et al. (1993) have suggested that the mitochondrial dysfunctional effects of Mn result in various oxidative stresses to cellular defense mechanisms (e.g., glutathione) and, secondarily, free radical damage to mitochondrial DNA. In view of the slow release of Mn from mitochondria (Gavin et al., 1992), such an indirect effect would help account for a progressive loss of function in the absence of ongoing Mn exposure (Brouillet et al., 1993), as Mn toxicity has been known to continue to progress in humans despite the termination of exposure (Cotzias et al., 1968; Rodier, 1955).

Because of the involvement of the dopaminergic system and extrapyramidal motor system in both Parkinson's disease and manganism, symptoms of the two diseases are somewhat similar, and several writers have suggested the possibility of a common etiology; however, many neurological specialists make a clear distinction in the etiologies and clinical features of Parkinson's disease and manganism (Barbeau, 1984; Langston et al., 1987).

Another primary endpoint of Mn toxicity has been male reproductive dysfunction, often manifesting in symptoms such as loss of libido, impotence, and similar complaints (e.g., Rodier, 1955; Cook et al., 1974). Some neuropathological evidence suggests that the hypothalamus is a site of Mn accumulation (Donaldson et al., 1973); thus, disturbance of the hypothalamic-pituitary-gonadal axis hormones might be expected (Deskin et al., 1981) and has been examined in a few occupational studies. Lauwerys et al. (1985) reported the results of a fertility questionnaire administered to male factory workers (n = 85) exposed to Mn dust. This study involved the same population of workers for which Roels et al. (1987) reported neurobehavioral disturbances. The range of Mn levels in the breathing zone was 0.07-8.61 mg/cu.m, with a median concentration of 0.97 mg/cu.m. Average length of exposure was 7.9 years (range of 1-19 years). A group of workers (n = 81) with a similar workload was used as a control group. The number of births expected during different age intervals of the workers (16-25, 26-35, and 36-45 years) was calculated on the basis of the reproductive experience of the control employees during the same period. A decrease in the number of

children born to workers exposed to Mn dust during the ages of 16-25 and 26-35 was observed. No difference in the sex ratio of the children was found. The same apparent LOAEL(HEC) (0.34 mg/cu.m) that was identified in Roels et al. (1987) for neurobehavioral effects is identified in this study for human reproductive effects.

However, a more recent report from the same group of investigators (Gennart et al., 1992), based on 70 of the alkaline battery plant workers evaluated by Roels et al. (1992), indicated that the probability of live birth was not different between the Mn-exposed and control workers. Also, in the study by Roels et al. (1992), serum levels of certain hormones related to reproductive function (FSH, LH, and prolactin) were not significantly different for the full group of 92 Mn workers vs. 102 controls. The latter results are partially supported by a preliminary report by Alessio et al. (1989), who found that serum FSH and LH levels were not significantly different in 14 workers generally exposed to <1 mg Mn/cu.m compared to controls, although prolactin and cortisol levels were significantly higher in the Mn-exposed workers. It is possible that differences in the forms of Mn to which workers were exposed in these studies may have contributed to the similarities and differences in the results, but insufficient information exists to substantiate this speculation.

Average concentrations of airborne Mn differed slightly in the reports of Gennart et al. (1992) and Roels et al. (1992), evidently because only a subset of Mn workers, presumably with different job functions, was used in the Gennart et al. (1992) analysis. The median respirable dust concentration was 0.18 mg/cu.m, and the median total dust concentration (comparable to Roels et al., 1987, and Lauwerys et al., 1985) was 0.71 mg/cu.m. Thus, if 0.34 mg/cu.m is identified as a LOAEL(HEC) based on the reports of Lauwerys et al. (1985) and Roels et al. (1987), 0.25 mg/cu.m total dust is the NOAEL(HEC) for reproductive effects based on the report of negative findings by Gennart et al. (1992).

The respiratory system is another primary target for Mn toxicity; numerous reports of Mn pneumonitis and other effects on the respiratory system have appeared in the literature, dating back to 1921 (NAS, 1973). In their cross-sectional study of workers exposed to mixed Mn oxides and salts (described in the Principal and Supporting Studies), Roels et al. (1987) found that significantly greater prevalences of coughs during the cold season, dyspnea during exercise, and recent episodes of acute bronchitis were reported in the exposed group on a self-administered questionnaire. However, objectively measured lung function parameters were only slightly altered and only in Mn-exposed smokers (also see Saric and Lucic-Palaic, 1977, regarding a possible synergism between Mn and smoking in producing respiratory symptoms). In their more recent study, Roels et al. (1992) found no significant differences between MnO2-exposed and control workers in responses to a questionnaire regarding respiratory symptoms. Nor were objective spirometric measurements significantly different for the two groups. The LOAEL(HEC) for respiratory effects is 0.34 mg/cu.m total dust, based on the Roels et al. (1987) study, and the NOAEL(HEC) is 0.05 mg/cu.m respirable dust, based on the Roels et al. (1992) study. In view of the near equivalence of the geometric mean total dust concentrations in the 1987 and 1992 studies by Roels et al. (0.94 and 0.95 mg/cu.m, respectively), there in fact may be little difference between the LOAEL(HEC) and the NOAEL(HEC) in terms of air concentrations; however, differences in the forms of Mn

(MnO2 vs. mixed Mn oxides and salts) to which the workers in the two studies were exposed make it difficult to compare these values quantitatively.

Nogawa et al. (1973) investigated an association between atmospheric Mn levels and respiratory endpoints in junior high school students. A questionnaire focusing on eye, nose, and throat symptoms and pulmonary function tests were given to students attending junior high schools that were 100 m (enrollment = 1258) and 7 km (enrollment = 648) from a ferromanganese plant. Approximately 97-99% of the students participated. Based on measurements obtained at another time by a government agency, the 5-day average atmospheric Mn level 300 m from the plant was reported to be 0.0067 mg/cu.m.

Significant increases in past history of pneumonia, eye problems, clogged nose, nose colds, throat swelling and soreness, and other symptoms were noted among the students in the school 100 m from the plant. Those living closest to the plant reported more throat symptoms and past history of pneumonia than did students living farther away. Pulmonary function tests revealed statistically significant decreases in maximum expiratory flow, forced vital capacity (FVC), forced expiratory volume at 1 second (FEV-1), and the FVC:FEV-1 ratio in the students attending the school closer to the plant, with some measures suggesting a relationship between performance and distance of residence from the plant.

Although the results from the study of Nogawa et al. (1973) suggest an association between ambient Mn exposure and respiratory problems, limitations in the study make it difficult to interpret. No direct measurements were made of atmospheric Mn levels either in the schools or homes, and exposure levels were inferred from the distance from the plant and other indirect measures of Mn in the environment. Also, the authors did not note whether socioeconomic variables were controlled, and this factor could well be confounded with both distance from the plant and health problems. A follow-up study by Kagamimori et al. (1973) suggested that, following reductions in Mn emissions (with apparently no reduction in sulfur dioxide or total dust) from the ferromanganese plant, students nearest the plant showed improvements in subjective symptoms and pulmonary function tests. As before, however, exposure levels were not adequately characterized to allow clear-cut conclusions.

Lloyd-Davies (1946) reported an increased incidence of pneumonia in men employed at a potassium permanganate manufacturing facility over an 8-year period. During that period, the number of workers in the facility varied from 40 to 124. Dust measurements were well described in terms of collection conditions and particle size and composition, but actual exposure levels were not evaluated. Air concentrations ranged from 9.6 to 83.4 mg/cu.m as MnO2, which constituted 41-66% of the dust. The incidence of pneumonia in the workers was 26 per 1000, compared to an average of 0.73 per 1000 in a reference group of over 5000 workers. Workers also complained of bronchitis and nasal irritation. In a continuation of this study, Lloyd-Davies and Harding (1949) reported the results of sputum and nasopharynx cultures for four men diagnosed as having lobar- or bronchopneumonia. With the exception of one of these cases, they concluded that Mn dust, without the presence of bacterial infection or other factors, caused the observed pneumonitis.

Evidence from several laboratory animal studies supports findings in Mn-exposed humans. For example, inhaled Mn has been shown to produce significant alterations in dopamine levels in the caudate and globus pallidus of Rhesus monkeys (Bird et al., 1984) and behavioral changes in mice (Morganti et al., 1985). However, species differences may complicate interpretation of certain neurobehavioral findings in laboratory animals. Unlike primates, rodents do not have pigmented substantia nigra, which is a brain region of relatively high Mn uptake and consequent involvement in neurobehavioral dysfunction. Nevertheless, rodent and primate studies show various neurochemical, neuropathological, and neurobehavioral effects resulting from Mn exposure. However, because most laboratory animal studies of Mn neurotoxicity involve exposure by routes other than inhalation, they are not described here.

Other endpoints of Mn toxicity also have been investigated with laboratory animal models of inhalation exposure. Experimental animal data qualitatively support human study findings of respiratory effects in that Mn exposure results in increased incidence of pneumonia in rats exposed to 68-219 mg/cu.m MnO2 for 2 weeks (Shiotsuka, 1984), pulmonary emphysema in monkeys exposed to 0.7-3.0 mg/cu.m MnO2 for 10 months (Suzuki et al., 1978), and bronchiolar lesions in rats and hamsters exposed to 0.117 mg/cu.m Mn3O4 for 56 days (Moore et al., 1975). Also, Lloyd-Davies and Harding (1949) induced bronchiolar epithelium inflammation, widespread pneumonia, and granulomatous reactions in rats administered 10 mg MnO2 by intratracheal injection, and pulmonary edema in rats administered 5-50 mg MnCl2 in the same fashion. However, no significant pulmonary effects were detected in other studies of rats and monkeys exposed to as much as 1.15 mg Mn/cu.m as Mn3O4 for 9 months (Ulrich et al., 1979a,b,c) or rabbits exposed to as much as 3.9 mg Mn/cu.m as MnCl2 for 4-6 weeks (Camner et al., 1985).

Laboratory animal studies also have shown that inhaled Mn may increase susceptibility to infectious agents such as Streptococcus pyogenes in mice (Adkins et al., 1980), Enterobacter cloacae in guinea pigs (Bergstrom, 1977), Klebsiella pneumoniae in mice (Maigetter et al., 1976), and Streptococcus hemolyticus in mice (Lloyd-Davies, 1946). In general, Mn concentrations were relatively high (>10 mg/cu.m) in these studies. However, Adkins et al. (1980) concluded that, based on the regression line of the relationship between concentration and mortality in Mn-exposed mice, exposure to <0.62 mg/cu.m would result in a mortality rate that differed from controls by at least 10%.

The developmental effects of Mn have been investigated primarily from the viewpoint of the nutritional role of this element and therefore have generally involved oral exposure. Some studies indicate that neonates of various species have a greater body burden of Mn than mature individuals have, possibly because neonates do not develop the ability to eliminate Mn--and thereby maintain Mn homeostasis--until some time after birth (Miller et al., 1975; Cotzias et al., 1976; Wilson et al., 1991). Moreover, some evidence suggests that the neonate's inability to maintain Mn homeostasis is due to a limitation in the elimination of Mn rather than in its gastrointestinal absorption (Bell et al., 1989), which would suggest a potentially greater vulnerability of young individuals to excessive Mn exposure regardless of the route of exposure.

Several studies have demonstrated neurochemical alterations in young rats and mice exposed postnatally to Mn by routes other than inhalation (e.g., Kontur and Fechter, 1988; Seth and Chandra, 1984; Deskin et al., 1981; Cotzias et al., 1976). The only inhalation study of the developmental toxicity of Mn appears to be that of Lown et al. (1984). Female mice were exposed to MnO2 7 hours/day, 5 days/week for 16 weeks prior to conception and for 17 days following conception (i.e., gestational days 1-18). For the first 12 weeks, the air concentration was 49.1 mg Mn/cu.m; all later exposures were at 85.3 mg Mn/cu.m. To separate prenatal and postnatal exposure effects, a cross-fostering design was used. Although mothers exposed to MnO2 prior to conception produced significantly worse pups per litter, prenatally exposed offspring showed reduced scores on various activity measures (open field, roto-rod, and exploration) and retarded growth that persisted into adulthood. A decrease in roto-rod performance was also observed in the offspring of nonexposed mice that were fostered to Mn-exposed females during lactation. Thus, balance and coordination were affected by either gestational or postpartum exposure to MnO2.

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.

M.24.1.3 Noncarcinogenic Assessment References

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M.24.2 CARCINOGENIC ASSESSMENT

Weight-of-Evidence Classification
Classification -- D; not classifiable as to human carcinogenicity

Basis -- Existing studies are inadequate to assess the carcinogenicity of manganese.

Human Carcinogenicity Data
None.

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.

Supporting Data for Carcinogenicity
None.

M.24.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Not available.

M.24.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Not available.

M.24.2.3 Carcinogenic Assessment References

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M.25 PCB 1248

Status of Data	*Last Revised*
Oral RfD Assessment	No data
Inhalation RfC Assessment	No data
Carcinogenicity Assessment	No data

M.25.1 NONCARCINOGENIC ASSESSMENT

M.25.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

The health effects data for Aroclor 1248 were reviewed by the U.S. EPA RfD/RfC Work Group and determined to be inadequate for the derivation of an oral RfD. The verification status for this chemical currently is NOT VERIFIABLE. For additional information on the health effects of this chemical, interested parties are referred to the U.S. EPA documentation listed
below.

NOT VERIFIABLE status indicates that the U.S. EPA RfD/RfC Work Group deemed the data base at the time of review to be insufficient to derive an oral RfD according to the current Agency guidelines. This status does not preclude the use of information in cited references for assessment by others.

Derivation of an oral RfD for Aroclor 1248 is not recommended because a Frank Effect (death of an infant) was noted at the lowest dose tested in a sensitive animal species, rhesus monkeys (Macaca mulatta). In general, Rhesus monkeys have shown adverse effects to PCB mixtures at doses 10-fold lower than in other species. The data indicated a dose-response relationship for this effect.

Schantz et al. (1989) evaluated neurobehavioral performance in offspring of rhesus monkeys that had been exposed to 0.03, 0.1 and 0.2 mg/kg-day of dietary Aroclor 1248 for different durations. Group I consisted of infants whose dams had received 0.03 mg/kg-day. Of the seven dams for this group, six delivered viable offspring. Necropsy of the infant who died at the time of weaning showed signs of PCB intoxication that included thymic atrophy and skin hyperpigmentation. Group II consisted of offspring of 4/8 females fed 0.1 mg/kg-day of Aroclor 1248. Of the eight dams of this group, one delivered a dead infant and one delivered an infant that died shortly after weaning with signs of PCB intoxication. Group III consisted of offspring of 3/7 females fed 0.2 mg/kg-day of Aroclor 1248. Of the seven females that were dams in this group, only three delivered live infants. Information on maternal toxicity was not provided in the report. Mild dermatological lesions and hyperpigmentation about the hairline developed in offspring in all treated groups during nursing, but no signs of toxicity were evident at the time of neurological testing (age 14 months). Offspring weights at birth and weaning were significantly reduced in Group III. Offspring in Groups I and II did not differ from controls on spatial, color or shape in two-choice discrimination reversal learning

tests, but decreased performance on a shape discrimination problem was observed in Group III when irrelevant cues were inserted. On the basis of thymic atrophy and chloracne and death of 1 of 7 infants, it is concluded that 0.03 mg/kg-day represents a FEL for developmental effects.

Adult female Rhesus monkeys were fed 0, 2.5 or 5 ppm (0, 0.1 and 0.2 mg/kg-day) of Aroclor 1248 incorporated in food pellets for up to 14 months (Barsotti et al., 1976; Barsotti, 1980). The exposure period ran from 7 months prior to breeding through gestation, and then for an additional 4 months until the infants were weaned. Some treated females began showing skin changes, such as hyperpigmentation and alopecia, characteristic signs of PCB intoxication, during the first 2 months of dosing. Monkeys with less body fat were the first to show clinical signs, regardless of the dose group to which they were assigned. All treated females showed signs of PCB intoxication to some degree by 6 months. A progressive increase in SGPT values was observed for all treated monkeys and this increase was found to be statistically significant ($p < 0.05$) by the 22nd month of the study, even though dosing stopped at the end of the l4th month. One female in each dose group developed severe shigellosis and died, and other dosed females developed clinical signs of shigellosis but did not die. Necropsies of deceased monkeys showed focal necrosis and lipid deposition of the liver, as well as marked subcutaneous edema. Increased menstrual duration was noted as well as occasional amenorrhea.

For the experimental breeding trial, conducted during the dosing period, all low-dose monkeys (8/8) conceived; 3/8 aborted and 5/8 delivered live infants. However, 3 of these 5 liveborn infants showed clinical signs of PCB toxicity and, being unable to withstand the stress of weaning, died when separated from their dams. Among the high-dose monkeys, 6/8 conceived. Among these six conceptions, four ended in abortion, one infant went to term, but was stillborn. Only one normal birth occurred among this group; however, at the time of weaning, this infant showed clinical signs of PCB toxicity and died.

The investigators realized that PCB mixtures might have latent effects that could appear long after dosing had ceased. Thus, they included three additional recovery breeding periods after dosing had been completed.

The first recovery breeding trial occurred approximately 22 months after the initiation of Aroclor 1248 dosing and 8 months after dosing had stopped. For the low-dose dams, 8/8 conceived. One of these eight conceptions resulted in abortion. Of the seven livebirths, two infants died at or before weaning. Among the high-dose mothers, 7/7 conceived. There was one abortion and one stillbirth among this group of seven mothers, and five livebirths. Among the group of five livebirths, three infants died at or before weaning.

A second recovery breeding trial was conducted approximately 36 months after the completion of Aroclor 1248 dosing. Among the low-dose mothers, 5/7 conceived. There was one stillbirth and four live births. All four of the liveborn infants survived past weaning and were available for behavioral testing at 14 months and 4 years of age. Among the high-dose mothers, 4/6 conceived for this breeding trial. There were no abortions among the four conceptions, but one stillbirth did occur; there were three livebirths.

The third recovery breeding trial was conducted 55 months after the completion of Aroclor 1248 dosing. Among the low-dose dams, 7/7 conceived. There were no abortions among this group but two stillbirths did occur. All five liveborn infants survived past weaning. For the high-dose mothers, only five had normal reproductive cycles and 4/5 conceived. Among the four conceptions, one ended in abortion, another infant was stillborn and two were born live.

In the first recovery breeding trial the average birth weights for the dosed groups were found to be reduced when compared with controls. For the second recovery breeding trial, the mean weight of the test group infants was 15 and 22% below the control group.

Results of this prolonged recovery period revealed impairment of reproductive function in female Rhesus monkeys lasting for more than 4 years after dosing ceased. In the groups of infants for which birth-weight data are available, a significant reduction in mean birth weight for PCB-exposed infants is evident.

Thomas and Hinsdill (1978) performed immunologic tests after Rhesus monkeys had been fed 0, 2.5 and 5 ppm dietary Aroclor 1248 for 11 months. All treated monkeys developed facial acne and edema and swollen eyelids to varying degrees after 6 months, with pronounced alopecia occurring in the 0.2 mg/kg-day group. Following the treatment period, the monkeys were inoculated with sheep red blood cells (SRBC) and tetanus toxoid. Anti-SRBC antibody titers were significantly reduced in the 0.2 mg/kg-day group at weeks 1 and 12 after inoculation, but antibody response to tetanus toxoid was not affected by treatment at either dosage level.

Groups of three female New Zealand white rabbits were fed 0, 10, 100 or 250 ppm of Aroclor 1248 for 4 weeks and bred with untreated males (Thomas and Hinsdill, 1980). No maternal toxicity was evident. Body-weight gain was significantly reduced in the offspring in the high-dose group.

Barsotti, D.A., R.J. Marlar and J.R. Allen. 1976. Reproductive dysfunction in rhesus monkeys exposed to low levels of polychlorinated biphenyls (Aroclor 1248). Food Cosmet. Toxicol. 14: 99-103.

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Schantz, S.L., E.D. Levin, R.W. Bowman et al. 1989. Effects of perinatal PCB exposure on discrimination-reversal learning in monkeys. Neurotoxicol. Teratol. 11: 243-250.

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Thomas, P.T. and R.D. Hinsdill. 1980. Perinatal PCB exposure and its effect on the immune system of young rabbits. Drug Chem. Toxicol. 3: 173-184.

M.25.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.25.1.3 Noncarcinogenic Assessment References

Barsotti, D.A., R.J. Marlar and J.R. Allen. 1976. Reproductive dysfunction in rhesus monkeys exposed to low levels of polychlorinated biphenyls (Aroclor 1248). Food Cosmet. Toxicol. 14: 99-103.

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M.25.2 CARCINOGENIC ASSESSMENT

This substance/agent has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential.

M.26 PCB 1254

Status of Data	*Last Revised*
Oral RfD Assessment	10/01/94
Inhalation RfC Assessment	No data
Carcinogenicity Assessment	No data

M.26.1 NONCARCINOGENIC ASSESSMENT

M.26.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses	UF	MF	RfD
Ocular exudate, inflamed and prominent Meibomian glands, distorted growth of finger and toe nails; decreased antibody (IgG and IgM) response to sheep erythrocytes	NOAEL: None	300	1	2E-5 mg/kg/day
Monkey Clinical and Immunologic Studies	LOAEL: 0.005 mg/kg-day			

Arnold et al., 1994a,b; Tryphonas et al., 1989, 1991a,b

Principal and Supporting Studies

Arnold, D.L., F. Bryce, R. Stapley et al. 1993a. Toxicological consequences of Aroclor 1254 ingestion by female Rhesus (Macaca mulatta) monkeys, Part 1A: Prebreeding phase - clinical health findings. Food Chem. Toxicol. 31: 799-810.

Arnold, D.L., F. Bryce, K. Karpinski et al. 1993b. Toxicological consequences of Aroclor 1254 ingestion by female Rhesus (Macaca mulatta) monkeys, Part 1B: Prebreeding phase -clinical and analytical laboratory findings. Food Chem. Toxicol. 31: 811-824.

Tryphonas, H., S. Hayward, L. O'Grady et al. 1989. Immunotoxicity studies of PCB (Aroclor 1254) in the adult rhesus (Macaca mulatta) monkey -- preliminary report. Int. J. Immunopharmacol. 11: 199-206.

Tryphonas, H., M.I. Luster, G. Schiffman et al. 1991a. Effect of chronic exposure of PCB (Aroclor 1254) on specific and nonspecific immune parameters in the rhesus (Macaca mulatta) monkey. Fund. Appl. Toxicol. 16(4): 773-786.

Tryphonas, H., M.I. Luster, K.L. White et al. 1991b. Effects of PCB (Aroclor 1254) on non-specific immune parameters in Rhesus (Macaca mulatta) monkeys. Int. J. Immunopharmacol. 13: 639-648.

Groups of 16 adult female rhesus monkeys ingested gelatin capsules containing Aroclor 1254 (Monsanto Lot No. KA634) in 1:1 glycerol: corn oil vehicle daily at dosages of 0, 5, 20, 40 or 80 ug/kg-day for more than 5 years. The Aroclor mixture contained 5.19 ppm of polychlorinated dibenzofurans and undetectable levels of polychlorinated dibenzo-p-dioxins (Truelove et al., 1990). At study initiation the monkeys were 11.1 +/- 4.1 years old (Tryphonas et al., 1991a,b; Arnold et al., 1993a,b). After 25 months of exposure the monkeys had achieved a pharmacokinetic steady-state based on PCB concentrations in adipose tissue and/or blood (Tryphonas et al., 1989). Results of general health and clinical pathology evaluations conducted during the first 37 months of exposure were reported by Arnold et al. (1993a,b). Results of immunologic assessments after 23 and 55 months of exposure were reported by Tryphonas et al. (1989, 1991a,b). Results of reproductive endocrinology evaluations after 24 or 29 months of exposure were reported by Truelove et al. (1990) and Arnold et al. (1993a). Effects on hydrocortisone levels during the first 22 months of exposure were reported by Loo et al. (1989) and Arnold et al. (1993b). All of the aforementioned evaluations were performed during the prebreeding phase of the study. Results of reproduction and histopathology evaluations in these monkeys are not fully available (Arnold, 1992).

General health status was evaluated daily, and body weight measurements, feed conversion ratio calculations, and detailed clinical evaluations were performed weekly throughout the study. Analyses of clinical signs of toxicity were limited to the occurrence of eye exudate, inflammation and/or prominence of the eyelid Meibomian (tarsal) glands, and particular changes in finger and toe nails (prominent nail beds, separation from nail beds, elevated nail beds, and nails folding on themselves). Each endpoint was analyzed for individual treatment-control group differences and dose-related trends with respect to incidence rate, total frequency of observed occurrences, and the onset time of the condition. With respect to effects on the eyes, the treatment-control group comparisons showed statistically significant (p less than or equal to 0.05) increases in the total frequency of inflamed and/or prominent Meibomian glands at 0.005, 0.02 and 0.08 mg/kg-day, and decreased onset time for these effects at 0.08 mg/kg-day. Significant dose-related trends (p less than or equal to 0.05) were observed for increased total frequencies of inflamed and/or prominent Meibomian glands, decreased onset time of inflamed and/or prominent Meibomian glands, and increased incidences of eye exudate. With respect to effects on finger and/or toe nails, the treatment-control group comparisons showed significantly (p less than or equal to 0.05) increased incidence of certain nail changes at 0.005 mg/kg-day (nail folding) and 0.08 mg/kg-day (elevated nails), increased total frequency of certain nail changes at 0.005 mg/kg-day (nail separation), 0.04 mg/kg-day (nail folding and separation) and 0.08 mg/kg-day (nail folding and separation, prominent beds, elevated nails), and decreased onset time of certain nail changes at 0.005 mg/kg-day (elevated nails) and 0.08 mg/kg-day (nail folding, prominent beds, elevated nails). Significant dose-related trends (p less than or equal to 0.05) were observed for certain nail changes (prominent beds, elevated nails) when adjusted

for onset time, total frequencies of certain nail changes (nail folding and separation, prominent beds, elevated nails), and decreases in onset time of certain nail changes (nail folding, prominent beds, elevated nails).

Immunologic assessment showed significant ($p < 0.01$ or < 0.05) reductions in IgG (at all doses of Aroclor 1254) and IgM (all doses but 0.02 mg/kg-day) antibody levels in response to injected sheep red blood cells (SRBC) after 23 months of exposure (Tryphonas et al., 1989). A significant ($p < 0.05$) decrease in the percent of helper T-lymphocytes, a significant ($p < 0.05$) increase in the percent and absolute level of suppressor T-lymphocytes (TS) and a significant ($p < 0.01$) reduction in TH/TS ratio was observed at 0.08 mg/kg-day. The antibody response to SRBC is an antigen-driven response that requires the interaction of several distinct cell types (i.e., antigen processing and presentation by macrophages, participation by T-helper cells and finally proliferation and differentiation of B cells into plasma cells that secrete the antibody), which result in the production and secretion of antibodies specific for SRBC from plasma cells. Perturbation in any of the cells or cell-to-cell interactions by physical, chemical or biological agents can result in aberrant antibody responses. The necessity for the interaction of the three principal cells of the immune system (i.e., macrophage, B lymphocyte and T lymphocyte), in response to SRBC, is the main reason why this response has been so widely used in immunotoxicity testing as a surrogate for infection with a pathogenic organism.

In a recent evaluation of the sensitivity and predictability of various immune function assays used for immunotoxicity testing in the mouse (Luster et al., 1992), the antibody plaque-forming cell (PFC) response to SRBC was found to show the highest association with immunotoxic compounds. Essentially this means that the antibody PFC response to SRBC is a very good predictor of immunotoxicants. Also, it has recently been demonstrated that measurement of serum antibody titer to SRBC using the ELISA assay is as sensitive as the PFC assay for determining the response to SRBC (Butterworth et al., 1993).

There were no exposure-related effects on total B-lymphocytes, total T-lymphocytes, total serum immunoglobulin levels, total serum protein, serum protein fractions after 23 months. No exposure-related effects on serum hydrocortisone levels were observed although the SRBC assay is considered a good surrogate (Tryphonas et al., 1989; Loo et al., 1989; Arnold et al., 1993b).

After 55 months of exposure, there was a significant dose-related decrease ($p < 0.0005$ for pairwise comparisons and trend test) in the IgM antibody response to injected SRBC at greater than or equal to 0.005 mg/kg-day at all times of evaluation (1-4 weeks postimmunization) (Tryphonas et al., 1991a). IgG antibody response to injected SRBC was significantly ($p < 0.01$) decreased only at 0.04 mg/kg-day, although the overall trend for dose-response was significant ($p = 0.033$). The antibody response to pneumococcus antigen did not differ significantly among all test groups (including controls) at any time tested and showed no dose-related trend. However, the antibody response to pneumococcus antigen is a T cell-independent response and the fact that there is no change with this antigen is not inconsistent with the depressed response to the T cell-dependent SRBC antigen. Other data corroborate the significance of Aroclor 1254 suppression of the antibody response to SRBC

and point to effects on T lymphocytes including the dose-related suppression of the Con A and PHA lymphoproliferative responses. The monkeys treated with greater than or equal to 0.005 mg/kg-day had significantly ($p < 0.0001$) lower mean percentage levels of total T-lymphocytes and significant trend for dose-response, but absolute numbers of T-lymphocytes were similar among test groups. Flow cytometric analysis showed no treatment-related effects on peripheral blood T-helper, T-suppressor or B-lymphocytes or TH/TS lymphocyte ratio. A statistically significant, dose-related increase was noted for thymosin alpha-1-levels but not for thymosin beta-2-levels. Serum complement activity was significantly ($p < 0.025$) increased at greater than or equal to 0.005 mg/kg-day but showed no significant ($p=0.1$) dose-related trend. Natural killer cell activity at effect or target ratios of 25:1, 50:1 or 75:1 was not significantly ($p > 0.05$) increased at any dosage, although there was a significant ($p=0.03$) dose-related trend. No signs of microbial infection were noted in any of the preceding reports.

Clinical pathology was evaluated during the first 37 months of the study (Arnold et al., 1993b). These evaluations included monthly measurements of hematology and serum biochemistry (including serum protein, RBC indices, semi-monthly measurements of thyroid function, and daily measurements of urinary porphyrins during the 33rd month of dosing). Significant ($p \leq 0.05$) decreases in average dose-group values compared with controls were found for serum cholesterol at 0.04 mg/kg-day, and reticulocyte count, serum cholesterol, total bilirubin, and alpha-1 + alpha-2-globulins at 0.08 mg/kg-day. Significant dose-related decreasing linear trends were also observed for reticulocyte count ($p=0.002$), cholesterol (p less than or equal to 0.001), and total bilirubin ($p=0.005$). Dose-related decreasing linear trends were also observed for red blood cell count ($p=0.019$), mean platelet volume ($p=0.034$), hematocrit ($p=0.064$), hemoglobin concentration ($p=0.041$). With regard to thyroid endpoints [serum thyroxine (T4), serum triiodothyronine (T3) uptake ratio, percent T3 uptake, and free thyroxine index], dose-response analysis consisted of group mean comparisons and an assessment of parallelism in the response profiles (an absence of parallelism would indicate time-dose interactive effects). No statistically significant changes were observed for any of the thyroid endpoints.

After approximately 2 years of dosing, each dose group was randomly divided into two test groups for daily analyses of serum progesterone and estrogen concentrations during one menstrual cycle (Truelove et al., 1990; Arnold et al., 1993b). There were no statistically significant differences between treated and control monkeys in menstrual cycle length or menses duration, and no apparent treatment-related effects on incidence of anovulatory cycles or temporal relationship between estrogen peak and menses onset, menses end or progesterone peak (Truelove et al., 1990; Arnold et al., 1993a,b).

To summarize the above, monkeys that ingested 0.005-0.08 mg/kg-day doses of Aroclor 1254 showed ocular exudate, prominence and inflammation of the Meibomian glands and distortion in nail bed formation. These changes were seen at the lowest dose tested, 0.005 mg/kg-day, and a dose-dependent response was demonstrated. Similar changes have been documented in humans for accidental oral ingestion of PCBs. Among the various immunologic function tests that were performed, the increases in IgM and IgG antibodies to sheep erythrocytes are most significant. IgG and IgM antibodies in response to SRBC were reduced after 23 months

of exposure but only the IgM antibodies were clearly decreased after 55 months. Particular importance is attributed to the immune response to sheep erythrocytes since it involves participation by the three principal cells of the immune system: the macrophage, B lymphocytes and T lymphocytes and has been shown to be the most predictive immunotoxicity test of those currently in use (Luster et al., 1992). On the basis the studies described, a LOAEL of 0.005 mg/kg-day was established for Aroclor 1254.

Uncertainty and Modifying Factors

UF -- A 10-fold factor is applied to account for sensitive individuals. A factor of 3 is applied to extrapolation from rhesus monkeys to humans. A full 10-fold factor for interspecies extrapolation is not considered necessary because of similarities in toxic responses and metabolism of PCBs between monkeys and humans and the general physiologic similarity between these species. A partial factor is applied for the use of a minimal LOAEL since the changes in the periocular tissues and nail bed see at the 0.05 mg/kg-day are not considered to be of marked severity. The duration of the critical study continued for approximately 25% of the lifespan of rhesus monkeys so that a reduced factor was used for extrapolation from subchronic exposure to a chronic RfD. The immunologic and clinical changes that were observed did not appear to be dependent upon duration which further justifies using a factor of 3 rather than 10 for extrapolation from subchronic to chronic, lifetime exposure. The total UF is 300.

MF -- None

Additional Studies/Comments

Human data available for risk assessment of Aroclor 1254 are useful only in a qualitative manner. Studies of the general population who were exposed to PCBs by consumption of contaminated food, particularly neurobehavioral evaluations of infants exposed in utero and/or through lactation, have been reported, but the original PCB mixtures, exposure levels and other details of exposure are not known (Kreiss et al., 1981; Humphrey, 1983; Fein et al., 1984a,b; Jacobson et al., 1984a, 1985, 1990a,b; Rogan et al., 1986; Gladen et al., 1988). Most of the information on health effects of PCB mixtures in humans is available from studies of occupational exposure. Some of these studies examined workers who had some occupational exposure to Aroclor 1254, but sequential or concurrent exposure to other Aroclor mixtures nearly always occurred, exposure involved dermal as well as inhalation routes (relative contribution by each route not known), and monitoring data are lacking or inadequate (Alvares et al., 1977; Brown and Jones, 1981; Colombi et al., 1982; Fischbein et al., 1979, 1982, 1985; Fischbein, 1985; Warshaw et al., 1979; Smith et al., 1982; Taylor et al., 1984; Lawton et al., 1985). Insufficient data are available in these studies to determine possible contributions of Aroclor 1254 alone, extent of direct skin exposure and possible contaminants. However, it is relevant to note that dermal and ocular effects, including skin irritation, chloracne, hyperpigmentation and eyelid and conjunctival irritation, have been observed in humans occupationally exposed to Aroclor 1254 and other Aroclor formulations.

Aroclor 1254 was fed to groups of eight female and four male adult rhesus monkeys once daily in dosages of 0, 5, 25 or 100 ug/kg for 14 months, followed by an observation period of 7

months (Levinskas et al., 1984). The Aroclor 1254 was dissolved in corn oil and offered to the animals in apple sauce prior to each day's feeding, and the control mixture (corn oil in applesauce) was used during the observation period. Dosages were adjusted biweekly for changing body weight as necessary. The monkeys were selected on the basis of a successful reproductive history, estimated to be at least 6 years old, and had been in captivity for 2-9 years. After 6 months of treatment the monkeys were bred to untreated males or females from the same colony over an 8-month period and offspring were observed for 2 months. Breeding was continued until conception was diagnosed by digital examination of the uterus and alterations in the menstrual cycle. Evaluations of adult animals included hematology and clinical chemistry. Urinalysis was also performed every 3 months during the study. Semen analyses were performed monthly from just prior to the start of treatment until the end of the treatment period. After 2 months of observation; sperm concentration, total sperm, sperm motility, percent abnormal cells and live/dead ratios were evaluated. Based upon these parameters, no effect was observed upon male reproductive capacity. Necropsies including histological examinations were performed on all adult animals that died during the study or were euthanized at the end of the observation period. Birth weight and somatic measurements were taken for all offspring of exposed females or males. The infants of the exposed females were subsequently evaluated monthly for body weight and complete blood cell counts were performed. Infants that did not show signs of intoxication were euthanized after 2 months and those showing signs were weaned, observed for reversal of signs, and euthanized at the end of the study along with the adults. Necropsies including histological examinations were performed on all infants that died or were euthanized.

Death or euthanasia in extremis occurred in 1/12, 0/12, 1/12 and 5/12 of the adult monkeys in the control, low-, mid- and high-dose groups, respectively. All of the deaths occurred in females except for one male in the high-dose group, and the only deaths considered to be related to treatment were in four of the high-dose animals (3 females, 1 male). Characteristic signs of PCB intoxication developed in the high-dose group after 9 months of exposure, including effects on the eyelids (redness and/or edema, wrinkling) in approximately half the animals and swelling of the lips in all animals. Other characteristic signs included bleeding gums, abnormal fingernail/toenail growth pattern and increased alopecia (including eyelashes) in several of the high-dose monkeys. In general, the signs of intoxication appeared to subside during the post-treatment period. Some of the monkeys in the mid-dose group showed signs of intoxication (swelling of the lips in one male and one female) after 15 and 18 months, respectively, and alopecia and abnormal nail growth, but no signs attributable to exposure occurred in the low-dose group. Hematologic effects at the high dose were observed including reduced packed cell volume, erythrocyte count, hemoglobin and platelet counts. In addition, increased serum iron and reduced serum cholesterol were observed, particularly in the monkeys that died. Some of the high-dose monkeys also had prolonged bleeding and improper healing at biopsy sites. Dermal histological changes characteristic of PCB poisoning were prominent in the high-dose group, occurring in 11/12 monkeys (8 females, 3 males), and included loss of secretory epithelium in the Meibomian (eyelid) glands and sebaceous glands, partial or total atrophy of sebaceous glands, follicular keratosis and/or squamous cysts. Dermal changes also occurred in four of the mid-dose monkeys, but not in the low-dose or control groups. Other histological alterations included squamous metaplasia in glandular ducts

of the tongue or lip (3 high-dose females, 1 high-dose male), subgingival epithelial cysts of the mandible (1 high-dose male, 1 high-dose female, 1 mid-dose male) and hyperplasia in the bile and pancreatic ducts and gall bladder (1 high-dose female). Nonspecific bone marrow alterations (decreased cellularity and/or granulocyte maturation) occurred in 6/12 high-dose monkeys (5 females, 1 male) and may have been compound-related because they correlated with the hematologic changes.

There was no apparent effect on male fertility based on conception rate following matings with the untreated females or the semen analyses (Levinskas et al., 1984). In the female control, low-, mid- and high-dose groups, the numbers of known pregnancies were 7, 7, 7 and 5, respectively, the numbers of live births were 6, 5, 7 and 1, respectively. Analysis of the preceding data showed that there was a statistically significant reduction in fertility in the high-dose group; this analysis refers only to the decreased number of live births. There was a clear exposure-related effect on birth weight and infant body weight gain. When compared with control group infants (mean birth weight 495.2 g) the 0.025 mg/kg-day infants (mean birth weight 392.2 g) showed a statistically significant reduction in birth weight ($p<0.005$). Most of the infants in the mid-dose group and all of the infants in the high-dose had abnormal clinical signs. These changes included being born with or developed dermal signs that were consistent with those in the adults (e.g., swollen lips, swollen eyelids and/or scanty eyelashes) and more severe at the high dose, and also developed pulmonary signs (e.g., respiratory wheezing).

Histological changes in the infants were generally similar to those observed in the adults. These effects included changes in the Meibomian and sebaceous glands, pancreatic ducts and bone marrow. Other histological changes included thymic atrophy in one mid-dose and the high-dose infant, and other effects in the high-dose infant (e.g., retarded kidney cortical maturation, bile duct hyperplasia and gastric mucosal gland cysts).

To summarize the above, no treatment-related effects were observed in the low-dose adults or their infants, indicating that 0.005 mg/kg-day is a NOAEL. For the mid-dose infants there was a 15% reduction in birth weight of infants that was statistically significant ($p<0.005$). When these infants reached 2 months of age the reduced body weight was 22% below controls and this difference was also found to be statistically significant ($p=0.05$). Ocular and dermal signs and/or histological changes characteristic of PCB intoxication developed in a some adults receiving 25 and 100 ug/kg-day, as well as in most of the infants in these groups. Based on these effects the 0.025 mg/kg-day dosage is a LOAEL. Other effects at the high dose included decreased adult survival, female fertility and numbers of live births, indicating that 0.1 mg/kg-day is a FEL. This FEL is supported by results of the Truelove study (Truelove et al., 1982).

Aroclor 1254 was fed to 1, 2 or 1 pregnant rhesus monkeys in reported average daily doses of 0, 0.1 or 0.2 mg/kg-day, respectively, 3 days/week for up to 267 days starting on gestation day 60 (Truelove et al., 1982). The exposure period included gestation and lactation. One of the adult monkeys in the low-dose group and the one adult in the high-dose group lost their fingernails after 233 and 242 days of PCB treatment, but other overt signs of intoxication were

not observed. There was a significant reduction in antibody production in response to injected SRBC in the exposed monkeys, but levels of antibody production to tetanus toxoid were not appreciably different from control. The two low-dosage monkeys delivered dead infants. The infant of the high-dosage monkey died at age 139 days; this infant showed impaired immune function as assessed by antibody production following SRBC injections. Hematological evaluation performed bimonthly following parturition in adults and the surviving infant were inconclusive. Although evaluation of the dead infants and other results of this study is complicated by the small number of animals, the characteristic dermal sign of PCB toxicity in the exposed monkeys and lack of effects in controls strongly indicate that the developmental toxicity is exposure-related. Therefore, based on the stillbirths, 0.1 mg/kg-day is a FEL in monkeys.

Groups of four young adult female rhesus monkeys were fed 0 or 0.28 mg/kg doses of Aroclor 1254 for 5 days/week for 114-121 weeks (Tryphonas et al., 1986a,b; Arnold et al., 1990). Groups of four mature adult female cynomolgus monkeys that had a poor breeding history were similarly exposed for 55-58 weeks (Tryphonas et al., 1986a; Arnold et al., 1990). The Aroclor mixture contained no detectable polychlorinated dibenzo-p-dioxin contaminants. Adjusting for the partial weekly exposure gives an average daily dosage of 0.2 mg/kg-day. Prominent clinical signs appeared in all exposed rhesus monkeys during the first 2-12 months of exposure, including facial and periorbital edema, loss of eyelashes, Meibomian gland enlargement and impaction, conjunctivitis, nail lesions progressing from dryness to detachment and gingival hyperplasia and necrosis of varying severity. Two of the exposed rhesus monkeys developed overwhelming infections of the eye or periodontal tissue after 27 months of exposure prompting sacrifice within 48 hours. The hematology and serum biochemistry evaluations showed various changes in the exposed rhesus monkeys, particularly slight or moderate normocytic anemia, depressed erythropoiesis in bone marrow and increased triglycerides and SGOT. The immunologic testing was inconclusive due to large interspecies variability. Pathology findings in the exposed rhesus monkeys included effects in the liver of three monkeys (30-55% increased relative liver weight, hepatocellular hypertrophy and necrosis, bile duct epithelial hypertrophy and hyperplasia, gall bladder epithelial hypertrophy), thyroid of two monkeys (enlargement, occasional follicular cell desquamation) and stomach of two monkeys (hypertrophic gastropathy). The cynomolgus monkeys had effects that were generally consistent with but less extensive and severe than those observed in the rhesus monkeys. After 38 weeks of exposure the rhesus monkeys were mated with untreated males; cynomolgus monkeys were not mated. The control and exposed rhesus monkeys became pregnant within 7 and 8 matings, respectively. Following extended post-implant bleeding all of the treated rhesus monkeys aborted within 30-60 days of gestation. Following recovery from the abortions the monkeys were bred again up to a maximum of seven times but none appeared to conceive. The menstrual cycle lengths and durations became erratic and longer during and subsequent to the breeding. Based on the abortions, reproductive impairment and pronounced overt signs of toxicity, the 0.2 mg/kg-day dosage is an FEL in monkeys.

Aulerich and Ringer (1977) performed a breeding study in which groups of eight female and two male adult mink were fed diets containing 0 or 2 ppm Aroclor 1254 for 39 weeks or until the kits were 4 weeks of age. The Aroclor was dissolved in acetone which was evaporated

from the diet prior to feeding. Using assumed values of 150 g/day for food consumption and 0.8 kg for body weight for female mink (Bleavins et al., 1980), the estimated dosage of Aroclor 1254 is 0.4 mg/kg-day. Approximately monthly determinations reportedly showed no statistically significant ($p < 0.05$) differences between the control and treated mink in body weight gain, hemoglobin, and hematocrit. Only two of seven mated females gave birth, producing one infant each. Of the two infants, one was born dead and the other had low body weight and was dead by age 4 weeks. Based on the reproductive and/or fetal toxicity resulting in nearly complete lack of births, 0.4 mg/kg-day is a FEL for Aroclor 1254 in mink.

Twelve female and four male adult ranch-bred mink (age 8 months, body weight not reported) were fed a diet containing 1 ppm Aroclor 1254 for 6 months (Wren et al., 1987a,b). Groups of 15 females and five males were used for unexposed controls. The mink were bred after approximately 12-14 weeks of exposure and exposure was continued until weaning at age 5 weeks. Using assumed values for food consumption and for body weight for female mink (Bleavins et al., 1980), the estimated dosage of Aroclor 1254 is 0.15 mg/kg-day. Offspring mortality during the first week of life was 75.8% higher in the exposed group than in the controls. Average body weight was significantly lower in the exposed offspring at age 3 and 5 weeks, but not at age 1 week, suggesting that transfer of PCBs by lactation may have contributed to the effect. There were no exposure-related effects on adult survival or mating performance, number of offspring per female mated or female that delivered, adult thyroid plasma T3 or T4 levels during the exposure period, adult scrotal diameter, offspring survival or relative liver weight at weaning or organ weights or histology (brain, kidney, adrenal, pituitary, thyroid).

Teratogenicity was not evaluated. The neonatal mortality indicates that 0.15 mg/kg-day is an FEL in mink.

Groups of 10 female Sprague-Dawley rats were fed 0, 1, 5, 10 or 50 ppm Aroclor 1254 in the diet for approximately 5-6 months (Byrne et al., 1987). The Aroclor was dissolved in acetone which was evaporated from the diet prior to feeding. Based on reported body weight and food consumption data the dosages are estimated to be 0.09, 0.43, 0.61 and 4.3 mg/kg-day. Serum thyroxine (T4) and triiodothyronine (T3) were evaluated at five different times during 140 and 175 days of treatment, respectively. Serum T4 levels were significantly reduced at 0.09 and 0.43 mg/kg-day by day 35 and at greater than or equal to 0.61 mg/kg-day by day 14. T3 levels were significantly reduced at 0.09 mg/kg-day by day 40 and at greater than or equal to 0.4 mg/kg-day by day 20. The suppressions were generally dose-related for T4 throughout the treatment period and T3 after 75 days. Disappearance rate of injected L-[125I] T4 was significantly decreased at greater than or equal to 0.09 mg/kg-day. Rats treated with only 0.43 or 0.61 mg/kg-day for approximately 5 months and challenged with i.p. injected TSH had diminished response of serum T4 and T3. Thyroid histology was not evaluated. There were no treatment-related effects on relative thyroid weight, body weight or food consumption. The findings of this study indicate that the decreased serum T3 and T4 resulted primarily from direct damage to the thyroid rather than suppression of the hypothalamo-pituitary axis or any enhanced peripheral catabolism (e.g., liver). Insufficient data are available to determine if the decreases in circulating thyroid hormones were physiologically significant. However, because

the effects are indicative of impaired organ function, they are at least potentially adverse and 0.09 mg/kg-day is considered to represent a LOAEL in rats.

Groups of 10 female Sprague-Dawley rats were fed 0, 1, 5, 10 or 50 ppm Aroclor 1254 in the diet for 5 months (Byrne et al., 1988). The Aroclor was dissolved in acetone which was evaporated from the diet prior to feeding. Using a rat food consumption factor of 0.05 kg food/kg body weight, the dosages are estimated to be 0.05, 0.25, 0.5 and 2.5 mg/kg-day. Serum levels of adrenal cortex hormones were evaluated in 8-10 rats 3-5 times during the treatment period. Serum corticosterone was significantly ($p<0.05$) decreased at greater than or equal to 0.25 mg/kg-day after approximately 60 days of exposure. Serum dehydroepiandrosterone and dehydroepiandrosterone sulfate were significantly ($p<0.05$) decreased at 0.25 and 0.5 mg/kg-day (not evaluated at other dosages) after approximately 100 days and 25 days of exposure, respectively. Serum corticosterone is the principal glucocorticoid in rats. Adrenal weight, adrenal histology and non-adrenal endpoints other than food consumption were not evaluated. Food consumption did not significantly differ between and among control and treatment groups. The results of this study are suggestive of toxicity to the adrenal rather than response to stress which would be expected to increase the release of glucocorticoids. Insufficient data are available to determine if the decreases in circulating adrenal cortex hormones were physiologically significant. However, because the effects are indicative of impaired organ function, they are at least potentially adverse. The dosages of 0.05 and 0.25 mg/kg-day therefore are considered to represent a NOEL and LOAEL, respectively, in rats.

Hepatotoxicity is a prominent effect of Aroclor 1254 that is well characterized in rats (U.S. EPA, 1990). The spectrum of effects includes hepatic microsomal enzyme induction, increased serum levels of liver-associated enzymes indicative of possible hepatocellular damage, liver enlargement, lipid deposition, fibrosis and necrosis. Estimated subchronic dosages as low as 1.25-2.5 mg/kg-day have been reported to produce increased liver weight and hepatic biochemical alterations in rats, but the lowest dosages producing signs of hepatic effects are generally higher than the lowest dosages that caused thyroid, adrenal and bone changes (Litterset et al., 1972; Bruckner et al., 1974; Kling and Gamble, 1982; Andrews et al., 1989). Rats fed 6.8 mg/kg-day for 8 months (Kimbrough et al., 1972) or an estimated dosage of 50 mg/kg-day for 30 days (Kling et al., 1978) developed fatty and necrotic degenerative hepatic histologic changes. Chronic dietary exposure to 1.25-5 mg/kg-day for approximately 2 years produced only preneoplastic and neoplastic liver lesions in rats (NCI, 1978; Ward, 1985).

A two-generation reproduction study was performed in which groups of 20 female and 10 male Sherman rats (age 3-4 weeks, body weight not reported) were fed 0, 1, 5, 20 or 100 ppm dietary Aroclor 1254 (Monsanto Lot No. AK-38) in peanut oil vehicle (Linder et al., 1974). Reported dosages were 0.06, 0.32, 1.5 and 7.6 mg/kg-day, and different controls were used for the less than or equal to 0.32 and greater than or equal to 1.5 mg/kg-day groups. Exposure times (before mating or conception-to-mating) ranged from 62-274 days. Exposure-related effects included increased relative liver weight in F1a weanlings at greater than or equal to 0.06 mg/kg-day, enlarged and vacuolated hepatocytes in F2a weanlings at

greater than or equal to 1.5 mg/kg-day, and 15-72% reduced litter size at greater than or equal to 1.5 mg/kg-day in the F1b, F2a and F2b generations and at 7.6 mg/kg-day in the F1a generation. Relative testes weights were increased in adult F1b males at 7.6 mg/kg-day (other groups not evaluated). The highest NOAEL is 0.32 mg/kg-day based on the increased liver weight without altered histology. The decreased litter size indicates that 1.5 mg/kg-day is a FEL.

A one-generation reproduction study was performed in which groups of 10 male and 10 female Sherman rats were fed 0, 100 or 500 ppm dietary Aroclor 1254 for 67 or 186 days prior to pair-mating for the F1a and F1b generations, respectively (Linder et al., 1974). The F0 rats received reported dosages of 0, 7.2 and 37.0 mg/kg-day and were sacrificed after a total exposure duration of 8 months for hematology, organ weight and liver histology evaluation. The study was terminated after the F1b pups were weaned. Effects in the P1 rats included increased liver weight in both sexes greater than or equal to 7.2 mg/kg-day, increased relative testis weight (absolute weight unchanged) at 37.0 mg/kg-day, decreased body weight gain in both sexes at 37.0 mg/kg-day, and changes in hematological values (reduced hematocrit and hemoglobin in both sexes, increased total leukocytes with normal differential count in females) at 37.0 mg/kg-day. Specific information on liver pathology was not reported but degenerative changes similar to those found in the Kimbrough et al. (1972) subchronic study were indicated for both dosages. Effects on the offspring included reduced survival to weaning at 7.2 mg/kg-day (85.9 and 68.1% survival in F1a and F1b pups, respectively, compared with 95.5% in controls), and reduced litter size and number and 100% pup mortality by day 3 in F1a rats at 37.0 mg/kg-day. The decreases in postnatal survival indicate that both dosages are FELs.

Groups of six to eleven female Wistar rats were fed 2.5, 26 or 269 ppm Aroclor 1254 in the diet during gestation and lactation (Overman et al., 1987). A control group was fed untreated diet that contained 0.02 ppm PCBs (i.e., no added PCBs). Using a rat food consumption factor of 0.05 kg food/kg body weight, the dosages are estimated to be 0.001, 0.13, 1.3 and 13.5 mg/kg-day. The following neurobehavioral endpoints were significantly delayed or reduced in the pups: appearance of the auditory startle response at 0.13 and 1.3 mg/kg-day at age 6 days (slightly delayed), development of righting ability at 1.3 mg/kg-day at days of age (slightly delayed) and performance on a motor coordination test at 1.3 mg/kg-day at age 7 and 8 days (slower performance). Grip strength and appearance of eye opening were not affected by exposure. Other effects attributable to exposure included increased relative liver weight in pups at weaning at greater than or equal to 1.3 mg/kg-day and reduced birth weight, 50% mortality by 2 days of age and retarded growth in pups at 13.5 mg/kg-day. There were no exposure-related effects on maternal weight gain, gestation length, litter size, pup sex ratios, number of live and dead pups or physical appearance, relative spleen and thymus weight or relative and absolute brain weight of pups. Brain PCB levels increased from birth to weaning in all groups. Based on the evidence for impaired motor coordination in developing infants the 0.13 and 1.3 mg/kg-day dosages are a NOAEL and LOAEL, respectively.

Dietary Aroclor 1254 was administered to groups of 4-10 female ICR mice in concentrations of 0, 1, 10 or 100 ppm from 90 days before mating through gestation day 18 (Welsch, 1985).

The investigators estimated the dosages to be 0.125, 1.25 and 12.5 mg/kg-day. No developmental toxicity was observed as judged by number of litters, number of dead and reabsorbed fetuses, fetal weight, incidence of gross malformations or skeletal development. Fetuses were not examined for internal malformations. Maternal effects other than significantly increased relative liver weight at greater than or equal to 0.125 mg/kg-day were not observed. No developmental effects were observed in mice treated with the same doses of PCB only on gestation days 6-18. Based on the increased maternal liver weight the highest NOAEL is 12.5 mg/kg-day.

Groups of seven adult male New Zealand white rabbits were fed dietary Aroclor 1254 in reported estimated dosages of 0, 0.18, 0.92, 2.10 or 6.54 mg/kg-day for 8 weeks (Street and Sharma, 1975). Immunological testing was started after 4 weeks of treatment at which time the rabbits were immunized with injected SRBCs. No treatment-related changes in serum antibody titers to SRBC (hemolysin and hemagglutination) were observed. SRBC-induced increases in serum gamma-globulin were consistently but not statistically significantly decreased by exposure, and the number of globulin-producing cells in popliteal lymph nodes was significantly decreased at 0.92 and 6.54 mg/kg-day. Skin sensitivity to tuberculin was generally lower in the treated groups but none of the decreases were statistically significant. Marked histologic atrophy of the thymus cortex was observed at 0.18 mg/kg-day and higher dosages except 0.92 mg/kg-day. There were no treatment-related effects on leukocyte count, histology of the spleen, thymus, liver, kidneys or spleen, relative kidney or adrenal weight, terminal body weight or food consumption. Relative liver and spleen weights were significantly increased at greater than or equal to 2.10 mg/kg-day; the increase in liver weight was 74% at the highest dosage. The 0.18 mg/kg-day dosage is a LOAEL based on the thymic cortical atrophy.

Limited specific information is available on the oral absorption of Aroclor 1254. Pregnant ferrets that ingested a single oral dose of Aroclor 1254 (approximately 0.06 mg/kg) absorbed approximately 85% of the initial amount (Bleavins et al., 1984). Studies predominately of individual chlorobiphenyl congeners indicate, in general, that PCBs are readily and extensively absorbed by animals. These studies have found oral absorption efficiency on the order of 75 to >90% in rats, mice and monkeys (Albro and Fishbein, 1972; Allen et al., 1974; Tanabe et al., 1981; Clevenger et al., 1989). A study of a non-Aroclor 54% chlorine PCB mixture prepared by the investigators provides direct evidence of absorption of PCBs in humans after oral exposure (Buhler et al., 1988), and indirect evidence of oral absorption of PCBs by humans is available from studies of ingestion of contaminated fish by the general population (Schwartz et al., 1983; Kuwabara et al., 1979). There are no quantitative data regarding inhalation absorption of PCBs in humans but studies of workers exposed suggest that PCBs are well absorbed by the inhalation and dermal routes (Maroni et al., 1981a,b; Smith et al., 1982; Wolff, 1985). PCBs distribute preferentially to adipose tissue and concentrate in human breast milk due to its high fat content (Jacobson et al., 1984b; Ando et al., 1985).

The metabolism of PCBs following oral and parenteral administration in animals has been extensively studied and reviewed, but studies in animals following inhalation or dermal exposure are lacking (Sundstrom and Hutzinger, 1976; Safe, 1980; Sipes and Schnellmann,

1987). Information on metabolism of PCBs in humans is limited to occupationally exposed individuals whose intake is derived mainly from inhalation and dermal exposure (Jensen and Sundstrom, 1974; Wolff et al., 1982; Schnellmann et al., 1983; Safe et al., 1985; Fait et al., 1989). In general, metabolism of PCBs depends on the number and position of the chlorine atoms on the phenyl ring of the constituent congeners (i.e., congener profile of the PCB mixture) and animal species. Although only limited data are available on metabolism of PCBs following inhalation exposure, there is no reason to suspect that PCBs are metabolized differently by this route.

Data exist on the in vitro hepatic metabolism and in vivo metabolic clearance of 2,2',3,3',6,6'-hexachlorobiphenyl and 4,4'-dichlorobiphenyl congeners in humans, monkeys, dogs and rats (Schnellmann et al., 1985). The hexachlorobiphenyl congener is a constituent of Aroclor 1254. For each congener, the Vmax values for metabolism in the monkey, dog and rat are consistent with the respective metabolic clearance values found in vivo. Thus, the kinetic constants for PCB metabolism obtained from the dog, monkey and rat hepatic microsomal preparations were good predictors of in vivo metabolism and clearance for these congeners. In investigations directed at determining which species most accurately predicts the metabolism and disposition of PCBs in humans, the in vitro metabolism of these congeners was also studied using human liver microsomes (Schnellmann et al., 1983, 1984). Available data suggest that metabolism of PCBs in humans would most closely resemble that of the monkey and rat. For example, the in vitro apparent Km and Vmax are comparable between humans and monkeys. These studies show consistency between the in vitro and in vivo findings and collectively indicate that metabolism of the two congeners is similar in monkeys and humans.

Confidence in the Oral RfD
Study -- Medium
Data Base -- Medium
RfD -- Medium

Confidence in the principal study is medium. Groups of 16 rhesus monkeys were tested at four dose levels and LOAEL was established on the basis of clinical signs and immunologic alterations. Data for female and male reproductive function and developmental data in a nonhuman primate species is taken from an unpublished study (Levinskas et al., 1984) which established a NOAEL for reproductive effects at 0.005 mg/kg-day. The Arnold study also included evaluation of reproductive function but the data have not been completely analyzed. Preliminary examination of the Arnold et al. data indicate that the LOAEL for female reproductive function may be 0.005 mg/kg-day. This inconsistency in effect levels for reproductive toxicity was viewed as a limitation to the data base. Furthermore, there is a limitation in the characterization of reproductive toxicology because results of an unpublished study have been considered. An extensive number of laboratory animal and human studies were available for review, including two-generation reproductive studies. The chronic, 2-year bioassays performed in F344 rats showed evidence of degenerative hepatocellular changes in addition to the neoplastic changs that were observed. Only limited assessment of nonhepatic changes were made. Human occupational and environmental data is available for commercial

PCB mixtures in general but not specifically for Aroclor 1254. The data base is rated medium on the basis of these considerations. Overall confidence in the RfD is medium.

M.26.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.26.1.3 Noncarcinogenic Assessment References

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M.26.2 CARCINOGENIC ASSESSMENT

This substance/agent has not been evaluated by the U.S. EPA for evidence of human carcinogenic potential.

M.27 PHENANTHRENE

Status of Data	*Last Revised*
Oral RfD Assessment	No data
Inhalation RfC Assessment	09/01/94
Carcinogenicity Assessment	12/01/90

M.27.1 NONCARCINOGENIC ASSESSMENT

M.27.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Not available at this time.

M.27.1.2 Reference Concentration for Chronic Inhalation Exposure

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

M.27.2 CARCINOGENIC ASSESSMENT

Weight-of-Evidence Classification
Classification -- D, not classifiable as to human carcinogenicity

Basis -- Based on no human data and inadequate data from a single gavage study in rats and skin painting and injection studies in mice.

Human Carcinogenicity
None.

Animal Carcinogenicity
Inadequate. Data from a rat gavage study and mouse skin application and injection studies are not adequate to assess the carcinogenicity of phenanthrene. Ten female Sprague-Dawley rats received a single oral dose of 200 mg phenanthrene in sesame oil (Huggins and Yang, 1962). No mammary tumors occurred. The observation period was not specified; however, based on the discussion of other experiments in the report it was probably at least 60 days. Controls were not reported.

Complete carcinogenic activity was not shown in two skin painting assays. Kennaway (1924) reported no tumors in 100 mice (strain and sex not specified) treated with phenanthrene (purity not specified) in 90% benzene (dose not reported) for 9 months. Roe and Grant (1964) reported in an abstract that mice (number, sex and strain not specified) did not develop tumors after dermal exposure to 5% phenanthrene (purity not specified, vehicle not specified) 3 times/week for 1 year.

Five studies of cancer-initiating activity in skin painting assays in mice have yielded one positive result. Groups of 30 female CD-1 mice received a single dermal application of 1.8 mg phenanthrene in benzene, followed by twice-weekly applications of tetradecanoylphorbol acetate (TPA, 3 mg), a promoter, for 35 weeks (Scribner, 1973). Phenanthrene used in the study was purified by preparative thin-layer chromatography (TLC) and determined to be homogeneous on TLC. It is stated in the report that the dose of TPA was 3 mg (5 umol); however, it is not clear whether this refers to the twice weekly or total dose. Controls were treated with TPA (6 mg); it is not clear whether controls received benzene (vehicle). The tumor incidence (skin papilloma) at 35 weeks was 12/30 (40%) in treated mice and 0/30 in TPA controls.

Tumor-initiating activity was not shown in the four other mouse skin painting studies. In the first study, male Swiss albino (Ha/ICR) mice (15 to 20/group) received 10 applications of a 0.1% solution of phenanthrene in acetone (total dose 1 mg) or acetone alone, followed by repeated applications of TPA (2.5 ug in acetone) 3 times/week for 20 weeks (LaVoie et al., 1981). Phenanthrene was >99.5% pure as determined by high pressure liquid chromatography (HPLC). No tumors occurred in treated or control mice. Wood et al. (1979) exposed female CD-1 mice (30/group) to a single application of 1.8 mg phenanthrene in acetone:ammonium hydroxide (1000:1) or vehicle alone, followed by TPA (10 ug) twice weekly for 35 weeks. Phenanthrene used in this study was >98% pure and homogeneous on HPLC. Tumor incidence (skin papillomas) out of 27-29 survivors in each group was 17% in treated mice and 7% in vehicle controls (not statistically different). In another study, albino mice (10/sex/dose, strain not specified) received four dermal applications of phenanthrene (total dose 1.2 mg, purity not specified) in acetone or to acetone alone, followed by croton oil once each week for 20 weeks (Roe, 1962). Tumor incidence (skin papillomas) was 4/19 (21%) in treated mice and 2/20 (10%) in vehicle controls. In the last study (Salaman and Roe, 1956), groups of 20 "S" strain mice (sex unspecified) received 10 dermal applications (3 times/week) of 18% phenanthrene (total dose 0.54 g, purity not specified) in acetone, followed by 18 weekly applications of croton oil. Controls were treated with 18 applications of croton oil; 10 controls survived until termination. The tumor incidence (skin papillomas) was 5/20 (25%) in treated mice and 4/10 (40%) in croton oil controls.

Parenterally administered phenanthrene was not shown to have tumorigenic activity in three studies. In the first (Buening et al., 1979), groups of Swiss Webster BLU:Ha ICR mice (100/group, approximately 50% of each sex) received intraperitoneal injections of phenanthrene (total dose 0.25 mg) in dimethyl sulfoxide (DMSO) or DMSO alone on days 1, 8, and 15 after birth. Phenanthrene was >98% pure and homogeneous on HPLC. Incidence of pulmonary tumors (adenomas) at 38 to 42 weeks was 1/18 (6%) and 5/17 (30%) in female and male treated mice and 7/38 (18%) and 2/10 (19%) in female and male controls; the apparent differences were not statistically significant. No hepatic tumors occurred in treated or control mice. One treated female mouse developed malignant lymphoma. In the second study (Grant and Roe, 1963), albino mice (sex, strain and group size not specified) received single subcutaneous injections of phenanthrene (40 ug, purity not specified) in an acetone/gelatin vehicle or only the vehicle. Incidence of pulmonary adenomas after 52-62 weeks was 3/39 (6%) in treated mice and 8/34 (24%) in vehicle controls. Other tumors reported were 4

hepatomas and 2 skin papillomas in treated mice, and 1 mammary adenocarcinoma, 1 hepatoma and 1 hemangioma in control mice. Finally in the Steiner (1955) study, groups of 40 to 50 male and female C57BL mice (numbers per sex not specified) received single subcutaneous injections of 5 mg phenanthrene (purity not specified) in tricaprylin. No tumors were reported in 27 surviving mice after 4 months. Vehicle controls were not reported.

Supporting Data for Carcinogenicity

Phenanthrene has not yielded positive results in assays for DNA damage in Bacillus subtilis and Escherichia coli (Rosenkrantz and Poirier, 1979; McCarroll et al., 1981). Tests for mutagenicity in Salmonella typhimurium have yielded positive (Oesch et al., 1981; Sakai et al., 1985; Bos et al., 1988) and negative results (Wood et al., 1979; McCann et al., 1975; LaVoie et al., 1981; Kaden et al., 1979; Bos et al., 1988). The results of phenanthrene in a fungi recombination assay (Simmon, 1979) and in tests for DNA damage in several mammalian cell cultures were not positive (Lake et al., 1978; Probst et al., 1981; Rice et al., 1984). A test for forward mutation in Chinese hamster ovary cells exposed to 1 ug/mL was not positive (Huberman and Sachs, 1976), whereas a test in human lymphoblast TK6 cells incubated with rat liver S9 (Arochlor) and 9 ug/mL phenanthrene yielded positive results (Barfknecht et al., 1981). Phenanthrene did not yield positive results in sister chromatid exchange and chromosome aberration assays in mammalian cell cultures (Popescu et al., 1977) or in cell transformation assays in several types of mammalian cells (5-40 ug/mL) (Marquardt and Heidelberger, 1972; Kakunaga, 1973; Evans and DiPaolo, 1975; Pienta et al., 1977).

Current theories regarding the mechanisms of metabolic activation of polycyclic aromatic hydrocarbons lead to predictions of a carcinogenic potential for phenanthrene. Jerina et al. (1978) considered phenanthrene to have a "bay-region" structure. It is metabolized by mixed function oxidases to reactive diol epoxides (Nordqvist et al., 1981; Vyas et al., 1982) that have been shown to be weakly mutagenic in some bacterial and mammalian cell assays (Wood et al., 1979). Evidence from in vivo assays indicates, however, that phenanthrene metabolites have a relatively low tumorigenic potential. The 1,2-, 3,4- and 9,10-dihydrodiol metabolites of phenanthrene did not show tumor initiating activity in mouse skin painting assays (Wood et al., 1979). The 1,2-diol-3,4-epoxides of phenanthrene did not produce lung tumors when injected into newborn mice (Buening et al., 1979). The relatively weak mutagenic and tumorigenic activity of phenanthrene diol epoxides is inconsistent with the "bay region theory" of PAH carcinogenesis. The reason for the inconsistency has not been elucidated. Phenanthrene epoxides have a relatively small molecular size (relative to other more active PAH epoxides such as chrysene diol epoxides) and as a result may have a lower affinity for DNA or may be transported less efficiently into the mammalian nucleus (Wood et al., 1979). While some studies have considered phenanthrene to have a "bay-region" structure, it may not clearly fall into this category.

M.27.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

None.

M.27.2.2 Quantitative Estimate of Risk from Inhalation Exposure

None.

M.27.2.3 Carcinogenic Assessment References

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M.28 PYRENE

Status of Data	*Last Revised*
Oral RfD Assessment	07/01/93
Inhalation RfC Assessment	Pending
Carcinogenicity Assessment	01/01/91

M.28.1 NONCARCINOGENIC ASSESSMENT

M.28.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses	UF	MF	RfD
Kidney effects (renal tubular pathology, decreased kidney weights)	NOAEL: 75 mg/kg/day	3000	1	3E-2 mg/kg/day
Mouse Subchronic Oral Bioassay	LOAEL: 125 mg/kg/day			

U.S. EPA, 1989

Principal and Supporting Studies

U.S. EPA. 1989. Mouse Oral Subchronic Toxicity of Pyrene. Study conducted by Toxicity Research Laboratories, Muskegon, MI for the Office of Solid Waste, Washington, DC.

Male and female CD-1 mice (20/sex/group) were gavaged with 0, 75, 125, or 250 mg/kg/day pyrene in corn oil for 13 weeks. The toxicological parameters examined in this study included body weight changes, food consumption, mortality, clinical pathological evaluations of major organs and tissues, and hematology and serum chemistry. Nephropathy, characterized by the presence of multiple foci of renal tubular regeneration, often accompanied by interstitial lymphocytic infiltrates and/or foci of interstitial fibrosis, was present in 4, 1, 1, and 9 male mice in the control, low-, medium-, and high-dose groups, respectively. Similar lesions were seen in 2, 3, 7, and 10 female mice in the 0, 75, 125, and 250 mg/kg treatment groups. The kidney lesions were described as minimal or mild in all dose groups. Relative and absolute kidney weights were reduced in the two higher dosage groups. Based on the results of this study, the low dose (75 mg/kg/day) was considered the NOAEL and 125 mg/kg/day the LOAEL for nephropathy and decreased kidney weights.

Uncertainty and Modifying Factors

UF -- An uncertainty factor of 3000 reflects 10 each for intra- and interspecies variability, 10 for the use of a subchronic study for chronic RfD derivation, and an additional 3 to account for the lack of both toxicity studies in a second species and developmental/reproductive studies.

MF -- None

Additional Comments

White and White (1939) fed six male rats (unspecified strain) a diet containing 2000 mg pyrene/kg for 40 days. The average reported food intake for two animals was 6.1 g/day, and the average body weight for these two animals was 94.3 g. A decrease in body weight gain was observed in two animals. The authors stated that this body weight gain was representative of the whole group; although there was no change in food intake. White and White (1939) also observed enlarged livers and increased hepatic lipid content in animals treated with pyrene, benzpyrene or methylcholanthrene in the diet; however, incidence data were not reported and it is unclear whether this effect occurred in the pyrene treated rats. Interpretation of this study is further complicated by the lack of experimental controls and statistical analysis, small sample size, and incomplete reporting of histopathology results.

Confidence in the Oral RfD

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

Confidence in the principal study is medium, as it was a well-designed experiment that examined a variety of toxicological endpoints and identified both a NOAEL and LOAEL for the critical effect. Confidence in the data base is low, due to the lack of supporting subchronic, chronic, and developmental/reproductive studies. Accordingly, confidence in the RfD is low.

M.28.1.2 Reference Concentration for Chronic Inhalation Exposure

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

M.28.1.3 Noncarcinogenic Assessment References

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M.28.2 CARCINOGENIC ASSESSMENT

Weight-of-Evidence Classification
Classification -- D, not classifiable as to human carcinogenicity

Basis -- Based on no human data and inadequate data from animal bioassays.

Human Carcinogencity Data
None.

Animal Carcinogenicity Data
Inadequate. Groups of 14-29 newborn male and 18-49 newborn female CD-1 mice on 1, 8, and 15 days of age received intraperitoneal injections of pyrene (purity unknown) in dimethyl sulfoxide (DMSO) (total dose = 40, 141 or 466 ug/mouse), or DMSO alone (Wislocki et al., 1986). Tumors were evaluated in animals that died spontaneously after weaning and in all remaining animals at 1 year after exposure. The mid-dose group was initiated 10 weeks after the other groups and had a separate vehicle control. The survival rate in the high-dose groups (male and female) was 25 to 35%; most of the mice died between the last injection and weaning. This high mortality was not observed in the control, low- or mid-dose groups (the survival rates were not stated). A statistically significant increase in the incidence of liver carcinomas occurred in the mid-dose males (3/25) relative to their vehicle control group (0/45), but not in the high-dose males (1/14) or low-dose males (0/29) or in female mice, when compared with their respective controls. The incidences of total liver tumors (adenomas and carcinomas), lung tumors or malignant lymphomas were not statistically significantly elevated in treated animals. The results of this 1-year experiment were not considered to be positive because of the overall lack of tumorigenic response in the short-term.

Mouse skin-painting assays of pyrene as a complete skin carcinogen or as an initiator of carcinogenicity were either not positive or inconclusive (Badger et al., 1940; Horton and Christian, 1974; Van Duuren and Goldschmidt, 1976; Salaman and Roe, 1956; Scribner, 1973).

A subcutaneous pyrene injection did not produce tumors in Jackson A mice; the mice were observed for 18 months after injection (Shear and Leiter, 1941).

Supporting Data for Carcinogenicity
In DNA damage assays in Escherichia coli and Bacillus subtilis pyrene was not mutagenic (Ashby and Kilbey, 1981). In bacterial gene mutation tests both positive (Kinae et al., 1981; Bridges et al., 1981; Matijasevic and Zeiger, 1985; Sakai et al., 1985; Kaden et al., 1979; Bos et al., 1988) and negative (McCann et al., 1975; LaVoie et al., 1979; Ho et al., 1981; Bos et al., 1988) results have been reported. The consensus conclusion on the international collaborative study (which involved 20 bacterial test sets) was that protocol or evaluation criteria were critical factors in individual test verdicts. Pyrene induced increased incidence of mitotic gene conversion but not other genetic endpoints in yeast (de Serres and Hoffman,

1981). Pyrene did not induce an increase in sex-linked recessive lethals in Drosophila (Valencia and Houtchens, 1981).

Mixed results have also been observed in mammalian assays in vitro, again with protocol and evaluation criteria being a factor in at least some of the cases. In the collaborative study Evans and Mitchell (1981) concluded pyrene was positive for SCE induction in CHO cells when all concentrations were different from controls, but no apparent increase when the concentration was increased 10-fold. In the same volume, two other laboratories reported pyrene negative both for SCE and for chromosome aberrations in CHO cells (Brookes and Preston, 1981). Tong et al. (1981) also reported that pyrene did not induce SCE in a rat liver epithelial cell system. Jotz and Mitchell (1981) reported pyrene was positive in the L5178Y mouse lymphoma gene mutation assay.

Pyrene did not induce chromosome aberrations (as detected by micronuclei) or SCE in bone marrow of several mouse strains receiving i.p. injections of pyrene (Purchase and Ray, 1981). Results of mammalian cell transformation assays in a variety of cell types have not been positive (DiPaolo et al., 1969; Pienta et al., 1977; Casto, 1979; Chen and Heidelberger, 1969; DiPaolo et al., 1972; Kakunaga, 1973; Evans and DiPaolo, 1975).

M.28.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

None.

M.28.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

None.

M.28.2.3 Carcinogenic Assessment References

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M.31 THALLIUM(I) SULFATE

Status of Data	*Last Revised*
Oral RfD Assessment	09/01/90
Inhalation RfC Assessment	no data
Carcinogenicity Assessment	09/01/90

M.31.1 NONCARCINOGENIC ASSESSMENT

M.31.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses	UF	MF	RfD
No adverse effects	NOAEL: 0.25 mg/kg/day	3000	1	8E-5 mg/kg/day
Rat Oral Subchronic Study	LOAEL: None			

U.S. EPA, 1986

Prinicipal and Supporting Studies

U.S. EPA. 1986. Subchronic (90-day) toxicity of thallium sulfate in Sprague-Dawley rats. Office of Solid Waste, Washington, DC.

In a 90-day subchronic study, Sprague-Dawley rats (20/sex/group) were treated by gavage with 0, 0.01, 0.05, and 0.25 mg/kg/day of an aqueous solution of thallium sulfate (approximately 0.008, 0.04, and 0.20 mg Tl/kg/day). Data generated from this study included body and organ weights, food consumption, hematology and clinical chemistry parameters, neurotoxicologic examinations, ophthalmologic examinations, and histopathology and neuropathology. No mortality was observed, but apparent dose-related increases in the incidence of alopecia, lacrimation, and exophthalmos were observed throughout the study. No differences between the control groups and groups receiving thallium sulfate were observed in body weights, body weight gains, food consumption, or absolute and relative organ weights. Moderate dose-related changes were observed in some blood chemistry parameters: increased SGOT, LDH, and sodium levels, and decreased blood sugar levels. The only grossly observed finding at necropsy thought to be treatment-related was alopecia, especially in female rats; however, microscopic evaluations did not reveal any histopathologic alterations. Based on the results of this study the 0.25 mg/kg/day thallium sulfate is considered a NOAEL. By applying an uncertainty factor of 3000 to this NOAEL, an RfD of 8E-5 mg/kg/day can be derived.

Uncertainty and Modifying Factors

UF -- The UF of 3000 includes factors of 10 to extrapolate from subchronic to chronic data, 10 for intraspecies extrapolation and 10 to account for interspecies variability, and a factor of 3 to account for lack of reproductive and chronic toxicity data.

MF -- None

Additional Comments

Groups of rats (5/sex/dose) were fed diets containing nominal concentrations of thallium acetate of 0, 5, 15, or 50 ppm (Downs et al., 1960). An additional group (30 ppm) was added after the study had been initiated (time not specified). Animals were allowed ad lib access to these diets for 15 weeks. The 50 ppm level resulted in 100% mortality by week 5. The 30 ppm level resulted in 100% mortality by week 9. By week 15, 4/10 control animals died (2/sex), making interpretation of survival in remaining dose groups difficult (15 ppm, 3/5 males and 1/5 females died; 5 ppm, 2/6 males and 0/4 females died). At termination, the only gross finding was alopecia in the 15 and 30 ppm groups. The authors stated that there was a slight increase in kidney weight (doses not specified, data not shown). The authors reported that histopathologic evaluations did not indicate treatment-related pathology.

Male Wistar rats (10/group) were administered drinking water containing 10 ppm T1SO4 (approximately 0.7 mg T1/kg/day based on reported thallium consumptions [270 ug T1/rat] and body weights [350-370 g] for 30 or 60 days; controls were pair fed. After 60 days of treatment, the following testicular effects were observed: disarrangement of the tubular epithelium, cytoplasmic vacuolation and distention of the smooth endoplasmic reticulum of the Sertoli cells, reduced testicular beta-glucuronidase activities, high concentrations of thallium in the testes, and reduced sperm motility (Formigli et al., 1986).

Eighty female Sprague-Dawley rats were administered drinking water containing thallium sulfate at a concentration of 10 mg Tl/L (approximately equivalent to a dose of 1.4 mg T1/kg/day based on reported T1 intakes and an assumption that the rats weighed 200 g). Mortality was 15 and 21% after 40 and 240 days of treatment, respectively. Functional and histopathological changes were observed in the peripheral nerves including changes in motor and sensory action potentials and histopathological changes in the sciatic myelin sheath and axonal destruction characterized by Wallerian degeneration, mitochondrial degeneration, neurofilamentous clustering, and elevated lysozomal activity (Manzo et al., 1983).

Confidence in the Oral RfD

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

Confidence in the critical study is rated low because of uncertainties in the results (i.e., vehicle vs. control differences) and because supporting studies show adverse health effects at doses slightly higher than the NOAEL. The data base provided only one subchronic study and some anecdotal human data, thus, a low confidence was assigned. Until additional chronic and reproductive studies are available, confidence in the RfD is considered low.

M.31.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.31.1.3 Noncarcinogenic Assessment References

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M.31.2 CARCINOGENIC ASSESSMENT

Weight-of-Evidence Classification
Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on the lack of carcinogenicity data in animals and humans.

Human Caricinogenicity Data
Inadequate. Medical records for 86 workers (sex and length of employment not reported) occupationally exposed to thallium at a magnesium seawater battery factory and 79 unexposed workers matched for age, length of employment, shift pattern, and type of work were examined (Marcus, 1985). No increase in incidence of benign neoplasms (site not specified) were observed. This study is limited by the examination of medical records only, lack of exposure quantitation, the small cohort, and unknown length of observation.

In another study, the health effects associated with exposure to thallium in 128 men (age 16 to 62 years) exposed for 1 to 42 years (average=19.5 years) in three cement manufacturing plants were reported (Schaller et al., 1980). Analyses of roasted pyrites and electro-filter dust confirmed the presence of thallium in various production areas in the plants. Urinary thallium was elevated in the workers. The health evaluation, consisting of a medical history and a physical exam, did not show any indication of thallium poisoning. However, this health evaluation was not adequate to detect any oncogenic response.

Animal Carcinogenicity Data

None. Several subchronic and chronic animal studies on thallium and compounds are available; however, they were not designed to examine carcinogenic endpoints (reviewed in U.S. EPA, 1988).

Supporting Data for Carcinogenicity

Thallium (I) salts were not mutagenic in reverse mutation assays using Salmonella typhimurium strains TA98, TA100, TA1535,and TA1538 and Escherichia coli strains B/r WP2 try and WP2 hcr try; use of hepatic homogenates was not specified (Kanematsu et al., 1980). Positive results were obtained at 0.001M for thallium nitrate in the Rec assay using Bacillus subtilis strains H17 and M45; use of hepatic homogenates was not specified (Kanematsu\et\al., 1980; Kada et al., 1980). Negative results were obtained in a screening of thallium nitrate for induction of mitotic gene coversion and reverse mutation in the yeast, Saccharomyces cerevisiae (Singh, 1983). Thallium nitrate produced negative effects on cell division in S. cerevisiae and E. coli. (Loveless et al., 1954). Cytotoxic levels (1000 ug/mL) of thallium acetate caused depressed DNA synthesis in Chinese hamster ovary cells (Garrett and Lewtas, 1983). Single-strand DNA breaks occurred in mouse and rat embryo fibroblasts exposed to thallium carbonate at E-6 to E-4M (Zasukhina et al., 1983). Thallium carbonate (0.5-0.005 ug/kg/day) was positive in a dominant lethal test in male white rats (Zasukhina et al., 1983).

M.31.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

None.

M.31.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

None.

M.31.2.3 Carcinogenic Assessment References

Garrett, N.E. and J. Lewtas. 1983. Cellular toxicity in Chinese hamster ovary cell cultures. I. Analysis of cytotoxicity endpoints for twenty-nine priority pollutants. Environ. Res. 32: 455-465.

Kada, T., K. Hirano and Y. Shirasu. 1980. Screening of environmental chemical mutagens by the Rec-assay system with Bacillus subtilis. In: Chemical Mutagens: Principles and Methods for Their Detection, F. deSerrres and A. Hollaender, Ed. 6: 149-173.

Kanematsu, N., M. Hara and T. Kada. 1980. Rec assay and mutagenicity studies on metal compounds. Mutat. Res. 77: 109-116.

Loveless, L.E., E. Spoerl and T.H. Weisman. 1954. A survey of effects of chemicals on division and growth of yeast and Escherichia coli. J. Bacteriol. 68: 637-644.

Marcus, R.L. 1985. Investigation of a working population exposed to thallium. J. Soc. Occup. Med. 35(1): 4-9.

Schaller, K.H., G. Manke, H.J. Raithel, G. Buhlmeyer, M. Schmidt and H. Valentin. 1980. Investigations of thallium-exposed workers in cement factories. Int. Arch. Occup. Environ. Health. 47(3): 223-231.

Singh, I. 1983. Induction of reverse mutation and mitotic gene conversion by some metal compounds in Saccharomyces cerevisiae. Mutat. Res. 117: 149-152.

U.S. EPA. 1988. Health and Environmental Effects Document for Thallium and Compounds. Prepared by the Environmental Criteria and Assessment Office, Cincinnati, OH, for the Office of Solid Waste and Emergency Response, Washington, DC.

Zasukhina, G.D., I.M. Vasilyeva, N.I. Sdirkova, et al. 1983. Mutagenic effect of thallium and mercury salts on rodent cells with different repair activities. Mutat. Res. 124(2): 163-173.

M.32 1,3,5-TRINITROBENZENE

Status of Data	*Last Revised*
Oral RfD Assessment	02/01/95
Inhalation RfC Assessment	No data
Carcinogenicity Assessment	10/01/91

M.32.1 NONCARCINOGENIC ASSESSMENT

M.32.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD
Increased splenic weight mg/kg/day	NOAEL: 3 ppm m-dinitrobenzene in drinking water, converted to 0.51 mg/kg/day 1,3,5-trinitrobenzene	10,000	1	5E-5

Rat Subchronic Oral LOAEL: 8 ppm
Study m-dinitrobenzene

Cody et al., 1981

*Conversion Factors: Authors calculated that 3 ppm m-dinitrobenzene wasequivalent to 0.4 mg/kg/day intake in males; molecular weight ratio of1,3,5-TNB to m-DNB is 214/168; thus, 0.40 mg/kg/day x (214/168) = 0.51mg/kg/day

Principal and Supporting Studies

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.

Data on the toxicity of 1,3,5-trinitrobenzene are insufficient for derivation of an RfD. Therefore, it is necessary to derive an RfD by analogy to the structurally similar 1,3-dinitrobenzene (m-dinitrobenzene). The LD50 of m-dinitrobenzene in rats is 83 mg/kg and the LD50 of 1,3,5-trinitrobenzene in rats is 450 mg/kg (RTECS, 1983), indicating that the additional nitro group results in reduced toxicity. Therefore, it is appropriate to derive an RfD for 1,3,5-trinitrobenzene based on the RfD for m-dinitrobenzene. Carworth Farm rats (20/sex/group) were exposed to 0, 3, 8, or 20 ppm m-dinitrobenzene in drinking water for 16 weeks. The concentration of 20 ppm decreased body weight gain in females, decreased hemoglobin concentrations, caused testicular atrophy in males and was associated with splenic enlargement with 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. In an independent study, Cody et al. (198l) treated

male rats with 0, 3 or 8 ppm m-dinitrobenzene in drinking water for 90 days. At both m-dinitrobenzene exposure levels, rats showed significant increases in activity on the running wheel. This change in activity did not appear to be adverse. The concentration of 3 ppm is thus a NOAEL and 8 ppm is a LOAEL.

Based on water consumption and body weight data, Cody et al. (1981) calculated that the 3 ppm level corresponded to a mean daily intake of 0.40 mg/kg in males. When adjusted for molecular weight differences, the corresponding equivalent intake would be 0.51 mg/kg/day 1,3,5-trinitrobenzene.

Uncertainty and Modifying Factors
UF -- 10 for extrapolation from subchronic to chronic exposure, 10 for interspecies extrapolation, 10 for sensitive members of the human population, and 10 for the derivation of an RfD by analogy to a stucturally similar dinitrobenzene.

MF -- None

Additional Comments
Korolev et al. (1977) administered 1,3,5-trinitrobenzene orally to rats, mice, and guinea pigs and found moderately toxic effects in the blood, liver, and CNS. The investigators estimated the maximum permissible limit in surface water as 0.4 mg/L, or 0.8 mg/day, assuming a 70 kg human drinks 2 L of water daily. Further details of this Russian study were not provided in the available abstract.

Confidence in the Oral RfD
Study -- Medium
Data Base -- Low
RfD -- Low

Confidence in the study is medium because both a NOAEL and a LOAEL were identified, adequate numbers of animals were treated by an appropriate route (drinking water), and suitable endpoints were investigated. The lack of toxicological or pharmacokinetic data on 1,3,5-trinitrobenzene itself, however, makes confidence in the data base and RfD low.

M.32.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.32.1.3 Noncarcinogenic Assessment References

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.

Korolev, A.A., T.V. Voitsekhovakaya, M.V. Bogdanov, M.V. Arsen'eva, and T.A. Zakharova. 1977. Experimental data for hygienic standardizzation of dinitrotoluene and trinitrobenzene in reservoir waters. Gig. Sanit. 10: 17-20. (Rus.)

RTECS (Registry of Toxic Effects of Chemical Substances). 1983. Online.

U.S. EPA. 1985. Health and Environmental Effects Profile forDinitrobenzenes (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.

M.32.2 CARCINOGENIC ASSESSMENT

This substance/agent has been evaluated by the U.S. EPA for evidence of human carcinogenic potential. This does not imply that this agent is necessarily a carcinogen. The evaluation for this chemical is under review by an inter-office Agency work group. A risk assessment summary will be included on IRIS when the review has been completed.

M.33 2,4,6-TRINITROTOLUENE

Status of Data	*Last Revised*
Oral RfD Assessment	02/01/93
Inhalation RfC Assessment	No data
Carcinogenicity Assessment	07/01/93

M.33.1 NONCARCINOGENIC ASSESSMENT

M.33.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

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)

Principal and Supporting Studies

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. 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.

Uncertainty and Modifying Factors

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

Additional Comments

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.

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.

M.33.1.2 Reference Concentration for Chronic Inhalation Exposure

Not available at this time.

M.33.1.3 Noncarcinogenic Assessment 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)

M.33.2 CARCINOGENIC ASSESSMENT

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.

Human Carcinogenicity Data
None. There are extensive human toxicity data, but they are not useful in the evaluation of carcinogenicity.

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.

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).

M.33.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

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

Dose-Response Data
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

Additional Comments
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.

Discussion of Confidence
Both rat and mouse studies (U.S. DOD, 1984a,b), were well conducted with appropriate number of animals per sex per dose.

M.33.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Not available.

M.33.2.3 Carcinogenic 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.

M.34 ZINC

Status of Data	*Last Revised*
Oral RfD Assessment	10/01/92
Inhalation RfC Assessment	no data
Carcinogenicity Assessment	02/01/91

M.34.1 NONCARCINOGENIC ASSESSMENT

M.34.1.1 Noncarcinogenic Reference Dose for Chronic Oral Exposure

Critical Effect	Experimental Doses*	UF	MF	RfD
47% Decrease in erythrocyte superoxide dismutase (ESOD) concentration in adult females after 10 weeks of zinc exposure Human Diet Supplement Study Yadrick et al., 1989	NOAEL: None LOAEL = 59.72 mg/day (1.0 mg/kg/day)	3	1	3E-1 mg/kg/day

*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).

Principal and Supporting Studies

Yadrick, M.K., M.A. Kenney and E.A. Winterfeldt. 1989. Iron, copper, and zinc status: Response to supplementation with zinc or zinc and iron in adult females. Am. J. Clin. Nutr. 49: 145-150.

The oral RfD is based on a clinical study which investigated the effects of oral zinc supplements on copper and iron balance. This study is supported by several other studies which indicate that zinc supplementation can alter copper balance. The effects on copper and iron biochemistry are considered of concern since long-term iron or copper deficiency could result in significant adverse effects. For example, zinc supplementation therapy with megadoses of up to 5 g/day, as well as smaller amounts of 150 mg/day, taken for 1 to 2 years have produced copper deficiency anemia (Fischer et al., 1984). In addition, several studies have investigated the effects of zinc supplementation on the high-density lipoprotein (HDL) levels of adult males.

These have been added as supporting studies because the observed change in HDL values in males may be significant since a sustained decrease in HDL concentrations may be associated with increased risk of coronary artery disease when combined with a parallel increase in low-density lipoprotein (LDL) cholesterol.

A 10-week study of zinc supplementation in 18 healthy women given zinc gluconate supplements twice daily (50 mg zinc/day, or 1.0 mg/kg-day, see below) resulted in a decrease of erythrocyte superoxide dismutase (ESOD) activity (Yadrick et al., 1989). ESOD concentrations declined over the 10-week supplementation period and at 10 weeks were significantly different ($p < 0.05$) from values during the pretreatment period. By 10 weeks, ESOD activity had declined to 53% of pretreatment levels. Change in enzyme activity is considered a better indicator of altered copper status than a measure of metal concentration in tissue or plasma. This has been documented by studies in rats fed copper-deficient or high-zinc diets, in which copper metalloenzyme activity is greater and precedes changes in plasma or tissue levels of copper (L'Abbe and Fischer, 1984a,b). Ceruloplasmin concentrations were not altered. Serum zinc was significantly increased. There was also a significant decline in serum ferritin and hematocrit values at 10 weeks. Such a decrease could pose a significant risk to the iron status of women.

No measurements were made of dietary zinc or copper in this study. However, a level of dietary zinc can be estimated at 9.72 mg/day for females (20- to 30-years old) from the results of the FDA Total Diet Study for 1982-1986 (Pennington et al., 1989). The LOAEL of 1.0 mg/kg-day was calculated from the sum of these dietary estimates and the supplemental zinc dose using an assumed body weight of 60 kg for adult females, as shown in the conversion factor section.

Support for considering the intake of 50 mg/kg-day supplemental zinc as a threshold LOAEL is provided by Fischer et al. (1984) which also suggests that zinc affects copper balance at doses of 0.95 mg/kg-day in males. Healthy men given 25 mg of zinc as gluconate twice daily for a 6-week period displayed a significant decrease ($p < 0.05$) in erythrocyte superoxide dismutase (ESOD) activity at the end of 6 weeks exposure. There were no differences between serum copper levels or ceruloplasmin activity in the 13 members of the supplement group compared with controls. Serum zinc levels were significantly increased in the supplement group after 2 weeks.

Prasad et al. (1978) fed a patient with sickle cell anemia supplements of 150 to 200 mg zinc/day for 2 years. The supplement resulted in copper deficiency; serum copper and plasma ceruloplasmin levels were decreased. When copper was administered, the plasma ceruloplasmin levels became normal. In a follow-up study, of 13 patients on zinc therapy (similar treatment levels assumed), 7 patients had ceruloplasmin levels at the lower limit of normal after 24 weeks of dosing.

In a 9-week study, Festa et al. (1985) fed nine male students diets containing 2.6 mg copper/day and 1.8-20.7 mg zinc/day for 1- to 2-week periods. This study indicated that fecal copper excretion was influenced by the amount of zinc in the diet and the length of time it was

administered. Typically, after 1-2 weeks at 18.5 mg/day (just 3.5 mg/day higher than the adult RDA), subjects lost significantly more copper in the feces. Plasma copper concentrations were unchanged.

Groups of 9, 13 or 9 healthy white men were administered 0, 50, or 75 mg/kg-day zinc as zinc gluconate, respectively, for 12 weeks (Black et al., 1988). The subjects were given instructions to avoid foods high in calcium, fiber and phytic acid, dietary constituents that have a negative impact on zinc absorption. Subjects were also told to restrict their intake of zinc-rich foods in order to minimize the variation in daily dietary zinc. Three-day dietary records were collected on a biweekly basis. These records indicated that the dietary zinc intakes of the three treatment groups were 12.5, 14.0, and 9.5 mg/day for the groups receiving 0, 50, and 75 mg/kg-day supplement, respectively. Based on the average body weights for each treatment group, these doses correspond to a total zinc intake of 0.16, 0.85, and 1.10 mg/kg-day.

Biweekly blood samples were collected from all subjects and analyzed for total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, zinc, and copper. Urinary zinc and copper values were also determined. There was a general decline in the mean serum HDL-cholesterol for the 75-mg supplement group between weeks 6 and 12. HDL values for this group were significantly lower than those for the placebo group at weeks 6 and 12 ($p < 0.05$). When the mean HDL-cholesterol level of these subjects was compared to population percentile norms, there was a decline from the 92nd to the 77th percentile (Simko et al., 1984) in 6 weeks, followed by a relative stabilization of HDL values for the remaining 6-week test period. There was also a decline in the HDL values for the 50-mg group between weeks 8 through 12; however, this decline was not significantly different ($p \leq 0.05$) 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.

Uncertainty and Modifying Factors

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

Additional Studies/Comments

Zinc is an essential nutrient with RDA values ranging from 5 to 15 mg/day for different age and sex categories (NRC, 1989). The RDA is an estimate of the zinc needed for growth, development, metabolism and tissue maintenance for over 98% of the healthy American

population. For 79% of a 70-year lifetime (55 years), the proposed RfD of 0.3 mg/kg-day supplies adequate zinc to meet these requirements in adolescents and adults without any concurrent physiological impairment. It does not supply the RDA for infants, preadolescent children or, possibly, for lactating women.

The RfD of 0.3 mg/kg-day is expected to be without adverse effects when consumed on a daily basis over an extended period of time. It neither induces a nutritional deficiency in healthy, non-pregnant, adult humans consuming the average American diet nor causes undesirable inhibition of normal lipid transport.

When the three studies monitoring HDL-cholesterol are considered as a group, they show a consistent lowering of HDL-cholesterol levels in response to the addition of zinc to the diet, an effect which is reversed with cessation of the zinc supplementation. The data of Black et al. (1988) indicate that the depressed HDL values can persist for up to 12 weeks. Data are available from all 3 studies at 6 weeks. However, in the Hooper et al. (1980) study, the 6-week data represent HDL status 1 week after supplement administration ended. Additional data will be needed to clarify whether or not this change is significant with longer exposure.

Supplemental zinc does not appear to have the same effect on females that it has on males. Healthy adult females were given supplemental zinc doses of 0, 15, 50 or 100 mg/day zinc as zinc acetate for 60 days (Freeland-Graves et al., 1982). Plasma cholesterol, HDL-cholesterol, and zinc were monitored at biweekly intervals. A transitory decrease in HDL values was noted at 4 weeks, but only in the group receiving the 100-mg/day supplement (1.8 mg/kg-day based on a 60-kg body weight and 8.1 mg/day zinc in the diet [from diet records]). This decrease in HDL values was not apparent at 6 and 8 weeks. Serum zinc levels were also highest in these subjects at 4 weeks.

A very slight but statistically significant ($p = 0.04$) 2-mg/dL increase in HDL cholesterol was seen in a group of 22 elderly male and female subjects (sex ratio unknown) 8 weeks after they ceased using zinc supplements (Goodwin et al., 1985). Serum zinc values fell from 92 to 86 μg/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.

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.

M.34.1.2 Reference Concentration for Chronic Inhalation Expsoure

Not available at this time.

M.34.1.3 Noncarcinogenic Assessment References

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M.34.2 CARCINOGENIC ASSESSMENT

Weight-of-Evidence Classification
Classification -- D; not classifiable as to human carcinogenicity

Basis -- Based on inadequate evidence in humans and animals.

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.

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.

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.

M.34.2.1 Quantitative Estimate of Carcinogenic Risk from Oral Exposure

None.

M.34.2.2 Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

None.

M.34.2.3 Carcinogenic Assessment References

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APPENDIX N

AIR DISPERSION MODELING

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1.0 INTRODUCTION

A screening-level air dispersion modeling analysis of inhalable particulate emissions from contaminated sites at Tooele Army Depot-North Area (TEAD-N) has been performed to support the health risk assessment for the facility. The health risk assessment focuses on future, long-term health effects caused by working at or living near the TEAD-N facility. Results of the air dispersion modeling analysis correspond to the exposure to airborne contaminants through inhalation.

Two air quality impact scenarios were evaluated: (1) impacts associated with construction activity at each SWMU, and (2) impacts associated with natural wind erosion at each SWMU. Each required a separate air quality estimation approach. In each case, air quality impacts were estimated and reported for each SWMU separately.

The contaminants, heavy metals and organic compounds, considered in the analysis were assumed to be soil-bound (i.e., small amounts of contaminant adsorbed to the surface of soil particles) and emitted to the atmosphere as particulate matter. Only the inhalable fraction of these particulates (< 10 microns in diameter or PM_{10}) was evaluated. This particulate matter could be entrained into the atmosphere by the construction activity and natural wind erosion.

Because of the long period of time that these contaminants have been in the soil, volatilization of the organic compounds was assumed to be negligible. Therefore, gaseous emissions were not considered.

The PM_{10} emission rates for each scenario and the fraction of each contaminant in the soil were used to estimate air quality impacts of the individual contaminants. The assumption here is that the contaminant is uniformly mixed within the soil. Subsurface soil concentrations were used with the construction activity scenario and surface soil concentrations were used with the natural wind erosion scenario.

2.0 SITE DESCRIPTION

TEAD-N is located in the Tooele Valley of northern Utah and covers approximately 100 square kilometers (km). It is surrounded by three small towns located just beyond the facility boundaries–Grantsville (0.8 km to the north), Tooele (adjacent to TEAD-N to the east), and Stockton (3.4 km to the south). The terrain within TEAD-N boundaries is generally uniform

with a moderate slope toward the north. The facility is surrounded by rugged terrain to the south, west, and east.

The contaminated sites at TEAD-N are referred to as solid waste management units (SWMUs). Of the SWMUs at TEAD-N, only eight were identified for dispersion modeling through a preliminary health risk screening analysis based on contaminant concentrations in the soil. The

eight SWMUs considered in this analysis are listed in Table 1. Refer to the main body of this report for a location map of these SWMUs at TEAD-N.

The SWMUs were treated as ground-level area sources in the air dispersion modeling analysis.

Table 1. SWMUs Considered in the Air Dispersion Modeling Analysis

SWMU	Total Area (acres)	Paved (acres)	Uncontaminated (acres)	Total Contaminated Area (acres)
6	40.23	-	-	40.23
7	391.20	-	-	391.20
8	10.41	-	-	10.41
22	0.34	0.06	-	0.28
23	5.73	1.33	-	4.40
32	0.05	-	-	0.05
35	42.50	-	10.60	31.90
40	59.48	-	-	59.48

3.0 EMISSION RATE ESTIMATES

3.1 INHALABLE PARTICULATE MATTER (PM_{10})

PM10 emission rates were estimated for two scenarios: construction activity and natural wind erosion. In both cases, estimation methods were taken from the U.S. Environmental Protection Agency's *Compilation of Air Pollutant Emission Factors; Volume I-Stationary Point and Area Sources* (AP-42).

3.1.1 Construction Activity

AP-42 Section 11.2.4.3 suggests a particulate matter emission factor from construction activity as 1.2 tons per acre of construction per month. This value applies to construction with medium activity level, moderate silt level, and in a semi-arid climate. Since this is the only emission factor available for general construction activity, it is assumed that the TEAD-N facility meets these characteristics.

This emission factor is representative of total suspended particulates (<30 microns in diameter). This emission factor was conservatively equated to the PM_{10} emission factor since no scaling factor was available in AP-42 for the PM_{10} fraction.

Since the SWMUs were treated as area sources in the air dispersion modeling, this emission factor was converted to an area source emission rate. For area sources, the air quality model required an emission rate with units of grams per second per square meter ($g/s/m^2$). In this case, the PM_{10} emission rate is:

(Equation 1)

$$PM_{10}\ Emission\ Rate = 1.2\frac{ton}{month \cdot acre} \times \frac{2000\ lb}{ton} \times \frac{month}{30.4\ days} \times \frac{acre}{43560\ ft^2}$$

$$= 0.00181\ \frac{lb}{day \cdot ft^2} \times \frac{453.6\ g}{lb} \times \frac{day}{86400\ \text{sec}} \times \frac{10.764\ ft^2}{m^2}$$

$$= 0.00010\ \frac{g}{\text{sec} \cdot m^2}.$$

3.1.2 Natural Wind Erosion

AP-42 Section 11.2.7.3 suggests an emission factor for wind generated particulate emissions of erodible and non-erodible surface material subject to disturbance. The emission factor is expressed as:

(Equation 2)

$$PM_{10}\ Emission\ Factor = k \times N \times EP$$

$$= 40.93\ \frac{g}{m^2 \cdot year}$$

where

k	=	Particle size multiplier (0.5 for PM_{10})
N	=	Number of disturbances, such as tilling or grading, to the erodible surface per year (1 disturbance is conservatively assumed for TEAD-N for natural wind erosion of an undisturbed area)
EP	=	Erosion potential in g/m^2 for a dry, exposed surface

The erosion potential (EP) is estimated as:

(Equation 3)

$$EP = [58 \times (u^* - u^*(t))^2] + [25 \times (u^* - u^*(t)]$$

$$= 81.87 \frac{g}{m^2}$$

where

u* = Friction velocity in m/sec
u*(t) = Threshold friction velocity in m/sec (assumed to be 0.43 m/sec which is the most conservative value in AP-42 Table 11.2.7-1)

The friction velocity (u*) is estimated as:

(Equation 4)

$$u* = 0.053 \times u^+_{10}$$

$$= 1.4 \frac{m}{\text{sec}}$$

where

u^+_{10} = Fastest mile in m/sec (60 mi/hr or 26.8 m/sec is the average for the National Weather Service observation site at Salt Lake City, Utah according to information in *Local Climatological Data, Part IV-Western Region* (U.S. Dept. of Commerce, NOAA, National Climactic Data Center)).

The PM_{10} emission factor of 40.93 g/m^2 per year was converted to an area source emission rate (for dispersion modeling) with units of g/s/m^2. For the dispersion modeling to yield the most conservative results, it was assumed that all erodible PM_{10} was entrained into the atmosphere during a one-hour period. Therefore, the per second emission rate is:

(Equation 5)

$$\underset{Rate}{\overset{PM_{10}\ Emission}{}} = 40.93 \frac{g}{m^2 \cdot hour} \times \frac{hour}{3600\ \text{sec}}$$

$$= 0.01137 \frac{g}{m^2 \cdot \text{sec}}.$$

3.2 CONTAMINANTS

Since it was assumed that the contaminants are uniformly mixed within the soil, the emission rate of each contaminant considered in the analysis was estimated by scaling the PM_{10} emission rate by the contaminant fraction in the soil. Subsurface soil concentrations were used for the construction activity scenario. Surface soil concentrations were used for the natural wind erosion scenario.

3.2.1 Subsurface Soil Concentrations (for Construction Activity)

Subsurface soil concentrations are presented in Table 2. Both central tendency exposure (CTE) and reasonable maximum exposure (RME) values were used in the construction activity analysis.

3.2.2 Surface Soil Concentrations (for Natural Wind Erosion)

Surface soil concentrations are presented in Table 3. Both CTE and RME values were used in the natural wind erosion analysis.

4.0 DISPERSION MODELING APPROACH

Two air quality impact scenarios were evaluated: (1) construction activity at each SWMU, and (2) natural wind erosion at each SWMU. The same dispersion modeling approach was used for both scenarios to estimate the worst-case (i.e., maximum, normalized 1-hour) impacts. However, separate approaches were used to convert the maximum 1-hour impacts to annual impacts. In each case, air quality impacts were estimated and reported for each SWMU separately.

The construction activity scenario assumed continuous construction activity and continuous PM_{10} emissions (i.e., 8,760 hours per year). All emissions were conservatively assumed to occur during worst-case dispersion. Therefore, the annual impacts were equal to the maximum 1-hour impacts.

Table 2. Subsurface Soil Concentrations for the Construction Activity Scenario

SWMU	Area of Concern	COPC	Concentration (mg/kg)		Fraction	
			CTE	RME	CTE	RME
6	Northeast Revetment Area	Aluminum	13,894	13,894	1.39E-02	1.39E-02
		Antimony	22.4	22.4	2.24E-05	2.24E-05
		Arsenic	22.3	22.3	2.23E-05	2.23E-05
		Chromium	46.3	46.3	4.63E-05	4.63E-05
		Copper	593	593	5.93E-04	5.93E-04
		Iron	53,680	53,680	5.37E-02	5.37E-02
		Lead	262	262	2.62E-04	2.62E-04
		Thallium	46.4	46.4	4.64E-05	4.64E-05
		Zinc	5,169	5,169	5.17E-03	5.17E-03
	Hot Spot at Test Pit 3	1,3,5-Trinitrobenzene	17	17	1.70E-05	1.70E-05
7	Firing Point	Arsenic	16.7	16.7	1.67E-05	1.67E-05
	Northwest Test Area Trench	Aluminum	42,400	42,400	4.24E-02	4.24E-02
		Beryllium	1.79	1.79	1.79E-06	1.79E-06
		Thallium	41.6	41.6	4.16E-05	4.16E-05
8	Bullet Stop	Arsenic	17.6	17.6	1.76E-05	1.76E-05
		Zinc	9,900	9,900	9.90E-03	9.90E-03
		Lead	172	172	1.72E-04	1.72E-04
	Firing Lines	Chromium	40.2	40.2	4.02E-05	4.02E-05
13	---	Diethyl Phthalate	0.71	4.43	7.10E-07	4.43E-06

Table 2. Subsurface Soil Concentrations for the Construction Activity Scenario (continued)

SWMU	Area of Concern	COPC	Concentration (mg/kg)		Fraction	
			CTE	RME	CTE	RME
22	---	2,4,6-Trinitrotoluene	65.7	65.7	6.57E-05	6.57E-05
		Chromium	56.9	56.9	5.69E-05	5.69E-05
23	Building 1343 Outfall	Chromium	54.6	54.6	5.46E-05	5.46E-05
	Outfall Near Building 1344	Chromium	537	537	5.37E-04	5.37E-04
		PAHs	0.057	0.057	5.70E-08	5.70E-08
	Building 1345 Outfall	Chromium	66.6	66.6	6.66E-05	6.66E-05
		PCBs	0.16	0.16	1.60E-07	1.60E-07
	Asphalt and Stained Area	Chromium	116	116	1.16E-04	1.16E-04
		PAHs	0.12	0.12	1.20E-07	1.20E-07
31	---	none				
32	---	Chromium	54	54	5.40E-05	5.40E-05
35		none				
36		none				
40	Remainder of SWMU	Arsenic	7.57	7.57	7.57E-06	7.57E-06

Table 3. Surface Soil Concentrations for the Natural Wind Erosion Scenario

SWMU	Area of Concern	COPC	Concentration (mg/kg)		Fraction	
			CTE	RME	CTE	RME
6	SWMU as a Whole	Arsenic	15	15	1.50E-05	1.50E-05
		Copper	77.8	77.8	7.78E-05	7.78E-05
		Lead	90.4	90.4	9.04E-05	9.04E-05
7	SWMU as a Whole	Aluminum	20,524	20,524	2.05E-02	2.05E-02
		Beryllium	1	1	1.0E-06	1.0E-06
		Manganese	732	732	7.32E-04	7.32E-04
		Thallium	30.8	30.8	3.08E-05	3.08E-05
8	SWMU as a Whole	Aluminum	17,438	17,438	1.74E-02	1.74E-02
		Antimony	31.5	31.5	3.15E-05	3.15E-05
		Arsenic	8.7	8.7	8.70E-06	8.70E-06
		Copper	52.3	52.3	5.23E-05	5.23E-05
		Lead	537	537	5.37E-04	5.37E-04
13	SWMU as a Whole	Chloromethane *(on-site adult resident)*	0.0018	0.0019	1.80E-09	1.90E-09
		Chloromethane *(on-site child resident)*	0.0018	0.0032	1.80E-09	3.20E-09
		Chloromethane *(on-site laborer)*	0.0048	0.0023	4.80E-09	2.30E-09
22	SWMU as a Whole	1,3,5-Trinitrobenzene	0.84	0.84	8.40E-07	8.40E-07
		2,4,6-Trinitrotoluene	2,157	2,157	2.16E-03	2.16E-03
		RDX	49.4	49.4	4.94E-05	4.94E-05

Table 3. Surface Soil Concentrations for the Natural Wind Erosion Scenario (continued)

SWMU	Area of Concern	COPC	Concentration (mg/kg)		Fraction	
			CTE	RME	CTE	RME
23	SWMU as a Whole	Anthracene *(on-site adult resident)*	0.04	0.1	4.00E-08	1.00E-07
		Anthracene *(on-site child resident)*	0.04	0.16	4.00E-08	1.60E-07
		Anthracene *(on-site laborer)*	0.10	0.11	1.00E-07	1.10E-07
		carcinogenic PAHs *(on-site adult resident)*	0.06	0.16	6.00E-08	1.60E-07
		carcinogenic PAHs *(on-site child resident)*	0.06	0.26	6.00E-08	2.60E-07
		carcinogenic PAHs *(on-site laborer)*	0.17	0.19	1.70E-07	1.90E-07
		Cadmium	2.9	2.9	2.90E-06	2.90E-06
		Chromium	67.5	67.5	6.75E-05	6.75E-05
		Lead	82.7	82.7	8.27E-05	8.27E-05
		PCBs	1.38	1.38	1.38E-06	1.38E-06
		Phenanthrene *(on-site adult resident)*	0.08	0.25	8.00E-08	2.50E-07
		Phenanthrene *(on-site child resident)*	0.08	0.42	8.00E-08	4.20E-07
		Phenanthrene *(on-site laborer)*	0.20	0.30	2.00E-07	3.00E-07
		Pyrene *(on-site adult resident)*	0.88	2.07	8.80E-07	2.07E-06
		Pyrene *(on-site child resident)*	0.88	3.2	8.80E-07	3.20E-06
		Pyrene *(on-site laborer)*	2.27	2.44	2.27E-06	2.44E-06

Table 3. Surface Soil Concentrations for the Natural Wind Erosion Scenario (continued)

SWMU	Area of Concern	COPC	Concentration (mg/kg)		Fraction	
			CTE	RME	CTE	RME
31	SWMU as a Whole	carcinogenic PAHs *(on-site adult resident)*	0.0025	0.0062	2.50E-09	6.20E-09
		carcinogenic PAHs *(on-site child resident)*	0.0025	0.010	2.50E-09	1.00E-08
		carcinogenic PAHs *(on-site laborer)*	0.0067	0.0075	6.70E-09	7.50E-09
32	SWMU as a Whole	Arsenic	16.1	16.1	1.61E-05	1.61E-05
		Cadmium	4.01	4.01	4.01E-06	4.01E-06
35	SWMU as a Whole	Arsenic	31.1	31.1	3.11E-05	3.11E-05
		delta-Benzenehexachloride *(on-site adult resident)*	0.005	0.01	5.00E-09	1.00E-08
		delta-Benzenehexachloride *(on-site child resident)*	0.005	0.017	5.00E-09	1.70E-08
		delta-Benzenehexachloride *(on-site laborer)*	0.014	0.012	1.40E-08	1.20E-08
		alpha-Chlordane *(on-site adult resident)*	1.4	1.82	1.40E-06	1.82E-06
		alpha-Chlordane *(on-site child resident)*	1.4	2.93	1.40E-06	2.93E-06
		alpha-Chlordane *(on-site laborer)*	3.47	2.17	3.47E-06	2.17E-06
		gamma-Chlordane *(on-site adult resident)*	1.17	1.52	1.17E-06	1.52E-06
		gamma-Chlordane *(on-site child resident)*	1.17	2.45	1.17E-06	2.45E-06
		gamma-Chlordane *(on-site laborer)*	2.91	1.82	2.91E-06	1.82E-06

Table 3. Surface Soil Concentrations for the Natural Wind Erosion Scenario (continued)

SWMU	Area of Concern	COPC	Concentration (mg/kg)		Fraction	
			CTE	RME	CTE	RME
35 (cont.)		alpha-Endosulfan *(on-site adult resident)*	0.00001	0.00008	1.00E-11	8.00E-11
		alpha-Endosulfan *(on-site child resident)*	0.00001	0.00014	1.00E-11	1.40E-10
		alpha-Endosulfan *(on-site laborer)*	0.000017	0.00010	1.70E-11	1.10E-10
		Endrin *(on-site adult resident)*	0.03	0.21	3.00E-08	2.10E-07
		Endrin *(on-site child resident)*	0.03	0.35	3.00E-08	3.50E-07
		Endrin *(on-site laborer)*	0.075	0.25	7.50E-08	2.50E-07
		Heptachlor *(on-site adult resident)*	0.00007	0.00011	7.00E-11	1.10E-10
		Heptachlor *(on-site child resident)*	0.00007	0.00018	7.00E-11	1.80E-10
		Heptachlor *(on-site laborer)*	0.00019	0.00013	1.90E-10	1.30E-10
		Heptachlor Epoxide *(on-site adult resident)*	0.004	0.018	4.00E-09	1.80E-08
		Heptachlor Epoxide *(on-site child resident)*	0.004	0.03	4.00E-09	3.00E-08
		Heptachlor Epoxide *(on-site laborer)*	0.011	0.022	1.10E-08	2.20E-08
36	SWMU as a Whole	Barium	309	309	3.09E-04	3.09E-04
		Copper	100	100	1.00E-04	1.00E-04
		Lead	117	117	1.17E-04	1.17E-04
40	SWMU as a Whole	Arsenic	11	11	1.10E-05	1.10E-05
		Barium	133	133	1.33E-04	1.33E-04

Table 3. Surface Soil Concentrations for the Natural Wind Erosion Scenario (continued)

SWMU	Area of Concern	COPC	Concentration (mg/kg)		Fraction	
			CTE	RME	CTE	RME
40 (cont.)		Lead	31.9	31.9	3.19E-05	3.19E-05
		HMX	2.2	2.2	2.20E-06	2.20E-06
		RDX	3.4	3.4	3.40E-06	3.40E-06
		1,3,5-Trinitrobenzene	0.49	0.49	4.90E-07	4.90E-07

The natural wind erosion scenario assumed all erodible PM_{10} emissions were entrained into the atmosphere during a 1-hour period with the worst-case dispersion. All other hours had no emissions. In this case, the annual impacts were equal to the maximum 1-hour impact divided by 8,760. This approach was chosen for this scenario since wind erosion is not continuous; it is better described as a function of wind gustiness.

In reality, annual impacts result from varying combinations of atmospheric stability, wind speed, and wind direction, will be lower than the values estimated with the approaches outlined above.

4.1 MODEL SELECTION

The USEPA's SCREEN2 air dispersion model, version dated 92245, was selected to estimate the air quality impacts at selected sites surrounding TEAD-N. SCREEN2 is a single-source, screening-level model that has algorithms to estimate air quality impacts associated with area sources. For area source modeling, SCREEN2 provides estimates of 1-hour impacts based on simulated, hourly meteorology.

4.2 MODEL OPTIONS AND INPUT

The SCREEN2 model options used in the analysis were:

- Area source
- Normalized emission rate (1.0 $g/m^2/sec$)
- Receptor height (above ground-level)
- Rural dispersion coefficients
- Full meteorology
- Discrete receptors
- Flat terrain

With these options, SCREEN2 calculated the maximum (or worst-case) 1-hour impacts at each receptor for each SWMU.

4.2.1 Source Data

Each SWMU was modeled as an area source. The area source parameters used as input to SCREEN2 are presented in Table 4. For simplicity, each SWMU was treated as a square area. This is consistent with the area source data requirements of SCREEN2. The "length of side" presented for each SWMU corresponds to the side of a square area with a total area as shown in Table 1.

Table 4. SCREEN2 Area Source Parameters for Each SWMU

SWMU	Source Type	Emission Rate ($g/m^2/sec$)	Source Height (m)	Length of Side (m)
6	AREA	1.0	0	403
7	AREA	1.0	0	1258
7-a		1.0	0	786
7-b		1.0	0	723
7-c		1.0	0	666
8	AREA	1.0	0	205
22	AREA	1.0	0	34
23	AREA	1.0	0	133
32	AREA	1.0	0	14
35	AREA	1.0	0	359
40	AREA	1.0	0	491

Note that SWMU 7 is presented both in terms of a single area and as three sub-areas. Dividing SWMU 7 into sub-areas (7-a, 7-b, and 7-c) was necessary for some model runs due to the proximity of this SWMU to the property line. SCREEN2 cannot predict impacts at locations closer than (length of side $\div \sqrt{\pi}$).

Because all SWMUs represent ground-level surfaces, the sources heights were set to 0 m.

4.2.2 Urban/Rural Classification

The *Guideline on Air Quality Models* (USEPA) recommends selection of appropriate dispersion coefficients based on predominant land use within a 3-km radius, circular area centered on the facility and the land use classification scheme developed by Auer (1978). Evaluation of this area indicates that it is predominantly open prairie land which is considered rural use. Therefore, rural dispersion coefficients were selected.

4.2.3 Meteorology

SCREEN2 used screening meteorology that is included within the model. The full meteorology option was selected which directed SCREEN2 to calculate the maximum (or worst-case) 1-hour impact at each receptor based on the full range of stability and wind speed included within the model.

4.2.4 Receptors

For each SWMU, normalized 1-hour impacts were estimated by SCREEN2 at five receptors. The distances to these receptors are listed in Table 5, and represent the shortest distance to each location. For the three towns, the distance is to the nearest town boundary. For the "edge of SWMU", the distance is to the nearest location for which SCREEN2 will estimate an impact. This is equal to (length of side / $\sqrt{\pi}$) + 1 m. Due to this limitation of SCREEN2, it was necessary to subdivide SWMU 7 into three smaller areas for modeling at the SWMU's edge and at the property line.

Table 5. SCREEN2 Receptors for Each SWMU

SWMU	Distance from SWMU to Receptor (m)				
	Edge of SWMU	Property Line	Grantsville	Tooele	Stockton
6	228	323	8,476	6,768	4,878
7			8,262	8,659	6,372
7-a	443	488			
7-b	408	427			
7-c	376	378			
8	116	543	2,561	11,860	12,683
22	19	921	7,744	9,756	7,591
23	75	1,494	5,732	11,707	10,335
32	8	1,311	7,409	1,524	8,049
35	203	512	10,091	2,165	3,933
40	277	963	4,055	12,530	12,073

For screening-level modeling, the wind is assumed to blow directly from the source toward the receptor. Therefore, direction to each receptor is not relevant.

Since the SWMUs were modeled as ground-level area sources, the receptor terrain elevations were set to 0.0 m (i.e., flat terrain). This is an acceptable assumption since there is no plume rise associated with a ground-level area source. The plume center-line will remain at ground-level regardless of any increase in terrain elevation. However, each receptor was set at a height of 6 ft (1.83 m) above ground-level to represent the breathing level of an exposed person.

4.3 SPECIAL MODELING CONSIDERATIONS: CONVERTING 1-HOUR IMPACTS TO ANNUAL IMPACTS

SCREEN2 estimated the maximum, normalized 1-hour impact at each receptor based on the meteorological conditions that result in the worst-case dispersion. It was necessary to convert these impacts to normalized, annual impacts for use in the risk assessment. The conversion approach was dependent on the scenario.

4.3.1 Construction Activity

For the construction activity scenario, 8,760 hours of construction per year was conservatively assumed. Conservatively assuming that the maximum 1-hour impact predicted by SCREEN2 is representative of every hour during the year, the annual impact is then equivalent to the maximum 1-hour impact (i.e., (maximum 1-hour impact $\times$ 8,760) / 8,760).

4.3.2 Natural Wind Erosion

For the natural wind erosion scenario, it was conservatively assumed that all erodible PM_{10} (see Section 3.1.2) was entrained into the atmosphere during the hour with the worst-case dispersion. The maximum 1-hour impact from SCREEN2 was then assumed to occur for only one hour during the year. The remaining 8,759 hours of the year would have no impact since no erodible material would be left to entrain into the atmosphere. Therefore, the maximum annual average impact would equal the maximum 1-hour impact / 8,760.

4.4 SCREEN2 MODEL RESULTS

Estimated annual impacts of each contaminant are presented in Table 6 for the construction activity scenario and Tables 7 and 8 for the on-site and off-site natural wind erosion scenarios. Results using both the CTE and the RME soil concentrations (see Table 2 and Table 3) are presented.

For each SWMU and receptor, the annual impacts were calculated using the maximum, normalized 1-hour impact from SCREEN2 and the following equations:

Table 6. Annual Impacts for the CTE and RME Construction Activity Scenarios

SWMU	Area of Concern	COPC	CTE Annual Impacts ($\mu g/m^3$)	RME Annual Impacts ($\mu g/m^3$)
6	Northeast Revetment Area	Aluminum	53.9	53.9
		Antimony	0.0868	0.0868
		Arsenic	0.0864	0.0864
		Chromium	0.179	0.179
		Copper	2.3	2.3
		Iron	208	208
		Lead	1.02	1.02
		Thallium	0.18	0.18
		Zinc	20	20
	Hot Spot at Test Pit 3	1,3,5-Trinitrobenzene	0.0659	0.0659
7	Firing Point	Arsenic	0.226	0.226
	Northwest Test Area Trench	Aluminum	575	575
		Beryllium	0.0243	0.0243
		Thallium	0.564	0.564
8	Bullet Stop	Arsenic	0.238	0.238
		Zinc	134	134
		Lead	0.13	0.13
	Firing Lines	Chromium	0.126	0.126
13	---	Diethyl Phthalate	0.0026	0.0026
22	---	2,4,6-Trinitrotoluene	0.0523	0.0523
		Chromium	0.0453	0.0453

Table 6. Annual Impacts for the CTE and RME Construction Activity Scenarios (continued)

SWMU	Area of Concern	COPC	CTE Annual Impacts ($\mu g/m^3$)	RME Annual Impacts ($\mu g/m^3$)
23	Building 1343 Outfall	Chromium	0.143	0.143
	Outfall Near Building 1344	Chromium	1.41	1.41
		PAHs	0.0000629	0.000149
	Building 1345 Outfall	Chromium	0.174	0.174
		PCBs	0.000419	0.000419
	Asphalt and Stained Area	Chromium	0.304	0.304
		PAHs	0.000128	0.000314
31	---	none		
32	---	Chromium	0.0177	0.0177
35		none		
36		none		
40	Remainder of SWMU	Arsenic	0.0309	0.0309

Table 7. Annual Impacts for the CTE and RME On-site Natural Wind Erosion Scenarios

SWMU	Area of Concern	COPC	CTE Annual Impacts ($\mu g/m^3$)	RME Annual Impacts ($\mu g/m^3$)
6	SWMU as a Whole	Arsenic	0.000755	0.000755
		Copper	0.00391	0.00391
		Lead	0.0046	0.0046
7	SWMU as a Whole	Aluminum	3.61	3.61
		Beryllium	0.000176	0.000176
		Manganese	0.129	0.129
		Thallium	0.00523	0.00523
8	SWMU as a Whole	Aluminum	0.711	0.711
		Antimony	0.00128	0.00128
		Arsenic	0.000355	0.000355
		Copper	0.00213	0.00213
		Lead	0.022	0.022
13	SWMU as a Whole	Chloromethane *(on-site adult resident)*	0.0000000856	0.0000000904
		Chloromethane *(on-site child resident)*	0.0000000856	0.000000152
		Chloromethane *(on-site laborer)*	0.000000228	0.000000109
22	SWMU as a Whole	1,3,5-Trinitrobenzene	0.00000867	0.00000867
		2,4,6-Trinitrotoluene	0.0223	0.0223
		RDX	0.000510	0.000510

Table 7. Annual Impacts for the CTE and RME On-site Natural Wind Erosion Scenarios (continued)

SWMU	Area of Concern	COPC	CTE Annual Impacts ($\mu g/m^3$)	RME Annual Impacts ($\mu g/m^3$)
23	SWMU as a Whole	Anthracene *(on-site adult resident)*	0.00000136	0.00000340
		Anthracene *(on-site child resident)*	0.00000136	0.00000544
		Anthracene *(on-site laborer)*	0.00000340	0.00000374
		carcinogenic PAHs *(on-site adult resident)*	0.00000204	0.00000544
		carcinogenic PAHs *(on-site child resident)*	0.00000204	0.00000884
		carcinogenic PAHs *(on-site laborer)*	0.00000578	0.00000646
		Cadmium	0.0000986	0.0000986
		Chromium	0.00229	0.00229
		Lead	0.0028	0.0028
		PCBs	0.0000469	0.0000469
		Phenanthrene *(on-site adult resident)*	0.00000272	0.00000850
		Phenanthrene *(on-site child resident)*	0.00000272	0.0000143
		Phenanthrene *(on-site laborer)*	0.00000680	0.0000102
		Pyrene *(on-site adult resident)*	0.0000299	0.0000704
		Pyrene *(on-site child resident)*	0.0000299	0.000109
		Pyrene *(on-site laborer)*	0.0000772	0.0000829

Table 7. Annual Impacts for the CTE and RME On-site Natural Wind Erosion Scenarios (continued)

SWMU	Area of Concern	COPC	CTE Annual Impacts ($\mu g/m^3$)	RME Annual Impacts ($\mu g/m^3$)
31	SWMU as a Whole	carcinogenic PAHs *(on-site adult resident)*	0.0000000857	0.000000212
		carcinogenic PAHs *(on-site child resident)*	0.0000000857	0.000000356
		carcinogenic PAHs *(on-site laborer)*	0.000000230	0.000000257
32	SWMU as a Whole	Arsenic	0.0000686	0.0000686
		Cadmium	0.0000171	0.0000171
35	SWMU as a Whole	Arsenic	0.00152	0.00152
		delta-Benzenehexachloride *(on-site adult resident)*	0.000000244	0.000000488
		delta-Benzenehexachloride *(on-site child resident)*	0.000000244	0.000000829
		delta-Benzenehexachloride *(on-site laborer)*	0.000000683	0.000000585
		alpha-Chlordane *(on-site adult resident)*	0.0000683	0.0000887
		alpha-Chlordane *(on-site child resident)*	0.0000683	0.000143
		alpha-Chlordane *(on-site laborer)*	0.000169	0.000106
		gamma-Chlordane *(on-site adult resident)*	0.0000570	0.0000741
		gamma-Chlordane *(on-site child resident)*	0.0000570	0.000119
		gamma-Chlordane *(on-site laborer)*	0.000142	0.0000887

Table 7. Annual Impacts for the CTE and RME On-site Natural Wind Erosion Scenarios (continued)

SWMU	Area of Concern	COPC	CTE Annual Impacts ($\mu g/m^3$)	RME Annual Impacts ($\mu g/m^3$)
35 (cont.)		alpha-Endosulfan *(on-site adult resident)*	0.000000000488	0.00000000390
		alpha-Endosulfan *(on-site child resident)*	0.000000000488	0.00000000683
		alpha-Endosulfan *(on-site laborer)*	0.000000000829	0.00000000488
		Endrin *(on-site adult resident)*	0.00000146	0.0000102
		Endrin *(on-site child resident)*	0.00000146	0.0000171
		Endrin *(on-site laborer)*	0.00000366	0.0000122
		Heptachlor *(on-site adult resident)*	0.00000000341	0.00000000536
		Heptachlor *(on-site child resident)*	0.00000000341	0.00000000878
		Heptachlor *(on-site laborer)*	0.00000000926	0.00000000634
		Heptachlor Epoxide *(on-site adult resident)*	0.000000195	0.000000878
		Heptachlor Epoxide *(on-site child resident)*	0.000000195	0.00000146
		Heptachlor Epoxide *(on-site laborer)*	0.000000536	0.00000107
36	SWMU as a Whole	Barium	0.00938	0.00938
		Copper	0.00303	0.00303
		Lead	0.0036	0.0036
40	SWMU as a Whole	Arsenic	0.000583	0.000583
		Barium	0.00703	0.00703

Table 7. Annual Impacts for the CTE and RME On-site Natural Wind Erosion Scenarios (continued)

SWMU	Area of Concern	COPC	CTE Annual Impacts ($\mu g/m^3$)	RME Annual Impacts ($\mu g/m^3$)
40 (cont.)		Lead	0.0017	0.0017
		HMX	0.0000260	0.0000260
		RDX	0.000117	0.000117
		1,3,5-Trinitrobenzene	0.0000180	0.0000180

Table 8. Annual Impacts for the RME and CTE Off-site Natural Wind Erosion Scenario

			RME and CTE Annual Impacts ($\mu g/m^3$)				
SWMU	**Area of Concern**	**COPC**	**Edge of SWMU**	**Property Line**	**Grantsville**	**Tooele**	**Stockton**
6	SWMU as a Whole	Arsenic	0.000755	0.000647	0.0000864	0.000108	0.000146
		Copper	0.00391	0.00335	0.000448	0.000563	0.000756
		Lead	0.00455	0.0039	0.000521	0.000654	0.000878
7	SWMU as a Whole	Aluminum	3.61	3.55	0.538	0.527	0.595
		Beryllium	0.000176	0.000173	0.0000262	0.0000257	0.0000290
		Manganese	0.0129	0.126	0.0192	0.0188	0.0212
		Thallium	0.00542	0.00532	0.000807	0.000792	0.000893
8	SWMU as a Whole	Aluminum	0.711	0.320	0.113	0.0189	0.0174
		Antimony	0.00128	0.000578	0.000204	0.0000342	0.0000314
		Arsenic	0.000355	0.000160	0.0000563	0.00000945	0.00000868
		Copper	0.00213	0.000959	0.000339	0.0000568	0.0000522
		Lead	0.0219	0.00985	0.00348	0.00583	0.000536
13	Not Applicable						
22	Not Applicable						
23	Not Applicable						
31	Not Applicable						
32	Not Applicable						
35	Not Applicable						
36	Not Applicable						
40	SWMU as a Whole	Arsenic	0.0000583	0.000318	0.000153	0.0000586	0.0000611

Table 8. Annual Impacts for the RME and CTE Off-site Natural Wind Erosion Scenario (continued)

SWMU	Area of Concern	COPC	RME and CTE Annual Impacts ($\mu g/m^3$)				
			Edge of SWMU	Property Line	Grantsville	Tooele	Stockton
40 (cont.)		Barium	0.00704	0.00385	0.00186	0.000708	0.000738
		Lead	0.00169	0.000923	0.000445	0.000170	0.000177
		1,3,5-Trinitrobenzene	0.0000260	0.0000142	0.00000684	0.00000261	0.00000272
		HMX	0.000117	0.0000636	0.0000307	0.0000117	0.0000122
		RDX	0.000180	0.0000984	0.0000474	0.0000181	0.0000189

Construction Activity:

(Equation 6)

$$\text{Annual Impact}\left(\frac{\mu g}{m^3}\right) = \text{Maximum normalized 1-hour impact}\left(\frac{\mu g/m^3}{g/m^2/\text{sec}}\right) \times PM_{10}\text{ emission rate for construction}\left(\frac{g}{m^2 \cdot \text{sec}}\right) \times \text{Contaminant fraction in the subsurface soil} \times \frac{8760}{8760}$$

Natural Wind Erosion:

(Equation 7)

$$\text{Annual Impact}\left(\frac{\mu g}{m^3}\right) = \text{Maximum normalized 1-hour impact}\left(\frac{\mu g/m^3}{g/m^2/\text{sec}}\right) \times PM_{10}\text{ emission rate for wind erosion}\left(\frac{g}{m^2 \cdot \text{sec}}\right) \times \text{Contaminant fraction in the surface soil} \times \frac{1}{8760}$$

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APPENDIX O

ADULT EXPOSURES TO INORGANIC LEAD

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1.0 ADULT EXPOSURES TO INORGANIC LEAD

The USEPA has developed the Integrated Exposure Uptake Biokinetic (IEUBK) model to evaluate lead exposure in children. The model estimates blood lead levels resulting from all applicable routes of exposure. The agency has set a target blood lead level of 10 μg Pb/dL blood. That target was used in evaluating child lead exposures in the RA.

The agency has recognized that this approach is not appropriate for land use best described by non-residential adult exposure (USEPA 1994). Hence, the agency has recommended a short-term option based on a simple approach that approximates the more complicated biokenetics in humans. Models for adult exposure are available in the scientific literature that meet USEPA's short-term criterion. Of these, the model proposed by Bowers and colleagues (1994) is most nearly consistent with the approach in the IEUBK model (USEPA 1995). It has the added advantage of simplicity.

This model was modified by USEPA Region VIII in the risk assessment for the California Gulch Superfund site (USEPA 1995). That modified approach was used in the RA along with the site-specific occupational exposure parameters in Appendix L.

1.1 ADULT OCCUPATIONAL EXPOSURE MODEL

The Bowers model predicts a geometric mean blood lead level by summing the baseline level with the incremental increase predicted to occur through occupational exposure. Because the model seeks to evaluate occupational exposures, it assumes that exposures will occur through ingestion or inhalation of soil and dust and ingestion of water. Groundwater was not part of the Phase II investigation and is therefore not evaluated in the risk assessment. The baseline blood lead level accounts for uptake of lead from other, non-site related, water sources. The resulting equation then becomes:

(Equation 1)

$$[PbB_{GM}] = [PbB_{baseline}] + (BSF)(Uptake_{air} + Uptake_{soil/dust})$$

where

PbB_{GM} = Geometric mean blood lead level in adults potentially exposed to lead in soil or dust through occupational activities (μg Pb/dL blood)

$PbB_{baseline}$ = Baseline blood level resulting from non-occupational sources (μg Pb/dL blood)

BSF = Biokinetic slope factor relating blood lead to absorbed lead [(μg Pb/dL blood)/(μg /day)]

$Uptake_{air}$ = Uptake through inhalation (μg /day) given by:

(Equation 1a)

$$Uptake_{air} = A_a\ V_a\ ET\ t_a\ C_a$$

where

A_a = Lung deposition and absorption (unitless)
V_a = Ventilation rate during working hours (m^3/hr)
ET = Occupational exposure time (hr/day)
t_a = Time-activity pattern constant corresponding to the fraction of total days per year spent on site (unitless)
C_a = Concentration of lead in air ($\mu g/m^3$)

$Uptake_{soil/dust}$ = Uptake through inhalation (μg /day) given by:

(Equation 1b)

$$Uptake_{soil/dust} = A_{s/d}\ I_{s/d}\ t_{s/d}\ C_{s/d}$$

where

$A_{s/d}$ = Soil/dust absorption (unitless)
$I_{s/d}$ = Soil ingestion rate (g/day)
$t_{s/d}$ = Time-activity pattern constant corresponding to the fraction of waking hours spent on site (unitless)
C_a = Concentration of lead in soil ($\mu g/g$)

1.1.1 Baseline Adult Blood Lead Level ($PbB_{baseline}$)

Because the target levels cited above are total blood lead concentrations, a baseline adult concentration must be established to determine whether the incremental increase resulting from potential occupational exposures at TEAD-N cause the target levels to be exceeded. The primary sources for baseline data are epidemiological studies.

In the California Gulch risk assessment (USEPA 1995), two studies are cited. The NHANES III study (Brody et al. 1994) gives geometric mean (GM) values of 1.7 μg Pb/dL blood for

women aged 20 to 49, 2.0 μg Pb/dL blood for Hispanics, and 2.2 μg Pb/dL blood for African-Americans. The second source is the Leadville study conducted by the University of Cincinnati (EPA 1995). In this study, data were collected from 157 individuals aged 18 to 49. The GM of this data set was 2.7 μg Pb/dL blood. The data were not segregated by gender.

Bowers and coworkers also cite two studies, both conducted by the University of Cincinnati. The GM value for 48 adults in Butte, Montana, was 3.1 μg Pb/dL blood with a geometric standard deviation (GSD) of 1.94 μg Pb/dL blood (Butte-Silver Bow Department of Health/University of Cincinnati Department of Environmental Health 1991). In a similar study in Midvale, Utah, the GM value of a group of 43 adults was 2.2 μg Pb/dL blood with a GSD of 1.77 μg Pb/dL blood (Bornschien et al. 1990). Neither study reported the data by gender.

Midvale, Utah, is some 25 miles due east of Tooele, Utah. Because of the geographic proximity and a reported blood lead level higher than that reported for the female cohort in the NHANES III study, the Midvale geometric mean value of 2.2 μg Pb/dL blood was used as the baseline adult blood level for the site-specific laborer and construction worker in the risk assessment.

1.1.2 Biokinetic Slope Factor (BSF)

The biokinetic slope factor (BSF) proposed by Bowers and others is based on a study of adult humans exposed to lead in water (Pocock et al. 1983). A value of 0.375 μg Pb/dL blood per μg/day lead uptake was derived based on the concentration in "first-draw" water. Because the concentration in first-draw water would be expected to be higher than in water drawn after the pipes are flushed, a volume-weighted average lead concentration in water would have resulted in a higher estimated BSF.

In the California Gulch risk assessment, a different BSF is estimated based on the pharmacokinetic model developed by O'Flaherty (1993). Calculations by Murphy (USEPA 1995) are used to develop a variable BSF dependent on age, sex, and lead body burden. The California Gulch adopts a midpoint in this BSF range of 0.4 μg Pb/dL blood per μg/day lead uptake. This value was used in the current risk assessment, although the difference with the BSF proposed by Bowers is not significant to the numerical estimates.

1.1.3 Inhalation Exposure

Potential exposure through direct inhalation of lead bound to resuspended particulates was evaluated for the hypothetical occupational receptors defined by the exposure scenarios. Exposures were estimated based on the duration of exposure, the inhalation rate of the exposed individuals during the exposure, and the concentration of chemicals in the air breathed. The model used for estimating inhalation exposure is shown below:

$$Uptake_{air} = A_a\ V_a\ ET\ t_a\ C_a$$

where

A_a	=	Lung deposition and absorption (unitless)
V_a	=	Ventilation rate during working hours (m^3/hr)
ET	=	Occupational exposure time (hr/day)
t_a	=	Time-activity pattern constant corresponding to the fraction of total days per year spent on site (unitless)
C_a	=	Concentration of lead in air ($\mu g/m^3$)

1.1.3.1 *Lung Deposition and Absorption (A_a)*

The USEPA reviewed several deposition and absorption studies (USEPA 1986) and established a range of 28 percent to 70 percent for deposition of airborne particulate into the deep lung. Deposition varied with particle size (variable), ventilation rate (inversely), and work conditions (variable). One study indicated that deposition decreased from 80 percent for a mean particle density of 0.02 μm to 30 percent for a mean particle density of 0.09 μm. Another study corroborated these findings for this particle size range but measured an increase in deposition to 63 percent for a mean particle diameter of 1.0 μm. Finally, a study of lead in workplace air defined as fumes and fine or coarse dust measures a maximum deposition of 47 percent. Air dispersion modeling (Appendix N) was conducted for particles less than 10 μm in diameter. Based on EPA's review, a deposition fraction of 50 percent was assumed for this risk assessment.

Absorption from the deep lung was estimated to be greater than 90 percent and corroborated in several studies (USEPA 1986). To be health-protective, a deep-lung absorption fraction of 100 percent was assumed. Hence, A_a was set equal to 50 percent or 0.5.

1.1.3.2 *Ventilation Rate during Working Hours (V_a)*

The RME (reasonable maximum exposure) and CTE (central tendency exposure) ventilation rates for both the site-specific laborer and construction worker are given in Appendix N. For the RME site-specific laborer, $V_{a(RME)}$ is assumed to be 0.9 m^3/hr while for the CTE site-specific laborer, $V_{a(CTE)}$ is assumed to be 0.6 m^3/hr. For the RME construction worker, $V_{a(RME)}$ is assumed to be 1.2 m^3/hr while for the CTE construction worker, $V_{a(CTE)}$ is assumed to be 0.8 m^3/hr.

1.1.3.3 *Exposure Time (ET)*

Again, the daily exposure time is based on the respective occupational scenarios described in Appendix N. For the RME site-specific laborer, ET_{RME} is assumed to be 10 hr/day while for the CTE site-specific laborer, ET_{CTE} is assumed to be 2 hr/day. For the RME construction worker, ET_{RME} is assumed to be 10 hr/day while for the CTE construction worker, ET_{CTE} is assumed to be 8 hr/day.

1.1.3.4 *Time-Activity Pattern Constant (t_a)*

Inhalation occurs 24 hours per day every day. Daily inhalation while at work is accounted for by the product V_aET. The time-activity pattern constant accounts for the fraction of the year spent at work (i.e., on site).

Most laborers at TEAD-N currently work 4-day-weeks. For the RME, as a worst-case estimate, it is assumed that the laborer is working at one particular SWMU the entire working year or 192 days/yr. This assumes that the laborer spends approximately 4 weeks on vacation, sick leave, and holidays away from the site. Therefore, $t_{a(RME)}$ is 192/365 or 0.53.

For the CTE site-specific laborer, it is assumed that the laborer is working on various assignments at various SWMUs throughout the year. The time that the laborer will spending at any given SWMU is estimated to be 50 days/year. In this case, $t_{a(CTE)}$ is 50/365 or 0.14.

For the RME construction worker, it is assumed that the worker is working at the site for an exposure frequency of 140 days or a 6-month construction project. Therefore, $t_{a(RME)}$ is 140/365 or 0.38. For the CTE, it is assumed that the worker is working at the site for an exposure frequency of 60 days, equivalent to a 3-month construction project. In this case, $t_{a(CTE)}$ is 60/365 or 0.16.

1.1.3.5 *Concentration of Lead in Air (C_a)*

The concentration in air is based on average values and the air dispersion modeling described in Appendix N.

1.1.4 Ingestion Exposure

Exposure to soil lead through ingestion was estimated based on the duration of exposure, the ingestion rate during exposure, and the concentration of lead in the soil ingested. The model used for estimating ingestion exposure is shown below:

$$Uptake_{soil/dust} = A_{s/d}\ I_{s/d}\ t_{s/d}\ C_{s/d}$$

where

$A_{s/d}$	=	Soil/dust absorption (unitless)
$I_{s/d}$	=	Soil ingestion rate (g/day)
$t_{s/d}$	=	Time-activity pattern constant corresponding to the fraction of waking hours spent on site (unitless)
C_a	=	Concentration of lead in soil (μg/g)

1.1.4.1 *Soil/Dust Absorption ($A_{s/d}$)*

Studies have demonstrated that the gastrointestinal absorption of ingested lead varies directly with time from last meal (USEPA 1995). For lead ingested along with food or in the diet, the absorption fraction is 8 percent to 10 percent. Lead ingested by subjects who have fasted for 9 hours, was absorbed at a rate of 30 percent to 37 percent. For the occupational scenarios modeled in this risk assessment, it is assumed that workers arrive at work shortly after breakfast and have lunch approximately in the middle of the work day. USEPA (1995) considers an absorption fraction of 10 percent to be more representative of this scenario.

Lead in soil and mine wastes may be absorbed less extensively than lead in food and water. In fact, the current version of the IEUBK model assumes a relative bioavailability of lead from soil of 60 percent. Therefore, the net absorption fraction, $A_{s/d}$, is set at 6 percent.

1.1.4.2 *Soil Ingestion Rate ($I_{s/d}$)*

Under the CTE and RME scenarios for the site-specific worker, the ingestion rate of soil is assumed to be 10 and 50 mg/day, respectively (AIHC 1994; Finley et al. 1994). For the CTE and RME construction worker scenarios, the ingestion rate of soil is 240 and 480 mg/day, respectively (USEPA 1990). For both scenarios, the adult is assumed to engage in outdoor physical activity. The estimate is based on ingesting a 50 μm-thick layer of soil from the inside surfaces of the fingers and thumb of one hand twice daily for the RME scenario and once for the CTE scenario (USEPA 1990).

1.1.4.3 *Time-Activity Pattern Constant ($t_{s/d}$)*

Soil ingestion occurs only during waking hours, which are assumed to be 16 hours/day. Because the soil ingestion rate is in units of mass per day, the time-activity pattern constant must include a factor representing the fraction of the day on site.

Most laborers at TEAD-N currently work 10-hour days and 4-day-weeks. For the RME, as a worst-case estimate, it is assumed that the laborer is working at one particular SWMU the entire working year or 192 days/yr. This assumes that the laborer spends approximately 4

weeks on vacation, sick leave, and holidays away from the site. Therefore, $t_{s/d(RME)}$ is (10/16)(192/365) or 0.33.

For the CTE site-specific laborer, it is assumed that the laborer is working on various assignments at various SWMUs throughout the year, none longer than 2 hours. The time that the laborer will spending at any given SWMU is estimated to be 50 days/year. In this case, $t_{s/d(CTE)}$ is (2/16)(50/365) or 0.02.

For the RME construction worker, it is assumed that the worker is working at the site 10 hours/day for an exposure frequency of 140 days or a 6-month construction project. Therefore, $t_{s/d(RME)}$ is (10/16)(140/365) or 0.24. For the CTE, it is assumed that the worker is working at the site 8 hours/day for an exposure frequency of 60 days, equivalent to a 3-month construction project. In this case, $t_{s/d(CTE)}$ is (8/16)(60/365) or 0.08.

1.1.4.4 *Concentration of Lead in Soil ($C_{s/d}$)*

The concentration in soil is based on average values described in Appendix L.

1.1.5 Target Blood Lead Level

In the California Gulch risk assessment, USEPA Region VIII has proposed a target blood lead level for women of child-bearing age in the workforce. That level is 11.1 μg Pb/dL blood and is based on the specification that the 95th percentile of blood lead distribution in fetuses not exceed 10 μg Pb/dL blood . In addition, blood lead level ranges of 10 μg Pb/dL blood (pregnant women) to 40 μg Pb/dL blood (occupational adult population) have been proposed (Carrington and Bolger 1992; OSHA 1991) as appropriate.

In this risk assessment the target level of 11.1 μg Pb/dL blood is used. That value is fixed as the 95th percentile value for women of child-bearing age in the workforce. The model output, however, is a geometric mean value for blood lead concentration. The geometric mean target level is derived from the following equation (USEPA, 1995):

$$PbB_{GM}(\text{maternal}) = 11.1/GSD_p^{1.645}$$

where

GSD_p = observed geometric standard deviation from the population

As stated above , the observed GSD for the Midvale, Utah, study group was 1.77 μg Pb/dL blood. Appling that value yields a geometric mean target level of 4.34 μg Pb/dL blood.

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APPENDIX P

TENTATIVELY IDENTIFIED COMPOUNDS

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Tentatively Identified Compounds, SWMU 7

Site ID	Tentative ID	Value (ug/g)
CRP-94-01A	Unknown unsaturated oxy hydrocarbon	0.3
CRP-94-01A	Unknown penanone isomer	0.3
CRP-94-01A	Unknown chloro oxy hydrocarbon	0.3
CRP-94-01A	Unknown acid ester	2
CRP-94-01A	Unknown propanol isomer	0.3
CRP-94-01A	Hexadecanoic Acid	0.6
CRP-94-01A	Unknown alcohol	2
CRP-94-01A	Unknown alkane	0.4
CRP-94-01A	Unknown alcohol	0.9
CRP-94-01A	Unknown alcohol	0.3
CRP-94-01B	Unknown alkyl cyclic hydrocarbon	0.6
CRP-94-01B	Unknown pentanone isomer	0.3
CRP-94-01B	Oxy Chloro Hydrocarbon	0.4
CRP-94-01B	Unknown acid ester	2
CRP-94-01B	Hexadecanoic acid	0.3
CRP-94-01B	Unknown alcohol	1
CRP-94-01B	Unknown alkane	0.3
CRP-94-01B	Unknown alkane	0.3
CRP-94-01B	Unknown alcohol	0.8
CRP-94-01C	Unknown oxy heterocycle	0.4
CRP-94-01C	Unsaturated Branched Hydrocarbon	0.4
CRP-94-01C	Nitrogen Containing Hydrocarbon	0.3
CRP-94-01C	Unknown pentanone isomer	0.3
CRP-94-01C	Unknown chloro oxy hydrocarbon	0.3
CRP-94-01C	Unknown acid ester	2
CRP-94-01C	Hexadecanoic acid	0.4
CRP-94-01C	Unknown alcohol	1
CRP-94-01C	Unknown alkane	0.5
CRP-94-01C	Unknown alkane	0.3
CRP-94-01C	Unknown alkane	0.3
CRP-94-01C	Unknown alcohol	0.9
CRP-94-01C	Unknown alcohol	0.3
CRP-94-01D	Unknown oxy heterocycle	0.5
CRP-94-01D	Unknown pentanone isomer	0.4
CRP-94-01D	Unknown chloro oxy hydrocarbon	0.4
CRP-94-01D	Unknown acid ester	2
CRP-94-01D	Nitrogen Containing Anthracenedione	0.8
CRP-94-01D	Unknown alcohol	0.8
CRP-94-01D	Nitrogen Containing Benzene Compound	1
CRP-94-01D	Unknown alcohol	0.3
CRP-94-02A	Unknown unsaturated oxy hydrocarbon	0.3
CRP-94-02A	Unknown oxy heterocycle	0.4
CRP-94-02A	Unknown alcohol	0.3

Tentatively Identified Compounds, SWMU 7 (continued)

Site ID	Tentative ID	Value (ug/g)
CRP-94-02A	Branched unsaturated hydrocarbon	0.5
CRP-94-02A	Unknown unsaturated oxy hydrocarbon	0.3
CRP-94-02A	Unknown pentanone isomer	0.4
CRP-94-02A	Unknown alkoxy ethanol	0.6
CRP-94-02A	Unknown oxy chloro hydrocarbon	0.4
CRP-94-02A	Unknown acid ester	1
CRP-94-02A	Hexadecanoic Acid	0.5
CRP-94-02A	Unknown alcohol	1
CRP-94-02A	Unknown alkane	0.5
CRP-94-02A	Unknown alkane	0.5
CRP-94-02A	Unknown alcohol	0.3
CRP-94-02A	Unknown alkane	0.5
CRP-94-02A	Unknown alcohol	1
CRP-94-02B	Unknown unsaturated oxy hydrocarbon	0.4
CRP-94-02B	Unknown pentanone isomer	0.3
CRP-94-02B	Unknown acid ester	1
CRP-94-02B	Hexadecanoic acid	0.5
CRP-94-02B	Unknown alcohol	1
CRP-94-02B	Unknown alkane	0.3
CRP-94-02B	Unknown cyclic oxy hydrocarbon	0.4
CRP-94-02B	Unknown alcohol	0.6
CRP-94-02C	Unknown unsaturated alcohol	0.3
CRP-94-02C	Unknown penanone isomer	0.3
CRP-94-02C	Unknown oxy chloro hydrocarbon	0.3
CRP-94-02C	Unknown acid ester	2
CRP-94-02C	Hexadecanoic acid	0.3
CRP-94-02C	Unknown alcohol	1
CRP-94-02C	Unknown alcohol	0.5
CRP-94-02D	Multi-unsaturated hydrocarbon	0.5
CRP-94-02D	Unknown pentanone isomer	0.3
CRP-94-02D	Unknown oxy chloro hydrocarbon	0.5
CRP-94-02D	Unknown acid ester	2
CRP-94-02D	Hexadecanoic acid	0.3
CRP-94-02D	Unknown alcohol	0.9
CRP-94-02D	Unknown alkane	0.3
CRP-94-02D	MeCL2 + Alcohol	0.4
CRP-94-03A	Unknown oxy hydrocarbon	0.4
CRP-94-03A	Unknown alcohol	1
CRP-94-03A	Unknown alcohol	0.3
CRP-94-03B	Unknown oxy hydrocarbon	0.3
CRP-94-03B	Unknown oxy hydrocarbon	0.5
CRP-94-03B	Unknown alcohol	1
CRP-94-03B	Unknown alkane	0.3

Tentatively Identified Compounds, SWMU 7 (continued)

Site ID	Tentative ID	Value (ug/g)
CRP-94-03B	Unknown alcohol	0.3
CRP-94-03C	Unknown oxy hydrocarbon	0.4
CRP-94-03C	Unknown chlorinated hydrocarbon	0.3
CRP-94-03C	Unknown oxy hydrocarbon	2
CRP-94-03C	Unknown alcohol	1
CRP-94-03C	Unknown alkane	0.4
CRP-94-03C	Unknown alkane	0.4
CRP-94-03C	Unknown alcohol	0.3
CRP-94-03D	Unknown oxy hydrocarbon	0.5
CRP-94-03D	Unknown oxy hydrocarbon	0.5
CRP-94-03D	Unknown alcohol	0.8
CRP-94-03D	Unknown alkane	0.3
CRP-94-04A	Unknown oxy hydrocarbon	0.4
CRP-94-04A	Unknown oxy hydrocarbon	0.7
CRP-94-04A	Unknown alcohol	0.6
CRP-94-04B	Unknown oxy hydrocarbon	0.6
CRP-94-04B	Unknown oxy hydrocarbon	0.5
CRP-94-04B	Unknown alcohol	0.3
CRP-94-04C	Unknown oxy hydrocarbon	0.3
CRP-94-04C	Unknown oxy hydrocarbon	0.5
CRP-94-04C	Unknown alcohol	0.9
CRP-94-04D	Unknown oxy hydrocarbon	0.3
CRP-94-04D	Unknown oxy hydrocarbon	0.8
CRP-94-04D	Unknown alcohol	2
CRP-94-04D	Unknown alkane	0.3
CRP-94-04D	Unknown alkane	0.3
CRP-94-04D	Unknown alcohol	0.7
CRP-94-05A	Unknown oxy hydrocarbon	0.3
CRP-94-05A	Unknown oxy hydrocarbon	0.9
CRP-94-05A	Unknown alcohol	0.8
CRP-94-05A	Unknown alcohol	0.3
CRP-94-05B	Unknown oxy hydrocarbon	0.3
CRP-94-05B	Unknown oxy hydrocarbon	0.5
CRP-94-05B	Unknown alcohol	0.9
CRP-94-05C	Unknown oxy hydrocarbon	0.5
CRP-94-05C	Unknown oxy hydrocarbon	0.8
CRP-94-05C	Unknown alcohol	0.9
CRP-94-05D	Unknown oxy hydrocarbon	0.4
CRP-94-05D	Unknown oxy hydrocarbon	0.5
CRP-94-05D	Unknown alcohol	0.7
CRP-94-06A	Long chain alcohol	1
CRP-94-06A	Long chain alcohol	2
CRP-94-06A	Unknown alkane	2

Tentatively Identified Compounds, SWMU 7 (continued)

Site ID	Tentative ID	Value (ug/g)
CRP-94-06A	Unknown aldehyde	0.3
CRP-94-06A	Unknown alkane	0.7
CRP-94-06A	Long chain alcohol	1
CRP-94-06A	Long chain alcohol	0.3
CRP-94-06A	Unknown polycyclic compound	1
CRP-94-06B	Hexadecanoic acid	0.7
CRP-94-06B	Long chain alcohol	2
CRP-94-06B	Unknown cyclic compound	0.2
CRP-94-06B	Long chain alcohol	0.4
CRP-94-06B	Long chain alcohol	0.6
CRP-94-06C	Hexadecanoic acid	0.6
CRP-94-06C	Long chain alcohol	1
CRP-94-06C	Long chain alcohol	0.5
CRP-94-07A	Long chain alcohol	2
CRP-94-07A	Unknown polycyclic compounc	0.4
CRP-94-07A	Long chain alcohol	0.3
CRP-94-07A	Long chain alcohol	2
CRP-94-07A	Alkane	2
CRP-94-07A	Alkane	0.8
CRP-94-07A	Long chain alcohol	0.8
CRP-94-07A	Unknown polycyclic compound	0.4
CRP-94-07A	Alkane	0.3
CRP-94-07A	Polycyclic compound	1
CRP-94-07B	Hexadecanoic acid	0.5
CRP-94-07B	Long chain alcohol	0.7
CRP-94-07C	Hexadecanoic acid	0.4
CRP-94-07C	Long chain alcohol	0.8
CRP-94-07C	Long chain alcohol	0.3
CRP-94-08A	Unknown ketone	0.4
CRP-94-08A	Hexadecanoic acid	0.3
CRP-94-08A	Unknown oxy hydrocarbon	0.3
CRP-94-08A	Unknown oxy hydrocarbon	2
CRP-94-08A	Unknown polycyclic hydrocarbon	0.3
CRP-94-08A	Unknown oxy hydrocarbon	0.4
CRP-94-08A	Unknown oxy hydrocarbon	3
CRP-94-08A	Unknown alkane	0.4
CRP-94-08A	Unknown oxy hydrocarbon	0.9
CRP-94-08A	Unknown alkane	0.4
CRP-94-08A	Unknown oxy hydrocarbon	1
CRP-94-08A	Unknown polycyclic hydrocarbon	0.5
CRP-94-08A	Unknown oxy hydrocarbon	0.3
CRP-94-08A	Unknown polycyclic hydrocarbon	1
CRP-94-08B	Unknown oxy hydrocarbon	0.4

Tentatively Identified Compounds, SWMU 7 (continued)

Site ID	Tentative ID	Value (ug/g)
CRP-94-08B	Hexadecanoic acid	0.5
CRP-94-08B	Unknown oxy hydrocarbon	1
CRP-94-08B	Unknown oxy hydrocarbon	0.5
CRP-94-08B	Unknown oxy hydrocarbon	0.4
CRP-94-08C	Unknown oxy hydrocarbon	0.4
CRP-94-08C	Hexadecanoic acid	0.8
CRP-94-08C	Unknown oxy hydrocarbon	0.9
CRP-94-09A	Unknown ketone	0.4
CRP-94-09A	Unknown oxy hydrocarbon	0.3
CRP-94-09A	Unknown oxy hydrocarbon	1
CRP-94-09A	Unknown oxy hydrocarbon	2
CRP-94-09A	Unknown oxy hydrocarbon	2
CRP-94-09A	Unknown oxy hydrocarbon	1
CRP-94-09A	Unknown alkane	0.6
CRP-94-09A	Unknown oxy hydrocarbon	0.4
CRP-94-09A	Unknown alkane	0.3
CRP-94-09A	Unknown oxy hydrocarbon	0.6
CRP-94-09A	Unknown alkane	0.3
CRP-94-09A	Unknown alkane	0.8
CRP-94-09A	Unknown oxy hydrocarbon	0.6
CRP-94-09A	Unknown oxy hydrocarbon	0.6
CRP-94-09A	Unknown alkane	0.3
CRP-94-09A	Unknown polycyclic hydrocarbon	0.6
CRP-94-09A	Unknown polycyclic hydrocarbon	0.5
CRP-94-09B	Hexadecanoic acid	0.5
CRP-94-09B	Hexadecanoic acid	0.4
CRP-94-09B	Unknown oxy hydrocarbon	0.8
CRP-94-09B	Unknown oxy hydrocarbon	0.8
CRP-94-09B	Unknown acid	0.4
CRP-94-09B	Unknown acid	0.4
CRP-94-09C	Unknown oxy hydrocarbon	0.5
CRP-94-09C	Unknown oxy hydrocarbon	0.5
CRP-94-09C	Hexadecanoic acid	0.7
CRP-94-09C	Hecadecanoic acid	0.5
CRP-94-09C	Unknown oxy hydrocarbon	0.6
CRP-94-09C	Unknown oxy hydrocarbon	1
CRP-94-09C	Unknown oxy hydrocarbon	0.5
CRP-94-10A	Unknown ketone	0.3
CRP-94-10A	Unknown oxy hydrocarbon	0.9
CRP-94-10A	Unknown oxy hydrocarbon	0.6
CRP-94-10A	Unknown alkane	0.3
CRP-94-10A	Unknown oxy hydrocarbon	0.4
CRP-94-10A	Unknown alkane	0.4

Tentatively Identified Compounds, SWMU 7 (continued)

Site ID	Tentative ID	Value (ug/g)
CRP-94-10A	Unknown oxy hydrocarbon	0.3
CRP-94-10A	Unknown polycyclic hydrocarbon	0.6
CRP-94-10B	Hexadecanoic acid	0.4
CRP-94-10B	Unknown oxy hydrocarbon	1
CRP-94-10B	Unknown acid	0.4
CRP-94-10B	Unknown nitrogen containing oxy hydrocarbon	0.7
CRP-94-10B	Unknown oxy hydrocarbon	0.5
CRP-94-10C	Hexadecanoic acid	0.5
CRP-94-10C	Unknown oxy hydrocarbon	0.6
CRP-94-10C	Unknown oxy hydrocarbon	0.4
CRP-94-11A	Unknown oxy hydrocarbon	0.8
CRP-94-11A	Unknown alkane	0.7
CRP-94-11A	Unknown alkane	1
CRP-94-11A	Unknown oxy hydrocarbon	0.5
CRP-94-11A	Unknown alkane	1
CRP-94-11A	Unknown oxy hydrocarbon	0.6
CRP-94-11B	Unknnown alcohol	0.3
CRP-94-11B	Hexadecanoic acid	0.5
CRP-94-11B	Unknown oxy hydrocarbon	2
CRP-94-11B	Unknown oxy hydrocarbon	0.5
CRP-94-11C	Unknown oxy hydrocarbon	0.3
CRP-94-11C	Hexadecanoic acid	0.3
CRP-94-11C	Unknown oxy hydrocarbon	1
CRP-94-11C	Unknown oxy hydrocarbon	0.3
CRP-94-12A	Benzaldehyde, 4-chloro-or isomer	0.5
CRP-94-12A	Chloronitrogen compound	0.6
CRP-94-12A	Unknown phthalate	0.8
CRP-94-12A	Hexadecanoic acid or isomer	0.8
CRP-94-12A	Octadecanoic acid or isomer	0.8
CRP-94-12A	Possible amide	0.3
CRP-94-12A	Unknown polycyclic hydrocarbon	0.5
CRP-94-12A	Possible amide	0.8
CRP-94-12A	Possible nitrogen containing + unknown	0.5
CRP-94-12A	Oxy hydrocarbon	3
CRP-94-12A	Possible nitrogen aromatic	0.5
CRP-94-12A	Oxy hydrocarbon	0.3
CRP-94-12A	Alkane	0.8
CRP-94-12A	Possible chlorinated aromatic	0.6
CRP-94-12A	Alkane	0.8
CRP-94-12A	Oxy hydrocarbon	0.5
CRP-94-12A	Possible chlorinated aromatic	0.8
CRP-94-12A	Chlorinated unknown	0.3
CRP-94-12A	Alkane	0.7

Tentatively Identified Compounds, SWMU 7 (continued)

Site ID	Tentative ID	Value (ug/g)
CRP-94-12A	Oxy hydrocarbon	2
CRP-94-12A	Chlorinated	0.4
CRP-94-12A	Dichloro compound	0.5
CRP-94-12A	Oxy hydrocarbon	0.7
CRP-94-12A	Oxy hydrocarbon	0.3
CRP-94-12B	Oxy hydrocarbon	0.3
CRP-94-12B	Unknown phthalate	0.7
CRP-94-12B	Hexadecanoic acid or isomer	0.8
CRP-94-12B	Octadecanoic acid or isomer	0.3
CRP-94-12B	Oxy hydrocarbon	4
CRP-94-12B	Oxy hydrocarbon	0.4
CRP-94-12B	Unknown polycyclic	0.8
CRP-94-12B	Oxy hydrocarbon	1
CRP-94-12B	Oxy hydrocarbon	0.4
CRP-94-12C	Unknown phthalate	0.4
CRP-94-12C	Hexadecanoic acid or isomer	0.8
CRP-94-12C	Octadecanoic acid or isomer	0.3
CRP-94-12C	Oxy hydrocarbon	3
CRP-94-12C	Oxy hydrocarbon	0.3
CRP-94-12C	Oxy hydrocarbon	1
CRP-94-12C	Oxy hydrocarbon	0.3
CRP-94-13A	Unknown phthalate	0.8
CRP-94-13A	Hexadecanoic acid or isomer	0.8
CRP-94-13A	Oxyhydrocarbon	3
CRP-94-13A	Alkane	1
CRP-94-13A	Alkane	0.3
CRP-94-13A	Alkane	0.7
CRP-94-13A	Oxy hydrocarbon	0.3
CRP-94-13A	Alkane	0.4
CRP-94-13A	Oxy hydrocarbon	1
CRP-94-13A	Oxy hydrocarbon	0.4
CRP-94-13B	Oxy hydrocarbon	0.3
CRP-94-13B	Unknown phthalate	1
CRP-94-13B	Unknown phthalate	0.3
CRP-94-13B	Hexadecanoic acid or isomer	5
CRP-94-13B	Oxy hydrocarbon	3
CRP-94-13B	Oxy hydrocarbon	0.3
CRP-94-13B	Oxy hydrocarbon	1
CRP-94-13B	Oxy hydrocarbon	0.3
CRP-94-13C	Unknown phthalate	0.7
CRP-94-13C	Hexadecanoic acid or isomer	0.7
CRP-94-13C	Oxy hydrocarbon	3

Tentatively Identified Compounds, SWMU 7 (continued)

Site ID	Tentative ID	Value (ug/g)
CRP-94-13C	Oxy hydrocarbon	0.3
CRP-94-13C	Oxy hydrocarbon	0.9
CRP-94-13C	Oxy hydrocarbon	0.3
CRP-94-14A	Possible alkene	0.4
CRP-94-14A	Oxy hydrocarbon	0.3
CRP-94-14A	Unknown phthalate	0.7
CRP-94-14A	Hexadecanoic acid or isomer	1
CRP-94-14A	Octadecanoic acid or isomer	0.3
CRP-94-14A	Oxy hydrocarbon	3
CRP-94-14A	Oxy aromatic	0.5
CRP-94-14A	Oxy hydrocarbon	1
CRP-94-14A	Oxy hydrocarbon	0.3
CRP-94-14A	Oxy hydrocarbon	1
CRP-94-14A	Oxy hydrocarbon	0.3
CRP-94-14A	Oxy hydrocarbon	0.5
CRP-94-14B	Oxy hydrocarbon	0.6
CRP-94-14B	Unknown phthalate	1
CRP-94-14B	Hexadecanoic acid or isomer	1
CRP-94-14B	Oxy hydrocarbon	2
CRP-94-14B	Oxy hydrocarbon	0.3
CRP-94-14B	Oxy hydrocarbon	0.9
CRP-94-14B	Oxy hydrocarbon	0.3
CRP-94-14C	Oxy hydrocarbon	0.3
CRP-94-14C	Oxy hydrocarbon	0.3
CRP-94-14C	Unknown phthalate	0.7
CRP-94-14C	Hexadecanoic acid or isomer	0.6
CRP-94-14C	Oxy hydrocarbon	2
CRP-94-14C	Oxy hydrocarbon	0.3
CRP-94-14C	Oxy hydrocarbon	0.8
CRP-94-14C	Oxy hydrocarbon	0.3
CRP-94-15A	Unknown phthalate	0.7
CRP-94-15A	Hexadecanoic acid or isomer	0.5
CRP-94-15A	Oxy aromatic	0.6
CRP-94-15A	Oxy hydrocarbon	3
CRP-94-15A	Oxy hydrocarbon	0.4
CRP-94-15A	Oxy hydrocarbon	1
CRP-94-15A	Oxy hydrocarbon	0.3
CRP-94-15B	Oxy hydrocarbon	0.3
CRP-94-15B	Unknown phthalate	0.4
CRP-94-15B	Hexadecanoic acid or isomer	0.6
CRP-94-15B	Oxy hydrocarbon	3
CRP-94-15B	Oxy hydrocarbon	0.4
CRP-94-15B	Oxy hydrocarbon	0.9

Tentatively Identified Compounds, SWMU 7 (continued)

Site ID	Tentative ID	Value (ug/g)
CRP-94-15B	Oxy hydrocarbon	0.3
CRP-94-15C	Oxy hydrocarbon	0.3
CRP-94-15C	Unknown phthalate	0.9
CRP-94-15C	Hexadecanoic acid or isomer	0.8
CRP-94-15C	Oxy hydrocarbon	3
CRP-94-15C	Oxy hydrocarbon	0.5
CRP-94-15C	Oxy hydrocarbon	1
CRP-94-15C	Oxy hydrocarbon	0.4
CRS-94-01	Alcohol	0.6
CRS-94-01	Alkane	0.5
CRS-94-01	Alkane	1
CRS-94-01	Alkane	1
CRS-94-01	Alcohol	0.5
CRS-94-01	Alkane	0.3
CRS-94-02	Oxy hydrocarbon	0.3
CRS-94-02	Alcohol	0.7
CRS-94-02	Alkane	0.8
CRS-94-02	Alkane	2
CRS-94-02	Alkane	0.9
CRS-94-02	Alcohol	0.7
CRS-94-02	Polycyclic hydrocarbon	0.5
CRS-94-03	Alcohol	0.5
CRS-94-03	Alkane	0.5
CRS-94-03	Alkane	1
CRS-94-03	Alkane	0.8
CRS-94-04	Alcohol	0.9
CRS-94-04	Alkane	0.7
CRS-94-04	Alkane	1
CRS-94-04	Alkane	0.8
CRS-94-04	Alcohol	0.4
CRS-94-04	Alkane	0.3
CRS-94-04	Polycyclic hydrocarbon	0.5
CRS-94-05	Oxy hydrocarbon	0.8
CRS-94-05	Polycyclic hydrocarbon	0.4
CRS-94-05	Alkane	0.6
CRS-94-05	Alkane	1
CRS-94-05	Alkane	0.9
CRS-94-05	Oxy hydrocarbon	0.6
CRS-94-05	Alkane	0.3
CRS-94-06	Alcohol	0.7
CRS-94-06	Alkane	0.8
CRS-94-06	Alkane	2
CRS-94-06	Aldehyde	0.4

Tentatively Identified Compounds, SWMU 7 (continued)

Site ID	Tentative ID	Value (ug/g)
CRS-94-06	Alkane	3
CRS-94-06	Oxy hydrocarbon	2
CRS-94-06	Alkane	1
CRS-94-06	Polycyclic hydrocarbon	0.8
CRS-94-07	Oxy hydrocarbon	0.6
CRS-94-07	Alkane	0.5
CRS-94-07	Alkane	1
CRS-94-07	Alkane	0.9
CRS-94-07	Alcohol	2
CRS-94-07	Alkane	0.3
CRS-94-07	Polycyclic hydrocarbon	0.4
CRS-94-08	Oxy hydrocarbon	0.4
CRS-94-08	Alkane	0.5
CRS-94-08	Alkane	1
CRS-94-08	Alkane	1
CRS-94-08	Oxy hydrocarbon	0.5
CRS-94-08	Alkane	0.4
CRS-94-09	Oxy hydrocarbon	0.3
CRS-94-09	Alkane	0.3
CRS-94-09	Alkane	0.7
CRS-94-09	Alkane	0.6
CRS-94-09	Alcohol	0.8
CRS-94-10	Oxy hydrocarbon	0.3
CRS-94-10	Polycyclic hydrocarbon	0.3
CRS-94-10	Oxy hydrocarbon	0.3
CRS-94-10	Alkane	0.3
CRS-94-10	Alkane	0.5
CRS-94-10	Alkane	0.4
CRS-94-10	Alcohol	0.4
CRS-94-10	Unknown	0.3
CRS-94-11	Alkane	0.3
CRS-94-11	Alkane	0.3
CRS-94-11	Alkane	0.6
CRS-94-11	Alkane	0.6
CRS-94-11	Oxy hydrocarbon	0.6
CRS-94-12	Alcohol	0.4
CRS-94-12	Alkane	0.3
CRS-94-12	Alkane	0.6
CRS-94-12	Alkane	0.8
CRS-94-12	Cyclic oxy hydrocarbon	0.7
CRS-94-12	Alkane	0.4
CRS-94-13	Oxy hydrocarbon	0.6
CRS-94-13	Alkane	0.9

Tentatively Identified Compounds, SWMU 7 (continued)

Site ID	Tentative ID	Value (ug/g)
CRS-94-13	Alkane	0.7
CRS-94-13	Alcohol	0.3
CRS-94-13	Alkane	0.5
CRS-94-14	Unknown unsaturated oxy hydrocarbon	0.6
CRS-94-14	Unknown alcohol	1
CRS-94-14	Unknown alcohol	0.6
CRS-94-14	Unknown alkane	0.5
CRS-94-14	Unknown unsaturated alcohol	0.5
CRS-94-14	Unknown alkane	0.9
CRS-94-14	Unknown alkane	0.9
CRS-94-14	Unknown alcohol	0.3
CRS-94-14	Unknown alkane	0.5
CRS-94-15	Unknown unsaturated oxy hydrocarbon	0.3
CRS-94-15	Unknown alkyl pentanone isomer	0.3
CRS-94-15	Unknown alcohol	0.7
CRS-94-15	Unknown alkane	0.6
CRS-94-15	Unknown alkane	0.6
CRS-94-15	Unknown alkane	0.4
CRS-94-15	Unknown alcohol	0.3
CRS-94-16	Unknown alkyl pentanone isomer	0.3
CRS-94-16	Unknown alcohol	0.5
CRS-94-16	Unknown alkane	0.5
CRS-94-16	Unknown alkane	0.5
CRS-94-16	Unknown alkane	0.4
CRS-94-17	Unknown alkyl pentanone isomer	0.4
CRS-94-17	Unknown chloro oxy cyclic hydrocarbon	0.3
CRS-94-17	Unknown unsaturated oxy hydrocarbon	1
CRS-94-17	Unknown alcohol	1
CRS-94-17	Unknown alkane	0.4
CRS-94-17	Unknown alkane	0.7
CRS-94-17	Unknown alkane	0.5
CRS-94-17	Unknown alcohol	0.3
CRS-94-18	Unknown alkyl pentanone isomer	0.3
CRS-94-18	Unknown unsaturated oxy hydrocarbon	0.5
CRS-94-18	Unknown alcohol	1
CRS-94-18	Unknown alkane	0.6
CRS-94-18	Unknown alkane	0.7
CRS-94-18	Unknown alkane	0.3

Tentatively Identified Compounds, SWMU 13

Site ID	Tentative ID	Value (ug/g)
TDP-94-01A	Alkane	0.3
TDP-94-01B	Hexadecanoic acid	0.5
TDP-94-01B	Alcohol	1
TDP-94-01B	Oxy hydrocarbon	0.3
TDP-94-01B	Alcohol	0.4
TDP-94-02A	Oxy hydrocarbon	0.3
TDP-94-02A	Cyclic oxy hydrocarbon	0.3
TDP-94-02B	Hexadecanoic acid	0.6
TDP-94-02B	Alcohol	0.5
TDP-94-02B	Cyclic oxy hydrocarbon	1
TDP-94-03A	Unknown oxy hydrocarbon	0.4
TDP-94-03A	Unknown unsaturated hydrocarbon	0.8
TDP-94-03A	Unknown acid	0.8
TDP-94-03A	2-Pentanone, 4-hydroxy-4-methyl-	0.4
TDP-94-03A	Unknown oxy chlorinated hydrocarbon	0.4
TDP-94-03B	2-Pentanone, 4-hydroxy-4-methyl-	0.6
TDP-94-03B	Unknown oxy chlorinated hydrocarbon	0.5
TDP-94-03B	Unknown oxy chlorinated hydrocarbon	2
TDP-94-03B	Unknown oxy hydrocarbon	0.7
TDP-94-04A	Unknown oxy hydrocarbon	0.3
TDP-94-04A	Unknown oxy hydrocarbon	1
TDP-94-04A	Unknown oxy hydrocarbon	0.3
TDP-94-04A	Unknown unsaturated hydrocarbon	0.7
TDP-94-04A	Unknown acid	0.8
TDP-94-04A	2-Pentanone, 4-hydroxy-4-methyl-	0.5
TDP-94-04A	Unknown oxy chlorinated hydrocarbon	0.3
TDP-94-04B	2-Pentanone, 4-hydroxy-4-methyl-	0.5
TDP-94-04B	Unknown oxy chlorinated hydrocarbon	0.3
TDP-94-04B	Unknown oxy chlorinated hydrocarbon	1
TDP-94-04B	Unknown oxy hydrocarbon	0.5
TDP-94-05A	Unknown oxy hydrocarbon	0.3
TDP-94-05A	Unknown oxy hydrocarbon	2
TDP-94-05A	Unknown oxy hydrocarbon	0.3
TDP-94-05A	Unknown oxy hydrocarbon	0.3
TDP-94-05A	Unknown unsaturated hydrocarbon	0.9
TDP-94-05A	Unknown oxy hydrocarbon	0.3
TDP-94-05A	Unknown propenoic acid	1
TDP-94-05A	2-Pentanone, 4-hydroxy-4-methyl-	0.5
TDP-94-05A	Unknown oxy hydrocarbon	0.4
TDP-94-05B	2-Pentanone, 4--hydroxy-4-methyl-	0.8
TDP-94-05B	Unknown oxy chlorinated hydrocarbon	0.3
TDP-94-05B	Unknown oxy chlorinated hydrocarbon	2
TDP-94-05B	Unknown oxy hydrocarbon	0.3

Tentatively Identified Compounds, SWMU 13 (continued)

Site ID	Tentative ID	Value (ug/g)
TDP-94-06A	2-Pentanone, 4-hydroxy-4-methyl-	1
TDP-94-06A	Unknown oxy chlorinated hydrocarbon	0.4
TDP-94-06A	Unknown oxy chlorinated hydrocarbon	1
TDP-94-06B	2-Pentanone, 4-hydroxy-4-methyl-	0.6
TDP-94-06B	Unknown oxy chlorinated hydrocarbon	0.3
TDP-94-06B	Unknown oxy chlorinated hydrocarbon	1
TDP-94-06B	Unknown chlorinated hydrocarbon	0.4
TDP-94-07A	2-Pentanone, 4-hydroxy-4-methyl-	0.8
TDP-94-07A	Unknown oxy chlorinated hydrocarbon	0.8
TDP-94-07A	Unknown oxy hydrocarbon	0.3
TDP-94-07A	Unknown alkane	0.3
TDP-94-07A	Unknown alkane	0.3
TDP-94-07B	2-Pentanone, 4-hydroxy-4-methyl-	0.6
TDP-94-07B	Unknown oxy chlorinated hydrocarbon	0.3
TDP-94-07B	Unknown oxy chlorinated hydrocarbon	2
TDP-94-07B	Unknown oxy hydrocarbon	0.5
TDP-94-08A	Cyclic hydrocarbon	6
TDP-94-08A	Alkane	3
TDP-94-08A	Alkane	10
TDP-94-08A	Alkane	9
TDP-94-08A	Alkane	6
TDP-94-08A	Alkane	4
TDP-94-08A	Methyl Alkane	3
TDP-94-08A	Alkane	3
TDP-94-08A	Alkane	9
TDP-94-08A	Hydrocarbon	3
TDP-94-08A	Alkane	10
TDP-94-08A	Oxy hydrocarbon	3
TDP-94-08A	Alkane	4
TDP-94-08A	Alkane	6
TDP-94-08A	Cyclic hydrocarbon	4
TDP-94-08A	Alkane	3
TDP-94-08A	Alkane	10
TDP-94-08A	Cyclic hydrocarbon	4
TDP-94-08A	Alkane	20
TDP-94-08A	Alkane	3
TDP-94-08A	Alkane	5
TDP-94-08A	Alkane	10
TDP-94-08A	Cyclic hydrocarbon	5
TDP-94-08A	Alkane	10
TDP-94-08A	Alkane	6
TDP-94-08A	Alkane	20
TDP-94-08A	Unsaturated hydrocarbon	3

Tentatively Identified Compounds, SWMU 13 (continued)

Site ID	Tentative ID	Value (ug/g)
TDP-94-08A	Unsaturated hydrocarbon	6
TDP-94-08A	Alkane	5
TDP-94-08A	Alkane	10
TDP-94-08A	Cyclic hydrocarbon	7
TDP-94-08A	Alkane	8
TDP-94-08A	Alkane	10
TDP-94-08A	Alkane	10
TDP-94-08A	Hydrocarbon	10
TDP-94-08A	Alkane	6
TDP-94-08A	Cyclic hydrocarbon	5
TDP-94-08A	Cyclic hydrocarbon	9
TDP-94-08A	Alkane	6
TDP-94-08A	Alkane	20
TDP-94-08A	Unsaturated hydrocarbon	3
TDP-94-08A	Hydrocarbon	40
TDP-94-08A	Oxy hydrocarbon	4
TDP-94-08A	Cyclic hydrocarbon	8
TDP-94-08A	Alkane	3
TDP-94-08A	Alkane	6
TDP-94-08A	Alkane	30
TDP-94-08A	Unsaturated hydrocarbon	10
TDP-94-08A	Hydrocarbon	3
TDP-94-08A	Alkane	10
TDP-94-08A	Cyclic hydrocarbon	20
TDP-94-08A	Alkane	20
TDP-94-08A	Alkane	30
TDP-94-08A	Alkane	10
TDP-94-08A	Polycyclic hydrocarbon	20
TDP-94-08A	Hydrocarbon	20
TDP-94-08A	Alkane	10
TDP-94-08A	Alkane	20
TDP-94-08A	Alkane	4
TDP-94-08A	Alkane	3
TDP-94-08A	Alkane	3
TDP-94-08A	Alkane	10
TDP-94-08A	Alkane	10
TDP-94-08A	Cyclic hydrocarbon	8
TDP-94-08A	Alkane	7
TDP-94-08A	Alkane	6
TDP-94-08A	Polycyclic hydrocarbon	10
TDP-94-08A	Alkane	6
TDP-94-08A	Cyclic hydrocarbon	5
TDP-94-08A	Alkane	5

Tentatively Identified Compounds, SWMU 13 (continued)

Site ID	Tentative ID	Value (ug/g)
TDP-94-08A	Alkane	3
TDP-94-08B	Oxy hydrocarbon	0.5
TDP-94-08B	Alkane	0.3
TDP-94-08B	Oxy hydrocarbon	0.9
TDP-94-08B	Oxy hydrocarbon	0.3
TDP-94-09A	Hydrocarbon	0.3
TDP-94-09A	Oxy hydrocarbon	0.5
TDP-94-09A	Oxy hydrocarbon	0.5
TDP-94-09A	Unknown alkyl pentanone	0.8
TDP-94-09A	Unknown acid	6
TDP-94-09A	Unknown alcohol	0.7
TDP-94-09B	Oxy hydrocarbon	0.7
TDP-94-09B	Oxy hydrocarbon	1
TDP-94-09B	Unknown alkyl pentanone	0.7
TDP-94-09B	Unknown acid	6
TDP-94-09B	Hexadecanoic acid	0.3
TDP-94-09B	Unknown alcohol	0.9
TDP-94-10A	Oxy hydrocarbon	0.3
TDP-94-10A	Oxy hydrocarbon	0.4
TDP-94-10A	Alkane	0.3
TDP-94-10A	Alkane	0.4
TDP-94-10B	Oxy hydrocarbon	0.4
TDP-94-11A	Alkane	3
TDP-94-11A	Alkane	4
TDP-94-11A	Alkane	10
TDP-94-11A	Alkane	9
TDP-94-11A	Alkane	7
TDP-94-11A	Alkane	4
TDP-94-11A	Alkane	3
TDP-94-11A	Alkane	8
TDP-94-11A	Alkane	10
TDP-94-11A	Alkane	3
TDP-94-11A	Alkane	4
TDP-94-11A	Alkane	5
TDP-94-11A	Cyclic hydrocarbon	3
TDP-94-11A	Alkane	9
TDP-94-11A	Cyclic hydrocarbon	3
TDP-94-11A	Alkane	20
TDP-94-11A	Alkane	3
TDP-94-11A	Alkane	4
TDP-94-11A	Alkane	10
TDP-94-11A	Cyclic hydrocarbon	4
TDP-94-11A	Alkane	7

Tentatively Identified Compounds, SWMU 13 (continued)

Site ID	Tentative ID	Value (ug/g)
TDP-94-11A	Alkane	5
TDP-94-11A	Alkane	20
TDP-94-11A	Hydrocarbon	5
TDP-94-11A	Oxy hydrocarbon	5
TDP-94-11A	Alkane	5
TDP-94-11A	Cyclic hydrocarbon	5
TDP-94-11A	Alkane	6
TDP-94-11A	Alkane	9
TDP-94-11A	Alkane	10
TDP-94-11A	Alkane	9
TDP-94-11A	Hydrocarbon	3
TDP-94-11A	Cyclic hydrocarbon	4
TDP-94-11A	Cyclic hydrocarbon	7
TDP-94-11A	Oxy hydrocarbon	5
TDP-94-11A	Alkane	10
TDP-94-11A	Alkane	20
TDP-94-11A	Unsaturated hydrocarbon	7
TDP-94-11A	Alkane	4
TDP-94-11A	Cyclic Hydrocarbon	3
TDP-94-11A	Cyclic Hydrocarbon	10
TDP-94-11A	Alkane	3
TDP-94-11A	Hydrocarbon	3
TDP-94-11A	Hydrocarbon	20
TDP-94-11A	Unsaturated hydrocarbon	9
TDP-94-11A	Alkane	10
TDP-94-11A	Alkane	8
TDP-94-11A	Alkane	20
TDP-94-11A	Alkane	9
TDP-94-11A	Alkane	20
TDP-94-11A	Alkane	10
TDP-94-11A	Cyclic hydrocarbon	8
TDP-94-11A	Alkane	20
TDP-94-11A	Alkane	5
TDP-94-11A	Alkane	4
TDP-94-11A	Alkane	10
TDP-94-11A	Alkane	10
TDP-94-11A	Alkane	7
TDP-94-11A	Alkane	9
TDP-94-11A	Polycyclic hydrocarbon	10
TDP-94-11A	Hydrocarbon	7
TDP-94-11A	Polycyclic hydrocarbon	9
TDP-94-11A	Alkane	7
TDP-94-11A	Polycyclic hydrocarbon	3

Tentatively Identified Compounds, SWMU 13 (continued)

Site ID	Tentative ID	Value (ug/g)
TDP-94-11A	Polycyclic hydrocarbon	4
TDP-94-11A	Alkane	8
TDP-94-11B	Hydrocarbon	0.7
TDP-94-11B	Cyclic hydrocarbon	0.3
TDP-94-11B	Oxy hydrocarbon	2
TDP-94-11B	Usaturated hydrocarbon	0.3
TDP-94-11B	Oxy hydrocarbon	0.5
TDP-94-12A	Hydrocarbon	0.3
TDP-94-12A	Oxy hydrocarbon	0.5
TDP-94-12A	Alkane	0.3
TDP-94-12A	Alkane	0.3
TDP-94-12B	Oxy hydrocarbon	0.8
TDP-94-12B	Oxy hydrocarbon	0.4
TDP-94-13A	Unknown unsaturated oxy hydrocarbon	0.3
TDP-94-13A	Unknown acid	10
TDP-94-13A	Unknown alkane	0.3
TDP-94-13A	Unknown alkane	0.4
TDP-94-13B	Unknown alkyl pentanone	4
TDP-94-13B	Unknown acid	80
TDP-94-13B	Unknown alcohol	4
TDP-94-14A	Unknown alkyl pentanone	0.4
TDP-94-14A	Unknown acid	8
TDP-94-14A	Unknown alcohol	0.8
TDP-94-14A	Unknown alkane	0.3
TDP-94-14B	Unknown alkyl pentanone	1
TDP-94-14B	Unknown acid	8
TDP-94-14B	Hexadecanoic acid	0.4
TDP-94-14B	Unknown alcohol	1
TDP-94-15A	Unknown alkyl pentanone	0.5
TDP-94-15A	Unknown acid	6
TDP-94-15A	Unknown alcohol	0.7
TDP-94-15B	Unknown alkyl pentanone	0.6
TDP-94-15B	Unknown acid	7
TDP-94-15B	Hexadecanoic acid	0.8
TDP-94-15B	Unknown alcohol	1

Tentatively Identified Compounds, SWMU 23

Site ID	Tentative ID	Value (ug/g)
BRB-94-02A	Methyl ester	0.3
BRB-94-02A	Unknown alkane	0.3
BRB-94-02A	Unknown aromatic	0.6
BRB-94-02A	Unknown alcohol	0.7
BRB-94-02A	Unknown alkane	0.4
BRB-94-02A	Unknown alkane	0.3
BRB-94-02B	Unknown nitrogen containing oxy hydrocarbon	2
BRB-94-02B	Unknown oxy hydrocarbon	0.3
BRB-94-02B	Unknown oxy hydrocarbon	0.5
BRB-94-02B	Dodecane	0.7
BRB-94-02B	Unknown alkane	0.5
BRB-94-02B	Tridecane	0.8
BRB-94-02B	Unknown alkane	0.6
BRB-94-02B	Tetradecane	1
BRB-94-02B	Unknown alkane	0.5
BRB-94-02B	Pentadecane	0.6
BRB-94-02B	Hexadecane	0.4
BRB-94-02B	Heptadecane	0.6
BRB-94-02B	Unknown alkane	0.3
BRB-94-02B	Polynuclear aromatic, MW=198	0.4
BRB-94-02B	Unknown alkane	0.3
BRB-94-02B	Polynuclear aromatic, MW=198	0.4
BRB-94-02B	Polynuclear aromatic, MW=192	0.9
BRB-94-02B	Polynuclear aromatic, MW=192	1
BRB-94-02B	Hexadecanoic acid	0.7
BRB-94-02B	Polynuclear aromatic, MW=192	0.5
BRB-94-02B	Polynuclear aromatic, MW=212	0.4
BRB-94-02B	Unknown alkane	0.4
BRB-94-02B	Polynuclear aromatic, MW=212	0.4
BRB-94-02B	Polynuclear aromatic, MW=212	0.8
BRB-94-02B	Polynuclear aromatic, MW=206	0.4
BRB-94-02B	Polynuclear aromatic, MW=206	0.9
BRB-94-02B	Polynuclear aromatic, MW=206	0.6
BRB-94-02B	Polynuclear aromatic, MW=206	2
BRB-94-02B	Polynuclear aromatic, MW=206	0.4
BRB-94-02B	Polynuclear aromatic, MW=206	0.8
BRB-94-02B	Polynuclear aromatic, MW=206	0.6
BRB-94-02B	Heneicosane	0.5
BRB-94-02B	Polynuclear aromatic, MW=226	0.7
BRB-94-02B	Polynuclear aromatic, MW=226	0.3
BRB-94-02B	Polynuclear aromatic, MW=220	0.4
BRB-94-02B	Polynuclear aromatic, MW=220	0.8
BRB-94-02B	Polynuclear aromatic, MW=220	1

Tentatively Identified Compounds, SWMU 23 (continued)

Site ID	Tentative ID	Value (ug/g)
BRB-94-02B	Polynuclear aromatic, MW=220	1
BRB-94-02B	Docosane	1
BRB-94-02B	Polynuclear aromatic, MW=220	0.8
BRB-94-02B	Polynuclear aromatic, MW=220	0.7
BRB-94-02B	Polynuclear aromatic, MW=220	0.6
BRB-94-02B	Polynuclear aromatic, MW=220	0.4
BRB-94-02B	Polynuclear aromatic, MW=216	1
BRB-94-02B	Polynuclear aromatic, MW=234	0.5
BRB-94-02B	Polynuclear aromatic, MW=216	2
BRB-94-02B	Tricosane	2
BRB-94-02B	Polynuclear aromatic, MW=234	0.5
BRB-94-02B	Polynuclear aromatic, MW=216	1
BRB-94-02B	Polynuclear aromatic, MW=216	1
BRB-94-02B	Polynuclear aromatic, MW=234	1
BRB-94-02B	Polynuclear aromatic, MW=230	0.7
BRB-94-02B	Polynuclear aromatic, MW=236	0.5
BRB-94-02B	Polynuclear aromatic, MW=230	0.8
BRB-94-02B	Tetracosane	2
BRB-94-02B	Polynuclear aromatic, MW=230	1
BRB-94-02B	Polynuclear aromatic, MW=230	1
BRB-94-02B	Polynuclear aromatic, MW=230	2
BRB-94-02B	Polynuclear aromatic, MW=230	1
BRB-94-02B	Polynuclear aromatic, MW=230	0.6
BRB-94-02B	Pentacosane	3
BRB-94-02B	Polynuclear aromatic, MW=244	1
BRB-94-02B	Polynuclear aromatic, MW=248	1
BRB-94-02B	Hexacosane	2
BRB-94-02B	Polynuclear aromatic, MW=248	0.3
BRB-94-02B	Polynuclear aromatic, MW=242	1
BRB-94-02B	Polynuclear aromatic, MW=242	0.4
BRB-94-02B	Polynuclear aromatic, MW=262	0.3
BRB-94-02B	Unknown alkane	2
BRB-94-02B	Polynuclear aromatic, MW=256	0.6
BRB-94-02B	Polynuclear aromatic, MW=256	0.4
BRB-94-02B	Unknown alkane	1
BRB-94-02B	Polynuclear aromatic, MW=270	0.5
BRB-94-02B	Unknown alkane	1
BRB-94-02B	Unknown alkane	0.8
BRB-94-02B	Unknown alkane	0.5
BRB-94-02B	Unknown oxy hydrocarbon	0.3
BRB-94-02B	Unknown alkane	0.3
BRB-94-02C	Unknown nitrogen containing oxy hydrocarbon	0.5
BRB-94-02C	Unknown unsaturated hydrocarbon	0.8

Tentatively Identified Compounds, SWMU 23 (continued)

Site ID	Tentative ID	Value (ug/g)
BRB-94-02C	Unknown unsaturated hydrocarbon	0.6
BRB-94-02C	Hexadecanoic acid	0.4
BRB-94-02C	Unknown oxy hydrocarbon	1
BRB-94-02C	Unknown oxy hydrocarbon	0.4
BRB-94-04A	Unknown nitrogen containing hydrocarbon	1
BRB-94-04A	Dodecane	0.3
BRB-94-04A	Unknown hydrocarbon	0.4
BRB-94-04A	Tridecane	0.3
BRB-94-04A	Tetradecane	0.4
BRB-94-04A	Unknown alkane	0.3
BRB-94-04A	Polynuclear aromatic, MW=192	0.3
BRB-94-04A	Hexadecanoic acid	0.4
BRB-94-04A	Polynuclear aromatic, MW=206	0.5
BRB-94-04A	Polynuclear aromatic, MW=220	0.3
BRB-94-04A	Polynuclear aromatic, MW=220	0.3
BRB-94-04A	Unknown alkane	0.3
BRB-94-04A	Polynuclear aromatic, MW=216	0.3
BRB-94-04A	Polynuclear aromatic, MW=216	0.3
BRB-94-04A	Heptadecane	0.5
BRB-94-04A	Polynuclear aromatic, MW=216	0.4
BRB-94-04A	Unknown alkane	0.5
BRB-94-04A	Polynuclear aromatic, MW=230	0.3
BRB-94-04A	Polynuclear aromatic, MW=230	0.4
BRB-94-04A	Unknown oxy hydrocarbon	2
BRB-94-04A	Unknown alkane	0.8
BRB-94-04A	Polynuclear aromatic, MW=242	0.4
BRB-94-04A	Unknown alkane	0.8
BRB-94-04A	Unknown alkane	0.5
BRB-94-04A	Unknown alkane	0.6
BRB-94-04A	Unknown alkane	0.3
BRB-94-04B	Unknown nitrogen containing hydrocarbon	1
BRB-94-04B	Unknown oxy hydrocarbon	0.3
BRB-94-04B	Tridecane	0.3
BRB-94-04B	Tetradecane	0.5
BRB-94-04B	Unknown alkane	0.3
BRB-94-04B	Unknown alkane	0.3
BRB-94-04B	Polynuclear aromatic, MW=192	0.5
BRB-94-04B	Polynuclear aromatic, MW=192	0.6
BRB-94-04B	Unknown alkane	0.3
BRB-94-04B	Polynuclear aromatic, MW=212	0.4
BRB-94-04B	Polynuclear aromatic, MW=206	0.5
BRB-94-04B	Polynuclear aromatic, MW=206	0.3
BRB-94-04B	Polynuclear aromatic, MW=206	1

Tentatively Identified Compounds, SWMU 23 (continued)

Site ID	Tentative ID	Value (ug/g)
BRB-94-04B	Polynuclear aromatic, MW=206	0.5
BRB-94-04B	Polynuclear aromatic, MW=206	0.3
BRB-94-04B	Polynuclear aromatic, MW=206	0.4
BRB-94-04B	Unknown alkane	0.8
BRB-94-04B	Polynuclear aromatic, MW=226	0.3
BRB-94-04B	Polynuclear aromatic, MW=220	0.4
BRB-94-04B	Polynuclear aromatic, MW=220	0.5
BRB-94-04B	Polynuclear aromatic, MW=220	0.8
BRB-94-04B	Unknown alkane	0.6
BRB-94-04B	Polynuclear aromatic, MW=220 and 240	0.3
BRB-94-04B	Polynuclear aromatic, MW=220 and 240	0.3
BRB-94-04B	Polynuclear aromatic, MW=216	0.5
BRB-94-04B	Polynuclear aromatic, MW=216	0.8
BRB-94-04B	Tricosane	0.9
BRB-94-04B	Polynuclear aromatic, MW=216 and 254	0.6
BRB-94-04B	Polynuclear aromatic, MW=216	0.5
BRB-94-04B	Polynuclear aromatic, MW=234	0.5
BRB-94-04B	Polynuclear aromatic, MW=230	0.3
BRB-94-04B	Nonadecane	1
BRB-94-04B	Polynuclear aromatic, MW=230	0.6
BRB-94-04B	Polynuclear aromatic, MW=230	0.5
BRB-94-04B	Polynuclear aromatic, MW=230	0.7
BRB-94-04B	Polynuclear aromatic, MW=230	0.3
BRB-94-04B	Unknown alkane	2
BRB-94-04B	Polynuclear aromatic, MW=244	0.4
BRB-94-04B	Polynuclear aromatic, MW=248	0.5
BRB-94-04B	Unknown alkane	1
BRB-94-04B	Polynuclear aromatic, MW=242	0.6
BRB-94-04B	Unknown alkane	1
BRB-94-04B	Polynuclear aromatic, MW=256	0.4
BRB-94-04B	Unknown alkane	0.6
BRB-94-04B	Unknown alkane	0.5
BRB-94-04B	Unknown alkane	0.3
BRB-94-04C	Unknown nitrogen containing hydrocarbon	1
BRB-94-04C	Dodecane	0.7
BRB-94-04C	Unknown alkane	0.6
BRB-94-04C	Tridecane	0.9
BRB-94-04C	Unknown alkane	0.9
BRB-94-04C	Tetradecane	2
BRB-94-04C	Unknown alkane	0.5
BRB-94-04C	Pentadecane	1
BRB-94-04C	Hexadecane	0.7
BRB-94-04C	Heptadecane	0.9

Tentatively Identified Compounds, SWMU 23 (continued)

Site ID	Tentative ID	Value (ug/g)
BRB-94-04C	Polynuclear aromatic, MW=198	0.8
BRB-94-04C	Nonadecane	0.5
BRB-94-04C	Polynuclear aromatic, MW=198	0.7
BRB-94-04C	Polynuclear aromatic, MW=192	2
BRB-94-04C	Polynuclear aromatic, MW=192	2
BRB-94-04C	Polynuclear aromatic, MW=192	0.6
BRB-94-04C	Polynuclear aromatic, MW=192	1
BRB-94-04C	Polynuclear aromatic, MW=212	0.7
BRB-94-04C	Eicosane	0.9
BRB-94-04C	Polynuclear aromatic, MW=212	0.8
BRB-94-04C	Polynuclear aromatic, MW=212	2
BRB-94-04C	Polynuclear aromatic, MW=212	0.7
BRB-94-04C	Polynuclear aromatic, MW=212	0.7
BRB-94-04C	Polynuclear aromatic, MW=206	1
BRB-94-04C	Polynuclear aromatic, MW=206	2
BRB-94-04C	Polynuclear aromatic, MW=206	1
BRB-94-04C	Polynuclear aromatic, MW=206	4
BRB-94-04C	Polynuclear aromatic, MW=206	1
BRB-94-04C	Polynuclear aromatic, MW=206	2
BRB-94-04C	Polynuclear aromatic, MW=206	1
BRB-94-04C	Heneicosane	2
BRB-94-04C	Unknown oxy aromatic hydrocarbon	0.6
BRB-94-04C	Unknown oxy hydrocarbon	1
BRB-94-04C	Unknown oxy hydrocarbon	0.6
BRB-94-04C	Polynuclear aromatic, MW=220	0.8
BRB-94-04C	Polynuclear aromatic, MW=220	1
BRB-94-04C	Polynuclear aromatic, MW=220	2
BRB-94-04C	Polynuclear aromatic, MW=220	3
BRB-94-04C	Docosane	2
BRB-94-04C	Polynuclear aromatic, MW=220	2
BRB-94-04C	Polynuclear aromatic, MW=220	1
BRB-94-04C	Polynuclear aromatic, MW=220	1
BRB-94-04C	Polynuclear aromatic, MW=220	0.9
BRB-94-04C	Polynuclear aromatic, MW=216	3
BRB-94-04C	Polynuclear aromatic, MW=219 and 234	1
BRB-94-04C	Tricosane	7
BRB-94-04C	Polynuclear aromatic, MW=234	1
BRB-94-04C	Polynuclear aromatic, MW=216	2
BRB-94-04C	Polynuclear aromatic, MW=216	2
BRB-94-04C	Polynuclear aromatic, MW=234	2
BRB-94-04C	Polynuclear aromatic, MW=230	1
BRB-94-04C	Unknown oxy hydrocarbon	1
BRB-94-04C	Polynuclear aromatic, MW=230	1

Tentatively Identified Compounds, SWMU 23 (continued)

Site ID	Tentative ID	Value (ug/g)
BRB-94-04C	Tetracosane	4
BRB-94-04C	Polynuclear aromatic, MW=230	3
BRB-94-04C	Polynuclear aromatic, MW=230	2
BRB-94-04C	Polynuclear aromatic, MW=230	3
BRB-94-04C	Polynuclear aromatic, MW=230	1
BRB-94-04C	Polynuclear aromatic, MW=230	1
BRB-94-04C	Pentacosane	5
BRB-94-04C	Polynuclear aromatic, MW=244	0.5
BRB-94-04C	Polynuclear aromatic, MW=244	2
BRB-94-04C	Polynuclear aromatic, MW=248	2
BRB-94-04C	Hexacosane	3
BRB-94-04C	Polynuclear aromatic, MW=248	0.5
BRB-94-04C	Polynuclear aromatic, MW=242	2
BRB-94-04C	Polynuclear aromatic, MW=242	0.5
BRB-94-04C	Polynuclear aromatic, MW=262	0.5
BRB-94-04C	Heptacosane	2
BRB-94-04C	Polynuclear aromatic, MW=318	0.9
BRB-94-04C	Polynuclear aromatic, MW=256	0.5
BRB-94-04C	Unknown alkane	2
BRB-94-04C	Polynuclear aromatic, MW=314	0.5
BRB-94-04C	Unknown alkane	2
BRB-94-04C	Unknown alkane	1
BRB-94-04C	Unknown alkane	0.8
BRB-94-04C	Unknown alkane	0.5
BRB-94-05A	Unknown nitrogen containing hydrocarbon	0.3
BRB-94-05A	Unknown alkene	0.6
BRB-94-05A	Unknown alkene	0.4
BRB-94-05A	Hexadecanoic acid	0.3
BRB-94-05A	Unknown oxy hydrocarbon	1
BRB-94-05A	Unknown alkane	0.4
BRB-94-05A	Unknown alkane	0.3
BRB-94-05A	Unknown oxy hydrocarbon	0.3
BRB-94-05B	Unknown nitrogen containing hydrocarbon	0.7
BRB-94-05B	Hexadecanoic acid	0.3
BRB-94-05B	Polynuclear aromatic, MW=206	0.4
BRB-94-05B	Polynuclear aromatic, MW=220	0.3
BRB-94-05B	Polynuclear aromatic, MW=216	0.4
BRB-94-05B	Octadecane	0.3
BRB-94-05B	Nonadecane	0.4
BRB-94-05B	Unknown oxy hydrocarbon	2
BRB-94-05B	Unknown alkane	0.5
BRB-94-05B	Unknown alkane	0.6
BRB-94-05B	Unknown alkane	0.3

Tentatively Identified Compounds, SWMU 23 (continued)

Site ID	Tentative ID	Value (ug/g)
BRB-94-05B	Unknown alkane	0.5
BRB-94-05B	Unknown oxy hydrocarbon	0.4
BRB-94-05C	Unknown oxy hydrocarbon	0.7
BRB-94-10A	Unknown amide	0.5
BRB-94-10A	Unknown alkyl pentanone	0.3
BRB-94-10A	Unknown oxy heterocycle	0.5
BRB-94-10A	Propanetriol triacetate	0.6
BRB-94-10A	Hexadecanoic acid	1
BRB-94-10A	Unknown nitroso benzenamine	0.3
BRB-94-10A	Unknown alcohol	0.7
BRB-94-10A	Unknown alkane	0.6
BRB-94-10A	Unknown alkane	0.7
BRB-94-10A	Unknown alcohol	0.3
BRB-94-10B	Hexadecanoic acid	0.4
BRB-94-10B	Unknown alcohol	0.6
BRB-94-10C	Unknown alkyl pentanone	0.3
BRB-94-11A	Unknown amide	0.9
BRB-94-11A	Unknown unsaturated acid	0.7
BRB-94-11A	Polynuclear aromatic, MW=190 + unknown acid	0.8
BRB-94-11A	Polynuclear aromatic, MW=216	0.3
BRB-94-11A	Unknown alkane	0.3
BRB-94-11A	Unknown alkane	0.3
BRB-94-11A	Unknown alkane	0.6
BRB-94-11A	Polynuclear aromatic, MW=252	0.6
BRB-94-11A	Unknown alkane	0.4
BRB-94-11A	Polynuclear aromatic, MW=278	0.4
BRB-94-11B	Unknown unsaturated acid	0.3
BRB-94-11B	Hexadecanoic acid	0.8
BRB-94-11B	Unknown unsaturated acid	0.9
BRB-94-11B	Unknown acid	2
BRB-94-11B	Unknown alcohol	0.3
BRB-94-11B	Unknown alcohol	0.3
BRB-94-11B	Unknown branched alkane	0.3
BRB-94-11B	Unknown alcohol	0.6
BRB-94-11C	Unknown amide	0.4
BRB-94-12A	Unknown nitrogen containing oxy hydrocarbon	0.9
BRB-94-12A	Unknown unsaturated hydrocarbon	2
BRB-94-12A	Unknown unsaturated hydrocarbon	2
BRB-94-12A	Unknown unsaturated hydrocarbon	0.3
BRB-94-12A	Unknown oxy hydrocarbon	0.3
BRB-94-12A	Hexadecenoic acid or isomer	5
BRB-94-12A	Hexadecanoic acid	2

Tentatively Identified Compounds, SWMU 23 (continued)

Site ID	Tentative ID	Value (ug/g)
BRB-94-12A	Unknown Oxy hydrocarbon	0.3
BRB-94-12A	Oleic acid	1
BRB-94-12A	Ocadecanoic acid	0.4
BRB-94-12A	Unknown alcohol	1
BRB-94-12A	Docosanoic acid	0.5
BRB-94-12A	Unknown polycyclic hydrocarbon	0.3
BRB-94-12A	Unknown polycyclic with oxy hydrocarbon	2
BRB-94-12A	Unknown oxy hydrocarbon	0.5
BRB-94-12A	Unknown polycyclic hydrocarbon	0.4
BRB-94-12A	Unknown alkane	0.7
BRB-94-12A	Unknown oxy hydrocarbon	0.4
BRB-94-12A	Unknown alkane	0.3
BRB-94-12A	Unknown oxy hydrocarbon	0.3
BRB-94-12A	Unknown alkane	0.4
BRB-94-12A	Unknown alkane	3
BRB-94-12A	Unknown alkane	0.6
BRB-94-12A	Unknown alkane	0.9
BRB-94-12A	Unknown alkane	5
BRB-94-12A	Unknown oxy hydrocarbon	1
BRB-94-12A	Unknown alkane	0.3
BRB-94-12A	Unknown alkane	0.7
BRB-94-12A	Unknown polycyclic hydrocarbon	1
BRB-94-12A	Polynuclear aromatic, MW=269	0.3
BRB-94-12B	Unknown aromatic	0.3
BRB-94-12B	Unknown oxy hydrocarbon	0.8
BRB-94-12B	Unknown acid	0.6
BRB-94-12B	Unknown aldehyde	0.7
BRB-94-12B	Unknown aldehyde	0.6
BRB-94-12B	Unknown alcohol	1
BRB-94-12B	Unknown oxy aromatic	0.5
BRB-94-12B	Unknown oxy hydrocarbon	0.4
BRB-94-12B	Unknown polycyclic hydrocarbon	3
BRB-94-12B	Unknown polycyclic hydrocarbon	0.6
BRB-94-12B	Unknown polycyclic hydrocarbon	0.8
BRB-94-12B	Unknown alkane	0.5
BRB-94-12B	Unknown alcohol	0.4
BRB-94-12B	Unknown alkane	2
BRB-94-12B	Unknown alkane	0.4
BRB-94-12B	Unknown oxy hydrocarbon	0.3
BRB-94-12B	Unknown alkane	0.7
BRB-94-12B	Unknown alkane	3
BRB-94-12B	Unknown oxy hydrocarbon	0.5
BRB-94-12B	Unknown polycyclic hydrocarbon	1

Tentatively Identified Compounds, SWMU 23 (continued)

Site ID	Tentative ID	Value (ug/g)
BRB-94-12B	Unknown oxy hydrocarbon	0.9
BRB-94-12B	Unknown alkane	0.4
BRB-94-12B	Unknown polycyclic hydrocarbon	0.5
BRB-94-12B	Methandriol	0.3
BRB-94-12C	Unknown polycyclic hydrocarbon	3
BRB-94-12C	Unknown alcohol	0.8
BRB-94-12C	Unknown alkane	0.3
BRB-94-12C	Unknown alkane	0.3
BRB-94-12C	Unknown oxy hydrocarbon	0.3
BRB-94-14A	Unknown alcohol	0.3
BRB-94-15A	Unknown alkyl pentanone	0.3
BRB-94-15A	Hexadecanoic acid	0.5
BRB-94-15A	Unknown alcohol	0.6
BRB-94-15B	Unknown alcohol	0.3
BRB-94-17A	Adipate ester	0.3
BRP-94-01A	Unknown acid	0.3
BRP-94-01A	Unknown alcohol	1
BRP-94-01A	Unknown alkane	0.8
BRP-94-01A	Unknown alkane	1
BRP-94-01A	Unknown aldehyde	0.3
BRP-94-01A	Unknown alkane	0.4
BRP-94-01A	Unknown alcohol	0.5
BRP-94-01B	Hexadecanoic acid	0.4
BRP-94-01B	Unknown alcohol	1
BRP-94-01B	Unknown oxy hydrocarbon	0.3
BRP-94-01B	Unknown oxy hydrocarbon	0.4
BRP-94-01B	Unknown oxy hydrocarbon	0.4
BRP-94-01C	Hexadecanoic acid	0.4
BRP-94-01C	Unknown alcohol	0.7
BRP-94-03A	Unknown unsaturated hydrocarbon	0.3
BRP-94-03A	Hexadecanoic acid	0.5
BRP-94-03A	Unknonw alcohol	1
BRP-94-03A	Unknown alkane	0.5
BRP-94-03A	Unknown oxy hydrocarbon	0.6
BRP-94-03A	Unknown cyclic hydrocarbon	2
BRP-94-03A	Unknown alkane	0.7
BRP-94-03A	Unknown oxy hydrocarbon	0.3
BRP-94-03A	Unknown alkane	0.3
BRP-94-03A	Unknown oxy hydrocarbon	0.8
BRP-94-03B	Unknown aromatic	0.4
BRP-94-03B	Unknown alcohol	0.6
BRP-94-03C	Unknown alcohol	0.8
BRP-94-03C	Unknown alkane	0.3

Tentatively Identified Compounds, SWMU 23 (continued)

Site ID	Tentative ID	Value (ug/g)
BRP-94-06A	Unknown nitrogen containing hydrocarbon	0.8
BRP-94-06A	Unknown oxy hydrocarbon	0.4
BRP-94-06A	2-Pentanone, 4-hydroxy-4-methyl-	0.8
BRP-94-06A	Unknown oxy hydrocarbon	0.4
BRP-94-06A	Unknown oxy hydrocarbon	0.3
BRP-94-06A	Unknown cyclic alkene	0.4
BRP-94-06A	Unknown hydrocarbon	0.3
BRP-94-06A	Hexadecanoic acid	0.5
BRP-94-06A	Octadecanoic acid	0.3
BRP-94-06A	Unknown oxy hydrocarbon	1
BRP-94-06A	Unknown polycyclic hydrocarbon	0.6
BRP-94-06A	Unknown aromatic hydrocarbon	0.3
BRP-94-06A	Unknown alkane	0.7
BRP-94-06A	Octadecane	0.4
BRP-94-06A	Unknown alkane	0.8
BRP-94-06A	Unknown oxy hydrocarbon	0.3
BRP-94-06A	Unknown alkane	0.3
BRP-94-06A	Unknown oxy hydrocarbon	0.4
BRP-94-06A	Unknown polycyclic oxy aromatic hydrocarbon	0.5
BRP-94-06A	Unknown polycyclic oxy aromatic hydrocarbon	0.3
BRP-94-06A	Unknown polycyclic hydrocarbon	0.3
BRP-94-06B	Unknown nitrogen containing hydrocarbon	0.3
BRP-94-06B	Unknown alkene	1
BRP-94-06B	Unknown alkene	1
BRP-94-06B	2-Pentanone, 4-hydroxy-4-methyl	1
BRP-94-06B	Hexadecanoic acid	0.8
BRP-94-06B	Unknown oxy hydrocarbon	1
BRP-94-06B	Unknown oxy hydrocarbon	0.5
BRP-94-06B	Unknown oxy hydrocarbon	0.4
BRP-94-06B	Unknown oxy hydrocarbon	0.6
BRP-94-06C	Unknown oxy hydrocarbon	0.6
BRP-94-06C	Unknown oxy hydrocarbon	0.6
BRP-94-07A	Unknown ketone	0.3
BRP-94-07A	Unknown alkane	0.4
BRP-94-07A	Unknown nitrogen containing hydrocarbon	2
BRP-94-07A	Unknown alkane	0.4
BRP-94-07A	Unknown oxy hydrocarbon	0.7
BRP-94-07A	Unknown oxy hydrocarbon	0.3
BRP-94-07A	Unknown oxy hydrocarbon	0.3
BRP-94-07A	Unknown unsaturated hydrocarbon	0.3
BRP-94-07A	Unknown oxy hydrocarbon	0.4
BRP-94-07A	Unknown oxy hydrocarbon	0.3
BRP-94-07A	Unknown oxy hydrocarbon	0.3

Tentatively Identified Compounds, SWMU 23 (continued)

Site ID	Tentative ID	Value (ug/g)
BRP-94-07A	Unknown oxy hydrocarbon	0.4
BRP-94-07A	Hexadecanoic acid	0.9
BRP-94-07A	Octadecanoic acid	0.3
BRP-94-07A	Hexanedioic acid, bis (2-ethylhexyl or isomer)	0.3
BRP-94-07A	Unknown alcohol	0.8
BRP-94-07A	Unknown alkane	0.3
BRP-94-07A	Unknown alkane	0.3
BRP-94-07A	Unknown oxy hydrocarbon	0.3
BRP-94-07B	Unknown methyl ester	0.3
BRP-94-07B	Hexadecanoic acid	0.4
BRP-94-07B	Unknown alcohol	0.8
BRP-94-07C	Unknown alcohol	0.7
BRP-94-07C	Unknown alcohol	0.3
BRP-94-08A	Aromatic nitrogen heterocycle	0.5
BRP-94-08A	Octadecanoic acid	0.3
BRP-94-08A	Dichloro phosphate propanol	0.4
BRP-94-08A	Adipate ester	0.3
BRP-94-08A	Cyclohexanecarboxylic acid	1
BRP-94-08A	Unknown alcohol	0.6
BRP-94-08A	Unknown oxy alkane	0.6
BRP-94-08A	Unknown alkane	0.8
BRP-94-08A	Unknown alkane	0.6
BRP-94-08A	Unknown unsaturated oxy hydrocarbon	0.4
BRP-94-08B	Phthalic anhydride isomer	0.5
BRP-94-08B	Unknown alkyl benzene compound	0.4
BRP-94-08B	Sulfur containing benzene compound	0.3
BRP-94-08B	Cyclohexanecarboxylic acid	2
BRP-94-08B	Unknown alcohol	0.3
BRP-94-08C	Unknown alkyl benzene compound	0.4
BRP-94-08C	Unknown alkyl diphenyl isomer	0.3
BRP-94-08C	Cyclohexanecarboxylic acid	2
BRP-94-09A	Unknown styrene	0.6
BRP-94-09A	Unknown acid + aromatic	0.5
BRP-94-09A	Unknown methyl styrene	1
BRP-94-09A	Unknown acetophenone	1
BRP-94-09A	Unknown Phenylpropenal isomer	0.3
BRP-94-09A	Unknown dimethyl benzenamine isomer	0.4
BRP-94-09A	Nitrogen containing benzoic acid	5
BRP-94-09A	Unknown alkane	0.3
BRP-94-09A	Polychlorinated biphenyl	0.5
BRP-94-09A	Polychlorinated biphenyl	0.3
BRP-94-09A	Polychlorinated biphenyl	0.8
BRP-94-09A	Polychlorinated biphenyl	0.4

Tentatively Identified Compounds, SWMU 23 (continued)

Site ID	Tentative ID	Value (ug/g)
BRP-94-09A	Polychlorinated biphenyl	0.3
BRP-94-09A	Unknown acid	4
BRP-94-09A	Polychlorinated biphenyl	2
BRP-94-09A	Polychlorinated biphenyl	0.9
BRP-94-09A	Polychlorinated biphenyl	0.6
BRP-94-09A	Polychlorinated biphenyl	1
BRP-94-09A	Polychlorinated biphenyl	0.4
BRP-94-09A	Unknown diphenyl propenone isomer	0.3
BRP-94-09A	Polychlorinated biphenyl	0.4
BRP-94-09A	Polychlorinated biphenyl	0.7
BRP-94-09A	Polychlorinated biphenyl	0.4
BRP-94-09A	Polychlorinated biphenyl	0.6
BRP-94-09A	Polychlorinated biphenyl	2
BRP-94-09A	Polychlorinated biphenyl	1
BRP-94-09A	Polychlorinated biphenyl	1
BRP-94-09A	Polychlorinated biphenyl + unsaturated hydrocarbn	0.5
BRP-94-09A	Polychlorinated biphenyl	0.6
BRP-94-09A	Polychlorinated biphenyl	0.3
BRP-94-09A	Unknown acid	2
BRP-94-09A	Polychlorinated biphenyl	0.5
BRP-94-09A	Polychlorinated biphenyl	0.4
BRP-94-09A	Polychlorinated biphenyl	0.4
BRP-94-09A	Polychlorinated biphenyl + unsaturated hydrocarbon	0.5
BRP-94-09A	Unknown oxy benzene compound	0.5
BRP-94-09A	Unknown alcohol	0.3
BRP-94-09A	Unknown oxy phenyl compound	0.3
BRP-94-09A	Nitrogen containing aromatic compound	0.5
BRP-94-09A	Unknown alkyl di-acid	0.3
BRP-94-09A	Unknown oxy alkane	0.4
BRP-94-09A	Unknown polycyclic oxy hydrocarbon	0.3
BRP-94-09A	Unknown polyaromatic oxy compound	0.4
BRP-94-09A	Nitrogen containing aromatic compound	0.3
BRP-94-09A	Unknown alkyl naphthalene compound	0.4
BRP-94-09A	Unknown aromatic	0.3
BRP-94-09A	Unknown oxy aromatic	0.5
BRP-94-09A	Unknown oxy aromatic	2
BRP-94-09A	Polychlorinated biphenyl	0.4
BRP-94-09A	Polychlorinated biphenyl	0.5
BRP-94-09A	Polychlorinated biphenyl	0.4
BRP-94-09A	Polychlorinated biphenyl	0.3
BRP-94-09A	Polychlorinated biphenyl	0.7
BRP-94-09A	Polychlorinated biphenyl	0.3
BRP-94-09A	Polychlorinated biphenyl	0.3

Tentatively Identified Compounds, SWMU 23 (continued)

Site ID	Tentative ID	Value (ug/g)
BRP-94-09A	Polychlorinated biphenyl	0.8
BRP-94-09A	Polychlorinated biphenyl	0.6
BRP-94-09A	Polychlorinated biphenyl	0.6
BRP-94-09A	Polychlorinated biphenyl	0.4
BRP-94-09A	Polychlorinated biphenyl	0.3
BRP-94-09A	Polychlorinated biphenyl	0.3
BRP-94-09A	Unknown alkane	0.3
BRP-94-09A	Unknown alcohol	0.5
BRP-94-09A	Unknown alkane	0.4
BRP-94-09A	Unknown polycyclic hydrocarbon	0.4
BRP-94-09B	Unknown amide	0.6
BRP-94-09B	Unknown alkyl benzene compound	0.4
BRP-94-09B	Hexadecanoic acid	0.3
BRP-94-09B	Cyclohexanecarboxylic acid	2
BRP-94-09B	Unknown alcohol	0.4
BRP-94-09B	Unknown amide	0.3
BRP-94-09B	Unknown cyclic acid	0.7
BRP-94-09B	Unknown alcohol	0.8
BRP-94-09C	Unknown oxy aromatic compound	4
BRP-94-09C	Unknown oxy benzene compound	1
BRP-94-09C	Bis (propanediyl) benzene	0.3
BRP-94-09C	Unknown alkyl benzene compound	0.3
BRP-94-09C	Diphenyl butadiene isomer	0.3
BRP-94-09C	Hexadecanoic acid	0.3
BRP-94-09C	Unknown chlorinated oxy hydrocarbon	4
BRP-94-09C	Cyclohexanecarboxylic acid	2
BRP-94-09C	Unknown alcohol	0.5
BRP-94-09C	Unknown alcohol	0.7
BRP-94-09C	Unknown alcohol	0.3
BRP-94-13A	Unknown hydrocarbon	0.8
BRP-94-13A	Hexadecanoic acid	0.8
BRP-94-13A	Unknown alcohol	0.7
BRP-94-13B	Unknown alcohol	0.7
BRP-94-13B	Unsaturated hydrocarbon	0.3
BRP-94-13C	Hexadecanoic acid	0.3
BRP-94-13C	Unknown alcohol	0.8
BRS-94-01	Oxy hydrocarbon	0.3
BRS-94-01	Methyl ester	2
BRS-94-01	Unknown acid	2
BRS-94-01	Methyl ester	1
BRS-94-01	Unknown acid	0.4
BRS-94-01	Hexadecanoic acid	0.4
BRS-94-01	Unknown alcohol	1

Tentatively Identified Compounds, SWMU 23 (continued)

Site ID	Tentative ID	Value (ug/g)
BRS-94-01	Unknown hydrocarbon	0.3
BRS-94-01	Unknown alkane	0.3
BRS-94-01	Polynuclear aromatic MW=252	0.3
BRS-94-01	Unknown alcohol	0.3
BRS-94-01	Polynuclear aromatic MW=278	0.3
BRS-94-02	Methyl ester	0.4
BRS-94-02	Methyl ester	0.5
BRS-94-02	Hexadecanoic acid	0.3
BRS-94-02	Unknown alcohol	0.7
BRS-94-02	Unknown alkane	0.6
BRS-94-02	Unknown alkane	0.9
BRS-94-02	Unknown alkane	0.4
BRS-94-03	Methyl ester	0.3
BRS-94-03	Methyl ester	0.3
BRS-94-03	Methyl ester	0.3
BRS-94-03	Methyl ester	1
BRS-94-03	Alcohol	0.4
BRS-94-03	Alkane	0.3
BRS-94-04	Methyl ester	0.6
BRS-94-04	Methyl ester	0.5
BRS-94-04	Methyl ester	0.7
BRS-94-04	Methyl ester	0.3
BRS-94-04	Methyl ester	1
BRS-94-04	Methyl ester	0.3
BRS-94-05	Methyl ester	0.3
BRS-94-05	Methyl ester	0.3
BRS-94-05	Methyl ester	0.6
BRS-94-05	Methyl ester	0.4
BRS-94-05	Unknown alkene	0.6
BRS-94-05	Unknown alkane	0.4
BRS-94-05	Unknown alkane	0.4
BRS-94-05	Unknown aldehyde	0.4
BRS-94-06	Unknown acid	0.7
BRS-94-06	Methyl ester	0.3
BRS-94-06	Methyl ester	5
BRS-94-06	Methyl ester	5
BRS-94-06	Unknown acid	0.3
BRS-94-06	Oxy hydrocarbon	0.8
BRS-94-06	Methyl naphthalene	0.3
BRS-94-06	Aromatic hydrocarbon	0.3
BRS-94-06	Methyl fluorene isomer	0.3
BRS-94-06	Polynuclear aromatic, MW=180	0.3

Tentatively Identified Compounds, SWMU 23 (continued)

Site ID	Tentative ID	Value (ug/g)
BRS-94-06	Polynuclear aromatic, MW=180	0.4
BRS-94-06	Dibenzothiophene	0.3
BRS-94-06	Carbazole	0.5
BRS-94-06	Polynuclear aromatic, MW= 192	0.8
BRS-94-06	Polynuclear aromatic, MW= 192	0.8
BRS-94-06	Polynuclear aromatic, MW= 192	0.5
BRS-94-06	Polynuclear aromatic, MW= 192	0.5
BRS-94-06	Polynuclear aromatic, MW= 192	0.5
BRS-94-06	Polynuclear aromatic, MW= 218	0.3
BRS-94-06	Polynuclear aromatic, MW= 216	0.5
BRS-94-06	Polynuclear aromatic, MW= 216	1
BRS-94-06	Polynuclear aromatic, MW= 216	0.7
BRS-94-06	Polynuclear aromatic, MW= 216	0.6
BRS-94-06	Polynuclear aromatic, MW= 216	0.3
BRS-94-06	Polynuclear aromatic, MW= 230	0.5
BRS-94-06	Polynuclear aromatic, MW= 230	0.4
BRS-94-06	Polynuclear aromatic, MW= 234	0.3
BRS-94-06	Polynuclear aromatic, MW= 228	0.7
BRS-94-06	Polynuclear aromatic, MW= 230	0.3
BRS-94-06	Branched alkane, polynuclear aromatic	0.5
BRS-94-06	Polynuclear aromatic, MW= 228	0.5
BRS-94-06	Polynuclear aromatic, MW= 242	0.6
BRS-94-06	Polynuclear aromatic, MW= 240	0.5
BRS-94-06	Alkane, polynuclear aromatic	1
BRS-94-06	Unknown alkane	0.4
BRS-94-06	Polynuclear aromatic, MW= 252	0.7
BRS-94-06	Polynuclear aromatic, MW= 278	0.3
BRS-94-06	Polynuclear aromatic, MW= 278	0.8
BRS-94-07	Methly ester	0.3
BRS-94-07	Methly ester	0.3
BRS-94-07	Methly ester	1
BRS-94-07	Unknown alcohol	1
BRS-94-07	Unknown alkane	0.8
BRS-94-07	Unknown alkane	0.7
BRS-94-07	Unknown aldehyde	0.3
BRS-94-07	Unknown alkane	0.3
BRS-94-07	Unknown aldehyde	0.4
BRS-94-07	Unknown alcohol	0.3
BRS-94-07	Unknown aldehyde	0.3
BRS-94-08	Methly ester	0.4
BRS-94-08	Methly ester	0.3
BRS-94-08	Methly ester	0.3
BRS-94-08	Methly ester	0.3

Tentatively Identified Compounds, SWMU 23 (continued)

Site ID	Tentative ID	Value (ug/g)
BRS-94-08	Unknown alcohol	0.6
BRS-94-08	Unknown alkane	0.5
BRS-94-08	Unknown alkane	0.4
BRS-94-09	Methly ester	1
BRS-94-09	Methly ester	1
BRS-94-09	Unknown alkane	0.3
BRS-94-09	Unknown branched alkane	0.3
BRS-94-09	Unknown alkane	0.5
BRS-94-09	Unknown alkane	0.5
BRS-94-09	Unknown alkane	0.4
BRS-94-09	Unknown alkane	1
BRS-94-09	Unknown alkane	0.3
BRS-94-09	Unknown alkane	0.8
BRS-94-09	Unknown alkane	0.4
BRS-94-09	Unknown alkane	0.3
BRS-94-09	Unknown alkane	0.9
BRS-94-09	Methyl ester	0.4
BRS-94-09	Unknown acid	0.3
BRS-94-09	Unknown alkane	0.6
BRS-94-09	Unknown alkane	0.5
BRS-94-09	Unknown alkene	0.8
BRS-94-09	Unknown alkane	0.5
BRS-94-09	Unknown alkane	0.4
BRS-94-09	Methyl ester	0.3
BRS-94-09	Unknown aldehyde	0.4
BRS-94-09	Methyl ester	0.5
BRS-94-10	Unsaturated hydrocarbon	0.8
BRS-94-10	Methyl ester	0.7
BRS-94-10	Methyl ester	0.5
BRS-94-10	Unknown alkane	0.6
BRS-94-10	Unknown alkane	0.5
BRS-94-10	Unknown alkane	0.8
BRS-94-10	Unknown alkane	0.7
BRS-94-10	Unknown alkane	1
BRS-94-10	Unknown alkane	0.3
BRS-94-10	Unknown alkane	0.3
BRS-94-10	Unknown alkane	0.8
BRS-94-10	Unknown alkane	0.5
BRS-94-10	Unknown alkane	0.4
BRS-94-10	Unknown alkene	0.5
BRS-94-10	Unknown alkane	0.3
BRS-94-10	Unknown alkane	0.4
BRS-94-10	Methyl ester	0.3

Tentatively Identified Compounds, SWMU 31

Site ID	Tentative ID	Value (ug/g)
TBS-94-03	Unknown amide	0.7
TBS-94-03	Unknown methyl dihydrofuran isomer	0.3
TBS-94-03	Unknown unsaturated hydrocarbon	1
TBS-94-03	Unknown unsaturated oxy hydrocarbon	1
TBS-94-03	Unknown alcohol	0.9
TBS-94-06	Unknown amide	0.5
TBS-94-06	Unknown unsaturated hydrocarbon	0.9
TBS-94-06	Unknown unsaturated oxy hydrocarbon	0.7
TBS-94-06	Unknown alkyl pentanone isomer	0.3
TBS-94-06	Unknown alcohol	0.4
TBS-94-09	Unknown amide	0.7
TBS-94-09	Unknown unsaturated oxy hydrocarbon	0.3
TBS-94-09	Unknown amide	1
TBS-94-09	Methyl dihydrofuran isomer	0.3
TBS-94-09	Unknown unsaturated hydrocarbon	0.7
TBS-94-09	Unknown unsaturated hydrocarbon	0.4
TBS-94-09	Unknown unsaturated oxy hydrocarbon	0.3
TBS-94-09	Unknown unsaturated oxy hydrocarbon	0.7
TBS-94-09	Unknown alkyl pentanone isomer	0.3
TBS-94-09	Unknown alkyl pentanone isomer	0.3
TBS-94-09	Unknown oxy hydrocarbon	0.3
TBS-94-09	Nitrogen containing benzene compound	0.3
TBS-94-09	Unknown alcohol	1
TBS-94-09	Unknown alcohol	1
TBS-94-12	Unknown amide	0.4
TBS-94-12	Unknown unsaturated hydrocarbon	0.9
TBS-94-12	Unknown unsaturated oxy hydrocarbon	0.7
TBS-94-12	Unknown alkyl pentanone isomer	0.5
TBS-94-12	Unknown unsaturated oxy hydrocarbon	0.3
TBS-94-12	Hexadecanoic acid	0.3
TBS-94-12	Unknown alcohol	1
TBS-94-12	Unknown alkane	0.3
TBS-94-12	Unknown alkane	0.3
TBS-94-15	Unknown amide	0.5
TBS-94-15	Unknown unsaturated hydrocarbon	1
TBS-94-15	Unknown unsaturated oxy hydrocarbon	0.8
TBS-94-15	Unknown alkyl pentanone isomer	0.5
TBS-94-15	Unknown unsaturated oxy hydrocarbon	0.5
TBS-94-15	Unknown alcohol	1
TBS-94-18	Unknown amide	0.3
TBS-94-18	Unknown unsaturated hydrocarbon	0.4
TBS-94-18	Unkown alkyl pentanone isomer	0.3
TBS-94-18	Unknown unsaturated oxy hydrocarbon	0.4

Tentatively Identified Compounds, SWMU 31 (continued)

Site ID	Tentative ID	Value (ug/g)
TBS-94-18	Unknown alcohol	1
TBS-94-21	Unknown amide	0.5
TBS-94-21	Unknown alkyl pentanone isomer	0.3
TBS-94-21	Unknown unsaturated oxy hydrocarbon	0.4
TBS-94-21	Unknown alcohol	2
TBS-94-21	Unknown alkane	0.3
TBS-94-21	Unknown acid ester	0.3

Tentatively Identified Compounds, SWMU 32

Site ID	Tentative ID	Value (ug/g)
PPB-94-01A	Oxy hydrocarbon	0.5
PPB-94-01A	Hexadecanoic acid or isomer	0.3
PPB-94-01A	Oxy hydrocarbon	1
PPB-94-01A	Alkane	0.5
PPB-94-01A	Alkane	0.4
PPB-94-01A	Alkane	0.3
PPB-94-01B	Oxy hydrocarbon	0.5
PPB-94-01B	Hexadecanoic acid or isomer	0.6
PPB-94-01B	Oxy hydrocarbon	1
PPB-94-01B	Oxy hydrocarbon	0.3
PPB-94-01C	Oxy hydrocarbon	0.5
PPB-94-01C	Hexadecanoic acid or isomer	0.2
PPB-94-01C	Oxy hydrocarbon	1
PPB-94-03A	Unknown alcohol	0.4
PPB-94-03A	Unknown alkyl pentanone	0.4
PPB-94-03A	Unknown unsaturated oxy hydrocarbon	0.4
PPB-94-03A	Hexadecanoic acid	0.3
PPB-94-03A	Unknown alcohol	1
PPB-94-03A	Unknown unsaturated oxy hydrocarbon	0.5
PPB-94-03A	Unknown alkane	0.9
PPB-94-03A	Unknown alkane	0.7
PPB-94-03B	Unknown alcohol	0.5
PPB-94-03C	Unknown alcohol	0.7
PPB-94-03C	Unknown alcohol	0.3
PPB-94-08A	Oxy hydrocarbon	0.5
PPB-94-08A	Hexadecanoic acid or isomer	0.5
PPB-94-08A	Oxy hydrocarbon	1
PPB-94-08A	Alkane	0.6
PPB-94-08A	Alkane	0.7
PPB-94-08A	Alkane	0.6
PPB-94-08A	Oxy hydrocarbon	0.3
PPB-94-08B	Oxy hydrocarbon	0.6
PPB-94-08B	Hexadecanoic acid or isomer	0.4
PPB-94-08B	Oxy hydrocarbon	1
PPB-94-08B	Alkane	0.4
PPB-94-08B	Alkane	0.4
PPB-94-08C	Oxy hydrocarbon	0.3
PPB-94-08C	Unknown phthalate	1
PPB-94-08C	Alkane	0.3
PPB-94-08C	Alkane	0.4
PPB-94-08C	Acid ester	5
PPB-94-08C	Oxy hydrocarbon	1
PPB-94-08C	Oxy hydrocarbon	0.4

Tentatively Identified Compounds, SWMU 32 (continued)

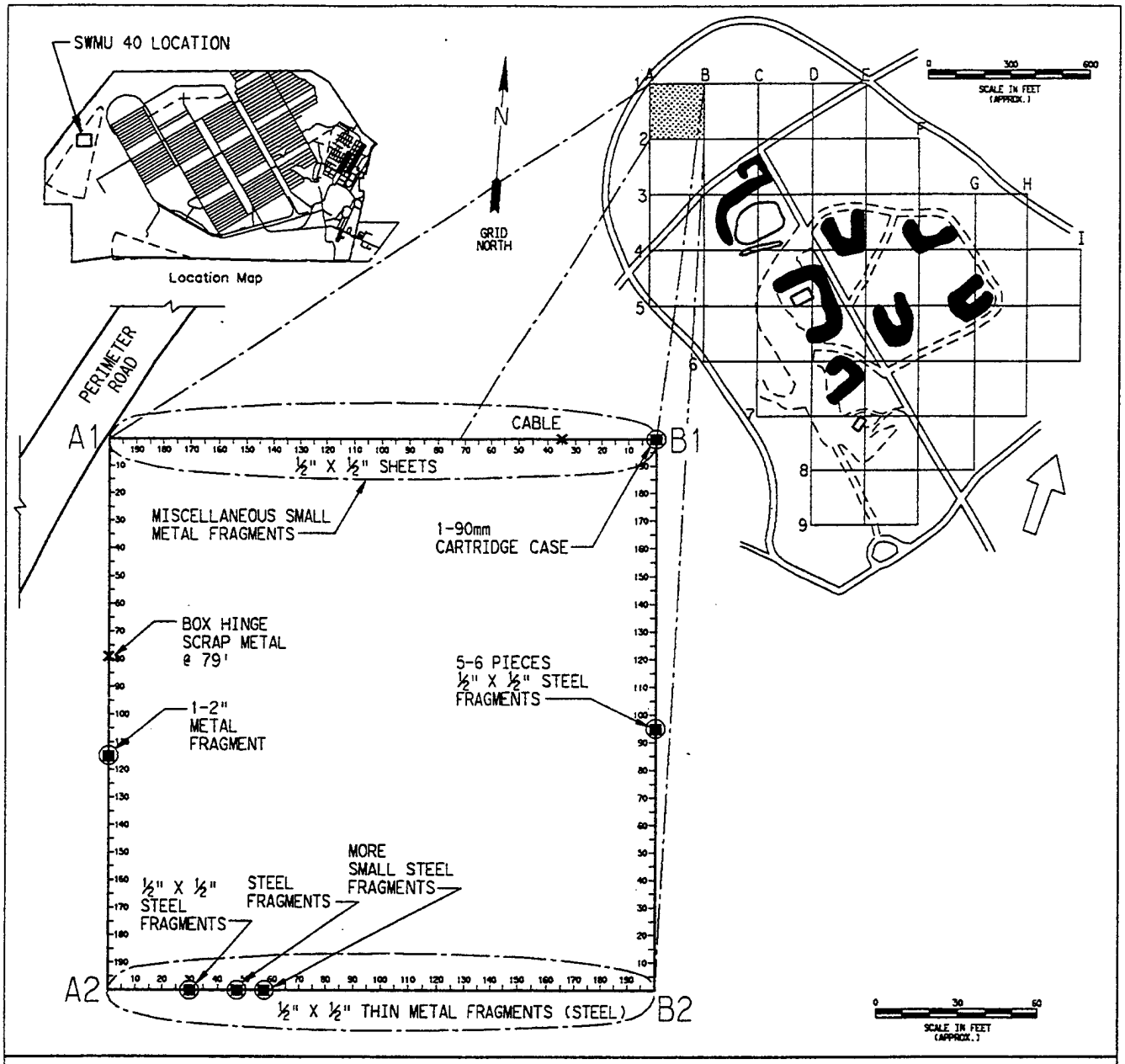
Site ID	Tentative ID	Value (ug/g)
PPS-94-05	Unknown unsaturated oxy hydrocarbon	0.5
PPS-94-05	Unknown acid	0.4
PPS-94-05	Unknown cyclic oxy hydrocarbon	0.4
PPS-94-05	Unknown alkane	0.4
PPS-94-05	Unknown alkane	1
PPS-94-05	Unknown alkane	2
PPS-94-05	Unknown alkane	0.7

Tentatively Identified Compounds, SWMU 35

Site ID	Tentative ID	Value (ug/L)
WW-1	Unknown alkyl pentanone isomer	5
WW-1	Unknown alkyl pentanone isomer	7

APPENDIX Q

UXO/DEBRIS WALKING SURVEY RESULTS FOR SWMU 40

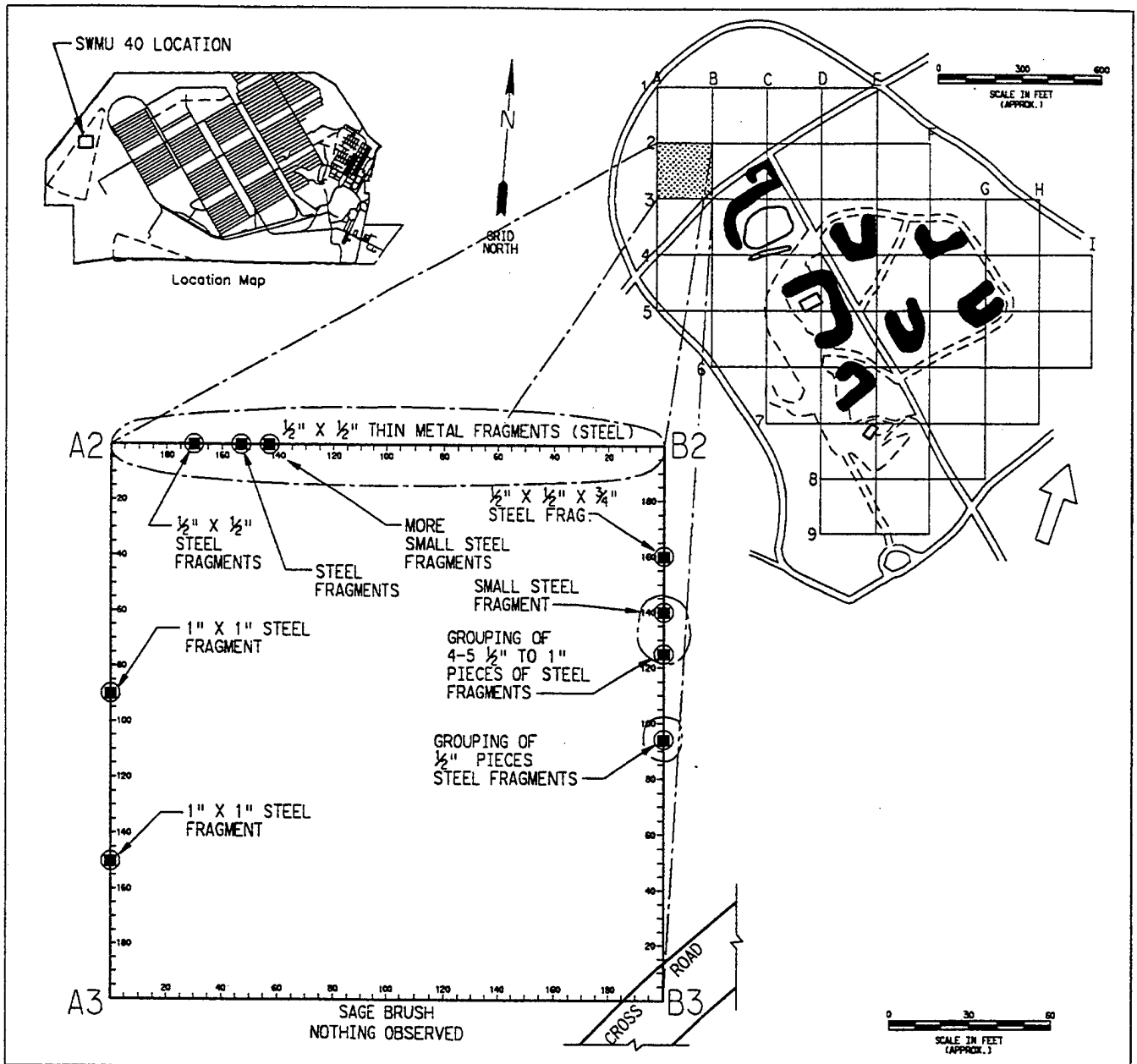


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG01.DGN

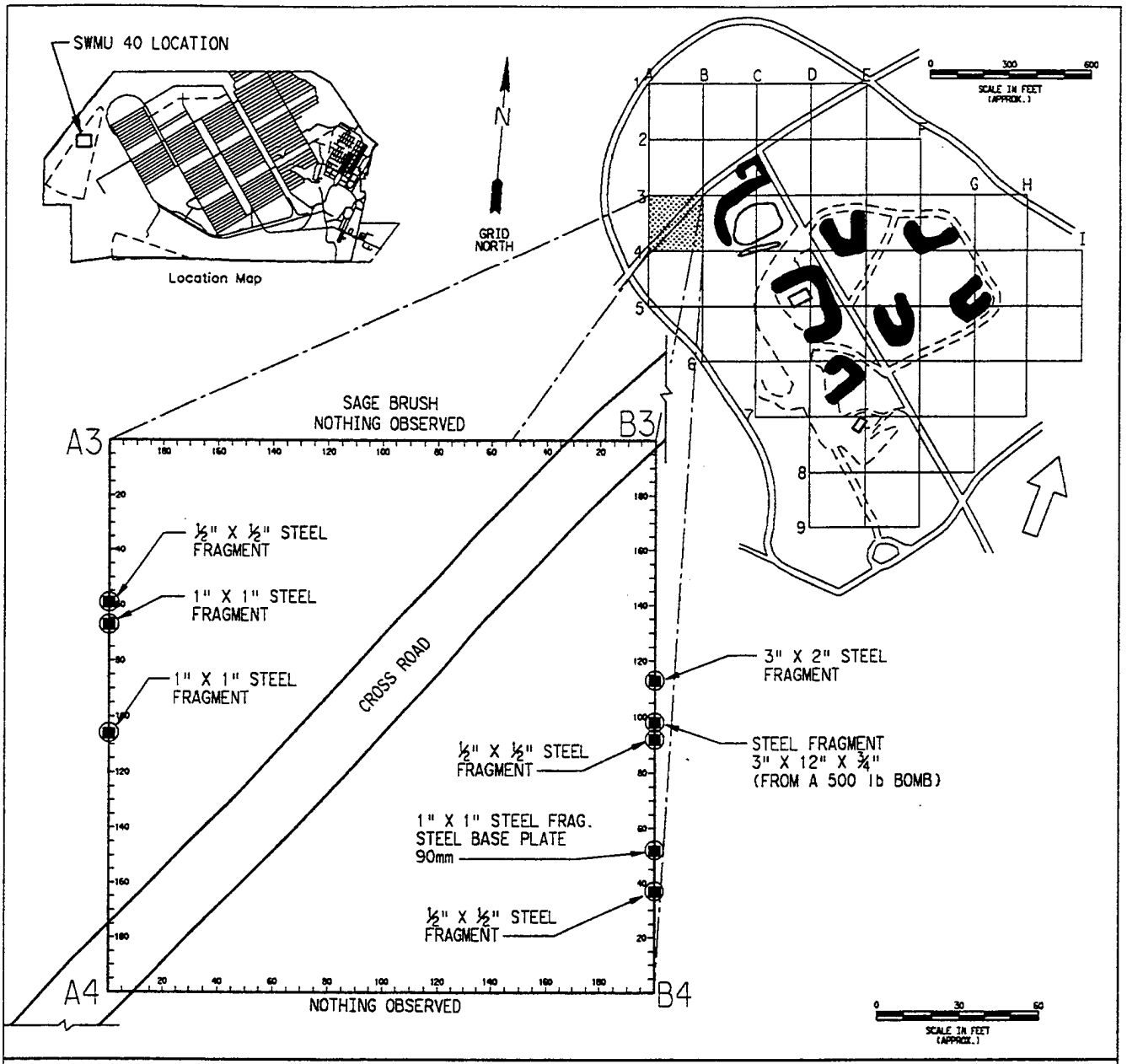


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG02.DGN

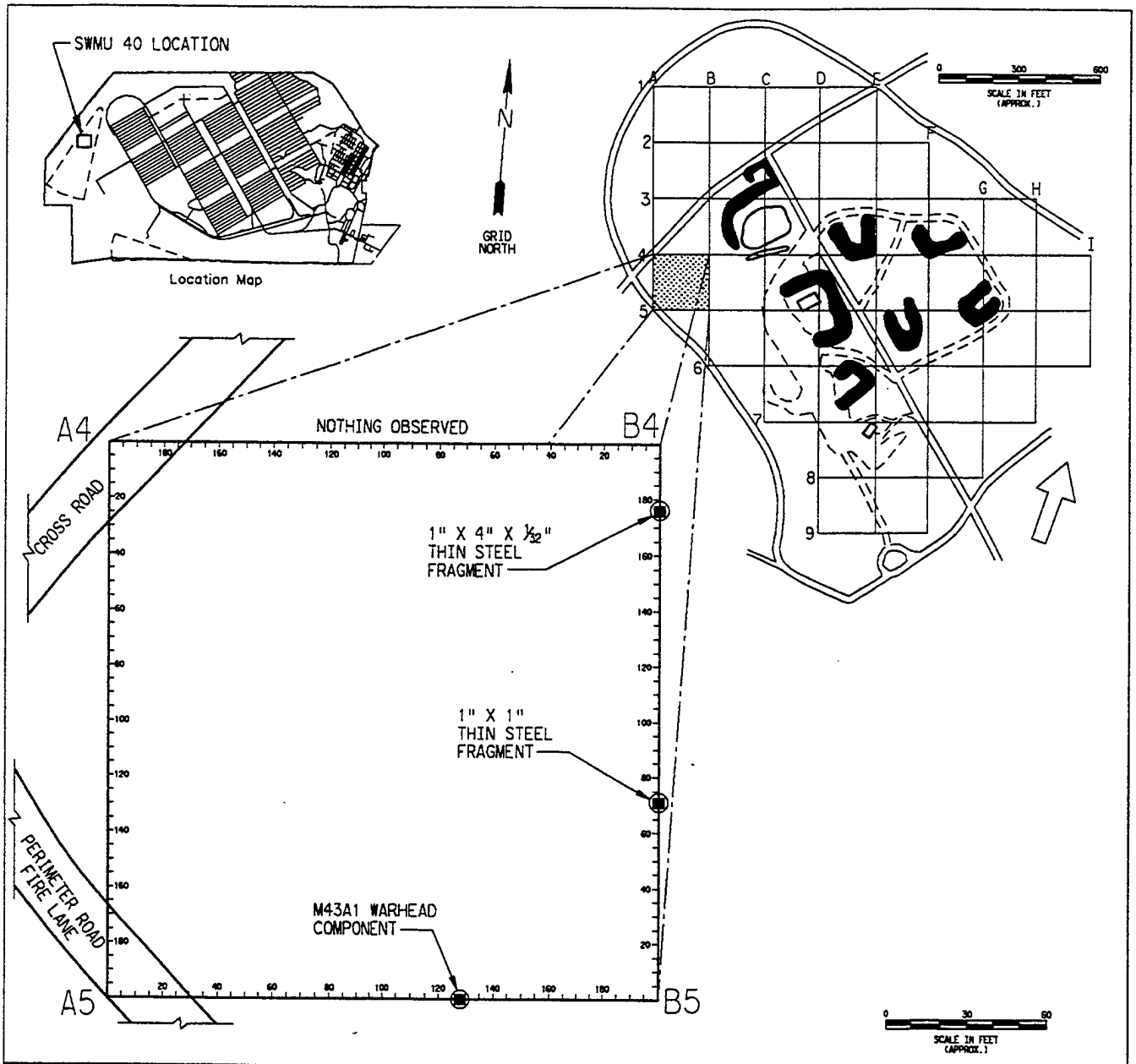


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
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- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG03.DGN

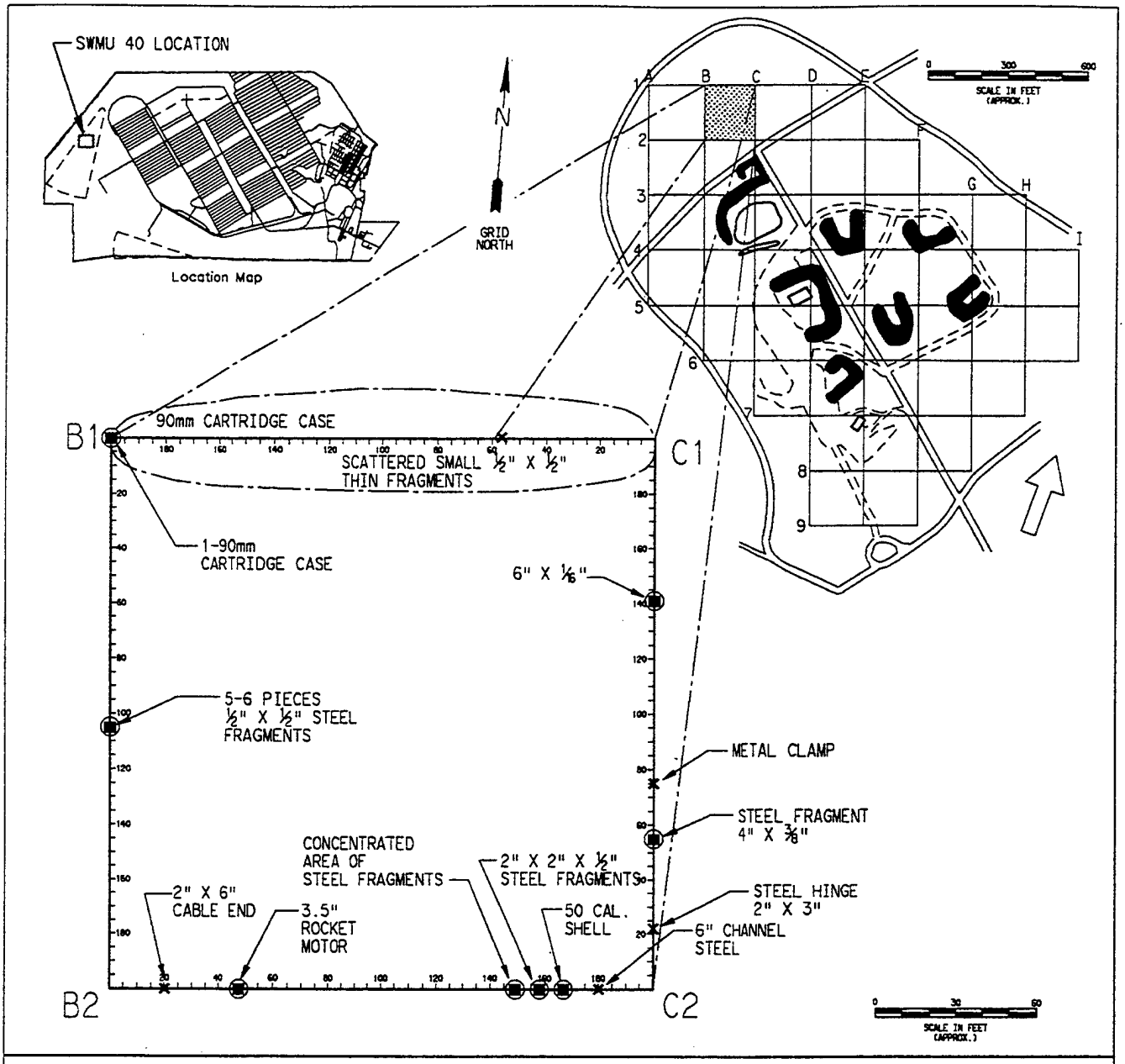


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG04.DGN

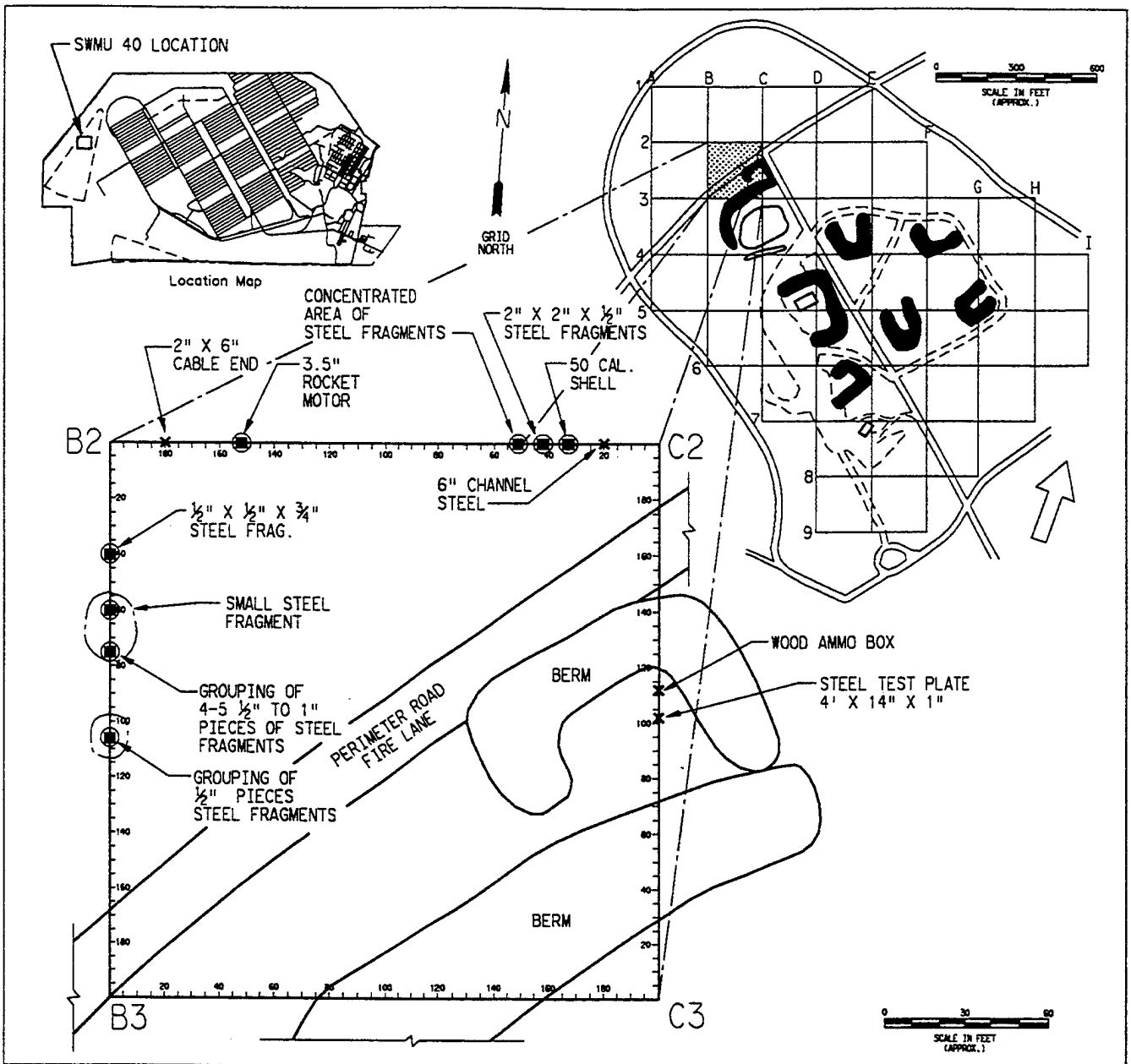


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG33.DGN

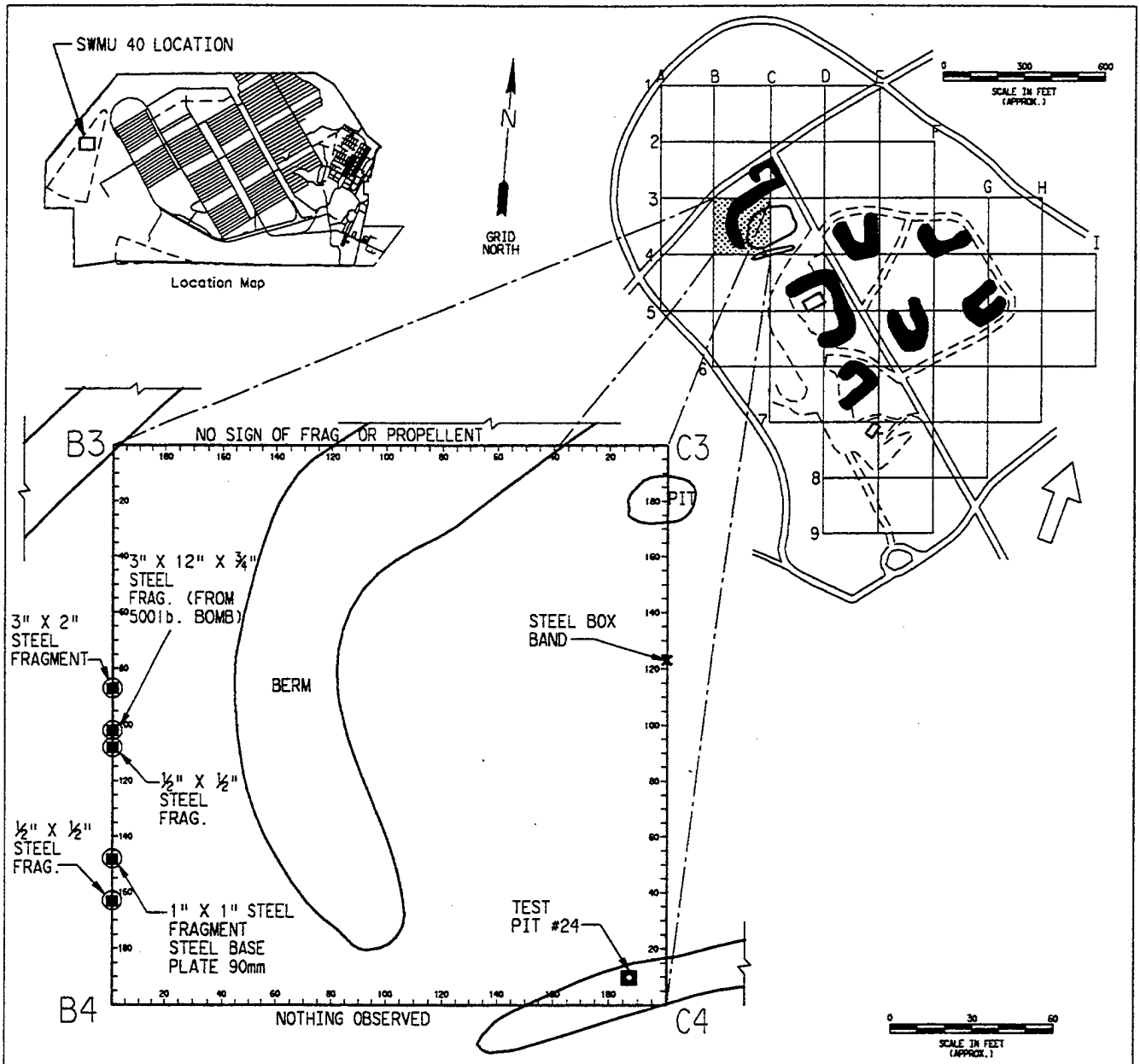


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
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- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
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- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG34.DGN

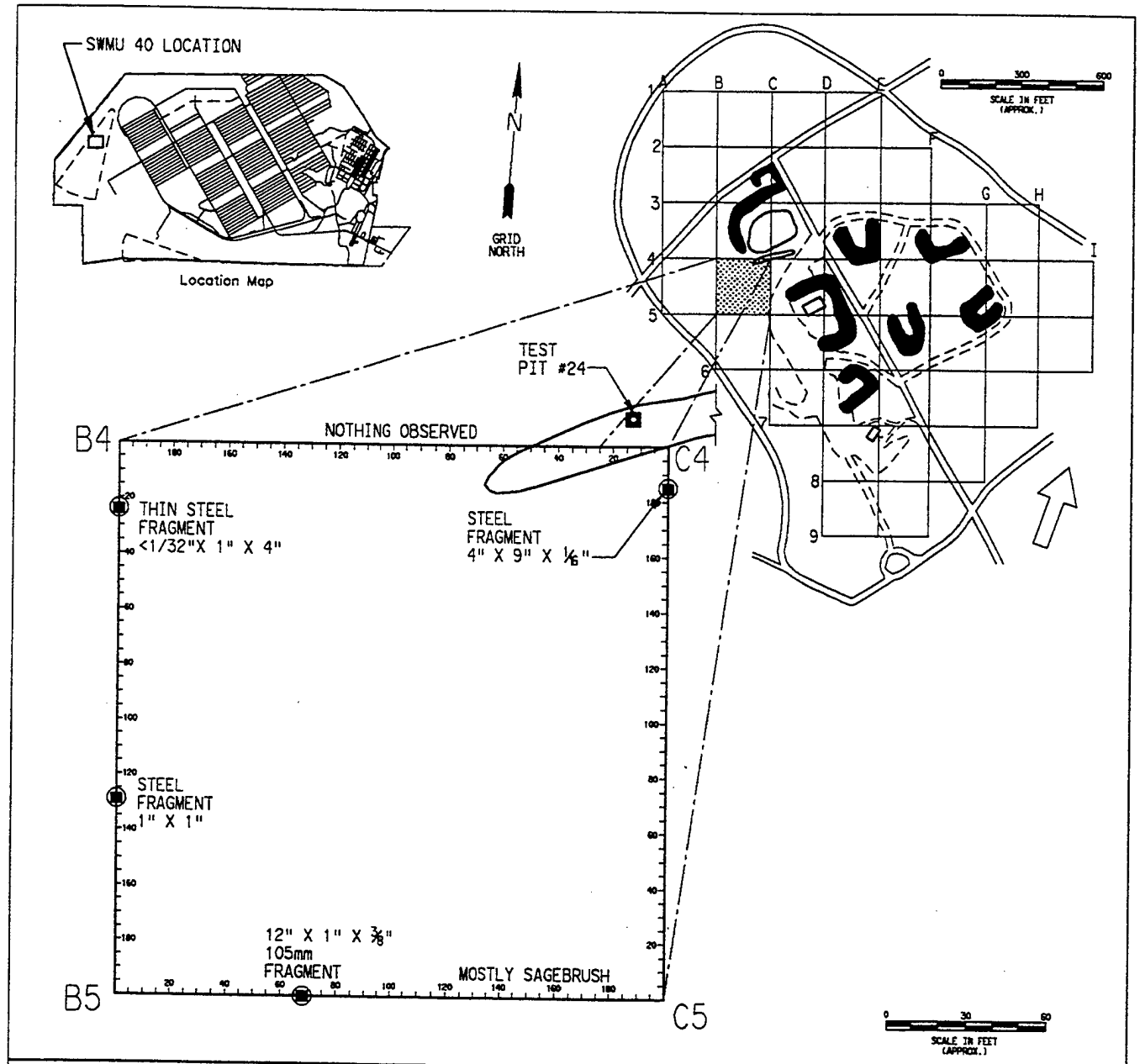


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
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- SAMPLING GRID
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- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG36.DGN

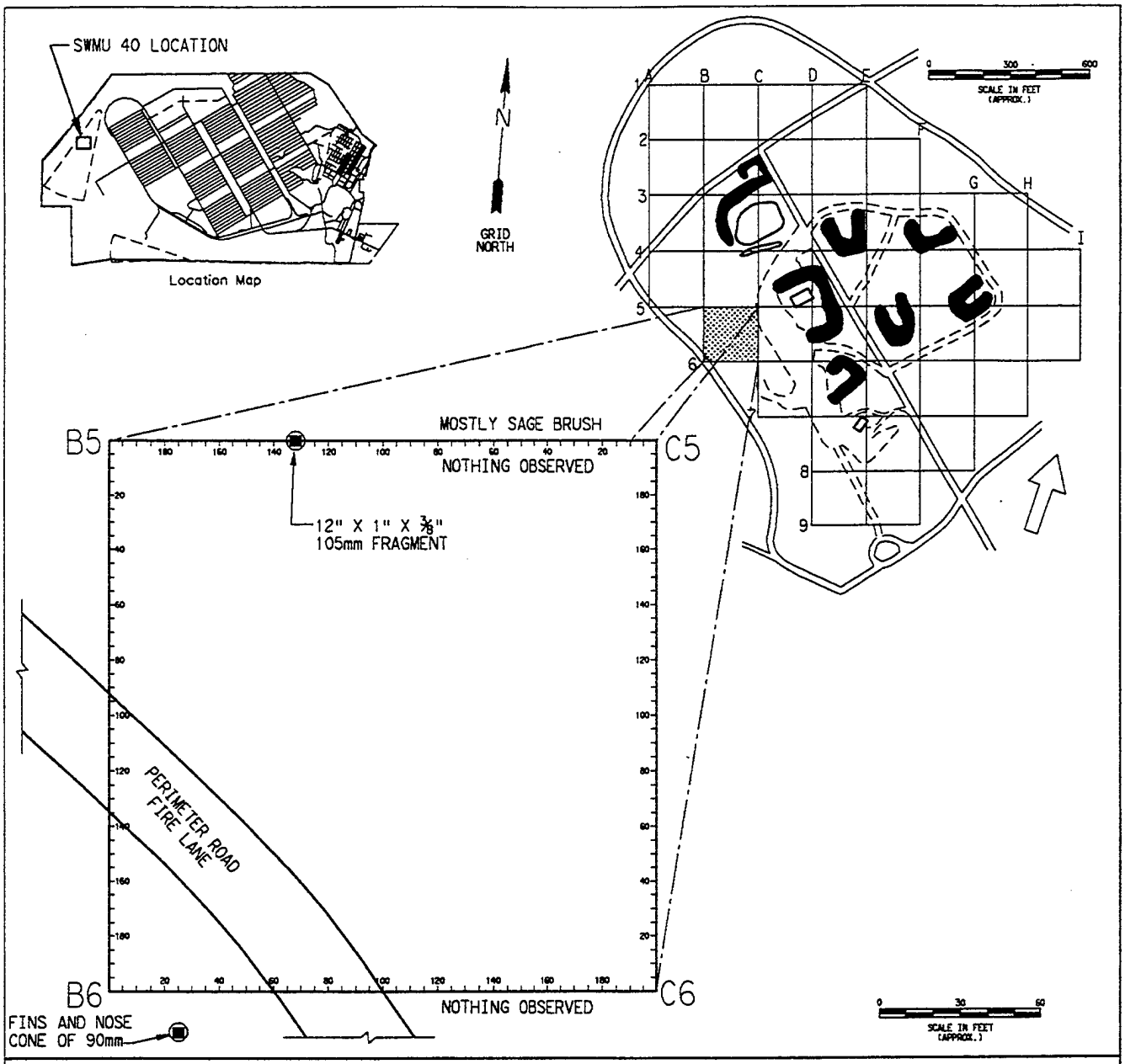


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
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- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
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- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG37.DGN

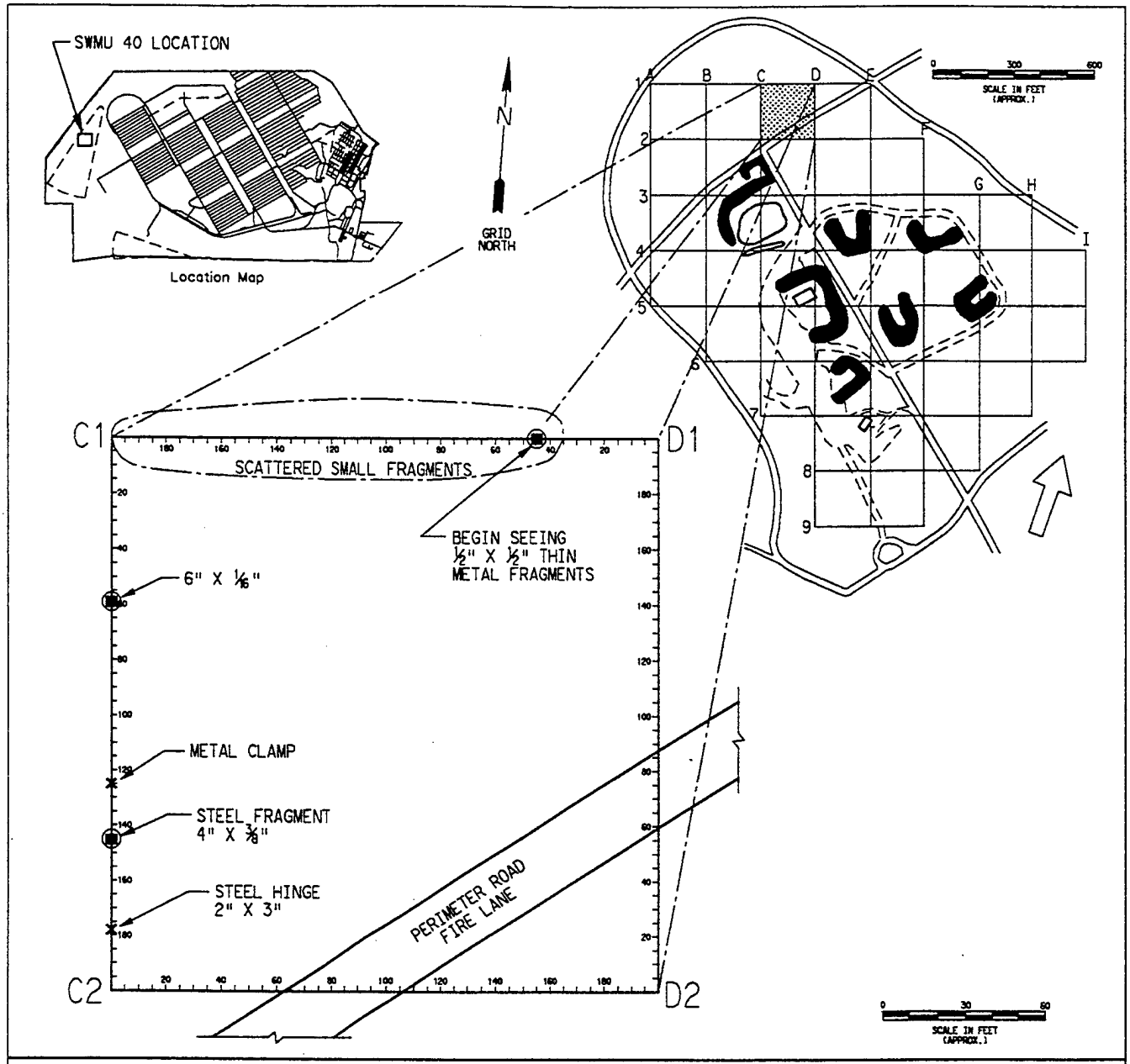


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
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- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG05.DGN

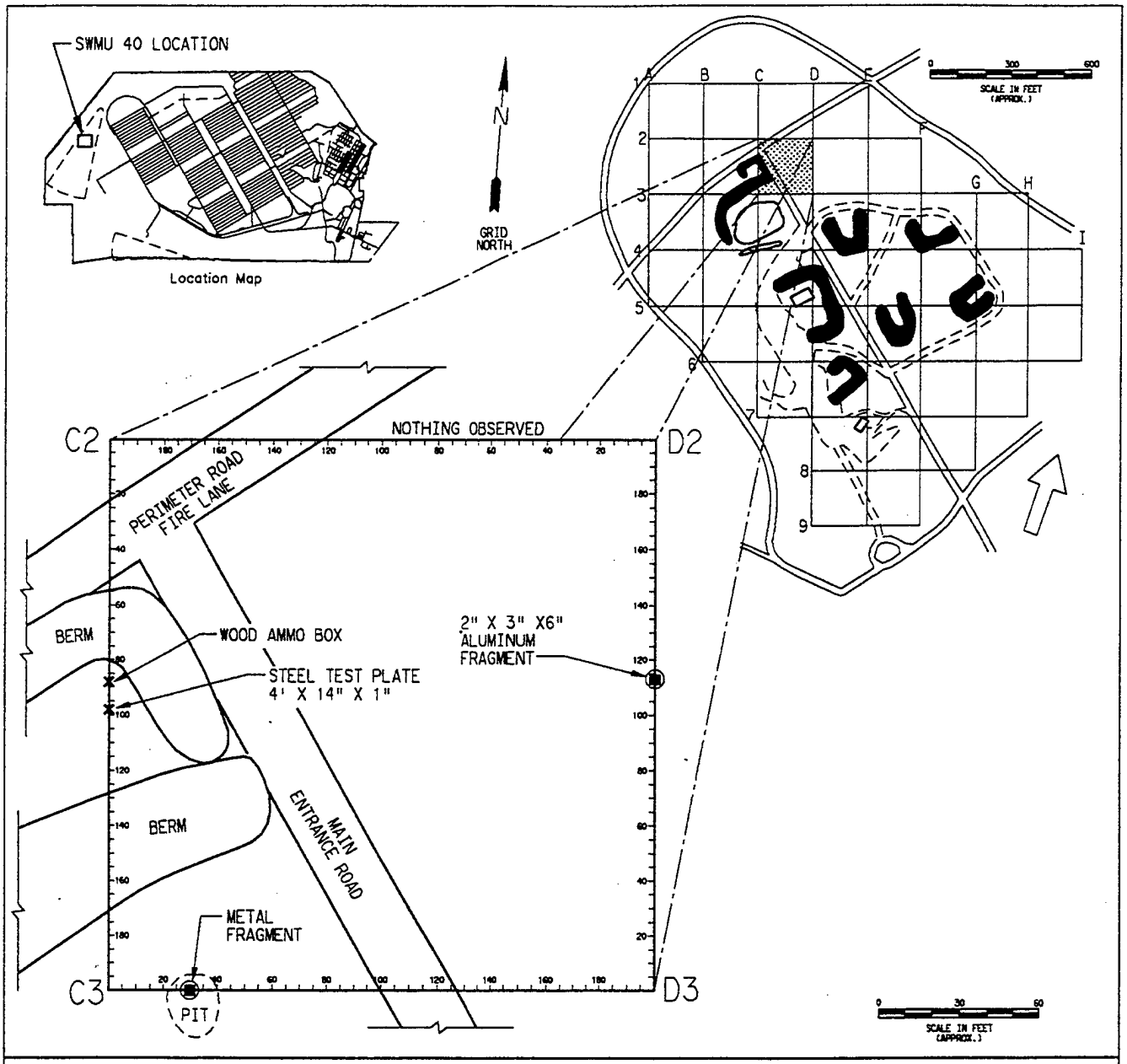


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG06.DGN

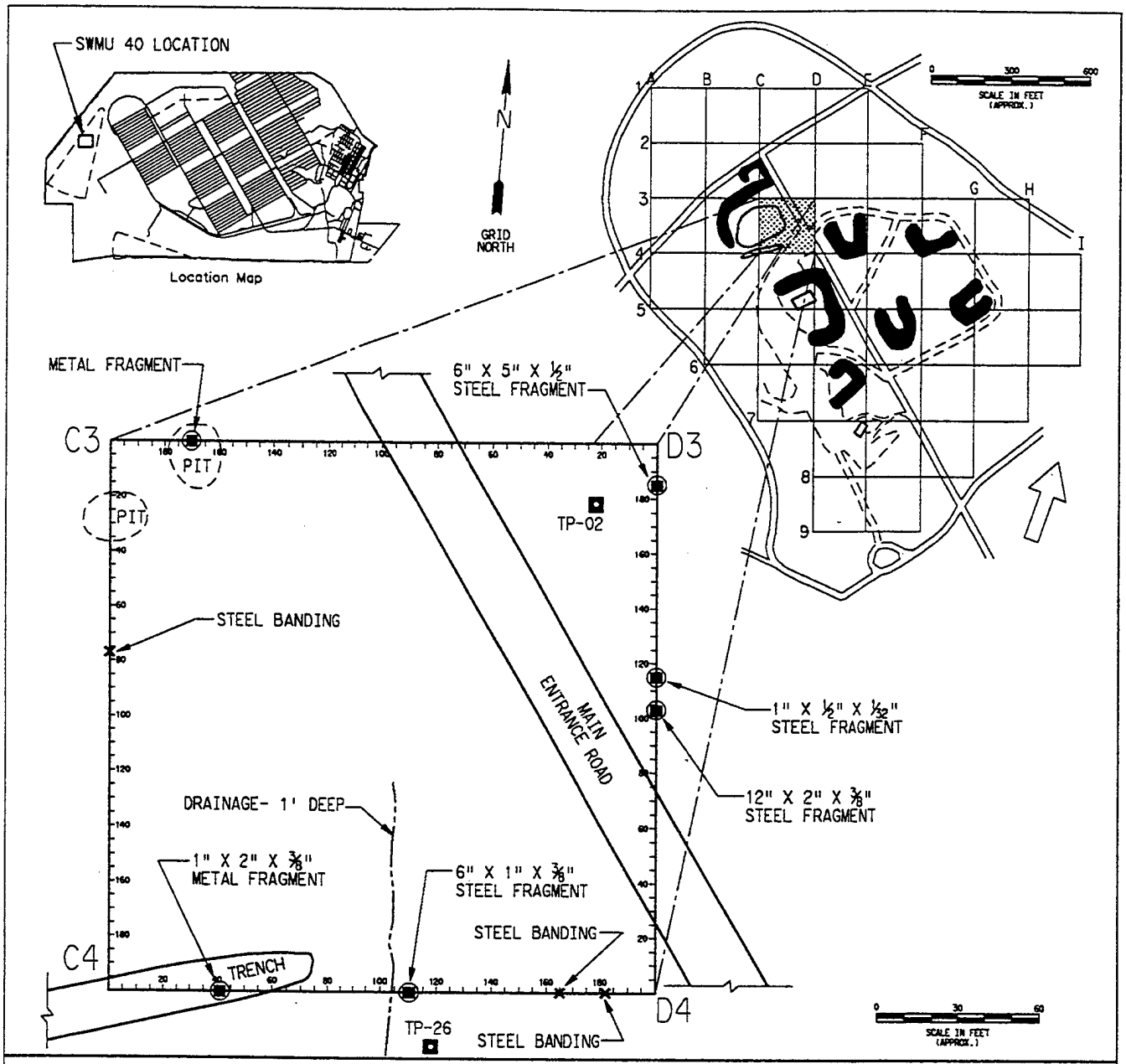


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG07.DGN

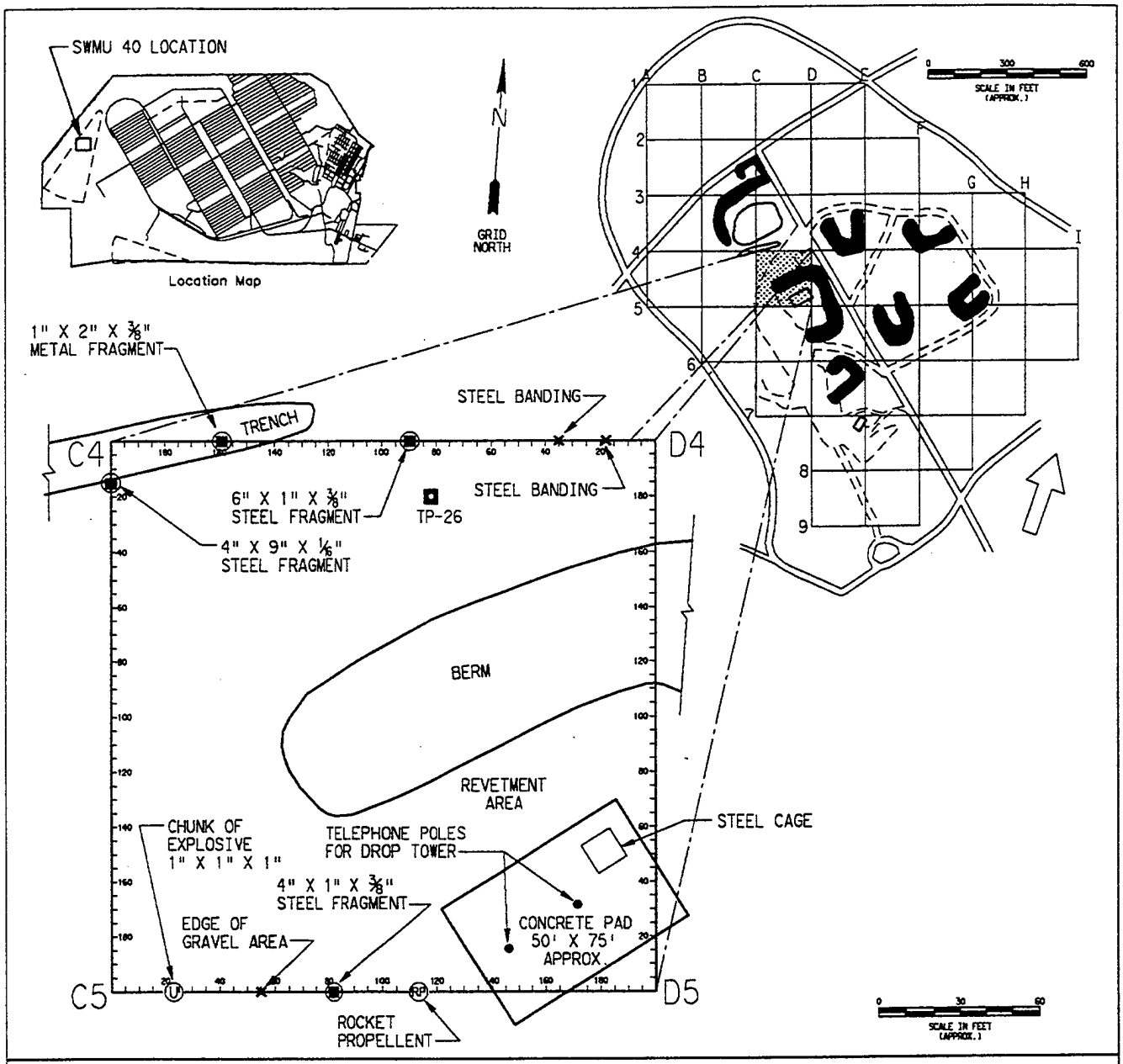


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG08.DGN

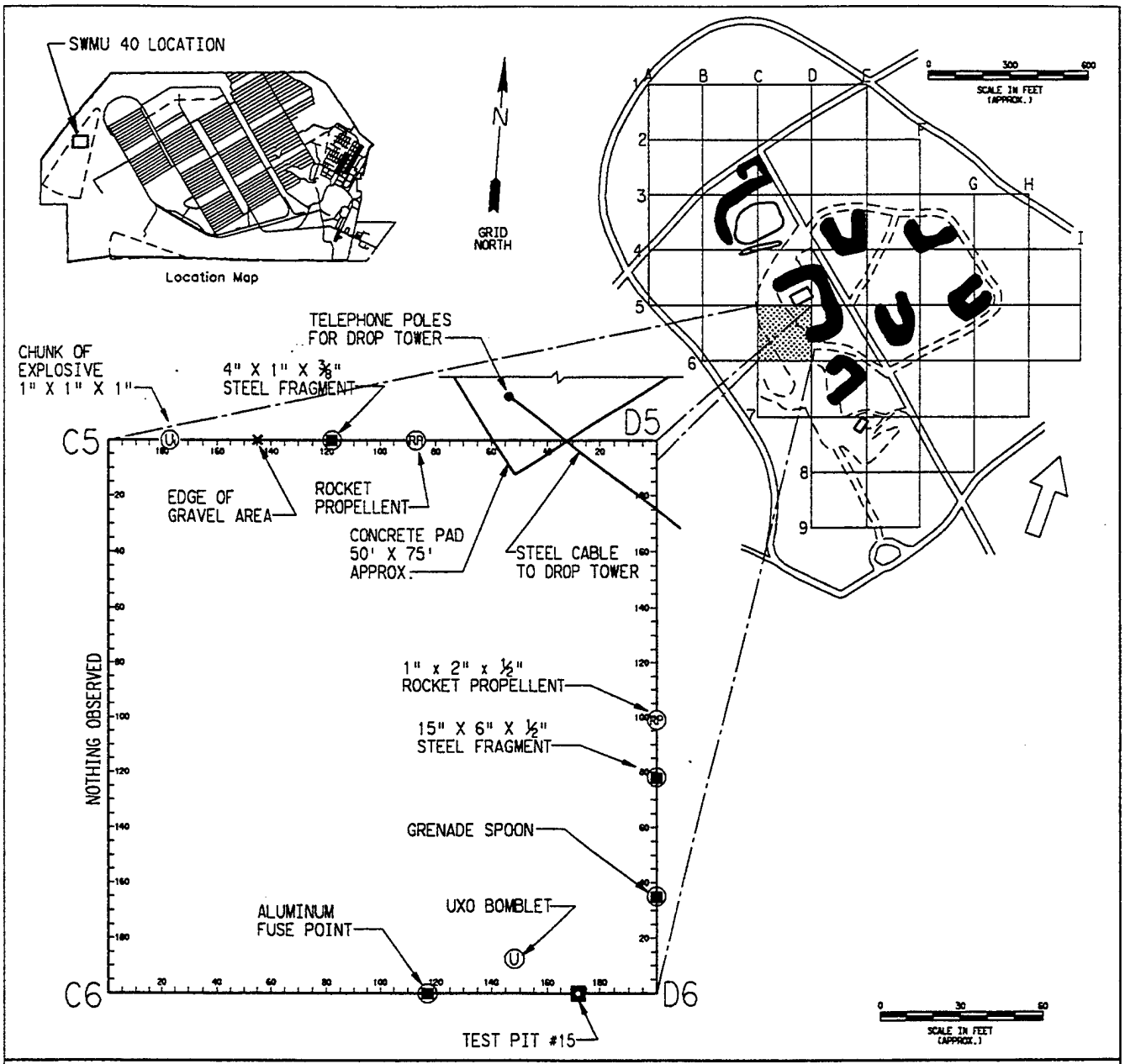


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG09.DGN

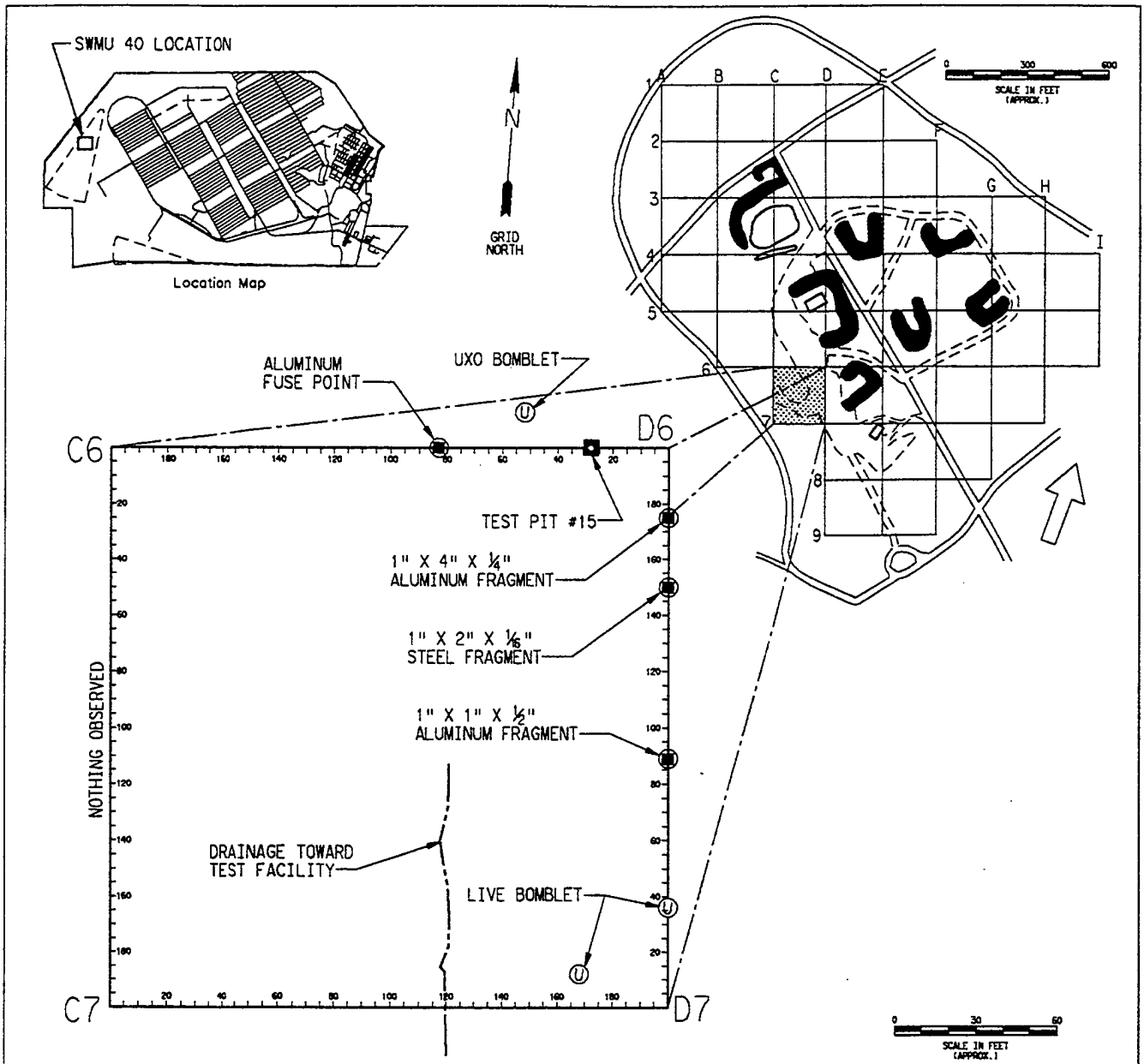


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
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- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG10.DGN

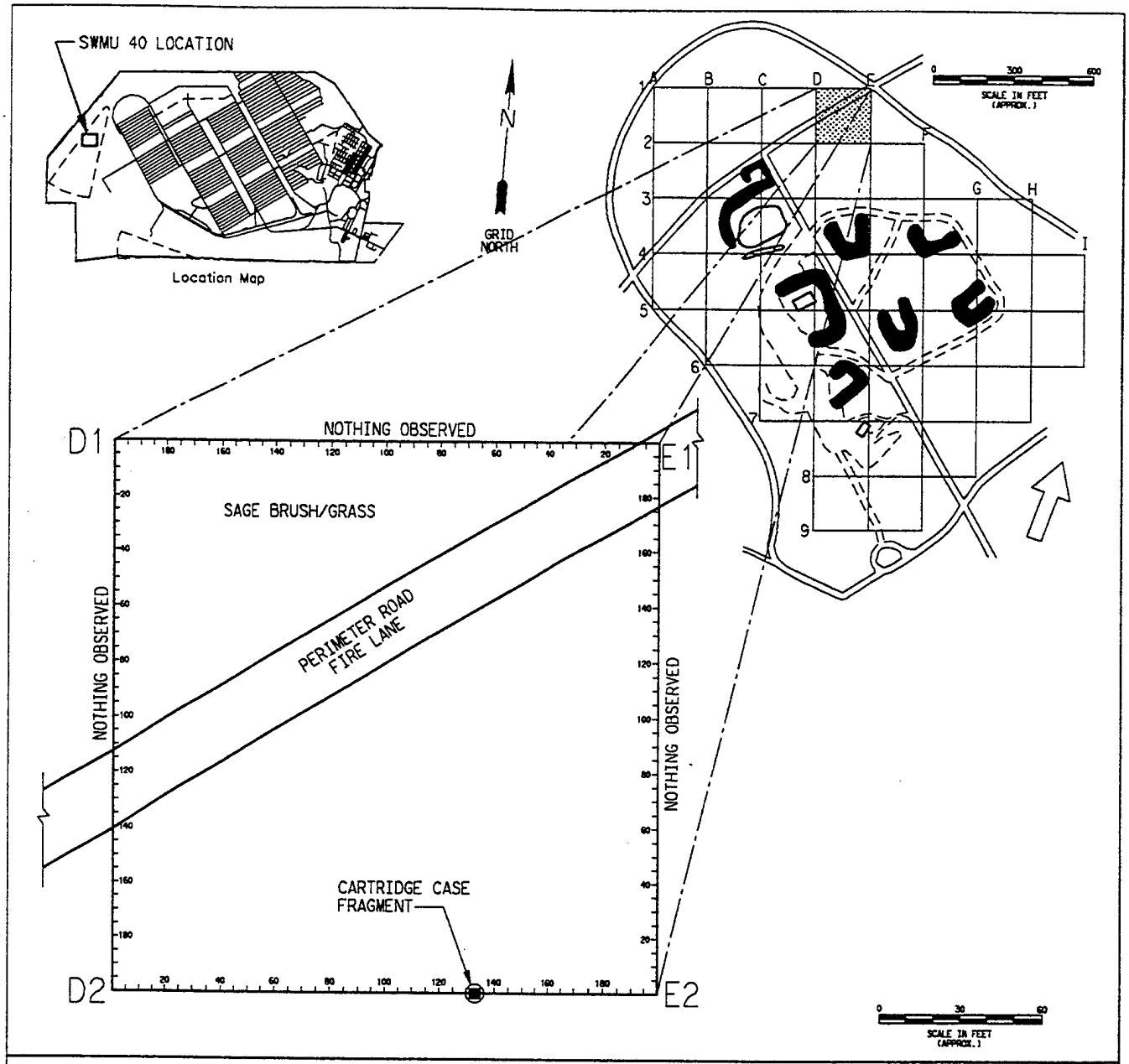


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG11.DGN

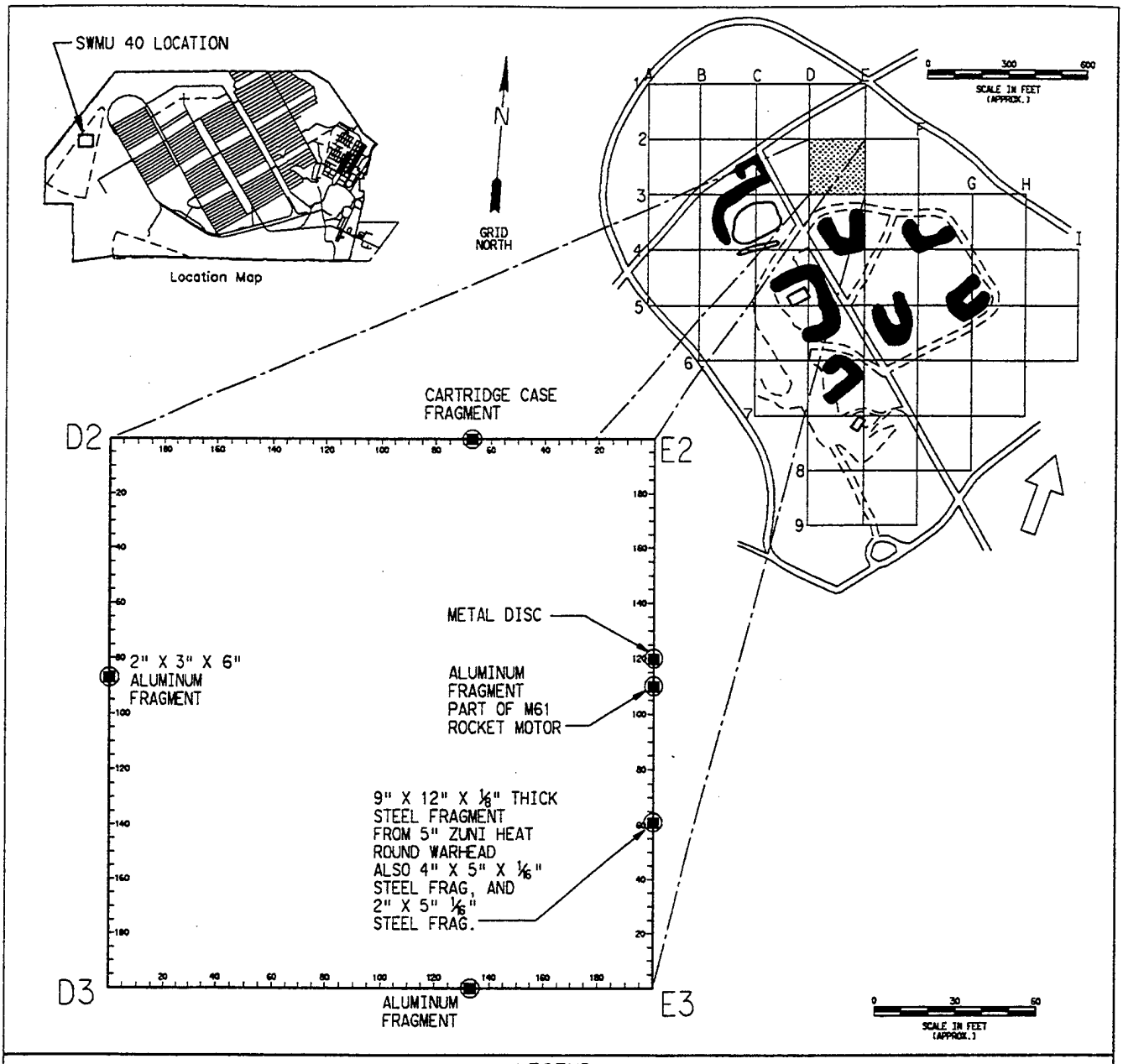


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG14.DGN

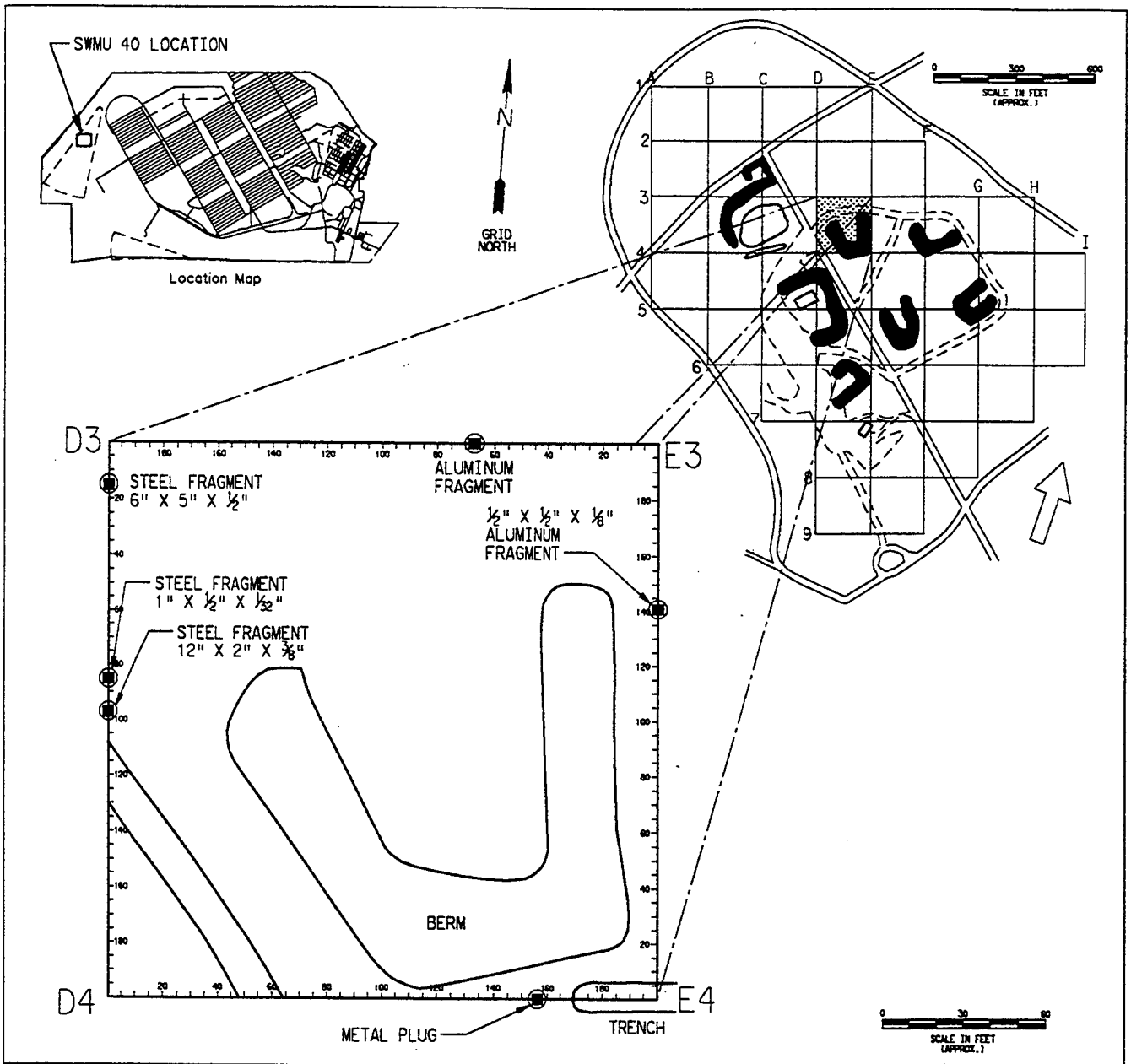


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG38.DGN

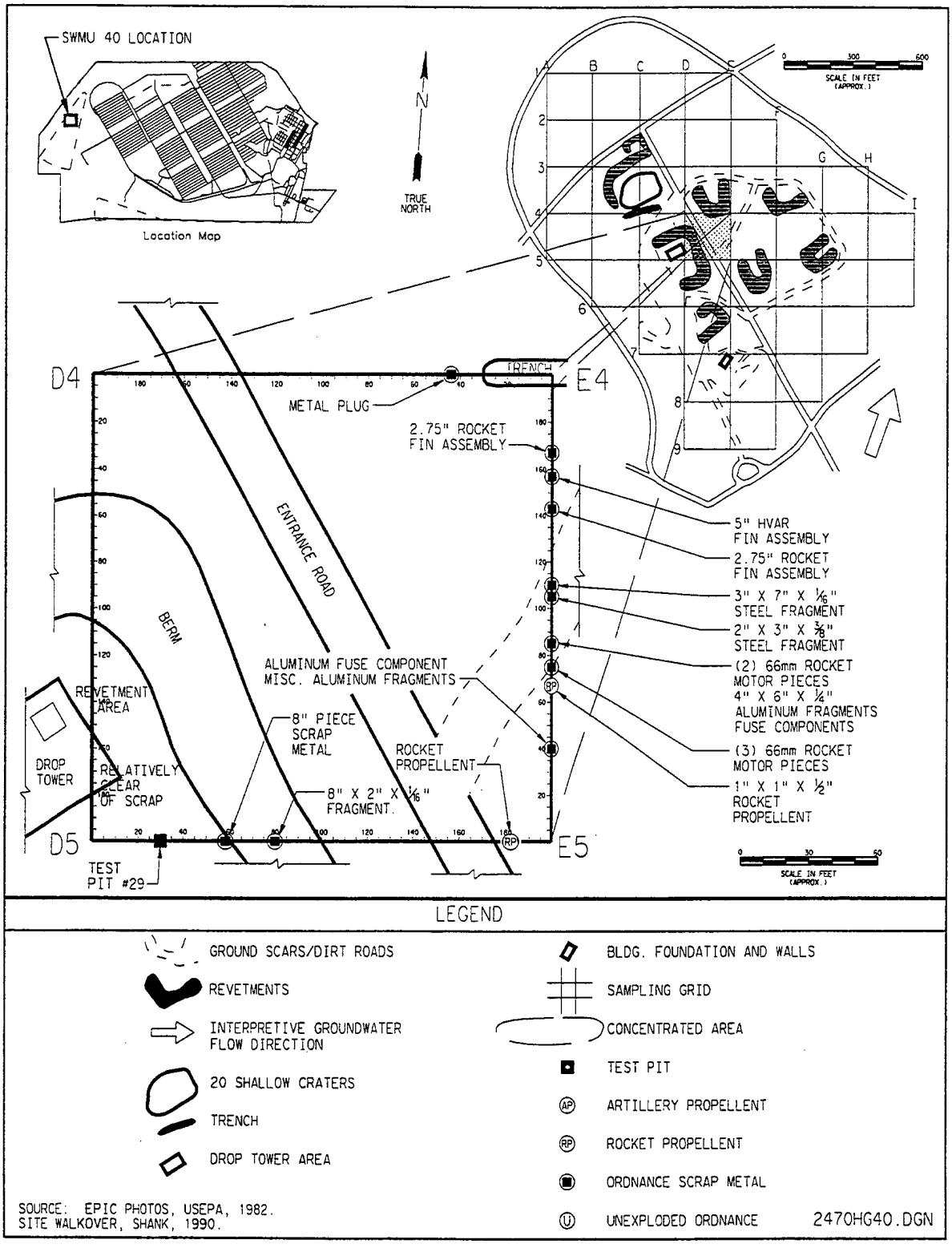


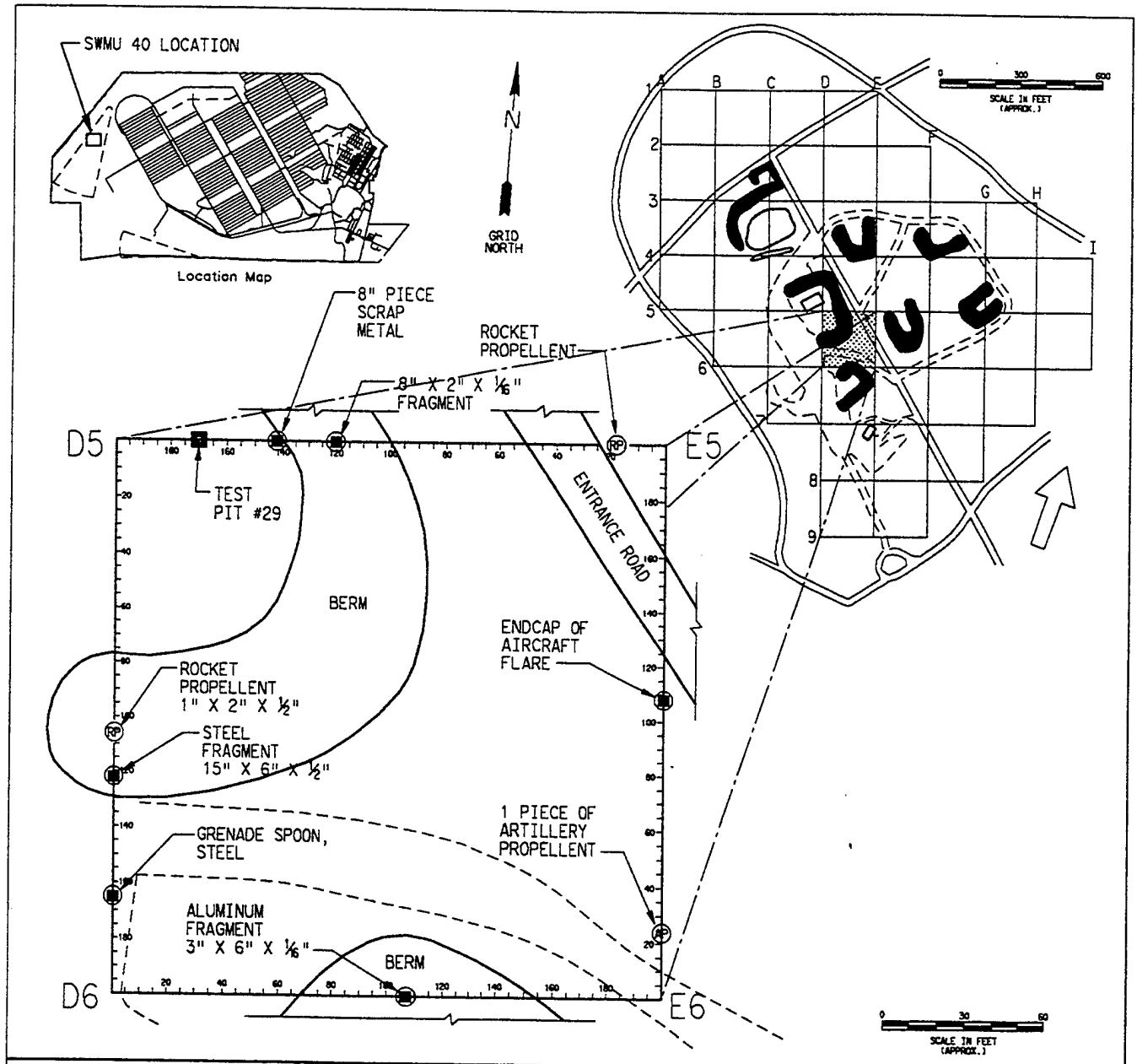
LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG39.DGN


SWMU 40 LOCATION
Location Map
N
TRUE NORTH
SCALE IN FEET (APPROX.)
TRENCH
METAL PLUG
2.75" ROCKET FIN ASSEMBLY
5" HVAR FIN ASSEMBLY
2.75" ROCKET FIN ASSEMBLY
3" X 7" X 1/16" STEEL FRAGMENT
2" X 3" X 3/8" STEEL FRAGMENT
(2) 66mm ROCKET MOTOR PIECES
4" X 6" X 1/4" ALUMINUM FRAGMENTS
FUSE COMPONENTS
(3) 66mm ROCKET MOTOR PIECES
1" X 1" X 1/2" ROCKET PROPELLENT
ENTRANCE ROAD
BERM
REVETMENT AREA
DROP TOWER
RELATIVELY CLEAR OF SCRAP
ALUMINUM FUSE COMPONENT
MISC. ALUMINUM FRAGMENTS
8" PIECE SCRAP METAL
ROCKET PROPELLENT
8" X 2" X 1/16" FRAGMENT.
TEST PIT #29
D4
D5
E4
E5
LEGEND
GROUND SCARS/DIRT ROADS
REVETMENTS
INTERPRETIVE GROUNDWATER FLOW DIRECTION
20 SHALLOW CRATERS
TRENCH
DROP TOWER AREA
BLDG. FOUNDATION AND WALLS
SAMPLING GRID
CONCENTRATED AREA
TEST PIT
ARTILLERY PROPELLENT
ROCKET PROPELLENT
ORDNANCE SCRAP METAL
UNEXPLODED ORDNANCE
SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.
2470HG40.DGN

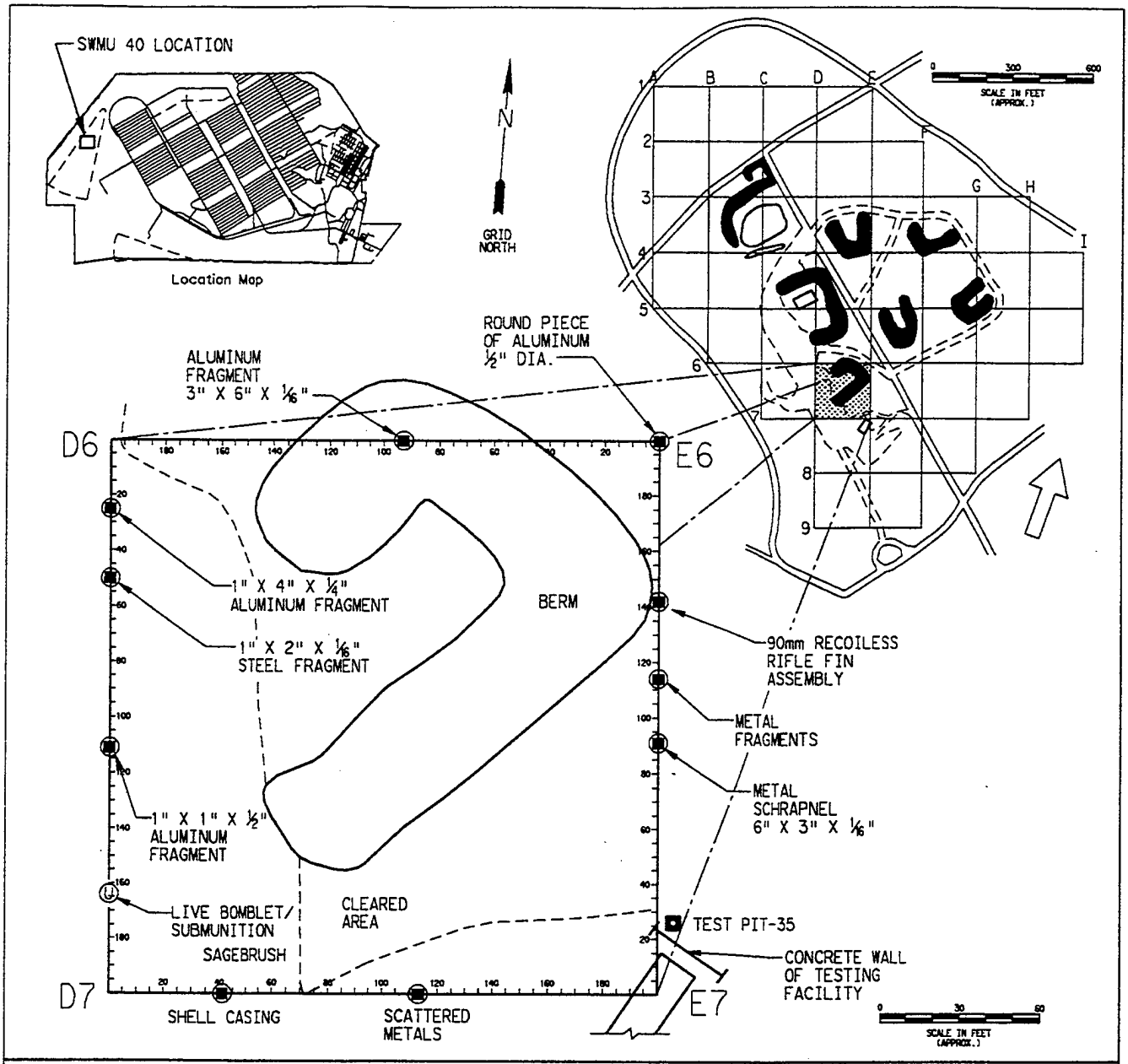


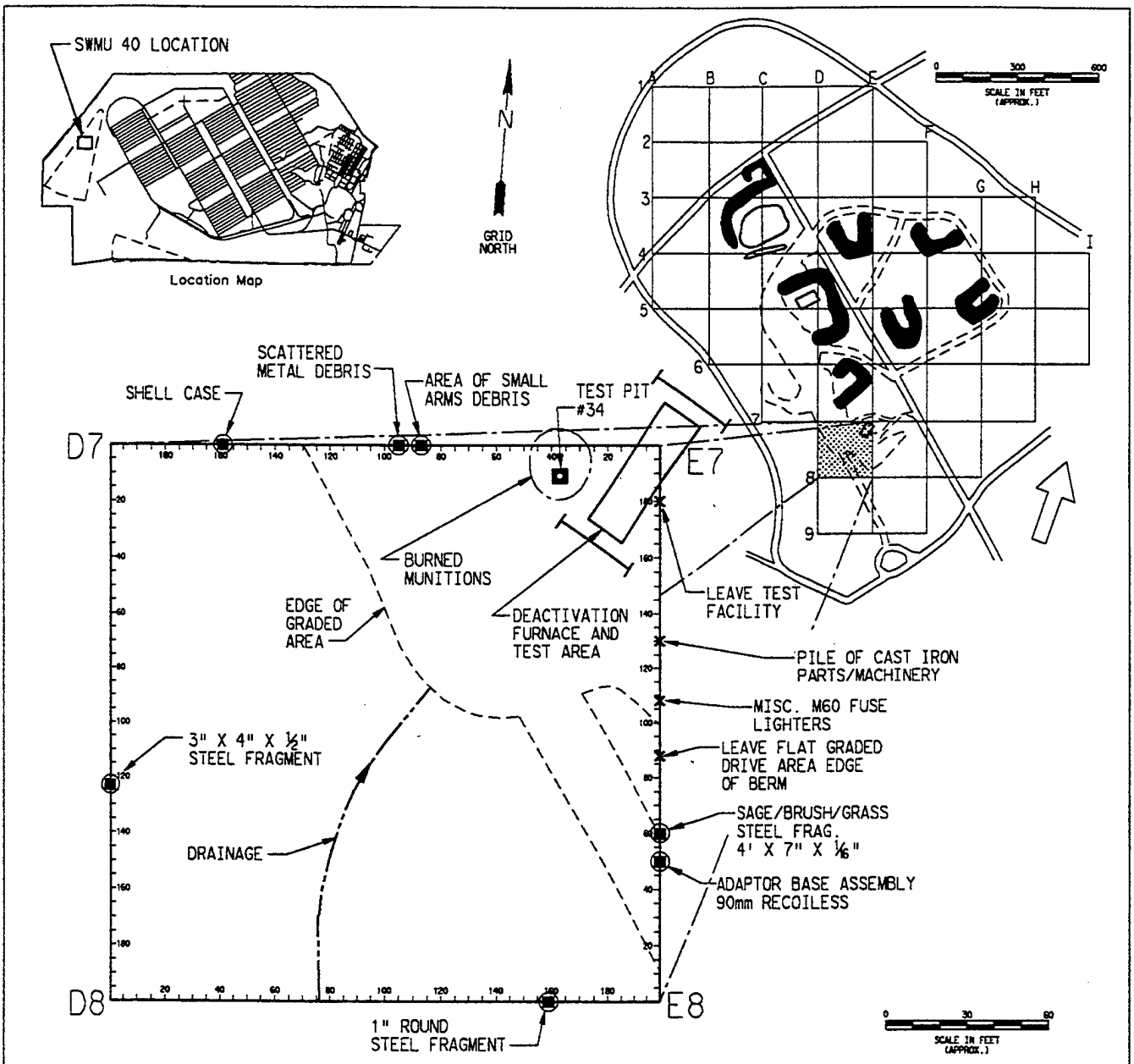
LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG41.DGN


SWMU 40 LOCATION
Location Map
GRID NORTH
SCALE IN FEET (APPROX.)
ROUND PIECE OF ALUMINUM ½" DIA.
ALUMINUM FRAGMENT 3" X 6" X 1/16"
D6
E6
D7
E7
1" X 4" X ¼" ALUMINUM FRAGMENT
1" X 2" X 1/16" STEEL FRAGMENT
BERM
90mm RECOILESS RIFLE FIN ASSEMBLY
METAL FRAGMENTS
METAL SCHRAPNEL 6" X 3" X 1/16"
1" X 1" X ½" ALUMINUM FRAGMENT
LIVE BOMBLET/ SUBMUNITION
SAGEBRUSH
CLEARED AREA
TEST PIT-35
CONCRETE WALL OF TESTING FACILITY
SHELL CASING
SCATTERED METALS
SCALE IN FEET (APPROX.)
LEGEND
GROUND SCARS/DIRT ROADS
REVETMENTS
INTERPRETIVE GROUNDWATER FLOW DIRECTION
20 SHALLOW CRATERS
TRENCH
DROP TOWER AREA
BLDG. FOUNDATION AND WALLS
SAMPLING GRID
CONCENTRATED AREA
TEST PIT
ARTILLERY PROPELLENT
ROCKET PROPELLENT
ORDNANCE SCRAP METAL
UNEXPLODED ORDNANCE
SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.
2470HG42.DGN

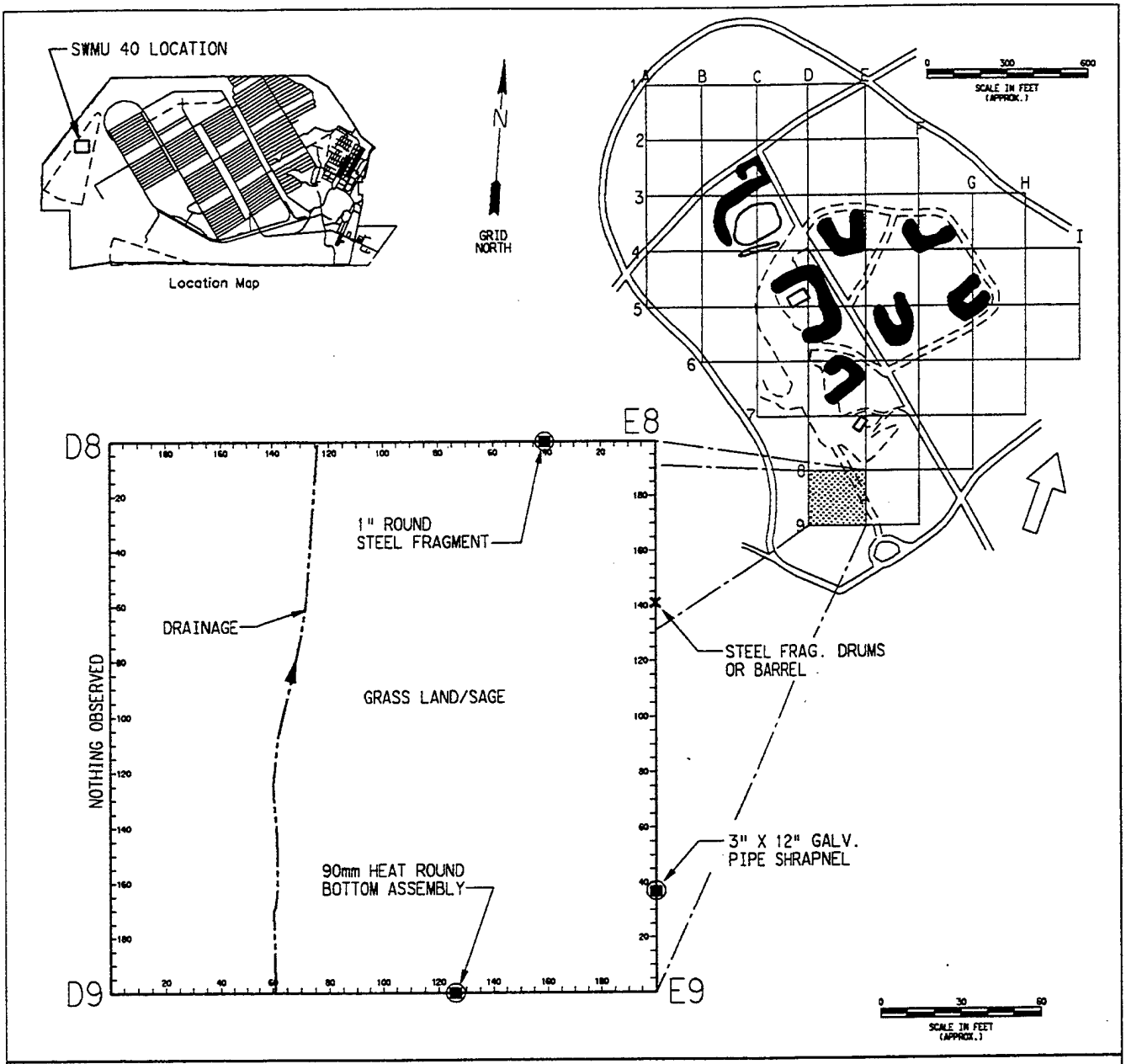


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG13.DGN

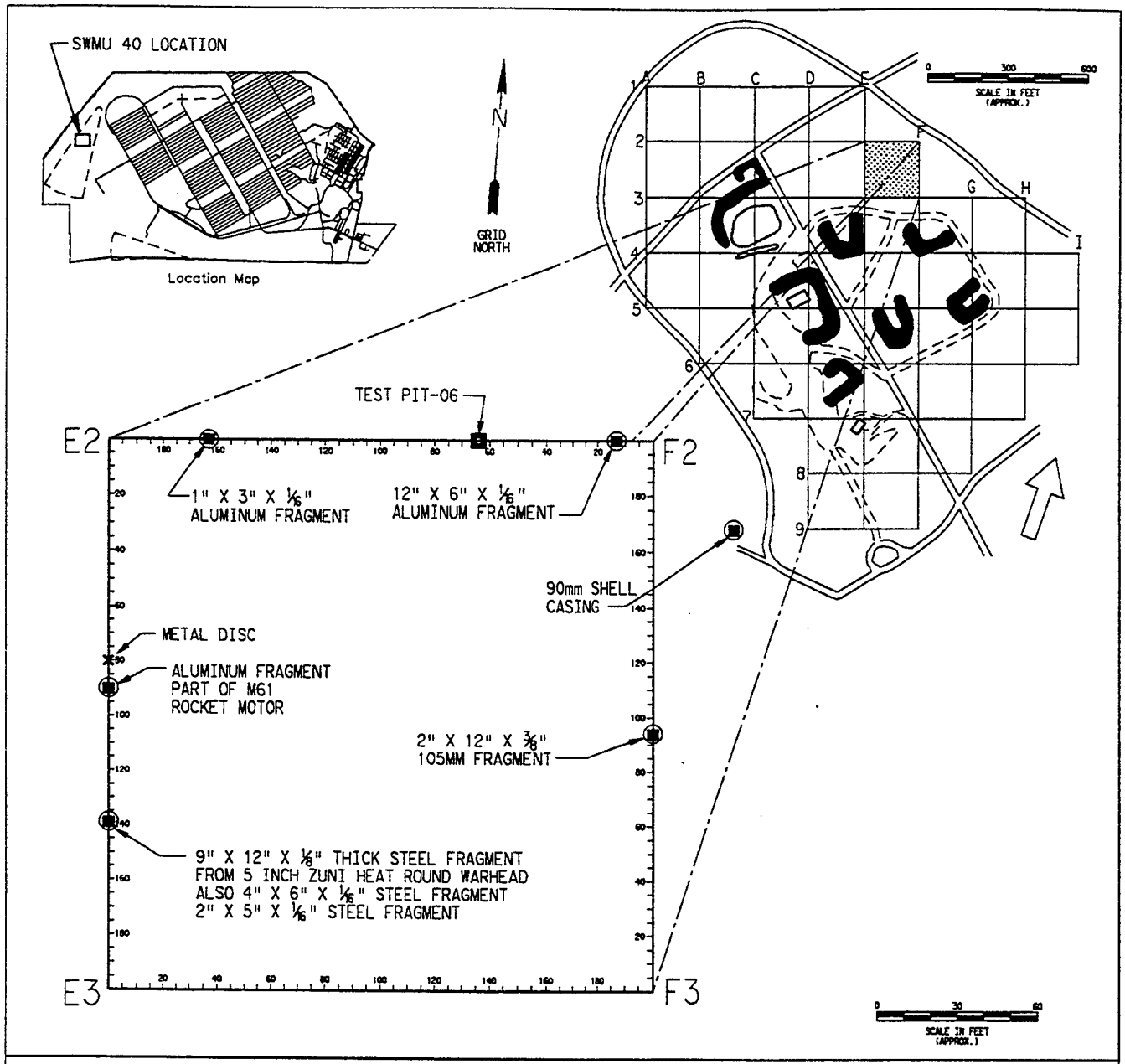


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG12.DGN

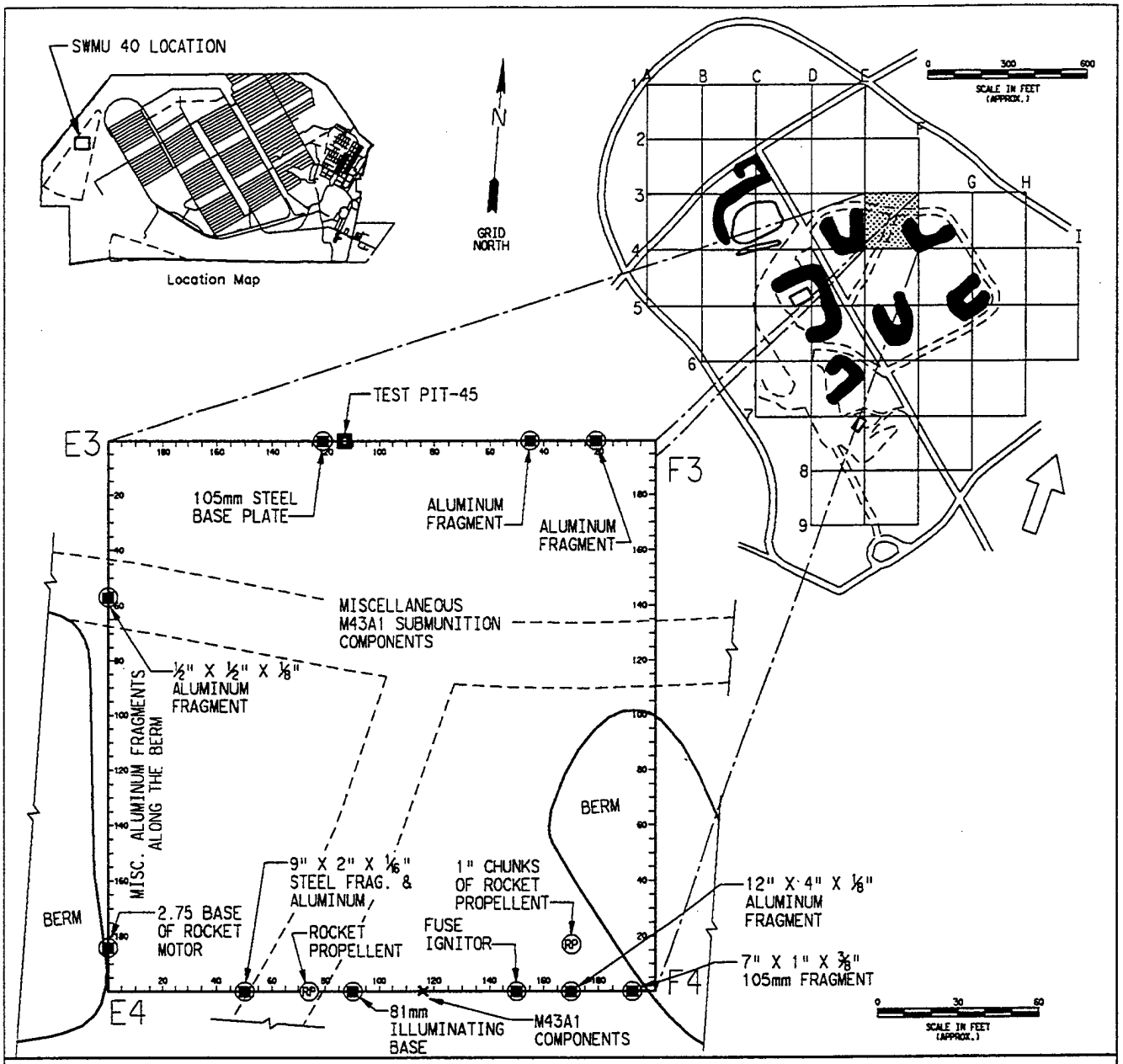


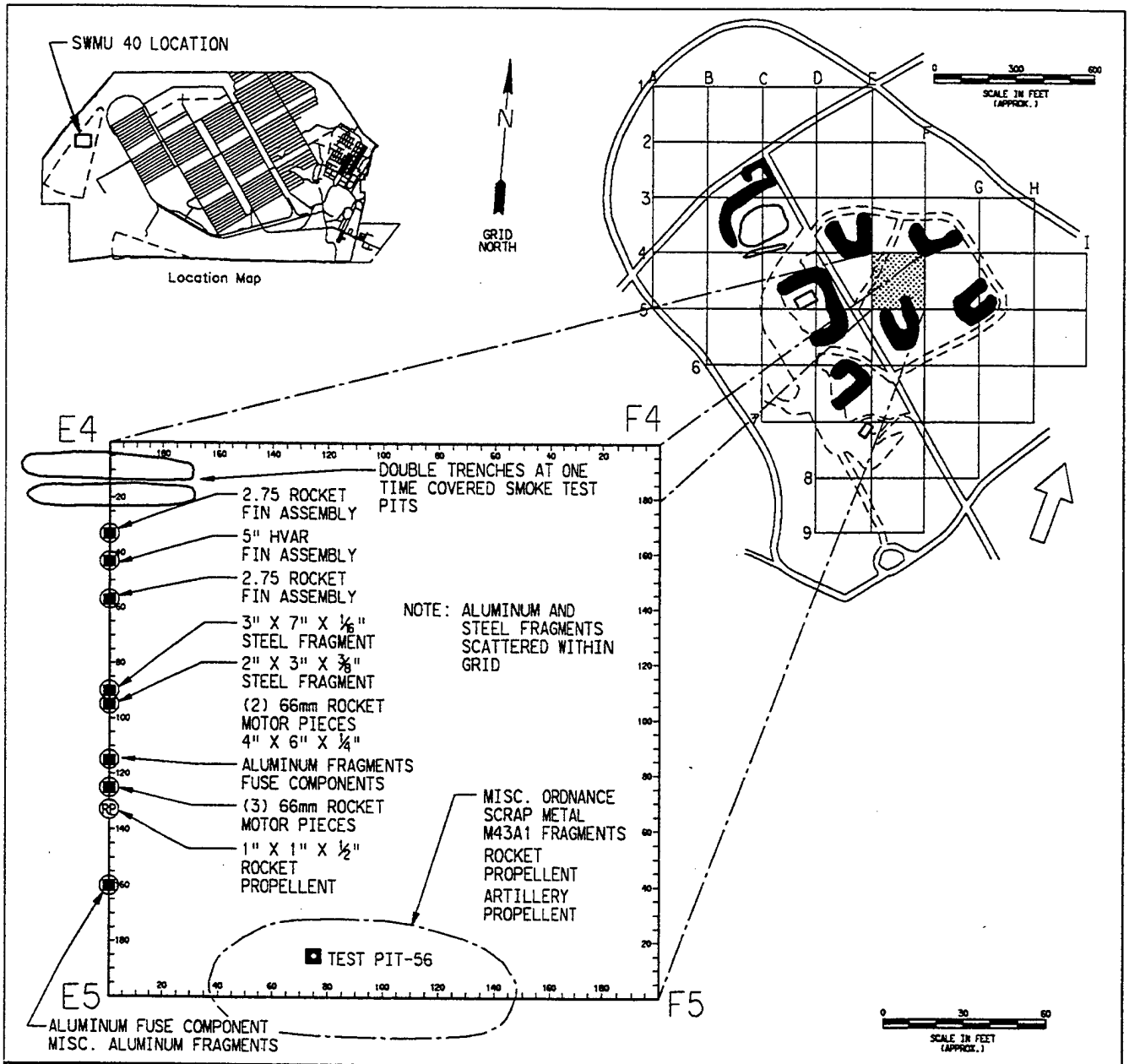
LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG15.DGN


SWMU 40 LOCATION
Location Map
GRID NORTH
SCALE IN FEET (APPROX.)
TEST PIT-45
105mm STEEL BASE PLATE
ALUMINUM FRAGMENT
ALUMINUM FRAGMENT
MISCELLANEOUS M43A1 SUBMUNITION COMPONENTS
½" X ½" X ⅛" ALUMINUM FRAGMENT
MISC. ALUMINUM FRAGMENTS ALONG THE BERM
BERM
2.75 BASE OF ROCKET MOTOR
9" X 2" X ⅛" STEEL FRAG. & ALUMINUM
ROCKET PROPELLENT
1" CHUNKS OF ROCKET PROPELLENT
FUSE IGNITOR
81mm ILLUMINATING BASE
M43A1 COMPONENTS
12" X 4" X ⅛" ALUMINUM FRAGMENT
7" X 1" X ⅜" 105mm FRAGMENT
E3
E4
F3
F4
LEGEND
GROUND SCARS/DIRT ROADS
REVETMENTS
INTERPRETIVE GROUNDWATER FLOW DIRECTION
20 SHALLOW CRATERS
TRENCH
DROP TOWER AREA
BLDG. FOUNDATION AND WALLS
SAMPLING GRID
CONCENTRATED AREA
TEST PIT
ARTILLERY PROPELLENT
ROCKET PROPELLENT
ORDNANCE SCRAP METAL
UNEXPLODED ORDNANCE
SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.
2470HG16.DGN

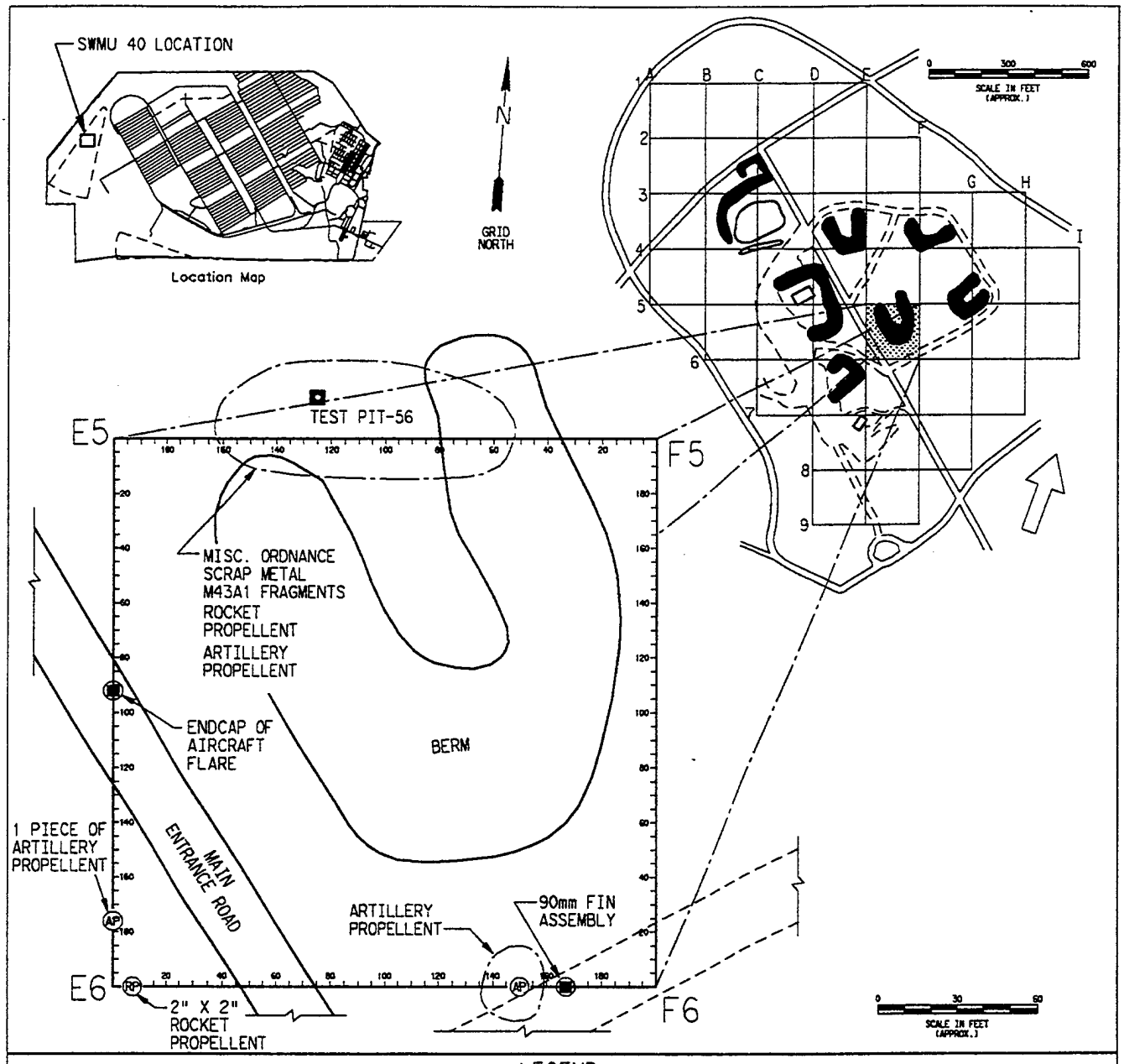


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG17.DGN

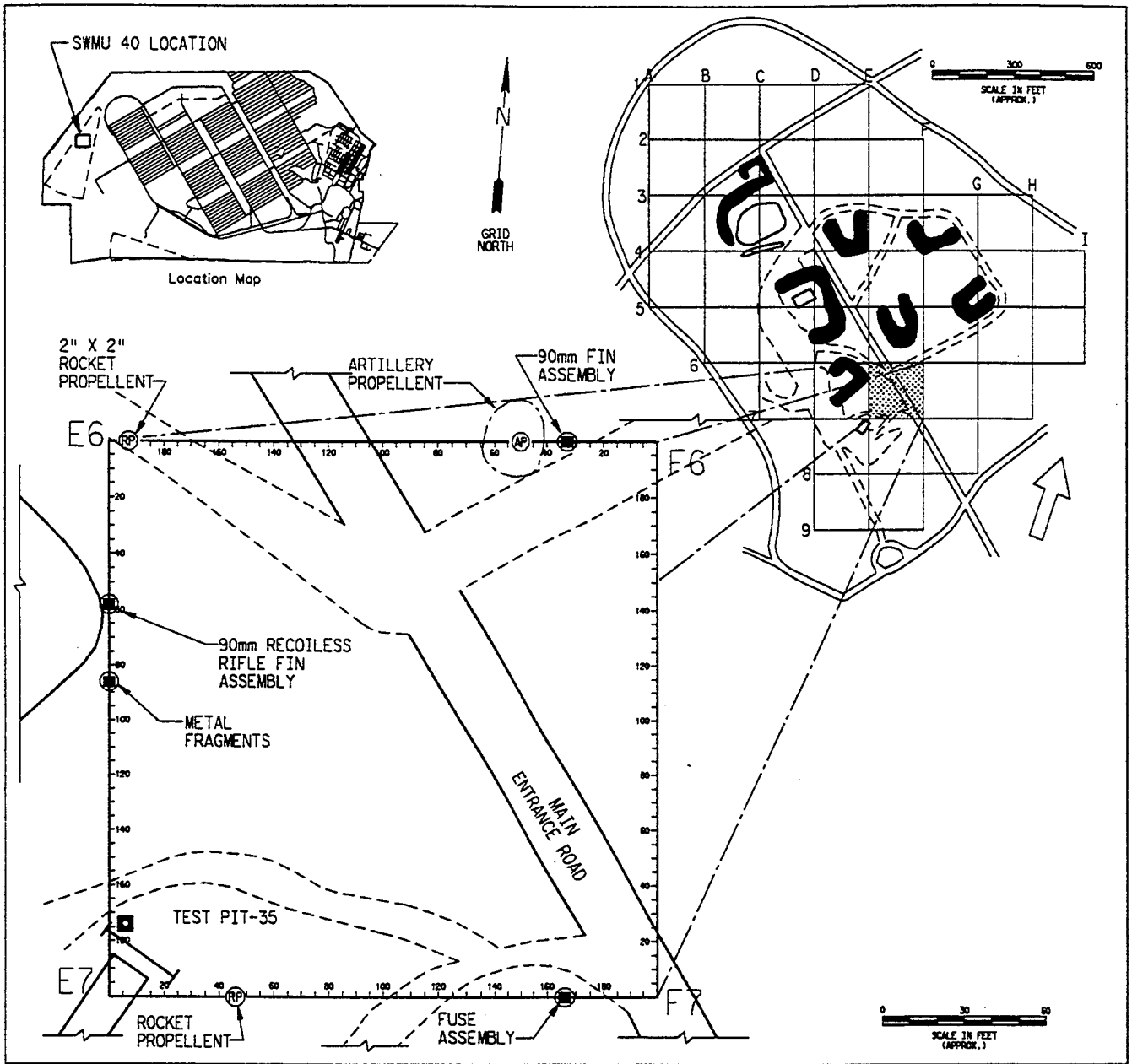


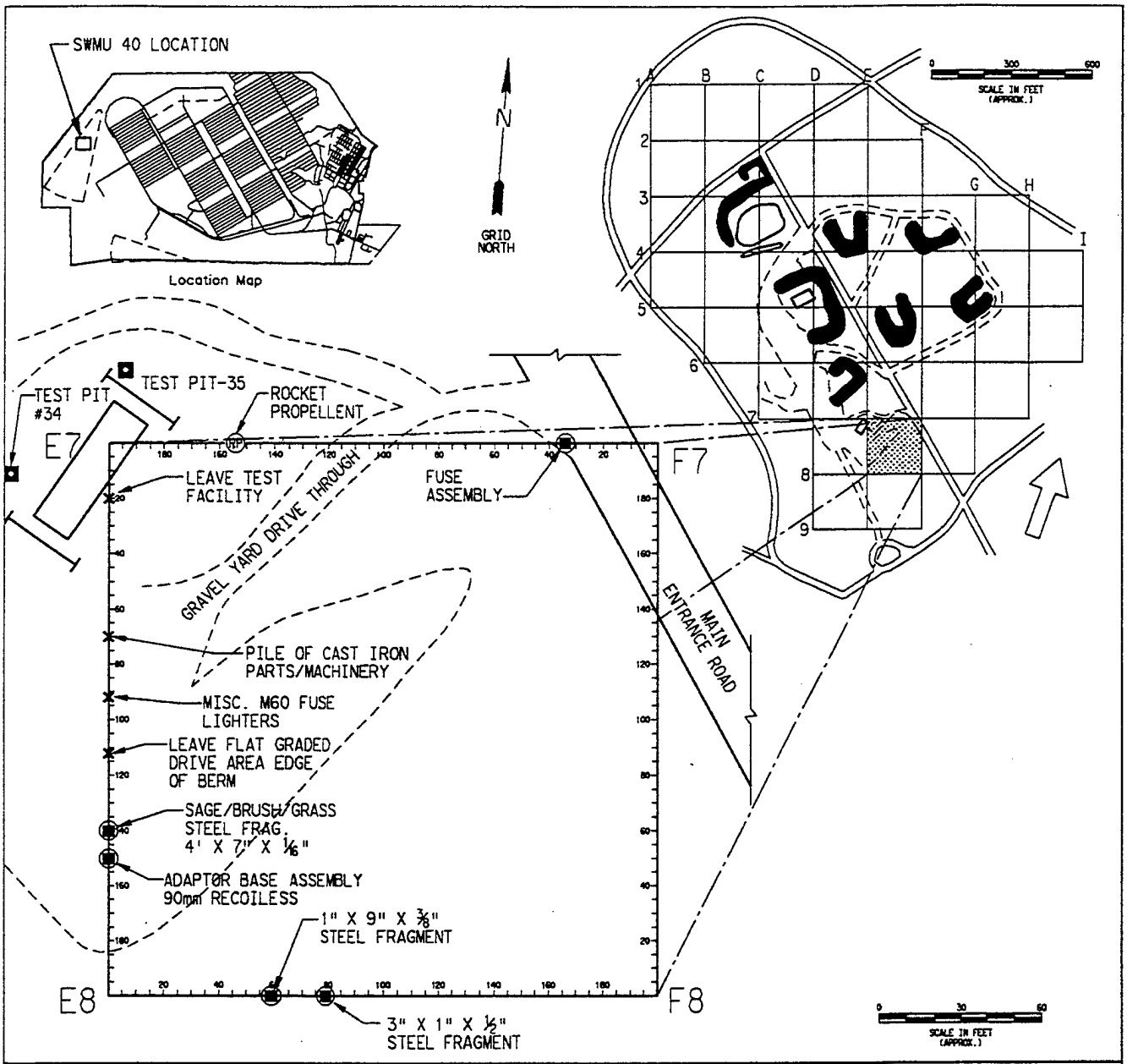
LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- ARTILLERY PROPELLENT
- ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG18.DGN


SWMU 40 LOCATION
Location Map
GRID NORTH
SCALE IN FEET (APPROX.)
2" X 2" ROCKET PROPELLENT
ARTILLERY PROPELLENT
90mm FIN ASSEMBLY
E6
E7
F7
90mm RECOILESS RIFLE FIN ASSEMBLY
METAL FRAGMENTS
MAIN ENTRANCE ROAD
TEST PIT-35
ROCKET PROPELLENT
FUSE ASSEMBLY
LEGEND
GROUND SCARS/DIRT ROADS
REVETMENTS
INTERPRETIVE GROUNDWATER FLOW DIRECTION
20 SHALLOW CRATERS
TRENCH
DROP TOWER AREA
BLDG. FOUNDATION AND WALLS
SAMPLING GRID
CONCENTRATED AREA
TEST PIT
ARTILLERY PROPELLENT
ROCKET PROPELLENT
ORDNANCE SCRAP METAL
UNEXPLODED ORDNANCE
SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.
2470HG19.DGN

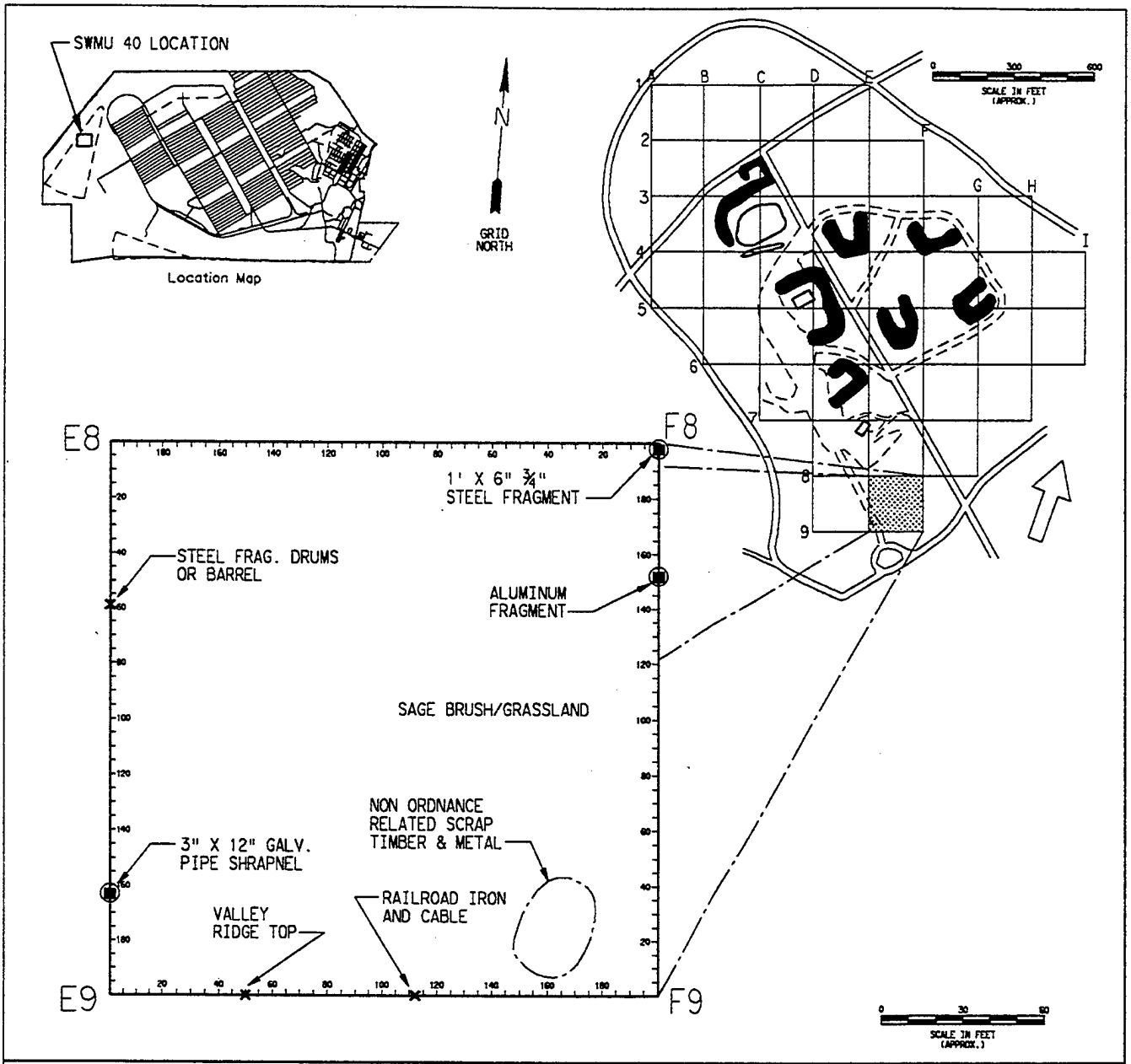


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG20.DGN

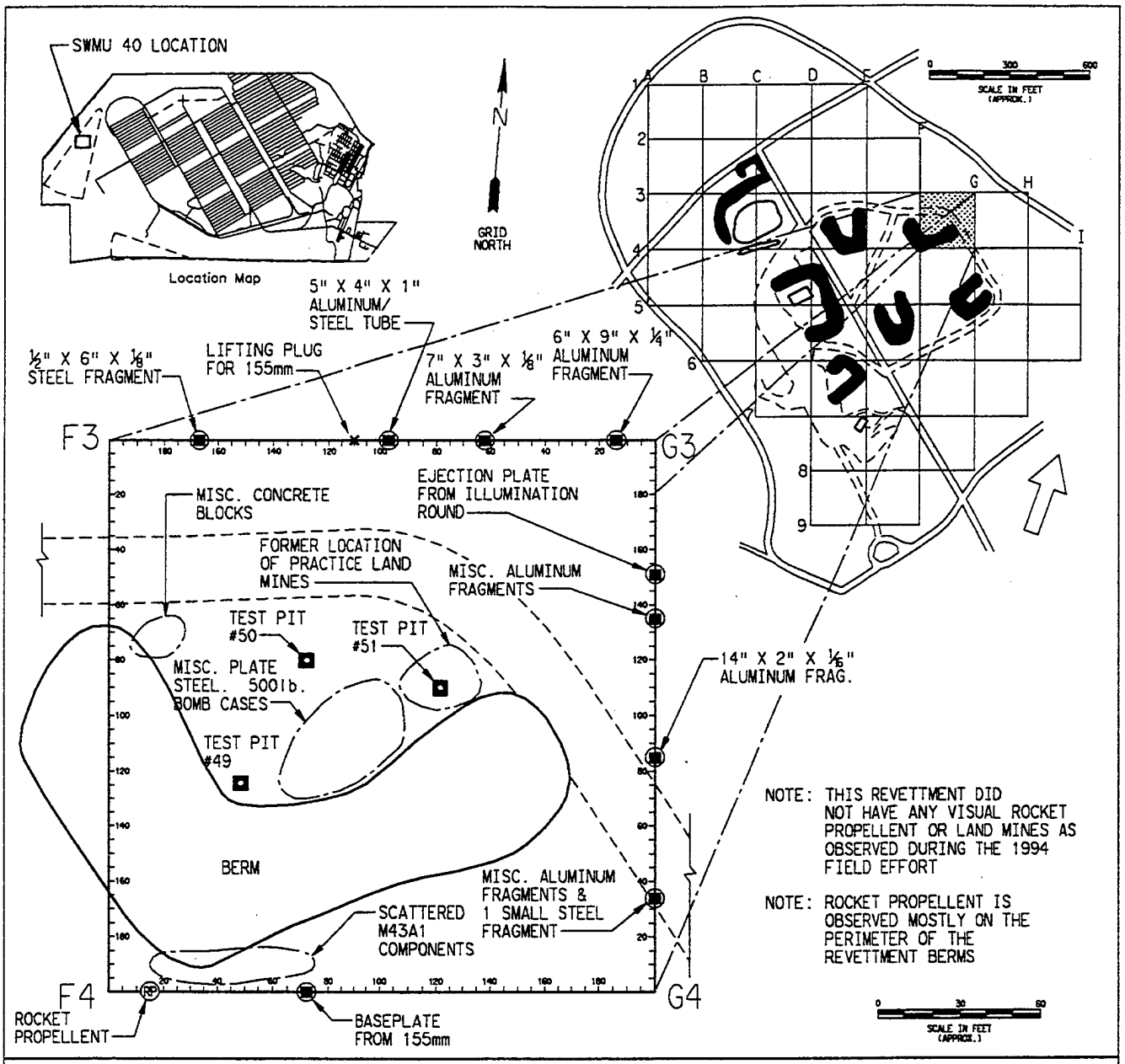


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG21.DGN

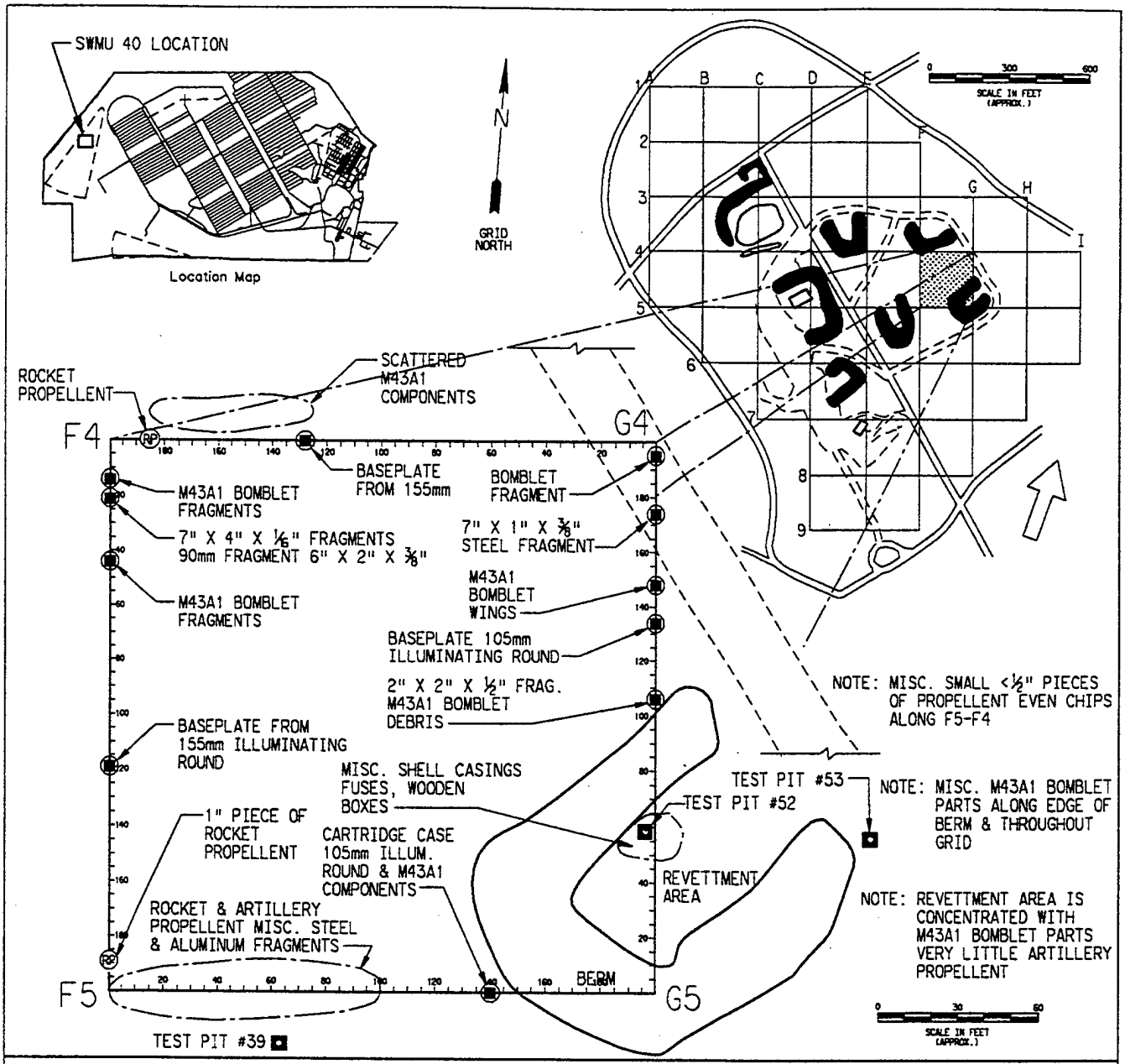


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG22.DGN

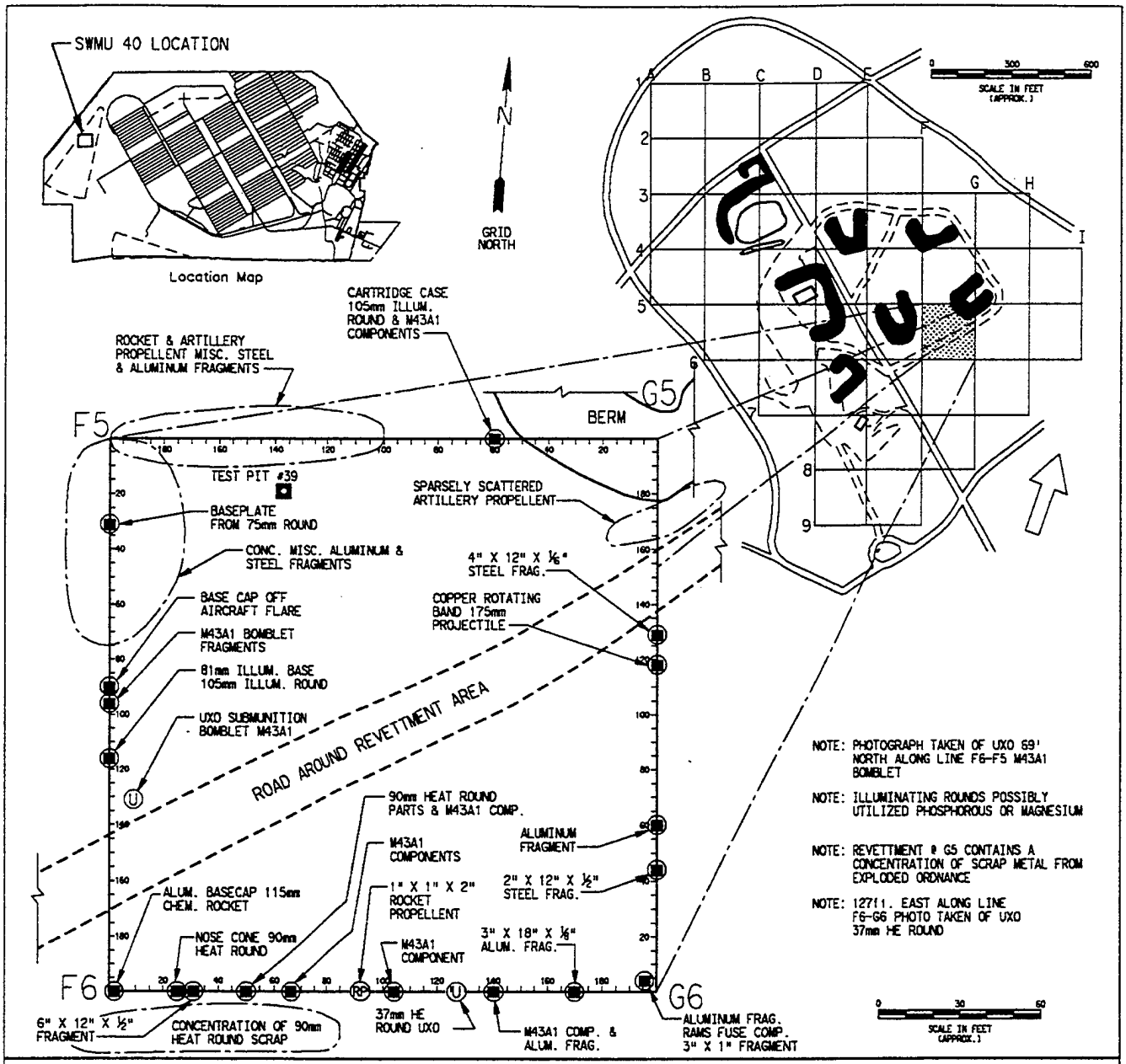


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG23.DGN

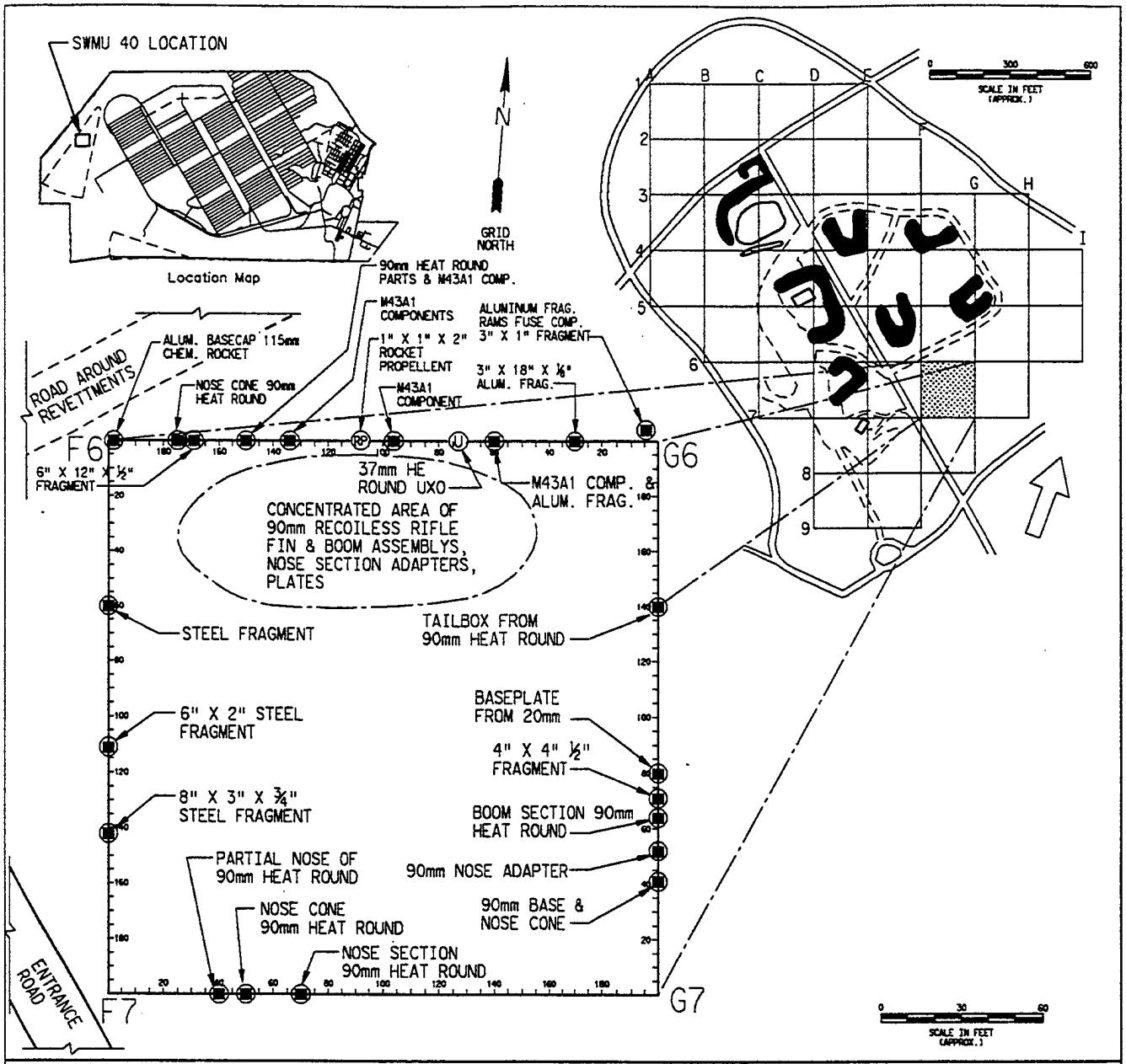


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG24.DGN

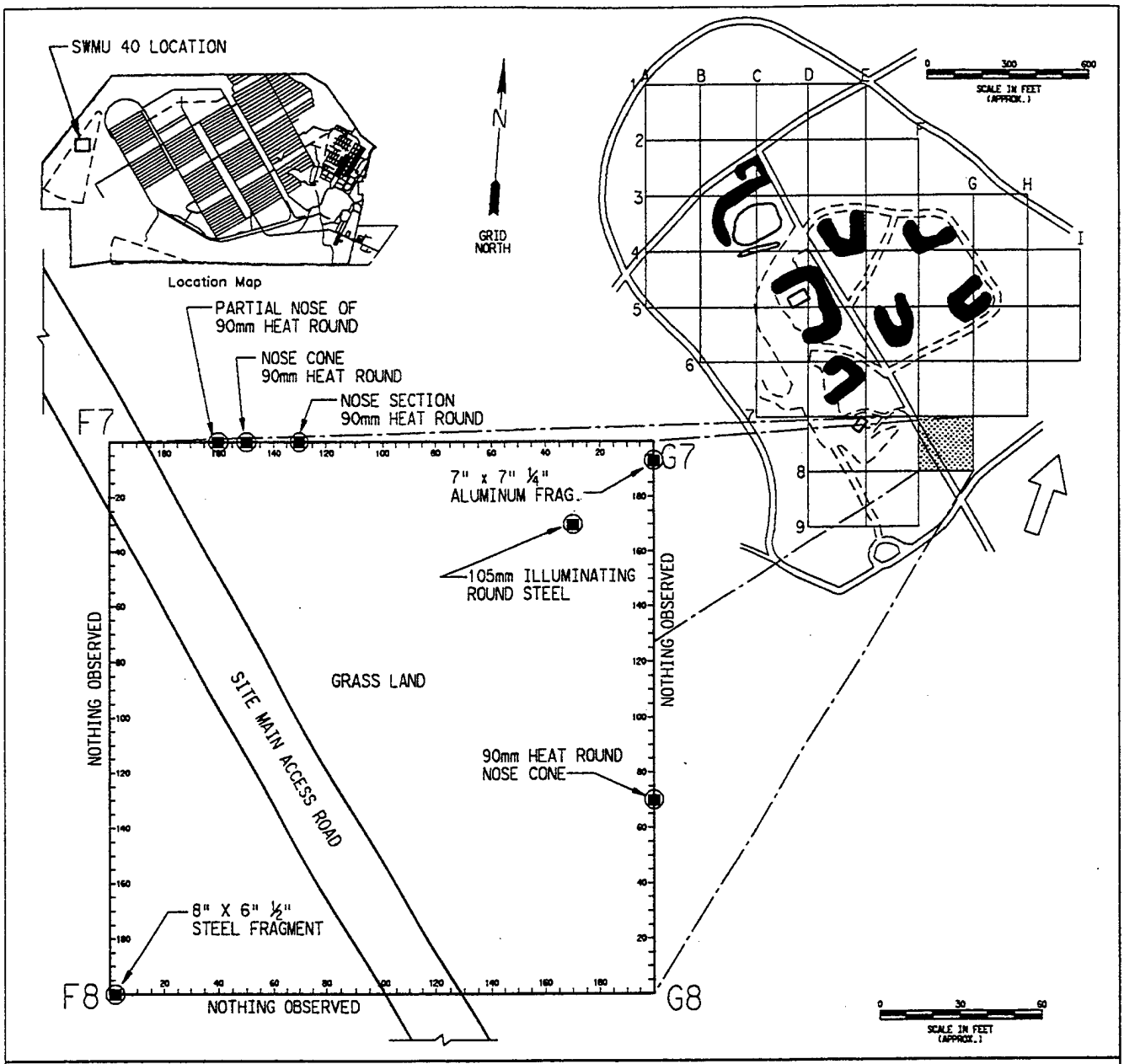


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG25.DGN

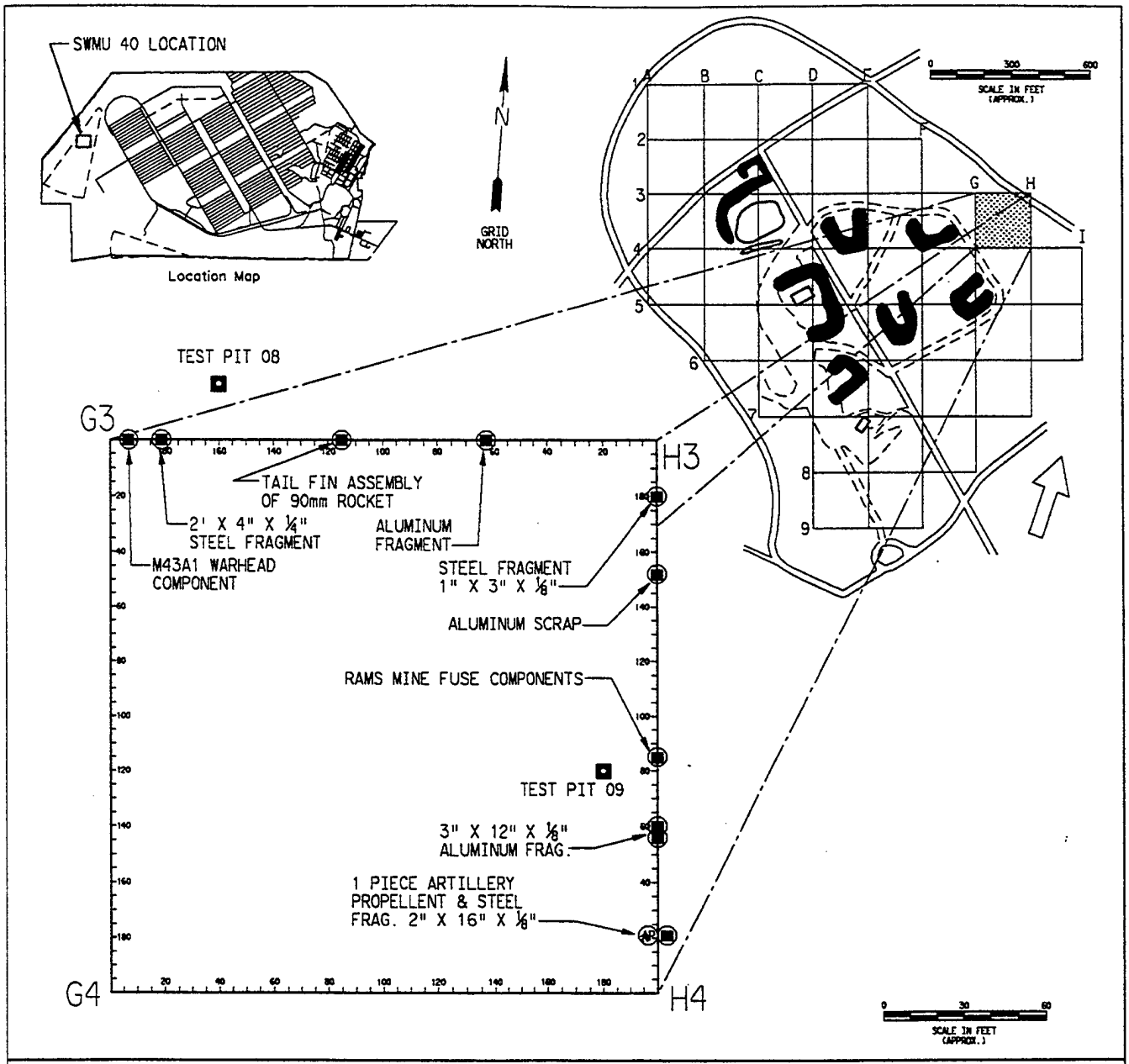


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG26.DGN

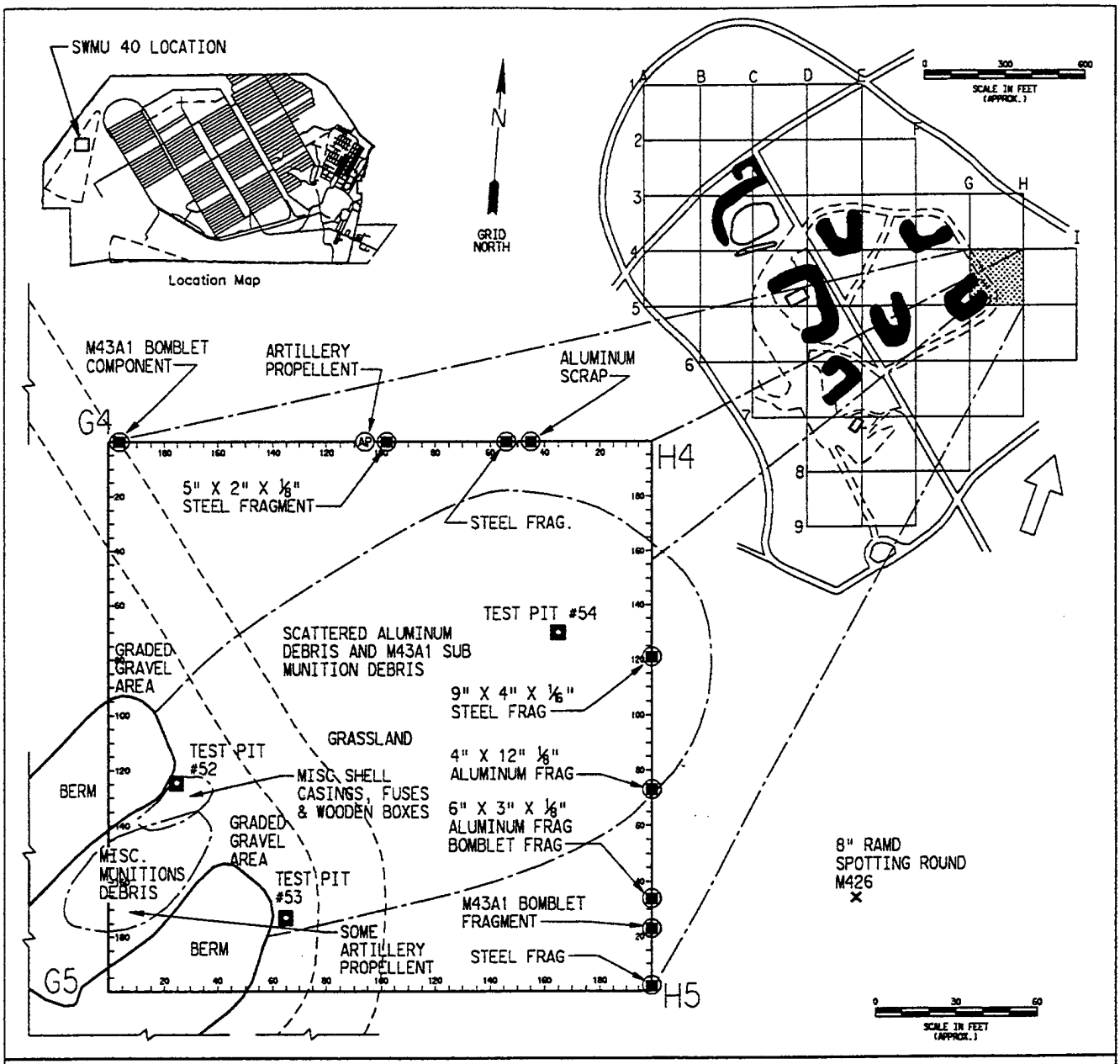


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG27.DGN

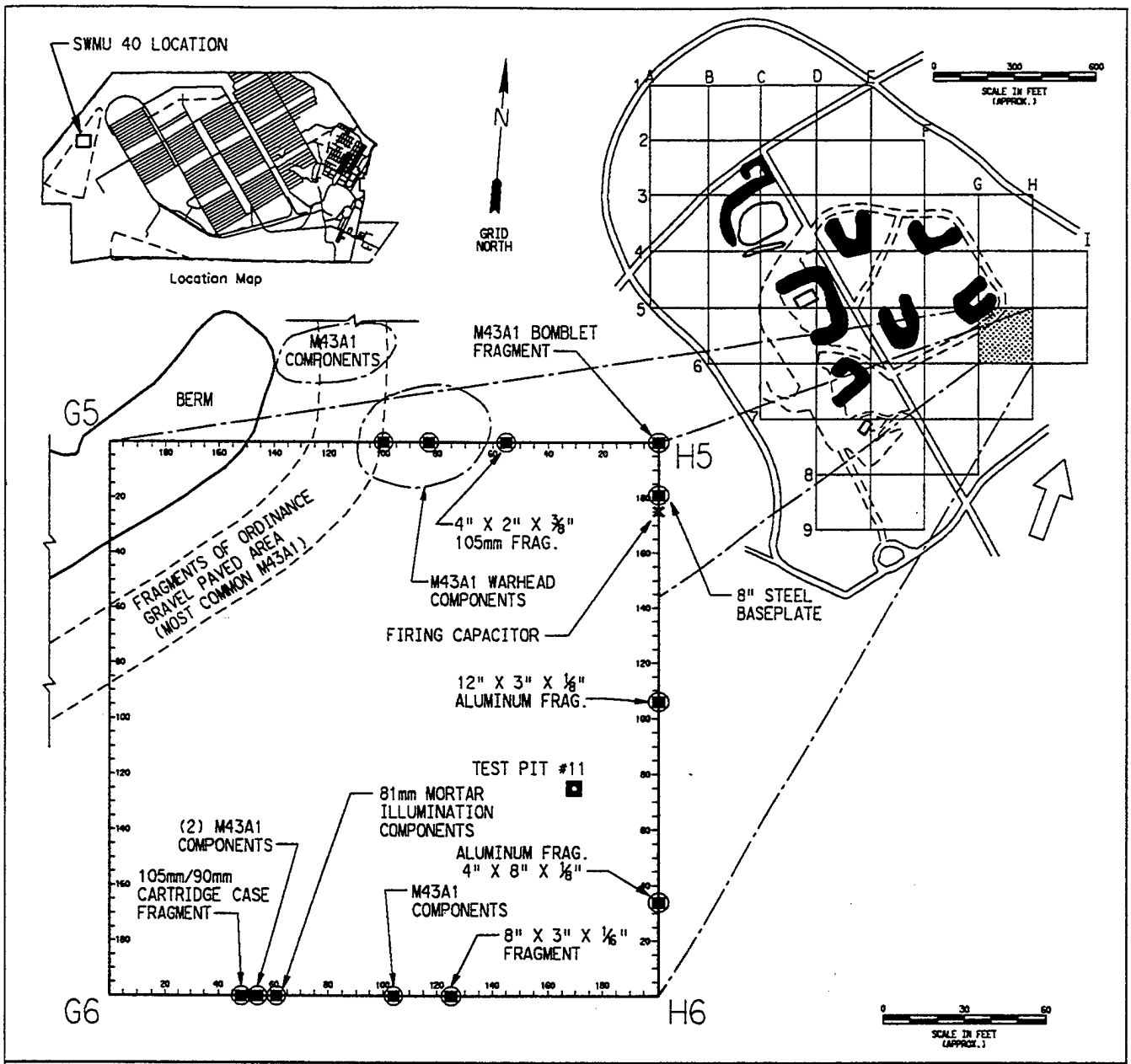


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG28.DGN

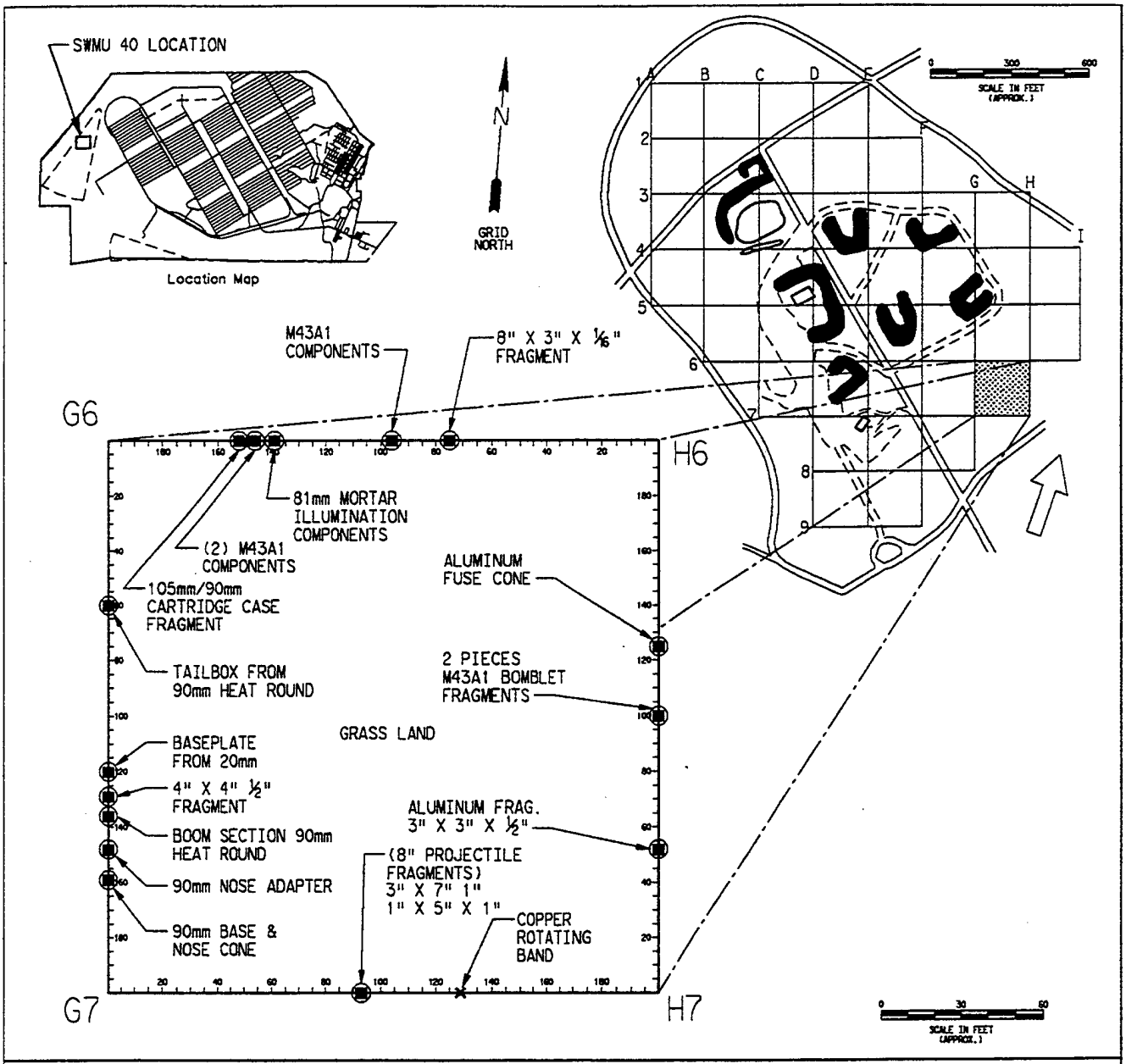


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG29.DGN

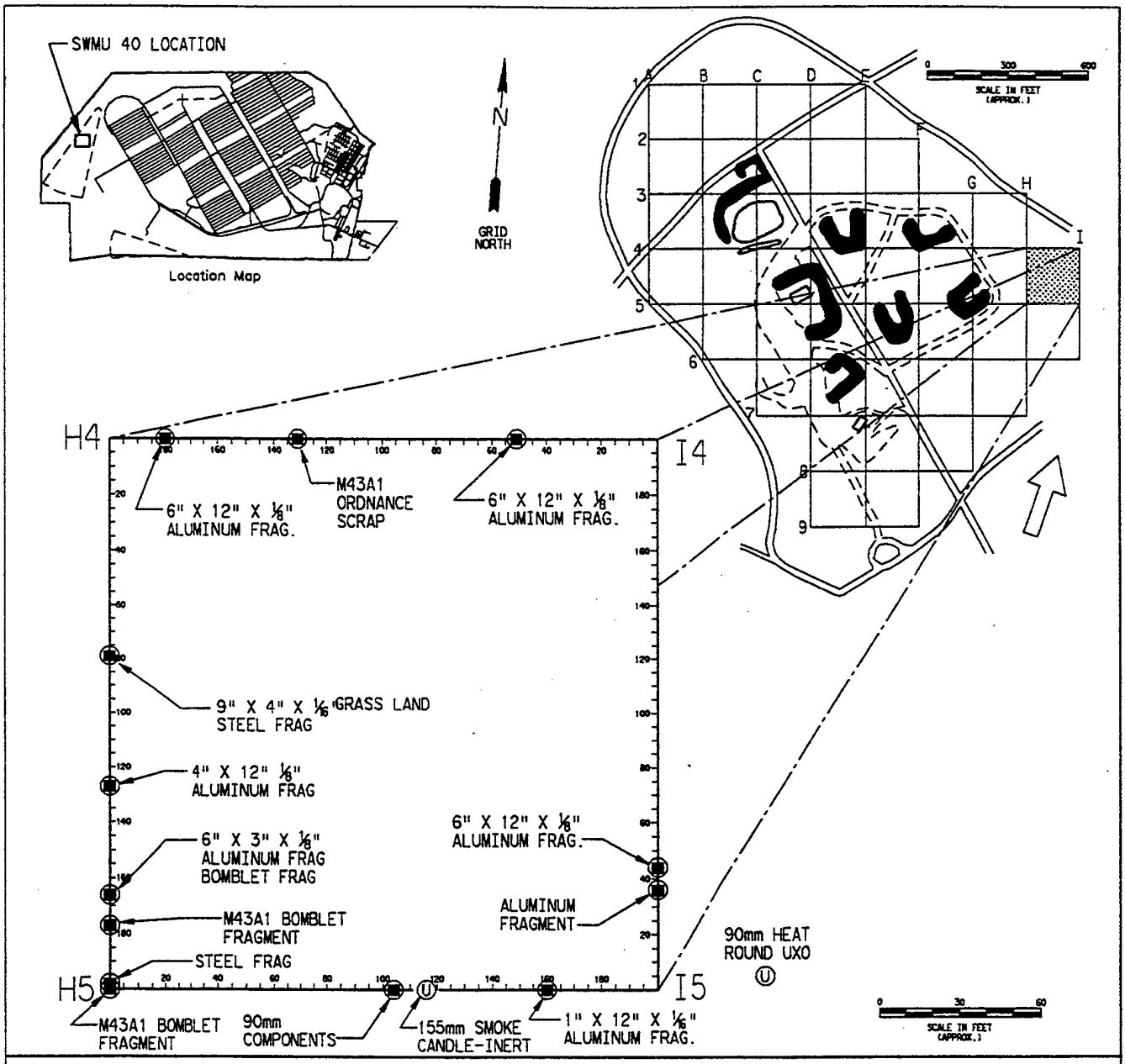


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG30.DGN

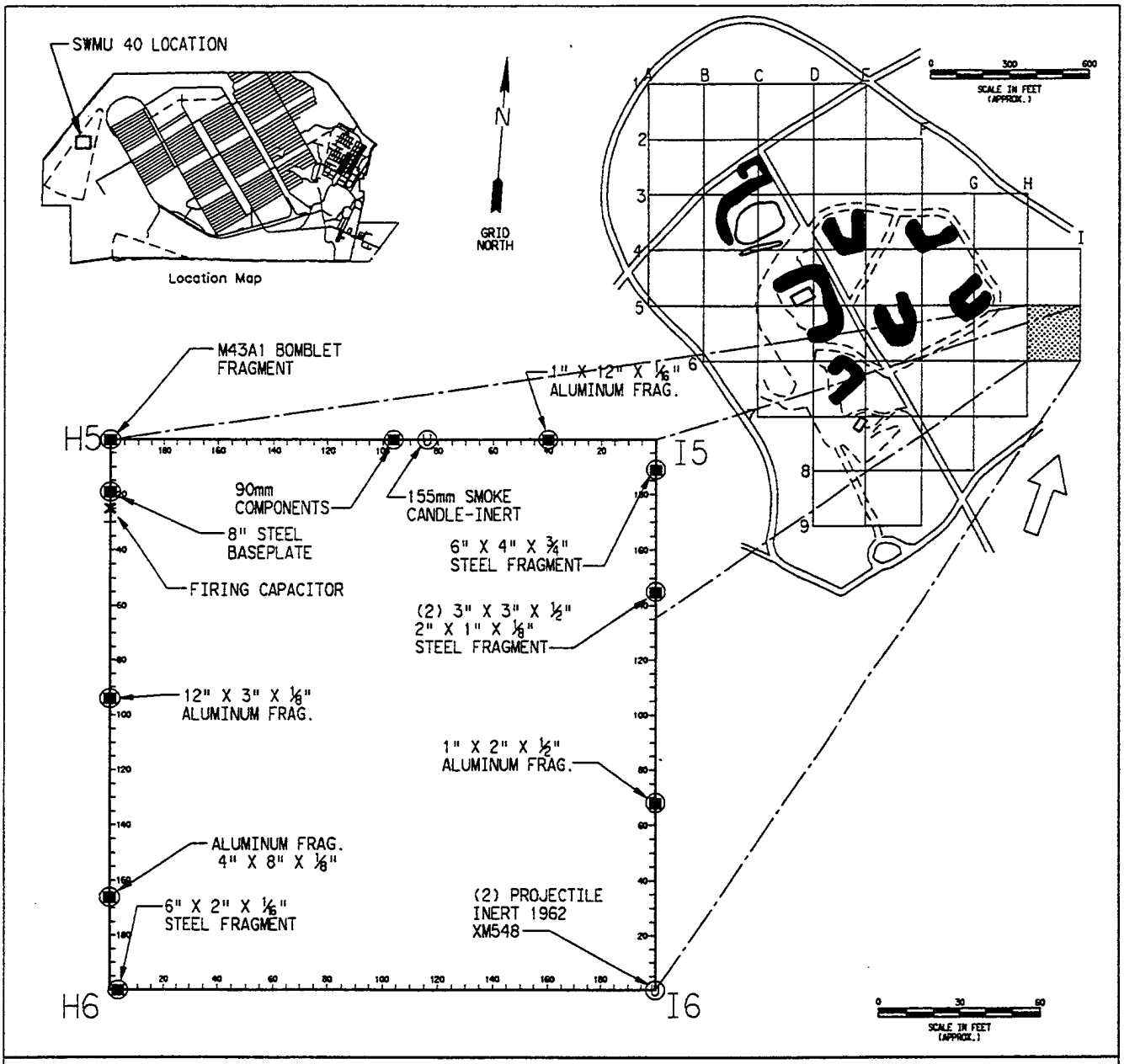


LEGEND

- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

SOURCE: EPIC PHOTOS, USEPA, 1982.
SITE WALKOVER, SHANK, 1990.

2470HG31.DGN



LEGEND

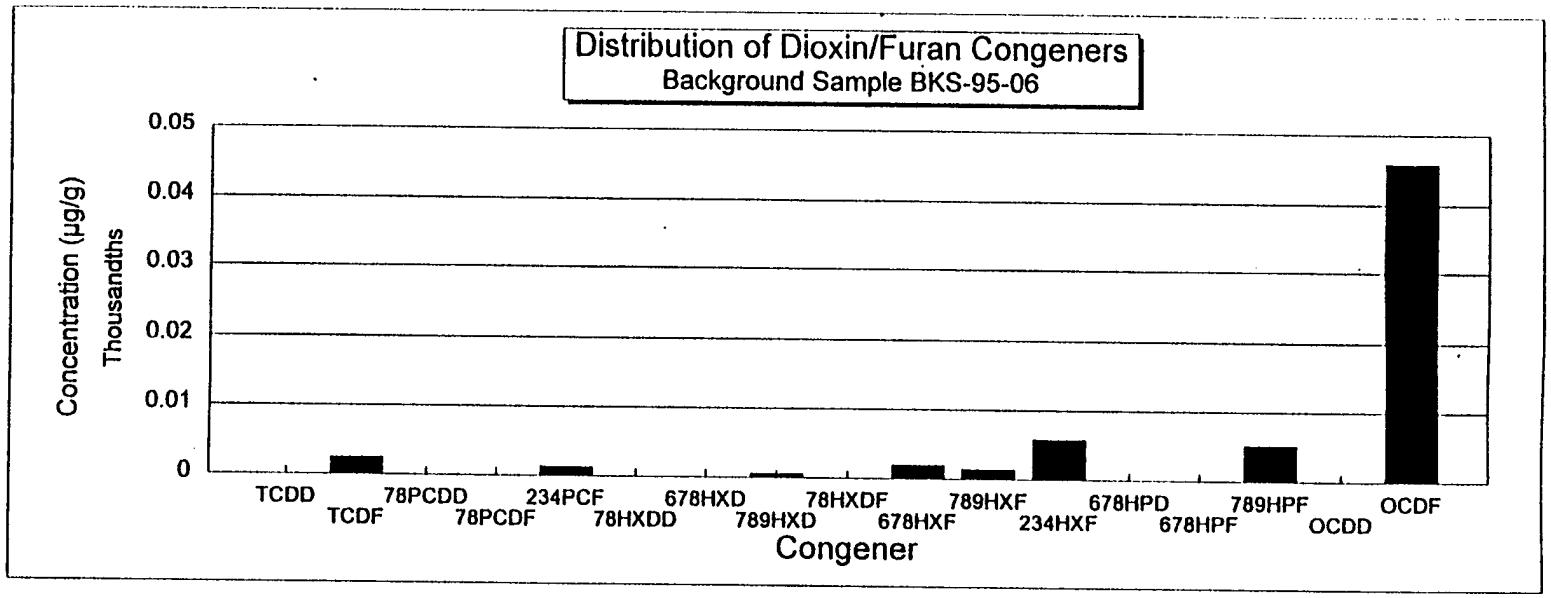
- GROUND SCARS/DIRT ROADS
- REVETMENTS
- INTERPRETIVE GROUNDWATER FLOW DIRECTION
- 20 SHALLOW CRATERS
- TRENCH
- DROP TOWER AREA
- BLDG. FOUNDATION AND WALLS
- SAMPLING GRID
- CONCENTRATED AREA
- TEST PIT
- (AP) ARTILLERY PROPELLENT
- (RP) ROCKET PROPELLENT
- ORDNANCE SCRAP METAL
- (U) UNEXPLODED ORDNANCE

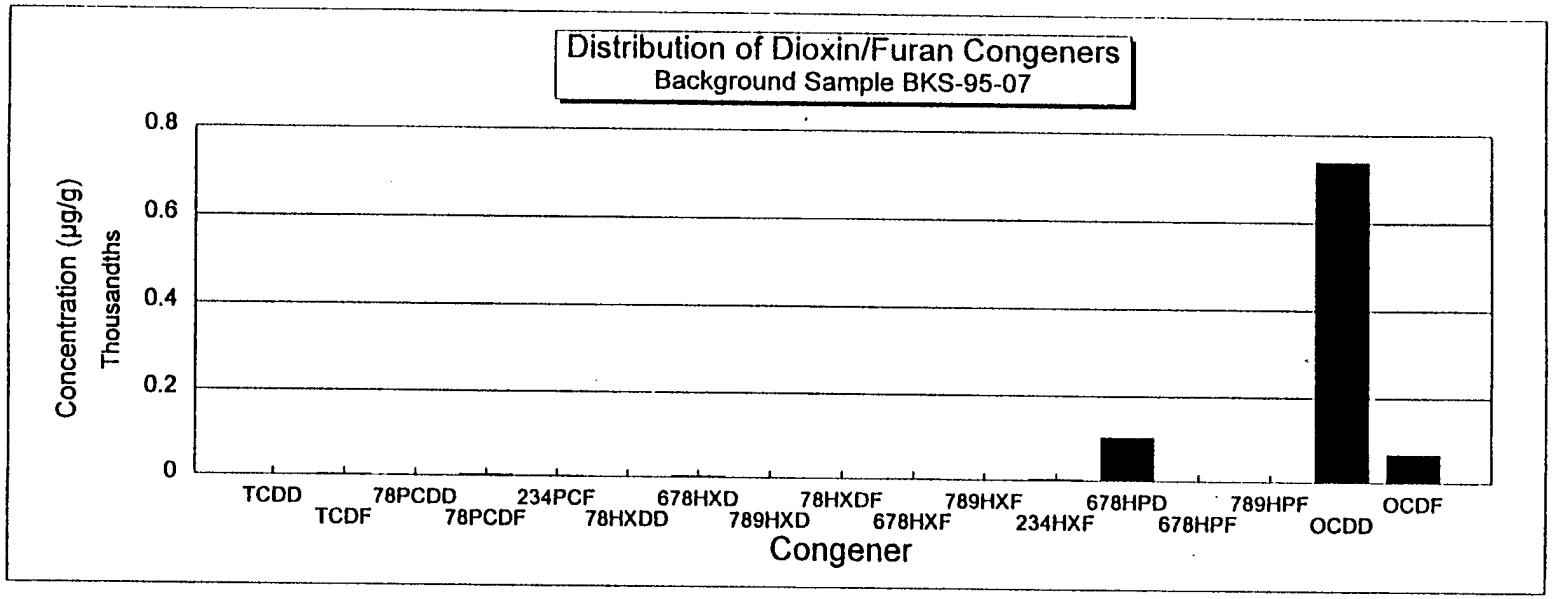
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SITE WALKOVER, SHANK, 1990.

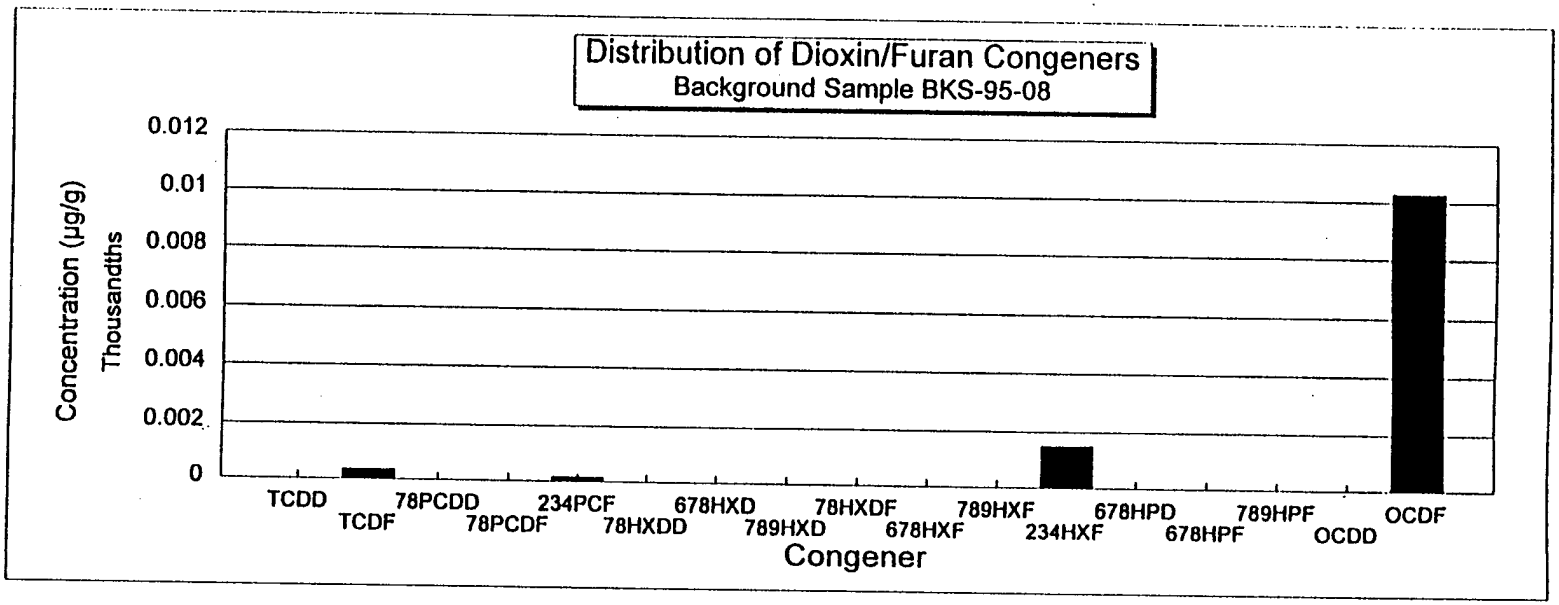
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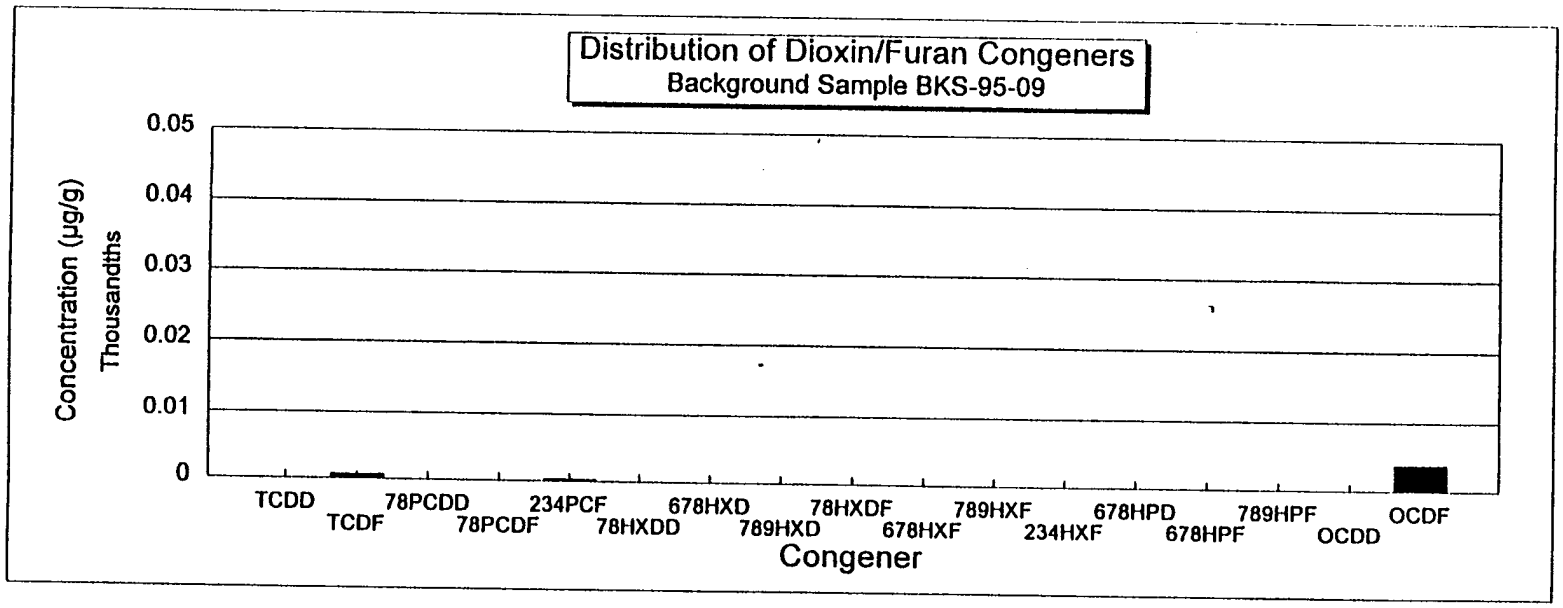
APPENDIX R

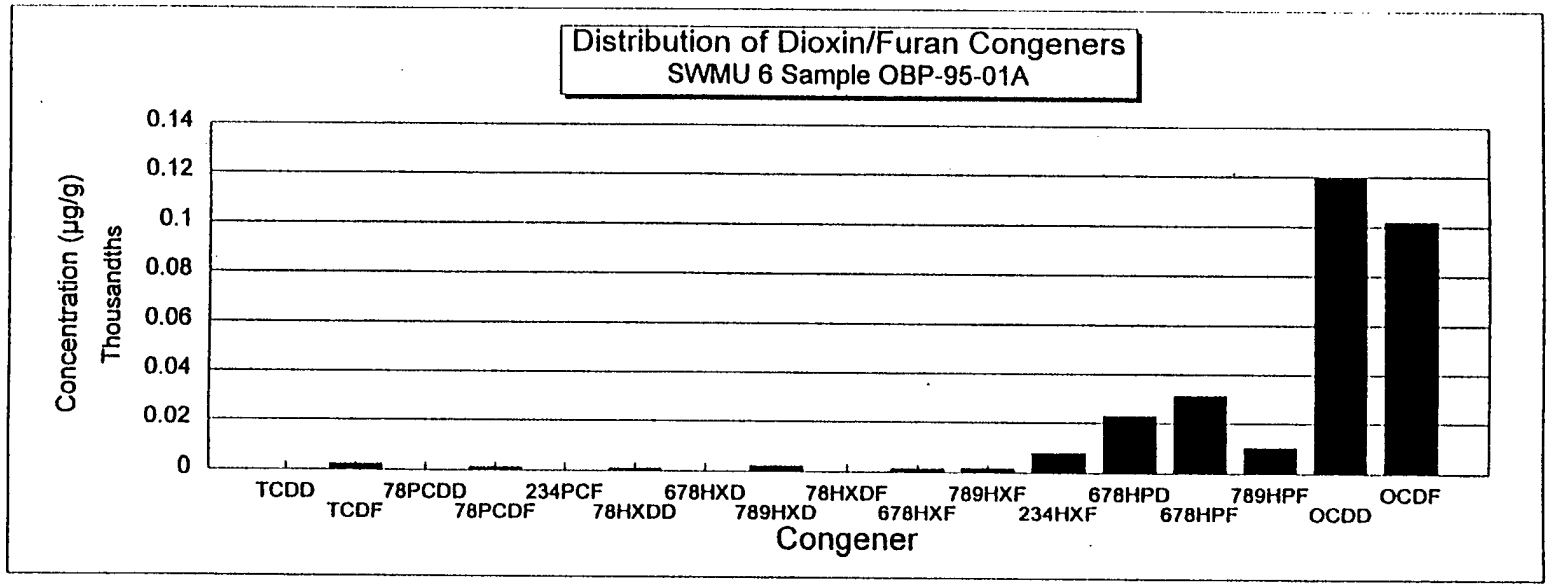
BAR GRAPHS FOR DISTRIBUTION OF DIOXIN/FURAN CONGENERS

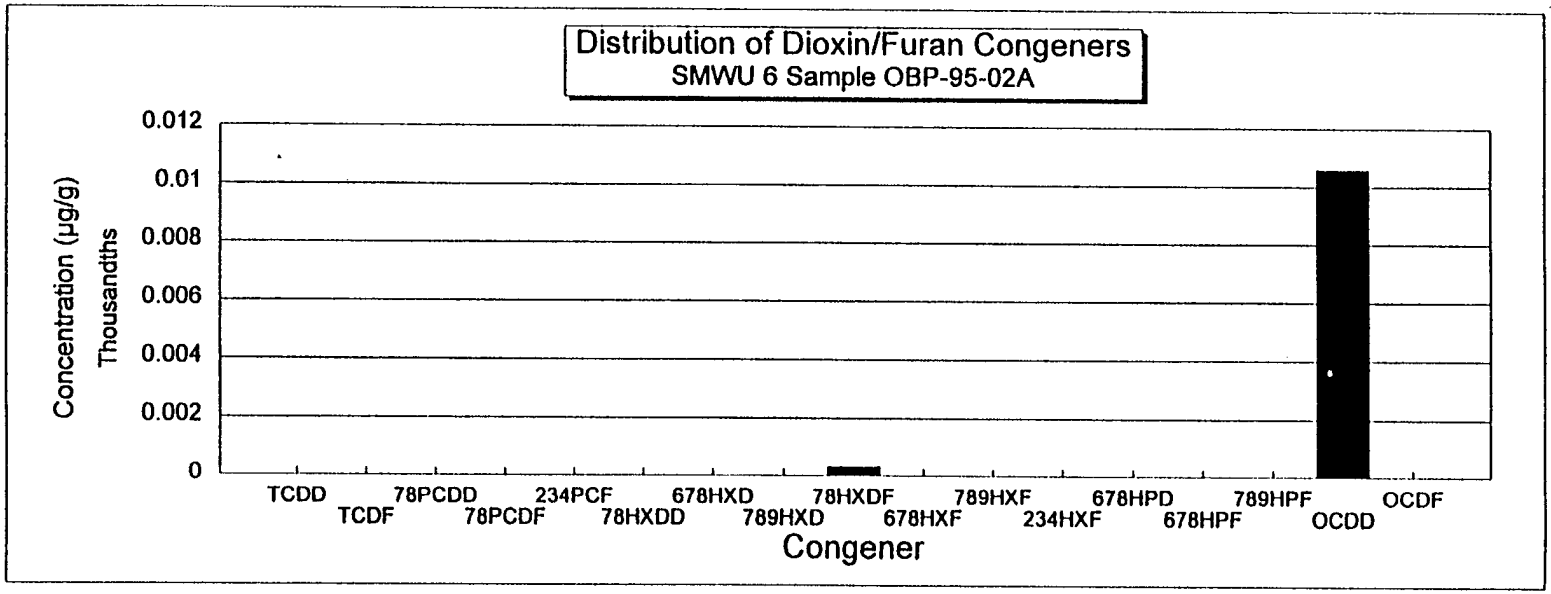

Distribution of Dioxin/Furan Congeners
Background Sample BKS-95-06
Concentration (µg/g)
Thousandths
0.05
0.04
0.03
0.02
0.01
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

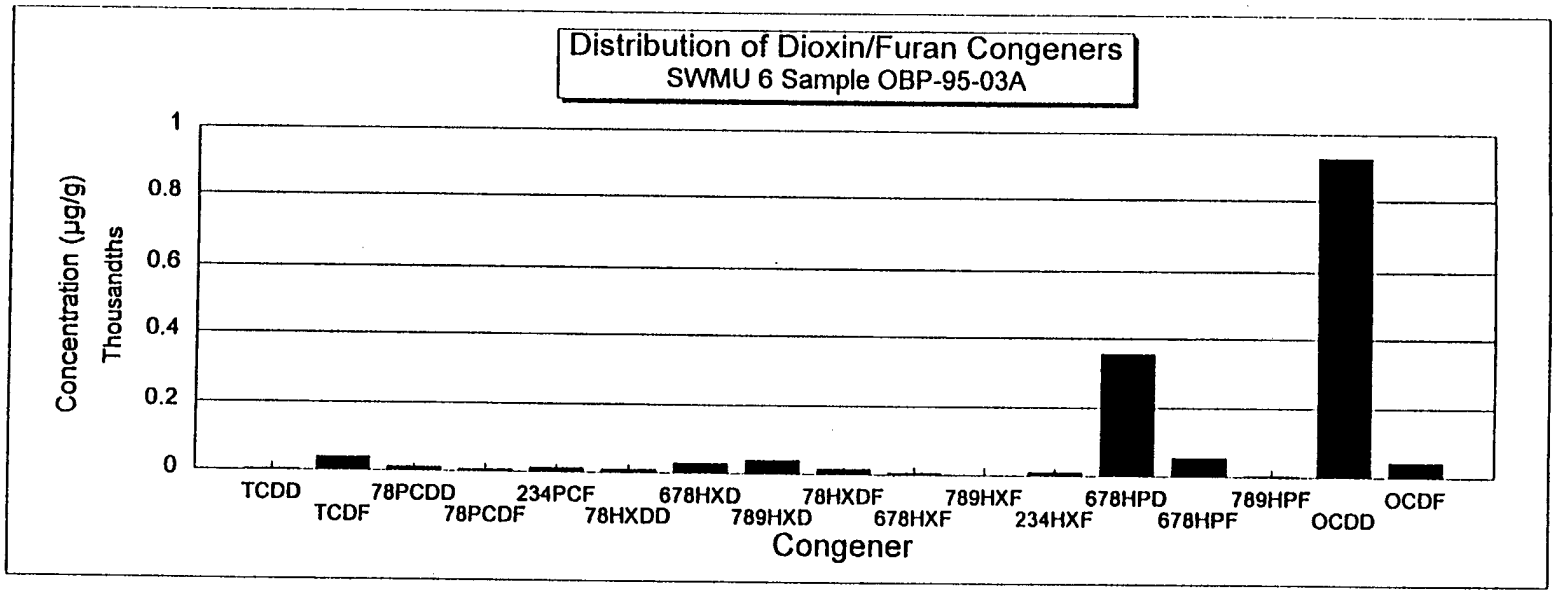

Distribution of Dioxin/Furan Congeners
Background Sample BKS-95-07
Concentration (µg/g)
Thousandths
0.8
0.6
0.4
0.2
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

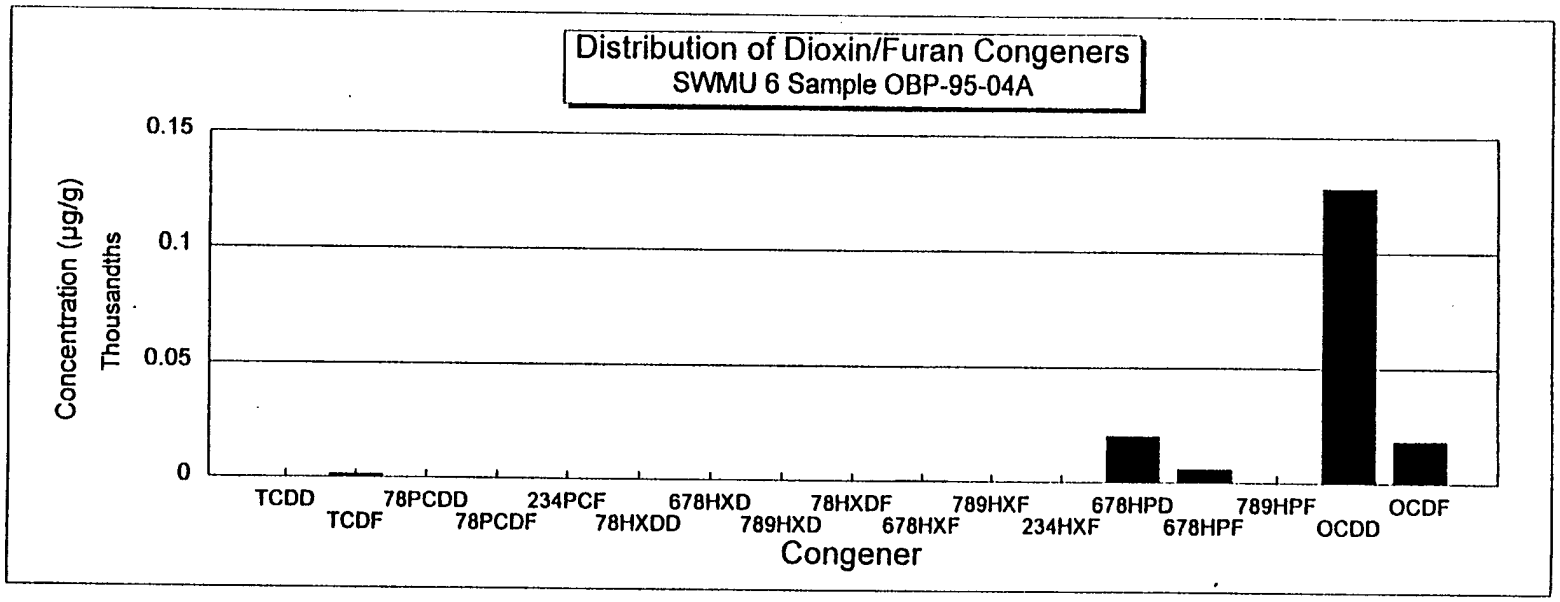

Distribution of Dioxin/Furan Congeners
Background Sample BKS-95-08
Concentration (µg/g)
Thousandths
0.012
0.01
0.008
0.006
0.004
0.002
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

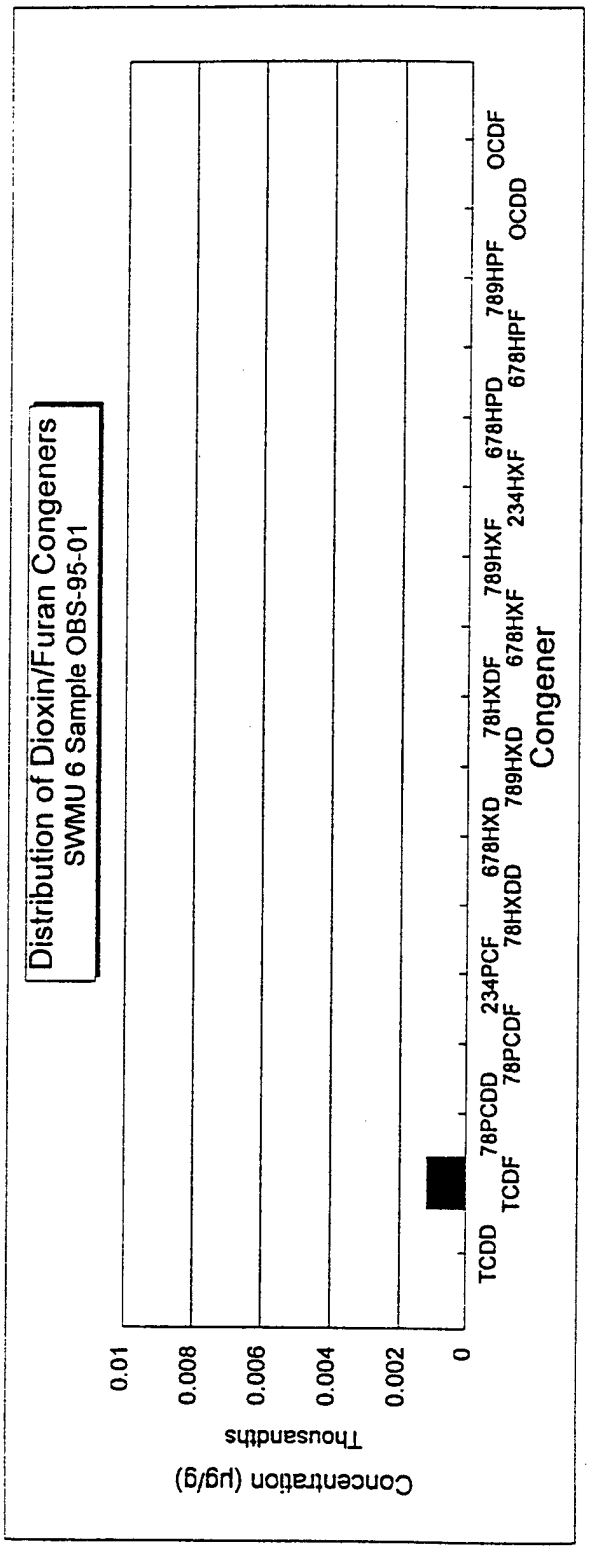

Distribution of Dioxin/Furan Congeners
Background Sample BKS-95-09
Concentration (µg/g)
Thousandths
0.05
0.04
0.03
0.02
0.01
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

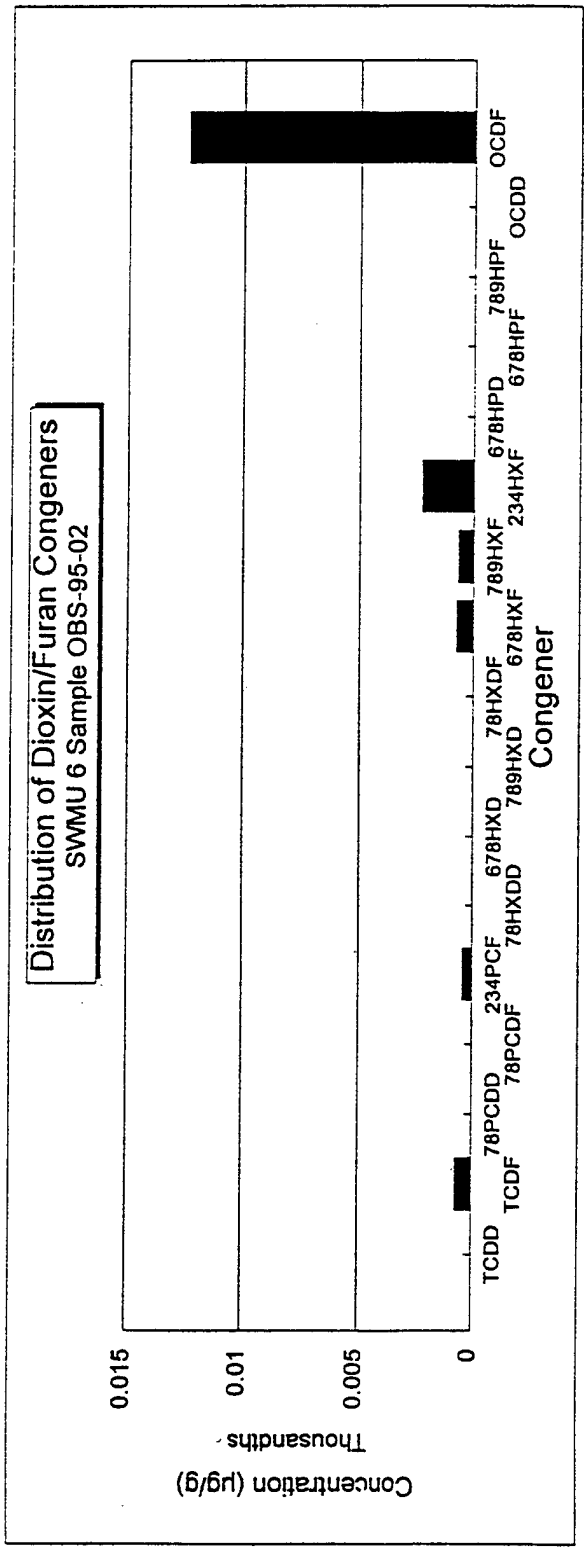

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBP-95-01A
Concentration (µg/g)
Thousandths
0.14
0.12
0.1
0.08
0.06
0.04
0.02
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

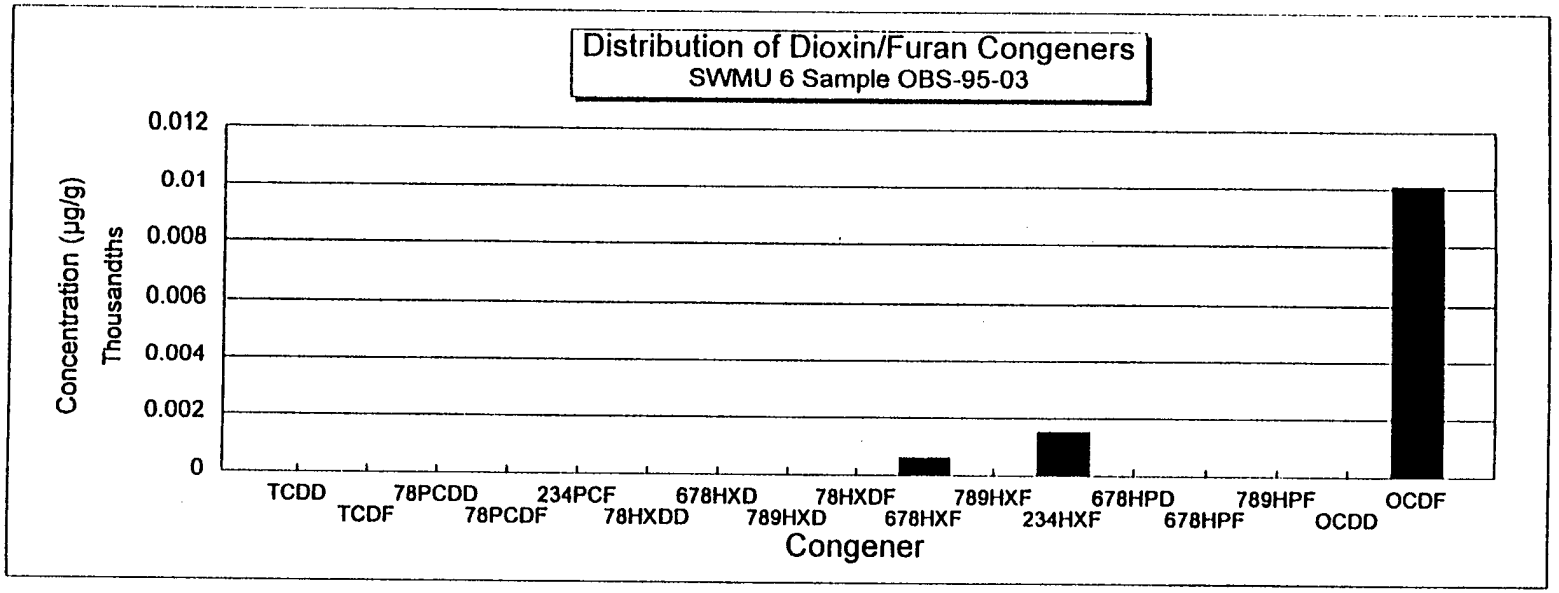

Distribution of Dioxin/Furan Congeners
SMWU 6 Sample OBP-95-02A
Concentration (µg/g)
Thousandths
0.012
0.01
0.008
0.006
0.004
0.002
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

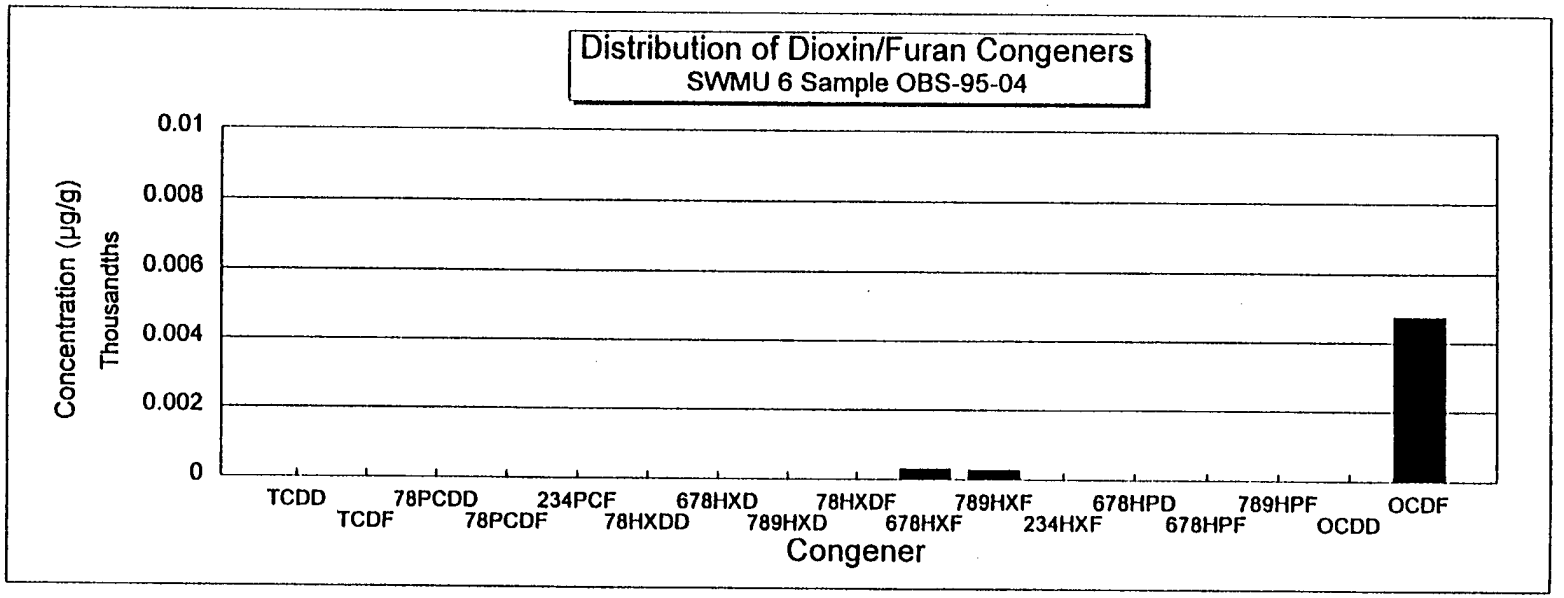

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBP-95-03A
Concentration (µg/g)
Thousandths
1
0.8
0.6
0.4
0.2
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

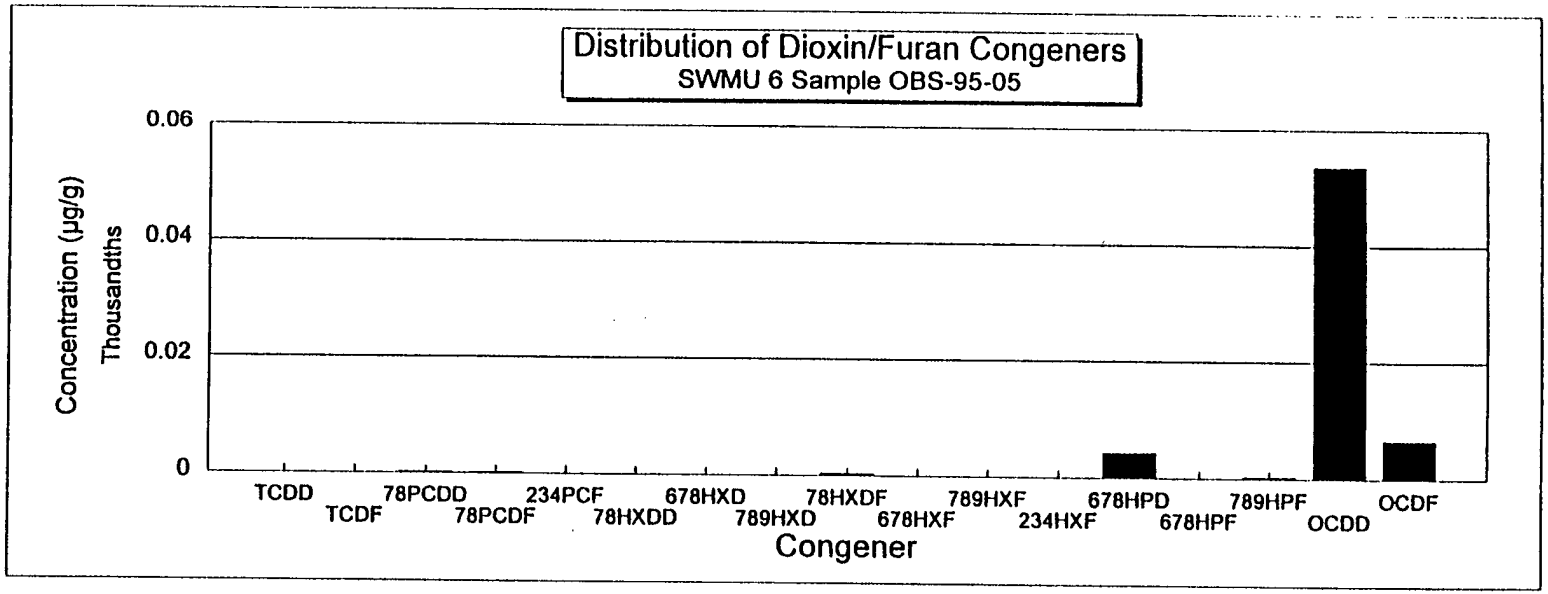

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBP-95-04A
Concentration (µg/g)
Thousandths
0.15
0.1
0.05
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

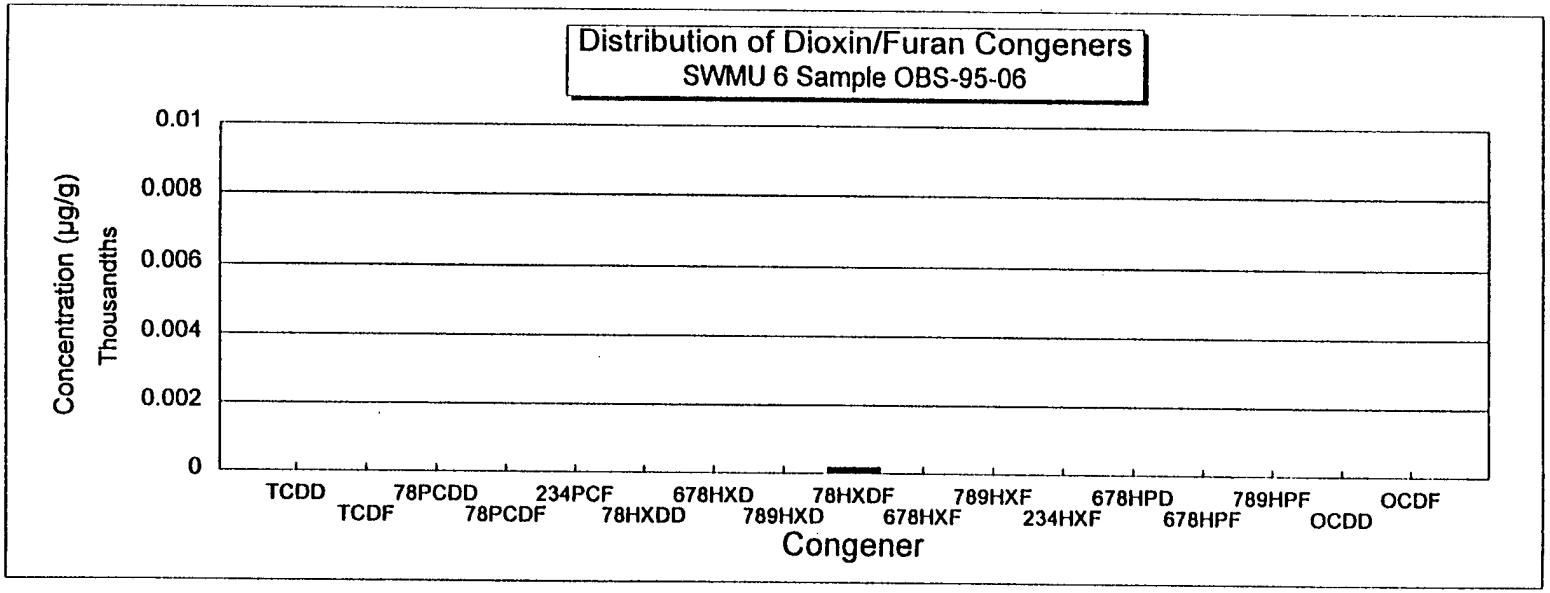

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-01
Concentration (µg/g)
Thousandths
0.01
0.008
0.006
0.004
0.002
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

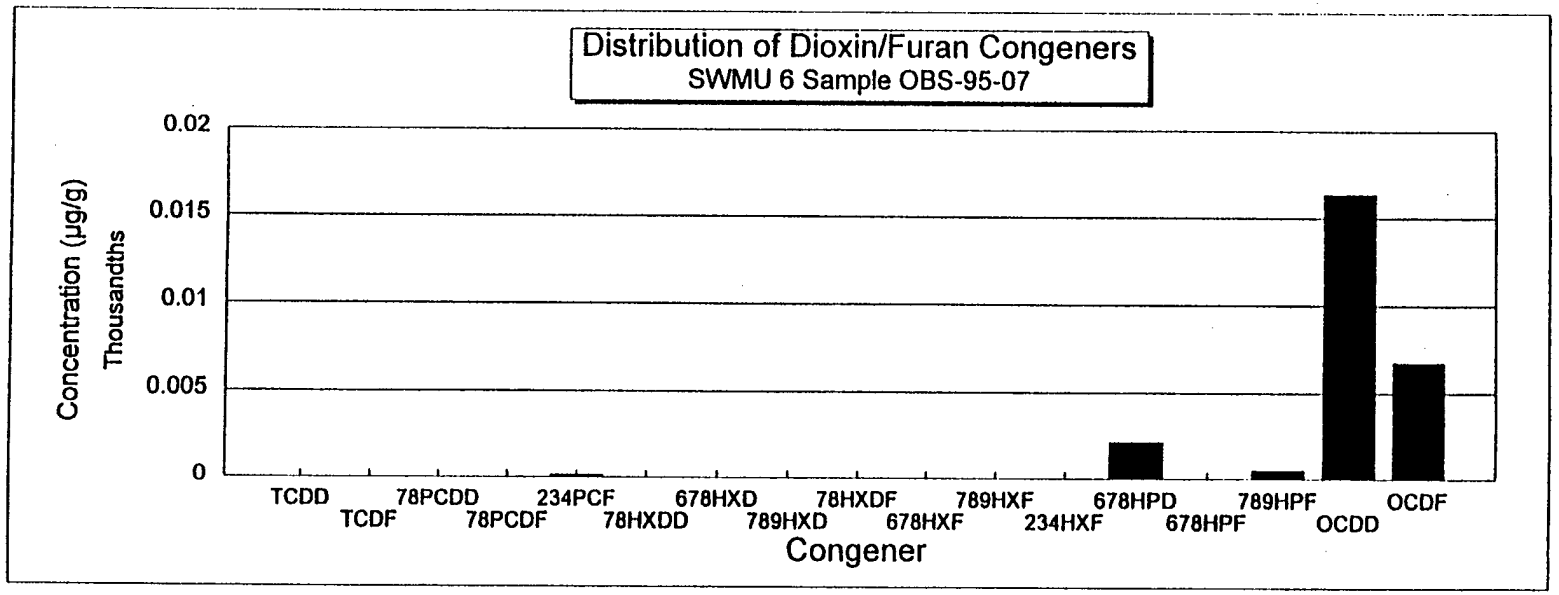

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-02
Concentration (µg/g)
Thousandths
0.015
0.01
0.005
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

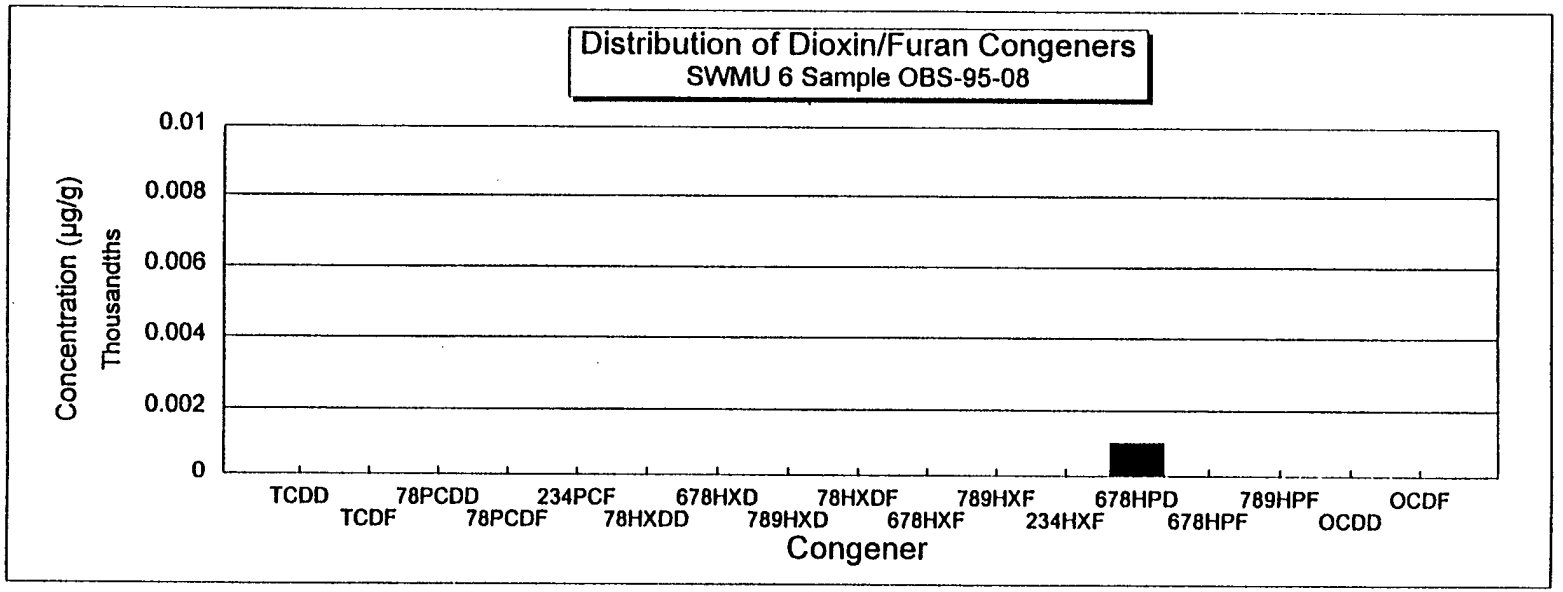

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-03
Concentration (µg/g)
Thousandths
0.012
0.01
0.008
0.006
0.004
0.002
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

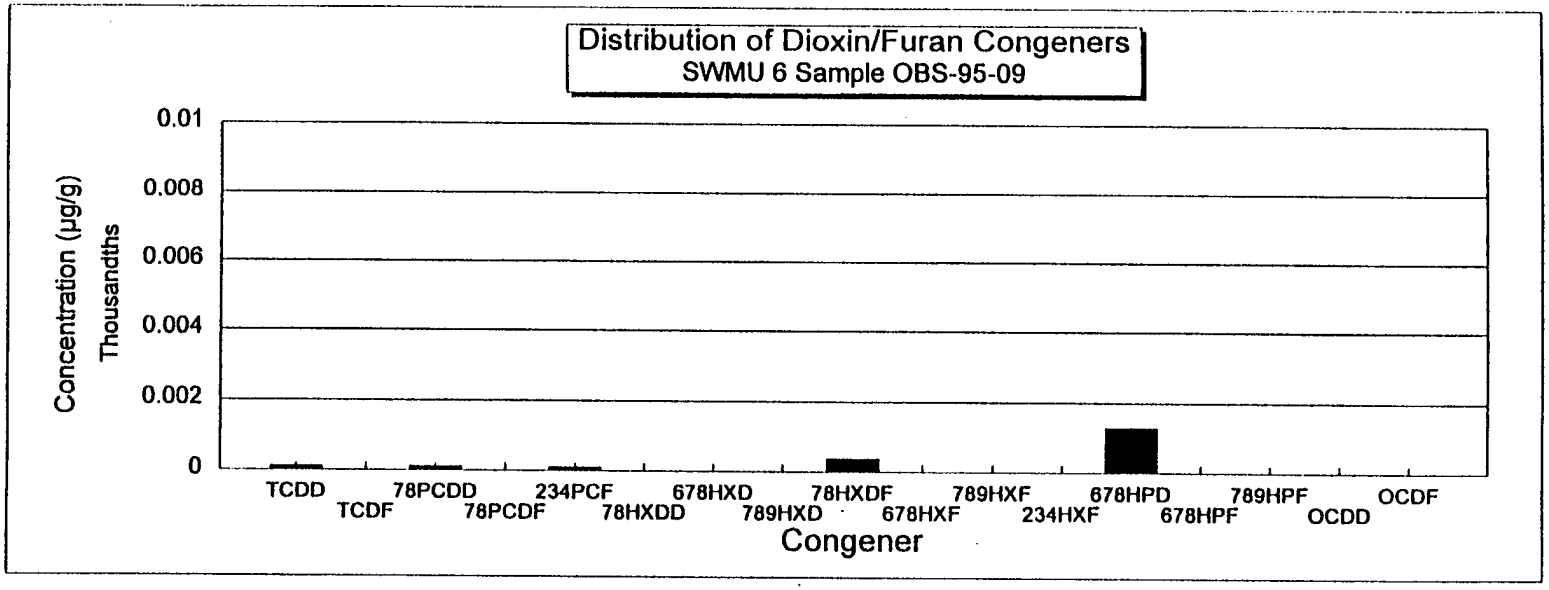

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-04
Concentration (µg/g)
Thousandths
0.01
0.008
0.006
0.004
0.002
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

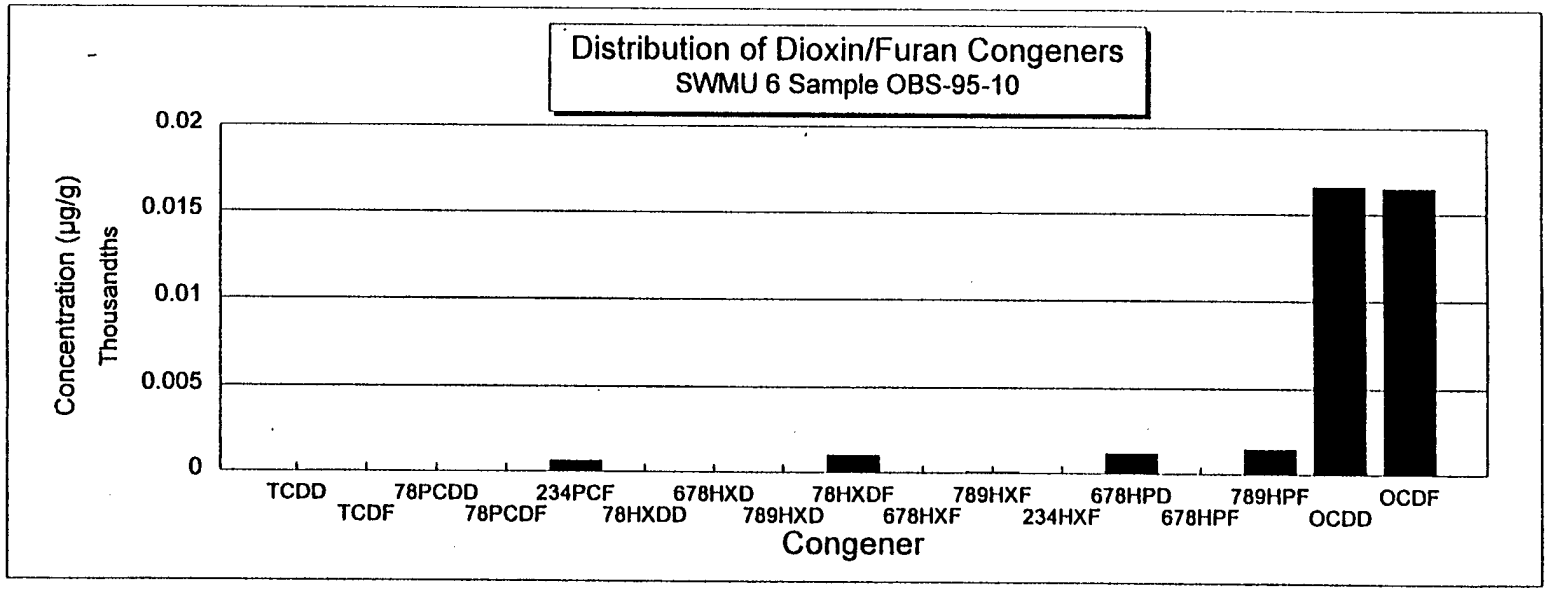

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-05
Concentration (µg/g)
Thousandths
0.06
0.04
0.02
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

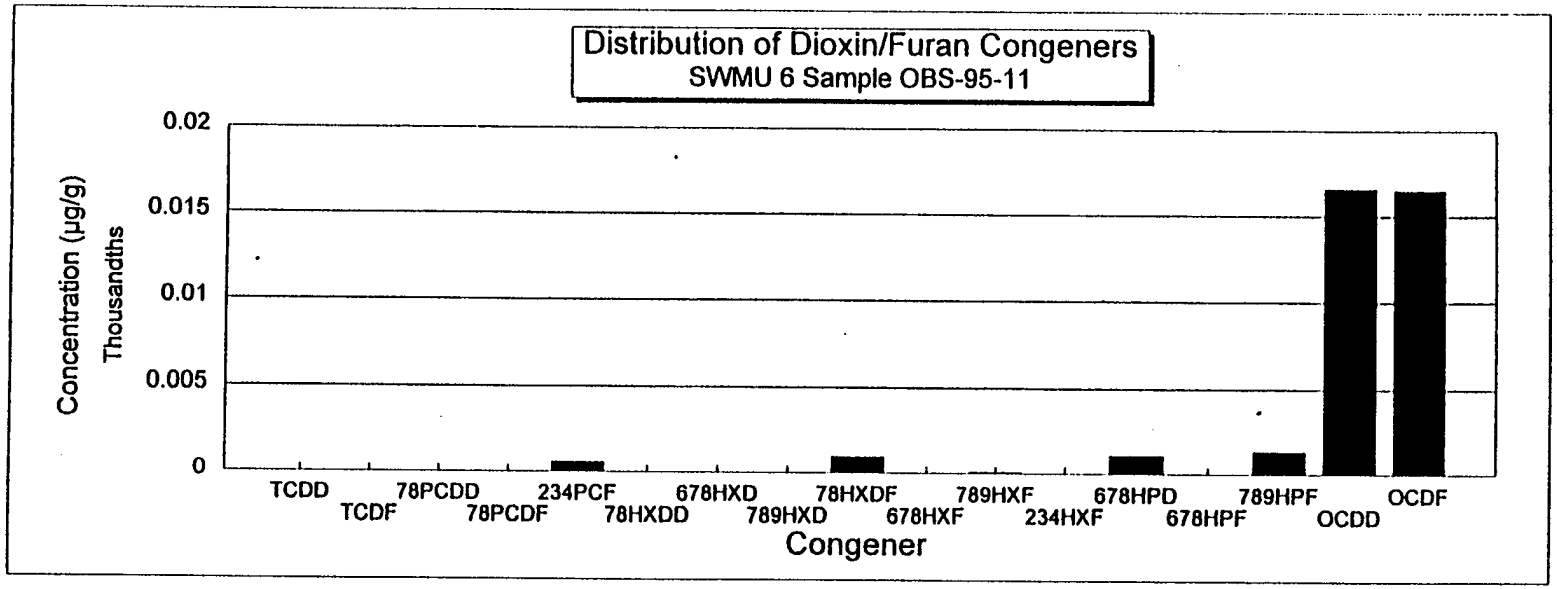

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-06
Concentration (µg/g)
Thousandths
0.01
0.008
0.006
0.004
0.002
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

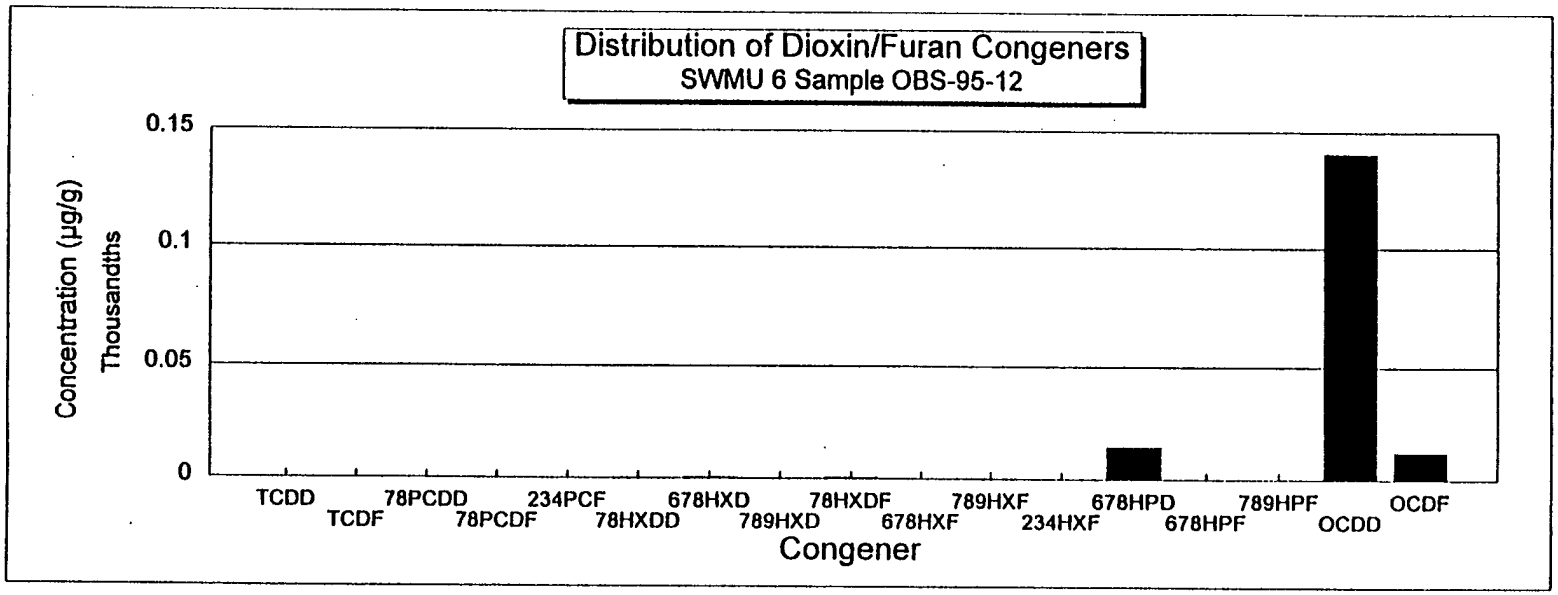

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-07
Concentration (µg/g)
Thousandths
0.02
0.015
0.01
0.005
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

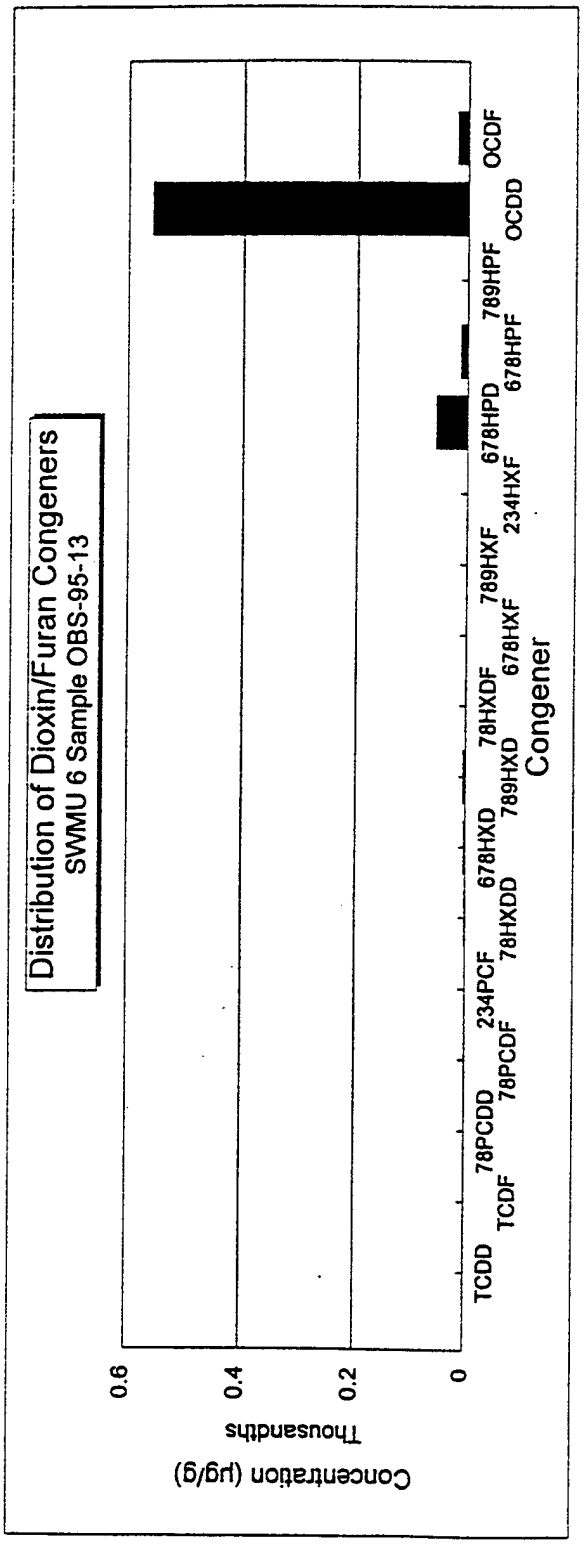

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-08
Concentration (µg/g)
Thousandths
0.01
0.008
0.006
0.004
0.002
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

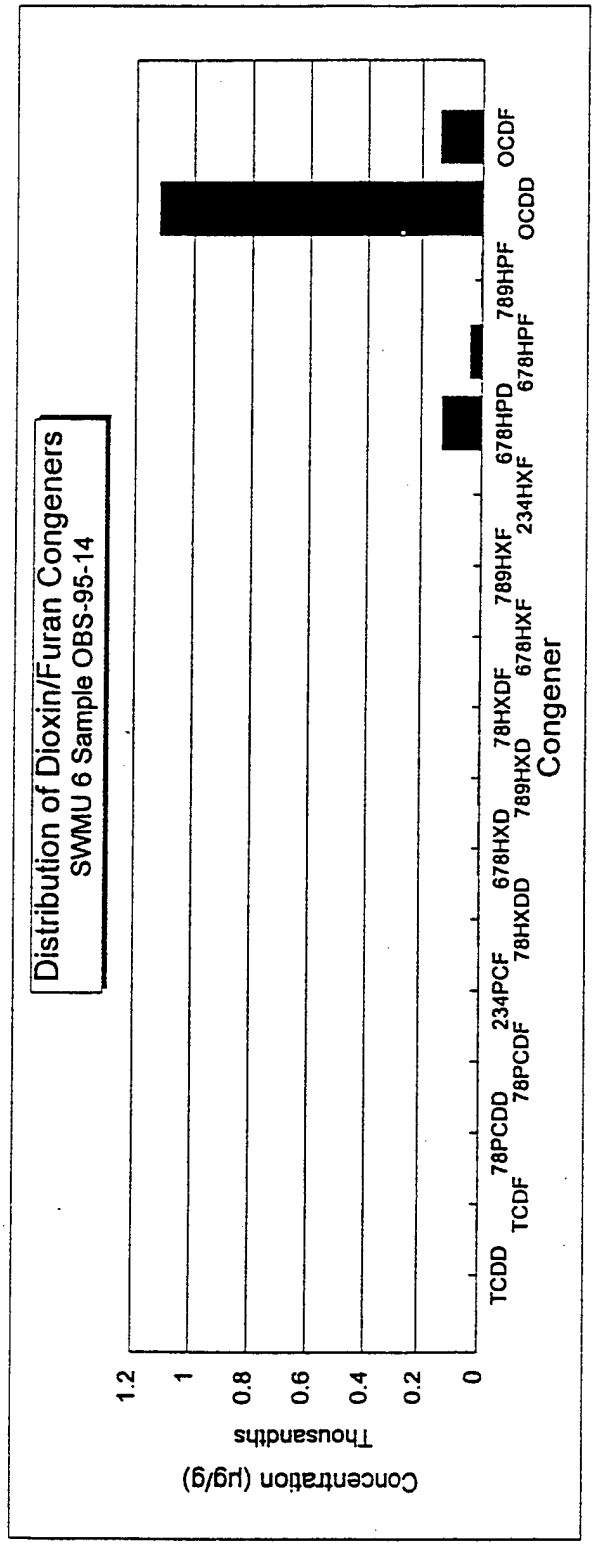

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-09
Concentration (µg/g)
Thousandths
0.01
0.008
0.006
0.004
0.002
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

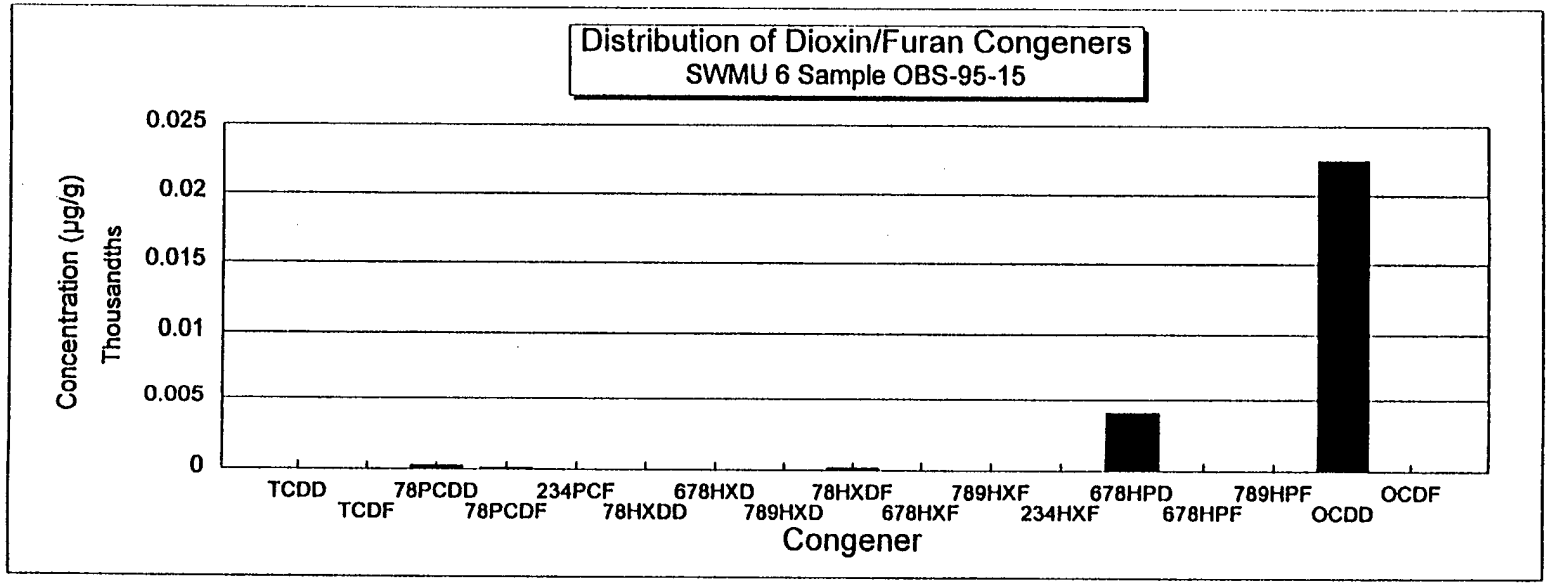

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-10
Concentration (µg/g)
Thousandths
0.02
0.015
0.01
0.005
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

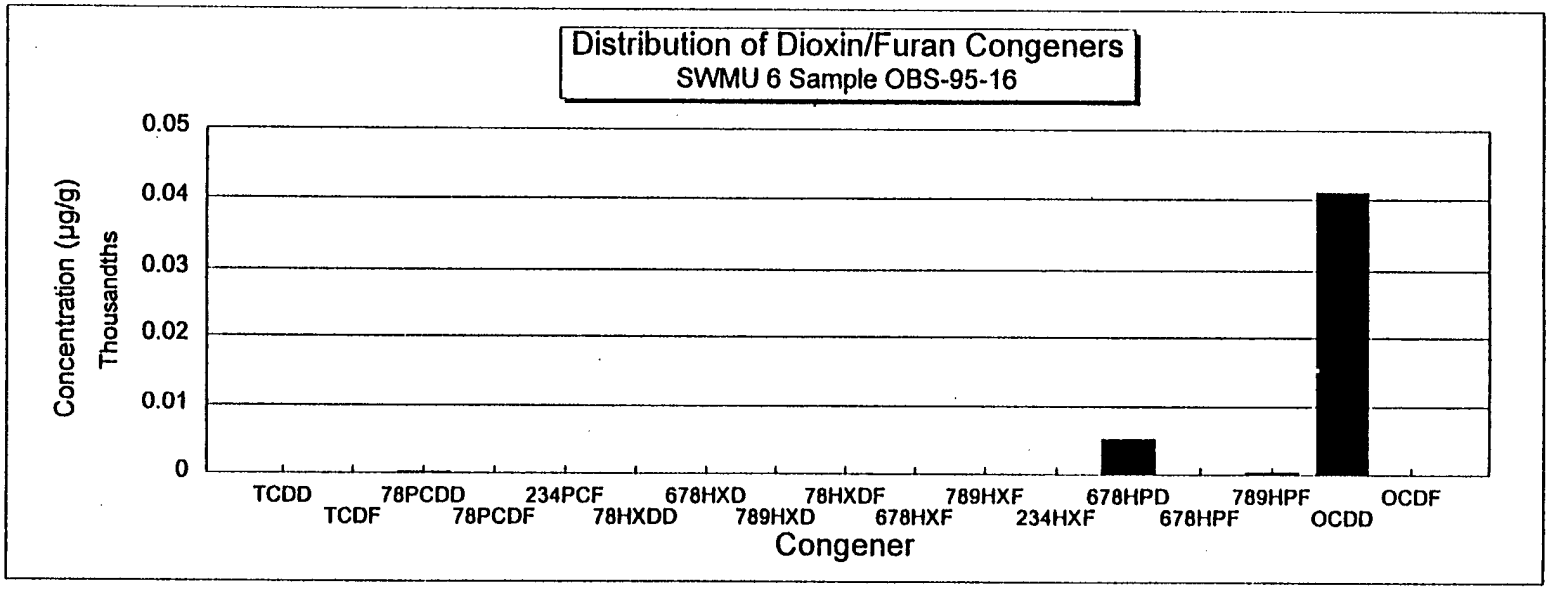

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-11
Concentration (µg/g)
Thousandths
0.02
0.015
0.01
0.005
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

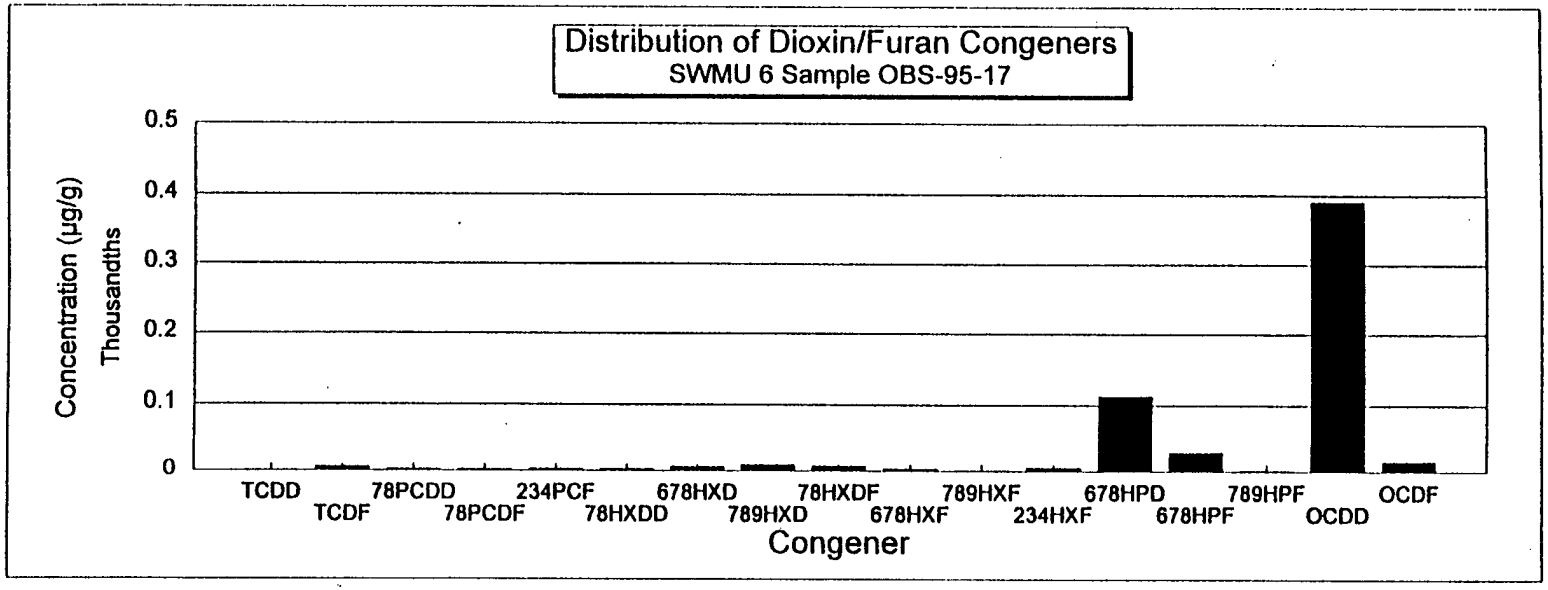

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-12
Concentration (µg/g)
Thousandths
0.15
0.1
0.05
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

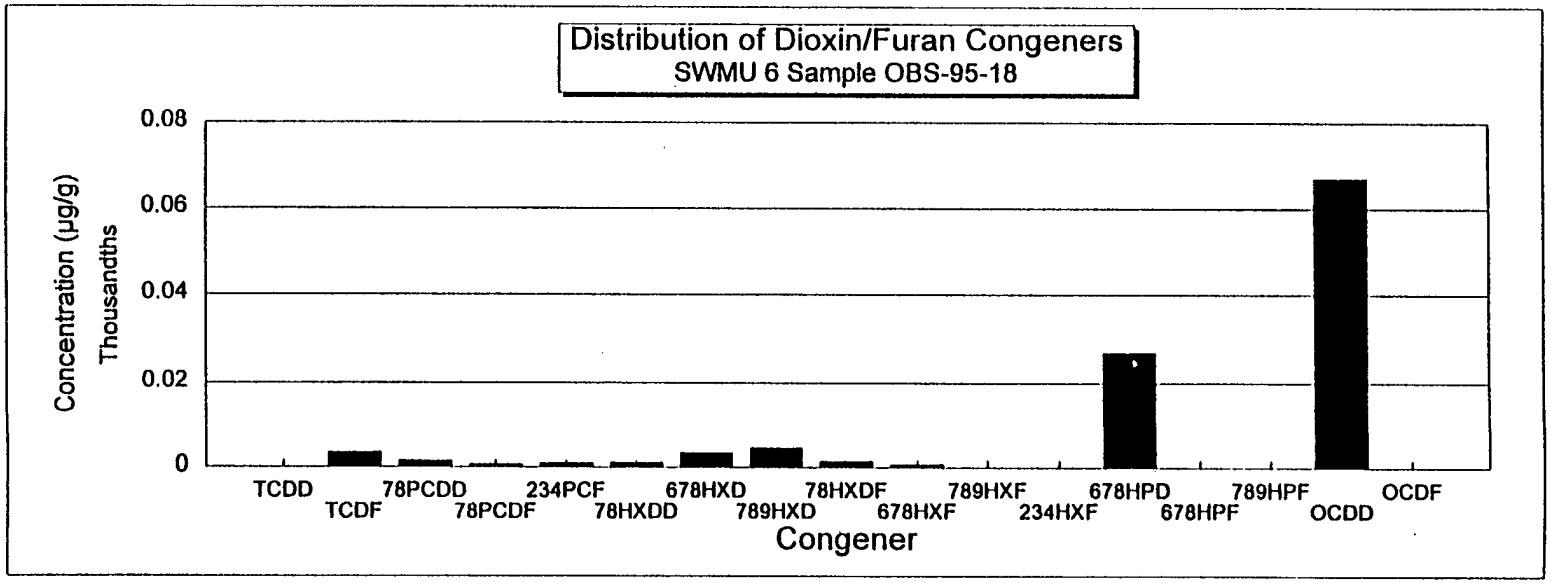

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-13
Concentration (µg/g)
Thousandths
0.6
0.4
0.2
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

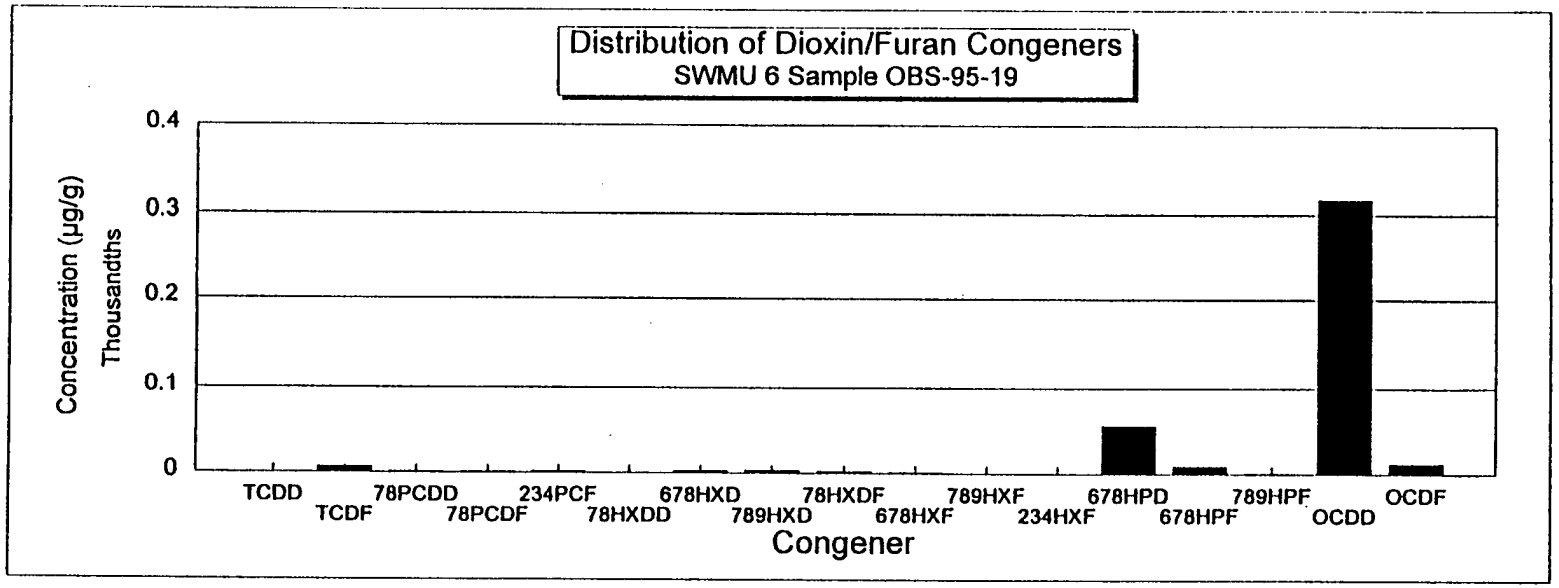

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-14
Concentration (µg/g)
Thousandths
1.2
1
0.8
0.6
0.4
0.2
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

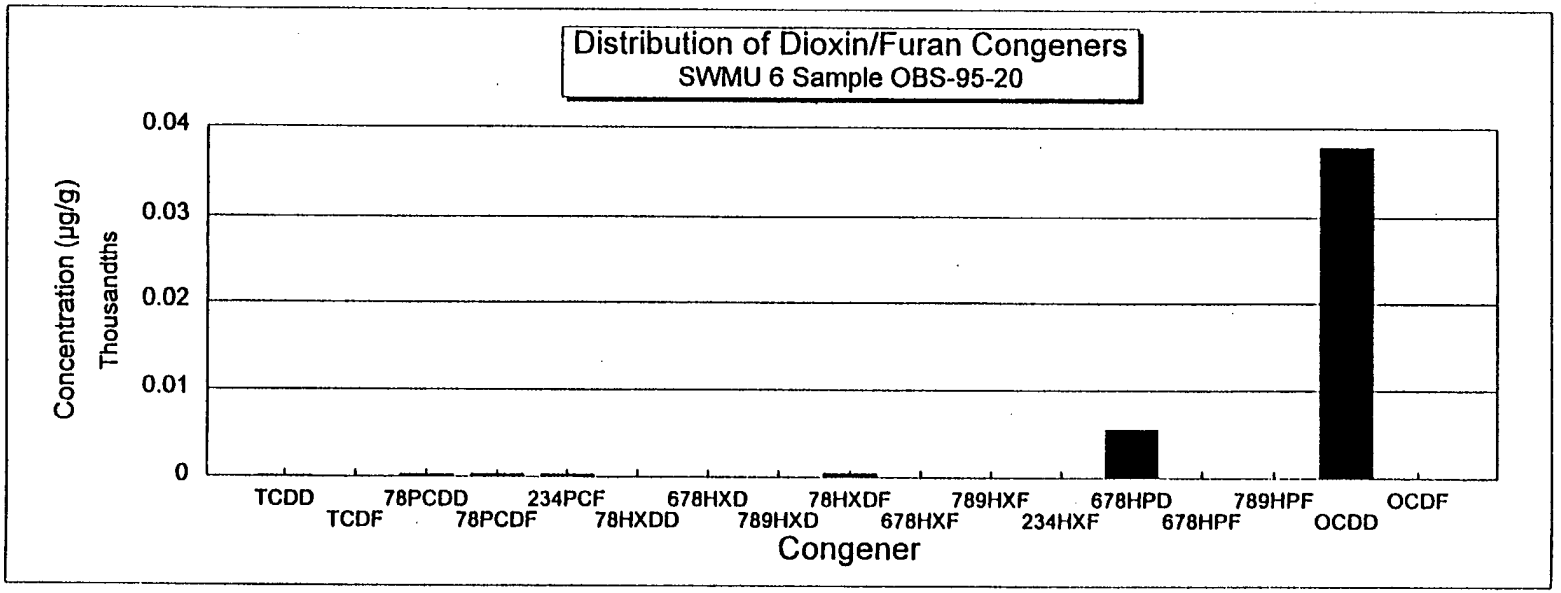

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-15
Concentration (µg/g)
Thousandths
0.025
0.02
0.015
0.01
0.005
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

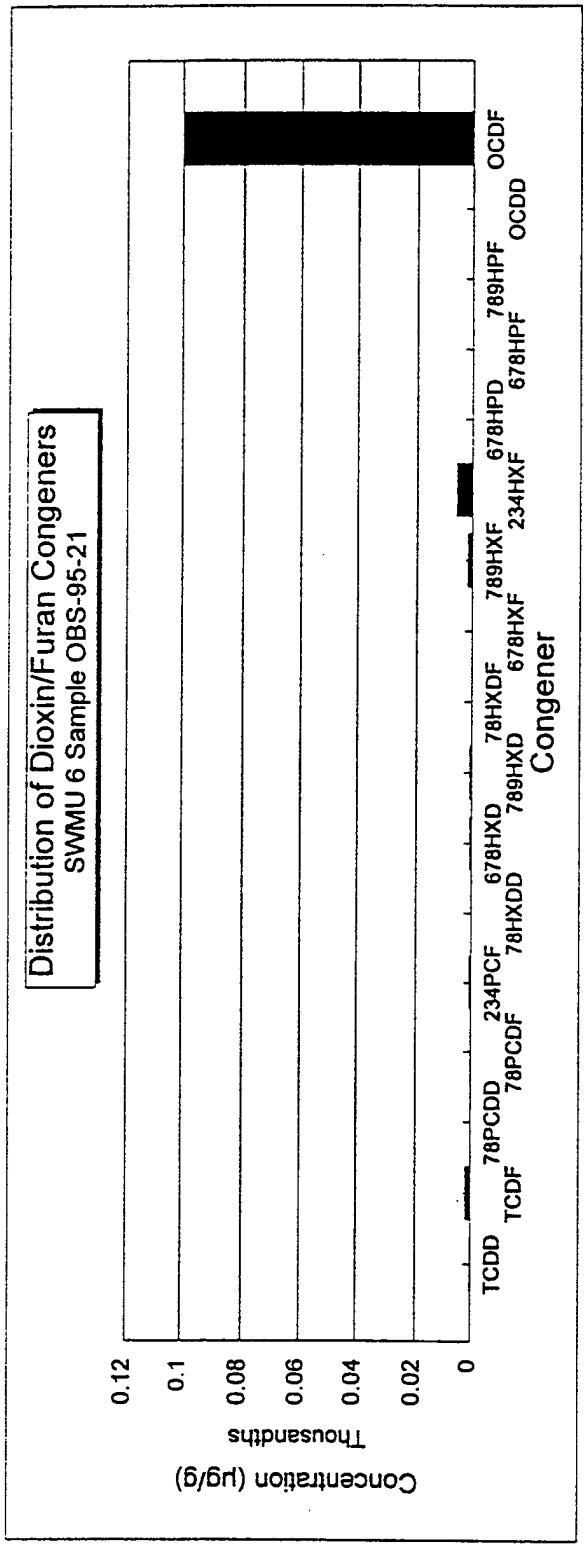

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-16
Concentration (µg/g)
Thousandths
0.05
0.04
0.03
0.02
0.01
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

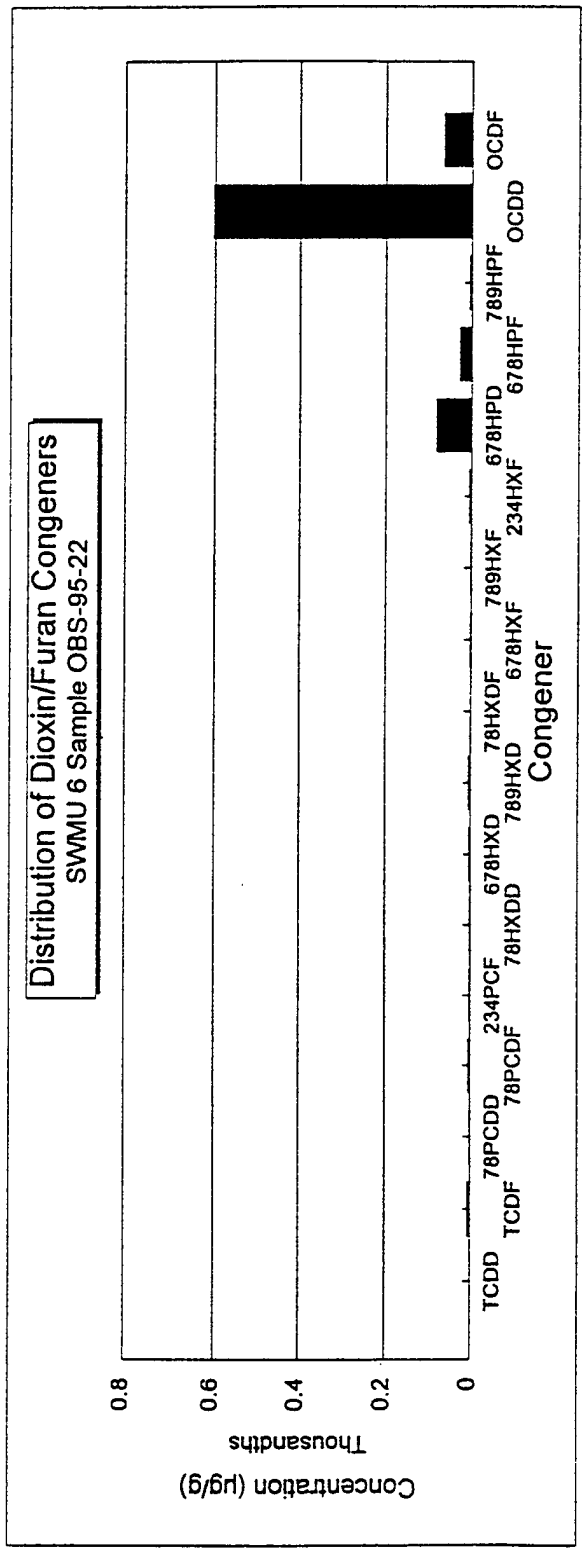

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-17
Concentration (µg/g)
Thousandths
0.5
0.4
0.3
0.2
0.1
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

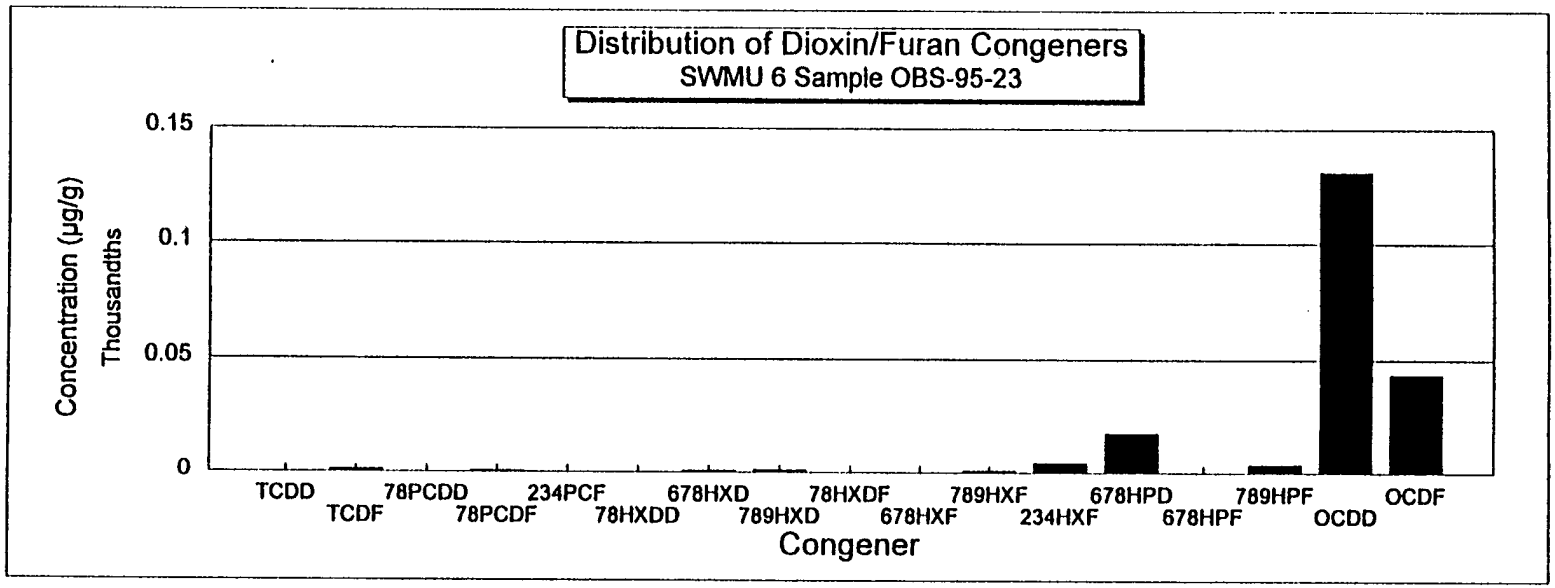

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-18
Concentration (µg/g)
Thousandths
0.08
0.06
0.04
0.02
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

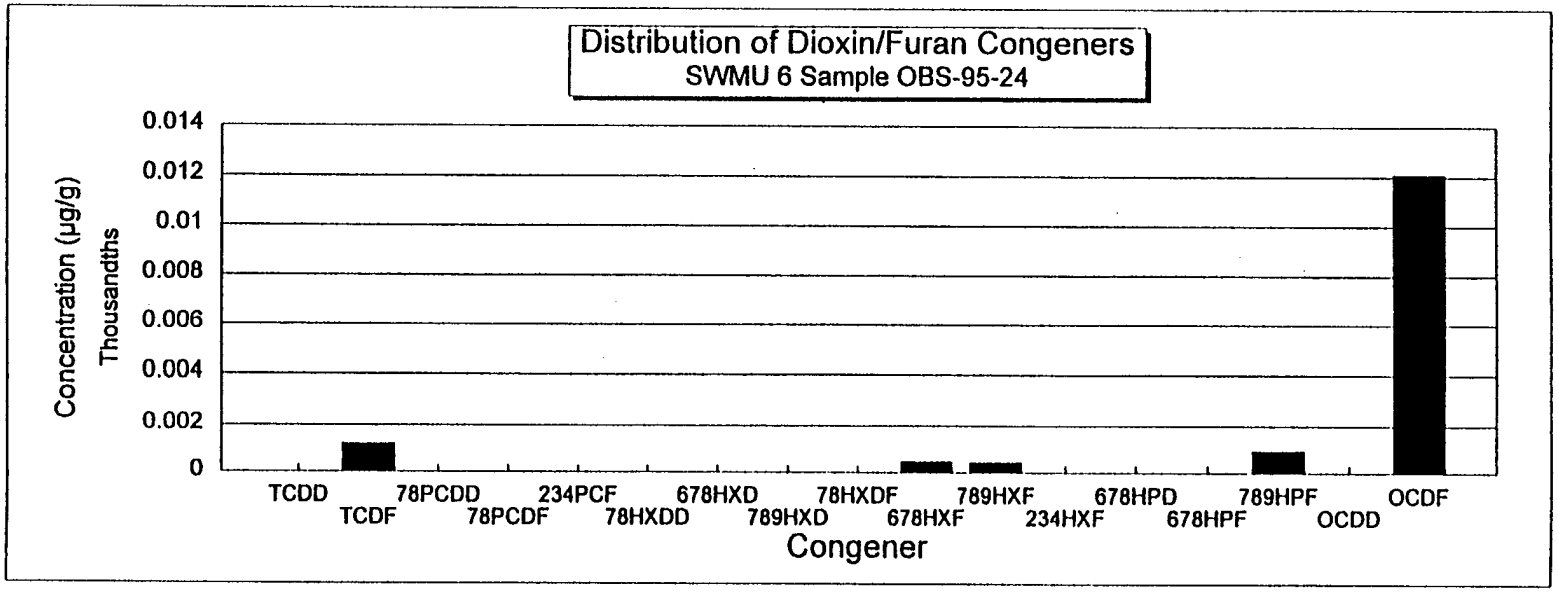

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-19
Concentration (µg/g)
Thousandths
0.4
0.3
0.2
0.1
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

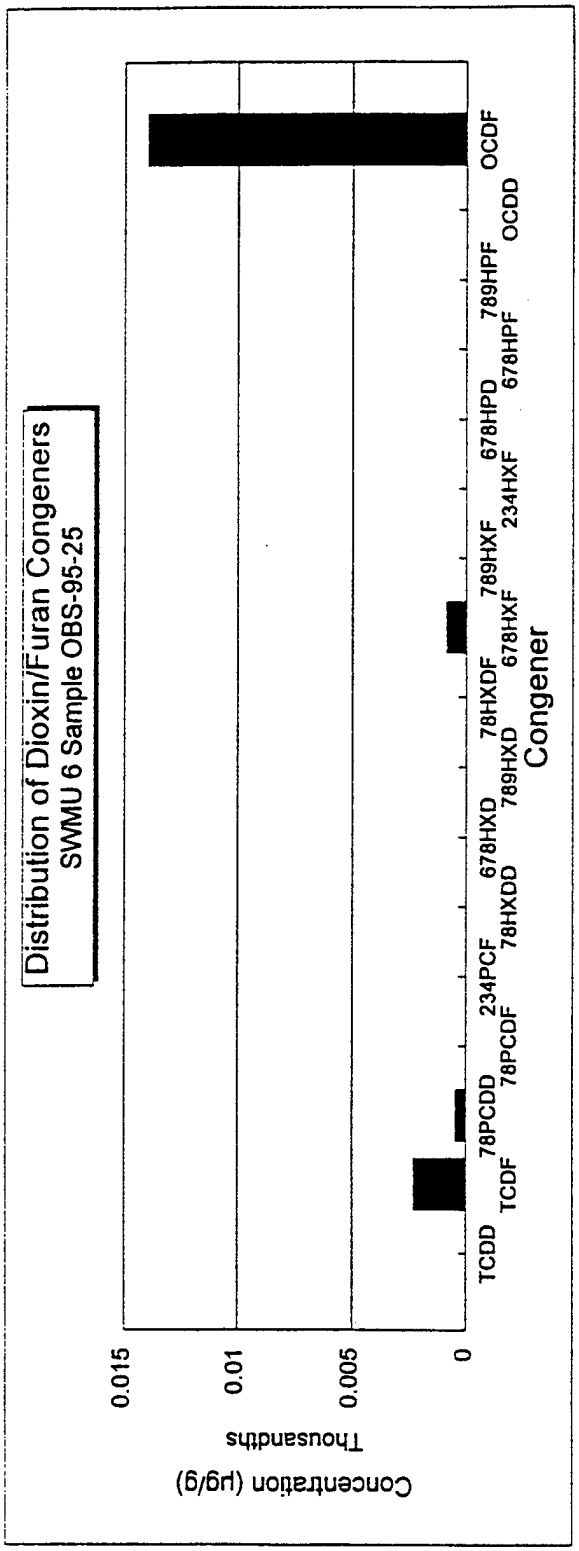

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-20
Concentration (µg/g)
Thousandths
0.04
0.03
0.02
0.01
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

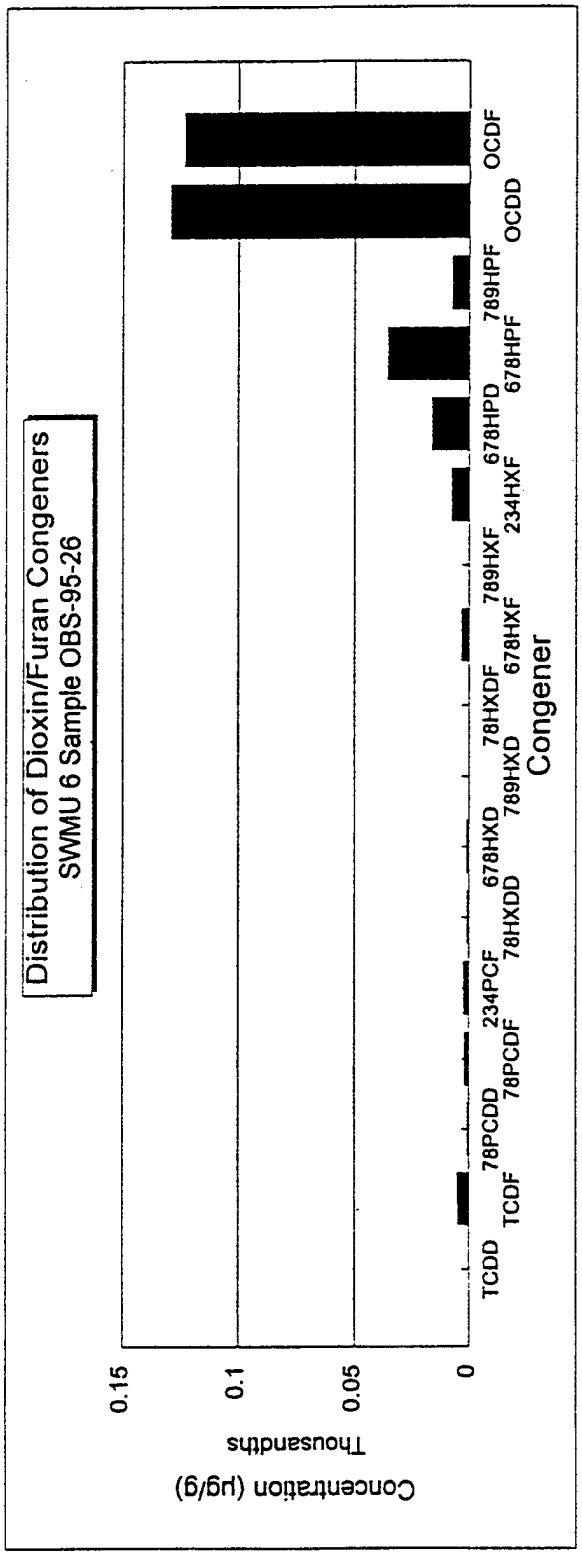

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-21
Concentration (pg/g)
Thousandths
0.12
0.1
0.08
0.06
0.04
0.02
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

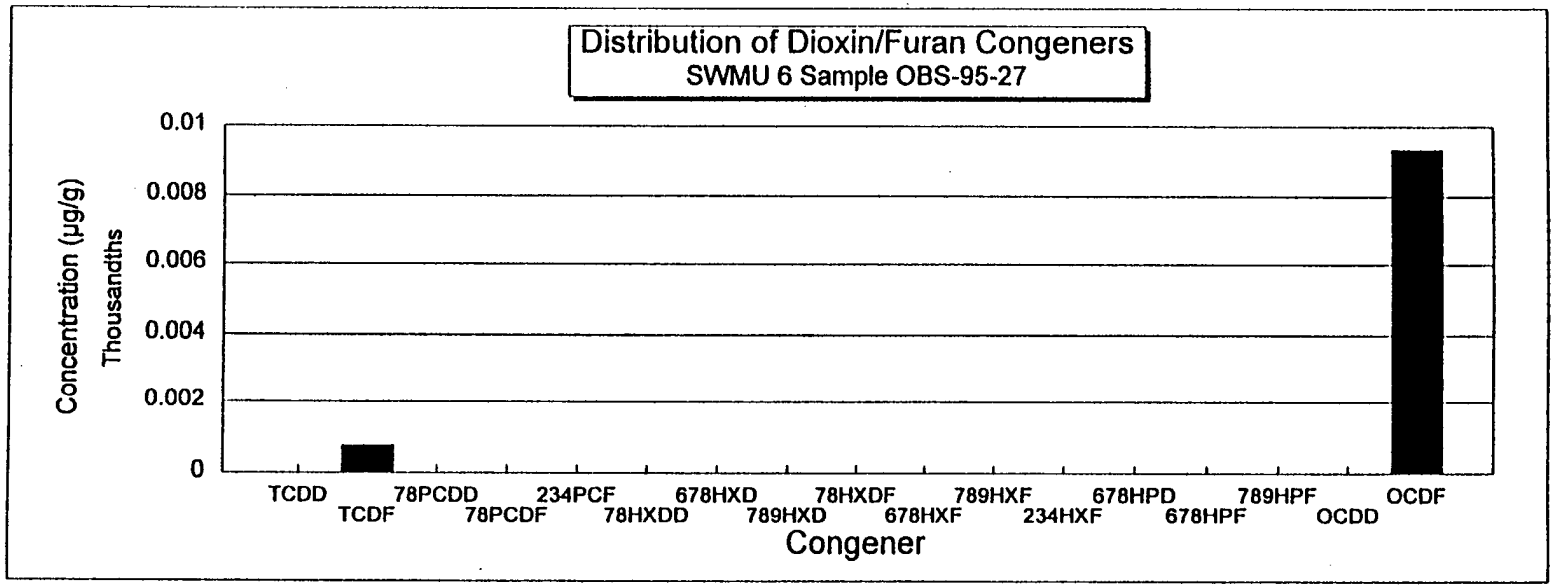

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-22
Concentration (pg/g)
Thousandths
0.8
0.6
0.4
0.2
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

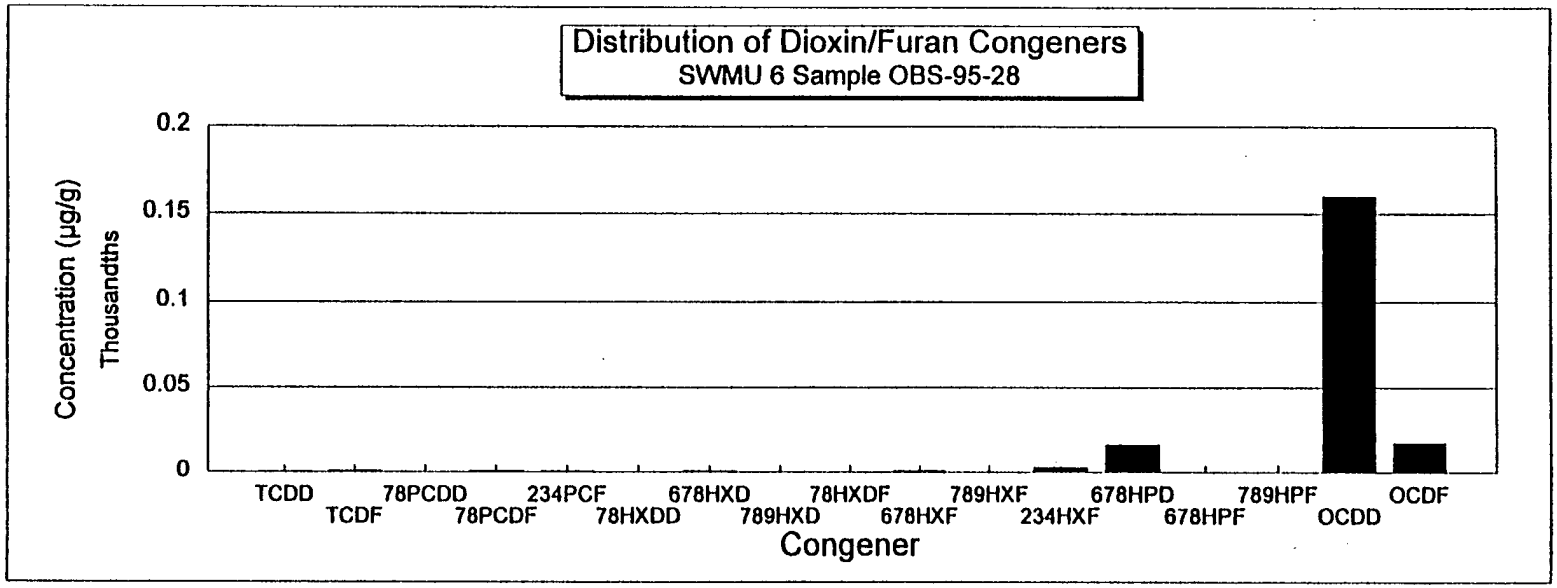

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-23
Concentration (µg/g)
Thousandths
0.15
0.1
0.05
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

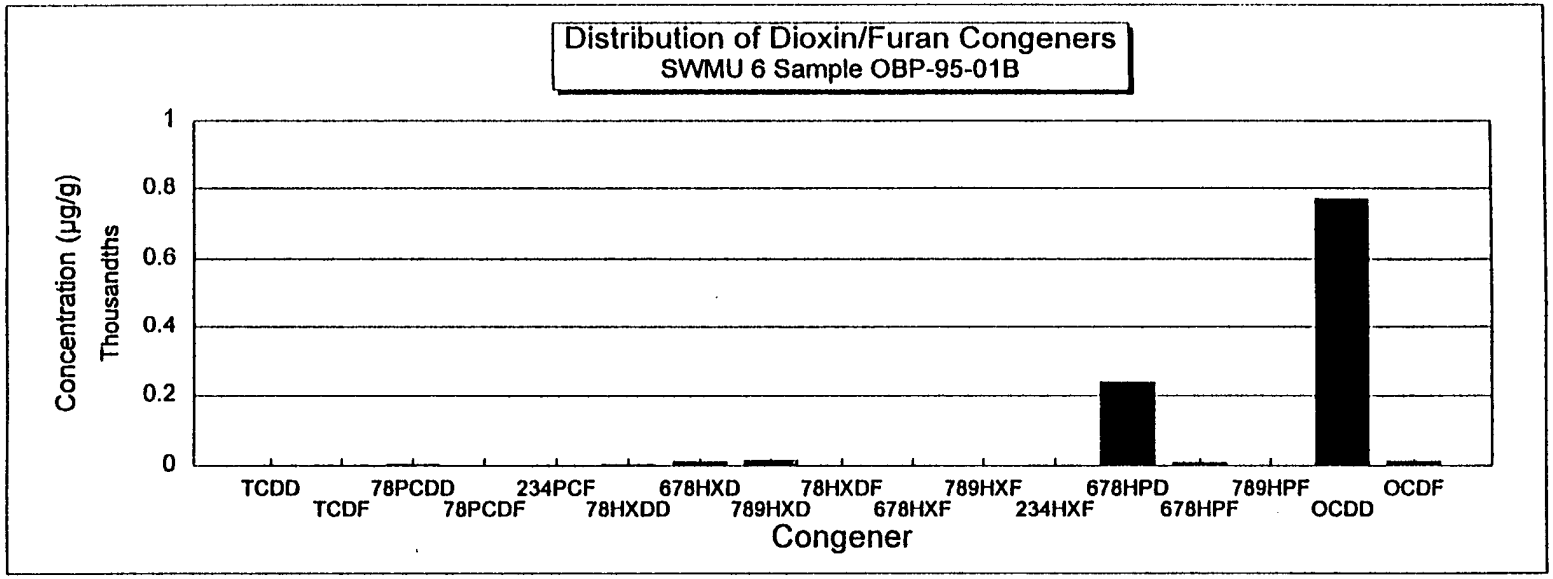

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-24
Concentration (µg/g)
Thousandths
0.014
0.012
0.01
0.008
0.006
0.004
0.002
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

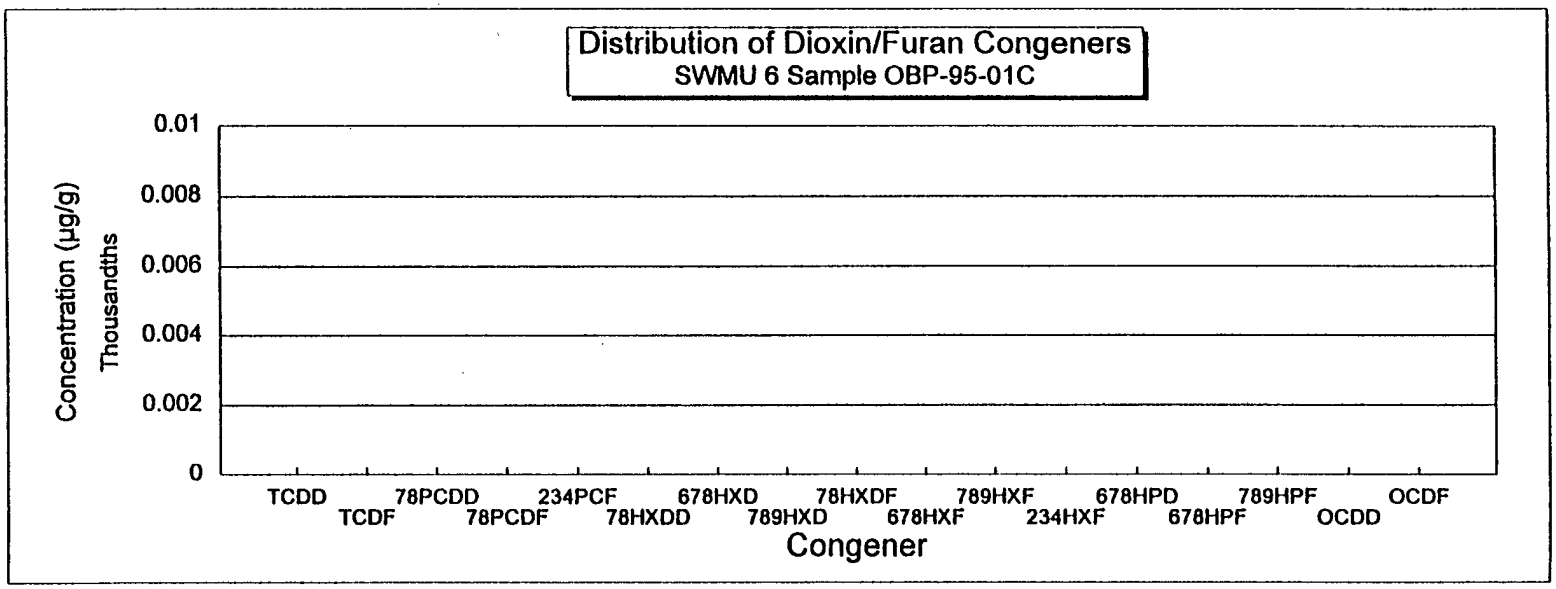

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-25
Concentration (µg/g)
Thousandths
0.015
0.01
0.005
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

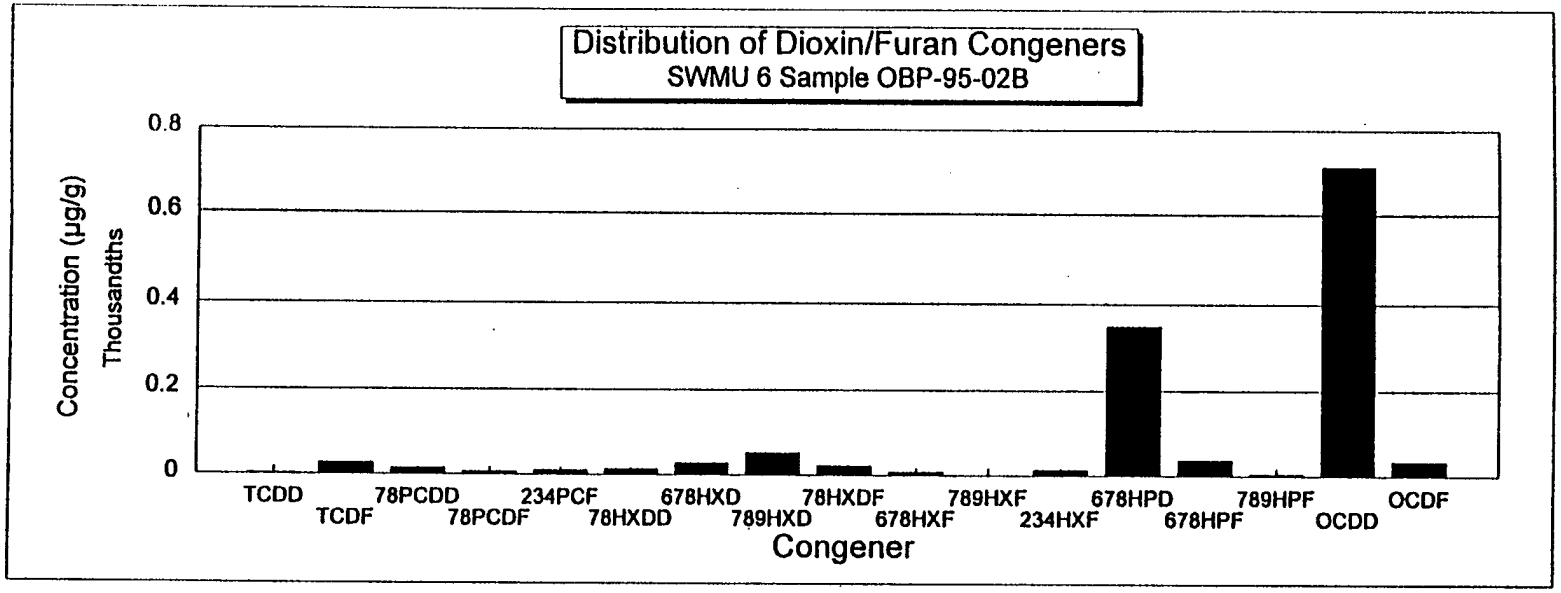

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-26
Concentration (µg/g)
Thousandths
0.15
0.1
0.05
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

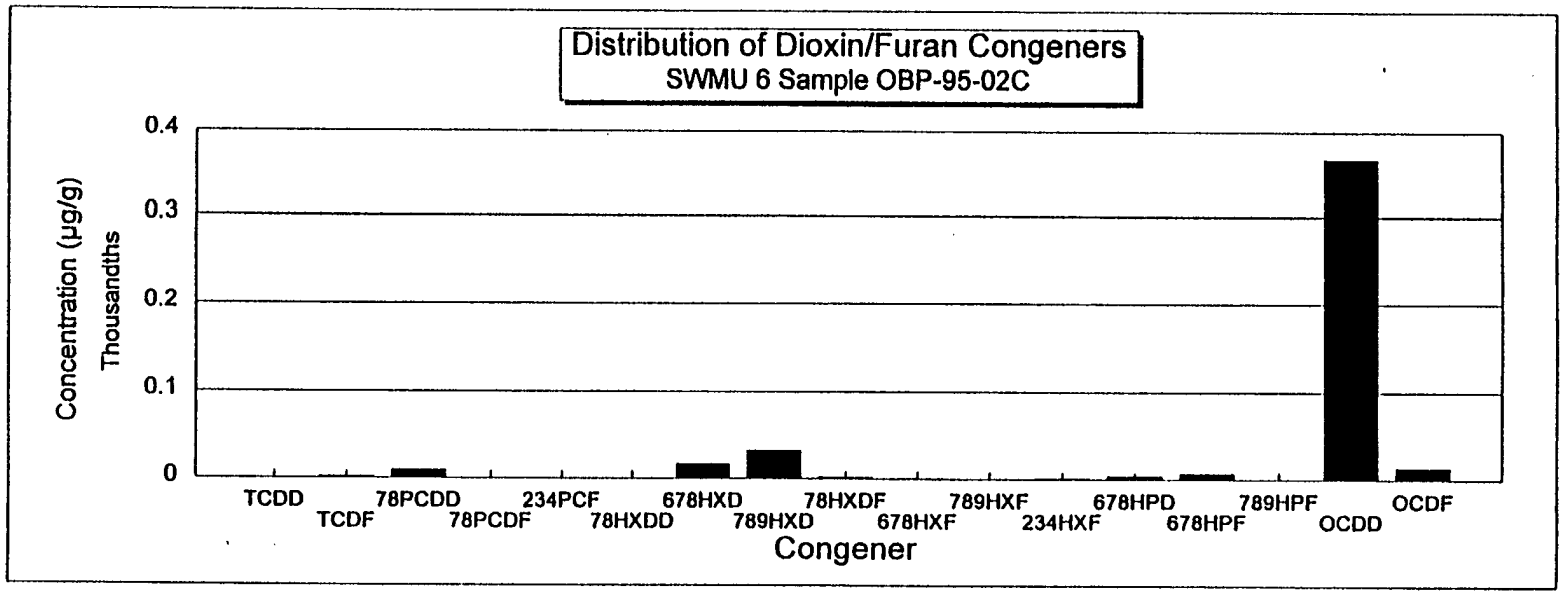

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-27
Concentration (µg/g)
Thousandths
0.01
0.008
0.006
0.004
0.002
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

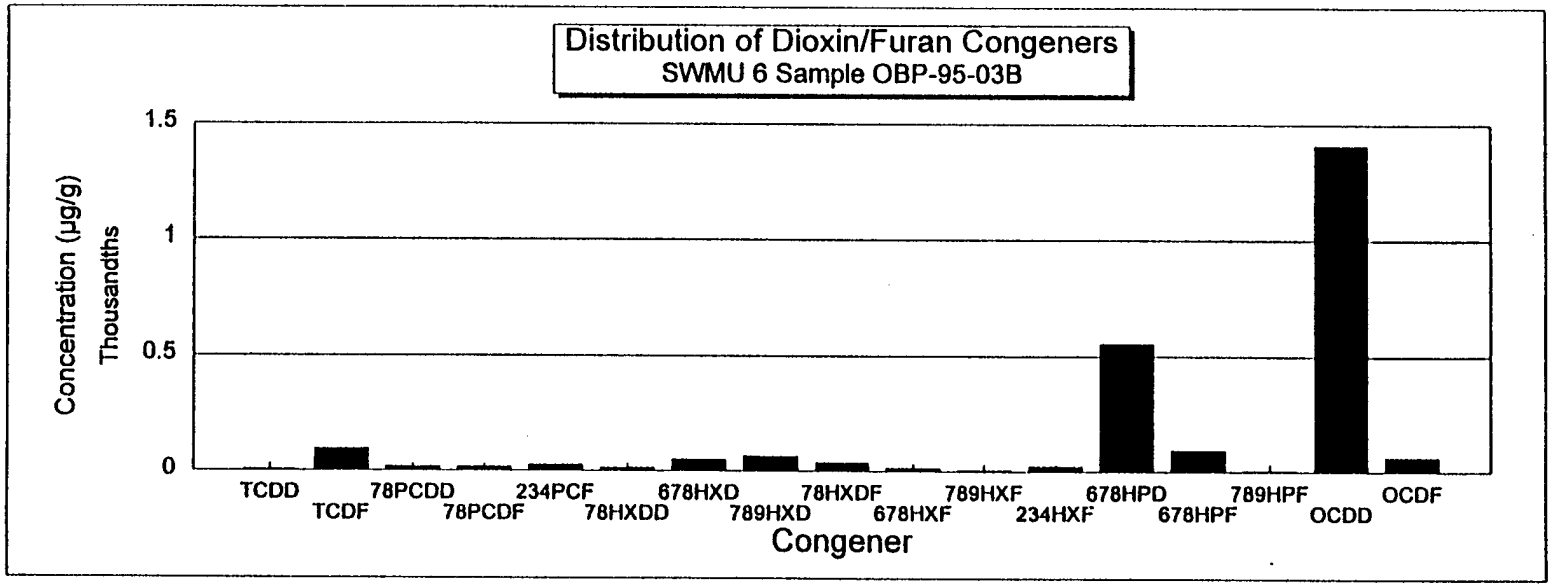

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBS-95-28
Concentration (µg/g)
Thousandths
0.2
0.15
0.1
0.05
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

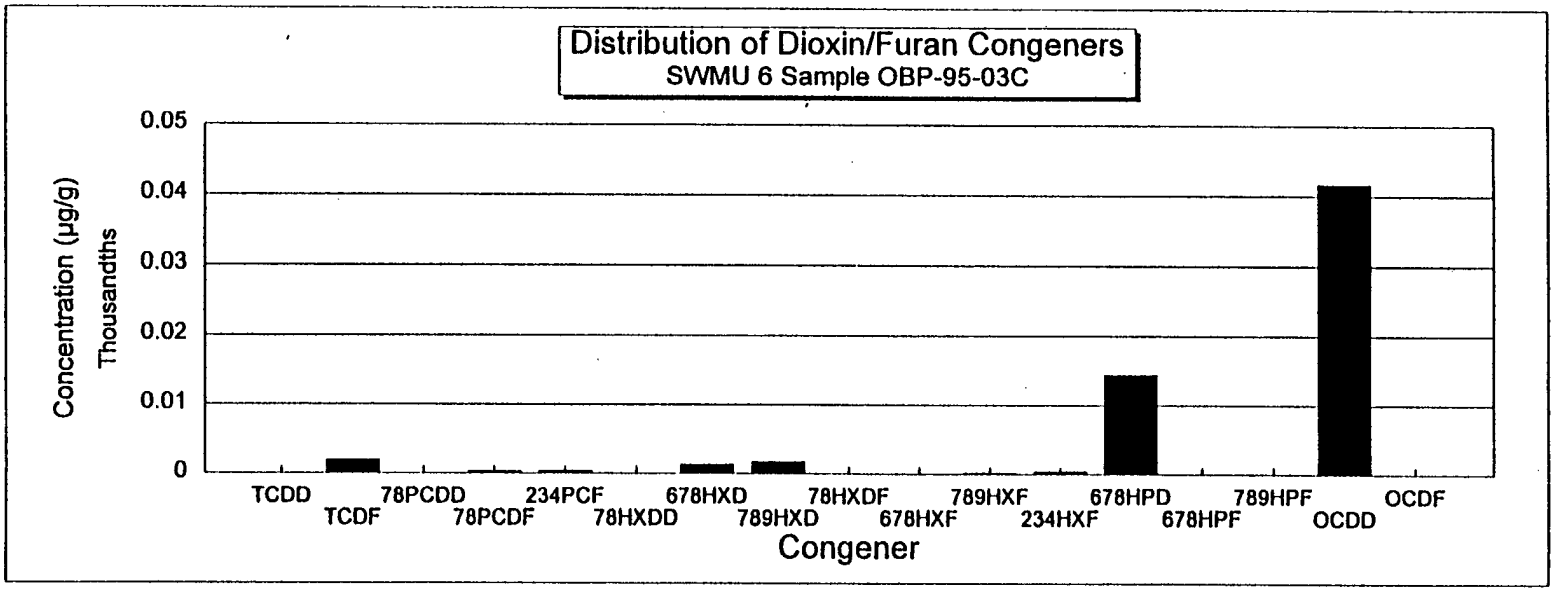

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBP-95-01B
Concentration (µg/g)
Thousandths
1
0.8
0.6
0.4
0.2
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

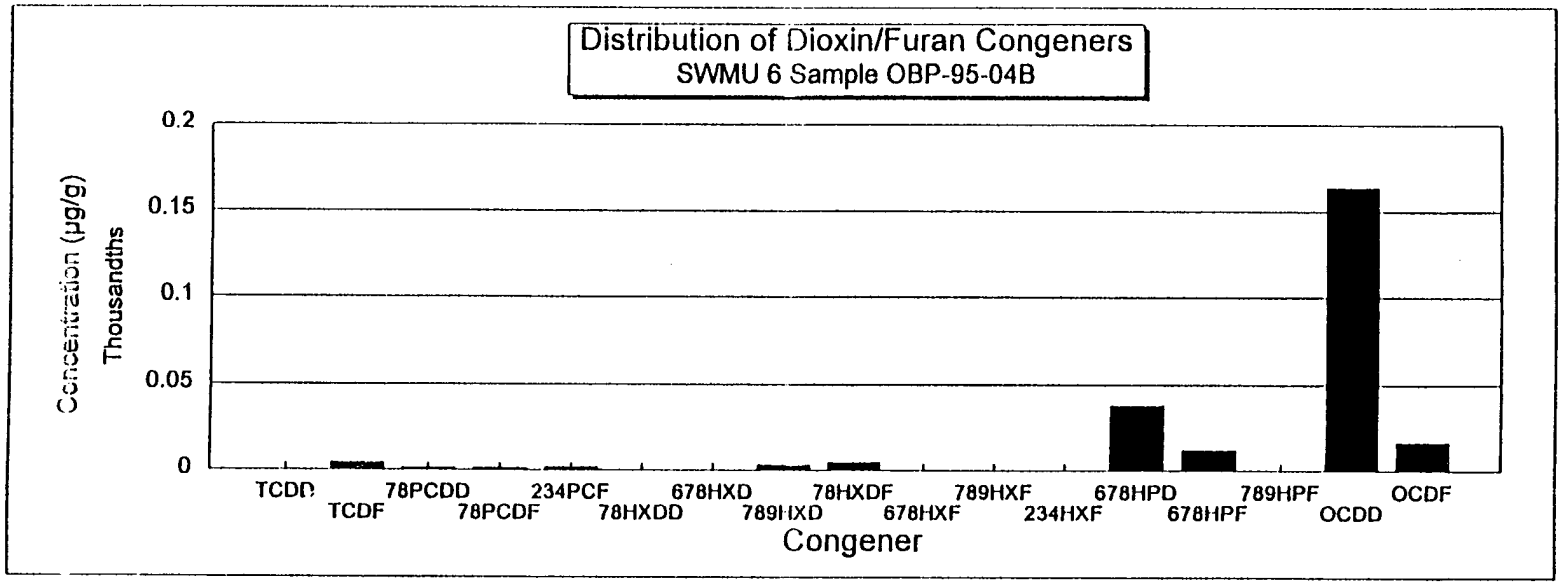

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBP-95-01C
Concentration (µg/g)
Thousandths
0.01
0.008
0.006
0.004
0.002
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener

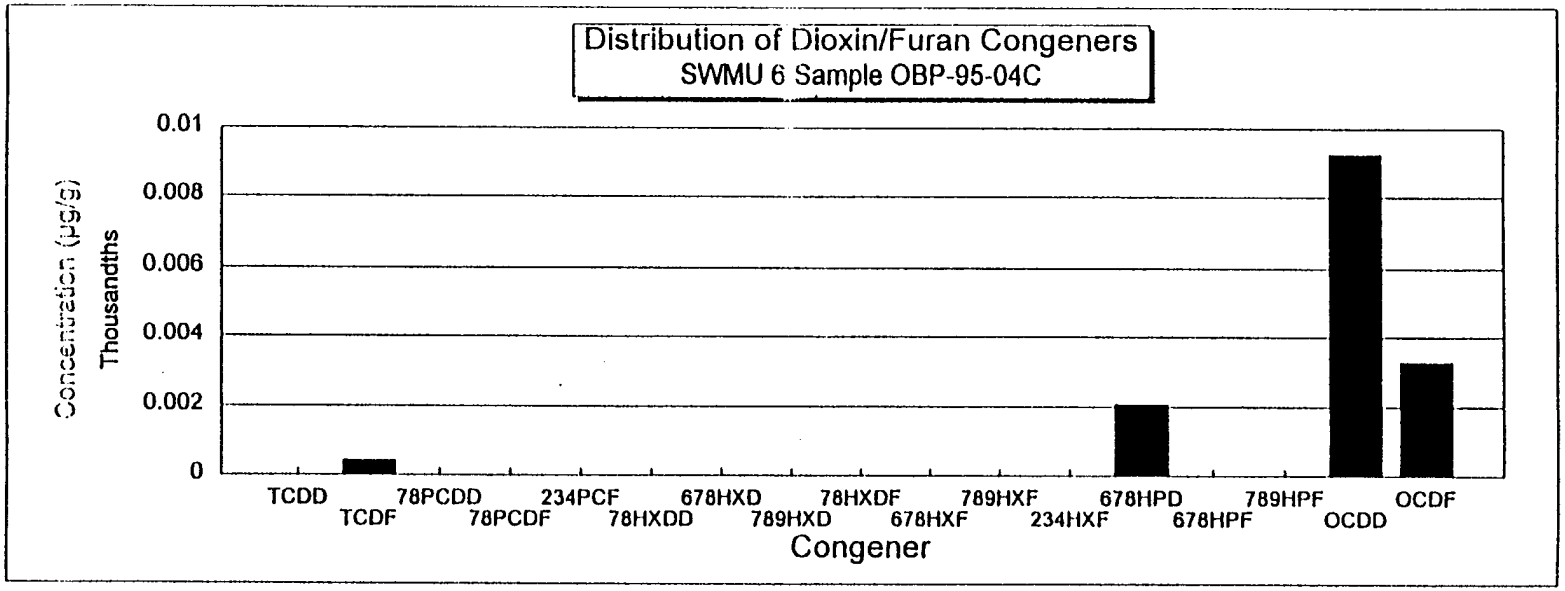

Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBP-95-02B
Concentration (µg/g)
Thousandths
0.8
0.6
0.4
0.2
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener


Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBP-95-02C
Concentration (µg/g)
Thousandths
0.4
0.3
0.2
0.1
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener


Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBP-95-03B
Concentration (µg/g)
Thousandths
1.5
1
0.5
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener


Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBP-95-03C
Concentration (µg/g)
Thousandths
0.05
0.04
0.03
0.02
0.01
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener


Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBP-95-04B
Concentration (µg/g)
Thousandths
0.2
0.15
0.1
0.05
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener


Distribution of Dioxin/Furan Congeners
SWMU 6 Sample OBP-95-04C
Concentration (µg/g)
Thousandths
0.01
0.008
0.006
0.004
0.002
0
TCDD
TCDF
78PCDD
78PCDF
234PCF
78HXDD
678HXD
789HXD
78HXDF
678HXF
789HXF
234HXF
678HPD
678HPF
789HPF
OCDD
OCDF
Congener