

US EPA ARCHIVE DOCUMENT

APPENDIX C.7

DERMAL APPENDIX

Updated Dermal Equations and Parameters

provided by USEPA Region 1

1999

Dermal Worksheet
Intermediate Variables for Calculating DA(event)
Wells G&H Superfund Site OU3

Chemical of Potential Concern	Media	Dermal Absorption Fraction (soil)	FA	Kp		T(event)		Tau		T*		B
			Value	Value	Units	Value	Units	Value	Units	Value	Units	Value
Heptachlor	Surface Water	--	0.8	8.6E-03	cm/hr	1	hr/event	12.99	hr	31.16	hr	0.1
Trichloroethylene	Surface Water	--	1	1.2E-02	cm/hr	1	hr/event	0.57	hr	1.37	hr	0.1
Chloroform	Surface Water	--	1	6.8E-03	cm/hr	1	hr/event	0.49	hr	1.18	hr	0.03
Tetrachloroethylene	Surface Water	--	1	3.3E-02	cm/hr	1	hr/event	0.89	hr	2.14	hr	0.2
Benzo(a)anthracene	Sediment/Soil	0.13	No data	No data	No data	No data	No data	No data	No data	No data	No data	No data
Benzo(a)pyrene	Sediment/Soil	0.13	No data	No data	No data	No data	No data	No data	No data	No data	No data	No data
Benzo(b)fluoranthene	Sediment/Soil	0.13	No data	No data	No data	No data	No data	No data	No data	No data	No data	No data
Benzo(k)fluoranthene	Sediment/Soil	0.13	No data	No data	No data	No data	No data	No data	No data	No data	No data	No data
Dibenz(a,h)anthracene	Sediment/Soil	0.13	No data	No data	No data	No data	No data	No data	No data	No data	No data	No data
Indeno(1,2,3-cd)pyrene	Sediment/Soil	0.13	No data	No data	No data	No data	No data	No data	No data	No data	No data	No data
Phenanthrene	Sediment/Soil	0.13	No data	No data	No data	No data	No data	No data	No data	No data	No data	No data
Aroclor 1248	Sediment/Soil	0.14	No data	No data	No data	No data	No data	No data	No data	No data	No data	No data
Aroclor 1254	Sediment/Soil	0.14	No data	No data	No data	No data	No data	No data	No data	No data	No data	No data
Aroclor 1260	Sediment/Soil	0.14	No data	No data	No data	No data	No data	No data	No data	No data	No data	No data
Arsenic	Sediment/Soil	0.03	No data	No data	No data	No data	No data	No data	No data	No data	No data	No data
Cadmium	Sediment/Soil	0.01	No data	No data	No data	No data	No data	No data	No data	No data	No data	No data

FA = Fraction Absorbed Water

Kp = Dermal Permeability Coefficient of
Compound in Water

T(event) = Event Duration

Tau = Lag Time

T* = Time to Reach Steady-State

B = Dimensionless Ratio of the Permeability Coefficient of a Compound Through the
Stratum Corneum Relative to its Permeability Coefficient Across the Viable Epidermis

TABLE C.7-1. DERMALLY ABSORBED DOSE CALCULATIONS
(Variable Definitions follow Table)

Reach	Station(s)	Timeframe	Receptor	Cancer/ Non-cancer	RME/ CT	A cm ²	t _{event} hr/event	EV event/day	EF days/yr	ED years	BW kg	AT days	Isc cm	IR cm ³ /day	ABSGI	Chemical	CAS No.	MWT	logKow	Kp 95% LCI	Kp (cm/hr) predicted	Kp (cm/hr) measured	Kp 95% UCI	Derm/Drink Kp	
01	NR, 22/TT-22, WH, and WG	Current	Adult	Non-cancer	RME	5700	1	1	26	24	70	8760	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	20%	
					CT	5700	0.5	1	26	7	70	2555	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	14%	
				Cancer	RME	5700	1	1	26	24	70	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	20%	
					CT	5700	0.5	1	26	7	70	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	14%	
			Child	Non-cancer	RME	2800	1	1	26	6	15	2190	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	10%	
					CT	2800	0.5	1	26	2	15	730	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	7%	
				Cancer	RME	2800	1	1	26	6	15	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	10%	
					CT	2800	0.5	1	26	2	15	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	7%	
	14	Current	Adult	Non-cancer	RME	5700	1	1	26	24	70	8760	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	20%	
					CT	5700	0.5	1	26	7	70	2555	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	14%	
				Cancer	RME	5700	1	1	26	24	70	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	20%	
					CT	5700	0.5	1	26	7	70	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	14%	
			Child	Non-cancer	RME	2800	1	1	26	6	15	2190	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	10%	
					CT	2800	0.5	1	26	2	15	730	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	7%	
				Cancer	RME	2800	1	1	26	6	15	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	10%	
					CT	2800	0.5	1	26	2	15	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	7%	
	WS/WSS	Current	Adult	Non-cancer	RME	5700	1	1	104	24	70	8760	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	20%	
					CT	5700	0.5	1	78	7	70	2555	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	14%	
				Cancer	RME	5700	1	1	104	24	70	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	20%	
					CT	5700	0.5	1	78	7	70	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	14%	
			Child	Non-cancer	RME	2800	1	1	104	6	15	2190	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	10%	
					CT	2800	0.5	1	78	2	15	730	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	7%	
				Cancer	RME	2800	1	1	104	6	15	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	10%	
					CT	2800	0.5	1	78	2	15	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	7%	
22/TT-22, 13/TT-27, WH, NT-1, NT-2, NT-3, WG, WW, and JY	Future	Adult	Non-cancer	RME	5700	1	1	78	24	70	8760	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	20%		
				CT	5700	0.5	1	26	7	70	2555	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	14%		
			Cancer	RME	5700	1	1	78	24	70	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	20%		
				CT	5700	0.5	1	26	7	70	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	14%		
		Child	Non-cancer	RME	2800	1	1	78	6	15	2190	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	10%		
				CT	2800	0.5	1	26	2	15	730	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	7%		
			Cancer	RME	2800	1	1	78	6	15	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	10%		
				CT	2800	0.5	1	26	2	15	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	7%		
		NR and 14	Future	Adult	Non-cancer	RME	5700	1	1	52	24	70	8760	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	20%
						CT	5700	0.5	1	52	7	70	2555	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	14%
					Cancer	RME	5700	1	1	52	24	70	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	20%
						CT	5700	0.5	1	52	7	70	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	14%
Child	Non-cancer			RME	2800	1	1	52	6	15	2190	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	10%		
				CT	2800	0.5	1	52	2	15	730	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	7%		
	Cancer			RME	2800	1	1	52	6	15	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	10%		
				CT	2800	0.5	1	52	2	15	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	7%		
WS/WSS	Future	Adult	Non-cancer	RME	5700	1	1	104	24	70	8760	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	20%		
				CT	5700	0.5	1	78	7	70	2555	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	14%		
			Cancer	RME	5700	1	1	104	24	70	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	20%		
				CT	5700	0.5	1	78	7	70	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	14%		
		Child	Non-cancer	RME	2800	1	1	104	6	15	2190	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	10%		
				CT	2800	0.5	1	78	2	15	730	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	7%		
			Cancer	RME	2800	1	1	104	6	15	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	10%		
				CT	2800	0.5	1	78	2	15	25550	1.0E-03	2000	1	Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01	7%		
U2	TT-30, CB-07, and AM	Current	Adult	Non-cancer	RME	5700	1	1	26	24	70	8760	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	7%	
					CT	5700	0.5	1	26	7	70	2555	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	5%	
				Cancer	RME	5700	1	1	26	24	70	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	7%	
					CT	5700	0.5	1	26	7	70	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	5%	
			Child	Non-cancer	RME	2800	1	1	26	6	15	2190	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	4%	
					CT	2800	0.5	1	26	2	15	730	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	3%	
				Cancer	RME	2800	1	1	26	6	15	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	4%	
					CT	2800	0.5	1	26	2	15	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	3%	
	CB-01, CB-02, CB-03, CB-04, and CB-06	Current	Adult	Non-cancer	RME	5700	1	1	104	24	70	8760	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	7%	
					CT	5700	0.5	1	78	7	70	2555	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	5%	
				Cancer	RME	5700	1	1	104	24	70	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	7%	
					CT	5700	0.5	1	78	7	70	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	5%	
			Child	Non-cancer	RME	2800	1	1	104	6	15	2190	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	4%	
					CT	2800	0.5	1	78	2	15	730	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	3%	
				Cancer	RME	2800	1																		

TABLE C.7-1. DERMALLY ABSORBED DOSE CALCULATIONS
(Variable Definitions follow Table)

Reach	Station(s)	Timeframe	Receptor	Cancer/ Non-cancer	RME/ CT	A cm ²	t _{event} hr/event	EV event/day	EF days/yr	ED years	BW kg	AT days	Isc cm	IR cm ³ /day	ABSGI	Chemical	CAS No.	MWT	logKow	Kp 95% LCI	Kp (cm/hr) predicted	Kp (cm/hr) measured	Kp 95% UCI	Derm/Drink Kp			
U2 (cont.)	16/TT-33, 09, and DA (cont.)		Child	Non-cancer	RME	2800	1	1	78	6	15	2190	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	4%			
					CT	2800	0.5	1	26	2	15	730	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	3%			
			Cancer	RME	2800	1	1	78	6	15	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	4%				
				CT	2800	0.5	1	26	2	15	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	3%				
			TT-30, CB-07, and AM	Future	Adult	Non-cancer	RME	5700	1	1	26	24	70	8760	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	7%	
							CT	5700	0.5	1	26	7	70	2555	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	5%	
	Cancer	RME			5700	1	1	26	24	70	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	7%				
		CT			5700	0.5	1	26	7	70	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	5%				
	Child	Non-cancer			RME	2800	1	1	26	6	15	2190	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	4%			
					CT	2800	0.5	1	26	2	15	730	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	3%			
	TT-31	Future	Adult	Non-cancer	RME	2800	1	1	26	6	15	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	4%			
					CT	2800	0.5	1	26	2	15	730	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	3%			
			Cancer	RME	2800	1	1	78	6	15	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	4%				
				CT	2800	0.5	1	26	2	15	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	3%				
			Child	Non-cancer	RME	2800	1	1	78	6	15	2190	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	4%			
					CT	2800	0.5	1	26	2	15	730	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	3%			
	CB-01, CB-02, CB-03, CB-04, and CB-06	Future	Adult	Non-cancer	RME	5700	1	1	104	24	70	8760	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	7%			
					CT	5700	0.5	1	78	7	70	2555	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	5%			
				Cancer	RME	5700	1	1	104	24	70	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	7%			
					CT	5700	0.5	1	78	7	70	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	5%			
			Child	Non-cancer	RME	2800	1	1	104	6	15	2190	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	4%			
					CT	2800	0.5	1	78	2	15	730	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	3%			
				Cancer	RME	2800	1	1	104	6	15	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	4%			
					CT	2800	0.5	1	78	2	15	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	3%			
			16/TT-33, 09, and DA	Future	Adult	Non-cancer	RME	5700	1	1	78	24	70	8760	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	7%	
							CT	5700	0.5	1	26	7	70	2555	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	5%	
						Cancer	RME	5700	1	1	78	24	70	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	7%	
							CT	5700	0.5	1	26	7	70	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	5%	
	Child	Non-cancer			RME	2800	1	1	78	6	15	2190	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	4%			
					CT	2800	0.5	1	26	2	15	730	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	3%			
		Cancer			RME	2800	1	1	78	6	15	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	4%			
					CT	2800	0.5	1	26	2	15	25550	1.0E-03	2000	1	Trichloroethylene	79016	131.4	2.42	4.7E-04	1.2E-02		2.9E-01	3%			
	05	05	Current	Adult	Non-cancer	RME	5700	1	1	78	24	70	8760	1.0E-03	2000	1	Chloroform	67663	119.4	1.97	2.8E-04	6.8E-03		1.7E-01	4%		
						5700	1	1	78	24	70	8760	1.0E-03	2000	1	Tetrachloroethylene	127184	165.8	3.40	1.3E-03	3.3E-02		8.4E-01	25%			
						CT	5700	0.5	1	26	7	70	2555	1.0E-03	2000	1	Chloroform	67663	119.4	1.97	2.8E-04	6.8E-03		1.7E-01	3%		
						5700	0.5	1	26	7	70	2555	1.0E-03	2000	1	Tetrachloroethylene	127184	165.8	3.40	1.3E-03	3.3E-02		8.4E-01	17%			
						Cancer	RME	5700	1	1	78	24	70	25550	1.0E-03	2000	1	Chloroform	67663	119.4	1.97	2.8E-04	6.8E-03		1.7E-01	4%	
							5700	1	1	78	24	70	25550	1.0E-03	2000	1	Tetrachloroethylene	127184	165.8	3.40	1.3E-03	3.3E-02		8.4E-01	25%		
							CT	5700	0.5	1	26	7	70	25550	1.0E-03	2000	1	Chloroform	67663	119.4	1.97	2.8E-04	6.8E-03		1.7E-01	3%	
							5700	0.5	1	26	7	70	25550	1.0E-03	2000	1	Tetrachloroethylene	127184	165.8	3.40	1.3E-03	3.3E-02		8.4E-01	17%		
						Child	Non-cancer	RME	2800	1	1	78	6	15	2190	1.0E-03	2000	1	Tetrachloroethylene	127184	165.8	3.40	1.3E-03	3.3E-02		8.4E-01	12%
								CT	2800	0.5	1	26	2	15	730	1.0E-03	2000	1	Chloroform	67663	119.4	1.97	2.8E-04	6.8E-03		1.7E-01	1%
Cancer							RME	2800	1	1	78	6	15	25550	1.0E-03	2000	1	Tetrachloroethylene	127184	165.8	3.40	1.3E-03	3.3E-02		8.4E-01	9%	
							CT	2800	0.5	1	26	7	70	25550	1.0E-03	2000	1	Chloroform	67663	119.4	1.97	2.8E-04	6.8E-03		1.7E-01	2%	
Future					Adult	Non-cancer	RME	5700	1	1	78	24	70	8760	1.0E-03	2000	1	Chloroform	67663	119.4	1.97	2.8E-04	6.8E-03		1.7E-01	4%	
							5700	1	1	78	24	70	8760	1.0E-03	2000	1	Tetrachloroethylene	127184	165.8	3.40	1.3E-03	3.3E-02		8.4E-01	25%		
							CT	5700	0.5	1	26	7	70	2555	1.0E-03	2000	1	Chloroform	67663	119.4	1.97	2.8E-04	6.8E-03		1.7E-01	3%	
							5700	0.5	1	26	7	70	2555	1.0E-03	2000	1	Tetrachloroethylene	127184	165.8	3.40	1.3E-03	3.3E-02		8.4E-01	17%		
						Cancer	RME	5700	1	1	78	24	70	25550	1.0E-03	2000	1	Chloroform	67663	119.4	1.97	2.8E-04	6.8E-03		1.7E-01	4%	
							5700	1	1	78	24	70	25550	1.0E-03	2000	1	Tetrachloroethylene	127184	165.8	3.40	1.3E-03	3.3E-02		8.4E-01	25%		
							CT	5700	0.5	1	26	7	70	25550	1.0E-03	2000	1	Chloroform	67663	119.4	1.97	2.8E-04	6.8E-03		1.7E-01	3%	
							5700	0.5	1	26	7	70	25550	1.0E-03	2000	1	Tetrachloroethylene	127184	165.8	3.40	1.3E-03	3.3E-02		8.4E-01	17%		
						Child	Non-cancer	RME	2800	1	1	78	6	15	2190	1.0E-03	2000	1	Chloroform	67663	119.4	1.97	2.8E-04	6.8E-03		1.7E-01	2%
								CT	2800	1	1	78	6	15	2190	1.0E-03	2000	1	Tetrachloroethylene	127184	165.8	3.40	1.3E-03	3.3E-02		8.4E-01	12%
							Cancer	RME	2800	1	1	78	6	15	25550	1.0E-03	2000	1	Chloroform	67663	119.4	1.97	2.8E-04	6.8E-03		1.7E-01	1%
								CT	2800	0.5	1	26	2	15	730	1.0E-03	2000	1	Tetrachloroethylene	127184	165.8	3.40	1.3E-03	3.3E-02		8.4E-01	9%
Future				Child	Non-cancer	RME	2800	1	1	78	6	15	2190	1.0E-03	2000	1	Chloroform	67663	119.4	1.97	2.8E-04	6.8E-03		1.7E-01	2%		
						2800	1	1	78	6	15	2190	1.0E-03	2000	1	Tetrachloroethylene	127184	165.8	3.40	1.3E-03	3.3E-02		8.4E-01	12%			
						CT	2800	0.5	1	26	2	15	730	1.0E-03	2000	1	Chloroform	67663	119.4	1.97	2.8E-04	6.8E-03		1.7E-01	1%		
						2800	0.5	1	26	2	15	730	1.0E-03	2000	1	Tetrachloroethylene	127184	165.8	3.40	1.3E-03	3.3E-02		8.4E-01	9%			
					Cancer	RME	2800	1	1	78	6	15	25550	1.0E-03	2000	1	Chloroform	67663	119.4	1.97	2.8E-04	6.8E-03		1.7E-01	2%		
						2800	1	1	78	6	15	25550	1.0E-03	2000	1	Tetrachloroethylene	127184	165.8	3.40	1.3E-03	3.3E-02		8.4E-01	12%			
						CT	2800	0.5	1	26	2	15	25550	1.0E-03	2000	1	Chloroform	67663	119.4	1.9							

TABLE C.7-1. DERMALLY ABSORBED DOSE CALCULATIONS
(Variable Definitions follow Table)

Reach	Station(s)	Timeframe	Receptor	Cancer/ Non-cancer	RME/ CT	Chem Assess	B	tau (hr)	t_star (hr)	FA for tau>3	Conc mg/cm3	DA_event mg/cm2-evt	DAD mg/kg-day	log(Ds/lsc)	Dsc/lsc	Dsc	b	c	t_star1 B>0.6	t_star3 B<=0.6
01	NR, 22/TT-22, WH, and WG	Current	Adult	Non-cancer	RME	Y	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	7.6E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	Y	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	5.3E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
				Cancer	RME	Y	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	2.6E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	Y	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	5.3E-11	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
			Child	Non-cancer	RME	N	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	1.7E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	N	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	1.2E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
				Cancer	RME	N	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	1.5E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	N	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	3.5E-11	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
	14	Current	Adult	Non-cancer	RME	Y	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	7.6E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	Y	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	5.3E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
				Cancer	RME	Y	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	2.6E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	Y	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	5.3E-11	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
			Child	Non-cancer	RME	N	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	1.7E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	N	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	1.2E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
				Cancer	RME	N	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	1.5E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	N	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	3.5E-11	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
	WS/WSS	Current	Adult	Non-cancer	RME	Y	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	3.0E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	Y	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	1.6E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
				Cancer	RME	Y	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	1.0E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	Y	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	1.6E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
			Child	Non-cancer	RME	N	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	6.9E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	N	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	3.7E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
				Cancer	RME	N	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	5.9E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	N	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	1.0E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
	22/TT-22, 13/TT-27, WH, NT-1, NT-2, NT-3, WG, WW, and JY	Future	Adult	Non-cancer	RME	Y	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	2.3E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	Y	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	5.3E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
				Cancer	RME	Y	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	7.8E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	Y	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	5.3E-11	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
			Child	Non-cancer	RME	N	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	5.2E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	N	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	1.2E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
				Cancer	RME	N	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	4.5E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	N	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	3.5E-11	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
	NR and 14	Future	Adult	Non-cancer	RME	Y	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	1.5E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	Y	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	1.1E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
				Cancer	RME	Y	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	5.2E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	Y	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	1.1E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
			Child	Non-cancer	RME	N	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	3.5E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	N	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	2.4E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
				Cancer	RME	N	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	3.0E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	N	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	7.0E-11	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
	WS/WSS	Future	Adult	Non-cancer	RME	Y	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	3.0E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	Y	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	1.6E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
				Cancer	RME	Y	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	1.0E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	Y	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	1.6E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
			Child	Non-cancer	RME	N	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	6.9E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	N	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	3.7E-09	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
				Cancer	RME	N	0.1	12.99	31.16	0.8	1.9E-09	1.3E-10	5.9E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
					CT	N	0.1	12.99	31.16	0.8	1.9E-09	9.2E-11	1.0E-10	-4.89E+00	1.28E-05	1.28E-08	3.4E-01	3.8E-01	N/A	31.16
U2	TT-30, CB-07, and AM	Current	Adult	Non-cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	3.0E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	2.1E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
				Cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	1.0E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	2.1E-08	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
			Child	Non-cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	6.8E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	4.8E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
				Cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	5.8E-08	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	1.4E-08	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
	CB-01, CB-02, CB-03, CB-04, and CB-06	Current	Adult	Non-cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	1.2E-06	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	6.3E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
				Cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	4.1E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	6.3E-08	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
			Child	Non-cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	2.7E-06	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	1.4E-06	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
				Cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	2.3E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	4.1E-08	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
16/TT-33, 09, and DA	Current	Adult	Non-cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	8.9E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37	

TABLE C.7-1. DERMALLY ABSORBED DOSE CALCULATIONS
(Variable Definitions follow Table)

Reach	Station(s)	Timeframe	Receptor	Cancer/ Non-cancer	RME/ CT	Chem Assess	B	tau (hr)	t_star (hr)	FA for tau>3	Conc mg/cm3	DA_event mg/cm2-evt	DAD mg/kg-day	log(Ds/lsc)	Dsc/lsc	Dsc	b	c	t_star1 B>0.6	t_star3 B<=0.6		
U2 (cont.)	16/TT-33, 09, and DA (cont.)	rent	Adult	Non-cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	2.0E-06	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	4.8E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
				Cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	1.7E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	1.4E-08	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
		TT-30, CB-07, and AM	Future	Adult	Non-cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	3.0E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37	
						CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	2.1E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37	
				Cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	1.0E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	2.1E-08	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
			Child	Non-cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	6.8E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	4.8E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
				Cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	5.8E-08	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	1.4E-08	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
	TT-31	Future	Adult	Non-cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	8.9E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	2.1E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
				Cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	3.0E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	2.1E-08	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
			Child	Non-cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	2.0E-06	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	4.8E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
				Cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	1.7E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	1.4E-08	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
			CB-01, CB-02, CB-03, CB-04, and CB-06	Future	Adult	Non-cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	1.2E-06	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
							CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	6.3E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
						Cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	4.1E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
							CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	6.3E-08	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
	Child	Non-cancer			RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	2.7E-06	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	1.4E-06	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
		Cancer			RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	2.3E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	4.1E-08	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
	16/TT-33, 09, and DA	Future			Adult	Non-cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	8.9E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
							CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	2.1E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
						Cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	3.0E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
							CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	2.1E-08	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37
			Child	Non-cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	2.0E-06	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	4.8E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
				Cancer	RME	N	0.1	0.57	1.37	1.0	2.0E-06	5.1E-08	1.7E-07	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
					CT	N	0.1	0.57	1.37	1.0	2.0E-06	3.6E-08	1.4E-08	-3.54E+00	2.91E-04	2.91E-07	3.4E-01	3.7E-01	N/A	1.37		
	05	05	Current	Adult	Non-cancer	RME	N	0.03	0.49	1.18	1.0	1.0E-06	1.3E-08	2.3E-07	-3.47E+00	3.40E-04	3.40E-07	3.2E-01	3.5E-01	N/A	1.18	
							Y	0.2	0.89	2.14	1.0	2.0E-06	1.7E-07	3.0E-06	-3.73E+00	1.87E-04	1.87E-07	4.1E-01	4.5E-01	N/A	2.14	
					Cancer	RME	N	0.03	0.49	1.18	1.0	1.0E-06	1.3E-08	7.9E-08	-3.47E+00	3.40E-04	3.40E-07	3.2E-01	3.5E-01	N/A	1.18	
							Y	0.2	0.89	2.14	1.0	2.0E-06	1.7E-07	1.0E-06	-3.73E+00	1.87E-04	1.87E-07	4.1E-01	4.5E-01	N/A	2.14	
Child				Non-cancer		RME	N	0.03	0.49	1.18	1.0	1.0E-06	1.3E-08	5.2E-07	-3.47E+00	3.40E-04	3.40E-07	3.2E-01	3.5E-01	N/A	1.18	
							Y	0.2	0.89	2.14	1.0	2.0E-06	1.7E-07	6.9E-06	-3.73E+00	1.87E-04	1.87E-07	4.1E-01	4.5E-01	N/A	2.14	
				Cancer	RME	N	0.03	0.49	1.18	1.0	1.0E-06	1.3E-08	4.5E-08	-3.47E+00	3.40E-04	3.40E-07	3.2E-01	3.5E-01	N/A	1.18		
						Y	0.2	0.89	2.14	1.0	2.0E-06	1.7E-07	5.9E-07	-3.73E+00	1.87E-04	1.87E-07	4.1E-01	4.5E-01	N/A	2.14		
Future					Adult	Non-cancer	RME	N	0.03	0.49	1.18	1.0	1.0E-06	1.3E-08	2.3E-07	-3.47E+00	3.40E-04	3.40E-07	3.2E-01	3.5E-01	N/A	1.18
								Y	0.2	0.89	2.14	1.0	2.0E-06	1.7E-07	3.0E-06	-3.73E+00	1.87E-04	1.87E-07	4.1E-01	4.5E-01	N/A	2.14
				Child		Non-cancer	RME	N	0.03	0.49	1.18	1.0	1.0E-06	1.3E-08	5.2E-07	-3.47E+00	3.40E-04	3.40E-07	3.2E-01	3.5E-01	N/A	1.18
								Y	0.2	0.89	2.14	1.0	2.0E-06	1.7E-07	6.9E-06	-3.73E+00	1.87E-04	1.87E-07	4.1E-01	4.5E-01	N/A	2.14
Cancer					RME	N	0.03	0.49	1.18	1.0	1.0E-06	1.3E-08	4.5E-08	-3.47E+00	3.40E-04	3.40E-07	3.2E-01	3.5E-01	N/A	1.18		
						Y	0.2	0.89	2.14	1.0	2.0E-06	1.7E-07	5.9E-07	-3.73E+00	1.87E-04	1.87E-07	4.1E-01	4.5E-01	N/A	2.14		
				Future	Adult	Non-cancer	RME	N	0.03	0.49	1.18	1.0	1.0E-06	1.3E-08	2.3E-07	-3.47E+00	3.40E-04	3.40E-07	3.2E-01	3.5E-01	N/A	1.18
								Y	0.2	0.89	2.14	1.0	2.0E-06	1.7E-07	3.0E-06	-3.73E+00	1.87E-04	1.87E-07	4.1E-01	4.5E-01	N/A	2.14
Child						Non-cancer	RME	N	0.03	0.49	1.18	1.0	1.0E-06	1.3E-08	5.2E-07	-3.47E+00	3.40E-04	3.40E-07	3.2E-01	3.5E-01	N/A	1.18
								Y	0.2	0.89	2.14	1.0	2.0E-06	1.7E-07	6.9E-06	-3.73E+00	1.87E-04	1.87E-07	4.1E-01	4.5E-01	N/A	2.14
				Cancer	RME	N	0.03	0.49	1.18	1.0	1.0E-06	1.3E-08	4.5E-08	-3.47E+00	3.40E-04	3.40E-07	3.2E-01	3.5E-01	N/A	1.18		
						Y	0.2	0.89	2.14	1.0	2.0E-06	1.7E-07	5.9E-07	-3.73E+00	1.87E-04	1.87E-07	4.1E-01	4.5E-01	N/A	2.14		
Future					Adult	Non-cancer	RME	N	0.03	0.49	1.18	1.0	1.0E-06	1.3E-08	2.3E-07	-3.47E+00	3.40E-04	3.40E-07	3.2E-01	3.5E-01	N/A	1.18
								Y	0.2	0.89	2.14	1.0	2.0E-06	1.7E-07	3.0E-06	-3.73E+00	1.87E-04	1.87E-07	4.1E-01	4.5E-01	N/A	2.14
				Child		Non-cancer	RME	N	0.03	0.49	1.18	1.0	1.0E-06	1.3E-08	5.2E-07	-3.47E+00	3.40E-04	3.40E-07	3.2E-01	3.5E-01	N/A	1.18
								Y	0.2	0.89	2.14	1.0	2.0E-06	1.7E-07	6.9E-06	-3.73E+00	1.87E-04	1.87E-07	4.1E-01	4.5E-01	N/A	2.14
Cancer					RME	N	0.03	0.49	1.18	1.0	1.0E-06	1.3E-08	4.5E-08	-3.47E+00	3.40E-04	3.40E-07	3.2E-01	3.5E-01	N/A	1.18		
						Y	0.2	0.89	2.14	1.0	2.0E-06	1.7E-07	5.9E-07	-3.73E+00	1.87E-04	1.87E-07	4.1E-01	4.5E-01	N/A	2.14		

DERMAL ABSORPTION CALCULATION EXAMPLE

Note: This EPA spreadsheet utilized as basis for Table C.7-1 calculations.

FOR ORGANIC CHEMICALS IN WATER (updated on 11/99)

Worksheet to Calculate Dermal Absorption of Organic Chemicals from Aqueous Media (updated 11/99)

Enter the Following Exposure Conditions: for site specific conditions, change values in Cells G5-G18

Concentration (mg/L*L/1000 cm3):	Conc =	1.0E-03 mg/cm3 (default value for purpose of illustration)
Input site specific concentrations in Column marked "Conc"		= 1 mg/L (1 ppm) = 1 ug/cm3 = 1000 ppb
Area exposed (cm2):	A =	5672.0 cm2
Event time (hr/event):	t_event =	0.5 hr/event (35 minutes/event)
Event frequency (events/day):	EV =	1.0 event/day
Exposure frequency (days/year):	EF =	26.0 days/yr
Exposure duration (years):	ED =	7.0 years
Body weight (kg):	BW =	70.0 kg
Averaging time (days):	AT =	2555.0 days
for carcinogenic effects, AT=70 years (25,550 days)		
for noncarcinogenic effects, AT=ED (in days)		
Skin thickness (assumed to be 10 um):	Lsc =	1.0E-03 cm

Default conditions for screening purposes:

Compare Dermal to Drinking: Adults showering for 35 minutes/day, compared to drinking 2L water/day

Dermal (mg/day) = DA_event * A * EV	IR =	2000.0 (cm3/day = L/day * 1000 cm3/L)
Drinking (mg/day) = Conc * IR * ABSIG	ABSGI =	1.0 (assumed 100% GI absorption)

IR: Ingestion rate of drinking water
ABSIG: Absorption fraction in GI tract

Refer to Appendix A for equations to evaluate DA_event and DAD

(*): outside of the Effective Prediction Domain (EPD) determined by the Flynn's measured Kp data

95% LCI and UCI are evaluated by Dr. Paul Pinsky in NCEA using SAS

CHEMICAL	CAS No.	MWT	logKow	Kp 95% LCI	Kp (cm/hr) predicted	Kp (cm/hr) measured	Kp 95% UCI	Chemicals outside EPD (*)	Derm/ Drink Kp	Chem Assess	B	tau (hr)	t_star (hr)
118 Heptachlor	76448	373.5	4.27	3.4E-04	8.6E-03		2.2E-01		14%	Y	0.1	12.99	31.16
	FA for tau>3	Conc (mg/cm3)	DA_event (mg/cm2-evt)	DAD (mg/kg-day)		log(Ds/lsc)	Dsc/lsc	Dsc		b	c	t_star1 B>0.6	t_star3 B<=0.6
	0.8	1.4E-09	6.8E-11	3.9E-10		-4.89E+00	1.28E-05	1.28E-08		3.4E-01	3.8E-01	#NUM!	31.16

APPENDIX C.8

TOXICITY PROFILES FOR COPCs

INTRODUCTION

This Appendix contains toxicity criteria and toxicity profiles for the chemicals selected as chemicals of potential concern (COPCs) for the Wells G&H Superfund Site OU3 baseline human health risk assessment. The chronic oral toxicity criteria for COPCs are summarized in Tables 3-5.1 and 3-6.1. Table C.8-1 presents the absolute oral bioavailability factors (i.e., oral to dermal adjustment factors) used to adjust the oral toxicity criteria for the COPCs evaluated in the dermal exposure pathways, as discussed in Section 3.0, subsection 3.4.3. Toxicity profiles for the COPCs are provided in the following pages.

VOLATILE ORGANICS

Chloroform

Chloroform is a colorless, volatile liquid that is widely used as a general solvent and as an intermediate in the production of refrigerants, plastics, and pharmaceuticals (Torkelson and Rowe, 1976; IARC, 1976). Chloroform is rapidly absorbed from the lungs and the gastrointestinal tract, and to some extent through the skin. It is extensively metabolized in the body, with carbon dioxide as the major end product. The primary sites of metabolism are the liver and kidneys. Excretion of chloroform occurs primarily via the lungs, either as unchanged chloroform or as carbon dioxide (ATSDR, 2002).

Target organs for chloroform toxicity are the liver, kidneys, and central nervous system. Liver effects (hepatomegaly, fatty liver, and hepatitis) were observed in individuals occupationally exposed to chloroform (Bomski et al., 1967). Several subchronic and chronic studies by the oral routes of exposure documented hepatotoxic effects in rats, mice, and dogs (Palmer et al., 1979; Munson et al., 1979; Heywood et al., 1979). Renal effects were reported in rats and mice following oral exposures (Roe et al., 1979; Reuber, 1976; Torkelson et al., 1976), but evidence for chloroform-induced renal toxicity in humans is sparse. Chloroform is a central nervous system depressant, inducing narcosis and anesthesia at high concentrations. Lower concentrations may cause irritability, lassitude, depression, gastrointestinal symptoms, and frequent and burning urination (ATSDR, 2002).

Developmental toxicity studies with rodents indicate that orally administered chloroform is toxic to dams and fetuses. Chloroform may cause sperm abnormalities in mice and gonadal atrophy in rats (Palmer et al., 1979; Reuber, 1979).

Epidemiological studies indicate a possible relationship between exposure to chloroform present in chlorinated drinking water and cancer of the bladder, large intestine, and rectum. Chloroform is one of several contaminants present in drinking water, but it has not been identified as the sole or primary cause of the excess cancer rate (ATSDR, 2002; U.S. EPA, 1985). In animal carcinogenicity studies, positive results included increased incidences of renal epithelial tumors in male rats, hepatocellular carcinomas in male and female mice, and kidney tumors in male mice (Jorgensen et al., 1985; Roe et al., 1979; NCI, 1976). Based on U.S. EPA guidelines, chloroform was assigned to weight-of-evidence Group B2, probable human carcinogen, on the basis of an increased incidence of several tumor types in rats and in three strains of mice.

ATSDR (Agency for Toxic Substances and Disease Registry). 2002. Toxicological Profile for Chloroform. Prepared by Syracuse Research Corporation. U.S. Public Health Service..

Bomski, H., A. Sobolweska and A. Strakowski. 1967. Toxic damage to the liver by chloroform in chemical industry workers. Arch. Gewerbepathol. Gewerbehyg. 24: 127-134. (In German; cited in ATSDR, 2002; Torkelson and Rowe, 1981; IARC, 1979)

Heywood, R., R.J. Sortwell, P.R.B. Noel, et al. 1979. Safety evaluation of toothpaste containing chloroform. III. Long-term study in beagle dogs. *J. Environ. Pathol. Toxicol.* 2: 835-851.

IARC (International Agency for Research on Cancer). 1979. Chloroform. In: Some Halogenated Hydrocarbons. IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol. 20. World Health Organization, Lyon, France, pp. 401-427.

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

Munson, A.E., L.E. Sain, V.M. Sanders, et al. 1982. Toxicology of organic drinking water contaminants: Trichloromethane, bromodichloromethane, dibromochloromethane, and tribromomethane. *Environ. Health Perspect.* 46: 117-126.

NCI (National Cancer Institute). 1976. Report on Carcinogenesis Bioassay of Chloroform. National Cancer Institute, Washington, DC. NTIS PB 264018.

Palmer, A.K., A.E. Street, F.J.C. Roe, et al. 1979. Safety evaluation of toothpaste containing chloroform. II. Long term studies in rats. *J. Environ. Path. Toxicol.* 2: 821-833.

Reuber, M.D. 1979. Carcinogenicity of chloroform. *Environ. Health Perspect.* 31: 171-182. (Cited in U.S. EPA, 1985)

Roe, F.J.C., A.A.K. Palmer, A.N. Worden, et al. 1979. Safety evaluation of toothpaste containing chloroform. I. Long-term studies in mice. *J. Environ. Toxicol.* 2: 799-819.

Torkelson, T.R. and V.K. Rowe. 1981. Halogenated aliphatic hydrocarbons containing chlorine, bromine and iodine. In: G.D. Clayton and E. Clayton, Eds. *Patty's Industrial Hygiene and Toxicology*, Vol. 2B. John Wiley & Sons, New York, pp. 3462-3469.

Torkelson, T.R., F. Oyen and V.K. Rowe. 1976. The toxicity of chloroform as determined by single and repeated exposure of laboratory animals. *Am. Ind. Hyg. Assoc.* 37: 697-704.

U.S. EPA. 1985. Health Assessment Document for Chloroform. Final Report. Office of Health and Environmental Assessment, Washington, DC. EPA/600/8-84/004F, NTIS PB86-105004/XAB.

Tetrachloroethene

Tetrachloroethene (PCE) is readily absorbed following inhalation and oral exposure (ATSDR, 2002). Tetrachloroethene vapors and liquid also can be absorbed through the skin (USEPA 1985a,b). The principal toxic effects of tetrachloroethene in humans and animals following acute

and longer-term exposures include CNS depression and fatty infiltration of the liver and kidney with concomitant changes in serum enzyme activity levels indicative of tissue damage (U.S. EPA 1985a,b; Buben and O'Flaherty 1985). Mice subchronically exposed to tetrachloroethene did not show any adverse liver effects at 20 mg/kg/day (Buben and O'Flaherty 1985).

In an NCI (1977) bioassay, increased incidence of hepatocellular carcinoma were observed in both sexes of B6C3F1 mice administered tetrachloroethylene (386–1,072 mg/kg/day) in corn oil by gavage for 78 weeks. Tetrachloroethene is currently under review by the Carcinogen Risk Assessment Verification Endeavor (CRAVE) and estimates of cancer potency were withdrawn by USEPA (1995). However, the USEPA National Center for Environmental Assessment currently classifies tetrachloroethene as a Group B2/C carcinogen (Probable/Possible Human Carcinogen).

Agency for Toxic Substances and Disease Registry (ATSDR). 2002. *Toxicological profile for tetrachloroethene*. Draft. U.S. Department of Health and Human Services. October.

Buben, J.A., and O'Flaherty, E.J. 1985. Delineation of the role of metabolism in the hepatotoxicity of trichloroethylene and perchloroethylene: A dose-effect study. *Toxicol. Appl. Pharmacol.* 78:105-122.

International Agency for Research on Cancer (IARC). 1979. *IARC Monographs on the evaluation of the carcinogenic risks of chemicals to humans. Vol. 20: Some Halogenated Hydrocarbons*. Lyon, France: World Health Organization.

National Cancer Institute (NCI). 1977. *Bioassay of tetrachloroethylene for possible carcinogenicity*. CAS No. 127-18-4. NCI Carcinogenesis Technical Report Series No. 13. DHEW (NIH) Publication No. 77-813

U.S. Environmental Protection Agency (USEPA). 1985a. *Health assessment document for tetrachloroethylene (perchloroethylene)*. Washington, D.C.: Office of Health and Environmental Assessment. July 1985. EPA 600/8-82-005F.

U.S. Environmental Protection Agency (USEPA). 1985b. *Drinking water criteria Document for tetrachloroethylene*. Washington, D.C.: Office of Drinking Water, Criteria and Standards Division. June 1985.

U.S. Environmental Protection Agency (USEPA). 1995. *Health effects assessment summary tables*. Cincinnati, OH: Office of Health and Environmental Assessment, Environmental Assessment and Criteria Office, Washington, D.C.: Office of Solid Waste and Emergency Response, Office of Remedial Response. FY-1995.

Trichloroethene

Absorption of trichloroethene (TCE) from the gastrointestinal tract is virtually complete. Absorption following inhalation exposure is proportional to concentration and duration of exposure (USEPA, 1985). TCE is a CNS depressant following acute and chronic exposures. In humans, single oral doses of 15–25 mL (21–35 grams) have resulted in vomiting and abdominal pain, followed by transient unconsciousness (Stephens, 1945). High-level exposure can result in death due to respiratory and cardiac failure (ATSDR, 2002). Hepatotoxicity has been reported in human and animal studies following acute exposure to TCE (ATSDR, 2002). Industrial use of TCE is often associated with adverse dermatological effects including reddening and skin burns on contact with the liquid form. These effects are usually the result of contact with concentrated solvent. However, no effects have been reported following exposure to TCE in dilute, aqueous solutions (USEPA, 1985).

TCE has caused significant increases in the incidence of hepatocellular carcinomas in mice (NCI, 1976), and renal tubular-cell neoplasms in rats exposed by gavage (NTP, 1983). TCE was mutagenic in *Salmonella typhimurium* and in *E. coli* (strain K-12), utilizing liver microsomes for activation (Greim *et al.*, 1977).

USEPA is currently reviewing the carcinogenicity of TCE. The National Center for Environmental Assessment (NCEA) currently classifies TCE as a Group B2/C (Probable/Possible Human Carcinogen) based on inadequate evidence in humans and sufficient evidence of carcinogenicity from animal studies.

Agency for Toxic Substances and Disease Registry (ATSDR). 2002. *Toxicological profile for trichloroethylene*. August 2002.

Greim, H., D. Bimboes, G. Egert, W. Giggelmann and M. Kramer. 1977. Mutagenicity and chromosomal aberrations as an analytical tool for *in vitro* detection of mammalian enzyme-mediated formation of reactive metabolites. *Arch. Toxicol.* 39:159.

National Cancer Institute (NCI). 1976. *Carcinogenesis bioassay of trichloroethylene*. CAS No. 79-01-6. Carcinogenesis Technical Report Series No. 2. PB-264 122.

National Toxicology Program (NTP). 1983. *Carcinogenesis studies of trichloroethylene (without epichlorohydrin), CAS No. 79-01-6, in F344/N rats and B6C3F₁ mice (gavage studies)*. Draft. NTP 81-84, NTP TR 243. August 1983.

Stephens, C. 1945. Poisoning by accidental drinking of trichloroethylene. *Br. Med. J.* 2:218.

U.S. Environmental Protection Agency (USEPA). 1985. *Health assessment document for trichloroethylene*. Environmental Criteria and Assessment Office. EPA/600/8-82/006F.

Vinyl Chloride

Vinyl chloride, a colorless gas, is a halogenated aliphatic hydrocarbon with the empirical formula of C_2H_3Cl . It is used primarily as an intermediate in the manufacture of polyvinyl chloride (PVC); limited quantities are used as a refrigerant and as an intermediate in the production of chlorinated compounds (ATSDR, 2002).

Vinyl chloride is rapidly absorbed from the gastrointestinal tract. Metabolism of vinyl chloride occurs primarily in the liver via oxidation by hepatic microsomal enzymes to polar compounds which can be conjugated with glutathione and/or cysteine. These covalently bound metabolites are then excreted in the urine (U.S. EPA, 1980, 1985).

For the oral route of exposure, the primary target organ of vinyl chloride toxicity in animals is the liver. Chronic oral administration of 1.7-14.1 mg/kg/day of vinyl chloride induced dose-related increases in nonneoplastic lesions of the liver of rats (Feron et al., 1981). Evidence of developmental toxicity was seen in rats exposed to vinyl chloride during the first trimester of gestation (Ungvary et al., 1978).

The carcinogenicity of vinyl chloride in humans has been demonstrated in a number of epidemiological studies and case reports, many of which associated occupational exposure to vinyl chloride to the development of angiosarcomas of the liver (U.S. EPA, 1985). Vinyl chloride has been shown to be carcinogenic in numerous animal studies. Oral administration of vinyl chloride induced liver, lung, and kidney tumors in rodents (Feron et al., 1981; Maltoni, 1977). EPA has classified vinyl chloride as a Group A chemical, human carcinogen (U.S. EPA, 1985).

SEMIVOLATILE ORGANICS

Polycyclic Aromatic Hydrocarbons (Carcinogenic)

Polycyclic aromatic hydrocarbons (PAHs) occur in the environment as complex mixtures containing numerous PAHs of varying carcinogenic potencies. Only a few components of these mixtures have been adequately characterized, and only limited information is available on the relative potencies of different compounds.

PAH absorption following oral exposure is inferred from the demonstrated toxicity of PAHs following ingestion (USEPA, 1984a). PAHs are also absorbed following dermal exposure (Kao *et al.*, 1985). Acute effects from direct contact with PAHs and related materials are limited primarily to phototoxicity; the primary effect is dermatitis (NIOSH, 1977). PAHs have also been shown to cause cytotoxicity in rapidly proliferating cells throughout the body; the hematopoietic system, lymphoid system, and testes are frequent targets (Santodonato *et al.*, 1981). Destruction of the sebaceous glands, hyperkeratosis, hyperplasia, and ulceration have been observed in mouse skin following dermal application of the cPAHs (Santodonato *et al.*, 1981). Benzo(a)pyrene has also been shown to have an immunosuppressive effect in animals (ATSDR, 2002). Nonneoplastic lesions have been observed in animals exposed to the more potent cPAHs, but only after exposure to levels well above those required to elicit a carcinogenic response. Benzo(a)pyrene has been

demonstrated to induce adverse developmental and reproductive effects in experimental animals following oral exposure (ATSDR, 2002). These effects were manifested as reduced pup weights during postnatal development, sterility, reduced fertility, and an increased incidence of stillborns and resorptions (ATSDR, 2002). cPAHs are believed to induce tumors both at the site of application and systemically. Studies in laboratory animals have demonstrated that the cPAHs benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, benzo(a)pyrene, chrysene, dibenz(a,h)anthracene and indeno(1,2,3-cd)pyrene have the ability to induce skin tumors following dermal exposure (ATSDR, 2002). Neal and Rigdon (1967) reported that oral administration of 250 ppm benzo(a)pyrene for approximately 110 days led to forestomach tumors in mice.

Benzo(a)pyrene, benzo(a)anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, and indeno(1,2,3-cd)pyrene are classified by USEPA in Group B2—Probable Human Carcinogen. USEPA has developed an oral slope factor for benzo(a)pyrene. Oral cancer slope factors for the other six cPAHs are derived by applying relative potency factors developed by USEPA (1993) to the oral slope factor for benzo(a)pyrene.

Agency for Toxic Substances and Disease Registry (ATSDR). 2002. *Toxicological profile for polycyclic aromatic hydrocarbons (PAHs)*. August 2002.

Brune, H., R.P. Deutsch-Wenzel, M. Habs, S. Ivankovic and D. Schmhl. 1981. Investigation of the tumorigenic response to benzo(a)pyrene in aqueous caffeine solution applied orally to Sprague-Dawley rats. *J. Cancer Res. Clin. Oncol.* 102:153-57.

Kao, J.K., F.K. Patterson and J. Hall. 1985. Skin penetration and metabolism of topically applied chemicals in six mammalian species including man: An *in vitro* study with benzo[a]pyrene and testosterone. *Toxicol. Appl. Pharmacol.* 81:502-516.

National Institute for Occupational Safety and Health (NIOSH). 1977. *Criteria for a Recommended Standard—Occupational Exposure to Coal Tar Products*. DHEW (NIOSH) 78-107.

Neal, J. and R.H. Rigdon. 1967. Gastric tumors in mice fed benzo(a)pyrene: A quantitative study. *Tex. Rep. Biol. Med.* 25:553-557.

Rabstein, L.S., R.L. Peters and G.H. Spahn. 1973. Spontaneous tumors and pathologic lesions in SWR/J mice. *J. Natl. Cancer Inst.* 50:751-758.

Santodonato, J., P. Howard and D. Basu. 1981. Health and ecological assessment of polynuclear aromatic hydrocarbons. *J. Environ. Pathol. Toxicol.* 5:1-364.

U.S. Environmental Protection Agency (USEPA). 1984. *Health effects assessment for polycyclic aromatic hydrocarbons (PAHs)*. Environmental Criteria and Assessment Office. EPA 540/1-86-013. September 1984.

U.S. Environmental Protection Agency (USEPA). 1993. *Provisional guidance for quantitative risk assessment of polycyclic aromatic hydrocarbons*. Office of Research and Development. EPA/600/R-93/089. July 1993.

Phenanthrene

Phenanthrene is a member of the polyaromatic hydrocarbons (PAH). PAHs constitute a class of non-polar compounds that contain two or more aromatic rings. They are ubiquitous in nature and are both naturally occurring and man-made. The database on the potential health effects of phenanthrene is limited.

Little data are available regarding the pharmacokinetics of phenanthrene. The intestinal absorption of phenanthrene is less dependent on the presence of bile in the stomach than is the absorption of the larger PAHs (such as benzo(a)pyrene) (Rahman et al, 1986).

Phenanthrene has been shown to be a skin photosensitizer in humans (Sax, 1984). Phenanthrene has a reported LD 50 of 700 mg/kg in mice (Simmon et al., 1979). Rats injected intraperitoneally evidenced liver effects (Yoshikawa et al, 1987).

There is equivocal evidence for cancer from dermal application of phenanthrene in rats (IARC, 1983). Phenanthrene is not a complete skin carcinogen (ATSDR, 2002). It is neither an initiator (LaVoie et al, 1981; Roe, 1962) nor a promoter (Roe and Grant, 1964). Higgins and Yang (1962) reported no tumor production within two months after the ingestion of 200 mg of phenanthrene by rats. There are limited data that suggest that phenanthrene is mutagenic (Wood et al., 1979). However, the majority of tests are negative (ATSDR, 2002).

Agency for Toxic Substances and Disease Registry (ATSDR) (2002) *Toxicological profile for polycyclic aromatic hydrocarbons*. U. S. Public Health Service.

Higgins, L. and Yang, Y. (1962) *Induction and extinction of mammary cancer*. Science 137:257-262.

International Agency for Research on Cancer (IARC) (1983) *Monograph on the evaluation of carcinogenic risk of chemicals to man, Phenanthrene*. 32:419-430.

LaVoie, K. et al. (1981) *Mutagenicity and tumor initiating activity and metabolism of phenanthrenes*. Cancer Res. 41:3441-3447.

Rahman, A., Barrowman, J.A., Rahimtula, A. (1986) *The influence of bile on the bioavailability of polynuclear aromatic hydrocarbons from the rat intestine*. Can J Physio Pharmacol 64:1214-1218.

Roe, F.J.C. (1962) *Effect of phenanthrene on tumour-initiation by 3,4-benzpyrene*. Br J Cancer 16:503-506.

Sax, N.I. (1984) *Dangerous Properties of Industrial Materials*. 6th edition. Van Nostrand Reinhold Company. N.Y.

Simmon, P. et al. (1979) *Mutagenic activity of chemicals carcinogens and related compounds in the intraperitoneal host-mediated assay*. J. Natl. Cancer Inst. 62:911-918.

Wood, R. et al. (1979) *Mutagenicity and tumorigenicity of phenanthrene and chrysene epoxides and diol epoxides*. Cancer Res. 39:4069-4077.

Yoshikawa, T. et al. (1987) *Toxicity of polycyclic aromatic hydrocarbons III. Effects of beta-naphthoflavone pretreatment on hepatotoxicity of compounds produced in the ozonation or NO₂-nitration of phenanthrene and pyrene by rats*. Vetern Human Toxicol. 29:113-117.

PESTICIDES AND POLYCHLORINATED BIPHENYLS

Aldrin

Aldrin is absorbed following ingestion (Farb *et al.*, 1973) and dermal exposure (Feldmann and Maibach, 1974). Aldrin is metabolically converted to dieldrin in fatty tissues (ACGIH, 1986) and both are considered to have similar chemical and toxic effects (USEPA, 1988). Acute symptoms of aldrin intoxication in humans and animals following ingestion indicate CNS stimulation manifested primarily as hyperexcitability, muscle twitching, convulsions, and depression (Borgmann *et al.*, 1952a; Hayes, 1982; Hodge *et al.*, 1967; Hoogendam *et al.*, 1962; Jager, 1970). Experimental studies indicate that dogs exposed for longer periods of time to levels as low as 1 mg/kg developed hepatic and renal toxicity (Fitzhugh *et al.*, 1964; Treon and Cleveland, 1955). Rats fed aldrin for 2 years developed hepatic lesions and nephritis at doses of 0.5 and 50 ppm, respectively (Fitzhugh *et al.*, 1964). Aldrin produced fetotoxic and/or teratogenic effects in hamsters fed a single oral dose of 50 mg/kg (approximately 84 ppm) and in mice fed a single oral dose of 25 mg/kg (approximately 6 ppm) (Ottolenghi *et al.*, 1974). Aldrin produced marked effects on fertility, gestation, viability, and lactation in mice given 25 mg/kg-day in a six-generation study (Deichmann, 1972).

Aldrin produces chromosomal aberrations in mouse, rat, and human cells and unscheduled DNA synthesis in rats and humans (Probst *et al.*, 1981). Chronic oral exposure to aldrin has produced an increase in hepatocellular tumors in mice (Davis, 1965; NCI, 1978). In contrast, chronic

feeding studies with aldrin in rats indicate that exposure was associated with nonneoplastic changes in the liver (NCI, 1978; Fitzhugh *et al.*, 1964). USEPA classified aldrin as a group B2 - Probable Human Carcinogen

Agency for Toxic Substances and Disease Registry (ATSDR). 2002. *Toxicological profile for Aldrin/Dieldrin*. April 2002.

American Conference of Governmental Industrial Hygienists (ACGIH). 1986. *Documentation of the threshold limit values and biological exposure indices*. 5th ed. Cincinnati, OH. pp. 17, 196.

Beyermann, K. and W. Eckrich. 1973. Gas-chromatographische bestimmung von insecticide-spuren in luft. *Z. Anal. Chem.* 265:4-7.

Borgmann, A., C. Kitselman, P. Dahm, J. Pankaskie and F. Dutra. 1952a. *Toxicological studies of aldrin on small laboratory animals*. Unpublished report of Kansas State College (As cited in ATSDR 2002).

Davis, L. 1965. Pathology report on mice fed dieldrin, aldrin, heptachlor, or heptachlor epoxide for two years. Internal FDA memorandum to Dr.A.J. Lehrman, July 19, 1965.

Deichmann, W. 1972. Toxicology of DDT and related chlorinated hydrocarbon pesticides. *J. Occup. Med.* 14:285.

Farb, R., T. Sanderson, B. Moore and A. Hayes. 1973. *Interaction: The effect of selected mycotoxins on the tissue distribution and retention of aldrin and dieldrin in the neonatal rat*. Paper presented at the 8th Inter-America Conference on Toxicology and Occupational Medicine.

Feldmann, R. and H. Maibach. 1974. Percutaneous penetration of some pesticides and herbicides in man. *Toxicol. Appl. Pharmacol.* 28:126-132.

Fitzhugh, O., A. Nelson and M. Quaife. 1964. Chronic oral toxicity of aldrin and dieldrin in rats and dogs. *Food Cosmet. Toxicol.* 2:551-562.

Hayes, W. 1982. *Pesticides studied in man*. Baltimore, MD: The Williams and Wilkins Co. pps. 234-247.

Hodge, H., A. Boyce, W. Deichmann and H. Kraybill. 1967. Toxicology and no-effect levels of aldrin and dieldrin. *Toxicol. Appl. Pharmacol.* 10:613-675.

Hoogendam, I., J. Versteeg and M. Devlieger. 1962. Electroencephalograms in insecticide toxicity. *Arch. Environ. Health* 4:92-100.

Jager, K. 1970. *Aldrin, dieldrin, endrin, and telodrin: An epidemiological and toxicological study of long-term occupational exposure*. New York: Elsevier Publishing Co. pp. 121-131.

National Cancer Institute (NCI). 1978. *Bioassay of aldrin and dieldrin for possible carcinogenicity*. DHEW Publication No. (NIH) 78-821. Technical Report Series No. 21.

Ottolenghi, A., J. Haseman and F. Suggs. 1974. Teratogenic effects of aldrin, dieldrin, and endrin in hamsters and mice. *Teratology* 9:11-16.

Probst, G., R. McMahon, L. Hill, D. Thompson, J. Epp and S. Neal. 1981. Chemically-induced unscheduled DNA synthesis in primary rat hepatocyte cultures: A comparison with bacterial mutagenicity using 218 chemicals. *Environ. Mutagenesis* 3:11-32.

Shell. 1984. *Review of mammalian and human toxicology, aldrin and dieldrin*. Review series HSE 84.003. Shell International Petroleum Maatschappij. B.V. The Hague.

Treon, J. and F. Cleveland. 1955. Toxicity of certain chlorinated hydrogen insecticides for laboratory animals, with special reference to aldrin and dieldrin. *Agric. Food Chem.* 3:402-408.

U.S. Environmental Protection Agency (USEPA). 1988. *Chemical profiles for extremely hazardous substances. Aldrin*. June 1988.

Benzene Hexachlorides (BHCs)

Technical-grade benzene hexachloride (BHC; also known as hexachlorocyclohexane) is composed mainly of *alpha*- (55–80%), *beta*- (5–14%), *delta*- (2–16%), *gamma*- (8–15%), and *epsilon*- (1–5%) isomers (ATSDR, 2002). BHC is absorbed by humans and animals following oral and dermal exposure (USEPA, 1985; Hayes, 1982). Absorption of the various isomers of BHC following ingestion is greater than 90% of the administered dose (Albro and Thomas, 1974). The *alpha*-, *beta*-, and *delta*-isomers of BHC primarily act as depressants of the CNS producing symptoms of tremors, prostration, and flaccidity of the entire musculature. *gamma*-BHC is a stimulant causing convulsions (Hayes, 1982). All the isomers induce hepatic enzymes (Hayes, 1982). For example, rats exhibited liver and kidney toxicity after ingesting *gamma*-BHC (1.55 mg/kg-day) for 12 weeks in the diet (Zoecon, 1983). Hepatocellular tumors have been observed in mice exposed to *alpha*- and *beta*-BHC in the diet (Ito *et al.*, 1973; Munir *et al.*, 1983; Thorpe and Walker, 1973; USEPA, 1987). The most tumorigenic isomer is *alpha*-BHC, followed by the *gamma*-, *beta*-, *delta*-, and *epsilon*-isomers (Hayes, 1982; USEPA, 1985, 1987). Various reproductive and developmental effects from exposure to *beta*- and *gamma*-BHC have been demonstrated in rodents (Hayes, 1982; USEPA, 1985).

USEPA classified both *alpha*-BHC and technical-grade BHC in Group B2 — Probable Human Carcinogens, *beta*-BHC in Group C — Possible Human Carcinogen, and *delta*-BHC in Group D — not classified as to human carcinogenicity. USEPA classified *gamma*-BHC (lindane) as a Group B2 — Probable Human Carcinogen.

Agency for Toxic Substances and Disease Registry (ATSDR). 2002. *Toxicological profile for alpha-, beta-, gamma- and delta-hexachlorocyclohexane*. May 2002.

Albro, P. and R. Thomas. 1974. Intestinal absorption of hexachlorobenzene and hexachlorocyclohexane isomers in rats. *Bull. Environ. Contam. Toxicol.* 12:289-294.

Hayes, W., Jr. 1982. *Pesticides studied in man*. Baltimore, MD: Williams and Wilkins.

Ito, N., H. Nagasaki and M. Arai. 1973. Histological and ultrastructural studies on the hepatocarcinogenicity of benzenehexachloride in mice. *J. Natl. Cancer Inst.* 51:817-826.

Munir, K.M., C.S. Soman and V. Samati. 1983. Hexachlorocyclohexane-induced tumorigenicity in mice under different experimental conditions. *Tumori.* 69:383-386.

Thorpe, E. and A.I.T. Walker. 1973. Toxicology of dieldrin (HEOD). II. Comparative long-term oral toxicity studies in mice with dieldrin, DDT, phenobarbitone, beta-HCH and gamma-HCH. *Food Cosmet. Toxicol.* 11:433-442.

U.S. Environmental Protection Agency (USEPA). 1985. *Drinking water criteria document for lindane*. Final Draft. Environmental Criteria and Assessment Office. ECAO-CIN-402. March 1985.

U.S. Environmental Protection Agency (USEPA). 1987. *Health effects profile for hexachlorocyclohexane*. Environmental Criteria and Assessment Office.

Zoecon Corporation (Zoecon). 1983. MRID No. 00128356.

Chlordane

Chlordane is a manmade pesticide used in the United States from 1948 to 1988. It was used to treat field crops and as a soil treatment to kill termites. Chlordane is not water soluble, and in soil, adsorbs strongly to the upper layers of soil especially heavy clayey soils and organic soils. Breakdown is slow; most is lost by evaporation in the first two to three days after application. However, chlordane can persist up to 20 years (ATSDR, 2002).

The effects observed in humans and animals exposed to chlordane do not appear to be route dependent. Absorption occurs readily by any route of exposure. Gastrointestinal symptoms are an early and consistent observation in acute human oral and inhalation exposure (Curley and Garrettson, 1969; Dadey and Kramer, 1953; USEPA, 1980; Olanoff *et al.*, 1983). Chlordane causes neurological effects in humans following acute or prolonged oral, inhalation, or dermal exposures. Neurological effects, such as headache, dizziness, irritability, muscle tremors, confusion, convulsions, and coma are the first signs reported. Central nervous system effects have been reported in children following oral exposure (Aldrich and Holmes, 1969). Jaundice has been

reported by persons living in homes treated with chlordane (USEPA, 1980). Subtle serum enzyme level changes were observed in pesticide application workers in Japan (Ogata and Izushi, 1991). Acute oral and parenteral studies of animals exposed to low levels of chlordane are reported to show enzyme induction, minor histochemical and histomorphological changes, and liver hypertrophy within hours of exposure (Casterline and Williams, 1971; Cram *et al.*, 1956; Den Tonkelaar and Van Esch, 1974; Hart *et al.*, 1963; Johnson *et al.*, 1986; Truhaut *et al.*, 1974, 1975).

Chlordane is classified by USEPA as Group B2 - Probable Human Carcinogen based on inadequate evidence of carcinogenicity from human studies and sufficient evidence of carcinogenicity from animal studies. USEPA developed an oral cancer slope factor based on hepatocellular carcinomas in mice.

Agency for Toxic Substances and Disease Registry (ATSDR). 2002. *Toxicological profile for chlordane*. May 2002.

Aldrich, F.D. and J.H. Holmes. 1969. Acute chlordane intoxication in a child: Case report with toxicological data. *Environ. Health* 19:129-132.

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Dieldrin

Dieldrin is a chlorinated cyclodiene insecticide that is structurally related to aldrin. Both aldrin and dieldrin are well absorbed through the lungs, skin, and gastrointestinal tract (Shell, 1984; Heath and Vanderkar, 1964; Hunter and Robinson, 1967, 1969; Sundaram *et al.*, 1978a,b; Iatropoulos *et al.*, 1975). Aldrin is metabolically converted to dieldrin in fatty tissues (ACGIH, 1986) and both are considered to have similar chemical and toxic effects (USEPA, 1988). Several human and animal studies have shown that adipose tissue is the primary storage depot for dieldrin, followed by the liver, brain, and whole blood (ATSDR, 2002). Acute symptoms of dieldrin intoxication in humans and animals following ingestion or inhalation indicate CNS stimulation manifested primarily as irritability, salivation, tremors, and convulsions. Experimental studies indicate that dogs exposed for longer periods of time to levels as low as 1 mg/kg developed hepatic and renal toxicity (Fitzhugh *et al.*, 1964; Treon and Cleveland, 1955; Walker *et al.*, 1969). Rats fed dieldrin for 2 years developed hepatic lesions and nephritis at doses of 0.5 and 50 ppm, respectively (Fitzhugh *et al.*, 1964). Dieldrin produced fetotoxic and/or teratogenic effects in hamsters fed a single oral dose of 50 mg/kg (approximately 84 ppm) and in mice fed a single oral dose of 25 mg/kg (approximately 6 ppm) (Ottolenghi *et al.*, 1974). Dieldrin produced marked effects on fertility, gestation, viability, and lactation in mice given 25 mg/kg-day in a six-generation study (Deichmann, 1972). Dieldrin produces chromosomal aberrations in mouse, rat, and human cells and unscheduled DNA synthesis in rats and humans (Probst *et al.*, 1981). Chronic oral exposure to dieldrin has produced an increase in hepatocellular tumors in mice (Davis, 1965; Epstein, 1975; NCI, 1978). In contrast, chronic feeding studies with dieldrin in rats indicate that exposure was associated with nonneoplastic changes in the liver (NCI, 1978; Fitzhugh *et al.*, 1964). Ingestion of dieldrin by laboratory animals results in a decreased immune response (Loose 1982; Loose *et al.*, 1981).

USEPA classified dieldrin as group B2 - Probable Human Carcinogen and developed an oral cancer slope factor based on the increased incidence of liver carcinoma observed in male and female C3H mice (Davis, 1965; Epstein, 1975) and in male B6C3F1 mice (NCI, 1978).

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4,4'-DDT, 4,4'-DDE, 4,4'-DDD

DDT (1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane) was a chemical widely used to control insects on agricultural crops and insects that carry diseases like malaria and typhus. Technical grade DDT is a mixture of three forms, 4,4'-DDT (85%), 2,4'-DDT (15%), and 2,2'-DDT (trace amounts) (ATSDR, 2002). All of these are white, crystalline, tasteless, and almost odorless solids. Also, DDE (1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene) and DDD (1,1-dichloro-2,2-bis(p-chlorophenyl)ethane) sometimes contaminate technical grade DDT. DDD was also used to kill pests; one form of DDD (2,4'-DDD) has been used medically to treat cancer of the adrenal gland (ATSDR, 2002). DDT is no longer used as a pesticide in the United States except in cases of public health emergency. The most prevalent isomers for DDT, DDE, or DDD in the environment are the 4,4'-isomers (ATSDR, 2002).

DDT is absorbed by humans and experimental animals from the gastrointestinal tract (USEPA, 1984, 1980). Jenson et al. (1957) reported that 95% of ingested DDT in rats is absorbed from the gastrointestinal tract. Absorption of DDT through the skin is minimal (USEPA, 1980). In humans, DDT and its metabolites, DDD and DDE, are stored primarily in adipose tissue; storage

of DDT in human tissues can last up to 20 years (NIOSH, 1978). Acute oral exposure to DDT in humans and animals may cause dizziness, confusion, tremors, convulsions, and paresthesia of the extremities. Allergic reactions in humans following dermal exposure to DDT have also been reported (USEPA, 1980). Long-term occupational exposure to DDT results in increased activity in hepatic microsomal enzymes, increased serum concentrations of enzymes and cholesterol, decreased serum concentrations of creatinine phosphokinase, increased blood pressure, and increased frequency of miscarriages (NIOSH, 1978). Blood, kidney, liver and neurological effects, immunosuppression, reduced fertility, embryotoxicity, and fetotoxicity have also been reported in animals following subchronic and chronic exposure to DDT (ATSDR, 2002; Laug *et al.*, 1950; NIOSH, 1978; McLachlan and Dixon, 1972; Schmidt, 1973). For example, monkeys subchronically exposed to 50 mg/kg-day DDT exhibited loss of equilibrium and rats chronically exposed to 16 mg/kg-day DDT exhibited tremors by week 26 (ATSDR, 2002). In addition, rats exposed, in a two-generation feeding study, to 0.35 mg/kg-day DDT had decreased fertility (Green, 1969). DDT has been shown to be carcinogenic in mice and rats at several dose levels or dosage regimens. The principal site of action is the liver, but an increased incidence of tumors of the lung and lymphatic system have also been reported in several investigations (NIOSH, 1978; Tomatis *et al.*, 1974; NCI, 1978).

4,4'-DDT, 4,4'-DDD, and 4,4'-DDE are classified by USEPA in Group B2 - Probable Human Carcinogen based on inadequate evidence of carcinogenicity from human studies and sufficient evidence of carcinogenicity from animal studies. For 4,4'-DDT, USEPA developed an oral cancer slope factor based on a number of carcinogenicity studies. USEPA developed an oral cancer slope factor for 4,4'-DDD based on an increased incidence of lung tumors in male and female mice, liver tumors in male mice, and thyroid tumors in male and female rats. USEPA developed an oral cancer slope factor for 4,4'-DDE based on an increased incidence of liver tumors in two strains of mice and hamsters, and thyroid tumors in male and female rats by diet.

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Heptachlor Epoxide

Heptachlor epoxide is a contaminant and metabolite of the insecticide, heptachlor. Heptachlor is readily absorbed from the gastrointestinal tract following oral exposure (ATSDR, 2002). Acute symptoms due to heptachlor exposure in humans include irritability, excessive salivation, labored respiration, muscle tremors, and convulsions (USEPA, 1987). Acute exposure of animals to heptachlor and heptachlor epoxide produced tremors, convulsions, paralysis, and hypothermia (USEPA, 1985). Chronic exposure of experimental animals to dietary concentrations of heptachlor or heptachlor epoxide has been associated with increased liver weight and hepatocellular carcinoma; heptachlor also induced hepatic lesions (USEPA, 1987; Velsicol, 1955; Dow Chemical, 1955; Davis, 1965; Epstein, 1976; NCI, 1977; Velsicol, 1973). In the presence of metabolic activation, both heptachlor and heptachlor epoxide induced unscheduled DNA synthesis in transformed human fibroblasts (Ahmed *et al.*, 1977). Heptachlor also increased the frequency of chromosomal aberrations in bone marrow cells of mice (Markarjan, 1966). Results of studies with rodents also indicate that heptachlor epoxide induces reproductive and developmental effects (USEPA, 1987).

Heptachlor epoxide is classified as Group B2 - Probable Human Carcinogens based on sufficient evidence of carcinogenicity in animal studies and inadequate evidence of carcinogenicity in humans. Using experiments in which mice exposed to dietary concentrations of heptachlor epoxide exhibited hepatocellular carcinomas (Davis, 1965; NCI, 1977; Velsicol, 1973), USEPA estimated an oral cancer slope factor for heptachlor epoxide.

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Polychlorinated Biphenyls (PCBs)

Polychlorinated biphenyls (PCBs) are complex mixtures of chlorinated biphenyls. There are 209 individual PCB congeners which comprise environmental and commercial mixtures of PCBs to varying degrees. The commercial PCB mixtures that were manufactured in the United States were given the trade name of "Aroclor." Aroclors are distinguished by a four-digit number (for example, Aroclor-1260). The last two digits in the Aroclor 1200 series represent the average percentage by weight of chlorine in the product. Each Aroclor contains numerous congeners; for example, Aroclor-1260 contains 80 individual congeners when analyzed by high resolution chromatography (Safe *et al.*, 1987). Not all of the congeners are equally toxic. In general, coplaner PCB molecules which are sterically similar to 2,3,7,8-tetrachloro-dibenzodioxin (TCDD) (3,3',4,4',5-penta-CB, 3,3',4,4',5,5'-hexa-CB and 3,3',4,4'-tetra-CB), exhibit the highest toxicity in laboratory animals (Kamrin and Fischer, 1991). The toxicity of an environmental mixture of

PCBs will largely be determined by the quantities of the highly toxic congeners that are present in the mixture.

PCBs in pure form are readily and extensively absorbed through the gastrointestinal tract and somewhat less readily through the skin; PCBs are presumably readily absorbed from the lungs, but few data are available that experimentally define the extent of absorption after inhalation (USEPA, 1985). Studies have found oral absorption efficiency on the order of 75% to >90% in rats, monkeys and ferrets (Albro and Fishbein, 1972; Allen *et al.*, 1974; Tanabe *et al.*, 1981; Bleavens *et al.*, 1984; Clevenger *et al.*, 1989). PCBs distribute preferentially to adipose tissue and concentrate in human breast milk due to its high fat content (ATSDR, 2002). The binding of PCBs to a soil or sediment matrix inhibits absorption by all routes (ATSDR, 2002).

Dermatitis and chloracne (a potentially disfiguring and long-term skin disease) have been the most prominent and consistent findings in studies of occupational exposure to PCBs. Several studies examining liver function in exposed humans have reported disturbances in blood levels of liver enzymes. Reduced birth weights, slow weight gain, reduced gestational ages, and behavioral deficits in infants were reported in a study of women who had consumed PCB-contaminated fish from Lake Michigan (USEPA, 1985). Reproductive, developmental, hepatic, immunotoxic, and immunosuppressive effects appear to be the most sensitive end points of PCB toxicity in nonrodent species, and the liver appears to be the most sensitive target organ for toxicity in rodents (USEPA, 1985). For example, adult monkeys exposed to dietary concentrations of 0.028 mg/kg-day Aroclor-1016 for approximately 22 months showed no evidence of overt toxicity; however, the offspring of these monkeys exhibited decreased birth weight and possible neurological impairment (Barsotti and Van Miller, 1984; Levin *et al.*, 1988; Schantz *et al.*, 1989, 1991).

A number of studies have suggested that PCB mixtures are capable of increasing the frequency of tumors including liver tumors in animals exposed to the mixtures for long periods (Kimbrough *et al.*, 1975; NCI, 1978; Schaeffer *et al.*, 1984; Norback and Weltman, 1985). In addition, studies have suggested that PCB mixtures can act to promote or inhibit the action of other carcinogens in rats and mice (USEPA, 1985). It is known that PCB congeners vary greatly in their potency in producing biological effects, such as cancer; however, USEPA generally considers Aroclor-1260 to be the Aroclor with the greatest tumorigenic potential and, therefore, conservatively uses this Aroclor to be representative of all PCB mixtures for the evaluation of carcinogenic effects. Nevertheless, USEPA has acknowledged that there is some evidence that mixtures containing highly chlorinated biphenyls are more potent inducers of hepatocellular carcinoma in rats than are mixtures containing less chlorine by weight following oral exposure. The responses are mostly limited to the livers in rats and mice, although there is a suggestion that some PCB mixtures may also affect the stomach of rats and monkeys (Chase *et al.*, 1989). Statistically significant increases in malignant tumors have not been observed in animal studies with PCB mixture containing less than 60 percent chlorine content (Chase *et al.*, 1989). There is some suggestive evidence that Aroclor-1254 induces hepatocellular adenomas and carcinomas combined in male rats based on the reclassification and reevaluation of the NCI (1978) tumor data conducted by Ward (1985).

However, the majority of tumors were benign (statistically significant alone), while the few malignant tumors (carcinomas) were not statistically elevated by themselves. At present, there is uncertainty as to whether or not Aroclor-1248, -1242, or -1232 are tumorigenic in animals. This is because there are no valid cancer bioassays for these mixtures (Chase *et al.*, 1989).

Existing epidemiological data do not indicate a consistent tumorigenic effect among individuals exposed to PCBs. ATSDR (2002) concluded that occupational studies involving predominantly inhalation and dermal exposures to PCBs have suggested an association between the development of liver, gastrointestinal, hematopoietic and skin cancer and PCB exposure. However, the majority of these studies were mortality studies that reported nonstatistically significant results, were confounded by concurrent exposure to other chemicals (many of which are considered to be potential carcinogens), had small sample sizes or number of deaths, or unquantified PCBs exposures. In addition, there is no consistent pattern of associations among the various studies, either with respect to type of human cancers observed or the nature and extent of PCB exposures.

USEPA classifies PCBs as Group B2 - Probable Human Carcinogens based on sufficient evidence in animal bioassays and inadequate evidence from studies in humans. USEPA recently revised the oral slope factor for PCBs to multiple possible slope factors corresponding to three different tiers. The appropriate tier for use depends on the level of risk and likely persistence of the congeners evaluated. The top tier, for "high risk and persistence," is considered most appropriate at this site. The criteria for use of this tier, suggested by USEPA, are as follows: (1) food chain exposures; (2) sediment or soil ingestion; (3) dust or aerosol inhalation; (4) dermal exposure, if an absorption factor has been applied; (5) presence of dioxin-like, tumor-promoting, or persistent congeners; and (6) early-life exposures. Dose-response data were generated based on the incidence of liver hepatocellular adenomas, carcinomas, cholangiomas, or cholangiocarcinomas in female Sprague-Dawley rats exposed to Aroclor-1260, -1254, -1242, and -1016 separately in one study (Brunner *et al.*, 1996) and only Aroclor-1260 in another study (Norback and Weltman, 1985).

Agency for Toxic Substances and Disease Registry (ATSDR). 2002. *Toxicological profile for polychlorinated biphenyls*. August 2002.

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INORGANICS

Antimony

Antimony is a metal which occurs both in the trivalent and pentavalent oxidation states (USEPA, 1980). Absorption of this metal via oral routes of exposure is low (10% for antimony, tartrate; 1% for all other forms) (ATSDR, 2002). Organic antimony is more toxic than the inorganic compounds due to increased absorption. Humans and animals exposed acutely by oral or inhalation exposures to either the trivalent or pentavalent forms of antimony displayed electrocardiogram (ECG) changes and myocardial lesions (USEPA, 1980). Pneumoconiosis has been observed in humans exposed by acute inhalation and dermatitis has occurred in individuals

exposed either orally or dermally. Following acute oral exposure to antimony trioxide or potassium antimony tartrate, both humans and laboratory animals (dogs) manifested nausea and vomiting (ATSDR, 2002). Humans and laboratory animals (i.e., rat and pig) chronically exposed to antimony compounds (antimony trioxide, pentoxide, and trisulfide) via inhalation manifested respiratory effects including macrophage proliferation, fibrosis and pneumonia at LOAELs ranging from 0.046 to 86.3 mg/m³ (ATSDR, 2002). Chronic oral exposure in rats (0.35 mg/kg-day) resulted in altered blood glucose and blood cholesterol levels and decreased lifespan (Schroeder *et al.*, 1970). A single report (Balyeava, 1967) noted an increase in spontaneous abortions, premature births, and gynecological problems in 318 female workers exposed to a mixture of antimony metal, antimony trioxide, and antimony pentasulfide dusts. No change in the incidence of cancer was observed in laboratory animals (i.e., rats, mice) fed 0.262 or 0.35 mg/kg-day antimony as potassium antimony tartrate for a lifetime.

Agency for Toxic Substances and Disease Registry (ATSDR). 2002. *Toxicological profile for antimony*. October 2002.

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Arsenic

Arsenic is difficult to characterize as a single analyte because it has complex chemistry. It may be trivalent or pentavalent and is widely distributed in nature (ATSDR, 2002). Both inorganic and organic forms of arsenic are readily absorbed via oral and inhalation routes. Soluble forms are more readily absorbed than insoluble forms (USEPA, 1984). Approximately 95% of soluble inorganic arsenic administered to rats is absorbed from the gastrointestinal tract (Coulson *et al.*, 1935; Ray-Bettley and O'Shea, 1975). Approximately 70–80% of arsenic deposited in the respiratory tract of humans has been shown to be absorbed (Holland *et al.*, 1959). Dermal absorption of the insoluble forms of arsenic is not significant (USEPA, 1984). At mining sites, arsenic is expected to occur in naturally occurring mineral assemblages with considerably lower bioavailability than expected in soluble inorganic arsenic salts (Davis *et al.*, 1992).

Acute exposure in humans by ingestion of metallic arsenic has been associated with gastrointestinal effects, hemolysis, and neuropathy (USEPA, 1984). Chronic human arsenicism (by drinking water ingestion) is associated with increased risk of nonmelanoma, typically nonlethal, skin cancer and a peripheral vascular disorder that results in gangrene of the extremities, especially feet, known as blackfoot disease (Tseng, 1977). Additionally, there is strong evidence to suggest ingested inorganic arsenic causes cancers of the bladder, kidney, lung,

and liver, and possibly other sites (Bates *et al.*, 1992; Chen *et al.*, 1992; Chen *et al.*, 1986). It is well known that hyperpigmentation and keratosis are also associated with chronic arsenicism (Neubauer, 1947) and arsenic can produce toxic effects on both the peripheral and CNS, precancerous dermal lesions, and cardiovascular damage (USEPA, 1984; Tseng, 1977). Arsenic is embryotoxic, fetotoxic, and teratogenic in several animal species (USEPA, 1984). No evidence of reproductive toxicity was found (Calabrese and Kenyon, 1991). Epidemiological studies of workers in smelters and in plants manufacturing arsenical pesticides have shown inhalation of arsenic is strongly associated with lung cancer and less so, with hepatic angiosarcoma (USEPA, 1984).

There is substantial evidence that establishes the nutritional essentiality of trace levels of arsenic. Deficiency has been shown to depress growth and impair reproduction in rats, minipigs, chickens, and goats (USEPA, 1988; NRC, 1989). Methylation of arsenic to less toxic, more rapidly excreted chemical species provides an effective detoxification mechanism *in vivo*. In humans, this system may become saturated at daily oral intake rates greater than 250–1,000 µg/day. For this reason, the dose-response curve for arsenic, for carcinogenicity and systemic toxicity, may have nonlinearities, i.e., a portion of the dose-response curve exists over which increases in dose do not result in comparable increases in physiological response (Petito and Beck, 1990).

USEPA classified arsenic as Group A - Human Carcinogen and derived an oral cancer slope factor based on two epidemiological studies (Tseng *et al.*, 1968; Tseng, 1977) which indicated an increased incidence of skin cancer in individuals exposed to arsenic in drinking water.

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Barium

The soluble salts of barium, an alkaline earth metal, are toxic in mammalian systems. They are absorbed rapidly from the gastrointestinal tract and are deposited in the muscles, lungs, and bone. Barium is excreted primarily in the feces.

At low doses, barium acts as a muscle stimulant and at higher doses affects the nervous system eventually leading to paralysis. Acute and subchronic oral doses of barium cause vomiting and diarrhea, followed by decreased heart rate and elevated blood pressure. Higher doses result in cardiac irregularities, weakness, tremors, anxiety, and dyspnea. A drop in serum potassium may account for some of the symptoms. Death can occur from cardiac and respiratory failure. Acute doses around 800 milligrams can be fatal to humans.

Subchronic and chronic oral or inhalation exposure primarily affects the cardiovascular system resulting in elevated blood pressure. A lowest-observed-adverse-effect level (LOAEL) of 0.51 mg barium/kg/day based on increased blood pressure was observed in chronic oral rat studies (Perry et al. 1983), whereas human studies identified a no-observed-adverse-effect level (NOAEL) of 0.21 mg barium/kg/day (Wones et al. 1990, Brenniman and Levy 1984). In the Wones et al. study, human volunteers were given barium up to 10 mg/L in drinking water for 10 weeks. No clinically significant effects were observed. An epidemiological study was conducted by Brenniman and Levy in which human populations ingesting 2 to 10 mg/L of barium in drinking water were compared to a population ingesting 0 to 0.2 mg/L. No significant individual differences were seen; however, a significantly higher mortality rate from all combined cardiovascular diseases was observed with the higher barium level in the 65+ age group. The average barium concentration was 7.3 mg/L, which corresponds to a dose of 0.20 mg/kg/day.

Subchronic and chronic inhalation exposure of human populations to barium-containing dust can result in a benign pneumoconiosis called “baritosis.” This condition is often accompanied by an elevated blood pressure but does not result in a change in pulmonary function. Exposure to an air concentration of 5.2 mg barium carbonate/m³ for 4 hours/day for 6 months has been reported to result in elevated blood pressure and decreased body weight gain in rats (Tarasenko et al. 1977). Reproduction and developmental effects were also observed. Increased fetal mortality was seen after untreated females were mated with males exposed to 5.2 mg/m³ of barium carbonate. Similar results were obtained with female rats treated with 13.4 mg barium carbonate/m³.

Barium has not been evaluated by the USEPA for evidence of human carcinogenic potential

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Cadmium

Gastrointestinal absorption of cadmium in humans ranges from 5 to 6% (USEPA, 1985a). Based on a comprehensive model for inhaled cadmium, the deposition rate of particulate airborne

cadmium is 5–50% (i.e., 5% of particles greater than 10 microns and up to 50% of particles less than 0.1 microns), and 50–100% of the cadmium deposited was absorbed (Nordberg *et al.*, 1985). Cadmium bioaccumulates in humans, particularly in the kidney and liver (USEPA, 1985a,b). Acute oral exposure to cadmium in laboratory animals resulted in systemic, immunological, neurological, developmental, and reproductive effects at doses of 2–138 mg/kg-day (ATSDR, 2002). Chronic oral or inhalation exposure of humans to cadmium has been associated with renal dysfunction, itai-itai disease (bone damage), hypertension, anemia, endocrine alterations, and immunosuppression. Renal toxicity occurs in humans chronically exposed to cadmium in food at LOAEL of 0.0075 mg/kg-day. In laboratory animals (i.e., rat, mouse) chronic oral exposure to cadmium results in increased blood pressure, hematological, and renal effects at LOAELs ranging from 0.014 to 57 mg/kg-day (ATSDR, 2002). Teratogenic and reproductive effects (i.e., decreased fetal and birth weight, delayed ossification, behavioral impairment, and reduced fertility) were reported in laboratory animals (i.e., rat, mice, dogs) subchronically exposed to cadmium in drinking water at LOAELs ranging from 0.04 to 40 mg/kg-day (ATSDR, 2002). Epidemiological studies have demonstrated a strong association between inhalation exposure to cadmium and cancers of the lung, kidney, and prostate (USEPA, 1985b; Thun *et al.*, 1985). In experimental animals, cadmium induces injection-site sarcomas and testicular tumors. When administered by inhalation, cadmium chloride is a potent pulmonary carcinogen in rats. Cadmium is a well-documented animal teratogen (USEPA, 1985b).

USEPA classified cadmium as Group B1 - Probable Human Carcinogen by inhalation. This classification applies to agents for which there is limited evidence of carcinogenicity in humans from epidemiologic studies.

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Chromium

Chromium exists in two states, as chromium (III) and as chromium (VI). Following oral exposure, absorption of chromium (III) has been reported to be 0.4% while absorption of chromium (VI) has been observed to be as high as 10% (ATSDR, 2002). However, chromium (VI) is rapidly reduced to chromium (III) after penetration of biological membranes and in the gastric environment (ATSDR, 2002). Chromium is an essential micronutrient and is not toxic in trace quantities (USEPA, 1980).

Alterations in liver enzyme activities were noted in rats administered an oral dose of 13.5 mg/kg-day chromium (VI) for 20 days (Kumar *et al.*, 1985). Rats subchronically administered higher concentrations of chromium VI (98 mg/kg-day) have exhibited adverse effects on renal function (Diaz-Mayans *et al.*, 1986). No significant changes, however, were detected in the livers or kidneys of rats exposed to 2.7 mg/kg-day or 3.5 mg/kg-day chromium (III) or chromium (VI), respectively, in the drinking water for 1 year (MacKenzie *et al.*, 1958; ATSDR, 2002). CNS effects including hypoactivity have been reported in rats when exposed to subchronic levels of 98 mg/kg-day chromium VI in drinking water (Diaz-Mayans *et al.*, 1986).

Workers exposed to 2 µg/m³ chromic acid vapors (mean duration of 2.5 years), a soluble chromium (VI) compound, exhibited atrophy and ulceration of the nasal mucosa and transient decrease in lung function (Lindberg and Hedenstierna, 1983). There is, however, insufficient scientific evidence that chromium (III) compounds by themselves elicit atrophy of the nasal mucosa or adverse respiratory effects in humans (ATSDR, 2002). Furthermore, epidemiological studies of worker populations have clearly established that inhaled chromium (VI) is a human carcinogen; the respiratory passages and the lungs are the target organs (Mancuso, 1975; USEPA, 1984).

Inhalation of chromium (III) or ingestion of chromium (VI) or (III) has not been associated with carcinogenicity in humans or experimental animals (USEPA, 1984). Oral exposure of pregnant mice (gestational days, 1 to 19) to 57 mg/kg-day chromium (VI) resulted in embryo-lethal effects (e.g., increased resorptions and postimplantation loss), reduced ossification and gross anomalies (Trivedi *et al.*, 1989). Chromium (III) does not appear to cause fetotoxic or teratogenic effects in rats (ATSDR, 2002). Reproductive effects in the form of decreased sperm count were noted in mice administered oral doses of 4.6 mg/kg-day chromium (VI) (225 ppm) and 3.5 mg/kg-day chromium (III) (172 ppm) for 7 weeks (Zahid *et al.*, 1990).

USEPA classified inhaled chromium (VI) in Group A—Human Carcinogen by the inhalation route. Inhaled chromium (III) and ingested chromium (III) and (VI) have not been classified with respect to carcinogenicity. Because carcinogenicity by the oral route of exposure can not be determined, chromium is classified as Group D for the oral exposure route.

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Copper

Copper is a reddish metal that occurs naturally in rock, soil, water, sediment, and air. Its average concentration in the earth's crust is about 50 parts copper per million parts soil. Copper also occurs naturally in plants and animals. It is an essential element for all known living organisms including humans and other animals.

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 adeno virus 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.

Hematological effects in workers employed in a copper processing factory have been reported by Finelli et al. (1981). However, interpretation of the study results is limited by the finding of elevated iron, lead, and cadmium in hair samples of exposed workers.

Metal fume fever, has been reported in factory workers exposed to copper dust or fumes (Armstrong et al. 1983; Gleason 1968; Stokinger 1981).

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).

Bionetics Research Labs (1968) studied the carcinogenicity of a copper-containing compound, copper hydroxyquinoline, in two strains of mice (B6C3F1 and B6AKF1). Groups of 18 male and 18 female 7-day-old mice were administered 1000 mg copper hydroxyquinoline/kg bw (180.6 mg Cu/kg) suspended in 0.5% gelatin daily until they were 28 days old, after which they were administered 2800 ppm (505.6 ppm Cu) in the feed for 50 additional weeks. No statistically significant increases in tumor incidence were observed in the treated 78-week-old animals. In the same study, Bionetics Research Labs (1968) administered a single subcutaneous injection of gelatin (control) or 1000 mg of copper hydroxyquinoline/kg bw (180.6 mg Cu/kg) suspended in 0.5% gelatin to groups of 28-day-old mice of both strains. After 50 days of observation, the male B6C3F1 had an increased incidence of reticulum cell sarcomas compared with controls. No tumors were observed in the treated male B6AKF1 mice, and a low incidence of reticulum cell sarcomas was observed in the treated female mice of both strains.

Gilman (1962) administered intramuscular injections containing 20 mg of cupric oxide (16 mg Cu), cupric sulfide (13.3 mg Cu), and cuprous sulfide (16 mg Cu) into the left and right thighs of 2- to 3-month-old Wistar rats. After 20 months of observations, no injection-site tumors were observed in any animals, but other tumors were observed at very low incidence in the animals

receiving cupric sulfide (2/30) and cuprous sulfide (1/30). As the relevance of the organic copper compound to the observation of sarcoma induction is uncertain and the incidence of tumors in rats treated i.m. with inorganic copper was very low, data are considered inadequate for classification.

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Lead

Lead is used extensively in the manufacture of storage batteries and was used in gasoline and paint. Lead is also a natural constituent of many soils, for which concentrations normally range from 10 to 30 mg lead per kilogram of soil (USEPA, 1980).

Lead can be absorbed by the oral, inhalation or dermal exposure routes (see section on Relative Absorption Factors). Gastrointestinal absorption of lead varies considerably depending upon chemical form, dietary intake, and age (Forbes and Reina, 1974; Barltrop and Meek, 1975). The deposition and absorption of inhaled lead depends upon particle size, chemical form and the rate and depth of breathing (Randall et al., 1975; Nozaki, 1966; Chamberlain et al., 1975). Once absorbed, lead is distributed to the various organs of the body, with most distribution occurring into mineralized tissues (ATSDR, 2002). Placental transfer to the developing fetus is possible (Bellinger et al., 1987). Inorganic lead is not known to be biotransformed within the body. Absorbed lead is excreted via the urinary or fecal routes (ATSDR, 2002)

Cases of acute lead poisoning in humans are not common and have not been studied in experimental animals as thoroughly as chronic lead poisoning. Symptoms of acute lead poisoning from deliberate ingestion by humans may include vomiting, abdominal pain, hemolysis, liver damage, and reversible tubular necrosis (USEPA, 1984). Subacute exposures in humans reportedly may produce a variety of neurological effects including dullness, restlessness, irritability, poor attention span, headaches, muscular tremor, hallucinations, and loss of memory. Nortier et al., (1980) report encephalopathy and renal damage to be the most serious complications of chronic toxicity in man and the hematopoietic system to be the most sensitive. For this reason, most data on the effects of lead exposure in humans are based upon blood lead levels. The effects of lead on the formation of hemoglobin and other hemoproteins, causing decreased levels, are reportedly detectable at lower levels of lead exposure than in any other organ system (Betts et al., 1973). Peripheral nerve dysfunction is observed in adults at levels of 30 to 50 mg/dL-blood. Children's nervous systems are reported to be affected at levels of 15 mg/dL-blood and higher (Benignus et al., 1981). In high doses, lead compounds may potentially cause abortions, premature delivery, and early membrane rupture (Rom, 1976).

Acute oral lethal doses of lead in animals depend upon chemical form, but generally range from 500 to 30,000 mg/kg. Several reproduction studies on the effects of subchronic oral exposure to lead in rats have been conducted (Kimmel et al., 1976; Grant et al., 1980; Fowler et al., 1980). These studies report that lead acetate administered in drinking water at various concentrations caused depressed body weights at 50 and 250 mg-Pb/L water, histological changes in the kidneys of offspring, cytotokaryomegaly of the tubular epithelial cells of the inner cortex at concentrations greater than or equal to 25 mg/L and postnatal developmental delays at 50 to 250 mg/L. Higher oral doses of lead may result in decreased fertility and fetotoxic effects in a variety of species (Hilderbrand et al., 1973). A reduction in the number of offspring of rats and mice exposed to 25 mg Pb/L drinking water with a chromium deficient diet was reported by Schroeder et al. (1970). Chronic oral exposure of female Long-Evans rats to lead (5 mg/PB/L-water) reportedly resulted in slight effects on tissue excitability, systolic blood pressure, and cardiac ATP concentrations (Kopp et al., 1980a,b).

Results of *in vitro* studies with human lymphocyte cultures using lead acetate were nearly equally positive and negative. Results of *in vivo* tests are also contradictory but suggest that lead may have an effect on chromosomes (sister chromatid exchange). Results for gene mutations, DNA modification, and recombinations in various microorganisms using lead acetate, lead nitrate and lead chloride were consistently negative with or without metabolic activation. Lead chloride has been reported to inhibit both DNA and RNA synthesis. In *in vitro* mammalian test systems, lead acetate gave conflicting results.

No epidemiological data regarding the oral carcinogenic potential of lead could be located in the available literature. Chronic inhalation may result in a statistically significant increase in deaths due to tumors in the digestive organs and respiratory systems in lead smelter workers and battery plant workers (Kang et al., 1980). Several studies have reported tumor formation in experimental animals orally administered specific lead salts, not normally ingested by humans (Zawirska and Medras, 1972; Boyland et al., 1962; Ito, 1973). The carcinogenicity of inhaled lead in experimental animals could not be located in the available literature. The USEPA has classified lead and lead compounds as Group B2 - Probable Human Carcinogens. Because the carcinogenic potential of lead appears to be weak, USEPA bases risk management decisions for this compound on neurodevelopmental effects rather than carcinogenicity.

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Manganese

Manganese is considered to be among the least toxic of the trace metals and, in fact, is considered to be an essential element (NRC, 1989). The oral absorption of dietary manganese ranges from 3 to 10%. However, manganese is absorbed to a greater extent following inhalation exposures. The National Research Council has established a provisional recommended dietary allowance for adults of 2 to 5 mg/day (NRC, 1989).

The effects following acute exposure to manganese are unknown. Chronic occupational exposure to manganese dust (0.02–2.6 mg/m³) has been associated with respiratory symptoms and pneumonitis (Chandra *et al.*, 1981) and higher levels have been associated with a condition known as manganism, a progressive neurological disease characterized by speech disturbances, tremors, and difficulties in walking. For example, male workers exposed to manganese dioxide, tetroxide and various salts (TWA of total airborne manganese dust ranged from 0.07 to 8.61 mg/m³) experienced an increased incidence of psychomotor disturbances (e.g., reaction time, hand-eye coordination and hand steadiness) (Roels *et al.*, 1987). Other effects observed in humans occupationally exposed to manganese dust include hematological (Chandra *et al.*, 1981; Flinn *et al.*, 1941; Kesic and Hausler, 1954), cardiovascular (Saric and Hrustic, 1975) and reproductive effects (Cook *et al.*, 1974; Emara *et al.*, 1971; Lauwerys *et al.*, 1985; Rodier, 1955). In adults, a safe intake of manganese from dietary sources ranges from 2 to 10 mg/day (10 mg/day = 0.14 mg/kg-day) (WHO, 1973; NRC, 1989; Schroeder *et al.*, 1966). Individuals who chronically ingested drinking water from natural wells containing manganese concentrations of

1,600–2,300 :g/L (0.06 mg/kg-day), showed a statistically significant increase in minor neurologic effects (neurologic exam scores) (Kondakis *et al.*, 1989). The dietary intake of manganese was unaccounted for in this study, and therefore, USEPA withdrew its previous assessment that used this study to determine a quantitative dose-response relationship for manganese in drinking water. Higher concentrations in drinking water (0.8 mg/kg-day) have resulted in symptoms including lethargy, increased muscle tonus, tremor and mental disturbances (Kawamura *et al.*, 1941).

Chronic oral exposure of rats to manganese chloride can also result in CNS dysfunction (Leung *et al.*, 1981; Lai *et al.*, 1982). Chronic inhalation exposure of experimental animals (monkeys, rats, mice, hamsters) has resulted in respiratory effects; however, other studies have demonstrated that these effects may be immunological in origin (ATSDR, 2002). Manganese has not been reported to be teratogenic; however, this metal has been observed to cause depressed reproductive performance and reduced fertility in humans and experimental animals (USEPA, 1984a).

Certain manganese compounds have been shown to be mutagenic in a variety of bacterial tests. Manganese chloride and potassium permanganate can cause chromosomal aberrations in mouse mammary carcinoma cells. Manganese was moderately effective in enhancing viral transformation of Syrian hamster embryo cells (USEPA, 1984a,b). USEPA established a weight-of-evidence classification for manganese of D (not classifiable as to human carcinogenicity).

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Mercury

In humans, inorganic mercury is absorbed following inhalation and oral exposure; however, only 7–15% of administered inorganic mercury is absorbed following oral exposure (USEPA, 1984; Rahola *et al.*, 1971; Task Group on Metal Accumulation, 1973; ATSDR, 2002). Organic mercury is almost completely absorbed from the gastrointestinal tract and is assumed to be well absorbed via inhalation in humans (USEPA, 1984).

A primary target organ for inorganic compounds is the kidney. Acute and chronic exposures of humans to inorganic mercury compounds have been associated with anuria, polyuria, proteinuria, and renal lesions (Goyer, 1996). Chronic occupational exposure of workers to elemental mercury vapors (0.026–0.2 mg/m³) has been associated with mental disturbances, tremors, and gingivitis (USEPA, 1984; ATSDR, 2002). Animals exposed to inorganic mercury for 12 weeks have exhibited proteinuria, nephrotic syndrome and renal disease (Druet *et al.*, 1978). Rats chronically administered inorganic mercury (as mercuric acetate) in their diet for 2 years exhibited a dose-related increase in glomerular nephritis at concentrations as low as 1.27 mg/kg-day (Fitzhugh *et al.*, 1950).

The CNS is a major target for organic mercury compounds. Adverse effects in humans, resulting from subchronic and chronic oral exposures to organic mercury compounds, have included destruction of cortical cerebral neurons, damage to Purkinje cells, and lesions of the cerebellum. Clinical symptoms following exposure to organic mercury compounds have included paresthesia, loss of sensation in extremities, ataxia, and hearing and visual impairment (WHO, 1976; ATSDR, 2002). Adverse kidney effects are also prominent in animals following chronic ingestion of organic mercury (0.5 ppm phenyl mercuric acetate or 0.015 mg Hg/kg-day) (Fitzhugh *et al.*, 1950). Embryotoxic and teratogenic effects, including malformations of the skeletal and genitourinary systems, have been observed in animals exposed orally to organic mercury (USEPA, 1984).

Both organic and inorganic compounds are reported to be genotoxic in eukaryotic systems (Leonard *et al.*, 1984). Elevated incidence of fetal resorption was observed in hamsters exposed to 31.4 mg/kg-day inorganic mercury (Gale, 1974). There is evidence to suggest methylmercury chloride induces renal tumors, mostly adenocarcinomas in two strains of male mice (ICR and B6C3F1) (Hirano *et al.*, 1986; Mitsumori *et al.*, 1981, 1990). However, monkeys, cats and rats chronically administered methyl mercury in the diet did not develop an elevated tumor incidence (Ikeda *et al.*, 1973; Charbonneau *et al.*, 1976; Vershuuren *et al.*, 1976). Furthermore, elevated cancer incidence has not been reported in humans who ingested methylmercury-contaminated fish in the Minamata area of Japan (Katsuna, 1968) or in humans who ingested methylmercury fungicide-treated grains in Iraq and were followed for 13 years (Greenwood, 1985).

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Nickel

Nickel in the ambient atmosphere typically exists as a constituent of suspended particulate matter (U.S. EPA, 1985). The greatest volume of nickel emitted into the atmosphere is the result of fossil fuel combustion. Other sources of nickel emissions are primary production, incinerators, metallurgy, chemical manufacturing, cement manufacturing, coke ovens, nickel recovery, asbestos mining/milling and cooling towers.

Studies of nickel absorption have shown that it is absorbed by all routes of exposure to varying degrees, primarily dependent on the chemical form (see section on Relative Absorption Factors). Absorbed nickel is bound to serum components and distributed to body organs, reaching highest concentrations in kidney and lung tissue (Whanger, 1973). Nickel is not known to be biotransformed. Excretion of absorbed nickel is primarily through urine, with minor excretory routes through hair and sweat (ATSDR, 2002).

Nickel carbonyl $\text{Ni}(\text{CO})_4$ is a particularly toxic form of nickel upon inhalation and causes chest pain, dry coughing, hyperpnea, cyanosis, occasional gastrointestinal symptoms, sweating, visual

disturbances and severe weakness. This is often followed by pulmonary hemorrhage, edema and cellular derangement. Survivors may be left with pulmonary fibrosis. In the workplace, nickel dermatitis may result at high nickel concentrations. At lower concentrations some susceptible individuals develop eczema-like lesions. The threshold for these health effects is much greater than exposures which occur in the ambient environment. The major adverse effects of nickel in man are dermatitis, chemical pneumonitis, and lung and nasal cancers.

Deaths occurred in rats and mice at concentrations greater than 3.3 and 1.7 mg/m³ nickel, respectively, upon extended inhalation exposure to NiSO₄ (Dunnick et al., 1987). Mice exposed to Ni₃S₂ died due to necrotizing pneumonia at 7.3 mg/m³ nickel (Benson et al., 1987). Prolonged exposure of hamsters to nickel oxide at 41.7 mg/m³ resulted in decreased survival due to emphysema (Wehner et al., 1975). Oral LD₅₀s in rats vary depending upon the nickel-containing compound to which the rats were exposed. These range from 355 mg compound/kg (118 mg Ni/kg) for nickel acetate (Haro, 1968) to greater than 5000 mg compound/kg for nickel oxide, nickel sulfide, and nickel subsulfide (Mastromatteo, 1986). Rats fed diets containing nickel sulfate hexahydrate at 0, 250, 500 and 1000 ppm nickel showed no adverse effects over three generations in fertility, gestation, viability or lactation.

Weak evidence exists for the mutagenicity of nickel in bacterial and mammalian cells. Nickel appears to induce chromosomal aberrations in cultured mammalian cells (Larramendy et al., 1981), but not in vivo (Waksvik and Boysen, 1982). Occupational studies of human exposure indicate that certain nickel compounds appear to be carcinogenic via inhalation. However, there is no evidence of carcinogenicity in mammals through ingestion or dermal exposure (U.S. EPA, 1985). Nickel subsulfide has been found to be carcinogenic via the inhalation route in rats (Ottolenghi et al., 1974). Studies on nickel exposure via the oral route are inadequate to reach conclusions on carcinogenicity (ATSDR, 2002).

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Selenium

Human and animal data suggest that many chemical forms of selenium produce similar effects. Selenium is known to be an essential micronutrient for humans and animals; therefore, inadequate as well as excessive selenium intake can cause negative health effects (ATSDR, 2002). One proposed mechanism of intermediate and chronic toxicity for selenium compounds is that under conditions of excess body levels of selenium, selenium atoms begin to replace the sulfur atoms in structural and enzymatic proteins (Shamberger, 1970), destroying the protein structural and functional integrity. This mechanism of action is unlikely to be organ specific; therefore, under this proposed mechanism, toxic levels of selenium would be expected to affect multiple organ systems. Furthermore, differential sensitivities of the various organ systems to selenium exposure would be expected on the basis of differential accumulation or retention of selenium compounds (Goyer, 1996).

The primary target organ in humans and in animals upon acute exposure to high concentrations of selenium by inhalation or oral routes is the lung, with cardiovascular, hepatic, and renal systems also affected. Lesser effects are observed in all other organ systems except the musculoskeletal system. The liver is the primary target organ for the oral toxicity of sodium selenite, sodium selenate, and organic forms of selenium in animals following intermediate and chronic exposure. In humans, liver cirrhosis or dysfunction are the result of chronic selenosis (ATSDR, 2002). Endocrine effects were found following intermediate oral exposure. Following chronic oral

exposure to selenium compounds, the primary effects in humans are dermal and neurological. As evidenced by populations in China, chronic exposure to high selenium levels in the diet can cause diseased nails and skin as well as hair loss. Higher levels can cause neurological problems including unsteady gait and paralysis. However, studies of populations living in areas of naturally occurring high selenium concentrations in the United States have not revealed adverse health effects in those populations (Yang *et al.*, 1989a,b). Following intermediate and chronic oral exposure to selenium compounds, the primary effects in livestock exposed to naturally occurring selenium in range plants are also dermal and neurological. Studies in animals with high selenium concentrations demonstrate that many organ systems retain selenium and are affected. The primary effects in laboratory animals exposed to inorganic selenium salts or to selenium-containing amino acids are cardiovascular, gastrointestinal, hematological, hepatic, dermal, immunological, neurological, and reproductive (ATSDR, 2002). Selenium is a teratogen in birds. However, studies of Chinese populations and laboratory animals have not found evidence of teratogenic effects in mammals (ATSDR, 2002).

USEPA has determined that selenium is not classifiable as to human carcinogenicity (Class D).

Agency for Toxic Substances and Disease Registry (ATSDR). 2002. *Toxicological profile for selenium*. August 2002.

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Thallium

Thallium and its salts are readily and rapidly absorbed through the skin, lungs, and mucous membranes of the mouth and gastrointestinal tract (ATSDR, 2002). Percutaneous absorption has also been reported to occur through rubber gloves (Rumack, 1986).

Thallium is acutely toxic to humans regardless of the chemical form of the compound or route of administration. Hundreds of cases of thallotoxicosis due to ingestion of thallium-based pesticides have been reported (ACGIH, 1986). Children poisoned by thallium ingestion have exhibited neurological abnormalities including mental retardation and psychoses (ACGIH, 1986). The effects of thallium toxicity are similar in humans and animals. The most commonly noted response to thallium exposure is alopecia, but neurological and gastrointestinal findings are frequently found. Such effects include ataxia, lethargy, painful extremities, peripheral neuropathies, convulsions, endocrine disorders, psychoses, nausea, vomiting, and abdominal pains (Bank, 1980). It has been noted that the degree and duration of exposure to thallium and its salts can influence the clinical picture of thallium intoxication. Subchronic feeding studies conducted with rats observed marked growth depression and a nearly complete loss of hair (USEPA, 1986; Clayton and Clayton, 1981).

Exposure to thallium salts during critical developmental stages in chicks and rats has been reported to be associated with the induction of adverse developmental outcomes (Karnofsky *et al.*, 1950). Pre- and postnatally exposed rat pups have exhibited hydronephrosis, fetal weight reduction and growth retardation (Clayton and Clayton, 1981; Gibson and Becker, 1970). Thallium has also been shown to cross the placenta and, presumably, enter the fetal blood system (Clayton and Clayton, 1981).

Thallium has not been demonstrated to be carcinogenic in humans or experimental animals and may have some antitumor activity (Clayton and Clayton, 1981).

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Vanadium

The absorption of vanadium through the gastrointestinal tract of animals is low (2.6% for vanadium pentoxide in rats) (Conklin *et al.*, 1982). Soluble vanadium compounds that are inhaled and deposited are readily absorbed (50–100%) (ATSDR, 2002). Because vanadium has low solubility, its absorption through skin is thought to be quite low, although no specific studies were located regarding dermal absorption (ATSDR, 2002).

Pentavalent vanadium compounds are generally considered to be more toxic than other valence states. Many incidents of short-term and long-term occupational exposures to vanadium, mainly vanadium pentoxide dust, have been reported. Inhalation causes respiratory tract irritation, coughing, wheezing, labored breathing, bronchitis, chest pains, eye and skin irritation and discoloration of the tongue (NIOSH, 1977; NAS, 1974). Humans subchronically exposed to vanadium pentoxide (0.1 mg/m³) via inhalation experienced respiratory irritation (Zenz and Berg, 1967). Experimental animals (i.e., rats, monkeys) subchronically exposed to vanadium compounds (vanadium pentoxide, bismuth orthovanadate) manifested alveolar proteinosis and increased pulmonary resistance at concentrations of 2.5–4.7 mg/m³ (Lee and Gillies, 1986; Knecht *et al.*, 1985). Effects seen in experimental animals following chronic inhalation exposure include fatty degeneration of the liver and kidneys, hemorrhage, and bone marrow changes (Browning, 1969).

Humans subchronically exposed to ammonium vanadyl tartrate (1.3 mg/kg-day) via capsules did not manifest any adverse effects (Dimond *et al.*, 1963). However, experimental animals (i.e., rats, mice) orally exposed to vanadium compounds (sodium metavanadate, sodium orthovanadate, ammonium metavanadate) exhibited mild systemic effects (decreased weight gain, vascular infiltration, spleen hypertrophy and increased ventricular pressure) at doses as low as 0.57 mg/kg-day (ATSDR, 2002). Rats chronically administered 0.77 mg/kg-day (5 ppm) vanadium in their drinking water showed no adverse effects (Schroeder *et al.*, 1970). Pre- and postnatally exposed rat pups have exhibited reduced pup weight and length and facial hemorrhage (ATSDR, 2002). Vanadium has not been demonstrated to be carcinogenic in humans or experimental animals.

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Zinc

Zinc is absorbed in humans following oral exposure (approximately 20–30%) (ATSDR, 2002); however, insufficient data are available to evaluate absorption following inhalation exposure (USEPA, 1984). Zinc is an essential trace element that is necessary for normal health and metabolism and therefore is nontoxic in trace quantities (Goyer, 1996). The National Research Council (NRC) recommends a dietary allowance of 10–15 mg/day for adults (NRC, 1989).

Exposure to zinc at concentrations that exceed recommended levels, however, has been associated with a variety of adverse effects. In humans, acute inhalation exposure to relatively high levels of zinc has been associated with gastrointestinal disturbances, dermatitis, and metal fume fever, a condition characterized by chest pain, cough, and dyspnea, as well as impaired pulmonary function characterized by reduced lung volumes (ATSDR, 2002).

Eighteen healthy women given supplements of zinc gluconate (1 mg/kg-day) for 10 weeks developed slight alterations in blood chemistry (decreased enzyme levels) (Yadrick *et al.*, 1989). Chronic oral exposure of humans to zinc (2 mg/kg-day) may cause decreased red blood cell count (Hale *et al.*, 1988). Experimental animals (rats, rabbits, mice) administered zinc in the diet (68–1,110 mg/kg-day) for durations up to 1 year manifested blood, liver, renal, and reproductive effects (ATSDR, 2002). An increased incidence of fetal resorption was noted in pregnant rats administered 200 mg/kg-day (Schlicker and Cox, 1968). In addition, increased preimplantation loss was observed in rats fed the same concentration for 18 days (Pal and Pal, 1987). There is no evidence that zinc is carcinogenic (ATSDR, 2002).

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TABLE C.8-1. ABSOLUTE ORAL BIOAVAILABILITY FACTORS

Chemical	Absolute Oral Bioavailability Factor	Reference
Antimony	0.15	ATSDR, 2002
Cadmium	0.01	McLellan <i>et al.</i> , 1978
Chromium	0.013	Donaldson and Barreras, 1996
Manganese	0.04	Davidson <i>et al.</i> , 1989
Mercury (inorganic)	0.07	USEPA, 2004
Vanadium	0.026	Conklin <i>et al.</i> , 1982

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APPENDIX C.9

RELATIVE BIOAVAILABILITY OF ARSENIC IN SEDIMENTS FROM THE ABERJONA RIVER

**RELATIVE BIOAVAILABILITY OF ARSENIC
IN SEDIMENTS FROM THE ABERJONA RIVER**

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- Margaret E. Dunsmore, BS, helped with all aspects of animal handling and dosing, as well as urine collection and sample preparation.
- Dr. John Drexler at the University of Colorado, Boulder, performed the characterization of the sediment samples and test materials, including *in vitro* testing of bioaccessibility and electron microprobe and particle size analyses of the test materials.
- Dr. Edward Hinderberger of L.E.T., Inc., Columbia, Missouri, provided prompt and reliable chemical analysis of all of the samples for total arsenic concentrations.

EXECUTIVE SUMMARY

The gastrointestinal absorption of arsenic from two composite sediment samples collected from the banks of the Aberjona River was measured using young swine. Groups of animals (four animals per dose group) were given oral doses of a reference material (sodium arsenate) or site sediment twice a day for 12 days. Urine excreted by each animal was collected on days 6/7, 8/9 and 10/11. The urinary excretion fraction (UEF) (the ratio of the amount excreted per 48 hours divided by the dose given per 48 hours) was calculated for sodium arsenate and each test material using linear regression analysis. The relative bioavailability (RBA) of arsenic in a test material compared to that in sodium arsenate was calculated as:

$$RBA = \frac{UEF(\text{test material})}{UEF(\text{sodium arsenate})}$$

The results are summarized below:

Test Material	Description	Arsenic Conc. (ppm)	Relative Bioavailability	
			Best Est.	90% CI
TM1	Composite sample of three sediments with arsenic concentrations greater than 500 ppm	676	37%	32% – 41%
TM2	Composite sample of three sediments with arsenic concentrations of 180-460 ppm	313	51%	46% – 56%

These data indicate that arsenic in site sediments is absorbed less extensively than arsenic in drinking water. Use of these site-specific data is likely to improve the accuracy of risk estimates for humans who may be exposed to the sediments.

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RELATIVE BIOAVAILABILITY OF ARSENIC IN ABERJONA RIVER SEDIMENTS

1.0 INTRODUCTION

Accurate assessment of the health risks resulting from oral exposure to any chemical frequently requires knowledge of the amount of the chemical absorbed from the gastrointestinal tract into the body. This information on absorption may be described either in absolute or relative terms:

Absolute Bioavailability (ABA) is the ratio of the amount of chemical absorbed compared to the amount of chemical ingested:

$$ABA = \frac{\text{Absorbed Dose}}{\text{Ingested Dose}}$$

This ratio is also referred to as the oral absorption fraction (AF_0).

Relative Bioavailability (RBA) is the ratio of the absolute bioavailability of some test material compared to the absolute bioavailability of some appropriate reference material, usually the chemical dissolved in water or some fully soluble form that completely dissolves when ingested:

$$RBA = \frac{ABA(\text{test material})}{ABA(\text{reference material})}$$

For example, if 100 ug of arsenic dissolved in drinking water were ingested and a total of 90 ug entered the body, the ABA would be 0.90 (90%). Likewise, if 100 ug of arsenic contained in soil were ingested and 30 ug entered the body, the ABA for soil would be 0.30 (30%). If the arsenic dissolved in water was used as the reference substance for describing the relative amount of arsenic absorbed from soil, the RBA would be $0.30/0.90 = 0.33$ (33%).

Using Relative Bioavailability Data to Improve Risk Calculations for Arsenic

When reliable data are available on the relative bioavailability of arsenic in a site medium (e.g., soil, sediment), this information can be used to improve the accuracy of exposure and risk calculations for that medium at that site as follows:

$$RfD(\text{adjusted}) = \frac{RfD(IRIS)}{RBA}$$

$$SF(\text{adjusted}) = SF(IRIS) \cdot RBA$$

Alternatively, it is also acceptable to adjust the dose (rather than the toxicity factors) as follows:

$$Dose(adjusted) = Dose(default) \cdot RBA$$

This adjustment in dose is mathematically equivalent to adjusting the toxicity factors as described above.

Purpose of This Study

USEPA Region 1 is currently investigating potential human health risks from arsenic in sediment samples from along the Aberjona River and associated wetlands and floodplain areas. This study was performed to obtain site-specific data on the relative bioavailability of arsenic in sediment samples from the site in order to improve accuracy and decrease uncertainty in human health risk evaluations.

2.0 STUDY DESIGN

This investigation of arsenic relative bioavailability was performed according to the basic design presented in Table 2-1. As shown, the study investigated arsenic absorption from sodium arsenate (the reference material) and from two site-specific sediments, each administered to groups of animals at three different dose levels for 12 days. All doses were administered orally.

2.1 Test Materials

2.1.1 Preliminary Characterization of Site Sediment Samples

Preparation of the two test materials for this study began by collecting 12 sediment samples from multiple locations along the Aberjona River. Each of these samples was characterized in order to support decisions as to which samples should be selected for use as dose material in the animal study, as well as to answer questions about how the dose material should be prepared and administered. Figure 2-1 is a flow chart that summarizes this characterization process.

Sample Description

The sampling locations of the 12 sediment samples span four basic regions of the Aberjona River. Sediment samples 1-3 were collected from the Halls Brook Holding Area, samples 4-6 were collected from the Wells G&H 38-acre Wetland, samples 7-9 were collected from the Cranberry Bog, and samples 10-12 were from Davidson Park. Samples were selected to cover a range of arsenic concentrations in sediments, and were also selected to provide reasonable spatial representativeness across the site.

Sample Preparation

One portion of each of the 12 samples was coarse-sieved through a 1 cm screen to remove large debris (sticks, leaf matter, stones, etc.). This screening was performed on the moist (un-dried) samples. A portion of this coarse-sieved material was removed for arsenic analysis, and a second portion was removed for *in vitro* bioaccessibility analysis (see below). The remaining portion was air dried and fine-sieved (using a 2 mm screen). This step was performed because it is considered probable that the fine-grained portion of the sediment is more likely to adhere to skin and be ingested by humans than the coarse-grained fraction.

Arsenic Concentration

The concentration of arsenic was measured in both the coarse- and fine-sieved samples by inductively coupled plasma atomic absorption spectrometry (ICP-AES). The results from these analyses are shown below:

River Segment	Sample	Arsenic Concentration (ppm)	
		Fine-sieved	Coarse-sieved
Halls Brook Holding Area	1	459	583
	2	527	590
	3	144	269
Wells G&H Wetland	4	145	411
	5	775	605
	6	176	156
Cranberry Bog	7	301	315
	8	832	560
	9	407	388
Davidson Park	10	43.4	37.0
	11	64.0	91.8
	12	67.1	74.9

As seen, the concentration of arsenic in the sediment samples is quite variable, both within a segment of river and between segments. In general, the concentration of arsenic in coarse-sieved and fine-sieved material tends to be similar (Figure 2-2). Thus, RBA results based on tests using fine-sieved material can be extrapolated to samples for which only bulk sample results are available.

In Vitro Bioaccessibility

In vivo absorption of arsenic from a solid medium such as sediment depends on the rate and extent to which arsenic dissolves from the solid medium into the fluids of the gastrointestinal tract. Dr. John Drexler at the University of Colorado has developed a standard procedure to measure the amount of arsenic that dissolves from a test material into a fluid that is similar to the gastric fluid of humans. The amount of arsenic that solubilizes in this test after a specified period of time (usually one hour) is referred to as the *in vitro* bioaccessibility (IVBA), and this value may be used as a preliminary qualitative indicator of potential *in vivo* RBA.

Figure 2-3 shows the IVBA for each of the 12 dried and fine-sieved sediment samples from the site. As seen, there is a range of values, and the IVBA appears to be inversely correlated with concentration (i.e., the most concentrated samples tend to have the lowest *in vitro* bioaccessibility, while the least concentrated samples tend to have the highest *in vitro* bioaccessibility). The basis for this apparent relationship is not known.

Effect of Drying

Each of the sediment samples collected in the field contained considerable moisture content. *A priori*, it was considered possible that drying the samples might alter (increase) the binding of

arsenic to the sediment particles, potentially resulting in a change (decrease) in bioavailability. In order to investigate this possibility, the IVBA of the dried and un-dried samples were compared. Because the moist, un-dried material could not be effectively sieved through the 2mm screen, the moist sample was selected manually to include as few coarse particles as possible. The results are shown in the following table and in Figure 2-4:

River Segment	Sample	In Vitro Bioaccessibility of Arsenic (%)	
		Dry	Moist (Un-dried)
Halls Brook Holding Area	1	40	2
	2	31	5
	3	70	5
Wells G&H Wetland	4	40	26
	5	12	16
	6	55	9
Cranberry Bog	7	37	12
	8	13	12
	9	15	13
Davidson Park	10	39	53
	11	49	53
	12	59	9
Average		38	18

As seen, drying the moist material does not appear to significantly influence the IVBA for some samples, and tends to increase rather than decrease the IVBA for other samples. The basis for this apparent change in IVBA is not known, but the results suggest that dried sediment will be as bioavailable or more bioavailable than un-dried sediments. On this basis, it was decided that the *in vivo* test of RBA would be performed using the dried materials.

Evaluation of Methyl Arsenic

Studies at other sites (e.g., Sanders et al. 1994) have revealed that arsenic in sediments may become methylated by microbial action at times when the oxygen tension in the sediments is low. Because methylated forms of arsenic might have different bioavailability (and different toxicity) than the inorganic forms, aliquots of the dried fine-sieved samples were analyzed for organic methyl arsenic. Samples were sent to West Coast Analytical Services, where they were extracted with carbonate buffer and analyzed for As+3, As+5, MMA, and DMA by ion chromatography-ICPMS. The results are summarized below:

Sample	Total Arsenic (ppm)		Extracted Arsenic (WCAS) (ppm)			
	WCAS	Drexler	As+3	DMA	MMA	As+5
1	630	459	ND	ND	ND	20
2	600	527	ND	ND	ND	ND
3	168	144	ND	ND	ND	ND
4	169	145	ND	ND	ND	ND
5	670	775	ND	ND	ND	ND
6	167	176	ND	ND	ND	ND
7	292	301	ND	ND	ND	ND
8	520	832	ND	ND	ND	ND
9	296	407	ND	ND	ND	ND
10	51	43.4	ND	ND	ND	ND
11	87	64	ND	ND	ND	10
12	83	67.1	ND	ND	ND	11
Detection Limit (ppm)	1		5	5	5	5

WCAS = West Coast Analytical Services

As seen, very low levels were observed for each analyte. Recovery of matrix spikes for As+3 and As+5 was poor, suggesting that recoveries of these species may be low. However, recovery of matrix spikes of MMA and DMA were high (89%). These results indicate that if MMA or DMA are present in the samples, they constitute only a very small fraction of the total arsenic.

Mineral Phase Speciation

Each of the 12 dried fine-sieved samples was characterized by electron microprobe analysis (EMPA) in order to provide preliminary data on the identity and relative abundance of the different mineral forms of arsenic present in the samples. The results are summarized in Table 2-2. As seen, these data suggest that arsenic exists mainly in association with particles of iron oxide, iron sulfate, and zinc-iron sulfate. The preliminary data are too limited to draw firm conclusions, but suggest that the presence of iron oxide is associated with higher arsenic concentrations and lower *in vitro* bioaccessibility, and that the presence of the iron-zinc sulfate complexes is associated with lower arsenic concentrations and higher *in vitro* bioaccessibility.

2.1.2 Test Material Selection and Preparation

Test materials for use in the *in vivo* study were selected by considering the results of the preliminary characterization of 12 site sediment samples (Section 2.1.1, above). Specifically, factors that were considered included the concentration level of arsenic in a sample and the degree to which different samples appear to be similar or dissimilar based on speciation and *in vitro* bioaccessibility testing. Based on the conclusion that the only clear pattern of difference among samples is the *in vitro* bioaccessibility (inversely related to concentration), three test materials were prepared by compositing samples with similar arsenic concentrations, as described below.

Test Material 1

Test Material 1 was prepared by compositing equal masses of dried fine-sieved material from samples 2, 5, and 8. These three samples were selected because they have the highest measured arsenic concentration values (all >500 ppm) and they tend to have low bioaccessibility (average = 19%). In addition, the three samples represent each of the three reaches of river (excluding the Davidson Park area), providing good spatial representativeness. These samples tend to be relatively enriched in the iron oxide form of arsenic.

Test Material 2

Test Material 2 was prepared by compositing equal masses of dried fine-sieved material from samples 1, 6, and 7. These three samples were selected because they have intermediate arsenic concentration values (180-460 ppm), intermediate bioaccessibility values (average = 44%), and represent each of the three upstream reaches of the river. These samples tend to be relatively enriched in the zinc-iron sulfate form of arsenic.

Test Material 3

Test Material 3 was prepared by compositing equal masses of all samples with an arsenic concentration less than 150 ppm (samples 3, 4, 10, 11, and 12). These are the samples with the highest apparent bioaccessibility (average = 51%), but the arsenic levels are too low (average = 93 ppm) to permit effective testing in animals. Although Test Material 3 was not used in the *in vivo* portion of the study, it underwent all of the same detailed characterization efforts as Test Materials 1 and 2.

Test Material Preparation

Each test material was prepared by combining equal masses of the appropriate sediment samples, as indicated above. The samples for a given test material were composited using a stainless steel bowl and mixing spoon, and characterized as detailed below.

2.1.3 Detailed Characterization of Test Materials

Arsenic Concentration

After compositing, the concentration of arsenic in each test material was measured by ICP/AES and by ICP/MS. The results are shown below:

Analytical Method	Arsenic Concentration (mg/kg)		
	TM1	TM2	TM3
ICP/MS	590	290	80
ICP/MS	652	318	93.6
ICP/AES	733	319	--
ICP/AES	730	324	--
Average	676.3	312.8	86.8
Standard Deviation	68.6	15.4	9.6

-- = Not measured

Concentration of Other Inorganics, Organic Carbon, and Sulfide

Each sample was analyzed for EPA's Target Analyte List (TAL) of inorganic chemicals, as well as for total organic content (TOC) and total sulfide content. Results are shown in Table 2-3.

Particle Speciation, Size, and Matrix Association

Each test material was characterized by electron microprobe analysis (EMPA) in order to identify the different mineral forms of arsenic that were present in the sample and to estimate how much of the total arsenic was present in each form. In addition, the size distribution of the particles was characterized along with the matrix association of each particle. The detailed data are presented in Appendix A and the results are summarized below.

Arsenic Phases

Speciation of the three test materials indicated that the arsenic in these samples is associated with four different types of mineral phase: iron oxide, iron pyrite, iron sulfate, and zinc sulfate. Estimates of the relative arsenic mass (an approximation of the fraction of the total arsenic present in each phase) are presented below:

Arsenic Speciation Data

Test Material	Number of Particles Counted	Relative Arsenic Mass			
		Iron Oxide	Pyrite	Iron Sulfate	Zinc Sulfate
TM1	186	69%	0%	29%	2%
TM2	123	16%	2%	27%	55%
TM3	57	24%	1%	59%	16%

As seen, arsenic is primarily associated with iron oxide in TM1, with zinc sulfate in TM2, and iron sulfate in TM3. These differences in mineral phase may influence the RBA of the arsenic in the materials.

It is important to note that these quantitative estimates of relative arsenic mass are based on examination of a limited number of arsenic-bearing particles in each sample (N = 57 to 186). Consequently, the quantitative values reported should not be considered to be highly precise, and apparent differences between samples may be partly due to random variation in the analysis rather than authentic differences in composition.

Particle Size Distribution

Particle size is a potentially important contributor to RBA because the fraction of a particle that undergoes dissolution in gastrointestinal fluids is likely related to the surface area to volume ratio (this ratio is larger for small particles than large particles). The distribution of particle sizes for arsenic-bearing grains in these test materials is summarized below:

Particle Size Distribution			
Test Material	Percent of Particles by Size Class		
	≤25 um	26-100 um	>100 um
TM1	79%	15%	6%
TM2	85%	14%	2%
TM3	72%	26%	2%

As seen above, in these test materials, a large majority of all arsenic-containing particles are small: an average of 79% of all particles are 25 um or less in size. This predominance of small particles may tend to increase the RBA compared to what would be expected for larger particles of similar composition.

Matrix Association

Arsenic-containing particles may be characterized according to their association with other particles into four types, as follows:

Matrix Association	Description
Liberated	A grain of arsenic-containing material that is not attached to or contained within any other particle
Rimming	Arsenic is present on the outer surface of a particle, usually as a consequence of adsorption or precipitation
Cemented	The arsenic-containing particle is loosely bound to or associated with other particles or phases that do not contain arsenic
Included	The arsenic-containing particle is entirely contained within another particle

In the first three types of matrix association, the arsenic is exposed at the surface of some or all of the particle, and hence the arsenic is available to be dissolved by gastrointestinal fluids. Particles that are fully included in other particles are not exposed to external fluids and are not likely to have high bioavailability. The distribution of matrix associations for arsenic-bearing particles in the test materials from this site is summarized below:

Particle Matrix Associations

Test Material	Percent of Particles by Matrix Class			
	Liberated	Rimming	Cemented	Included
TM1	27%	2%	67%	4%
TM2	22%	0%	78%	0%
TM3	37%	11%	53%	0%

As seen, relative few particles are fully included, and 96-100% of the particles are entirely or partially exposed to external fluids. This suggests that the RBA of the arsenic is likely to be determined primarily by mineral phase and/or particle size rather than by matrix association.

In Vitro Bioaccessibility

The details of the method used to measure the *in vitro* bioaccessibility of arsenic are described in USEPA (1999). In brief, 1.00 g of test substrate is placed into a 125-mL wide-mouth HDPE bottle. To this is added 100 mL of the extraction fluid (0.4 M glycine, pH 1.5). Each bottle is placed into a heated water bath (water temperature = 37°C) and rotated end-over-end. After a specified period of time (1, 2 or 4 hours), the bottles are removed, dried, and placed upright on the bench top to allow the soil to settle to the bottom. A 15-mL sample of supernatant fluid is removed directly from the extraction bottle into a disposable 20-cc syringe. After withdrawal of the sample into the syringe, a Luer-Lok attachment fitted with a 0.45-µm cellulose acetate disk filter (25 mm diameter) is attached, and the 15 mL aliquot of fluid is filtered through the attachment to remove any particulate matter. This filtered sample of extraction fluid is then analyzed for arsenic. The fraction of arsenic originally present in the sample that occurs in the dissolved phase at the end of the extraction procedure is the *in vitro* bioaccessibility (IVBA). IVBA results for the three test materials in this study are summarized below:

Test Material	Concentration (ppm)	IVBA		
		1 hr.	2 hr.	4 hr.
TM1	676	14%	16%	19%
TM2	313	35%	47%	51%
TM3	86.8	49%	57%	66%

As seen, IVBA values tend to increase slowly as a function of extraction time. In all cases, an inverse relationship is observed between IVBA and arsenic concentration in the sediment

sample, similar to the pattern that was observed previously during the preliminary characterization of the 12 site sediments samples (see Section 2.1, above).

2.2 Experimental Animals

Young swine were selected for use in these studies because they are considered to be a good physiological model for gastrointestinal absorption in children (Weis and LaVelle 1991). The animals were intact males of the Pig Improvement Corporation (PIC) genetically defined Line 26, and were purchased from Chinn Farms, Clarence, MO.

The animals were housed in individual stainless steel cages. All animals were held for several days prior to beginning exposure to test materials in order to allow them to adapt to their new environment and to ensure that all of the animals were healthy. Animals were assigned to dose groups at random. When exposure began (day zero), the animals were about 6 weeks old and weighed an average of about 12.1 kg. Animals were weighed every three days during the course of the study. On average, animals gained about 0.4 kg/day and the rate of weight gain was comparable in all groups, ranging from 0.38 to 0.46 kg/day. These body weight data are summarized in Figure 2-5.

2.3 Diet

Animals provided by the supplier were weaned onto standard pig chow purchased from MFA Inc., Columbia, MO. In order to minimize arsenic exposure from the diet, the animals were gradually transitioned from the MFA feed to a special feed (Zeigler Brothers, Inc., Gardners, PA) over the time interval from day -7 to day -3, and this feed was then maintained for the duration of the study. The feed was nutritionally complete and met all requirements of the National Institutes of Health-National Research Council. The typical nutritional components and chemical analysis of the feed is presented in Table 2-4. Each day every animal was given an amount of feed equal to 5% of the mean body weight of all animals on study. Feed was administered in two equal portions of 2.5% of the mean body weight at each feeding. Feed was provided at 11:00 AM and 5:00 PM daily. Previous analysis of feed samples indicated the arsenic level was generally below the detection limit (0.1 ppm), which corresponds to a dose contribution from food of less than 5 ug/kg-day (less than 50 ug/day).

Drinking water was provided *ad libitum* via self-activated watering nozzles within each cage. Previous analysis of samples from randomly selected drinking water nozzles indicated the arsenic concentration was less than the quantitation limit (about 1 ug/L). Assuming water intake of about 0.1 L/kg-day, this corresponds to a dose contribution from water of less than 0.1 ug/kg-day (1 ug/day).

2.4 Dosing

Animals were exposed to sodium arsenate (abbreviated in this report as "NaAs") or a test material (site sediment) for 12 days, with the dose for each day being administered in two equal portions given at 9:00 AM and 3:00 PM (two hours before feeding). Dose material was placed in the

center of a small portion (about 5 grams) of moistened feed (this is referred to as a “doughball”), and this was administered to the animals by hand.

The dose levels administered were based on the arsenic content of the test material, with target doses of 300, 600, and 900 ug/day for NaAs and each test material. The mass of each test material needed to provide these doses of arsenic were calculated based on a preliminary estimation of the arsenic concentration in the test materials. Actual administered arsenic doses were re-calculated after the study was completed using the mean of two ICP-AES measurements and two ICP-MS measurements. These actual administered doses are presented in Appendix B.

2.5 Collection and Preparation of Samples

Urine

Samples of urine were collected from each animal for three consecutive 48-hour periods, on days 6/7, 8/9 and 10/11 of the study. Collection began at 9AM and ended 48 hours later. The urine was collected in a stainless steel pan placed beneath each cage, which drained into a plastic storage bottle. Each collection pan was fitted with a nylon screen to minimize contamination with feces, spilled food, or other debris. Plastic diverters were used to minimize urine dilution with drinking water spilled by the animals from the watering nozzle into the collection pan, although this was not always effective in preventing dilution of the urine with water. Due to the length of the collection period, collection containers were emptied at least twice daily into a separate holding container. This ensured that there was no loss of sample due to overflow.

At the end of each collection period, the urine volume was measured and 60-mL portions were removed for analysis. A separate 250-mL aliquot was retained as an archive sample. Each sample was acidified by the addition of concentrated nitric acid. The samples were stored refrigerated until arsenic analysis.

2.6 Arsenic Analysis

Urine samples were assigned random sample numbers and submitted to the laboratory for analysis in a blind fashion. Details of urine sample preparation and analysis are provided in USEPA (1999). In brief, 25 mL samples of urine were digested by refluxing and then heating to dryness in the presence of magnesium nitrate and concentrated nitric acid. Following magnesium nitrate digestion, samples were transferred to a muffle furnace and ashed at 500°C. The digested and ashed residue was dissolved in hydrochloric acid and analyzed by the hydride generation technique using a Perkin-Elmer 3100 atomic absorption spectrometer. Preliminary tests of this method established that each of the different forms of arsenic that may occur in urine, including trivalent inorganic arsenic (As+3), pentavalent inorganic arsenic (As+5), monomethyl arsenic (MMA) and dimethyl arsenic (DMA), are all recovered with high efficiency.

Laboratory Quality Assurance

A number of quality assurance steps were taken during this project to evaluate the accuracy of the analytical procedures. Steps performed by the analytical laboratory included:

Spike Recovery

Randomly selected samples were spiked with known amounts of arsenic (usually 40 ug, as sodium arsenate) and the recovery of the added arsenic was measured. Recovery for individual samples ranged from 95% to 110%, with an average across all analyses of $103 \pm 4.5\%$ (N = 7).

Duplicate Analysis

Random samples were selected for duplicate analysis by the laboratory analyst. Duplicate results had a relative percent difference (RPD) of 0-17%, with an average of $2.6 \pm 5.0\%$ (N = 13).

Laboratory Control Standards

Four different types of laboratory control standards (LCS) were tested periodically during the analysis. These are samples for which a certified concentration of arsenic has been established. Results for these four types of LCS are summarized below:

LCS Type	Certified Value	Average Recovery	SEM	N
E.R.A. P081 - Metals WasteWatR	366 ng/mL	97%	1.7%	42
N.R.C.C. Dolt-2 Dogfish Liver	16.6 +/- 1.1 ug/g dry wt	84%	0.0%	2
N.R.C.C. Tort-2 Lobster	21.6 +/- 1.8 ug/g dry wt	99%	3.3%	3
N.I.S.T. Oyster 1566b	7.65 +/- 0.65 ug/g dry wt	97%	0.8%	3

As seen, recovery of arsenic from these standards was good in all cases, and no samples were outside the acceptance criteria specified by the suppliers.

Blanks

Blank samples run along with each batch of samples never yielded a measurable level of arsenic, with all values being reported as less than 0.03 ug of arsenic.

Blind Quality Assurance Samples

In addition to these laboratory-sponsored QA samples, an additional series of QA samples were submitted to the laboratory in a blind fashion. This included a number of Performance Evaluation (PE) samples (urines of known arsenic concentration) and a number of blind duplicates.

The results for the PE samples are shown in Figure 2-6. As seen, the PE samples included several different concentrations each of four different types of arsenic (As+3, As+5, MMA, and DMA). In all cases, there was good recovery of the arsenic.

The results for blind duplicates are shown in Figure 2-7. As seen, there was good agreement between results for the duplicate pairs.

Based on the results of all of the quality assurance samples and steps described above, it is concluded that the analytical results for samples of urine are of high quality and are suitable for derivation of reliable estimates of arsenic absorption from test materials.

3.0 DATA ANALYSIS

Figure 3-1 shows a conceptual model for the toxicokinetic fate of ingested arsenic. Key points of this model are as follows:

- In most animals (including humans), absorbed arsenic is excreted mainly in the urine over the course of several days. Thus, the urinary excretion fraction (UEF), defined as the amount excreted in the urine divided by the amount given, is usually a reasonable approximation of the oral absorption fraction or ABA. However, this ratio will underestimate total absorption, because some absorbed arsenic is excreted in the feces via the bile, and some absorbed arsenic enters tissue compartments (e.g., skin, hair) from which it is cleared very slowly or not at all. Thus the urinary excretion fraction should not be equated with the absolute absorption fraction.
- The relative bioavailability (RBA) of two orally administered materials (i.e., test material and reference material) can be calculated from the ratio of the urinary excretion fraction of the two materials. This calculation is independent of the extent of tissue binding and of biliary excretion:

$$RBA(test\ vs\ ref) = \frac{AF_o(test)}{AF_o(ref)} = \frac{D \cdot AF_o(test) \cdot K_u}{D \cdot AF_o(ref) \cdot K_u} = \frac{UEF(test)}{UEF(ref)}$$

Based on the conceptual model above, raw data from this study were reduced and analyzed as follows:

- The amount of arsenic excreted in urine by each animal over each collection period was calculated by multiplying the urine volume by the urine concentration:

$$\text{Excreted (ug/48hr)} = \text{Concentration (ug/L)} \cdot \text{Volume (L/48hr)}$$

- For each test material, the amount of arsenic excreted by each animal was plotted as a function of the amount administered (ug/48 hours), and the best fit straight line (calculated by linear regression) through the data (ug excreted per ug administered) was used as the best estimate of the urinary excretion fraction (UEF).
- The relative bioavailability of arsenic in a test material was calculated as:

$$RBA = UEF(test) / UEF(NaAs)$$

where sodium arsenate (NaAs) is used as the frame of reference.

- As noted above, each RBA value is calculated as the ratio of two slopes (UEFs), each of which is estimated by linear regression through a set of data points. Because of the variability in the data, there is uncertainty in the estimated slope (UEF) for each material. This uncertainty in the slope is described by the standard

error of the mean (SEM) for the slope parameter. Given the best estimate and the SEM for each slope, the uncertainty in the ratio may be calculated using Monte Carlo simulation. The probability density function describing the confidence around each slope (UEF) term was assumed to be characterized by a t-distribution with $n-2$ degrees of freedom :

$$\frac{UEF(measured) - UEF(true)}{SEM} \sim t_{n-2}$$

For convenience, this PDF is abbreviated $T(\text{slope}, \text{sem}, n)$, where slope = best estimate of the slope derived by linear regression, sem = standard deviation in the best estimate of the slope, and n = number of data points upon which the regression analysis was performed. Thus, the confidence distribution around each ratio was simulated as:

$$PDF(RBA) = \frac{T(\text{slope}, \text{sem}, n)_{test}}{T(\text{slope}, \text{sem}, n)_{ref}}$$

Using this equation, a Monte Carlo simulation was run for each RBA calculation. The 5th and 95th percentile values from the simulated distribution of RBA values were then taken to be the 90% confidence interval for the RBA.

4.0 RESULTS

4.1 Clinical Signs

The doses of arsenic administered in this study are below a level that is expected to cause toxicological responses in swine, and no clinical signs of arsenic-induced toxicity were noted in any of the animals used in the study.

4.2 Urinary Excretion Fractions

Detailed results from the study are presented in Appendix B. The results for urinary excretion of arsenic are summarized in Figures 4-1 to 4-3. Although there is variability in the data, most dose-response curves are approximately linear, with the slope of the best-fit straight line being equal to the best estimate of the urinary excretion fraction (UEF). The following table summarizes the best fit slopes (urinary excretion fractions) for sodium arsenate and each of the test materials.

Summary of UEF Values

Test Material	Slope (UEF) $\pm$ SEM
NaAs	0.892 $\pm$ 0.033
TM1	0.326 $\pm$ 0.021
TM2	0.456 $\pm$ 0.021

4.3 Calculation of Relative Bioavailability

As discussed above, the relative bioavailability of arsenic in a specific test material is calculated as follows:

$$\text{RBA}(\text{test vs. NaAs}) = \text{UEF}(\text{test}) / \text{UEF}(\text{NaAs})$$

The results are summarized below:

Test Material	Relative Bioavailability	
	Best Estimate	90% Confidence Interval
TM1	37%	32% - 41%
TM2	51%	46% - 56%

5.0 DISCUSSION AND RECOMMENDATIONS

The *in vivo* RBA results for two composite sediments collected from the Aberjona River study area range from 37% to 51%. These results clearly indicate that arsenic in Aberjona River site sediments is not as well absorbed as soluble arsenic, and it is appropriate to take this into account when evaluating potential risks to humans from incidental ingestion of sediments. Because each sediment sample tested during this study is a composite of three sub-samples collected from differing locations along the Aberjona River, each test material represents a fairly large spatial area, and the results for these two samples may be assumed to be generally applicable to the entire site.

Although RBA values can be applied in the site risk assessment process without any understanding of what factors are responsible for the observed RBA values, it is a matter of some interest to investigate the degree to which the RBA value is correlated with other factors. The following table compares the measured values for RBA with the arsenic concentration in the sample, the IVBA, and the primary mineral phase present in each test material:

Test Material	Concentration (ppm)	RBA	IVBA		Primary Form
			1 hr	4 hrs	
TM1	676	37%	14%	19%	Iron oxide
TM2	313	51%	35%	51%	Zinc sulfate
TM3	86.8	--	49%	66%	Iron sulfate

As seen, both RBA and IVBA show an inverse correlation with concentration in the sediment. This is plotted graphically in Figure 5-1. The basis of this apparent relationship is not known. Absolute values of IVBA at one hour tend to be lower than the measured RBA values, but the difference between RBA and IVBA tends to decrease after longer extraction times. Although the values for TM2 at 4 hours happen to be equal, the values for TM1 are not equivalent. These data suggest that IVBA is a good screen to evaluate the relative *in vivo* bioavailability of arsenic at different locations, but that it should not be used as a quantitative surrogate for *in vivo* RBA at this site. The data are not sufficient to establish an empiric relationship between mineral form and RBA, but the results suggest that arsenic in association with iron oxide is likely to be less bioavailable than other forms.

6.0 REFERENCES

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USEPA. 1999. Quality Assurance Project Plan for Vasquez Blvd-I70. Bioavailability of Arsenic in Site Soils Using Juvenile Swine as an Animal Model. Report prepared by ISSI Consulting Group for USEPA Region VIII. United States Environmental Protection Agency. September, 1999.

Weis, C.P., and LaVelle, J.M. 1991. Characteristics to consider when choosing an animal model for the study of lead bioavailability. In: The Proceedings of the International Symposium on the Bioavailability and Dietary Uptake of Lead. Science and Technology Letters 3:113-119.

TABLE 2-1 STUDY DESIGN

Group	Number of Animals	Material Administered	Target Dose (ug As/day)
1	3	Control	0
2	4	Sodium Arsenate	300
3	4	Sodium Arsenate	600
4	4	Sodium Arsenate	900
5	4	Test Material 1	300
6	4	Test Material 1	600
7	4	Test Material 1	900
8	4	Test Material 2	300
9	4	Test Material 2	600
10	4	Test Material 2	900

TABLE 2-2 PRELIMINARY (SEMI-QUANTITATIVE) SPECIATION RESULTS

Sample	Arsenic Concentration (ppm)	Bioaccessibility (%)	PARTICLE FREQUENCY*						PARTICLE SIZE (um)					
			Phase						Phase					
			Iron sulfide	Iron oxide	Iron sulfate	Zinc-Iron Sulfate	Tin oxide	Sodium sulfate	Iron sulfide	Iron oxide	Iron sulfate	Zinc-Iron Sulfate	Tin oxide	Sodium sulfate
1	459	40	3	2		2			2-8	20		5-80		
2	527	31	Tr	2	3	2		Tr		4-100	8-110	12-25		
3	144	70	3	1	2	Tr				1-8		8-30		
4	145	40	1	3	2	1			1-40	8-150	15-125	7-35		
5	775	12	Tr	3		Tr	Tr			8-250				
6	176	55	3		Tr	2			3-7			12-40		
7	301	37	3	1	2	2		2	2-10		3-22	4-80		8-35
8	832	13	Tr	3		2				35-220		15		
9	407	15		3		2				30-225		7-30		
10	43.4	39	2	1	Tr				3-7					
11	64.0	49	1	1	Tr				2-10	15-35				
12	67.1	59	1	1		Tr			1-15	14				

* Code: 3 = Most Common

2 = Common

1 = Relatively Infrequent

Tr = Trace

 = Majority of arsenic in probably in this phase

TABLE 2-3 COMPOSITION OF TEST MATERIALS

Analyte	Concentration (mg/kg) ^a		
	TM1	TM2	TM3
Aluminum	15000	11000	11000
Antimony	4.3	3.7	<1
Arsenic	676.3	312.8	86.8
Barium	75	98	60
Beryllium	0.96	0.62	0.54
Cadmium	15	16	1.9
Calcium	9100	10000	4100
Chromium	680	620	140
Cobalt	32	46	14
Copper	840	540	150
Iron	73000	38000	22000
Lead	410	350	130
Magnesium	2000	2600	4300
Manganese	510	610	430
Mercury	2.9	1.1	0.61
Nickel	28	35	22
Potassium	690	770	1300
Selenium	5.8	3.8	1.6
Silver	0.88	1.1	<1
Sodium	ND	<500	ND
Sulfides, Total	5.9	63	7.2
Thallium	1.7	4.4	1.4
Total Organic Carbon	210 g/kg	220 g/kg	120 g/kg
Vanadium	49	43	35
Zinc	3300	4500	830

ND = Not detected

^a All values are in units of mg/kg except where noted otherwise. All metals except mercury were measured by USEPA method 6010B. Mercury was measured by USEPA method 7471A, total sulfides were measured by USEPA method 9030B/9034, and total organic carbon was measured by USEPA method 9060. All data are based on single measurements except arsenic, which is based on the average of duplicate analysis by ICP-MS and duplicate analysis by ICP-AES.

Table 2-4 Typical Feed Composition

Nutrient Name	Amount	Nutrient Name	Amount
Protein	20.1021%	Chlorine	0.1911%
Arginine	1.2070%	Magnesium	0.0533%
Lysine	1.4690%	Sulfur	0.0339%
Methionine	0.8370%	Manganese	20.4719 ppm
Met+Cys	0.5876%	Zinc	118.0608 ppm
Tryptophan	0.2770%	Iron	135.3710 ppm
Histidine	0.5580%	Copper	8.1062 ppm
Leucine	1.8160%	Cobalt	0.0110 ppm
Isoleucine	1.1310%	Iodine	0.2075 ppm
Phenylalanine	1.1050%	Selenium	0.3196 ppm
Phe+Tyr	2.0500%	Nitrogen Free Extract	60.2340%
Threonine	0.8200%	Vitamin A	5.1892 kIU/kg
Valine	1.1910%	Vitamin D3	0.6486 kIU/kg
Fat	4.4440%	Vitamin E	87.2080 IU/kg
Saturated Fat	0.5590%	Vitamin K	0.9089 ppm
Unsaturated Fat	3.7410%	Thiamine	9.1681 ppm
Linoleic 18:2:6	1.9350%	Riboflavin	10.2290 ppm
Linoleic 18:3:3	0.0430%	Niacin	30.1147 ppm
Crude Fiber	3.8035%	Pantothenic Acid	19.1250 ppm
Ash	4.3347%	Choline	1019.8600 ppm
Calcium	0.8675%	Pyridoxine	8.2302 ppm
Phos Total	0.7736%	Folacin	2.0476 ppm
Available Phosphorous	0.7005%	Biotin	0.2038 ppm
Sodium	0.2448%	Vitamin B12	23.4416 ppm
Potassium	0.3733%		

Feed obtained from and nutritional values provided by Zeigler Bros., Inc

FIGURE 2-1 SAMPLE CHARACTERIZATION AND PREPARATION FLOW CHART

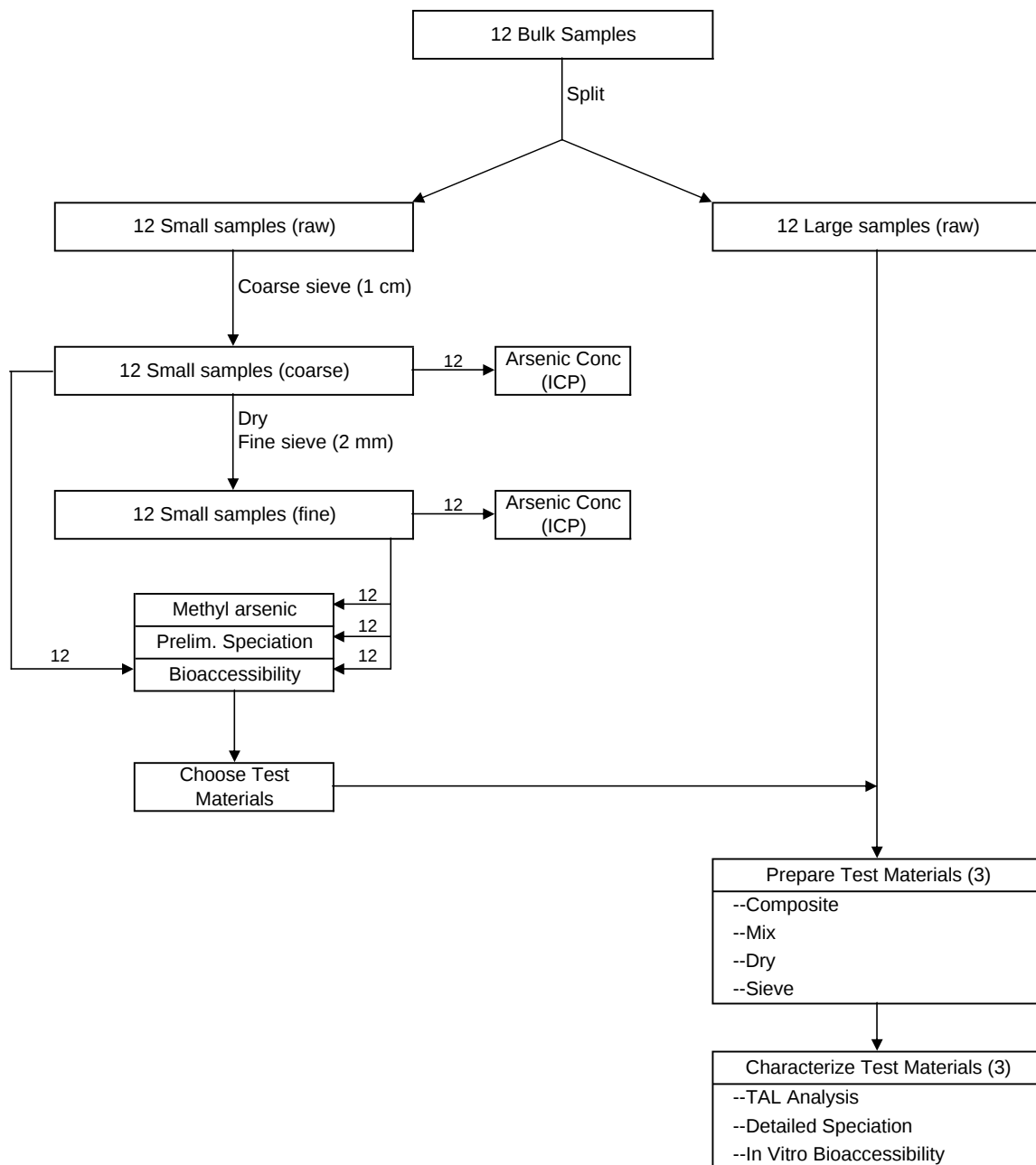


FIGURE 2-2 COMPARISON OF ARSENIC CONCENTRATIONS IN COARSE- AND FINE-SIEVED SAMPLES

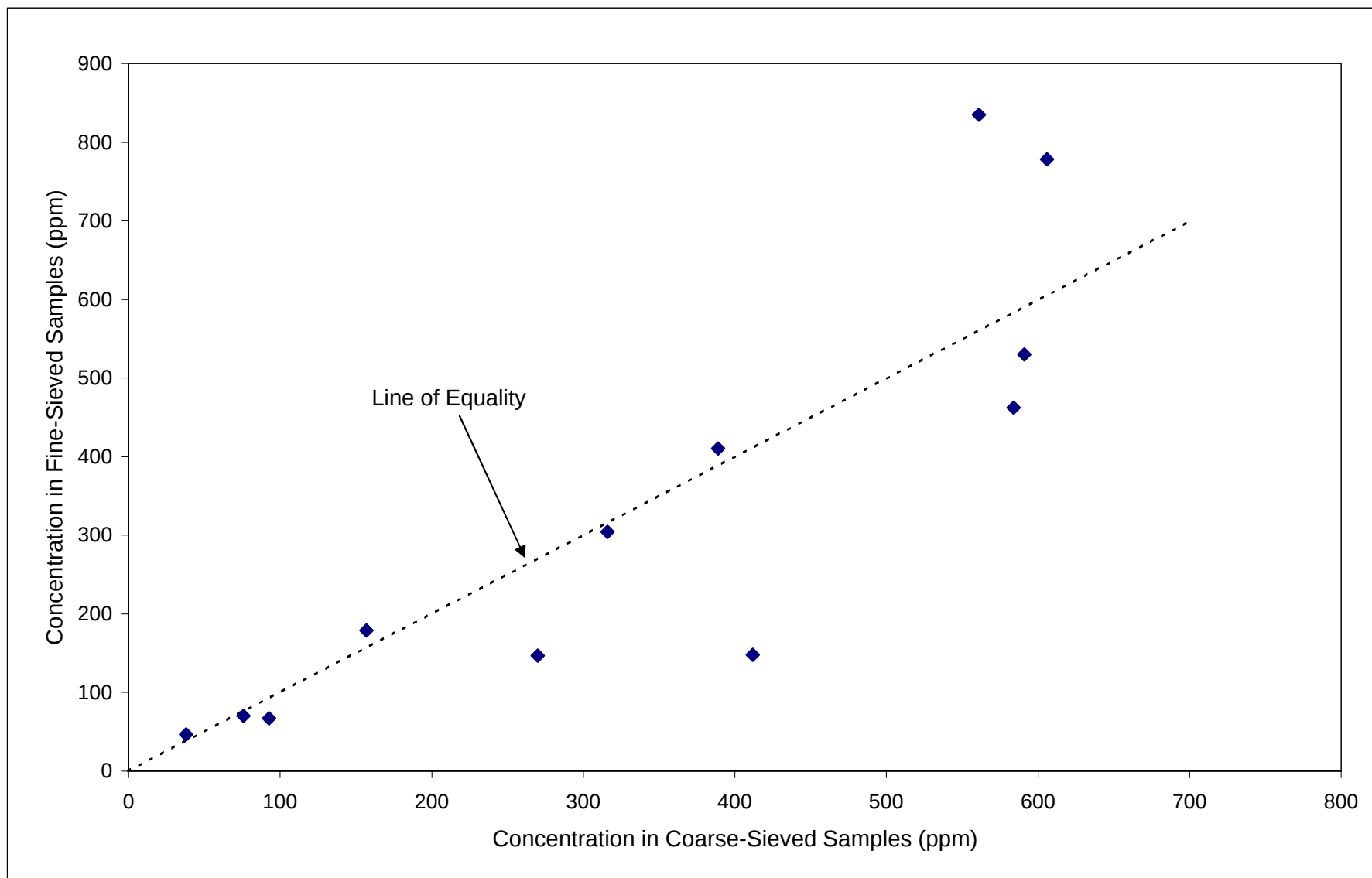


FIGURE 2-3 IN VITRO BIOACCESSIBILITY OF DRIED FINE-SIEVED SAMPLES

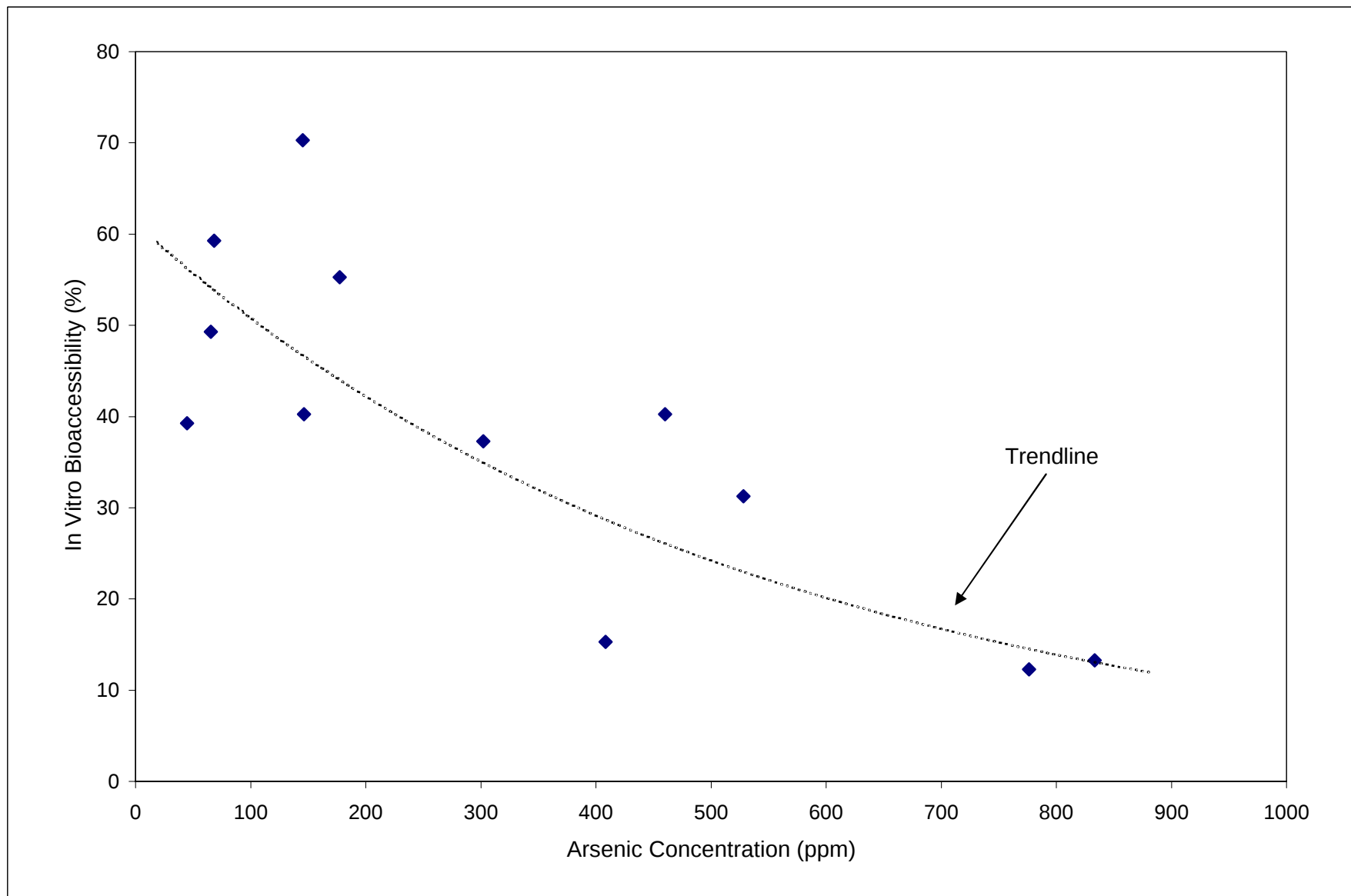


FIGURE 2-4 COMPARISON OF IN VITRO BIOACCESSIBILITY OF DRIED AND UN-DRIED FINE-SIEVED SAMPLES

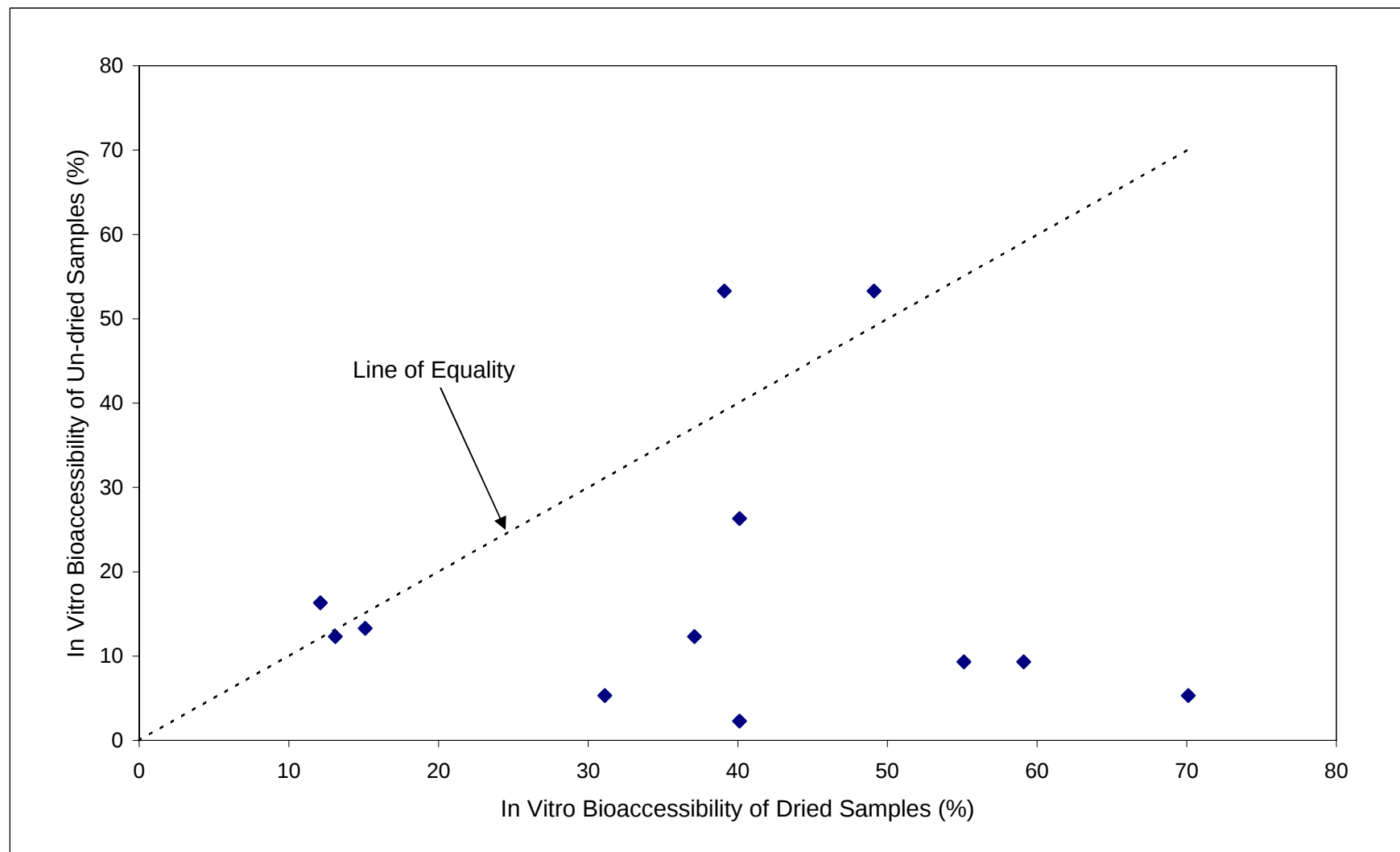


FIGURE 2-5 BODY WEIGHT GAIN

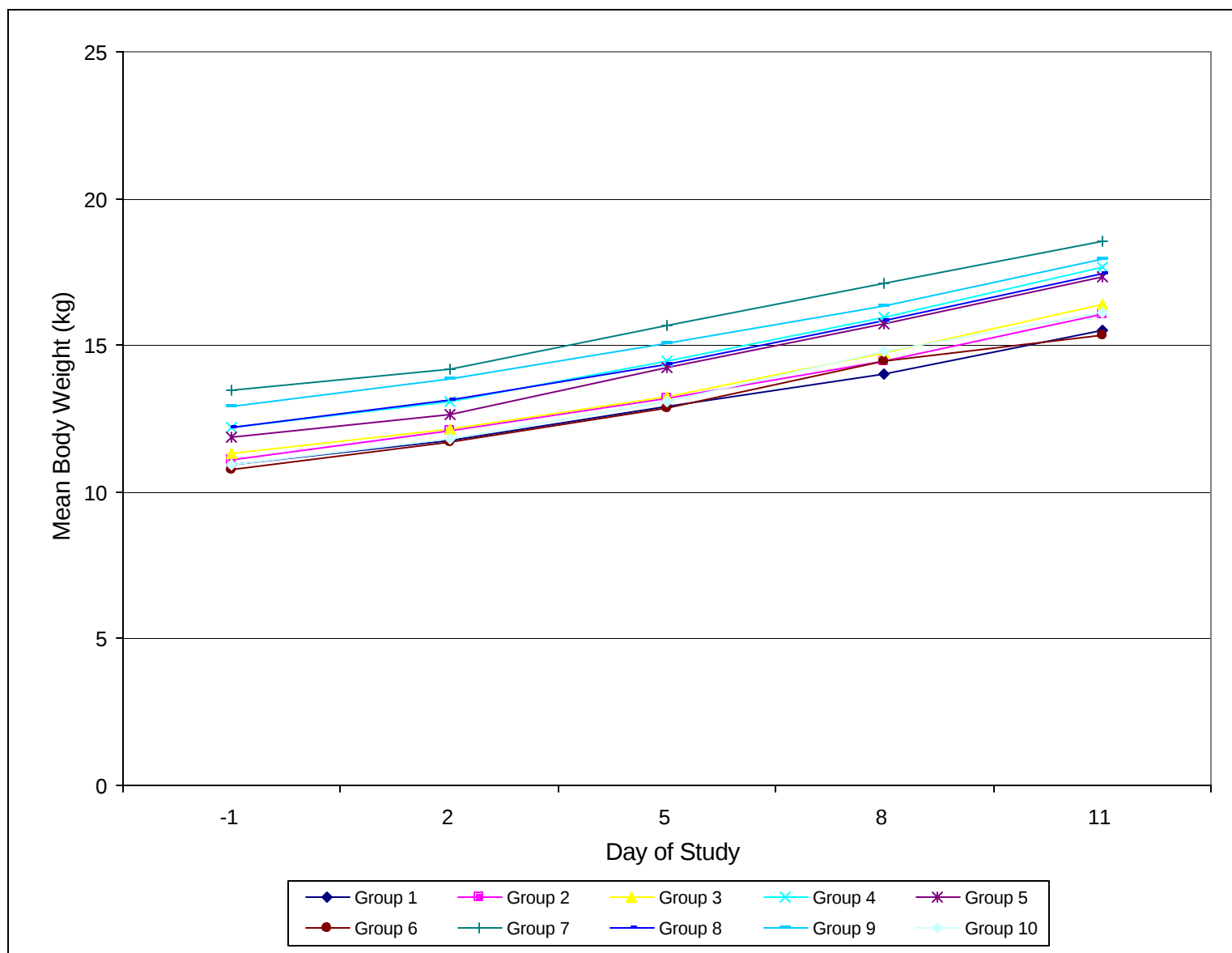


FIGURE 2-6 PERFORMANCE EVALUATION SAMPLES

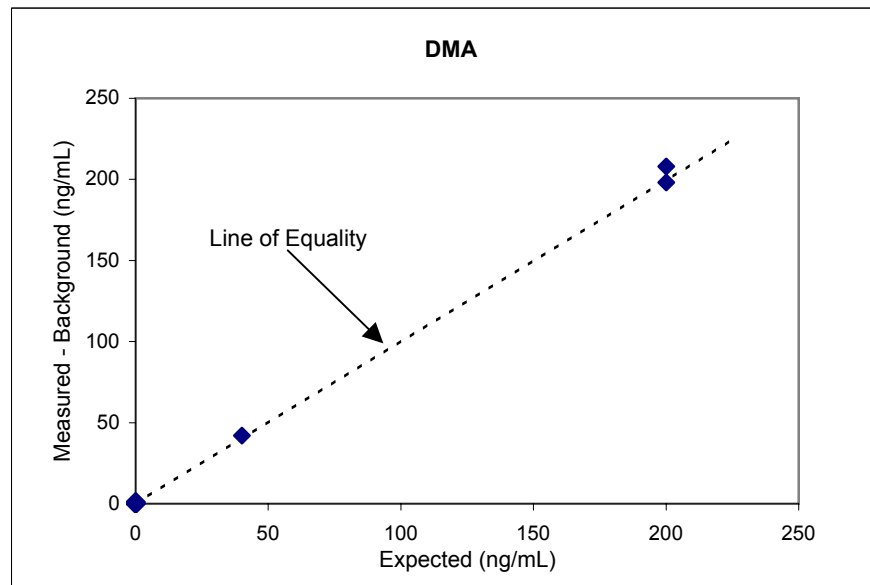
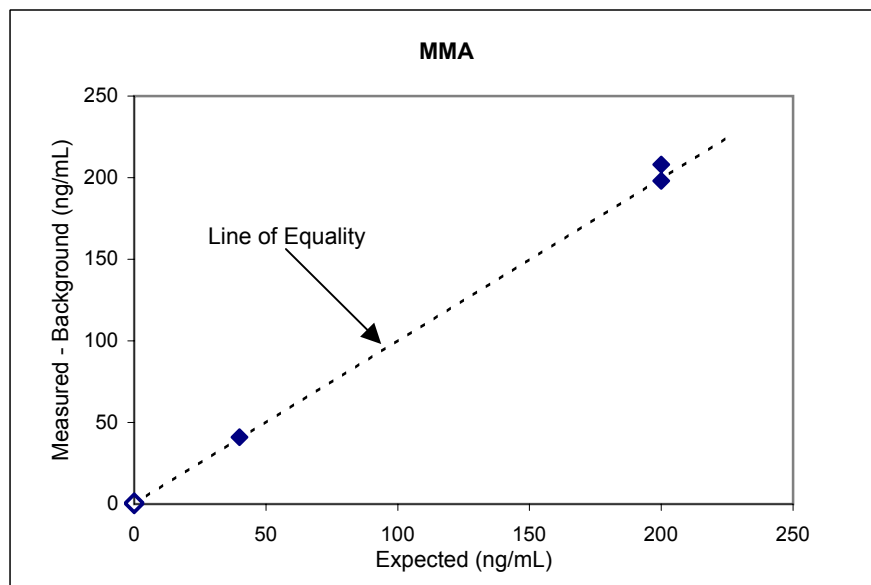
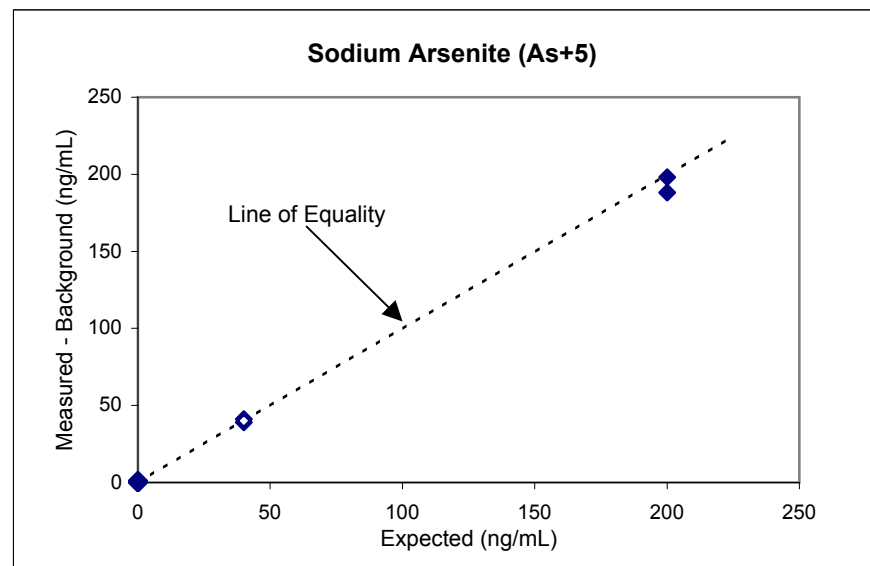
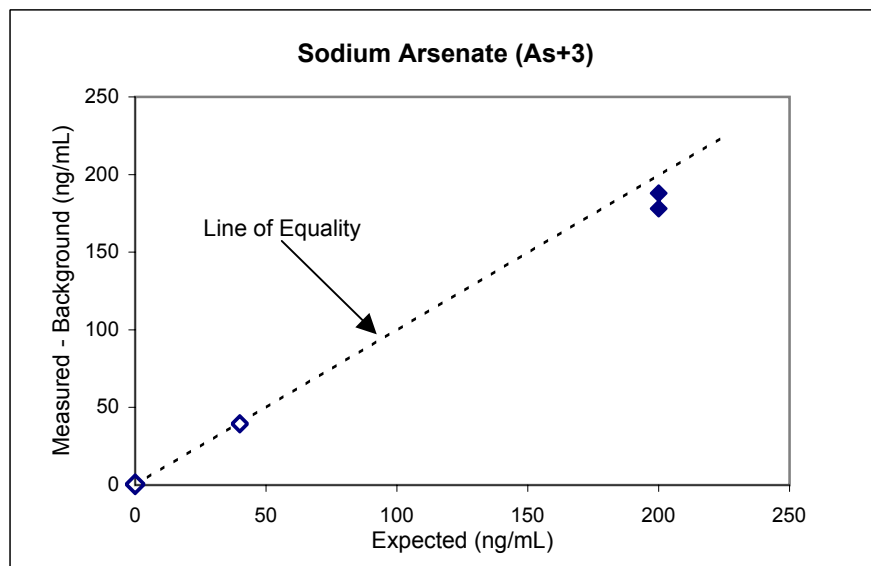


FIGURE 2-7 BLIND DUPLICATE SAMPLES

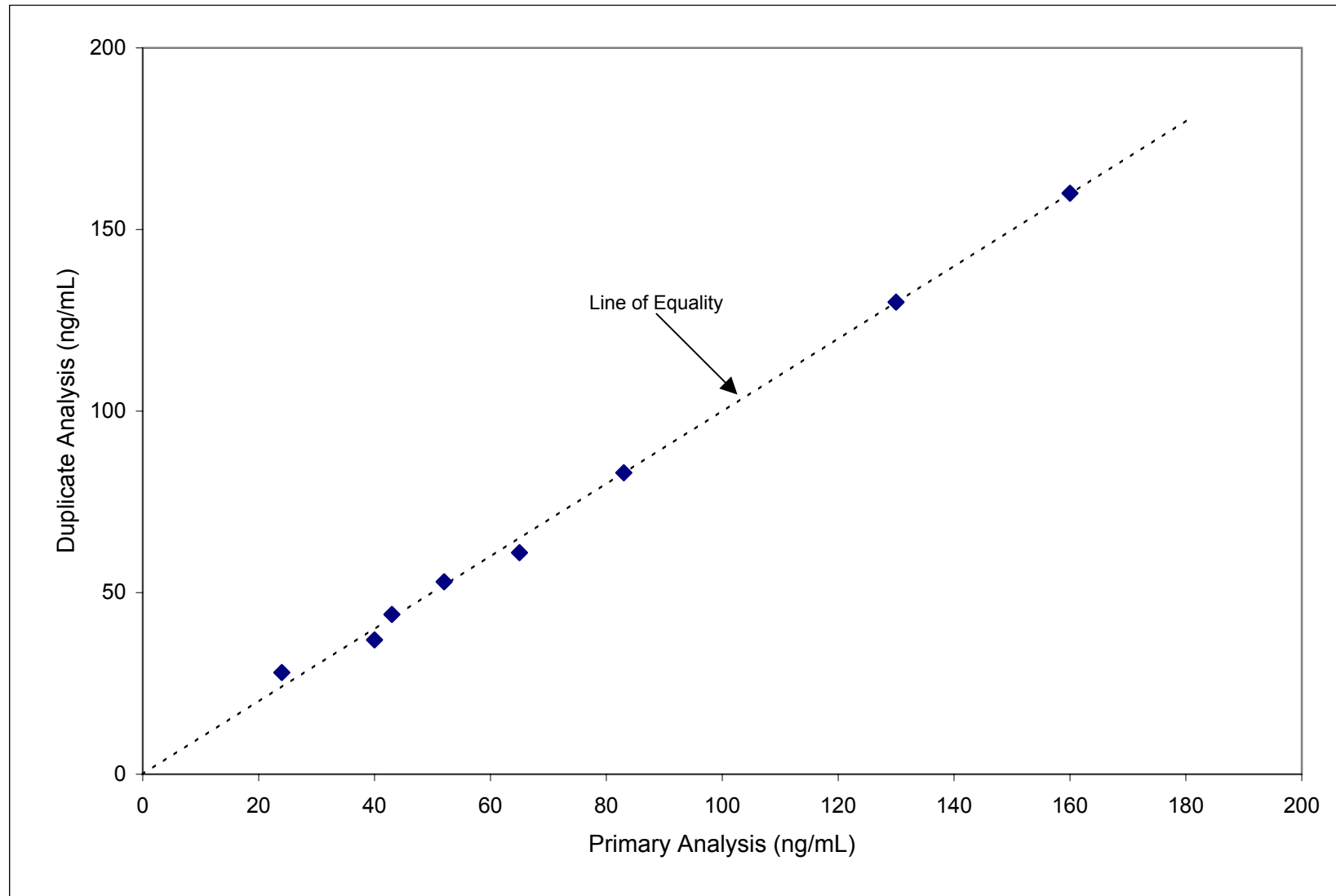
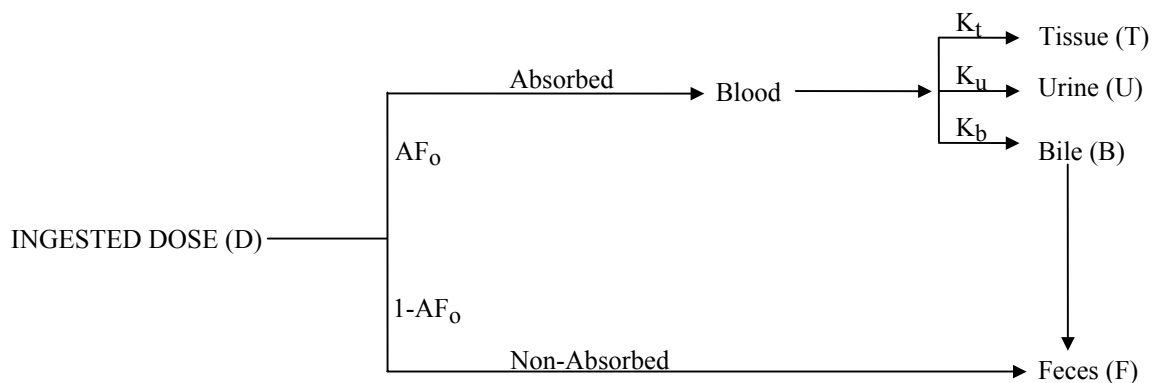


Figure 3-1. Conceptual Model for Arsenic Toxicokinetics



where:

D = Ingested dose (ug)

AF_0 = Oral Absorption Fraction

K_t = Fraction of absorbed arsenic which is retained in tissues

K_u = Fraction of absorbed arsenic which is excreted in urine

K_b = Fraction of absorbed arsenic which is excreted in the bile

BASIC EQUATIONS:

$$\text{Amount Absorbed (ug)} = D \cdot AF_0$$

$$\begin{aligned} \text{Amount Excreted (ug)} &= \text{Amount absorbed} \cdot K_u \\ &= D \cdot AF_0 \cdot K_u \end{aligned}$$

$$\begin{aligned} \text{Urinary Excretion Fraction (UEF)} &= \text{Amount excreted} / \text{Amount ingested} \\ &= (D \cdot AF_0 \cdot K_u) / D \\ &= AF_0 \cdot K_u \end{aligned}$$

$$\begin{aligned} \text{Relative Bioavailability (x vs. y)} &= \text{UEF(x)} / \text{UEF(y)} \\ &= (AF_0(x) \cdot K_u) / (AF_0(y) \cdot K_u) \\ &= AF_0(x) / AF_0(y) \end{aligned}$$

FIGURE 4-1 URINARY EXCRETION OF ARSENIC FROM SODIUM ARSENATE

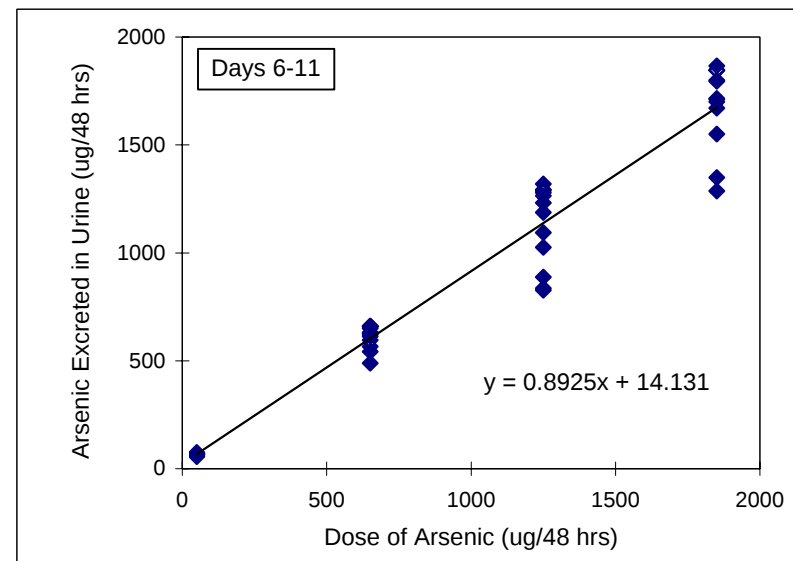
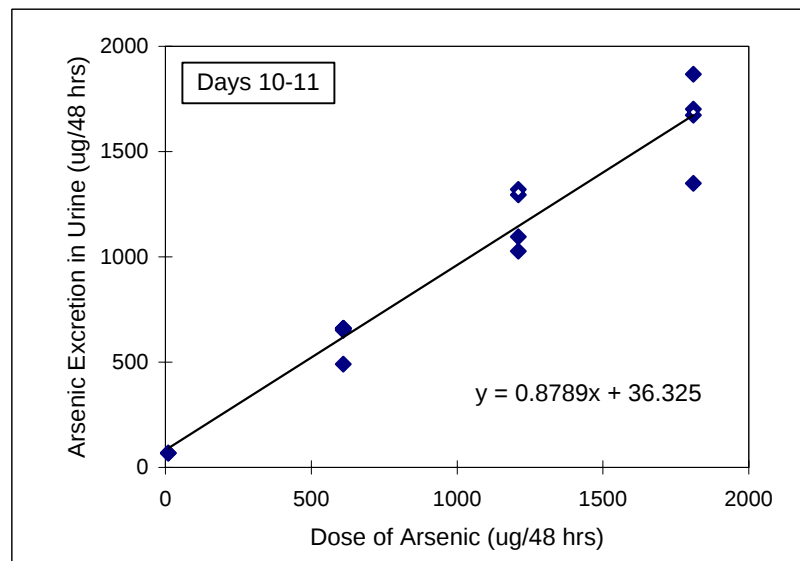
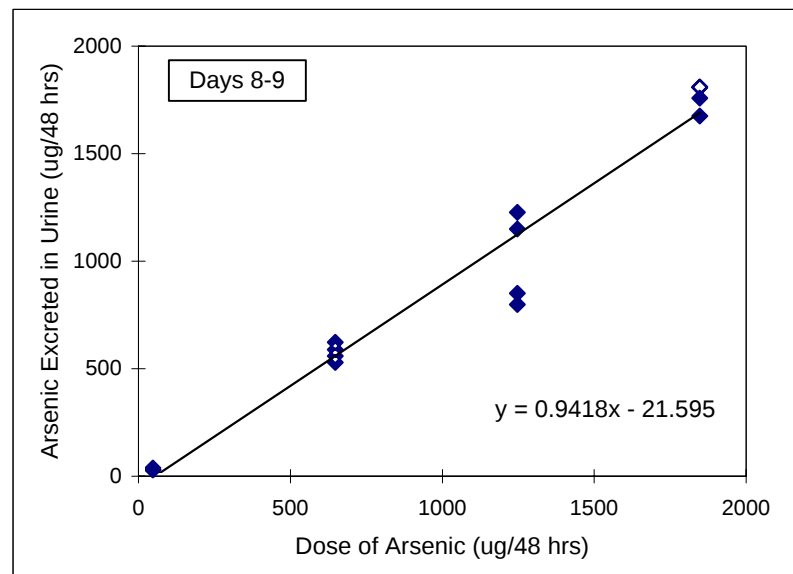
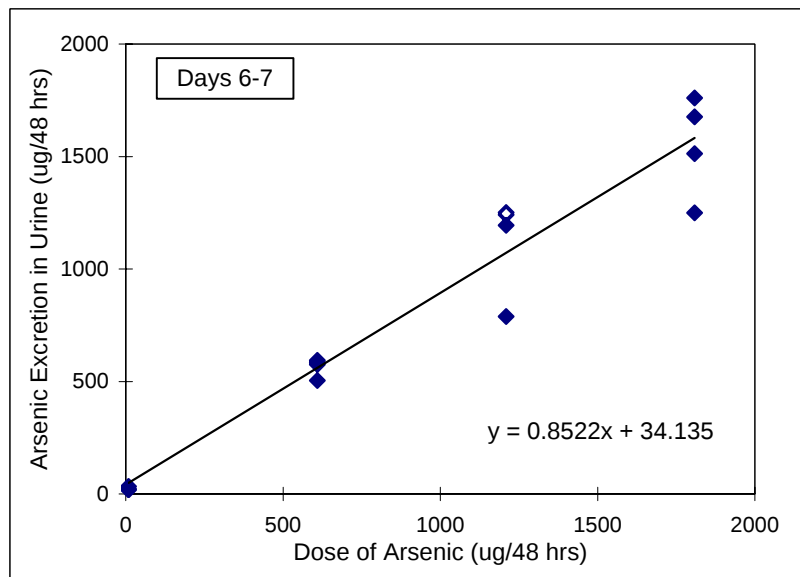


FIGURE 4-2 URINARY EXCRETION OF ARSENIC FROM TEST MATERIAL 1

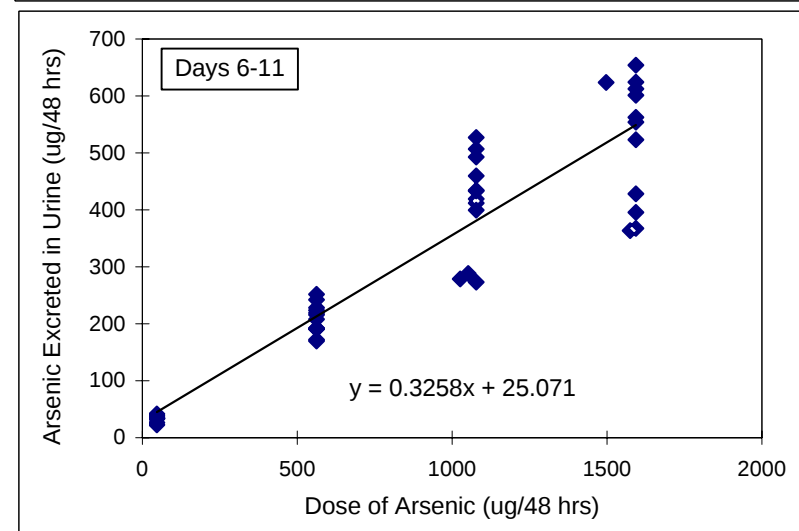
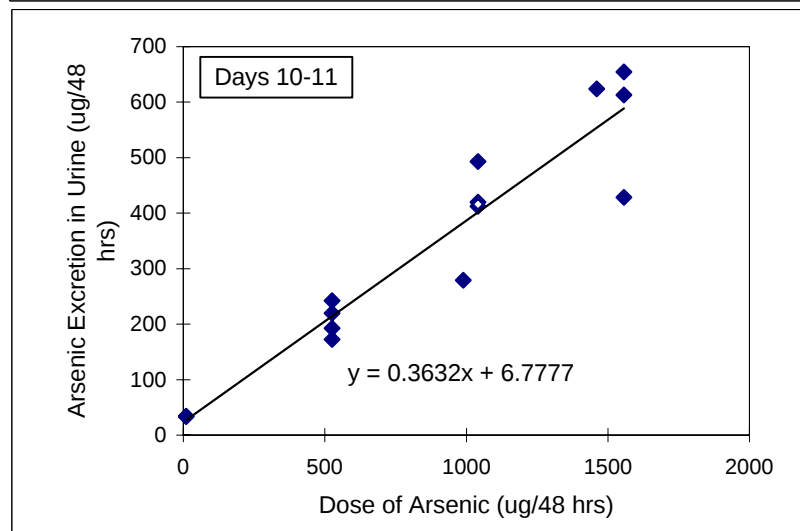
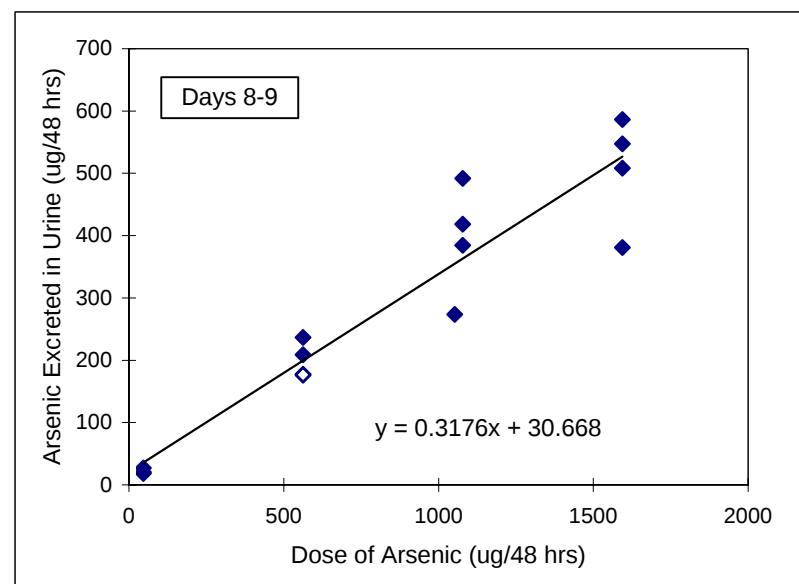
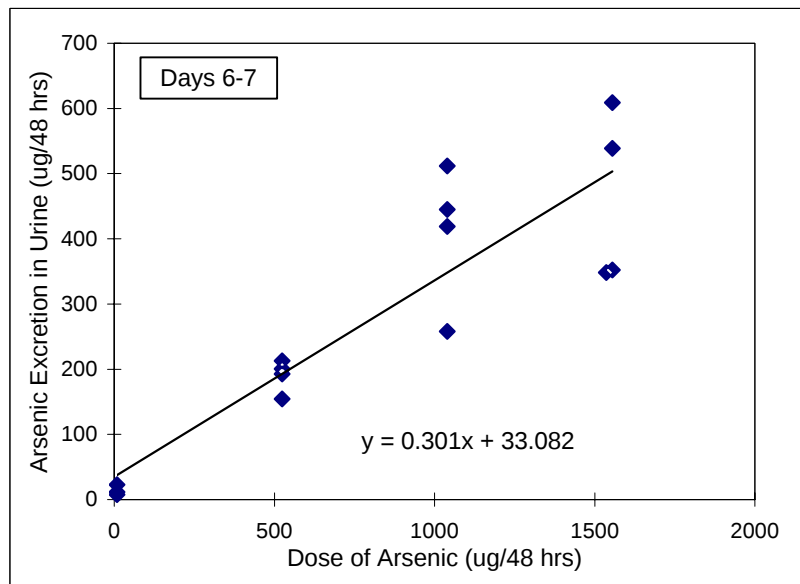


FIGURE 4-3 URINARY EXCRETION OF ARSENIC FROM TEST MATERIAL 2

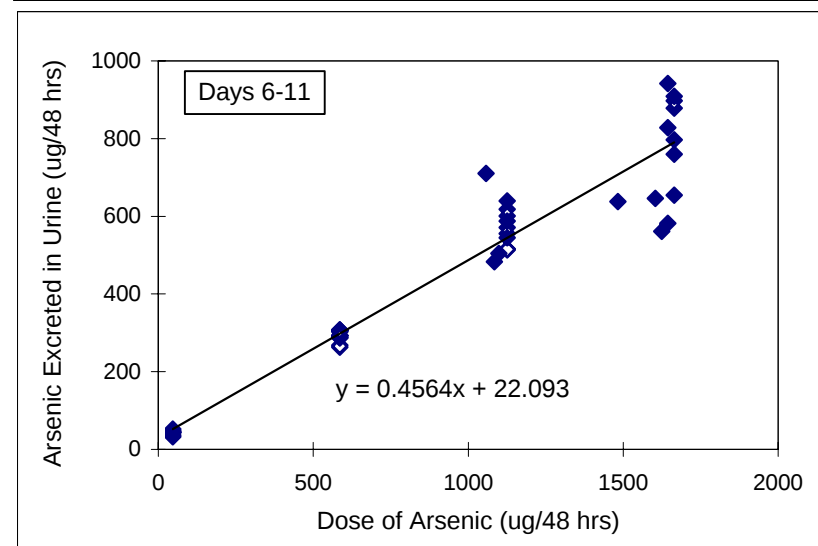
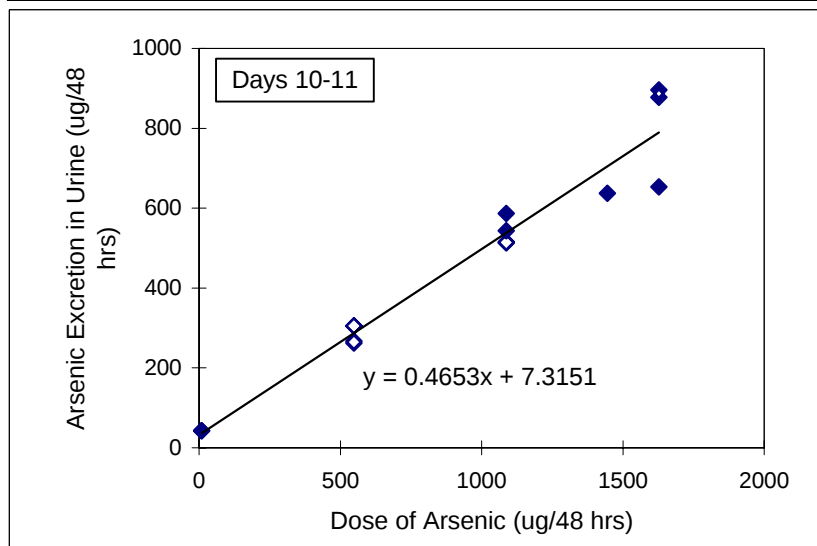
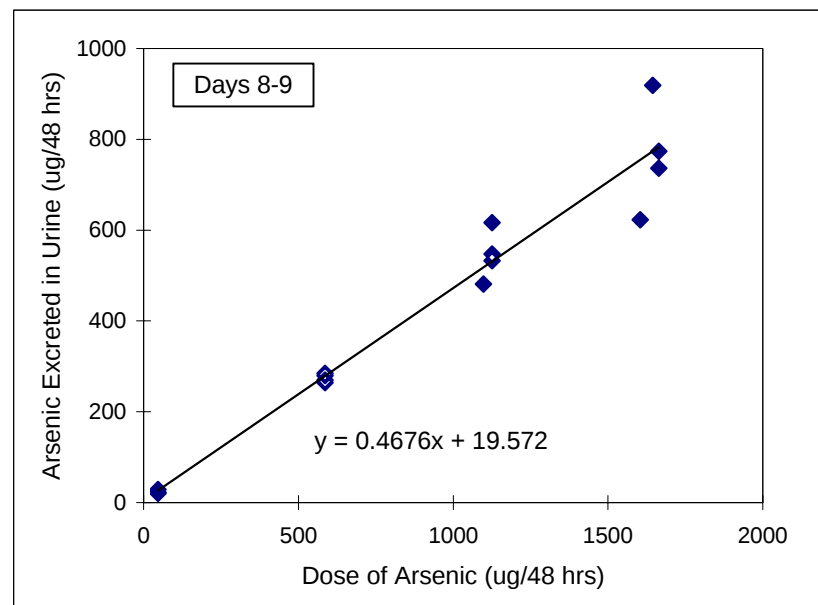
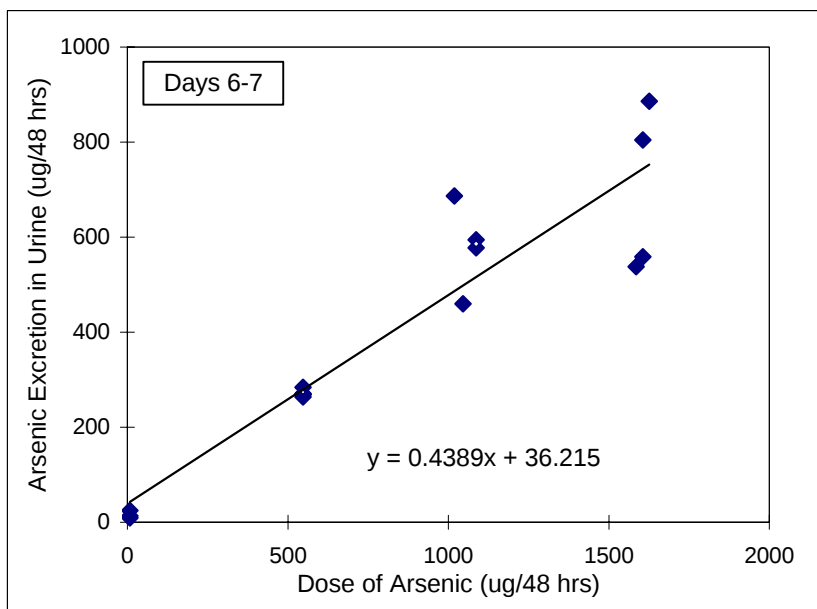
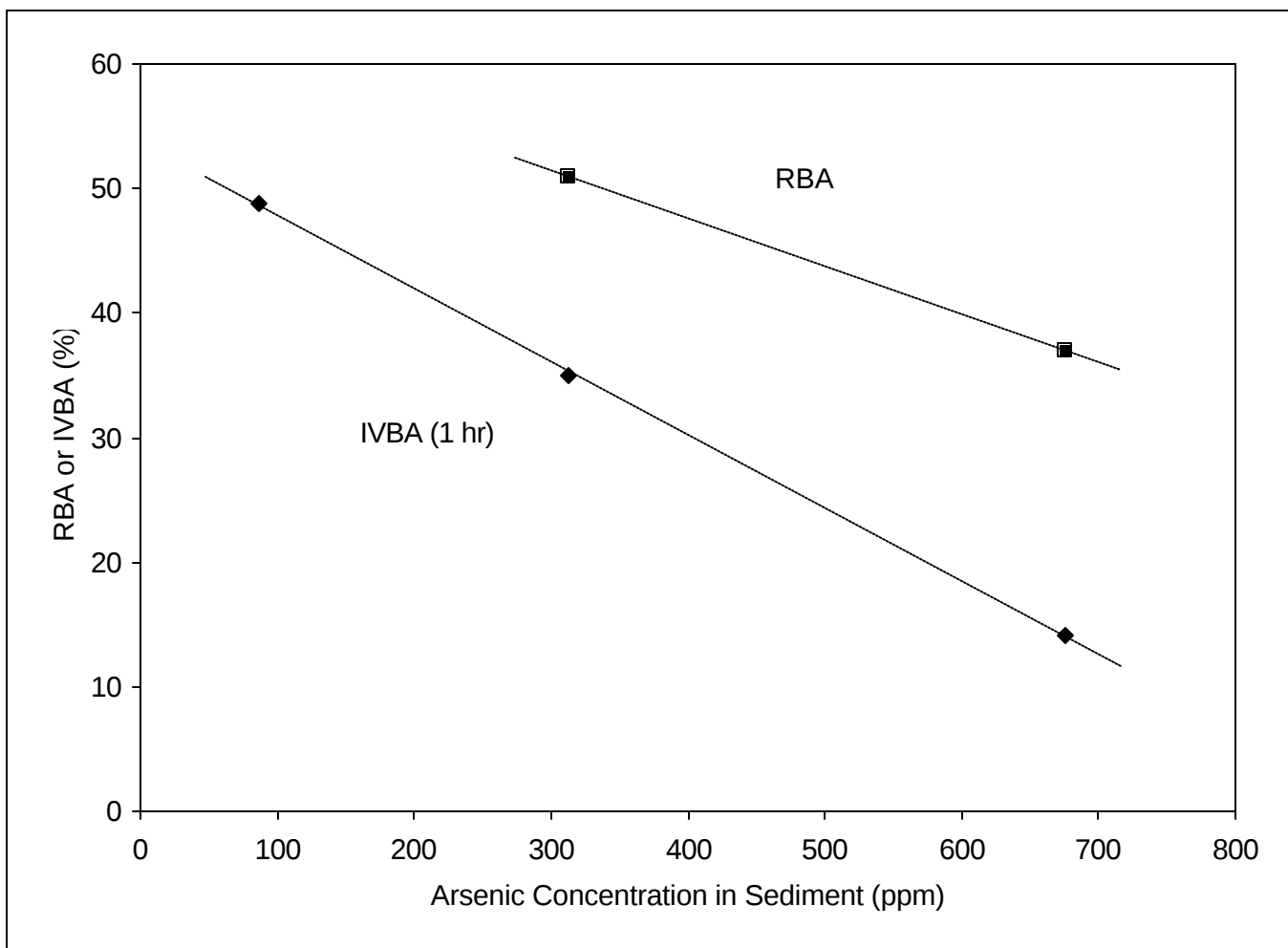


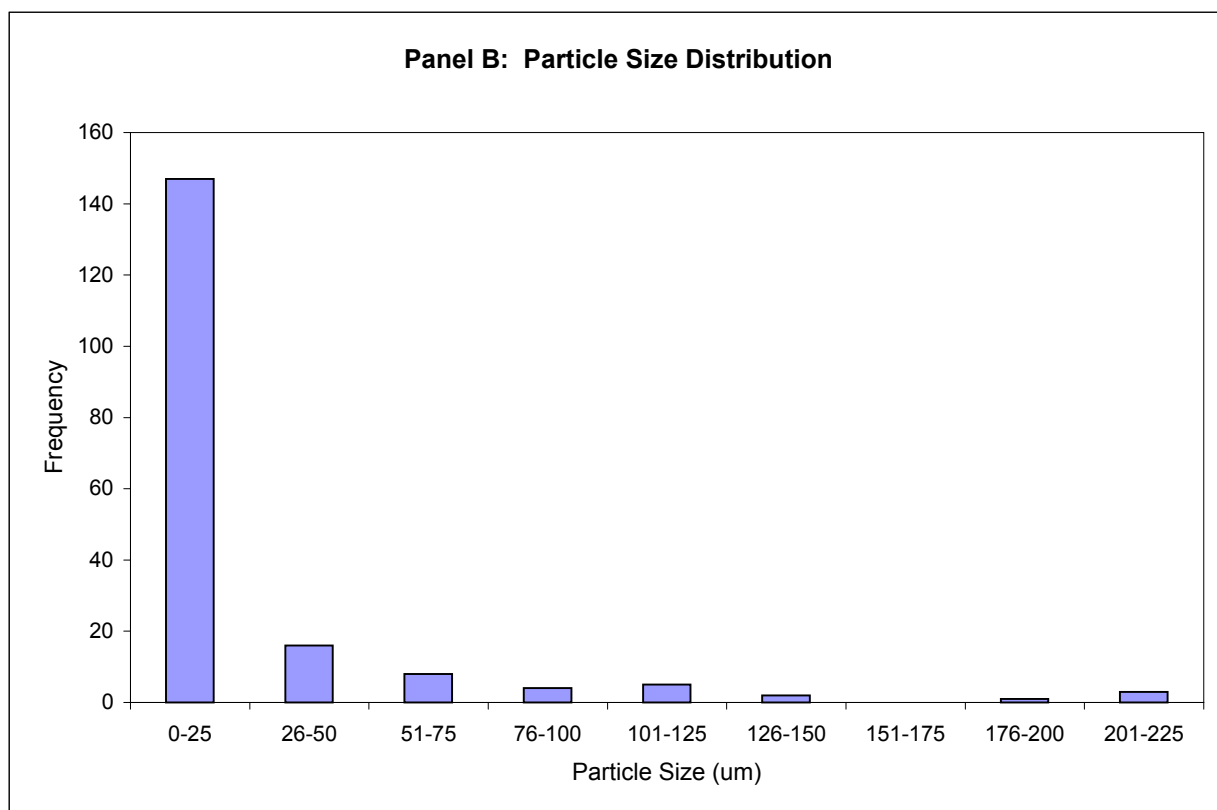
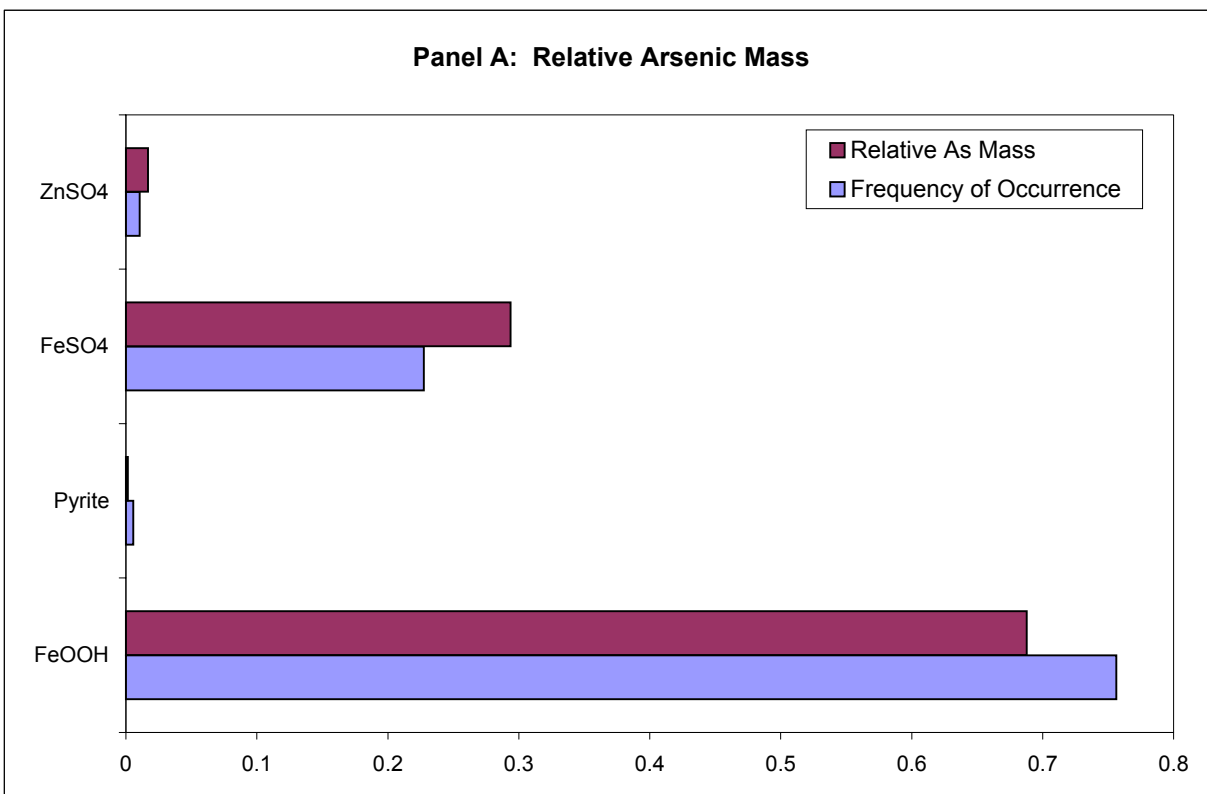
Figure 5-1. RBA and IVBA as a Function of Sediment Concentration



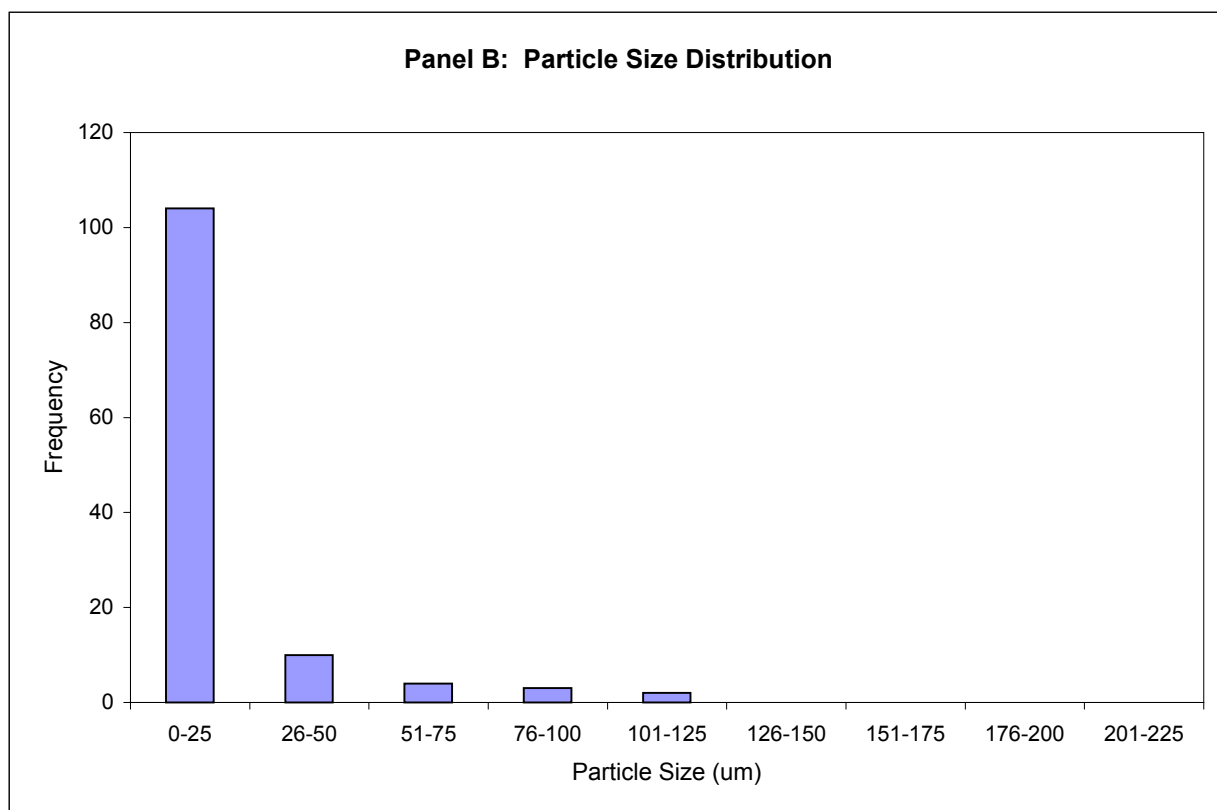
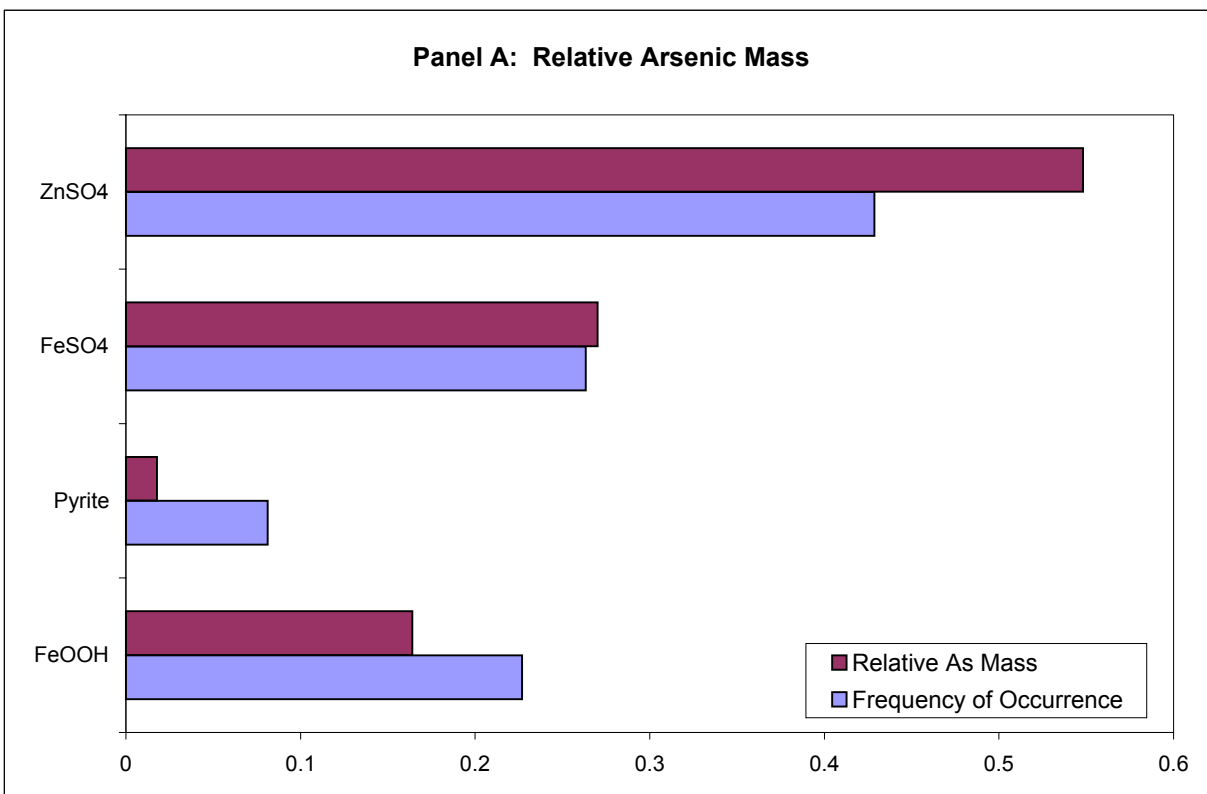
APPENDIX A

DETAILED ARSENIC SPECIATION RESULTS

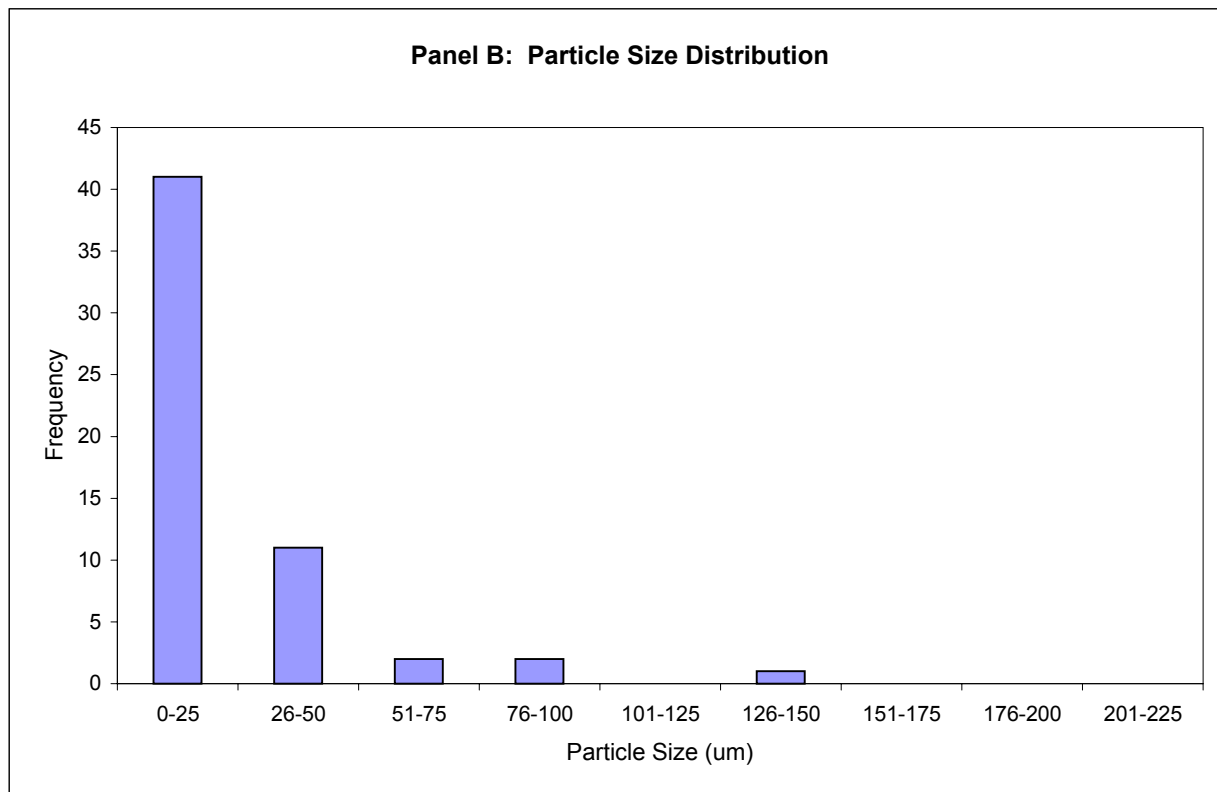
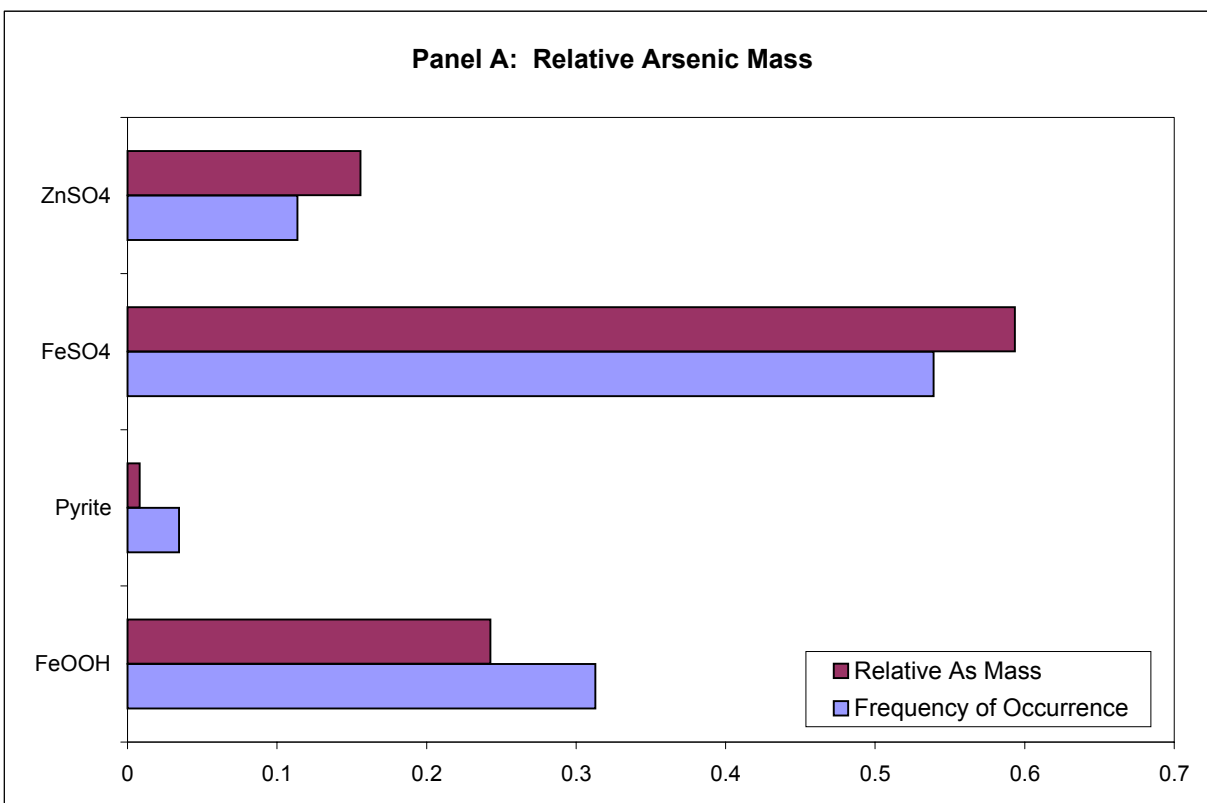
TEST MATERIAL 1 - SPECIATION AND PARTICLE SIZE DATA



TEST MATERIAL 2 - SPECIATION AND PARTICLE SIZE DATA



TEST MATERIAL 3 - SPECIATION AND PARTICLE SIZE DATA



APPENDIX B
DETAILED RESULTS

TABLE B-1 SCHEDULE

Study Day	Day	Date	Dose Administration	Feed Special Diet	Weigh	Dose Prep	Cull Pigs/ Assign Dose Group	48 hr Urine Collection	Sacrifice
-8	Tuesday	8/27/02							
-7	Wednesday	8/28/02			X		X		
-6	Thursday	8/29/02							
-5	Friday	8/30/02							
-4	Saturday	8/31/02			X				
-3	Sunday	9/1/02							
-2	Monday	9/2/02		X					
-1	Tuesday	9/3/02	X	X	X	X			
0	Wednesday	9/4/02	X	X					
1	Thursday	9/5/02	X	X					
2	Friday	9/6/02	X	X	X	X			
3	Saturday	9/7/02	X	X					
4	Sunday	9/8/02	X	X					
5	Monday	9/9/02	X	X	X	X			
6	Tuesday	9/10/02	X	X				↕	
7	Wednesday	9/11/02	X	X				↕	
8	Thursday	9/12/02	X	X	X	X		↕	
9	Friday	9/13/02	X	X				↕	
10	Saturday	9/14/02	X	X				↕	
11	Sunday	9/15/02	X	X	X			↕	
12	Monday	9/16/02							X

TABLE B-2 GROUP ASSIGNMENTS

Pig Number	Dose Group	Material Administered	Target Dose of Arsenic (ug/day)
324 338 349	1	Control	0
326 330 339 350	2	NaAs	300
310 316 322 340	3	NaAs	600
303 315 329 341	4	NaAs	900
301 318 344 347	5	TM1	300
309 327 343 346	6	TM1	600
306 308 317 331	7	TM1	900
304 311 314 321	8	TM2	300
307 313 325 332	9	TM2	600
328 337 342 348	10	TM2	900

TABLE B-3 BODY WEIGHTS AND ADMINISTERED DOSES, BY DAY

Body weights were measured on days -7, -4, -1, 2, 5, 8, and 11. Weights for other days are estimated, based on linear interpolation between measured values.

Group	Pig #	Day -7		Day -4		Day -1		Day 0		Day 1		Day 2		Day 3		Day 4		Day 5		Day 6		Day 7		Day 8		Day 9		Day 10		Day 11	
		BW	ugAs	BW	ugAs	BW	ugAs	BW	ugAs	BW	ugAs	BW	ugAs	BW	ugAs	BW	ugAs	BW	ugAs	BW	ugAs	BW	ugAs	BW	ugAs	BW	ugAs	BW	ugAs	BW	ugAs
		kg	per day	kg	per day	kg	per day	kg	per day	kg	per day	kg	per day	kg	per day	kg	per day	kg	per day	kg	per day	kg	per day	kg	per day	kg	per day	kg	per day	kg	per day
1	324	10.15	0	10.25	0	11.15	0	11.48	0	11.82	0	12.15	0	12.57	0	12.98	0	13.40	0	13.75	0	14.10	0	14.45	0	14.87	0	15.28	0	15.70	0
1	338	8.9	0	9.45	0	10.9	0	11.0	0	11.2	0	11.3	0	11.7	0	12.1	0	12.45	0	12.8	0	13.1	0	13.45	0	14.1	0	14.8	0	15.4	0
1	349	10	0	9.45	0	10.75	0	11.1	0	11.4	0	11.75	0	12.1	0	12.5	0	12.85	0	13.3	0	13.8	0	14.2	0	14.6	0	15.0	0	15.4	0
2	326	11.05	300	11.2	300	11.9	300	12.3	300	12.6	300	13	300	13.3	300	13.7	300	14	300	14.4	300	14.8	300	15.25	300	15.8	300	16.4	300	16.9	300
2	330	9.65	300	10.3	300	11.35	300	11.5	300	11.7	300	11.85	300	12.3	300	12.7	300	13.15	300	13.5	300	13.9	300	14.25	300	14.8	300	15.4	300	16	300
2	339	8.2	300	9	300	9.85	300	10.3	300	10.8	300	11.2	300	11.5	300	11.8	300	12.15	300	12.6	300	13.0	300	13.45	300	14.0	300	14.5	300	15.05	300
2	350	10.55	300	10.45	300	11.25	300	11.6	300	12.0	300	12.3	300	12.7	300	13.1	300	13.45	300	13.9	300	14.4	300	14.9	300	15.4	300	15.9	300	16.35	300
3	310	11.55	600	11.65	600	12.65	600	12.8	600	13.0	600	13.2	600	13.5	600	13.9	600	14.2	600	14.8	600	15.5	600	16.1	600	16.5	600	16.9	600	17.3	600
3	316	9.65	600	10.2	600	11.7	600	12.0	600	12.3	600	12.55	600	12.9	600	13.3	600	13.65	600	14.0	600	14.4	600	14.75	600	15.4	600	16.1	600	16.7	600
3	322	10.45	600	10.95	600	11.8	600	12.2	600	12.5	600	12.9	600	13.3	600	13.8	600	14.2	600	14.9	600	15.6	600	16.25	600	16.9	600	17.6	600	18.3	600
3	340	7.8	600	8.2	600	9.05	600	9.3	600	9.6	600	9.85	600	10.2	600	10.5	600	10.85	600	11.2	600	11.5	600	11.75	600	12.2	600	12.7	600	13.2	600
4	303	11.35	900	11.25	900	12.5	900	12.70	900	12.9	900	13.1	900	13.6	900	14.1	900	14.65	900	15.2	900	15.8	900	16.4	900	16.9	900	17.4	900	17.85	900
4	315	10.45	900	10.75	900	11.95	900	12.2	900	12.5	900	12.75	900	13.2	900	13.7	900	14.2	900	14.6	900	14.9	900	15.25	900	15.8	900	16.4	900	16.95	900
4	329	11.05	900	11.8	900	12.9	900	13.4	900	13.8	900	14.25	900	14.7	900	15.2	900	15.6	900	16.0	900	16.5	900	16.9	900	17.4	900	18.0	900	18.5	900
4	341	8.85	900	9.95	900	11.45	900	11.7	900	12.0	900	12.3	900	12.7	900	13.0	900	13.4	900	14.0	900	14.6	900	15.15	900	15.9	900	16.7	900	17.45	900
5	301	13.1	257.802	13.45	257.802	14.65	257.802	15.0	258	15.3	258	15.6	257.802	16.2	258	16.9	258	17.5	257.802	18.2	258	18.8	258	19.45	257.802	20.0	258	20.6	258	21.1	257.802
5	318	11.2	257.802	11.3	257.802	12.35	257.802	12.5	258	12.7	258	12.9	257.802	13.4	258	13.9	258	14.45	257.802	15.0	258	15.6	258	16.1	257.802	16.6	258	17.2	258	17.7	257.802
5	344	10.6	257.802	10.25	257.802	11.1	257.802	11.3	258	11.5	258	11.75	257.802	12.3	258	12.8	258	13.3	257.802	13.8	258	14.2	258	14.7	257.802	15.2	258	15.6	258	16.1	257.802
5	347	8.35	257.802	8.4	257.802	9.45	257.802	9.8	258	10.1	258	10.4	257.802	10.8	258	11.3	258	11.7	257.802	12.0	258	12.4	258	12.7	257.802	13.3	258	13.8	258	14.35	257.802
6	309	8.7	515.604	9.9	515.604	10.8	515.604	11.0	516	11.3	516	11.5	515.604	11.8	516	12.2	516	12.5	515.604	13.1	516	13.7	516	14.3	515.604	14.7	516	15.1	516	15.5	515.604
6	327	9.85	515.604	10.15	515.604	11.25	515.604	11.6	516	11.9	516	12.15	515.604	12.6	516	13.1	516	13.6	515.604	14.2	516	14.8	516	15.4	515.604	15.7	516	15.9	516	16.2	515.604
6	343	9.4	515.604	9.1	515.604	10.1	515.604	10.4	516	10.7	516	10.95	515.604	11.4	516	11.8	516	12.15	515.604	12.6	516	13.0	516	13.4	489.823	13.6	516	13.8	516	14.05	464.043
6	346	9.4	515.604	9.9	515.604	11	515.604	11.4	516	11.8	516	12.25	515.604	12.6	516	12.9	516	13.25	515.604	13.7	516	14.2	516	14.7	515.604	15.0	516	15.4	516	15.7	515.604
7	306	9.7	773.405	13.6	773.405	14.8	773.405	15.0	773	15.2	773	15.45	773.405	16.1	773	16.7	773	17.25	773.405	17.7	773	18.1	773	18.45	773.405	19.0	773	19.6	773	20.15	773.405
7	308	11.15	773.405	11.95	773.405	12.7	773.405	12.9	773	13.1	773	13.3	773.405	13.7	773	14.1	773	14.45	773.405	15.0	773	15.5	773	15.95	773.405	16.4	773	16.8	773	17.2	773.405
7	317	12.75	773.405	12.25	773.405	12.6	773.405	12.9	773	13.1	773	13.4	773.405	13.9	773	14.4	773	14.95	773.405	15.4	773	15.8	773	16.25	773.405	16.8	773	17.3	773	17.75	773.405
7	331	12.85	773.405	12.85	773.405	13.8	773.405	14.1	773	14.3	773	14.6	773.405	15.1	735	15.6	773	16.15	773.405	16.7	754	17.2	773	17.7	773.405	18.1	773	18.6	754	19	696.065
8	304	10.1	269.655	10.8	269.655	12.1	269.655	12.4	270	12.7	270	13.05	269.655	13.4	270	13.7	270	14	269.655	14.3	270	14.7	270	15	269.655	15.7	270	16.3	270	17	269.655
8	311	11.4	269.655	11.95	269.655	12.75	269.655	13.0	270	13.3	270	13.5	269.655	13.9	270	14.4	270	14.8	269.655	15.3	270	15.8	270	16.3	269.655	16.9	270	17.6	270	18.2	269.655
8	314	10.45	269.655	10.8	269.655	11.5	269.655	11.9	270	12.3	270	12.65	269.655	13.0	270	13.4	270	13.8	269.655	14.5	270	15.1	270	15.8	269.655	16.2	270	16.5	270	16.9	269.655
8	321	11.95	269.655	12.1	269.655	12.45	269.655	12.8	270	13.1	270	13.4	269.655	13.9	270	14.3	270	14.75	269.655	15.2	270	15.7	270	16.15	269.655	16.7	270	17.2	270	17.65	269.655
9	307	13.7	539.31	13	539.31	13.6	539.31	14.0	539	14.3	539	14.65	539.31	14.9	539	15.2	539	15.5	525.828	15.8	539	16.1	472	16.45	539.31	16.9	539	17.4	539	17.85	539.31
9	313	12.55	539.31	12.9	539.31	13.4	539.31	13.8	526	14.2	539	14.6	512.345	15.0	539	15.4	539	15.85	539.31	16.1	539	16.4	539	16.6	539.31	17.4	539	18.2	539	19	539.31
9	325	11.45	539.31	11.7	539.31	12.25	539.31	12.5	539	12.7	539	12.95	539.31	13.4	539	13.9	539	14.35	539.31	15.0	512	15.6	526	16.2	525.828	16.8	526	17.4	539	17.95	539.31
9	332	11.95	539.31	11.5	539.31	12.4	539.31	12.7	539	12.9	539	13.2	539.31	13.7	539	14.2	539	14.65	539.31	15.2	539	15.7	539	16.15	539.31	16.4	539	16.7	539	16.9	539.31
10	328	11.05	808.966	11.25	808.966	12.1	808.966	12.5	809	12.8	809	13.15	808.966	13.6	809	14.1	809	14.5	808.966	15.1	809	15.7	809	16.35	788.741	16.8	809	17.3	809	17.75	808.966
10	337	8.5	808.966	9.1	808.966	9.75	808.966	10.1	789	10.4	809	10.75	788.741	11.2	809	11.7	769	12.1	808.966	12.5	769	13.0	809	13.4	768.517	13.9	789	14.3	667	14.75	768.517
10	342	10.9	808.966	11.15	808.966	11.8	808.966	12.1	809	12.4	809	12.75	808.966	13.1	809	13.4	809	13.75	808.966	14.5	809	15.2	789	15.9	808.966	16.3	809	16.6	809	16.95	808.966
10	348	9.05	808.966	9.2	808.966	10.1	808.966	10.3	809	10.5	809	10.65	808.966	11.1	789	11.6	809	12.05	808.966	12.6	789	13.1	809	13.55	808.966	14.0	809	14.5	809	15	808.966

Missed Doses:

TABLE B-4 URINE VOLUMES - 48 HOUR COLLECTIONS

Units of Volume: mls

Group	Pig ID	Day		
		6-7 9/10-9/11	8-9 9/12-9/13	10-11 9/14-9/15
1	324	5400	6780	11620
	338	6960	7280	13800
	349	6100	4340	4460
2	326	6870	7640	14940
	330	3060	1900	3350
	339	19330	8320	18380
	350	12850	7640	10100
3	310	11150	3260	14060
	316	24060	50480	40840
	322	16940	8720	12400
	340	4840	3480	8100
4	303	10270	12800	13490
	315	12220	23700	16150
	329	21400	21620	26660
	341	5540	7260	8990
5	301	3360	2240	2020
	318	4960	4830	3440
	344	3440	4380	4010
	347	10700	10740	11690
6	309	18340	16790	19700
	327	6280	6360	9800
	343	7040	4480	9240
	346	22050	15820	16650
7	306	8220	8220	11620
	308	15500	11400	12200
	317	2520	2350	2150
	331	8180	8680	11180
8	304	5660	6600	4440
	311	23820	23920	29080
	314	6000	5250	4660
	321	10300	14600	7440
9	307	17000	21760	18000
	313	24830	16420	14660
	325	4360	4840	4050
	332	8910	6760	4290
10	328	15700	14470	21760
	337	3320	1400	3800
	342	14000	14200	33350
	348	3680	3840	4800

Volume measured by:

Date:

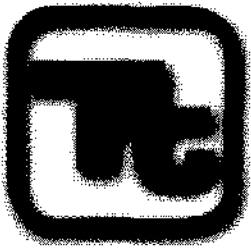
TE, CL, HH	HH, BL	HH, TN
9/12/02-9/13/02	9/14/02	9/16/02

TABLE B-5 URINE ANALYTICAL RESULTS

Tag Number	Pig Number	Group	Day	Material Administered	Target Dose (ug/d)	Q	Arsenic Conc in Urine	DL	Units
R1-01-0194	324	1	6/7	Control	0	<	1	1	ng/mL
R1-01-0265	338	1	6/7	Control	0		1	1	ng/mL
R1-01-0173	349	1	6/7	Control	0		3	1	ng/mL
R1-01-0163	326	2	6/7	NaAs	300		83	1	ng/mL
R1-01-0200	330	2	6/7	NaAs	300		160	2	ng/mL
R1-01-0191	339	2	6/7	NaAs	300		29	1	ng/mL
R1-01-0228	350	2	6/7	NaAs	300		45	1	ng/mL
R1-01-0232	310	3	6/7	NaAs	600		110	2	ng/mL
R1-01-0199	316	3	6/7	NaAs	600		49	1	ng/mL
R1-01-0112	322	3	6/7	NaAs	600		73	1	ng/mL
R1-01-0250	340	3	6/7	NaAs	600		160	2	ng/mL
R1-01-0167	303	4	6/7	NaAs	900		170	2	ng/mL
R1-01-0220	315	4	6/7	NaAs	900		101	1	ng/mL
R1-01-0263	329	4	6/7	NaAs	900		70	1	ng/mL
R1-01-0233	341	4	6/7	NaAs	900		300	4	ng/mL
R1-01-0136	301	5	6/7	TM1	300		56	1	ng/mL
R1-01-0261	318	5	6/7	TM1	300		42	1	ng/mL
R1-01-0260	344	5	6/7	TM1	300		57	1	ng/mL
R1-01-0159	347	5	6/7	TM1	300		14	1	ng/mL
R1-01-0148	309	6	6/7	TM1	600		24	1	ng/mL
R1-01-0187	327	6	6/7	TM1	600		66	1	ng/mL
R1-01-0156	343	6	6/7	TM1	600		36	1	ng/mL
R1-01-0208	346	6	6/7	TM1	600		23	1	ng/mL
R1-01-0121	306	7	6/7	TM1	900		65	1	ng/mL
R1-01-0165	308	7	6/7	TM1	900		39	1	ng/mL
R1-01-0193	317	7	6/7	TM1	900		138	1	ng/mL
R1-01-0171	331	7	6/7	TM1	900		42	1	ng/mL
R1-01-0225	304	8	6/7	TM2	300		49	1	ng/mL
R1-01-0183	311	8	6/7	TM2	300		11	1	ng/mL
R1-01-0117	314	8	6/7	TM2	300		44	1	ng/mL
R1-01-0118	321	8	6/7	TM2	300		25	1	ng/mL
R1-01-0177	307	9	6/7	TM2	600		40	1	ng/mL
R1-01-0152	313	9	6/7	TM2	600		23	1	ng/mL
R1-01-0234	325	9	6/7	TM2	600		104	1	ng/mL
R1-01-0172	332	9	6/7	TM2	600		66	1	ng/mL
R1-01-0114	328	10	6/7	TM2	900		56	1	ng/mL
R1-01-0164	337	10	6/7	TM2	900		160	2	ng/mL
R1-01-0147	342	10	6/7	TM2	900		57	1	ng/mL
R1-01-0186	348	10	6/7	TM2	900		150	2	ng/mL
R1-01-0120	324	1	8/9	Control	0		2	1	ng/mL
R1-01-0237	338	1	8/9	Control	0		3	1	ng/mL
R1-01-0123	349	1	8/9	Control	0		3.6	1	ng/mL
R1-01-0139	326	2	8/9	NaAs	300		75	1	ng/mL
R1-01-0221	330	2	8/9	NaAs	300		270	5	ng/mL
R1-01-0107	339	2	8/9	NaAs	300		73	1	ng/mL
R1-01-0243	350	2	8/9	NaAs	300		71	1	ng/mL
R1-01-0189	310	3	8/9	NaAs	600		240	5	ng/mL
R1-01-0213	316	3	8/9	NaAs	600		24	1	ng/mL
R1-01-0111	322	3	8/9	NaAs	600		130	2	ng/mL

Tag Number	Pig Number	Group	Day	Material Administered	Target Dose (ug/d)	Q	Arsenic Conc in Urine	DL	Units
R1-01-0145	340	3	8/9	NaAs	600		240	5	ng/mL
R1-01-0132	303	4	8/9	NaAs	900		140	2	ng/mL
R1-01-0257	315	4	8/9	NaAs	900		70	1	ng/mL
R1-01-0240	329	4	8/9	NaAs	900		83	1	ng/mL
R1-01-0188	341	4	8/9	NaAs	900		240	5	ng/mL
R1-01-0215	301	5	8/9	TM1	300		77	1	ng/mL
R1-01-0133	318	5	8/9	TM1	300		48	1	ng/mL
R1-01-0218	344	5	8/9	TM1	300		39	1	ng/mL
R1-01-0255	347	5	8/9	TM1	300		19	1	ng/mL
R1-01-0138	309	6	8/9	TM1	600		29	1	ng/mL
R1-01-0170	327	6	8/9	TM1	600		65	1	ng/mL
R1-01-0251	343	6	8/9	TM1	600		60	1	ng/mL
R1-01-0141	346	6	8/9	TM1	600		24	1	ng/mL
R1-01-0127	306	7	8/9	TM1	900		66	1	ng/mL
R1-01-0258	308	7	8/9	TM1	900		51	1	ng/mL
R1-01-0205	317	7	8/9	TM1	900		160	5	ng/mL
R1-01-0161	331	7	8/9	TM1	900		58	1	ng/mL
R1-01-0242	304	8	8/9	TM2	300		39	1	ng/mL
R1-01-0253	311	8	8/9	TM2	300		11	1	ng/mL
R1-01-0166	314	8	8/9	TM2	300		52	1	ng/mL
R1-01-0262	321	8	8/9	TM2	300		19	1	ng/mL
R1-01-0105	307	9	8/9	TM2	600		28	1	ng/mL
R1-01-0134	313	9	8/9	TM2	600		32	1	ng/mL
R1-01-0185	325	9	8/9	TM2	600		98	1	ng/mL
R1-01-0113	332	9	8/9	TM2	600		80	1	ng/mL
R1-01-0144	328	10	8/9	TM2	900		63	1	ng/mL
R1-01-0101	337	10	8/9	TM2	900		440	10	ng/mL
R1-01-0210	342	10	8/9	TM2	900		54	1	ng/mL
R1-01-0196	348	10	8/9	TM2	900		190	5	ng/mL
R1-01-0202	324	1	10/11	Control	0	<	1	1	ng/mL
R1-01-0239	338	1	10/11	Control	0		1	1	ng/mL
R1-01-0142	349	1	10/11	Control	0		3	1	ng/mL
R1-01-0192	326	2	10/11	NaAs	300		40	1	ng/mL
R1-01-0224	330	2	10/11	NaAs	300		130	2	ng/mL
R1-01-0229	339	2	10/11	NaAs	300		33	1	ng/mL
R1-01-0108	350	2	10/11	NaAs	300		60	1	ng/mL
R1-01-0209	310	3	10/11	NaAs	600		74	1	ng/mL
R1-01-0207	316	3	10/11	NaAs	600		31	1	ng/mL
R1-01-0131	322	3	10/11	NaAs	600		100	1	ng/mL
R1-01-0219	340	3	10/11	NaAs	600		120	2	ng/mL
R1-01-0254	303	4	10/11	NaAs	900		96	1	ng/mL
R1-01-0125	315	4	10/11	NaAs	900		102	1	ng/mL
R1-01-0236	329	4	10/11	NaAs	900		68	1	ng/mL
R1-01-0264	341	4	10/11	NaAs	900		180	5	ng/mL
R1-01-0109	301	5	10/11	TM1	300		110	2	ng/mL
R1-01-0231	318	5	10/11	TM1	300		58	1	ng/mL
R1-01-0176	344	5	10/11	TM1	300		43	1	ng/mL
R1-01-0128	347	5	10/11	TM1	300		13	1	ng/mL
R1-01-0227	309	6	10/11	TM1	600		24	1	ng/mL
R1-01-0129	327	6	10/11	TM1	600		40	1	ng/mL
R1-01-0115	343	6	10/11	TM1	600		28	1	ng/mL
R1-01-0204	346	6	10/11	TM1	600		24	1	ng/mL

Tag Number	Pig Number	Group	Day	Material Administered	Target Dose (ug/d)	Q	Arsenic Conc in Urine	DL	Units
R1-01-0160	306	7	10/11	TM1	900		51	1	ng/mL
R1-01-0150	308	7	10/11	TM1	900		52	1	ng/mL
R1-01-0143	317	7	10/11	TM1	900		190	5	ng/mL
R1-01-0248	331	7	10/11	TM1	900		54	1	ng/mL
R1-01-0238	304	8	10/11	TM2	300		62	1	ng/mL
R1-01-0178	311	8	10/11	TM2	300		9.5	1	ng/mL
R1-01-0217	314	8	10/11	TM2	300		50	1	ng/mL
R1-01-0214	321	8	10/11	TM2	300		32	1	ng/mL
R1-01-0252	307	9	10/11	TM2	600		31	1	ng/mL
R1-01-0245	313	9	10/11	TM2	600		33	1	ng/mL
R1-01-0256	325	9	10/11	TM2	600		120	2	ng/mL
R1-01-0216	332	9	10/11	TM2	600		120	2	ng/mL
R1-01-0149	328	10	10/11	TM2	900		39	1	ng/mL
R1-01-0246	337	10	10/11	TM2	900		160	5	ng/mL
R1-01-0174	342	10	10/11	TM2	900		26	1	ng/mL
R1-01-0103	348	10	10/11	TM2	900		130	2	ng/mL
R1-01-0222	2340	3	6/7	NaAs	600		160	2	ng/mL
R1-01-0180	2306	7	6/7	TM1	900		61	1	ng/mL
R1-01-0244	2307	9	6/7	TM2	600		37	1	ng/mL
R1-01-0104	2329	4	8/9	NaAs	900		83	1	ng/mL
R1-01-0247	2346	6	8/9	TM1	600		28	1	ng/mL
R1-01-0110	2314	8	8/9	TM2	300		53	1	ng/mL
R1-01-0212	2330	2	10/11	NaAs	300		130	2	ng/mL
R1-01-0182	2344	5	10/11	TM1	300		44	1	ng/mL
R1-01-0151	2348	10	10/11	TM2	900		130	2	ng/mL
R1-01-0157	AsCtrl	PE		Control	0		3	1	ng/mL
R1-01-0206	AsCtrl	PE		Control	0		2	1	ng/mL
R1-01-0119	AsIA200	PE		Sodium arsenate	200		180	4	ng/mL
R1-01-0124	AsIA200	PE		Sodium arsenate	200		190	5	ng/mL
R1-01-0198	AsIA40	PE		Sodium arsenate	40		42	1	ng/mL
R1-01-0158	AsIA40	PE		Sodium arsenate	40		41	1	ng/mL
R1-01-0122	AsIB200	PE		Sodium arsenite	200		190	4	ng/mL
R1-01-0175	AsIB200	PE		Sodium arsenite	200		200	5	ng/mL
R1-01-0106	AsIB40	PE		Sodium arsenite	40		43	1	ng/mL
R1-01-0230	AsIB40	PE		Sodium arsenite	40		41	1	ng/mL
R1-01-0241	AsOA200	PE		MMA	200		200	4	ng/mL
R1-01-0130	AsOA200	PE		MMA	200		210	5	ng/mL
R1-01-0135	AsOA40	PE		MMA	40		43	1	ng/mL
R1-01-0169	AsOA40	PE		MMA	40		43	1	ng/mL
R1-01-0116	AsOB200	PE		DMA	200		200	4	ng/mL
R1-01-0203	AsOB200	PE		DMA	200		210	5	ng/mL
R1-01-0249	AsOB40	PE		DMA	40		44	1	ng/mL
R1-01-0154	AsOB40	PE		DMA	40		44	1	ng/mL



LETTER OF TRANSMITTAL

TETRA TECH NUS, Inc.
55 Jonspin Road
Wilmington, Massachusetts 01887
(978) 658-7899

Correspondence No. RAC1-EPA-5235

TO: Diane Silverman
Metcalf & Eddy, Inc
701 Edgewater Drive
Wakefield, MA 01880-5371

DATE: 06-24-04 JOB NO.: N4123-0132
ATTENTION:
REGARDING: Arsenic Bioavailability Study
Industri-plex Site
Woburn, MA

WE ARE SENDING YOU ☒ Attached ☐ Under separate cover via _____ the following items:
☐ Shop drawings ☐ Prints ☐ Plans ☐ Samples ☐ Specifications
☐ Copy of letter ☐ Change Order ☐ Submittals ☐ _____

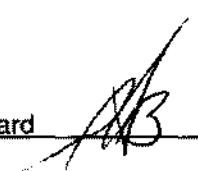
COPIES	DATE	NUMBER	DESCRIPTION
1			<u>Arsenic Bioavailability Sample Log Sheets</u>

THESE ARE TRANSMITTED as checked below:

- | | | |
|---|---|---|
| ☐ For approval | ☐ Approved as submitted | ☐ Resubmit _____ copies for approval |
| ☒ For your use/File | ☐ Approved as noted | ☐ Submit _____ copies for distribution |
| ☐ As requested | ☐ Return for corrections | ☐ Return _____ corrected prints |
| ☐ For review and comment | ☐ _____ | |

REMARKS:

COPY TO: N4123-1.0

SIGNED: Gordon Bullard 

Bioavailability Study - 7/9/02 - Re-sample 8/15/02

10.

1



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD-ABS-01-081502

Tetra Tech NUS Job No./PMS: N4123-0322

QC Information:

NA

(if applicable)

Sample Method:

Scoop w/ Stainless Steel Scoop

Depth Sampled:

0-0.5 feet

Sample Date & Time:

8/15/2002

1115

hours

Duplicate:

NA

hours

Sampler(s):

J. Daniel

K. O'Neill

J. Lambert

16 Bullard

(circle appropriate)

Data Recorded By:

J. Daniel

(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

Soil

Trip Blank*

☒ Sediment☐ Rinsate Blank*☐ Lagoon/Pond☐ Field Duplicate collected☒ Grab☐ Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

Wet, muck, abundant organics.
sandy

*include DIUF lot # in "other"

ANALYSIS	Check	NOTES/SKETCH:
As - Bioavailability	☒	old HBO108 Location
		DO 17.57
		DO% 236.9%
		Temp 30.13°C
		Bar. 165.7
		~ 0.4ft of Surface water over muck.
		Resample of location Sampled on 7/8/02 See sketch on 7/8/02 Log Sheet Sample ~ 5ft South of Sample Collected on 7/8/02



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD- ABS- 02-081502

Tetra Tech NUS Job No./PMS: N4123-0322

QC Information:

NA

(if applicable)

Sample Method: Scoop w/ Stainless Steel Scoop

Depth Sampled: 0-0.5 feet

Sample Date & Time: 8/15/2001

1320 hours

Duplicate: NA hours

Sampler(s): J. Danielli / K. O'Neill / J. Lambert

(military)
16 Bullard

(circle appropriate)

Data Recorded By:

J. Danielli

(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil☐ Trip Blank*☒ Sediment☐ Rinsate Blank*☐ Lagoon/Pond☐ Field Duplicate collected☒ Grab☐ Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

mucky Peat - Wet

Brown - Black

*include DIUF lot # in "other"

ANALYSIS	Check	NOTES/SKETCH: Old HB02-04 Location	Resample of location sampled on 7/8/02 See sketch on 7/8/02 Log sheet Sample ~ 3ft West of sample collected on 7/8/02.
As - Bioavailability	☒	DO 1.03 mg/L	
		DO% 13.6 %	
		Temp 28.53 °C	
		Bar	
		Pressure mmHg	
		765.3	
		Surface Water - @ Sediment Surface	



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Tetra Tech NUS Job No./PMS: N4123-0322

Sample ID: IPSD-ABS-03-081302

QC Information: NA (if applicable)

Sample Method: Scoop w/ Stainless Steel Scoop

Depth Sampled: 0-0.5 feet

Sample Date & Time: 8/15/2008 1054 hours Duplicate: NA hours

Sampler(s): J. Daniel / K. O'Neill / J. Lambert / ^(Military) G. Bullard (circle appropriate)

Data Recorded By: J. Daniel (Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil ☐ Trip Blank*
☒ Sediment ☐ Rinsate Blank*
☐ Lagoon/Pond ☐ Field Duplicate collected
☒ Grab ☐ Composite

Other _____

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.) Wet, Black, muck.

*include DIUF lot # in "other"

ANALYSIS	Check	NOTES/SKETCH: old HB03-0889 location 8/15/08
As - Bioavailability	☒	DD 0.89mg/L
		DO 11.7 %
		Temp 26.77°C
		Bar.
		Pressure 766.7 mmHg
		~ 0.15 ft of S. water over muck.

Re-sample of location
 Sampled on 7/8/02
 See sketch on 7/8/02
 Log sheet.
 Sample 2 ft ^{5H} South
 From sample
 Collected on
 7/8/02.



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD-ABS-04 - 081502

Tetra Tech NUS Job No./PMS: N4123- 0322

QC Information:

NA

(if applicable)

Sample Method:

Scoop w/ Stainless Steel Scoop

Depth Sampled:

0-0.5 feet

Sample Date & Time:

8/15/2002

0827

hours

Duplicate:

NA

hours

Sampler(s):

J. Daniel

K. O'Neil

J. Lambert

G. Bullard

(circle appropriate)

Data Recorded By:

J. Daniel

(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil☐ Trip Blank*☒ Sediment☐ Rinsate Blank*☐ Lagoon/Pond☐ Field Duplicate collected☒ Grab☐ Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

No Standing Water

Mucky Peat - abundant organic

DK Brown Moist

*include DIUF lot # in "other"

ANALYSIS	Check	NOTES/SKETCH: Old SD-12-01 Location
As - Bioavailability	☒	DO 0.65 mg/L
		DO% 7.2%
		Temp 20.98°C
		Bar. 766.4 mmHg
		Pressure

Re-sample of location
 Sampled on 7/8/02
 See sketch on 7/8/02
 Log Sheet
 Sample 2 ft from sample
 location on 7/8/02.



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD-ABS-05 - 081502

Tetra Tech NUS Job No./PMS: N4123- 0322

Information: NA

(if applicable)

Sample Method:

Scoop w/ Stainless Steel Scoop

Depth Sampled:

0-0.5 feet

Sample Date & Time:

8/15/2002

0853

hours

Duplicate:

NA

hours

Sampler(s):

J. Danielli

K. O'Neill

J. Lambert

G. Bullard

(circle appropriate)

Data Recorded By:

J. Danielli

(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

Soil

☒ Sediment

Lagoon/Pond

☒ Grab

Trip Blank *

Rinsate Blank *

Field Duplicate collected

Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

No Standing Water

Peak - abundant Organics

Dk. Brown

*include DIUF lot # in "other"

ANALYSIS	Check	NOTES/SKETCH:
As - Bioavailability	☒	Old NW-07 Location
		DO 0.28 mg/L
		DO% 3.1%
		Temp. 20.36 °C
		Bar.
		Pressure = 766.1 mmHg
		Depth to water below Surface = 0.55 ft
		Re-sample of location Sampled on 7/8/02. See sketch on 7/8/02 Log sheet. Sample 5 ft from sample location on 7/8/02.



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD - ABS - 010 - 081502

Tetra Tech NUS Job No./PMS: N4123- 0322

QC Information:

NA

(if applicable)

Sample Method:

Scoop w/ Stainless Steel Scoop.

Depth Sampled:

0-0.5 feet

Sample Date & Time:

8/15/2002

0919

hours

Duplicate:

NA

hours

Sampler(s): J. Daniel

K. O'Neill

J. Lambert

G. Bullard

(circle appropriate)

Data Recorded By:

J. Daniel

(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil☐ Trip Blank *☒ Sediment☐ Rinsate Blank *☐ Lagoon/Pond☐ Field Duplicate collected☒ Grab☐ Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

No standing water. Sulfide odor
Wet - DK Brown - Mucky Peat

*include DIUF lot # in "other"

ANALYSIS	Check	NOTES/SKETCH: old WS08 Location
As - Bioavailability	☒	DO 1.65 mg/L
		DO% 19.8%
		Temp. 24.23 °C
		Bar.
		Pressure 766-3 mmHg
		Depth to water = 0.1 ft
		from surface of Sediment.

Re-sample of
location Sampled on
7/8/02
See sketch on
7/8/02 Log Sheet
Sample 5 ft South
from 7/8/02 location



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD - ABS - 07 - 081502

Tetra Tech NUS Job No./PMS: N4123- 0322

QC Information:

NA

(if applicable)

Sample Method:

Scoop w/ Stainless Steel Scoop

Depth Sampled:

0-0.5 feet

Sample Date & Time:

8/15/2002

0949 hours

Duplicate: NA hours

Sampler(s): J. Danieli /

K. O'Neill /

J. Lambert

16 Bullard

(circle appropriate)

Data Recorded By:

J. Danieli

(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil☒ Sediment☐ Lagoon/Pond☒ Grab☐ Trip Blank*☐ Rinsate Blank*☐ Field Duplicate collected☐ Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

muck - wet - abundant decompose
No standing water Plant Fiber material

*include DIUF lot # in "other"

ANALYSIS	Check	NOTES/SKETCH: Old CB03-06 Location	Resample of location sampled on 7/8/02
As - Bioavailability	☒	DO 0.19 mg/L	See sketch on 7/8/02 log sheet
		DO% 2.2 %	Sample 2ft West of sample collected on 7/8/02
		Temp 24.13 °C	
		Bar.	
		Pressure 766.0 mmHg	
		Depth to water: 0.3ft - below surface of Sediment.	



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID: IPSD - ABS - 08 - 081502Tetra Tech NUS Job No./PMS: N4123- 0322QC Information: NA (if applicable)

Sample Method: _____

Depth Sampled: 0 - 0.5 feetSample Date & Time: 8/15/2002 1000 hours Duplicate: NA hoursSampler(s): J. Danieli / K. O'Neill / J. Lambert / G. Bullard (circle appropriate)Data Recorded By: J. Danieli
(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil	☐ Trip Blank*
☒ Sediment	☐ Rinsate Blank*
☐ Lagoon/Pond	☐ Field Duplicate collected
☒ Grab	☐ Composite

Other _____

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.) muck - abundant decomposedLeaves & Root matterDK Brown
*include DIUF lot # in "other"

ANALYSIS	Check	NOTES/SKETCH: Old CB03-11 location.	Resample of location sampled on 7/8/02.
As - Bioavailability	☒	<u>000273 - 08/15/02</u> <u>mg/L</u>	
		<u>DO% 3.6 %</u>	
		<u>Temp. 20.63 °C</u>	
		<u>Bar.</u>	
		<u>Pressure. 766.4 mmHg</u>	
		<u>Depth to water = 0.3 Ft below</u> <u>Surface of Sediment</u>	
			<u>See sketch on</u> <u>7/8/02 log sheet</u> <u>Sample 2ft South</u> <u>of Sample</u> <u>Collected on 7/8/02</u>



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD - ABS - 09 - 081502

Tetra Tech NUS Job No./PMS: N4123- 082a

QC Information: NA

(if applicable)

Sample Method:

Scoop w/ Stainless Steel Scoop

Depth Sampled:

0-0.5 feet

Sample Date & Time:

8/15/2002

1014

hours

Duplicate:

NA

hours

Sampler(s):

J. Daniel

K. O'Neill

J. Lambert

(military)

K. Bullard

(circle appropriate)

Data Recorded By:

J. Daniel

(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

Soil

Trip Blank *

☒ Sediment

Rinsate Blank *

☐ Lagoon/Pond

Field Duplicate collected

☒ Grab

Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

NO Standing Water
MUCK - MOIST - decayed leaves

*include DIUF lot # in "other"

ANALYSIS	Check	NOTES/SKETCH: Old CB03-09 Location	Resample of location sampled on 7/8/02
As - Bioavailability	☒	DO 2.02	See Sample log
		DO% 22.10%	Sheet for 7/8/02 for sketch
		Temp 20.52°C	Sample 2ft + North
		Bar	From Sample collected on 7/8/02
		Pressure: 7660 mmHg	
		Depth to water from surface of sed: 0.3ft	



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD - ABS - 10 - 081502

Tetra Tech NUS Job No./PMS: N4123- 0322

QC Information:

NA

(if applicable)

Sample Method:

Scoop w/ stainless steel scoop

Depth Sampled:

0-0.5 feet

Sample Date & Time:

8/15/2008

1446

hours

Duplicate: NA

hours

Sampler(s): J. Danielli / K. O'Neill / J. Lambert

(military)

G. Bullard

(circle appropriate)

Data Recorded By:

J. Danielli

(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil☐ Trip Blank*☒ Sediment☐ Rinsate Blank*☐ Lagoon/Pond☐ Field Duplicate collected☒ Grab☐ Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

MUCK - w/ Roots - trace sand + Gravel

No standing water @ sample location

*include DIUF lot # in "other"

water within 1 ft of sample location.

ANALYSIS	Check	NOTES/SKETCH: old 8007-10 location
As - Bioavailability	☒	DO 10.06mg/L
		100% 141.3%
		Temp. 33.17°C
		Bar.
		Pressure 765.0mmHg

Resample of location sampled on 7/8/02. See sketch on 7/8/02 log sheet.

Sample ~.3ft south of sample collected on 7/8/02



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD - ABS - 11 - 081502

Tetra Tech NUS Job No./PMS: N4123- 0302

QC Information:

NA

(if applicable)

Sample Method:

Scoop w/ Stainless Steel Scoop

Depth Sampled:

0-0.5 feet

Sample Date & Time:

8/15/2001

1430

hours

Duplicate: NA hours

Sampler(s):

J. Daniel

K. O'Neill

J. Lambert

(Military)

G. Bullard

(circle appropriate)

Data Recorded By:

J. Daniel

(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil☐ Trip Blank*☒ Sediment☐ Rinsate Blank*☐ Lagoon/Pond☐ Field Duplicate collected☒ Grab☐ Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

Muck - Roots

*include DIUF lot # in "other"

ANALYSIS	Check	NOTES/SKETCH:
As - Bioavailability	A	Old SP07-04 location
		DO 8.81 mg/L
		DO % 117.3 %
		Temp 30.50 °C
		Bar.
		Pressure 765.3 mmHg
		Re Sample of location sampled on 7/8/02 See Sketch on 7/8/02 log sheet
		water depth 0.46'



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD-ABS-12 - 081502

Tetra Tech NUS Job No./PMS: N4123-0322

QC Information:

NA

(if applicable)

Sample Method:

Scoop w/ Stainless Steel Scoop

Depth Sampled:

0-0.5

feet

Sample Date & Time:

8/15/2002

1405

hours

Duplicate:

NA

hours

Sampler(s):

J. Daniel

/ K. O'Neill

/ J. Lambert

/ ^(Military) G. Bullard

(circle appropriate)

Data Recorded By:

J. Daniel

(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil☐ Trip Blank*☒ Sediment☐ Rinsate Blank*☐ Lagoon/Pond☐ Field Duplicate collected☒ Grab☐ Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

*include DIUF lot # in "other"

ANALYSIS	Check	NOTES/SKETCH: old 5007-05 location	Resample of location sampled on 7/8/02
As - Bioavailability	☒	DO = 1.86 mg/L	See sketch on 7/8/02 log sheet
		DO% = 26.1 %	
		Temp = 30.75 °C	
		Bar.	
		Pressure = 766.7 mmHg	
			water depth 0.52'
			Sample ~ 2-5ft
			East of sample
			Collected on 7/8/02



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD-ABS-01-074802

Tetra Tech NUS Job No./PMS: N4123-0322

AC Information: NA (if applicable)

Sample Method:

Scoop w/ Stainless Steel Scoop

Depth Sampled:

0-0.5 feet

Sample Date & Time:

7/8/2002

1130

hours
(military)

Duplicate: NA hours

Sampler(s):

J. Danieli

K. O'Neill

J. Lambert

(circle appropriate)

Data Recorded By:

J. Danieli
(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil☐ Trip Blank*☒ Sediment☐ Rinsate Blank*☐ Lagoon/Pond☐ Field Duplicate collected☒ Grab☐ Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

Organic muck, wet, Black,
Trace silt + sand
~ 12" of water over sediment
*include DIUF lot # in "other" Blue shanon water

ANALYSIS

Check

As - Bioavailability

☒

NOTES/SKETCH: Old HB01-08 location

DO: 2.78

DO%: 32.7%

Temp: 26.99°C

Bar.

Pressure 762.2 mmHg

N ↑

K.R.

Tracks →

7 MBTA
Right of Way

Halls Brook Holding Area

Teradyne



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID: _____

IPSD - ABS-02 - 070802

Tetra Tech NUS Job No./PMS: N4123- 0322

AC Information: NA (if applicable)

Sample Method: _____

Scoop w/ stainless steel scoop

Depth Sampled: 0-0.5 feet

Sample Date & Time: 1/8/2002 1705 hours

Duplicate: NA hours

Sampler(s): J. Danieli / K. O'Neill / J. Lambert

(circle appropriate)

Data Recorded By: _____

J. Danieli
(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil ☐ Trip Blank*
☒ Sediment ☐ Rinsate Blank*
☐ Lagoon/Pond ☐ Field Duplicate collected
☒ Grab ☐ Composite

Other _____

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

OK Brown, organic muck w/ fine
 Root matter & phragmites (decayed) - soupy.
 Initially 2" of water Dush over debris -
 *include DIUF lot # in "other" more water

ANALYSIS	Check	NOTES/SKETCH: Old HB02-04 Location
As - Bioavailability	☒	Temp: 26.92 °C Bar.: 762.5 mmHg DO: 1.58 mg/L DO%: 19.1% RR Tracks N MBTA Right of Way HBHA road Tetachyne ABS-02



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD-ABS-03-070802

Tetra Tech NUS Job No./PMS: N4123- 0322

QC Information: NA (if applicable)

Sample Method:

Scoop w/ Stainless Steel

Depth Sampled:

0-0.5

feet

Scoop

Sample Date & Time:

7/8/2002

1610

hours

Duplicate: NA hours

(military)

Sampler(s):

J. Daniel

K. O'Neill

J. Lambert

(circle appropriate)

Data Recorded By:

J. Daniel

(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil☐ Trip Blank*☒ Sediment☐ Rinsate Blank*☐ Lagoon/Pond☐ Field Duplicate collected☒ Grab☐ Composite

Other _____

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

Wet, dk brown, organic muck

w/ sticks and sand.

~ 6" of water over sediment.

*include DIUF lot # in "other"

ANALYSIS	Check	NOTES/SKETCH: Old ABS-09 location = ~5ft from old location - closer to phragmites to west.
As - Bioavailability	☒	DO 1.22 mg/L N DOO% 16.1 % Temp 28.77 Bar. Pressure: 263.4
	☐	
	☐	
	☐	
	☐	
	☐	
		Halls Brook Holding Area Roux SW Station ABS-03 mishawum Rd.



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD-ABS-04-070802

Tetra Tech NUS Job No./PMS

N1123 0322

QC Information:

NA

(if applicable)

Sample Method:

Scoop w/ Stainless Steel Shovel

Depth Sampled:

0-0.5 feet

Sample Date & Time:

7/8/2002

1045

hours

Duplicate: NA hours

Sampler(s):

J. Danieli /

K. O'Neill /

J. Lambert

(circle appropriate)

Data Recorded By:

J. Danieli

(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: Check all that apply)

☐ Soil☒ Sediment☐ Lagoon/Pond☒ Grab

Other

☐ Trip Blank*☐ Rinsate Blank*☐ Field Duplicate collected☐ Composite

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

Brown, wet, muck,

abundant organics

water ~ 2" over sediment.

*include DIUF lot # in "other"

ANALYSIS	Check	NOTES/SKETCH: Old SD-12-ME Location
As - Bioavailability	X	ABS-04 Location. Wetlands Tree Line Shooting Range Rifle Club.

DO Reading

% = 41.4%

mg/L = 3.27 mg/L

Temp = 19.40°C

Bar. Pressure = 764.8



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSP-ABS-05-070802

Tetra Tech NUS Job No./PMS: N4123-0322

QC Information:

N/A

(if applicable)

Sample Method:

scoop w/ stainless steel scoop

Depth Sampled:

0-0.5

feet

Sample Date & Time:

7/8/2002

1115

hours

Duplicate:

N/A

hours

Sampler(s):

J. Danielli /

K. O'Neil /

J. Lambert

(military)

(circle appropriate)

Data Recorded By:

J. Danielli

(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil☐ Trip Blank*☒ Sediment☐ Rinsate Blank*☐ Lagoon/Pond☐ Field Duplicate collected☒ Grab☐ Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

Brown, Peat, trace sand and silt
roots - abundant organics.

*include DIUF lot # in "other"

ANALYSIS	Check	NOTES/SKETCH:
As - Bioavailability	X	Old WG-07 Location. II III wetlands I I DO 5.50 mg/L DO% 62.4 Temp 19.24 °C Bar. 765.4 mmHg Pressure ABS-05 (X) II Road Shooting Range Rifle Club N



SAMPLE LOG SHEET - SEDIMENT

Sample ID: IPSD-ABS-06-070802

0322

QC Information: NA (if applicable)

Sample Method: Scoop w/ Stainless Scoop

Depth Sampled: 0-0.5 feet

Sample Date & Time: 7/8/2002 11:45 hours Duplicate: NA hours

Sampler(s): J. Danielli / K. O'Neill / J. Lambert

Data Recorded By: J. Kinnell (Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil	☐ Trip Blank*
☒ Sediment	☐ Rinsate Blank*
☐ Lagoon/Pond	☐ Field Duplicate collected
☒ Grab	☐ Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.) moist, abundant organs -
Roots, Peat.

*include DIUF lot # in "other"

[illegible]



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD-ABS-08-070802

Tetra Tech NUS Job No./PMS: N4123-0322

QC Information:

NA

(if applicable)

Sample Method:

Scoop w/ stainless steel scoop

Depth Sampled:

0-0.5

feet

Sample Date & Time:

7 / 8 / 2008

1315

hours

Duplicate:

NA

hours

Sampler(s):

J. Danieli

K. O'Neill

J. Lamber

(circle appropriate)

Data Recorded By:

J. Danieli

(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil☐ Trip Blank*☒ Sediment☐ Rinsate Blank*☐ Lagoon/Pond☐ Field Duplicate collected☒ Grab☐ Composite

Other:

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

~4 inches of water over sediment
 water has a green / manganese flocc
 (natural) - 2-3 partially decayed leaves
 *include DIUF lot # in "other" over sediment removed
 organic muck.

ANALYSIS	Check	NOTES/SKETCH: old CBO 3-11 location
As - Bioavailability	☒	DO = 0.73
		DOO16 = 8.7
		Temp = 20.53
		Bar. Pressure = 763.5

wetlands

ABS-08

standing water

embankment - 34' drop

30 ft

road

trees



TETRA TECHNUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD-ABS-09-070602

Tetra Tech NUS Job No./PMS: N4123-0322

QC Information: **NP** (if applicable)

Sample Method:

Scoop with stainless steel scoop

Depth Sampled:

0-0.5 feet

Sample Date & Time:

7/8/2002

1308

08 hours
(military)

Duplicate: **NA** hours

110

(circle appropriate)

Sampler(s):

U. Daniel

(K. O'Neill)

J. Lambert

Data Recorded By:

J. Daniels

(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

Soil

 Sediment

Lagoon/Pond

Grab

Trio Blank*

Rinsate Blank*

Field Duplicate collected

Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.) ~ 1" of water over sediment

~~234 partially degraded leaves~~
Abundant organic muck, brown.
*include DIUF lot # in "other" Flac. on water
Surface

[illegible]

NOTES/SKETCH: Old CR03-09 location.

PO : 3.59 mg/L

DO% : 25.8%

Temp: 18.91

Boy.

Bar. pressure: 763.9 mmHg

Hand-drawn map of the Ab. River area. The map shows a river flowing from the top right towards the bottom left. A bridge is located on the river. To the left of the river is a line of trees. To the right of the river is a line of trees. At the bottom left, there is a circled 'X' labeled 'Wetlands'. The text 'ABS-09.' is written at the bottom left.



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD-ABS-10-020802

Tetra Tech NUS Job No./PMS: N4123-0322

QC Information: NA (if applicable)

Sample Method:

Scoop w/ Stainless Steel Scoop.

Depth Sampled:

0-0.5 feet

Sample Date & Time:

7/8/2002

1447-1500 hours
(military)

Duplicate: NA hours

Sampler(s):

J. Daniel

K. O'Neill

J. Lambert

(circle appropriate)

Data Recorded By:

J. Daniel
(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil☒ Sediment☐ Lagoon/Pond☒ Grab☐ Trip Blank*☐ Rinsate Blank*☐ Field Duplicate collected☐ Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.)

Organic muck w/ wood
debris sticks - trace silt.
~ 12" of water over sediment.

*include DIUF lot # in "other"

ANALYSIS	Check
As - Bioavailability	☒

NOTES/SKETCH:

Old SD07-02 location. SD07-04 (FW) location

DO: 6.85 mg/L 14.16 ? mg/L

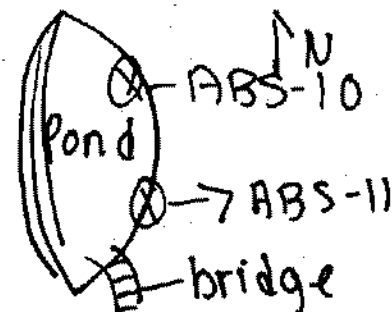
DO%: 82.6 % 190.5 % ?

Temp: 24.98 °C 31.06

Bar.

Pressure: 763.4 mmHg 763.8 mmHg

7/8/02



SAMPLE LOG SHEET - SEDIMENT

IPSD-ABS-11-070802

QC Information:

AD

(if applicable)

Scoop w/ stainless
Steel Scoop

Depth Sampled: 0-0.5 feet

Sample Date & Time: 7 / 8 / 2001

1447

hours

Duplicate: ~~1) 1~~ hours

Sampler(s): J. Danieli/ K. O'Neill/ J. Lambert

(circle appropriate)

Data Recorded By:

J. Daniels
(Signature)

Survey Meter/Monitor Reading: **Not used**

TYPE OF SAMPLE: (Check all that apply)

Soil

v Sediment

 Lagoon/Pond

✓ Grab

Trip Blank*

Rinsate Blank*

Field Duplicate collected

Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.) Organic muck w/ fine sand (2)

and wooded debris - sticks
~12" of water over sediment.

*include DIUF lot # in "other"

[illegible]

NOTES/SKETCH: Old SP07-04 CFW) location

DO: 6.85 mg/L

DO%: 82.6 %

Temp: 24.98 °C

Bar.

Pressure: 763.4 mmHg

A hand-drawn map of the study area. At the top, there is a north arrow pointing upwards, labeled 'N'. Below the arrow is a large, irregular shape representing a pond, labeled 'Pond'. To the right of the pond is a location marked 'ABS-1'. Below 'ABS-1' is a small structure labeled 'bridge' with a ladder-like shape next to it, indicating a crossing point.

Cross Street



TETRA TECH NUS, INC.

SAMPLE LOG SHEET - SEDIMENT

Site Name: Industri-Plex

Sample ID:

IPSD - ABS-12-070802

QC Information:

Tetra Tech NUS Job No./PMS: N4123-0322

(if applicable)

Sample Method:

Scoop w/ Stainless Steel Scoop

Depth Sampled: 0-0.51 feet

Sample Date & Time: 7/2/2002

1420 hours
(military)

Duplicate: NA hours

Sampler(s): J. Danieli / K. O'Neill / J. Lambert

(circle appropriate)

Data Recorded By:

J. Danieli
(Signature)

Survey Meter/Monitor Reading: Not used

TYPE OF SAMPLE: (Check all that apply)

☐ Soil ☐ Trip Blank*
☒ Sediment ☐ Rinsate Blank*
☐ Lagoon/Pond ☐ Field Duplicate collected
☒ Grab ☐ Composite

Other

Description: (Sand, Clay, Muck, Peat, Dry, Moist, Wet, Etc.) ~6' of water over sediment

Silty, sandy, organic muck w/ partially decayed leaves + sticks.

*include DIUF lot # in "other"

ANALYSIS	Check	NOTES/SKETCH: Old SD07-05 (M+E) location.
As - Bioavailability	X	DO = 6.61 $\leq N$ DO% = 80.1% Temp = 24.93 Bar. Pressure = 764.6 mmHg
		Pond Davidson Park ABS-12 Restaurant Cross Street

APPENDIX C.10

LEAD MODEL CALCULATIONS

IEUBK Model Information

Adult Lead Model Information

TABLE C.10-1 (RAGS D IEUBK LEAD WORKSHEET)
Site Name: Wells G&H Superfund Site OU3
Receptor: Young Child (1 to 6 years) Exposure to Media as Described

1. Lead Screening Questions

Medium	Lead Concentration Used in Model Run		Basis for Lead Concentration Used For Model Run	Lead Screening Concentration		Basis for Lead Screening Level
	Value	Units		Value	Units	
Sediment	337	mg/kg	Average Detected Value	400	mg/kg	Recommended Soil Screening Level
Water	4	ug/L	Model Default	15	ug/L	Recommended Drinking Water Action Level

2. Lead Model Questions

Question	Response for Residential Lead Model
What lead model (version and date) was used?	IEUBKwin32 Model 1.0 build 252
Where are the input values located in the risk assessment report?	Located in Appendix C.10, Tables C.10-3 and C.10-4
What range of media concentrations were used for the model?	Refer to Table C.10-3
What statistics were used to represent the exposure concentration terms and where are the data on concentrations in the risk assessment that support use of these statistics?	Arithmetic mean concentrations from Tables 3-3.2.RME/CT and 3-3.3.RME/CT
Was soil sample taken from top 2 cm? If not, why?	No
Was soil sample sieved? What size screen was used? If not sieved, provide rationale.	No
What was the point of exposure/location?	The maximum exposure scenario occurred at Station 22/TT-22
Where are the output values located in the risk assessment report?	Located in Appendix C.10, Table C.10-4 and Figure C.10-1
Was the model run using default values only?	No
Was the default soil bioavailability used?	Yes
Was the default soil ingestion rate used?	Yes
If non-default values were used, where are the rationale for the values located in the risk assessment report?	Located in Appendix C.10, Table C.10-3

3. Final Result

Medium	Result	Comment/PRG ¹
Sediment	Input value of 337 mg/Kg in sediment results in 1.472% of young children above a blood lead level of 10 ug/dL. Geometric mean blood lead = 3.594 ug/dL. This does not exceed the blood lead goal as described in the 1994 OSWER Directive of no more than 5% of children exceeding 10 ug/dL blood lead.	Based on site conditions, a PRG calculation is not necessary.

1. Attach the IEUBK text output file and graph upon which the PRG was based as an appendix. For additional information, see www.epa.gov/superfund/programs/lead

TABLE C.10-2 (RAGS D ADULT LEAD WORKSHEET)
Site Name: Wells G&H Superfund Site OU3
Receptor: Adult Non-Resident, Exposure to Media as Described

1. Lead Screening Questions

Medium	Lead Concentration used in Model Run		Basis for Lead Concentration Used For Model Run	Lead Screening Concentration		Basis for Lead Screening Level
	Value	Units		Value	Units	
Sediment	6765	mg/kg	Average Detected Value	750	mg/kg	Recommended Soil Screening Level

2. Lead Model Questions

Question	Response
What lead model was used? Provide reference and version	Adult Model associated with EPA-540-R-03-001
If the EPA Adult Lead Model (ALM) was not used provide rationale for model selected.	N/A
Where are the input values located in the risk assessment report?	Located in Appendix C.10, Table C.10-5
What statistics were used to represent the exposure concentration terms and where are the data on concentrations in the risk assessment that support use of these statistics?	Arithmetic mean concentrations from Tables 3-3.2.RME/CT and 3-3.3.RME/CT
What was the point of exposure and location?	The maximum exposure scenario occurred at Station 22/TT-22
Where are the output values located in the risk assessment report?	Located in Appendix C.10
What GSD value was used? If this is outside the recommended range of 1.8-2.1), provide rationale in Appendix C.10.	1.8
What baseline blood lead concentration (PbB ₀) value was used? If this is outside the default range of 1.7 to 2.2 provide rationale in Appendix C.10	2.0
Was the default exposure frequency (EF; 219 days/year) used?	No
Was the default BKSF used (0.4 ug/dL per ug/day) used?	Yes
Was the default absorption fraction (AF; 0.12) used?	Yes
Was the default soil ingestion rate (IR; 50 mg/day) used?	Yes
If non-default values were used for any of the parameters listed above, where are the rationale for the values located in the risk assessment report?	Located in Appendix C.10

3. Final Result

Medium	Result	Comment/RBRG ¹
Sediment	Input value of 6765 ppm in soil results in 0.2% of receptors above a blood lead level of 10 ug/dL and geometric mean blood lead = 2.0 ug/dL. This does not exceed the blood lead goal as described in the 1994 OSWER Directive of no more than 5% of children (fetuses of exposed women) exceeding 10 ug/dL blood lead.	Based on site conditions, a RBRG calculation is not necessary.

1. Attach the ALM spreadsheet output file upon which the Risk Based Remediation Goal (RBRG) was based and description of rationale for parameters used. For additional information, see www.epa.gov/superfund/programs/lead

TABLE C.10-3. SEDIMENT/SOIL AND SURFACE WATER IEUBK MODEL INPUTS

Sediment

Station	Average Concentration (mg/Kg)		CT Exposure Frequency (days/yr) ¹		Time-weighted conc. (mg/Kg) ²	
	Current	Future	Current	Future	Current	Future
NR	161		13		102	
14	68		13		68	
22/TT-22	6765		13		337	
13/TT-27	--	700	--	13	--	121
WH	1493		13		150	
NT-1	--	468	--	13	--	113
NT-2	--	420	--	13	--	111
NT-3	--	466	--	13	--	113
WG	429		13		112	
WW	--	300	--	13	--	107
JY	--	523	--	13	--	115
WS/WSS	295		39		121	
TT-30	425		13		112	
TT-31	--	277	--	13	--	106
CB-01	317		39		123	
CB-02	119		39		102	
CB-03	196		39		110	
CB-04	208		39		112	
CB-06	137		39		104	
CB-07	149		13		102	
16/TT-33	117		13		101	
09	30		13		30	
AM	150		13		102	
KF	97		13		97	
08	43		13		43	
07/DP	251		13		105	
LP	82	83	13		82	83
AS	573		13		117	
05	266		13		106	
03	124		13		101	
01	19		13		19	

Sediment/Soil

AJRW-SD	185	13	103
AJRW-SO	298	13	107
Sum =			210

Surface Water³

Reach	Average conc (ug/L)	SA ⁴ (cm ³)	PC (cm/hr)	ET (hrs/event)	EV (events/day)	EF ⁴ (days/yr)	ED (yrs)	CF1 (L/cm ³)	AT-N (days)	Intake ⁵ (ug/day)
01	13	2800	1E-03	0.5	1	78	2	0.001	730	4E-03
Upper 02	4.8	2800	1E-03	0.5	1	78	2	0.001	730	1E-03
Lower 02	0.43	2800	1E-03	0.5	1	78	2	0.001	730	1E-04
03	4.3	2800	1E-03	0.5	1	78	2	0.001	730	1E-03
04	5.7	2800	1E-03	0.5	1	78	2	0.001	730	2E-03
05	0.42	2800	1E-03	0.5	1	78	2	0.001	730	1E-04
Upper 06	3.2	2800	1E-03	0.5	1	78	2	0.001	730	1E-03
Lower 06	4.1	6600	1E-03	0.5	1	5	2	0.001	730	2E-04

Notes

- (1) Adjusted by fraction ingested term (50%)
- (2) Time-weighted over one year using MADEP background value (MADEP, 2002) of 100 mg/Kg. If average concentration is less than 100 mg/Kg, the average concentration is used.
Time-weighted conc = (Average Conc. * Exposure Freq. + Bkgd. Conc * (365 - Exposure Freq.)) / 365
- (3) Parameters for intake calculation are CT values defined in Table 3-4.1.CT
- (4) Maximum CT exposure frequency (EF) from stations within reach used
- (5) Intake = EPC * SA * PC * ET * EV * EF * ED * CF1 / AT. Surface water intakes (ug/day) are 2-3 orders of magnitude less than the water consumption intakes. Therefore, these intakes are considered negligible and have not been included in the model run.

Indoor Dust Lead Levels = MADEP Bkgd (100 mg/Kg) * 0.7 = 70 mg/Kg [Assumption]

TABLE C.10-4. IEUBK TEXT OUTPUT FOR STATION 22/TT-22 (MAXIMUM)

LEAD MODEL FOR WINDOWS Version 1.0 Build 252

=====
===

Model Version: 1.0 Build 252
User Name:
Date:
Site Name:
Operable Unit:
Run Mode: Research

=====
===

The time step used in this model run: 1 - Every 4 Hours (6 times a day).

***** Air *****

Indoor Air Pb Concentration: 30.000 percent of outdoor.
Other Air Parameters:

Age	Time Outdoors (hours)	Ventilation Rate (m ³ /day)	Lung Absorption (%)	Outdoor Air Pb Conc (ug Pb/m ³)
.5-1	1.000	2.000	32.000	0.100
1-2	2.000	3.000	32.000	0.100
2-3	3.000	5.000	32.000	0.100
3-4	4.000	5.000	32.000	0.100
4-5	4.000	5.000	32.000	0.100
5-6	4.000	7.000	32.000	0.100
6-7	4.000	7.000	32.000	0.100

***** Diet *****

Age	Diet Intake(ug/day)
.5-1	5.530
1-2	5.780
2-3	6.490
3-4	6.240
4-5	6.010
5-6	6.340
6-7	7.000

***** Drinking Water *****

Water Consumption:

Age	Water (L/day)
.5-1	0.200
1-2	0.500
2-3	0.520
3-4	0.530
4-5	0.550
5-6	0.580
6-7	0.590

Drinking Water Concentration: 4.000 ug Pb/L

***** Soil & Dust *****

Age	Soil (ug Pb/g)	House Dust (ug Pb/g)
.5-1	337.000	70.000
1-2	337.000	70.000
2-3	337.000	70.000
3-4	337.000	70.000
4-5	337.000	70.000
5-6	337.000	70.000
6-7	337.000	70.000

***** Alternate Intake *****

Age	Alternate (ug Pb/day)
.5-1	0.000
1-2	0.000
2-3	0.000
3-4	0.000
4-5	0.000
5-6	0.000
6-7	0.000

***** Maternal Contribution: Infant Model *****

Maternal Blood Concentration: 2.500 ug Pb/dL

CALCULATED BLOOD LEAD AND LEAD UPTAKES:

Year	Air (ug/dL)	Diet (ug/day)	Alternate (ug/day)	Water (ug/day)
.5-1	0.021	2.542	0.000	0.368
1-2	0.034	2.633	0.000	0.911
2-3	0.062	2.988	0.000	0.958
3-4	0.067	2.907	0.000	0.988
4-5	0.067	2.855	0.000	1.045
5-6	0.093	3.034	0.000	1.110
6-7	0.093	3.360	0.000	1.133

Year	Soil+Dust (ug/day)	Total (ug/day)	Blood (ug/dL)	
.5-1	4.457	7.387	4.0	
1-2	7.016	10.595	4.4	
2-3	7.091	11.098	4.1	
3-4	7.176	11.138	3.9	
4-5	5.420	9.387	3.3	
5-6	4.913	9.150	2.9	
6-7	4.655	9.242	2.6	

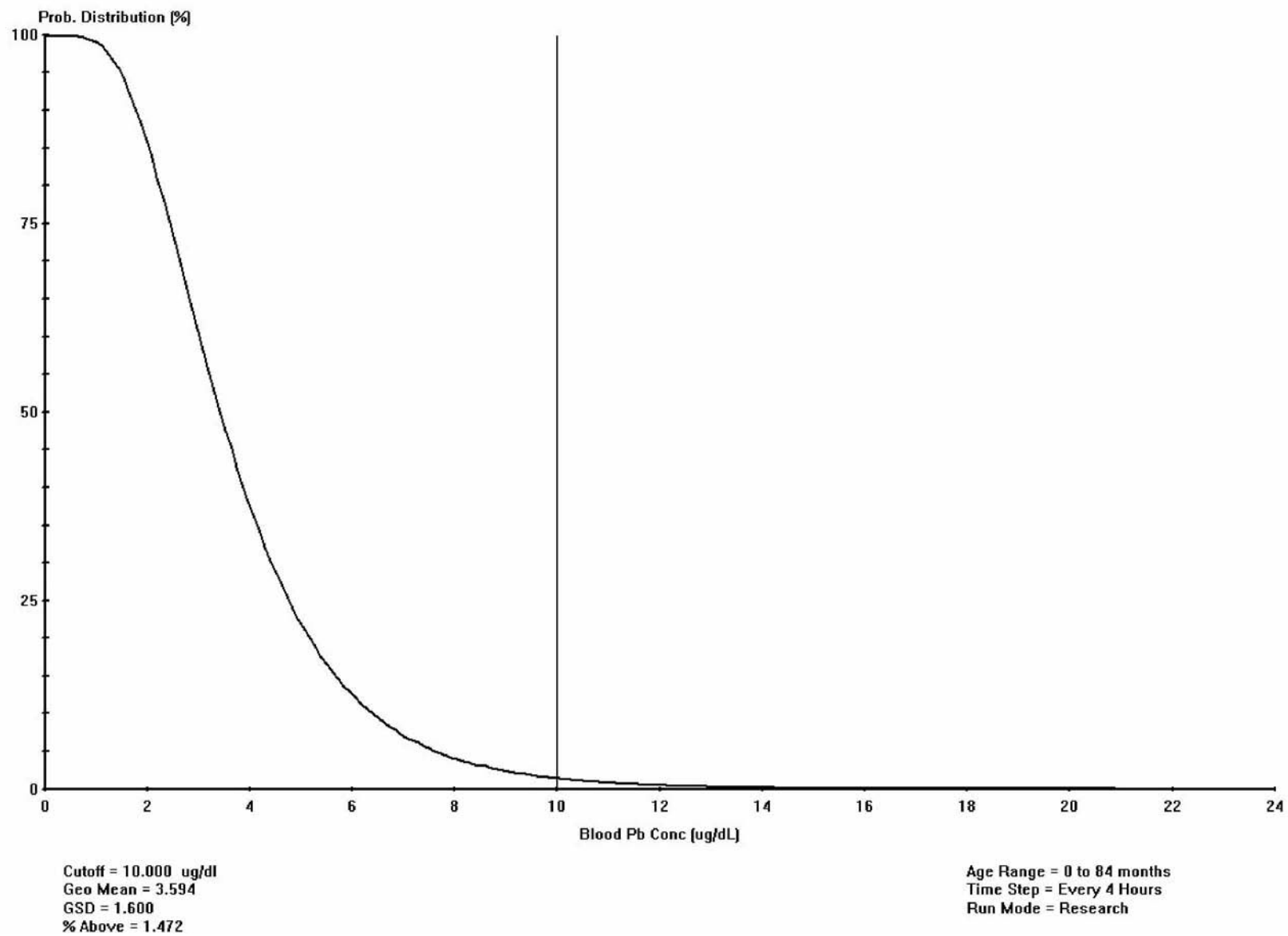


FIGURE C.10-1. IEUBK GRAPHICAL OUTPUT FOR STATION 22/TT-22 (MAXIMUM)

TABLE C.10-5. SEDIMENT/SOIL ADULT LEAD MODEL INPUTS

Sediment/Soil

Station	Average Concentration (mg/Kg)		CT Exposure Frequency (days/yr) ¹	
	Current	Future	Current	Future
NR		161		13
14		68		13
22/TT-22		6765		13
13/TT-27	--	700	--	13
WH		1493		13
NT-1	--	468	--	13
NT-2	--	420	--	13
NT-3	--	466	--	13
WG		429		13
WW	--	300	--	13
JY	--	523	--	13
WS/WSS		295		39
TT-30		425		13
TT-31	--	277	--	13
CB-01		317		39
CB-02		119		39
CB-03		196		39
CB-04		208		39
CB-06		137		39
CB-07		149		13
16/TT-33		117		13
09		30		13
AM		150		13
KF		97		13
08		43		13
07/DP		251		13
LP	82	83		13
AS		573		13
05		266		13
03		124		13
01		19		13
AJRW-SD		185		13
AJRW-SO		298		13
SC05		398		83
SC06		343		83
SC07		237		83
SC08		185		83
SC11		578		83
SC12		955		83
SC13		370		83

Notes

- (1) Adjusted by fraction ingested term (50%)

Calculations of Preliminary Remediation Goals (PRGs)

Exposure Variable	PbB Equation ¹		Description of Exposure Variable	Units
	1*	2**		
PbS	X	X	Soil lead concentration	ug/g or ppm
R _{fetal/maternal}	X	X	Fetal/maternal PbB ratio	--
BKSF	X	X	Biokinetic Slope Factor	ug/dL per ug/day
GSD _i	X	X	Geometric standard deviation PbB	--
PbB ₀	X	X	Baseline PbB	ug/dL
IR _S	X		Soil ingestion rate (including soil-derived indoor dust)	g/day
IR _{S+D}		X	Total ingestion rate of outdoor soil and indoor dust	g/day
W _S		X	Weighting factor; fraction of IR _{S+D} ingested as outdoor soil	--
K _{SD}		X	Mass fraction of soil in dust	--
AF _{S,D}	X	X	Absorption fraction (same for soil and dust)	--
EF _{S,D}	X	X	Exposure frequency (same for soil and dust)	days/yr
AT _{S,D}	X	X	Averaging time (same for soil and dust)	days/yr
PbB_{adult}	PbB of adult worker, geometric mean			ug/dL
PbB_{fetal, 0.95}	95th percentile PbB among fetuses of adult workers			ug/dL
PbB_i	Target PbB level of concern (e.g., 10 ug/dL)			ug/dL
P(PbB_{fetal} > PbB_i)	Probability that fetal PbB > PbB_i, assuming lognormal distribution			%

¹ Equation 1 does not apportion exposure between soil and dust ingestion (excludes W_S, K_{SD}).

When IR_S = IR_{S+D} and W_S = 1.0, the equations yield the same PbB_{fetal,0.95}.

*Equation 1, based on Eq. 1, 2 in USEPA (1996).

PbB_{adult} =	$(PbS * BKSF * IR_{S+D} * AF_{S,D} * EF_S / AT_{S,D}) + PbB_0$
PbB_{fetal, 0.95} =	$PbB_{adult} * (GSD_i^{1.645} * R)$

**Equation 2, alternate approach based on Eq. 1, 2, and A-19 in USEPA (1996).

PbB_{adult} =	$PbS * BKSF * [(IR_{S+D}) * AF_S * EF_S * W_S] + [K_{SD} * (IR_{S+D}) * (1 - W_S) * AF_D * EF_D] / 365 + PbB_0$
PbB_{fetal, 0.95} =	$PbB_{adult} * (GSD_i^{1.645} * R)$

Calculations of Preliminary Remediation Goals (PRGs)

Exposure Variable	Units	Values for Maximum Conc. 39-day Exposure Frequency			
		Using Equation 1		Using Equation 2	
		GSDi = 1.8		GSDi = 1.8	
PbS	ug/g or ppm	317		317	
R _{fetal/maternal}	--	0.9		0.9	
BKSF	ug/dL per ug/day	0.4		0.4	
GSD _i	--	1.8		1.8	
PbB ₀	ug/dL	2.0		2.0	
IR _S	g/day	0.050		--	
IR _{S+D}	g/day	--		0.050	
W _S	--	--		1.0	
K _{SD}	--	--		0.7	
AF _{S, D}	--	0.12		0.12	
EF _{S, D}	days/yr	39		39	
AT _{S, D}	days/yr	365		365	
PbB_{adult}	ug/dL	2.1		2.1	
PbB_{fetal, 0.95}	ug/dL	4.9		4.9	
PbB_t	ug/dL	10.0		10.0	
P(PbB_{fetal} > PbB_t)	%	0.2%		0.2%	

Calculations of Preliminary Remediation Goals (PRGs)

Exposure Variable	Units	Values for Maximum Conc. 13-day Exposure Frequency			
		Using Equation 1		Using Equation 2	
		GSDi = 1.8		GSDi = 1.8	
PbS	ug/g or ppm	6765		6765	
R _{fetal/maternal}	--	0.9		0.9	
BKSF	ug/dL per ug/day	0.4		0.4	
GSD _i	--	1.8		1.8	
PbB ₀	ug/dL	2.0		2.0	
IR _S	g/day	0.050		--	
IR _{S+D}	g/day	--		0.050	
W _S	--	--		1.0	
K _{SD}	--	--		0.7	
AF _{S, D}	--	0.12		0.12	
EF _{S, D}	days/yr	13		13	
AT _{S, D}	days/yr	365		365	
PbB_{adult}	ug/dL	2.6		2.6	
PbB_{fetal, 0.95}	ug/dL	6.1		6.1	
PbB_t	ug/dL	10.0		10.0	
P(PbB_{fetal} > PbB_t)	%	0.6%		0.6%	

Calculations of Preliminary Remediation Goals (PRGs)

Exposure Variable	Units	Values for Maximum Conc. 39-day Exposure Frequency			
		Using Equation 1		Using Equation 2	
		GSDi = 1.8		GSDi = 1.8	
PbS	ug/g or ppm	955		955	
R _{fetal/maternal}	--	0.9		0.9	
BKSF	ug/dL per ug/day	0.4		0.4	
GSD _i	--	1.8		1.8	
PbB ₀	ug/dL	2.0		2.0	
IR _S	g/day	0.050		--	
IR _{S+D}	g/day	--		0.050	
W _S	--	--		1.0	
K _{SD}	--	--		0.7	
AF _{S, D}	--	0.12		0.12	
EF _{S, D}	days/yr	83		83	
AT _{S, D}	days/yr	365		365	
PbB_{adult}	ug/dL	2.5		2.5	
PbB_{fetal, 0.95}	ug/dL	6.0		6.0	
PbB_t	ug/dL	10.0		10.0	
P(PbB_{fetal} > PbB_t)	%	0.6%		0.6%	