DEPARTMENT: Health

State of Minnesota

Office Memorandum

DATE: October 14, 2005

TO: MDH US Steel Site File

FROM: Carl Herbrandson, Ph.D., Toxicologist, MDH Site Assessment and Consultation

PHONE: 651 / 215-0925

SUBJECT: **Development of Sediment Screening Values**

In December 2002, the Minnesota Department of Health developed sediment screening values (SSVs) for the US Steel Site on the Saint Louis River in Duluth, MN (Attachment 1; MDH 2002). These values were used by US Steel to determine "Chemicals of Interest" in site-associated sediments (US Steel 2003; 2005).

MDH updated the SSVs in a report, "Human Health Screening Values for St. Louis River Sediments: US Steel Site", on August 5, 2005 (Attachment 2; MDH 2005). The updated SSVs reflect new health-based toxicity values and small changes that resulted from corrections to the methods used to calculate the original SSVs. The SSV report describes the assumptions and limitations that should be considered when the SSVs are used. An Appendix to the report (Attachment 3) contains the equations used to calculate the SSVs, as well as additional information supporting SSV derivation.

Minnesota Department of Health (2005). Human Health Screening Values For St. Louis River Sediments: US Steel Site. Site Assessment and Consultation Unit, Environmental Health Division, St. Paul, MN. August 8, 2005.

Minnesota Department of Health (2002). MDH health-based sediment screening values. Carl Herbrandson, Site Assessment and Consultation, St. Paul, MN. Email to US Steel, URS, Minnesota Pollution Control Agency, December 13, 2002.

US Steel (2005). Chemicals of Interest in Sediments and Surface Water - USS Former Duluth Works. Mark Barnes, Project Manager, Pittsburgh, PA. Submittal to Minnesota Pollution Control Agency, August 31, 2005.

US Steel (2003). Former Duluth Works Sediment Characterization and Tier I Risk Assessment Work Plan. Prepared by URS Corporation, Pittsburgh, PA. September 5, 2003.

Attachment 1

From: "Carl Herbrandson" <Carl.Herbrandson@state.mn.us>
To: <carl_crane@URSCorp.com>, <pei-fung_hurst@URSCorp.com>, <mazak@uss.com>
Date: 12/13/2002 11:58:24 AM
Subject: MDH health-based sediment screening values

Margaret, Carl and Pei-fung,

Attached is an ammended spreadsheet containing MDH Health-based Sediment screening value for the St. Louis River. I have included a few more pieces of information, including equations used (at the bottom of the Screening Values-ExposureRoutes worksheet) and route-to-route extrapolation for the inhalation route for the sparingly volatile organics (utility in inderstanding the chemical, not much of an effect on screening). In addition, I found a small error in the previous spreadsheet, so please dispose of it.

Thanks

Carl

Carl Herbrandson, Ph.D.
Toxicologist, Minnesota Department of Health
121 E Seventh Pl, Suite 220
St. Paul, MN 55101

651/215-0925 fax 651/215-0975
carl.herbrandson@health.state.mn.us

CC: "Ginny Yingling" <Ginny.Yingling@health.state.mn.us>, "Helen Goeden" <Helen.Goeden@state.mn.us>, <Andrew.Streitz@state.mn.us>, <Laura.Solem@state.mn.us>, <Susan.Johnson@state.mn.us>

The attached file (MDH Sediment Screening Values.xls) contains screening values that were developed for use in the St. Louis River. The 'Screening Values' worksheet contains values calculated for the following possible routes of exposure:

1. Direct ingestion of suspended sediment during swimming or wadding;
2. Direct dermal exposure to suspended sediment;
3. Indirect exposure by ingestion of fish that accumulate contaminants from sediment;
4. Indirect exposure by ingestion of water contaminated by sediment and suspended sediment;
5. Indirect exposure by dermal exposure to water contaminated by sediment and suspended sediment;
6. Indirect exposure by inhalation of air containing chemicals that partition from sediments to water and volatilize.

Default exposure parameters, default chemical-specific parameters and equations used to calculate the screening values are included in worksheets 'Exposure parameters' and 'Chemical specific data'. References are included in the worksheets.

MDH recommends that the 'Screening Values-ExposureRoutes' worksheet be used to screen individual chemicals and to determine the relative importance of different routes of exposure. However, specific areas of a site should be screened using a hazard index (HI) methodology that considers the potential additivity of health effects with specific endpoints (HI < 1)and a total cancer risk ($1x10^{-5}$) approach for carcinogens. The 'Sediment ScreeningCalcs' worksheet performs these calculations automatically when the appropriate data (sediment sample data) are input.

The screening values were produced using the best available information, primarily from EPA guidance documents. However, if new information becomes available, MDH may change some values. Changes are anticipated if and when regulatory or health agencies (e.g. EPA) update default factors, or significant new data become available.

Attachment 1

MDH Sediment Screening Values 12-02--.xls

Cancer Risk = 1.00E-05
Chronic HQ = 0.2

Chemical	Human Health-based Sediment Screening Values (mg/kg) Cancer-based	Non-cancer	Carcinogens: Direct Exposure From Sediment/Suspended Sediment — Direct ingestion f(Susp Sed Ing) % ttl	Dermal exposure f(Derm Exp Susp Sed) % ttl	Biota/Sed Accumulation — Fish ingestion f(Fish Ing, BSAF) % ttl	Indirect ** From Water - EqP from sediment — Direct ingestion f(Water Ing,EqP) % ttl	Dermal exposure f(Derm Exp Water,EqP) % ttl	Indirect ## From Air - EqP/Henry's — Inhalation Only f(InhR, Henry, EqP) % ttl	Non-Carcinogens: Direct Exposure From Sediment/Suspended Sediment — Direct ingestion f(Susp Sed Ing) % ttl	Dermal exposure f(Derm Exp Susp Sed) % ttl	Biota/Sed Accumulation — Fish ingestion f(Fish Ing, BSAF) % ttl	Indirect ** From Water - EqP from sediment — Direct ingestion f(Water Ing,EqP) % ttl	Dermal exposure f(Derm Exp Water,EqP) % ttl	Indirect ## From Air - EqP/Henry's — Inhalation Only f(InhR, Henry, EqP) % ttl	If water (direct Ingestion/dermal) is the driving route of exposure ** — Cancer Water Screening (mg/L)	Non-cancer Water Screening (mg/L)	If Inhalation is the driving route of exposure (e.g. VOCs) ## — Inhalation-Cancer Air Screening (ug/m³)	Inhalation-Cancer Water Screening (mg/L)	Inhalation-Non-cancer Air Screening (ug/m³)	Inhalation-Non-cancer Water Screening (mg/L)
Inorganics																					
Arsenic	**20**	**35**	16%	82%	Not Evaluated	Not Evaluated	Not Evaluated	Not Evaluated	42%	59%								1.2		0.859	
Cadmium		**97**							35%	65%								2.66		0.572	
Chromium III		**420000**									**Not Evaluated (no BSAF)**									150000	
Chromium VI		**1700**							101%									0.399		2.86	
Copper		**10000**							97%	**Not Evaluated (no ABS_D)**		**Not Evaluated (no EqP)**	**Not Evaluated (no EqP, DA and ABS_D)**	**Not Evaluated (no EqPs sed:water:air)**						3710	
Cyanide		**5600**							100%											2000	
Lead *		**100**																			
Mercury (inorganic in sediment/SW; methylmercury in fish)		**0.014**							0% (as Hg^0)		98%										
Nickel		**5600**							100%											1.43	
Zinc		**84000**							100%											30100	
VOCs																					
Benzene	**0.0035**	**0.0094**	0%	Not Evaluated	Not Evaluated	0%	0%	99%	0%	**Not Evaluated (ABS_D)**	**Not Evaluated (no BSAF/BCF)**	3%	2%	95%	0.602	0.329	614	0.00273	1720	0.00764
Ethyl benzene		**0.37**							0%			1%	3%	95%		2.79			28600	0.0886
Styrene		**4.3**							0%			1%	3%	95%		7.48			28600	0.25
Toluene		**0.21**							0%			1%	2%	95%		1.93			11400	0.0424
Xylenes (mixed)		**0.063**										0%	0%	99%		18			2860	0.0132
PAHs (Polynuclear Aromatic Hydrocarbons)																					
Acenaphthene		**7.9**							0%	0%	73%	1%	15%	11%		0.511			6010	0.735
Acenaphthylene (toxicity surrogate - acenaphthene)		**24**							0%	1%	58%	3%	25%	14%		0.685			6010	1.27
Anthracene		**170**							0%	1%	81%	1%	14%	3%		2.59			31500	11.8
Benzo(a)pyrene Equivalents	**0.077**		0%	7%	81%	0%	13%	0%							0.0000319		5.32	0.114		
Fluoranthene		**48**							0%	3%	58%	1%	37%	0%		0.134			4010	10.4
Fluorene		**18**							0%	1%	65%	3%	25%	9%		0.459			4010	1.22
Methylnaphthalene (toxicity surrogate - naphthalene)		**0.3**							0%	0%	8%	0%	1%	90%		0.504			85.9	0.00525
Naphthalene		**0.1**							0%	0%	3%	0%	1%	95%		0.504			85.9	0.00437
Perylene (toxicity surrogate - pyrene)		**33**							0%	2%	53%	0%	43%	0%		0.00995			3150	0.906
Phenanthrene (toxicity surrogate - anthracene)		**130**							0%	1%	62%	2%	25%	8%		1.84			31500	5.85
Pyrene		**41**							0%	3%	66%	1%	28%	0%		0.106			3150	7.7
Polychlorinated Biphenyls																					
PCBs (Polychlorinated Biphenyls)	**0.013**	**0.0032**	0%	0%	97%	0%	1%	0%	0%	0%	100%	0%	1%	0.3%	0.000235	0.0000312	8.41	0.000489	2	0.000117
DIOXINS/FURANS																					
~~2,3,7,8-TCDD (or 2,3,7,8-TCDD equivalents)~~	**7.7E-08**	**9.5E-07**	0%	0%	100%	0%	0%	0%	0%	0%	99%	0%	0%	0.0%	2.81E-10	1.93E-09	0.000145	1E-07	1.14E-03	7.89E-07
Other Organics																					
Carbazole	**2.6**		0%	0%	75%	1%	7%	15%							0.565		838	0.253			
Dibenzofuran (unsubstituted)		**1.3**							0%	1%	78%	2%	18%	1.1%		0.0467			401	0.754
Hexachlorobenzene	**0.15**	**1**	0%	2%	35%	0%	13%	48%	0%	2%	30%	1%	21%	42.1%	0.000695	0.00295	10.5	0.000194	80.1	0.00148
Octachlorostyrene		**0.013**							0%	1%	102%	0%	0%	0.0%		0.0000423			3.01	0.00207

Exposure to individual chemicals in sediment in the St. Louis River at above-listed levels for reasonable-maximal durations, should not result in adverse health effects or significant health risks. The potential for interactive or additive effects are not evaluated in the above table.

Human health-based screening values for many chemicals are not protective of aquatic plants and organisms.

* The lead screening value is not based on a direct evaluation of hazard, but it is equivalent to the Minnesota bare-soil standard of 100 ppm. (Minn Statute 144.9501)

** If percent contribution of water exposure pathway is significant and sediment concentration exceeds screening value, water analysis may be performed. Water extract from well-mixed suspended sediment/water mixture should not exceed Water Screening value.

If percent contribution of inhalation pathway is significant and sediment concentration exceeds screening value, water analysis and/or air analysis may be performed. Water extract from well-mixed suspended sediment/water mixture should not exceed Water Screening value. Air analysis (for screening) should be conducted on sample collected from an enclosure such as a flux chamber over turbid water/disturbed sediment, or from headspace of a sediment/water sample.

Sediment Screening Assessment

	Chemical	CAS No.	MDH 8/2002 Draft Screening Values (mg/kg)	Site Concen	Site HQ	Non-Cancer Endpoints (1) CV/BLD	CNS/PNS	EYE	GI/LIV	IMMUN	KIDN	REPRO (incl devel)	SKIN	THYROID	WHOLE BODY	CANCER (2) ELCR	Class
	Inorganics:																
	Arsenic	7440382	20		0.000	0.000	0.000						0.000			0.00E+00	A
	Cadmium	7440439	97		0.000						0.000					NA	B1
	Chromium III	16065831	420000		0.000												NA
	Chromium VI	18540299	1700		0.000											NA	A
	Copper	7440508	10000		0.000				0.000								D
	Cyanide, free	57125	5600		0.000		0.000							0.000	0.000		NA
	Lead	7439921	100		0.000							0.000				NA	B2
	Mercury	various	0.014		0.000		0.000			0.000		0.000					D
	Nickel	various	5600		0.000										0.000	NA	A
	Zinc	7440666	84000		0.000	0.000											D
	Volatile Organics																
	Benzene	71432	0.0035		0.000	0.000				0.000		0.000				0.00E+00	A
	Ethyl benzene	100414	0.37		0.000				0.000		0.000	0.000					
	Styrene	100425	4.3		0.000	0.000	0.000		0.000								
	Toluene	108883	0.21		0.000		0.000			0.000							
	Xylenes (mixed)	1330207	0.063		0.000		0.000										
	Polyaromatic Hydrocarbons																
	Acenaphthene	83329	7.9		0.000				0.000								NA
	Acenaphthylene (toxicity surrogate - acenaphthene)		24		0.000				0.000								
	Anthracene	120127	170		0.000												D
	Benzo[a]pyrene equivalents (see BaP equiv. Calculation worksheeet)	50328	0.077		0.000											0.00E+00	B2
	Fluoranthene	206440	48		0.000	0.000			0.000		0.000						D
	Fluorene	86737	18		0.000	0.000											D
	Methylnaphthalene (toxicity surrogate naphthalene)		0.3		0.000	0.000		0.000									
	Naphthalene	91203	0.1		0.000	0.000		0.000									
	Perylene (toxicity surrogate - pyrene)		33		0.000						0.000						
	Phenanthrene (toxicity surrogate - anthracene)	85018	130		0.000												
	Pyrene	129000	41		0.000						0.000					NA	D
	Polychlorinated Biphenyls																
	PCBs (Polychlorinated Biphenyls)	1336363	0.0032		0.000					0.000		0.000				0.00E+00	B2
	Dioxins and Furans																
	2,3,7,8-TCDD (see 2,3,7,8-TCDD equivalents Calculation worksheet)	1746016	7.70E-08		0.000					0.000		0.000				0.00E+00	B2
	Other Organics																
	Carbazole	86748	2.6		0.000											0.00E+00	B2
	Dibenzofuran	132649	1.3		0.000						0.000						NA
	Hexachlorobenzene	118741	0.15		0.000				0.000							0.00E+00	B2
	Octachlorostyrene		0.013		0.000	0.000			0.000		0.000			0.000			
	Cumulative Screening Risk =					0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.000	0.00E+00	

(1) CV/BLD - cardiovascular/blood system; CNS/PNS - central/peripheral nervous system; EYE; GI/LIV - gastrointestinal/liver system; IMMUN - immune system; KIDN - kidney; REPRO - reproductive system (incl. developmental effects); SKIN - skin irritation or other effects; THYROID; 'WHOLE BODY - increased mortality, decreased growth rate, etc.

(2) Class A - Known human carcinogen

Class B - Probable human carcinogen (B1 - limited evidence in humans; B2 - inadequate evidence in humans but adequate in animals)

Class C - Possible human carcinogen

Class D - Not Classifiable

NA - No EPA Classification Available.

Chemical Information

Chemical	CAS No.	Sediment Dermal Absorption Value	(Source)	Sediment/Oral RfD Absorption Adjustment Factor		Adjustment of Toxicity Value For Dose Absorbed in Toxicity Study		FA	Kp	B	τ	t*	Source (Chemical Parameters for Calculation of Dose Absorbed from Water)
Inorganics													
Arsenic	7440-38-2	0.03	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Default	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001		1.00E-03				Exhibit B-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Cadmium	7440-43-9	0.001	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	use toxicity value based on diet	0.025	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001		1.00E-03				Exhibit B-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
						0.05	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001						
Chromium III	16065-83-1			1	toxicity value based on diet study	0.013	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001		1.00E-03				Exhibit B-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Chromium VI	18540-29-9			0.5	toxicity value based on drinking water study	0.025	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001		2.00E-03				Exhibit B-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Copper	7440-50-8			1	Default	0.6	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001		1.00E-03				Exhibit B-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Cyanide	57-12-5			1	Default	1	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001		1.00E-03				Default for inorganics, Exhibit 3-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Lead	7439-92-1								1.00E-04				Exhibit 3-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Mercury (inorganic in sediment/SW; methylmercury	22967-92-6/7439-97-6			1	Default	0.07	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001		1.00E-03				Exhibit B-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Methyl Mercury						0.9							
Nickel	various			1	Default	0.04	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001		2.00E-04				Exhibit B-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Zinc	7440-66-6			1	Default	1	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001		6.00E-04				Exhibit B-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
VOCs													
Benzene	71-43-2			1	Default	0.5	inhalation study - approx. 50% absorption	1	1.50E-02	1.00E-01	2.90E-01	7.00E-01	Exhibit B-3. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Ethyl benzene	100-41-4			1	Default	0.9	ATSDR toxicological profile information	1	4.90E-02	2.00E-01	4.20E-01	1.01E+00	Exhibit B-3. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Toluene	108-88-3			1	Default	0.9	ATSDR toxicological profile information	1	3.10E-02	1.00E-01	3.50E-01	8.40E-01	Exhibit B-3. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Xylenes (mixed)	1330-20-7			1	Default	0.9	Default	1	5.30E-02	2.00E-01	4.20E-01	1.01E+00	Exhibit B-3. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Styrene	100-42-5			1	Default	0.9	Default	1	3.70E-02	1.00E-01	4.10E-01	9.80E-01	Exhibit B-3. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
PAHs (Polynuclear Aromatic Hydrocarbons)													
Acenaphthene	83-32-9	0.13	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	1.19E-01	5.68E-01	7.60E-01	1.80E+00	EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001. Calculated based on equations 3.8, A.1, A.4 & A.5; Exhibits A-5 and B-3
Acenaphthylene (toxicity surrogate - acenaphthene)		0.13	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	8.90E-02	5.68E-01	7.60E-01	1.80E+00	Surrogate - acenaphthene
Anthracene	120-12-7	0.13	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	1.19E-01	4.23E-01	7.45E-01	1.80E+00	EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001. Calculated based on equations 3.8, A.1, A.4 & A.5
Benzo(a)pyrene Equivalents	50-32-8	0.13	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	7.00E-01	4.30E+00	2.69E+00	1.17E+01	Exhibit B-3. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Fluoranthene	206-44-0	0.13	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	2.20E-01	1.20E+00	1.45E+00	5.68E+00	Exhibit B-3. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Fluorene	86-73-7	0.13	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	7.97E-02	4.00E-01	9.40E-01	2.25E+00	EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001. Calculated based on equations 3.8, A.1, A.4 & A.5
Methylnaphthalene (toxicity surrogate - naphthalene)		0.13	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	4.70E-02	2.00E-01	5.60E-01	1.34E+00	Based on Naphthalene
Naphthalene	91-20-3	0.13	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	4.70E-02	2.00E-01	5.60E-01	1.34E+00	Exhibit B-3. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Perylene (toxicity surrogate - pyrene)		0.13	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	1.63E+00	9.92E+00	2.70E+00	1.22E+01	Exhibit B-3. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Phenanthrene (toxicity surrogate - anthracene)	85-01-8	0.13	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	1.40E-01	7.00E-01	1.06E+00	4.11E+00	Exhibit B-3. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Pyrene	129-00-0	0.13	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	2.10E-01	1.15E+00	1.42E+00	5.45E+00	EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001. Calculated based on equations 3.8, A.1, A.4 & A.6
DIOXINS/FURANS													
~~2,3,7,8-TCDD (or 2,3,7,8-TCDD equivalents)~~	1746-01-6	0.03	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	assume same as dietary study	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	0.5	8.10E-01	5.60E+00	6.82E+00	3.01E+01	Exhibit B-3. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Other Organics													
Carbazole		0.1	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Default	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	2.49E-02	1.24E-01	9.00E-01	2.17E+00	EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001. Calculated based on equations 3.8, A.1, A.4 & A.5
Dibenzofuran (unsubstituted)	132-64-9	0.1	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Default	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	7.93E-02	3.95E-01	9.16E-01	2.20E+00	EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001. Calculated based on equations 3.8, A.1, A.4 & A.5
Hexachlorobenzene	118-74-1	0.1	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Default	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	0.9	1.30E-01	9.00E-01	4.22E+00	1.62E+01	Exhibit B-3. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001
Octachlorostyrene	29082-74-4	0.1	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Default	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	1.67E-01	1.25E+00	1.41E+01	5.15E+01	EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001. Calculated based on equations 3.8, A.1, A.4 & A.6
PCBs (Polychlorinated Biphenyls)	1336-36-3	0.14	Exhibit 3-4. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	1	Default	1	No adjustment. Exhibit 4-1. EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001	0.5	5.42E-01	3.80E+00	4.40E+00	1.89E+01	EPA RAGS Vol I Part E, Supplemental Guidance for Dermal Risk Assessment Review Draft. Sept 2001. Calculated based on equations 3.8, A.1, A.4 & A.6

Chemical	MW	Log Kow	Log Kow Source	BSAF Used	BSAF Range	Source	Volatile?	Diffusivity in air	Diffusivity in water	Koc	Henry's (atm-m3/mol)	Solubility (mg/l)	Source of information
Arsenic							No						
Cadmium							No						
Chromium III							No						
Chromium VI							No						
Copper							No						
Cyanide							No						
Lead													
Mercury (inorganic in sediment/SW; methylmercury				8	1.4 - 45.7	MDH calculation based on St. Louis River data - 8 (mg/kg FW fish)/(mg/kg DW sediment). Other info: (mg/kg DW fish tissue)/(mg/kg DW sediment) - - Hildegrand et al - rock bass and hog suckers 1.4. Sorenson et al - northern pike 10.1. Wren & MacCrimmon - northern pike 24 - 27.1. Lindqvist - yellow perch 45.7.	Yes	3.07E-02	6.30E-06	52 (Kd)	1.14E-02		EPA SSL Technical Background Document (1996)
Methyl Mercury													
Nickel							No						
Zinc							No						
VOCs				Note: units for organics is [mg/kg fish lipid]/mg/kg sediment organic carbon]									
Benzene	78.1						Yes	8.80E-02	9.80E-06	6.50E+01	5.50E-03	1.80E+03	EPA SSL Technical Background Document (1996)
Ethyl benzene	106						Yes	7.90E-02	7.80E-06	2.20E+02	7.90E-03	1.50E+02	EPA Region IX, PRG tables (9/95)
Toluene	92.1						Yes	7.80E-02	8.60E-06	2.60E+02	6.60E-03	8.80E+02	EPA Region IX, PRG tables (8/96)
Xylenes (mixed)	106						Yes	7.20E-02	8.40E-06	2.40E+02	5.30E-03	2.00E+02	EPA Region IX, PRG tables (9/95)
Styrene	104						Yes	7.10E-02	8.00E-06	9.10E+02	2.80E-03	3.10E+02	EPA Region IX, PRG tables (4/98)
Acenaphthene	154	4.15		2.3	0.083 - 2.3	EPA 1997 Screening method 0.29; 0.083 from Washington Dept Health 1995; van der Oost et al. 1994 0.5 to 2.3	Yes	4.00E-02	8.00E-06	5.00E+03	2.00E-04	4.00E+00	EPA Region IX, PRG tables (1999)
Acenaphthylene (toxicity surrogate - acenaphthene)	152	3.94		0.6	0.05 - 0.6	EPA 1997 screening method; 0.29; 0.05 from Washington Dept Health 1995; van der Oost et al, 1994 0.1 to 0.6				6.92E+03	1.16E-04		
Anthracene	178	4.35		0.6	0.05 - 0.6	EPA 1997 screening method; 0.29; 0.05 from Washington Dept Health 1995; van der Oost et al, 1994 0.1 to 0.6	Yes	3.20E-02	7.70E-06	2.40E+04	6.50E-05	4.30E-02	EPA Region IX, PRG tables (1999)
Benzo(a)pyrene Equivalents	252	6.39		0.1	0.02 - 0.29	EPA 1997 Screening method 0.29; 0.105 from Washington Dept Health 1995; van der Oost 1994 0.02 to 0.1	No			9.55E+05	1.15E-06		
Fluoranthene	202	4.93		0.2	0.05 - 0.29	EPA Screening method 0.29; 0.105 from Washington Dept Health 1995; van der Oost 1994 0.05 to 0.2	No			4.90E+04	9.46E-06		
Fluorene	170	4.02		0.6	0.083 - 0.6	EPA Screening method 0.29; 0.083 from Washington Dept Health 1995; van der Oost et al, 1994 0.1 to 0.6	Yes	6.10E-02	7.90E-06	8.00E+03	8.00E-05	2.00E+00	EPA Region IX, PRG tables (1999)
Methylnaphthalene (toxicity surrogate - naphthalene)	142	3.72		2.3		use naphthalene as surrogate				3.16E+03	4.00E-04		
Naphthalene	128	3.17		2.3	0.29 - 2.3	EPA 1997 Screening method 0.29; van der Oost et al. 1994 0.5 to 2.3	Yes	5.90E-02	7.50E-06	1.20E+03	4.80E-04	3.10E+01	EPA Region IX, PRG tables (1999)
Perylene (toxicity surrogate - pyrene)	252	6.7		0.2		surrogate - pyrene				3.89E+05	8.50E-05		
Phenanthrene (toxicity surrogate - anthracene)	178	4.35		0.6	0.1 - 0.6	EPA 1997 Screening method 0.29; 0.105 from Washington Dept Health 1995; van der Oost 1994 0.1 to 0.6		3.33E-02	7.47E-06	1.41E+04	1.32E-04	9.94E-01	Texas Draft Risk Reduction Program, Appendix VII, 1996
Pyrene	202	4.93		0.2	0.05 - 0.29	EPA 1997 Screening method 0.29; 0.105 from Washington Dept Health 1995; van der Oost et al, 1994 0.05 to 0.2	Yes	2.70E-02	7.20E-06	6.80E+04	1.00E-05	1.40E-01	EPA Region IX, PRG tables (1999)
~~2,3,7,8-TCDD (or 2,3,7,8-TCDD equivalents)~~	322	7.02		1	0.003 - 2.27	0.059 for 2,3,7,8-TCDD and 0.025 for dioxin-toxic equivalents EPA '97; 1 for dioxins/furans from WA Dept Ecology '97; isomer specific BSAF range from 0.003 to 2.268 from WA Dept Hea	No	4.70E-02	8.00E-06	1.40E+07	3.55E-05	1.93E-05	Texas Draft Risk Reduction Program, Appendix VII, 1996
Carbazole	167	3.23		1		EPA 1997 Screening method - default value of 1				3.39E+03	8.12E-05		EPA SSL Technical Background Document (1996)
Dibenzofuran (unsubstituted)	168	4		1		EPA 1997 Screening method - default value of 1	Yes	6.00E-02	1.00E-05	7.80E+03	1.30E-05	3.10E+00	EPA Region IX, PRG tables (4/98)
Hexachlorobenzene	285	5.86		0.1	0.057 - 0.105	0.09 EPA '97; 0.057 WA '97; 0.105 WA '95	No	5.42E-02	5.91E-06	8.00E+04	1.32E-03	6.20E+00	EPA SSL Technical Background Document 1996
Octachlorostyrene	380	6.29		0.98		Great Lakes Water Quality Initiative Technical Support Document for the Procedure to Determine Bioaccumulation Factors (EPA 1995)				1.40E+07	3.55E-05		used value for TCDD
PCBs (Polychlorinated Biphenyls)	290	6.3		2.6	1.4 - 4.1	1.85 EPA '97; 2.6 WA '97; 1.392-4.134 WA'95	No	1.04E-01	1.00E-05	5.25E+05	4.20E-04	5.52E-03	Texas Draft Risk Reduction Program, Appendix VII, 1996

Chemical	RfD (mg/kg/day)	Basis of RfD	RfC (mg/m3)	Basis of RfC	Oral Slope Factor (mg/kg/d)-1	Basis of Oral Cancer Slope Factor	Inhalation Unit Risk (ug/m3)-1	Basis of Inhalation Unit Risk Factor	Inhalation Unit Risk (ug/m3)-1	Basis
Arsenic	3.00E-04	Drinking water study in humans. Target organ - skin, cardiovascular and nervous systems. Uncertainty factor applied - 3. Confidence in RfD - medium (IRIS 12/95).	3.00E-05	CalEPA OEHHA REL 2001	1.50E+00	Epidemiological study of humans exposed via drinking water. Cancer target organs - skin, lung, liver, kidney and bladder. Note: CPF based on skin cancer incidence only. Class A (IRIS 12/95)	4.00E-03	Human occupational studies. Cancer target organ - lung. Class A (IRIS 12/95).	4.00E-03	IRIS
Cadmium	1.00E-03	Toxicokinetic modeling, based on diet, of critical target organ concentrations in humans. Target organ - kidney. Uncertainty factor - 10. Confidence in RfD - high (IRS 12/95).	2.00E-05	CalEPA OEHHA REL 2001	NA	NA	1.80E-03	Occupational studies in humans. Cancer target organ - lung. Class B1 (IRIS 12/95).	1.80E-03	MDH HRV + IRIS
	5.00E-04	Toxicokinetic modeling, based on water, of critical target organ concentrations in humans. Target organ - kidney. Uncertainty factor - 10. Confidence in RfD - high (IRS 12/95).								
Chromium III	1.50E+00	No effect observed. IRIS 1998.	NA		NA	NA	NA	NA		
Chromium VI	3.00E-03	No effect observed. IRIS 1998.	1.00E-04	IRIS 1998	NA	NA	1.20E-02	Occupational studies in humans. Cancer target organ - lung. Class A carcinogen (IRIS 12/95).	1.20E-02	MDH HRV + IRIS
Copper	3.70E-02	Single oral dose in humans. Target organ - gastrointestinal. Uncertainty factor - NA. Confidence in RfD - NA (HEAST 1995).	NA		NA	NA	NA	NA		
Cyanide	2.00E-02	Dietary study in rats. Target organ - nervous system, thyroid, and whole body. Uncertainty factors applied - 100. Confidence in RfD - medium (IRIS 12/95)	NA		NA	NA	NA	NA		
Lead	NA		NA		NA					
Mercury (inorganic in sediment/SW; methylmercury	3.00E-04		3.00E-04	IRIS 1995	NA	NA	NA	NA		
Methyl Mercury	1.00E-04	Methyl mercury exposure to humans. Target organ - developing nervous system. Uncertainty factor - 10. Confidence in RfD - medium (IRIS 12/95).		NA						
Nickel	2.00E-02	Dietary study in rats. Target organ - suppression of body and organ weights. Uncertainty factor - 300. Confidence in RfD - medium (IRIS 12/95).	5.00E-05	Nickel and Nickel Compounds, except nickel oxide (e.g., nickel chloride, nickel sulfate, nickel carbonate, nickel subsulfide) CalEPA OEHHA REL 2000	NA	NA	4.80E-04	Occupational exposure of humans to nickel subsulfide. Cancer target organ - respiratory system. Class A. (IRIS 12/95).		
Zinc	3.00E-01	Dietary supplement study in humans. Target organ - blood system. Uncertainty factor - 3. Confidence in RfD - medium (IRIS 12/95).	NA		NA	NA	NA	NA		
Benzene	3.00E-03	Provisional value. Target organ - blood and immune system. Uncertainty factor - 3000. Confidence - medium. (STSC memo 1996)	**6.00E-02**	**CalEPA REL (2000). Target organs: blood, developmental, nervous system. Uncertainty factor - 10.**	5.50E-02	Range 1.5E-2 to 5.5E-2. IRIS 2000	8.30E-06	studies in humans. Cancer target organ - blood system. Class A (IRIS	7.80E-06	MDH HRV + IRIS
Ethyl benzene	1.00E-01	Oral study in rats. Target organ - liver and kidney. Uncertainty factor applied - 1000. Confidence in RfD - low (IRIS 12/95).	1.00E+00	IRIS 1995 Target organs: developmental. Uncertainty factors - 300	NA	NA	NA	NA	NA	
Toluene	**4.00E-02**	**Oral study in rats. Target organ - immune system. Uncertainty factor - 1000. External Review Draft IRIS Summary (8/19/02)**	4.00E-01	MDH HRV 2002	NA	NA	NA	NA	NA	
Xylenes (mixed)	**7.00E-01**	**Oral study in rats. Target organ - nervous system. Uncertainty factor - 1000. External Review Draft IRIS Summary 3/2002**	**1.00E-01**	**Inhalation study in rats. Target organ - nervous system. Uncertainty factor - 300. External Review Draft IRIS Summary 3/2002**	NA	NA	NA	NA	NA	
Styrene	2.00E-01	Oral study in dogs. Target organ - liver and blood systems. Uncertainty factor - 1000. Confidence in RfD - medium (IRIS 12/95)	**1.00E+00**	MDH HRV 2002	NA	Under review	NA	Under review	NA	
Acenaphthene	6.00E-02	Oral study in mice. Target organ - liver. Uncertainty factor - 3000. Confidence in RfD - low (IRIS 12/95).	2.10E-01	EPA SSL Guidance, Appendix B 1996	NA	NA	NA	NA	NA	
Acenaphthylene (toxicity surrogate - acenaphthene)	6.00E-02	Oral study in mice. Target organ - liver. Uncertainty factor - 3000. Confidence in RfD - low (IRIS 12/95).	2.10E-01		NA	NA	NA	NA	NA	
Anthracene	3.00E-01	Oral study in mice. Target organ - none observed at the doses evaluated. Uncertainty factor - 3000. Confidence in RfD - low (IRIS 12/95).	1.10E+00	EPA SSL Guidance, Appendix B 1996	NA	NA	NA	NA	NA	
Benzo(a)pyrene Equivalents	NA	NA	NA		7.30E+00	Geometric mean of four slope factors obtained by different modeling methods using 2 different studies in more than 1 sex and species. Class B2 (IRIS 12/95)	1.70E-03	Inhalation study in hamsters. (HEAST 1995)	9.00E-04	NCEA + CalEPA REL
Fluoranthene	4.00E-02	Oral study in mice. Target organ - kidney, blood, and liver. Uncertainty factor - 3000. Confidence in RfD - low (IRIS 12/95).	1.40E-01	EPA SSL Guidance, Appendix B 1996	NA	NA	NA	NA	NA	
Fluorene	4.00E-02	Oral study in mice. Target organ - blood system. Uncertainty factor - 3000. Confidence in RfD - low (IRIS 12/95).	1.40E-01	EPA SSL Guidance, Appendix B 1996	NA	NA	NA	NA	NA	
Methylnaphthalene (toxicity surrogate - naphthalene)	2.00E-02	Oral study in rats. Target organ - blood system and decreased body weight. Uncertainty factor - 3000. Confidence in RfD - low. (IRIS 9/98)	3.00E-03		NA	NA			NA	
Naphthalene	2.00E-02	Oral study in rats. Target organ - blood system and decreased body weight. Uncertainty factor - 3000. Confidence in RfD - low. (IRIS 9/98)	3.00E-03	IRIS 1998	NA	NA	NA	NA	NA	
Perylene (toxicity surrogate - pyrene)	3.00E-02	Oral study in mice. Target organ - kidney. Uncertainty factor - 3000. Confidence in RfD - low (IRIS 12/95).	1.10E-01		NA	NA	NA	NA	NA	
Phenanthrene (toxicity surrogate - anthracene)	3.00E-01	Oral study in mice. Target organ - none observed at the doses evaluated. Uncertainty factor - 3000. Confidence in RfD - low (IRIS 12/95).	1.10E+00		NA	NA	NA		NA	
Pyrene	3.00E-02	Oral study in mice. Target organ - kidney. Uncertainty factor - 3000. Confidence in RfD - low (IRIS 12/95).	1.10E-01	EPA SSL Guidance, Appendix B 1996	NA	NA	NA	NA	NA	
~~2,3,7,8-TCDD (or 2,3,7,8-TCDD equivalents)~~	2.30E-09	Based on PTMI (Provisional Tolerable Monthly Intake) value of 70 pg/kg BW. WHO Joint FAO/WHO Expert Committee on Food Additives. June 2001.	4.00E-08	CalEPA OEHHA REL 2000	9.00E+05	SAB Dioxin Reassessment Draft 2000	3.30E+01	Oral study. HEAST 1995).	3.30E+01	HEAST
Carbazole	NA	NA	NA		2.00E-02	HEAST			NA	
Dibenzofuran (unsubstituted)	4.00E-03	Provisional toxicity value. Target organ - kidney. Uncertainty factor - 3000. Confidence in RfD - low. (ECAO memo, 8/94).	NA		NA	NA	NA	NA	NA	
Hexachlorobenzene	8.00E-04	Chronic feeding study in rats. Target organ - liver. Uncertainty factor - 100. Confidence in RfD - medium. (IRIS 12/96)	NA		1.60E+00	Dietary study in rats. Cancer target organ - liver. Class B2 (IRIS 12/96)	4.60E-04	Based on oral studies. Class B2 (IRIS 12/96)	NA	
Octachlorostyrene	3.00E-05	ADI from New York State Health Department. Based on 90 day and 12 month study in rats (Chu et al, 1984, Chu et al, 1986). Dietary study in rats. Target organs - blood system, thyroid, liver, and kidney. Uncertainty factor - 1000.	NA		NA	NA	NA	NA	NA	
PCBs (Polychlorinated Biphenyls)	2.00E-05	1016 RfD 7E-5; 1254 RfD 2E-5 (IRIS 1996)	NA		2.00E+00	Dietary studies in rats. Cancer target organ - liver. Class B2. (IRIS 10/96)	5.70E-04	Based on oral studies. Cancer target organ - liver. (CalEPA 1997)	5.70E-04	IRIS

EPA, September 1997- The Incidence and Severity of Sediment Contamination in Surface Waters fo the United States. Vol. 1 (EPA 823-R-97-006)

WA Department of Ecology December 1997 - Developing Health-Based Sediment Quality Criteria for Cleanup Sites: A Case Study Report.

WA Department of Health June 1995 - Tier I Report. Development of Sediment Quality Criteria for the Protection of Human Health. Based on chemical class and Kow

van der Oost, et al., 1994. Biochemical markers in feral Roach (Rutilus Rutilus) in relation to the bioaccumulation of organic trace pollutants. Chemo 29(4)801-817.

Draft Wader/Swimmer Screening Exposure Input Parameters

Variable	Definition	Value Utilized		Rationale/Reference
Sediment Ingestion Rate (mg/day)		57	(1-6)	Calculated based on ingestion of 0.1 l water/event (EPA 1989a) - - 1 event/day
		57	(>6-16)	for wading and **2** events/day during swimming for 1 - 16 yr; 1 event/day for wading and
		32	(>16 - 18)	swimming for >16 - 18 yr; and 0.05 l/event x 1 event/day for >18 yr
		16	(>18 - 33)	and average suspended sediment concentration of 320 mg/liter
		37	(age-adjusted)	(use site specific suspended value if available)
Surface Water Ingestion Rate (L/day)		0.18	(1-6)	Calculated based on ingestion of 0.1 l water/event (EPA 1989a) - - 1 event/day
		0.18	(>6-16)	for wading and **2** events/day during swimming for 1 - 16 yr; 1 event/day for wading and
		0.10	(>16 - 18)	swimming for >16 - 18 yr; and 0.05 l/event x 1 event/day for >18 yr
		0.05	(>18 - 33)	(use site specific suspended value if available)
		0.12	(age-adjusted)	
Skin surface area in contact with sediment (cm2)		5168	(1-6)	Approximately 20% of mean total body surface area during wading +
		9114	(>6-16)	approx. 90% of mean total body surface area during swimming (EPA 1997)
		11670	(>16 - 18)	weighted ave = [(0.2 x total body SA x # days wading) + (0.9 x total body SA x # days swimming)]/EF
		11312	(>18 - 33)	
		9551	(age-adjusted)	
Skin adherence factor for sediment (mg/cm2)		0.52	(1-6)	2 mg/cm2 during wading and 0.02 mg/cm2 (I.e., thin layer of fine sediment) during
		0.52	(>6-16)	swimming (EPA 1997)
		0.62	(>16 - 18)	weighted ave = [(2 mg/cm2 x # days wading) + (0.02 mg/cm2 x # days swimming)]/EF
		0.80	(>18 - 33)	
		0.65	(age-adjusted)	
Sediment dermal absorption factor		Chemical Specific		
SW dermal dose absorbed per unit area per event		chemical specific - calculated (see Table for DA)		
Skin surface area in contact with surface water (cm2)		5168	(1-6)	Approximately 20% of mean total body surface area during wading +
		9114	(>6-16)	approx. 90% of mean total body surface area during swimming (EPA 1997)
		11670	(>16 - 18)	
		11312	(>18 - 33)	
		9551	(age-adjusted)	
Number of SW contact events per day		1.8	(1-6)	1 event/day for wading and **2** event per day for swimming for 1 - 16 yr; 1 event/day
		1.8	(>6-16)	for wading and 1 event/day for swimming for > 16 yr receptor.
		1.0	(>16 - 18)	weighted ave = [# event/day x # days wading) + (# events/day x # days swimming)]/EF
		1.0	(>18 - 33)	
		1.4	(age-adjusted)	
Fish Ingestion Rate (kg fish/day)		0.01	(1-6)	
(used directly for inorganics)		0.015	(>6-16)	
		0.020	(>16 - 18)	
		0.03	(>18 - 33)	30 g/day used for adult recreational anglers (corresponds to 50th %
		0.0212	(age-adjusted)	(Exposure Factors, EPA 1997) and median (Mercury Report, EPA 1997)
Lipid Content	(kg lipid/kg fish)	0.015		Median lipid content for cool and warm water fish (MPCA WQ - 7050 Rule)
Fish Lipid Ingestion Rate (kg lipid/day)		0.00015	(1-6)	
for organics		0.00023	(>6-16)	Calculated - Fish ingestion rate (IRf) x lipid content
		0.00030	(>16 - 18)	
		0.00045	(>18 - 33)	
		0.00032	(age-adjusted)	
Fish Tissue Concentration				
organics:	(mg/kg lipid)	BSAF * normalized Cs		(for organics)
Mercury:	(mg/kg fish tissue)	BSAF * Cs		(for mercury)
Biota-sediment accumulation factor		Chemical specific		
Organics: (kg fish lipid/kg organic carbon)				
Inorganics: (kg fish tissue/kg sediment)				
Fraction of organic carbon in sediment		0.02		Site-specific
Fraction of fish ingested from impacted area		1		Note - use upper-bound value with central tendency for ingestion rate
Sediment and Surface Water				
Exposure Frequency (days/year)		69	(1-6)	Approx. 2 d/wk wading in May & Sept + 4 d/wk swimming June - August
		69	(>6-16)	Approx. 2 d/wk wading in May & Sept + 4 d/wk swimming June - August
		56	(>16 - 18)	Approx. 2 d/wk wading in May & Sept + 3 d/wk swimming June - August
		43	(>18 - 33)	Approx. 2 d/wk wading for May & Sept + 2 d/wk swimming June - Aug
		56	(age-adjusted)	
Fish Ingestion exposure frequency (day/yr)		350		Ingestion rate utilized is **daily average** (EPA 1996)
ED	Exposure duration (years)	6	(1-6)	Assume receptor continues to reside in local community
		10	(>6-16)	Apply 90% residence time at same location (based on 1990 census information
		2	(>16 - 18)	for Mpls/St. Paul metro area.
		15	(>18 - 33)	Use site specific information if available.
		33	(age-adjusted)	
BW	Body weight (kg)	15	(1-6)	
		39	(>6-16)	Average body weight for age group (EPA 1997)
		60	(>16 - 18)	
		70	(>18 - 33)	
		50	(age-adjusted)	
AT	Averaging Time (days)	2190	(1-6)	
		3650	(>6-16)	Noncancer Evaluation AT = exposure duration
		730	(>16 - 18)	
		5475	(>18 - 33)	
		12045	(age-adjusted)	
		25550		Cancer Evaluation AT = 70 year lifetime

Minnesota Department of Health

Human Health Screening Values for ST. LOUIS RIVER SEDIMENTS: US STEEL SITE

(SSV Report)

DULUTH, ST. LOUIS COUNTY, MINNESOTA

EPA FACILITY ID: MND039045430

August 8, 2005

Minnesota Department of Health
Environmental Health Division
625 North Robert Street
St. Paul, Minnesota 55155

FOREWORD

This document summarizes human health-based sediment screening values developed for the St. Louis River Estuary, St. Louis County, Minnesota. It is based on a formal evaluation prepared by the Minnesota Department of Health (MDH). A number of steps are necessary for this evaluation:

- **Evaluating exposure:** MDH scientists begin by reviewing available information about environmental conditions in the river. The first task is to find out how much contamination is present, and how people might be exposed to it. Usually, MDH does not collect its own environmental sampling data. We rely on information provided by the Minnesota Pollution Control Agency (MPCA), U.S. Environmental Protection Agency (EPA), and other government agencies, private businesses, and the general public.

- **Evaluating health effects:** If there is evidence that people are being exposed—or could be exposed—to hazardous substances, MDH scientists will take steps to determine whether that exposure could be harmful to human health. Their report focuses on public health; that is the health impact on the community as a whole—and is based on existing scientific information.

- **Developing recommendations:** In the evaluation report, MDH outlines its conclusions regarding any potential health threat posed by contamination, and offers recommendations for reducing or eliminating human exposure to contaminants. The role of MDH in dealing with individual sites is primarily advisory. For that reason, the evaluation report will typically recommend actions to be taken by other agencies—including EPA and MPCA. However, if an immediate health threat exists, MDH will issue a public health advisory warning people of the danger, and will work to resolve the problem.

- **Soliciting community input:** The evaluation process is interactive. MDH starts by soliciting and evaluating information from various government agencies, the individuals or organizations responsible for cleaning up the site, and community living near the site. Any conclusions about the site are shared with the individuals, groups, and organizations that provided the information. Once an evaluation report has been prepared, MDH seeks feedback from the public.

If you have questions or comments about this report, we encourage you to contact us.

Please write to: Community Relations Specialist
Site Assessment and Consultation Unit
Minnesota Department of Health
121 East Seventh Place/Suite 220
Box 64975
St. Paul, MN 55164-0975

OR call us at: (612) 215-0778 ***or*** 1-800-657-3908
(toll free call—press "4" on your touch tone phone)

Table Of Contents

List of Attachments

List of Appendices

Executive Summary

The St. Louis River empties into Lake Superior through the ports of Duluth Minnesota and Superior Wisconsin. Historically, the twin ports have been industrial centers with considerable coke, iron and steel making. This report (SSV Report) evaluates reasonable, maximal exposures to chemicals in St. Louis River sediments, and calculates Sediment Screening Values (SSVs) that are protective of human health.

At the request of the Minnesota Pollution Control Agency (MPCA), The Minnesota Department of Health (MDH) developed criteria in December 2002 for screening sediments at the US Steel site in the St. Louis River Estuary. This report contains some modifications of the 2002 criteria and is intended to clarify the derivation of SSVs for US Steel and other interested parties.

Sediments in this document are assumed to lie under water, and an analysis of exposures to upland, tidal or beach sediments is not included. The ingestion of water-covered sediments is calculated from estimated water ingestion and the suspended sediment concentration in water. The amount of sediment ingested with water is much lower than would be expected if beach or soil-like sediments were considered. Therefore it is important that potential exposures in these areas be evaluated separately.

Chemical contaminants in sediments partition into porewater and surface water. In addition, fish and other aquatic organisms accumulate some chemicals from food and sediment that they ingest, or from direct partitioning of the chemicals from water to biological tissues. Furthermore, under some conditions, a few chemical contaminants (notably volatile organic compounds) will partition from water into air. This contributes additional human exposure to the contaminants through inhalation of the volatile chemical.

Six different routes of exposure are quantitatively evaluated in this document: sediment ingestion, dermal sediment exposure, surface water ingestion, dermal surface water exposure, fish consumption, and inhalation. Contributions to a total exposure by different potential routes of exposure are compared. SSVs were only developed for chronic and lifetime exposure durations. Short-term exposures to some chemicals, such as polycyclic aromatic hydrocarbons (PAHs), may result in irritation at the point of contact or other adverse effects. However, toxicity data are not available to determine a threshold exposure for effects of these short exposures. Given the general lack of toxicity criteria for short exposures to chemicals, acute exposures are not addressed in this document.

The SSV Report provides an overview of the default exposure values and health criteria used to develop the SSVs. It includes a discussion of issues considered for selection of default values. Furthermore, the document contains a discussion of appropriate use of the SSVs. The Appendices comprise a glossary of variables used in all equations, as well as a description of equations used to calculate the SSVs . In addition the Appendices contain further technical arguments for selection of some default values.

While the SSVs were developed for the US Steel Site, they do not rely on site-specific data. Therefore, they can be applied to other sites as well. The SSVs are screening values and should

only be used in the initial (screening) evaluations of contaminated sediments. The SSV Report and attached Appendices include all equations needed to calculate sediment screening values. Furthermore, they show how to calculate the percent contribution for each route of exposure. It is expected that the default values and models used in this report may change as additional data are acquired and models become more refined.

Results and Discussion

As expected, quantitative evaluation showed that dominant exposure to chemicals with different chemical and physical characteristics will occur by different routes of exposure. For example: the dominant exposure to volatile organic compounds in sediments is likely by inhalation, while exposure to bioaccumulative compounds in sediments comes mostly from fish ingestion.

Metals

Metals occur in sediments in many different chemical and mineral forms. Some metal compounds are very stable, while others are labile. Therefore, partitioning between the solid (sediment) phase and liquid (dissolved) phase is not easily predicted from an analysis of metal concentration in sediments. Consequently, ingestion and dermal risk from metals in surface water should be calculated from measured surface water concentrations and not from partitioning calculations. However, it should be noted that most metals are not easily absorbed through the skin. Therefore, dermal absorption is not a significant pathway for most metals. As a result, only the ingestion route of exposure was evaluated for most metals.

Mercury

The dominant route of exposure to mercury is by the consumption of fish containing methyl mercury in their muscle tissue. Currently there is a mercury (methyl mercury) fish consumption advisory for the St. Louis River Estuary. St. Louis River sediment and fish tissue data are used to calculate a waterbody-specific biota sediment accumulation factor (BSAF). The BSAF was then used to calculate a mercury SSV of 0.02 mg/kg. This SSV is lower than the ambient levels of mercury in the estuary, which are elevated due to regional and local sources. But the SSV is the same as regional background mercury concentrations.

Volatile Organic Compounds

The fate of volatile organic compounds (VOCs) discharged into an aquatic system is either degradation to less toxic chemicals or volatilization from the water into air. VOCs are not persistent. However, if other more persistent organic chemicals are also present, VOCs may bind to these chemicals and remain in the sediments for longer periods. VOC releases from sediments will enter overlying water and then volatilize into air. As the VOCs are released into water, and then into air, they are diluted. These dilutions make it unlikely that VOCs in sediment will result in adverse health impacts. However, if sediment concentrations of individual VOCs exceed the SSVs, water concentrations should be measured. Because dermal exposures from surface water represent the second greatest pathway for VOC exposure, both the dermal and inhalation exposures and risks should then be recalculated from surface water concentration data.

Polycyclic Aromatic Hydrocarbons

Some lighter PAHs, such as naphthalene, are volatile and exposures are likely to be mainly from inhalation. For most PAHs, dermal exposure to the compound dissolved in surface water results

in the largest exposure. However, the largest human exposure to a group of carcinogenic PAHs may result from fish consumption. Unfortunately, available analytical data on these PAHs in fish tissue are at detection limits 1-2 orders of magnitude above levels of concern. PAHs are expected to accumulate in fatty tissues of fish (including the skin). While older and larger fish may have more PAHs than smaller fish, this age and size dependence is possibly less-pronounced than for other accumulating compounds, such as methyl mercury and PCBs. In the absence of site-specific information about PAHs in fish tissue, fish consumption advice for mercury and PCBs should be used.

Dioxins and PCBs

As anticipated, quantitative evaluation also demonstrated that the fish consumption pathway is far more important than any other route of exposure for dioxins and PCBs (polychlorinated biphenyls). Background dioxin concentrations exceed the calculated SSV by a large amount. However, there are dietary and health benefits to diets that include fish. Furthermore, other food sources also contain dioxins. Because the calculated SSV is small relative to background, an appropriate screening concentration for dioxin is background. The PCB SSV is similar to PCB background concentrations.

Conclusions and Recommendations

Chemical concentrations in sediments at or below the human health-based Sediment Screening Values (SSVs) developed in this report are considered safe for the general public. Alternatively however, sediment concentrations greater than the screening values should not be considered unsafe, because the values were developed from conservative measures of exposure, bioavailability and toxicity. If there are local exceedances of these health values, site-specific conditions need to be evaluated prior to concluding that sediments may impact health. In addition while this document evaluates a suite of persistent chemicals, it does not evaluate all chemicals of concern in sediments.

Recommendations are:

- Analytical detection limits for chemicals of concern in sediments and fish tissue should be similar to detection limits in Tables E-1a and E-1b.
- If chemical concentrations in sediments adjacent to the US Steel Site exceed the Sediment Screening Values (or background levels for TCDD-TEQs), further evaluation is needed to determine whether chemicals in the sediments may impact public health.
    - If chemical concentrations in sediments are below the values developed in this document, the screened chemicals in sediments will not adversely impact the health of the public.
    - Further evaluation may be needed to determine whether or not the health of special populations, such as subsistence fishers, are protected.

1 Contamination of the Lower St. Louis River by US Steel and Other Industrial Operations

The lower St. Louis River is bounded by the Fond du Lac Dam upstream, and Lake Superior at the River's outlet. The lower St. Louis River is often called a freshwater estuary, because seiches regularly occur, reversing the flow of the river more than 10 miles upstream (Stortz and Sydor 1980). The lower half of this portion of the River is the Duluth/Superior Harbor, a port for Great Lakes and ocean-going vessels.

In the last hundred years, there have been many anthropogenic sources of pollution to the St. Louis River. These include paper mills, steel mills, coking ovens, shipbuilding and repair, cargo-loading docks, petroleum refinery, treated and untreated municipal wastes, and storm sewer runoff. Wastes include nutrients for bacteria and phytoplankton, inert particulates, inorganic acids and bases, metals, other inorganic compounds, and organic compounds. Most of these chemicals have been diluted or chemically degraded over time such that they do not represent a significant human health hazard. However some chemicals, or related long-lived degradation products are persistent and remain in the aquatic environment for extremely long times. Persistent chemicals are typically metals or groups of similar long-lived organic chemicals (e.g. polycyclic aromatic hydrocarbons (PAHs), polychlorinated biphenyls (PCBs)). Sediments often act as repositories for these persistent chemicals, and high concentrations of contaminants can be found in some areas. These sediments act not only as a repository or a sink for the chemicals, but can also be a reservoir, or source of these chemicals in a dynamic environment, and a source of exposure for aquatic organisms, wildlife and even people.

The St. Louis River Community Action Committee (St. Louis River CAC) wrote a report in 2000 that reviews industrial development and impacts to the St. Louis River Estuary from the 1870's to the present (St. Louis River CAC 2000). Two 1997 joint reports from the Minnesota Pollution Control Agency (MPCA) and the U.S. Environmental Protection Agency (EPA) provide sediment sampling data on many of the contaminated areas in the Duluth/Superior Harbor and much of the lower St. Louis River (the St. Louis River Estuary) (EPA and MPCA 1997a; 1997b). In addition, EPA conducted sampling of areas not identified as hotspots as part of their Regional Environmental Monitoring and Assessment Program (REMAP; EPA 1995) (see Attachment 1 for sample locations) and the MPCA has also conducted sediment sampling in many areas of the lower St. Louis River. These data are available from the EPA and MPCA, respectively.

The Minnesota Department of Health (MDH) has reviewed data from 2 large historic industrial sites in the Lower St. Louis River. They are known as the St. Louis River Interlake/Duluth Tar Site (SLRIDT) and the St. Louis River US Steel Site (US Steel). The SLRIDT site encompasses about 130 acres of land, and an additional 85 acres of water in 3 inlets. The site is the former location of pig iron and coking plants, a water/gas (coal gasification) plant, as well as tar and chemical companies. Industrial operations on the SLRIDT site ceased in about 1961 (EPA 2003b). The US Steel site located in the Morgan Park area of Duluth, Minnesota began operation in 1915. The facilities on-site included coke ovens, a coke by-products plant, open-hearth and blast furnaces, a blooming mill, a billet mill, and a merchant mill. Also, a continuous rod mill, wire mill, nail mill, pot annealing equipment, staple and woven fence machines, nail

cleaning, bluing and coating facilities, rod and wire cleaning facilities, and galvanizing facilities operated onsite at different times. In addition, from about 1918 until 1929 benzene and toluene were produced on-site. Operation of the steel mill continued until 1975 when open hearth and blast furnaces were shut down. The coking plant ceased operations in 1979 (MPCA 1989). Attachment 1 shows the location of the US Steel Site on the St. Louis River. Attachment 2 is an aerial photo of the US Steel facility in 1951 (from Tweed Museum Exhibition, 1992). Attachments 3 - 5 show surface water and material flowing from the site into the St. Louis River in 1967 (Federal Water Pollution Control Administration 1967-8).

In 1983, both the SLRIDT and US Steel Sites were added to the National Priorities List (NPL) by EPA under a single Comprehensive Environmental Response, Compensation, and Liability Information System (CERCLIS; Superfund) number (MND039045430). In 1984, MPCA placed both sites on the Minnesota Permanent List of Priorities (PLP) as separate sites.

There are numerous other areas where historical industrial, urban and municipal discharges of liquid and solid waste impacted the lower St. Louis River. These include a large number of coking ovens, manufactured gas plants, as well as storm and sanitary sewer outputs. In addition, there may have been impacts from industries upstream of the lower river. Further contamination also likely occurred from deposition of particulates and chemicals from air emitted from local and/or regional industrial facilities. MDH has not reviewed these activities or additional point and area sources of pollution to the St. Louis River.

2 Development of Sediment Screening Values for US Steel

The sediment screening values in this report were initially developed in December 2002 at the request of MPCA and U.S. Steel, as sediment screening values for the US Steel Site in the St. Louis River. Differences between present values and values initially submitted to the MPCA and US Steel reflect new health-based toxicity values and small changes that resulted from corrections to the methods.

The MPCA lists human health-based Sediment Quality Targets (SQTs) for PCBs, Benzo[a]Pyrene, 2,3,7,8-Tetrachlorodibenzo-p-dioxin and a number of pesticides. These SQTs have been published in an EPA report (2000a; see Attachment 6 for Human Health SQTs - Table 15). However, the SQTs were originally calculated by the New York State Department of Environmental Conservation (New York State DEC 1999). They are defined as site-specific chemical benchmarks and chemical concentrations that provide "a level of protection." The differences between the SQTs and the Sediment Screening Values developed in this document are discussed in Section 7.2.

Six different routes-of-exposure were evaluated to develop screening values. Along with screening values, the relative contributions of various routes of exposure were evaluated for each chemical. In addition, this document describes the methods and default parameters used to develop the screening values. All variables used in this report are defined in Appendix A. Equations used in calculations are described in the attached Appendices.

Note that this report evaluates the effect of sediment contaminants on *human* health and does not evaluate the affect of these contaminants on aquatic plants and animals, or on wildlife. Often the effects of exposure on aquatic organisms are more severe, and criteria developed for their protection are more restrictive. Therefore, comparison of sediment concentrations with ecological metrics or criteria is necessary to evaluate the impact of sediment contamination on the environment.

3 Exposure to Contaminants

The lower half of the St. Louis River estuary has been an industrial area for over 100 years. But it is also a high-use recreational area. People wade and swim in areas of the estuary throughout the summer. People fish the entire length of the lower river in the summer and the winter, and there are reports of families (primarily immigrant) using this resource as their primary food source (i.e., as a subsistence fishery). In addition, the lower St. Louis River is used for recreational boating: canoeing, motor boating, sailing, jet-skiing and water-skiing are common in the lower river.

Numerous activities can lead to exposure to chemicals in contaminated sediments. For screening purposes it should be assumed that even small areas of contaminated sediments may be located in a spot that individuals frequent for recreational purposes. Wading or swimming in contaminated areas can expose an individual directly to contaminated sediments, to contaminated suspended sediments and to contaminated water. Chemicals in the contaminated sediments and water may be incidentally ingested or they may be absorbed through the skin (dermally). Some contaminants may be volatile, and people in or near the water may breathe them. In addition, some of these chemicals are readily taken up by aquatic organisms, but only slowly metabolized and excreted by these organisms (e.g. PCBs, methylmercury) and they accumulate in the tissue of aquatic organisms. If people eat these organisms, they consume contaminants found in edible tissues.

Human health-based sediment values are derived by assuming reasonable, maximal exposure of a hypothetical individual to chemicals found in sediments. They are calculated using the sum of the internal doses from direct exposure to sediments, as well as indirect exposures. Direct exposures include dermal contact, ingestion, or inhalation of chemicals of concern (COCs) found as volatile chemicals in overlying air, dissolved or suspended with solids in water, or in the sediments themselves. Indirect exposures are typically limited to consumption of bioaccumulated chemicals in fish and other aquatic or terrestrial animals and plants. The relative importance of different routes of exposure depends on the specific COC, human activity in the waterbody of concern, uptake of COCs by aquatic organisms and plants, and likelihood of consumption of fish, wildlife and wild plants.

The percent contribution by different potential routes of exposure are calculated for individual chemicals. Therefore, the effect of removing a route of exposure from a chemical-specific evaluation can be calculated (see Section 7.3). Screening can proceed with default values and adjustments can be made (e.g. by removing an exposure route) in subsequent site-specific analyses.

Direct human exposure to sediments generally occurs while swimming and wading in contaminated sediments or water during the summer. The MPCA and MDH developed reasonable maximal exposure scenarios (RMEs) for wading and swimming in the St. Louis River. These scenarios are discussed in detail below. Additionally, MDH has developed an indirect RME for consumption of contaminated fish tissue that does not account for individuals who are subsistence fishers, or others who eat diets containing large amounts of self-caught fish. Wild rice may be harvested in parts of the St. Louis River, but MDH is unaware of any wild rice harvest in the lower St. Louis River. Further, MDH is not aware of any non-fish species that are harvested and consumed from the St. Louis River. Crayfish may be a small portion of the diet of some individuals, but there are no available consumption data, or data on contamination in crayfish. It is reasonable to assume that consumption of non-fish species for most individuals may be accounted for in a relative source contribution adjustment. Therefore, indirect exposures are limited solely to fish ingestion.

Modeled exposures were grouped into 3 age ranges: 1 through 6 year-olds; 7 through 17 year-olds; and adults (assumed to be 18 through 33 years-old). The sum of these potential exposures is 33 years; which is the 90th percentile estimate for living in one's current home (EPA 1997a). This assumption of 33 years may not be conservative, as it does not include the possibility that individuals may move inside a community. Exposed individuals are assumed to have median body weights and skin surface areas for the age groups of interest (Table 1; EPA 1997a).

Table 1 - Receptor Characteristics

	Potential Exposure	Averaging time	Body Weight *	Body surface area *
	Years	Years	kg	cm^2
Chronic Exposures	**1 - 6**	**6**	16	6,730
	7 - 17	**11**	43	13,500
	18 - 33	**16**	70	18,200
Lifetime Yearly Average Exposures				
	33 *	**70 ****	51	14,000

* (EPA 1997a), ** (EPA 1989)

3.1 What are sediments?

Sediments are materials that sink to the bottom of waterbodies. Materials that are exposed to air, but were at one time covered with water (e.g. flood plains and areas above those covered by normal tides) may be upland sediments. In addition, intertidal zones, or shallow areas and beaches that are under water at different times (such as parts of days or years) are also sediments. A third type of sediments are in areas that are typically covered by water.

Exposures to each of these types of sediment are likely to be different. If a child is playing along the shore of a lake or river, exposures to upland sediment are likely to be similar to exposures to soil in a sandbox or at a playground. In addition, contaminated dried sediments above flood elevations will not directly affect the fish tissue concentration of sediment-associated contaminants. Therefore, upland sediments should be evaluated as if they are soils.

Exposure to intertidal sediments are likely to be among the highest exposures to soils or sediments over a given duration. Small children playing where the water meets the shore are often playing in a medium that more resembles mud than water or dry soil. As children play in deeper and deeper water, exposure to sediments (especially ingestion exposure) likely becomes related to the amount of water they ingest and the amount of suspended sediment in the water.

This document calculates sediment screening values for sediments that are typically covered by water. Exposures to intertidal and water-covered sediments may be similar at different times, but it is appropriate in most cases to evaluate intertidal sediments differently than either sediment or soils. If the sediment screening values are to be used to evaluate intertidal sediments, MDH recommends: increase the sediment ingestion by a factor of 4 to adjust ingestion to soil ingestion levels (EPA 1997a); adjust the dermal-sediment adherence and contact area to reflect potential exposure to mud, and; adjust the dermal-water exposure to reflect exposures of a child playing in the intertidal zone.

3.2 Frequency of contact with sediments

Direct contact with chemicals from sediments (i.e. contact excluding fish consumption) in the lower St. Louis River generally occurs from May through September. Local residents wade along the river from May through September. Swimming in the river is typically limited to the months of June, July, and August. Table 2 shows reasonable maximum frequencies of wading and swimming in the lower St. Louis River, based on discussions with residents. During the summer, children may swim twice a day, but most exposures are assumed to occur once a day and only 2-4 days a week. The duration of each event, wading or swimming, is assumed to be ½ hour.

Reasonable maximums from EPA, used to estimate the frequency and duration that an individual may swim are similar to MDH defaults. EPA suggests that an individual may swim 1 hour per event, 1 event per day and 150 days per year for 30 years, when averaged over a lifetime (EPA 2002a). Reasonable maximum exposures scenarios used in this document are shown in Tables 2 and 3.

Table 2 - Exposure Event Frequency

Age (yr)	Wading Events			
	May, September		June, July, August	
	8.6 weeks		12.9 weeks	
	events/day	days/week	events/day	days/week
1 - 6	1	2	0	0
7 - 17	1	2	0	0
18 - 33	1	2	0	0
1 - 33	1.0	2.0	0	0

Age (yr)	Swimming Events			
	May, September		June, July, August	
	8.6 weeks		12.9 weeks	
	events/day	days/week	events/day	days/week
1 - 6	0	0	2	4
7 - 17	0	0	2	4
18 - 33	0	0	1	2
1 - 33	0	0	1.5	3.0

3.3 Default exposure assumptions

General exposure default assumptions and references are listed in Tables 3 through 8.

Acute, or short exposures to many chemicals may result in adverse health effects. Often health effects are limited to irritation that will subside when the exposure ceases. However, high acute exposures to some chemicals have more serious health endpoints (e.g. benzene with an acute MDH Health Risk Value of 1,000 microgram per cubic meter based on a developmental endpoint). While acute workplace and first-responder exposure limits are available for many chemicals, there are very few acute toxicity criteria available that are protective of public health. Furthermore, calculating acute sediment screening values would require an additional layer of complexity in this document. Therefore, acute sediment screening values are not calculated in this document. They may need to be addressed in a future document.

Exposures that can lead to chronic health impacts generally occur over periods of months to years. Chronic values developed here are from yearly average exposures. For the non-cancer endpoints, children ages 1-6 have the potential to be the most highly exposed group (Tables 3 - 8; see Appendix B for calculations). Therefore, data related to exposure of 1-6 year-olds are used for calculating all non-cancer screening values.

It is assumed that a reasonably maximally exposed individual may use the lower St. Louis River for 33 years of their life (EPA 1997a; Table 1). For the purpose of evaluating cancer risk, it is assumed that 33-year exposures (1-33 year olds from Table 1) are the total lifetime exposure. By convention, cancer risk is determined by averaging lifetime exposures over a 70 year lifetime (EPA 1989; Table 1). Some discussion of less-than-lifetime exposure risk is included in a later section (10) on children's health.

3.3.1 Sediment ingestion

EPA *Risk Assessment Guidance for Superfund* (EPA 1989) recommends using soil ingestion rates as sediment ingestion rates in risk assessments. Therefore, on an event day a child may ingest about 250 mg of intertidal or upland sediments (EPA 1997a). Arguably, 250 mg/event-day may be a high estimate of the amount of submerged sediments that are ingested by a child playing in the water. Because this document is calculating screening values for sediments under water, the calculated ingested sediment per event day is limited to the amount of water that may be ingested times the concentration of suspended sediment in the water (*Equation A-1*, Appendix B*)*.

Surface water ingestion is discussed in Section 3.3.2 and surface water ingestion amounts for different age groups are listed in Table 4. The responsible party at the St. Louis River Interlake Duluth Tar site conducted a "step-down" test that was used to measure suspended sediment concentrations in water during swimming and wading (IT Corporation 1996; 1997). The 75% confidence limit of the mean suspended sediment from samples (n=6) was 371 milligrams per liter (mg/L). This value and the surface water ingestion rate (Table 4) are used in *Equation A-1*, Appendix B, to calculate the modeled sediment ingestion rate (Table 3). Note the modeled sediment ingestion for 1 - 6 year-olds, 92.5 mg/hr and 9.25 mg/hr while swimming and wading, is considerably lower than the EPA estimated daily ingestion rate of 250 mg/day for a young child playing outdoors (1997a).

The sediment ingestion rate for swimming and wading, body weight (Table 1), event frequency (Table 2) and event duration (Table 3) are used in *Equation A-2*, Appendix B, to calculate average daily intakes of 0.818 and 0.306 mg/(kg · d) for 1 - 6 year-olds and 7 - 17 year-olds, respectively (Table 3). Adult (18 - 33 year-olds) intake is calculated to be 0.00946 mg/(kg · d). Average daily sediment intake rates for evaluating carcinogenic chemicals were calculated using *Equation A-4*, Appendix B.

Sediment ingestion assumptions for days when exposures occur (event days) are listed in Table 3.

Table 3 - Sediment Ingestion

	Potential Exposure	Wading		Swimming		Suspended Sediment Concentration	Event Duration	Sed_{Ing}
	Years	mg / hr	events / yr	mg / hr	events / yr	mg/L	hr/event	$mg_{sed}/(kg_{bw} \cdot d)$
Chronic Exposures	**1 - 6**	9.25	17.2	92.5	103	* 370	0.5	0.818
	7 - 17	9.25	17.2	92.5	103			0.306
	18 - 33	0.185	17.2	18.5	25.8			0.00946
Lifetime Yearly Average Exposures								Sed_{Ing-c}
	33	4.85	17.2	56.6	65.6	* 370	0.5	0.12

Sed_{Ing} = sediment ingested (Appendix *Equation A-2*) ($mg_{sed}/(kg_{BW} \cdot d)$)

Sed_{Ing-c} = sediment ingested - lifetime average (Appendix *Equation A-4*) ($mg_{sed}/(kg_{BW} \cdot d)$)

* 75% C.L. mean of SLRIDT Site data (IT Corporation 1997)

3.3.2 Surface water ingestion

Swimming and wading typically result in incidental ingestion of some water. The Superfund Exposure Assessment Manual (EPA 1988) states that 50 milliliters per hour (mL/hr) swimming is a reasonable estimate for incidental ingestion by an adult. Because children playing in water ingest considerably more water than adults, it is assumed that children and adolescents ingest five times the adult ingestion. There are no published data on water ingestion during wading. We assume that a reasonable adult ingestion for screening purposes is $1/100^{th}$ of the adult swimming ingestion and, because the difference between wading and swimming for children is not discrete, ingestion by 1-17 year-olds is $1/10^{th}$ of their swimming ingestion (see Table 4). Using these data, assumed body weight (Table 1) and event frequency (Table 2), average daily surface water intake rates were calculated with *Equation A-3*, Appendix B. Results are shown in Table 4. Average daily surface water intake rates for evaluating carcinogenic chemicals were calculated using *Equations A-5*, Appendix B.

Table 4 - Surface Water Ingestion

	Potential Exposure	Wading		Swimming		Event Duration	SW_{Ing}
	Years	ml / hr	events / yr	ml / hr	events / yr	hr/event	$L/(kg_{bw} \cdot d)$
Chronic Exposures	**1 - 6**	25	17.2	250	103	0.5	0.00221
	7 - 17	25	17.2	250	103		0.00083
	18 - 33	0.5	17.2	* 50	25.8		0.0000256
Lifetime Yearly Average Exposures							SW_{Ing-c}
	33	13.1	17.2	153	59.2	0.5	0.000326

SW_{Ing} = water ingested (Appendix *Equation A-3*) (L/(kg·d))
SW_{Ing-c} = water ingested - lifetime average (Appendix *Equation A-5*) (L/(kg·d))
* (EPA 1988)

3.3.3 Dermal exposure to sediment

Dermal exposure to sediments occurs during any wading or swimming event. Contaminated sediment in 4 to 10 foot water depths should not be excluded from a dermal screening assessment. Dermal guidance published by EPA (EPA 2001b) suggests that sediment in deeper water will wash off before an individual reaches shore, but there is no reference for this assertion. Suspended fines may adhere to the skin, and may not be removed without washing.

Dermal exposure to a chemical in sediment occurs by the non-active transfer of a fraction of chemical from sediment that adheres to skin, into the body. A sediment film covering the skin will typically stay on the skin until it is washed away with soap. Therefore, for this report it is assumed that the sediment remains on the skin for about 24 hours (the length of time soil was on skin in the studies that were used to determine the dermal absorbed fraction) (EPA 2001b). Further, it is assumed that if more than one swimming or wading event occurs during a single day, dermal exposure to sediment only occurs once during that day (i.e. total exposures during one year is equal to the number of event-days, not the number of events).

The relationship between activities and sediment adherence to skin is discussed in Appendix B.

The EPA Exposure Factors Handbook (EPA 1997a) was used as guidance in determining a percent of the total body surface area exposed to sediment during wading and swimming. Values from Tables 1,2 and 5, and *Equations A-6* and *A-7* from Appendix B are used to calculate dermal contact with sediments shown in Table 5.

Table 5 - Sediment Dermal Contact

	Potential Exposure	Wading			Swimming			Sed_{Derm}
	Years	% total Surface Area	Adherence (mg/cm^2)	event-days / yr	% total Surface Area	Adherence (mg/cm^2)	event-days / yr	$mg_{sed}/(kg_{bw}\cdot d)$
Chronic Exposures	**1 - 6**	20%	* 1	17.2	** 90%	0.2	51.6	14.5
	7 - 17			17.2			51.6	10.9
	18 - 33			17.2		0.07	25.8	3.62
Lifetime Yearly Average Exposures								Sed_{Derm-c}
	33	20%	* 1	17.2	** 90%	0.137	39.1	3.78

Sed_{Derm} = dermal sediment contact (Appendix *Equation A-6*) ($mg_{sed}/(kg_{bw}\cdot d)$)
Sed_{Derm-c} = dermal sediment contact - lifetime average (Appendix *Equation A-7*) ($mg_{sed}/(kg_{bw}\cdot d)$)
* (Massachusetts DEP 2002)
** (EPA 1997a)

3.3.4 Dermal exposure to surface water

Dermal exposure to surface water occurs during any wading or swimming event. Dermal exposure to a chemical in water is based on the fraction of that chemical in water non-actively transferred through the skin and into the body. Exposure only occurs while the event is taking place. Therefore, more than one event during a single day results in more than one exposure. Difficulties in approximating the internal doses that result from dermal exposure to chemicals in water are related to problems in resolving issues such as: competing, time-dependent actions such as slow transfer of non-polar organic chemicals through the skin and desquamation of skin (loss of dead skin); activity-dependent renewal of water in contact with skin, and; inherently low non-polar organic chemical concentration in water.

The percent of the total body surface area that is exposed to surface water during wading and swimming is assumed to be the same as the amount that is exposed to sediment. Values used to calculate dermal contact with surface water are listed in Tables 1,2 and 6. Results from *Equations A-8* and *A-9*, Appendix B, are shown in Table 6.

Table 6 - Surface Water Dermal Contact

	Potential Exposure	Wading		Swimming		SW_{Derm}
	Years	% total Surface Area	events / yr	% total Surface Area	events / yr	$cm^2/(kg_{bw} \cdot d)$
Chronic Exposures	**1 - 6**	20%	17.2	* 90%	103	109
	7 - 17		17.2		103	82.3
	18 - 33		17.2		25.8	19
Lifetime Yearly Average Exposures						SW_{Derm-c}
	33	20%	17.2	* 90%	59.2	26.7

SW_{Derm} = surface area exposed to surface water (Appendix *Equation A-8*) ($cm^2/(kg \cdot d)$)
SW_{Derm-c} = surface area exposed to surface water - lifetime average (Appendix *Equation A-9*) ($cm^2/(kg \cdot d)$)
* (EPA 1997a)

3.3.5 Inhalation exposure to volatile chemicals

Inhalation exposures to chemicals are calculated by determining the proportion of time an individual may be exposed over an entire year. This ratio times the potential exposure concentration should not exceed health values for inhalation exposure. Fractions of years and lifetime spent wading or swimming are presented in Table 7 (results of *Equations A-10* and *A-11*, Appendix B).

Table 7 - Inhalation Fraction

	Potential Exposure	Wading	Swimming	Event Duration	Inh_{frac}
	Years	events / yr	events / yr	hr/event	(Unitless)
Chronic Exposures	**1 - 6**	17.2	103	0.5	0.00686
	7 - 17	17.2	103		0.00686
	18 - 33	17.2	25.8		0.00245
Lifetime Yearly Average Exposures					Inh_{frac-c}
	33	17.2	59.2	0.5	0.00223

Inh_{frac} = fraction of time onsite (Appendix *Equation A-10*) (unitless)
Inh_{frac-c} = time onsite - lifetime average (Appendix *Equation A-11*) (unitless)

3.3.6 Fish consumption exposure

According to the EPA, reasonable rates of fish ingestion range from 17.5 grams / day (g/d) intake for the general population to 142.4 g/d for subsistence fishers (EPA 2000b). On the other hand, a single fish meal-per-week consumption rate (or 30 g/d) is the basis for all Minnesota human health-based water quality standards in Minnesota Rules (Chapters 7050 and 7052). Therefore for this report, it is assumed that a reasonable fish consumption rate for an adult is 30 g/d. This intake rate is based on the presumed ingestion of a single 210 g meal per week by a 70 kilogram (kg) adult. Ingestion for all age groups is scaled to this rate (Table 8). Calculations use data from Tables 1 and 8, and *Equations A-12* and *A-13*, Appendix B.

Table 8 - Fish Ingestion

	Potential Exposure	Meal Frequency	Amount Consumed	Ing_{Fish}
	Years	meals / wk	grams / meal	$g_{fish}/(kg_{bw} \cdot d)$
Chronic Exposures	**1 - 6**			0.431
	7 - 17			0.431
	18 - 33	1	210	0.431
Lifetime Yearly Average Exposures				Ing_{Fish-c}
	33			0.203

Ing_{fish} = fish ingestion rate (Appendix *Equation A-11*) ($g_{fish}/(kg_{bw} \cdot d)$)
Ing_{fish-c} = fish ingestion rate - lifetime average (Appendix *Equation A-12*) ($g_{fish}/(kg_{bw} \cdot d)$)

4 Potential Chemicals of Concern

Potential chemicals of concern can be categorized as metallic compounds and other inorganic and organic compounds. Metals are rarely found in the environment in the elemental form but exist in many different compounds (species). The relative prevalence of chemical species (mostly inorganic compounds) is often determined by chemical conditions in the environment, including pH (acidity) and Eh (oxidation-reduction or redox potential). In addition, some metals may exist in toxicologically important organic compounds (e.g. monomethyl mercury or trimethyl tin). Most non-metallic inorganic compounds discharged into sediments degrade rapidly and, therefore, are not found in high enough concentrations to adversely impact human health following exposure. However, some cyanide compounds can affect human health and there were historic sources of cyanides in the lower St. Louis River, so cyanides are evaluated.

Organic compounds can remain unchanged in the environment for varying lengths of time from very short to very long. Compounds with short half-lives (i.e. hours) will not accumulate in sediments and, therefore, exposure to them in sediments is unlikely unless there is a recent release. However, some compounds with relatively short half-lives, such as benzene and other volatile organic compounds (VOCs), may remain in sediments for extended periods of time if they were released in large enough quantities or if they are constituents of a highly contaminated organic layer. Of potentially greater concern are more persistent organic chemicals such as polycyclic aromatic hydrocarbons (PAHs) and non-polar chlorinated organics. PAHs are major constituents of petroleum products and wastes from burning organic fuels (e.g. coal tar). High concentrations of PAHs can sometimes form a non-aqueous phase liquid (NAPL) in sediments.

Chemicals that bioaccumulate in the aquatic food chain are of concern because fish consumption is often the largest source of these chemicals for people. Bioaccumulative chemicals are efficiently passed up the aquatic food chain and only slowly excreted by biota. As a result, animals at the top of the food chain may have the highest concentrations of bioaccumulative chemicals in their tissues. People who consume large amounts of predatory fish may also ingest large amounts of bioaccumulative chemicals.

The US EPA and Environment Canada have identified 12 substances as Level I Binational Toxics Strategy (BNS) substances: aldrin/dieldrin, benzo[a]pyrene, chlordane, DDT, hexachlorobenzene, alkyl-lead, mercury and compounds, mirex, octachlorostyrene, PCBs, dioxins and furans, and toxaphene (EPA 1998a). These substances are priority contaminants to be addressed during environmental sampling and remedial action in the Great Lakes Region.

Bioaccumulative chemicals that are not addressed in this report include: tin, palladium and thallium that can form organic-metal compounds similar to those formed by mercury (non-polar metal-carbon bonds: Bailey et al. 1978); persistent organic pesticides (e.g. aldrin/dieldrin, chlordane, DDT, mirex, and toxaphene) and their metabolites, and; organic compounds for which there are only limited environmental data including: perfluorinated alkyl compounds, polybrominated diphenyl ethers (PBDEs), chlorinated PAHs (primarily naphthalenes), polybrominated dibenzodioxins (PBDDs) and polybrominated dibenzofurans (PBDFs). Relevant health criteria are not available for most of these chemicals.

4.1 Inorganic chemicals and metals

Calculating exposure to metals from all six routes-of-exposure addressed in this SSV Report is not possible given the limitations of current partitioning models. Different species of each metal may have considerably different solubility. Equilibriums between total metal concentration in interstitial water (dissolved) and total metal in sediment (solid) will likely be different in areas with different substrate and different chemistry. Acid volatile sulfides (AVS), organic matter and iron oxides in sediments can control the solubility and availability of most metals. Furthermore, if sediment is disturbed, as it will be when someone wades through it, oxidation-reduction conditions can change causing reactions that may enhance or decrease the availability of the metal. The magnitudes of these effects are uncertain, as is the time-dependence of these reactions in the environment.

As a result of these uncertainties, evaluation of potential exposures to metals from sediments does not include evaluation of exposures to metals partitioning from sediment into water, or into air. For most metals, only a single route of exposure was evaluated: direct ingestion of metals in sediment. Evaluation of dermal exposure to metals is limited to potential exposures to arsenic and cadmium as recommended by EPA (2001b).

4.1.1 Mercury

Data from the upper St. Louis River (above Cloquet) suggest that the concentration of mercury in sediments that are not directly impacted by human activity may be about 0.02 mg/kg (Glass et al. 1999). Possible sources of mercury to the St. Louis River include: effluent from municipal waste and upstream paper plants; as well as local effluent and air emissions from coal-burning facilities, and; air emissions from regional taconite processing facilities. Currently in Minnesota, commercially burned coal contains about 0.1 mg/kg mercury (MPCA 2000). Mercury content of coal in years past was likely higher. Most mercury that is burned in coal will go up the furnace stack and be locally, regionally, or globally distributed. It is not known what percent of mercury in coal burned in industrial facilities along the St. Louis River has deposited locally and regionally. In addition, some mercury may be found in coal tar sludge from coke ovens, manufactured gas plants, and other heavy industry facilities. Large amounts of elemental

(liquid) mercury can also be found in industrial meters and electrical switches, as well as in meter and switch repair shops.

Mercury in the aquatic environment may be elemental, inorganic, or organic. Elemental mercury is not soluble or reactive; it volatilizes slowly over time. Therefore, elemental mercury is not often encountered at high concentrations in the environment unless there has been a spill. Concentrations in outdoor air are typically between 1 and 4 nanograms per cubic meter (ng/m^3) (Slemr et al. 2003).

Inorganic mercury is primarily found in sediments, with very low concentrations found in surface water. REMAP total mercury sediment concentrations in the St. Louis River estuary ranged from 0.005 - 0.702 mg/kg (EPA 1995). REMAP data are from analyses of cores from three types of sediments: Class 1, shallow areas; Class 2, channel areas; and Class 3, Thomson Reservoir above Fond du Lac Dam (see Attachment 1 for sample locations). Only Class 1 and Class 2 data were acquired in the St. Louis River estuary. Therefore, all REMAP data cited for this document are data from Class 1 and Class 2 cores. It is necessary to note that REMAP data are not background concentrations (background in this document does not mean pre-anthropogenic background, but signifies chemical concentrations without a defined local or regional source), but the data set may be a good representation of the ambient concentrations in areas that have not been identified as sources of contaminants. However, in dynamic systems such as rivers and waterbodies affected by seiches, contaminants can accumulate far from the sources of contamination. Analysis of REMAP data (see Appendix C) appears to agree with upstream data from Glass et al. (1999), suggesting that background concentrations of mercury in the St. Louis River may be about 0.02 mg/kg.

Total mercury (mostly inorganic) in surface water in the St. Louis River is about 4 ng/L (unfiltered water, STORET data (EPA STORage and RETrival database) reviewed by MPCA). Methylmercury is also found in sediments and surface water, but it typically accounts for only about 1 - 10 % total mercury (Krabbenhoft et al. 1999). Small aquatic organisms appear to take up both inorganic mercury and organic mercury (Becker and Bigham 1995; Lasorsa and Allen-Gil 1995; Tremblay et al. 1996). When small organisms are consumed by larger organisms, the methylmercury accumulates, so that fish and other piscivores at the top of the aquatic food chain typically have the highest concentrations of mercury, about 95% as methylmercury (Bloom 1992).

Potential exposures to both methylmercury, through ingestion of fish tissue, and inorganic mercury, ingested directly from sediments, are calculated in this report.

4.1.2 Cadmium

The mean concentration of cadmium in soils worldwide is about 0.5 mg/kg (Kabata-Pendias and Pendias 2001). However, cadmium concentrations can be elevated in areas where soils are impacted by anthropogenic activity. Cadmium may be emitted from metal processing facilities and some smelters. Cadmium has also been used in paints; however this use has been mostly curtailed. Elevated levels of cadmium can be found in phosphate fertilizers.

Cadmium, and to a lesser extent lead, can accumulate in aquatic organisms. However, they typically accumulate in the hepatopancreas (Chou et al. 2000) or liver. Many people do eat the hepatopancreas of lobster (tomalley), but MDH does not know if individuals eat the hepatopancreas of crayfish caught in the St. Louis River. Fish liver (e.g. whitefish liver) is a delicacy that is served in some restaurants in northern Minnesota. Consumption of fish livers was not evaluated in this document because there are no liver tissue concentration data and no liver consumption data for the St. Louis River estuary. The potential accumulation of cadmium in aquatic species, and the subsequent ingestion of these organisms by people was not evaluated. Cadmium exposure by ingestion of sediment and dermal exposure to cadmium in sediment were evaluated. However, it is likely that the cadmium ecological lowest effect level of 0.6 mg/kg in sediments (Ontario Ministry of the Environment 1993) will be more restrictive than human health-based values.

A joint EPA, MPCA study (1993 'Mudpuppy' Study, EPA and MPCA 1997a) found cadmium in sediment between 0.52 and 7.4 mg/kg (mean = 2.3 mg/kg, standard deviation = 1.4 mg/kg, median = 2.05 mg/kg). The two highest cadmium concentrations were found in samples, adjacent to the US Steel site (7.4 and 5.5 mg/kg).

There are no cadmium data that describe ambient concentrations in identified non-source areas of the St. Louis River Estuary. Cadmium sediment concentration in the REMAP study was determined using a method that is intended to simultaneously extract metals (SEM) including, lead, cadmium, copper, nickel and zinc, and acid volatile sulfides (AVS) from sediments (for information on AVS/SEM see Di Toro et al. 1992; Hansen et al. 1996). While this method can be used to determine the potential toxicity to benthic organisms in the environment, it is not useful for determining metal concentrations in sediments or bioavailability of metals in sediment to people.

4.1.3 Lead

A study by Boerngen and Shacklette (1981) showed subsurface soil concentrations of lead in rural areas of Minnesota to be between <10 and 20 mg/kg. Lead concentrations can be elevated in areas where soils are impacted by anthropogenic activity. Lead may be emitted from metal processing facilities and some smelters. In addition, lead was historically used in paint, and high concentrations of lead are often found in soil adjacent to old houses. Lead can also be found at elevated levels in sediments. Furthermore, as noted in the previous section (4.1.2), some lead may accumulate in non-muscle tissue of fish and other aquatic organisms.

Lead exposure was not evaluated by any route in this document. Because there is no known threshold exposure below which lead has no effect, lead exposure is generally associated with some health hazard. In the absence of more specific criteria, it is reasonable to apply soil criteria to sediment as recommended by EPA (EPA 1989). The current Minnesota Soil Reference Value (SRV) for lead is 400 mg/kg (MPCA 1999). This criterion applied to soil is reasonably anticipated to result in blood lead concentrations below 10 micrograms per deciliter (µg/dL) (MDH 2002a). *Minnesota Statute 144.9504* applies a more restrictive standard of 100 mg/kg for bare soil at a property under a number of conditions including: 1) a child has a blood lead level at or greater than 20 µg/dl, 2) a child has a blood lead level 15-19.9 µg/dl that persists for 3 months,

or 3) a pregnant woman has a blood lead level at or above 10 µg/dl. In addition, *Minnesota Statute 144.9503* states that priority sites for primary prevention of toxic lead exposure include census tracts with median soil lead concentrations equal to or greater than 100 mg/kg. It is likely that a protective ecological sediment value, such as the Ontario lowest effect level of 31 mg/kg (Ontario Ministry of the Environment 1993) will be more restrictive than a value based on public health policy.

The EPA, MPCA 'Mudpuppy' Study (1993 'Mudpuppy' Study, EPA and MPCA 1997a) looked at lead concentrations near potential sources and found a range of lead concentrations in sediment between 1.5 and 548 mg/kg (mean = 58.3 mg/kg, standard deviation = 105 mg/kg, median = 17.0 mg/kg). The highest concentrations in this data set (548 and 289 mg/kg) were measured in sediment adjacent to the US Steel site.

Lead REMAP data for the St. Louis River was restricted to AVS/SEM data, and is not useful for human risk characterization.

4.1.4 Arsenic

Background concentration of arsenic in soil in Minnesota is about 5 mg/kg (Boerngen and Shacklette 1981; mean 5.5 mg/kg, median 4 mg/kg, n=37). Elevated arsenic levels are often found in areas contaminated by smelters and ore-processing industries, as well as coal fired industries. In addition, arsenic has been used historically in pesticides and wood preservatives. Ash from burning chromated copper arsenate (CCA) treated wood can have very high concentrations of arsenic, chrome and copper. Arsenic may also be found in high concentrations in some 'natural' soil supplements.

The REMAP study did not measure total arsenic concentrations in sediments in the St. Louis River Estuary. The 1993 'Mudpuppy' Study (EPA and MPCA 1997a) looked at arsenic concentrations near potential sources and found a range of arsenic concentrations in sediment between 0.4 and 33.5 mg/kg (mean = 9.6 mg/kg, standard deviation = 8.3 mg/kg, median = 6.8 mg/kg). The highest concentration in this data set (33.5 mg/kg) was measured in sediment adjacent to the US Steel site.

Non-mineralized arsenic chemical species in the environment are generally soluble. However, arsenic mobility may be limited by its affinity for and adsorption to clays, hydroxides and organic materials (Kabata-Pendias and Pendias 2001). Potential exposures to arsenic by ingestion and dermal contact with sediments are evaluated.

4.2 Persistent organic compounds

Historic human activity in the St. Louis River area has resulted in deposition of persistent organic compounds in river sediments. Canada and the United States have developed a Binational Toxics Strategy (BNS) to address these toxicants. Not all of the BNS targeted substances have been evaluated, but the methods used to calculate sediment screening values in this report can be used to calculate similar values for other persistent organics. Organic pollutants in this report that are evaluated for all 6 routes of exposure are: polychlorinated dibenzo-p-dioxins, polychlorinated dibenzofurans, polychlorinated biphenyls,

hexachlorobenzene, octachlorostyrene, polycyclic aromatic hydrocarbons, dibenzofuran, carbazole and volatile organics; benzene, ethyl benzene, styrene, toluene, and xylenes.

4.2.1 Chlorinated organics

4.2.1.1 Polychlorinated dibenzo-p-dioxins, polychlorinated dibenzofurans and dioxin-like polychlorinated biphenyls

Polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and polychlorinated biphenyls (PCBs) are persistent non-polar organic compounds. PCDDs and PCDFs are groups of 75 and 135 similar chemicals, respectively, that are not intentionally produced. Instead, they are either inadvertent byproducts of production (e.g. 2,4,5-trichlorophenoxy acetic acid), or byproducts formed from flue gases during the burning of organic compounds (e.g. coal, plastics). Natural processes, such as fires and volcanoes can also produce PCDDS and PCDFs. PCBs are a group of 209 chlorinated organics that were produced for use in high temperature oils and as insulating coolants in electric transformers. In addition, some PCBs can be accidental products of manufacturing processes that form PCDDs and PCDFs. Some carcinogenic PCB (cPCB) congeners behave toxicologically like dioxins and have been identified by the World Health Organization's (WHO) as dioxin-like (Van den Berg et al. 1998).

The MDH, U.S. EPA, National Toxicology Program (NTP) and the International Agency for Cancer Research (IARC) have characterized 2,3,7,8-Tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) as a "human carcinogen". The MDH and the U.S. EPA have classified the complex mixtures of PCBs, PCDDs and PCDFs to which people are exposed as "likely human carcinogen(s)". Subsets of the PCBs, PCDDs and PCDFs in mixtures are also likely to be carcinogenic to humans (see Table 9). While these congeners have different potencies, it is believed that they act through the same mechanism. MDH recommends utilization of the WHO 1998 toxic equivalency factor (TEF_{WHO98}) scheme (Van den Berg et al. 1998) to weight each compound's relative cancer risk. Potency is scaled relative to the toxicity of 2,3,7,8-TCDD, which is the most studied and, apparently, the most toxic chemical in this group. Total 2,3,7,8-TCDD Toxic Equivalency (TCDD-TEQ) exposure concentration is equal to:

$$\text{TCDD-TEQ } \{ mg_{sed}/(kg_{bw} \cdot d) \} = \sum Exp_i * TEF_i \quad \text{for i = each chemical with a } TEF_{WHO98} \qquad \textit{Equation 1.}$$

Where:

Exp_i = the exposure concentration for each dioxin or dioxin-like compound (mg/(kg·d))

TEF_i = the TEF_{WHO98} for each dioxin or dioxin-like compound

Note that Exp_i is an exposure concentration for each dioxin-like compound and not the sediment concentration. Because the physical characteristics of different dioxin-like chemicals can be different, partitioning between skin and sediment, or intestinal lumen and sediment, can be different for different congeners. For example, if the octanol-water coefficient (K_{ow}) between 2 different dioxin-like compounds varies by a factor of 10 (e.g. 2,3,7,8-TCDD and 1,2,3,4,7,8-HCDD: see Table 9 below), the calculated partitioning between water and sediment would then vary by a factor of about 10, resulting in a 10-fold difference in estimates of the concentration in water that is ingested or contacts the skin.

In practice the actual difference in total risk may be less than this implies, because contributions by different congeners are both over and underestimated, and generally, only a few congeners contribute most of the risk in any environment. While screening values used in conjunction with TCDD-TEQs can provide a rough estimate of the potential dioxin and dioxin-like risk, a measure of uncertainty can be removed in subsequent analyses by calculating potential exposure, at a specific site. Specific chemical and physical data for the individual dioxin and dioxin-like compounds can be used to calculate TCDD-TEQs for individual chemicals. These TCDD-TEQs can then be added and evaluated to see if they exceed exposure values for 2,3,7,8-TCDD. However, this is beyond the scope of this document. (For additional information on the toxicity of dioxin and dioxin-like compounds please see MDH 2003b; 2003a).

The TEF_{WHO98} values and partition information (Log K_{ow}) are listed in Table 9 below.

Table 9 - TCDD-TEFs

Compound	TCDD-TEF$_{WHO98}$*	Log K_{ow}**
Polychlorinated Dibenzo-p-dioxins (PCDDs)		
2,3,7,8-TetraCDD	1	6.8
1,2,3,7,8-PentaCDD	1	6.64
1,2,3,4,7,8-HexaCDD	0.1	7.8
1,2,3,6,7,8-HexaCDD	0.1	
1,2,3,7,8,9-HexaCDD	0.1	
1,2,3,4,6,7,8-HeptaCDD	0.01	8.0
1,2,3,4,6,7,8,9-OctaCDD	0.0001	8.2
Polychlorinated Dibenzofurans (PCDFs)		
2,3,7,8-TetraCDF	0.1	6.1
1,2,3,7,8-PentaCDF	0.05	6.79
2,3,4,7,8-PentaCDF	0.5	6.5
1,2,3,4,7,8-HexaCDF	0.1	7.0
1,2,3,6,7,8-HexaCDF	0.1	
2,3,4,6,7,8-HexaCDF	0.1	
1,2,3,7,8,9-HexaCDF	0.1	
1,2,3,4,6,7,8-HeptaCDF	0.01	7.4
1,2,3,4,7,8,9-HeptaCDF	0.01	6.9***
1,2,3,4,6,7,8,9-OctaCDF	0.0001	8.0
Polychlorinated Biphenyls (cPCBs)		
3,3'4,4'-TetraCB (PCB 77)	0.0001	6.5
3,4,4',5-TetraCB (PCB 81)	0.0001	6.36
2,3,3',4,4'-PentaCB (PCB 105)	0.0001	6.0
2,3,4,4',5-PentaCB (PCB 114)	0.0005	6.65
2,3',4,4',5-PentaCB (PCB 118)	0.0001	7.12
2',3,4,4',5-PentaCB (PCB 123)	0.0001	6.74
3,3',4,4',5-PentaCB (PCB 126)	0.1	6.89
2,3,3',4,4',5-HexaCB (PCB 156)	0.0005	7.16
2,3,3',4,4',5'-HexaCB (PCB 157)	0.0005	7.19
2,3',4,4',5,5'-HexaCB (PCB 167)	0.00001	7.09
3,3',4,4',5,5'-HexaCB (PCB 169)	0.01	7.46
2,3,3',4,4',5,5'-HeptaCB (PCB 189)	0.0001	7.71

* (Van den Berg et al. 1998)
** (EPA 2000c)
*** (Lancaster University 2003)

Background concentrations of dioxin-like compounds in sediments are also characterized as TCDD-TEQs. The EPA Dioxin Reassessment (EPA 2000c) states that background TCDD-TEQs for US lakes (11 lakes and reservoirs) are between 0.189 and 18.5 ng/kg, with a mean of 5.8 ng/kg (total PCDDs / PCDFs from 9.1 to 2,916 ng/kg; total cPCBs from 83 to 2159 ng/kg) (Cleverly 2004). These concentrations are well above the Sediment Screening Values calculated in this document. Therefore, in most cases background concentrations may be used as site-specific sediment screening values.

Data are insufficient to evaluate background concentrations for dioxin-like compounds in St. Louis River sediments. The 1993 'Mudpuppy' Study (40 sample locations specifically targeting potential source areas) evaluated 2,3,7,8-Tetrachlorodibenzo-p-dioxin and 2,3,7,8-Tetrachlorodibenzofuran (EPA and MPCA 1997a). 2,3,7,8-Tetrachlorodibenzo-p-dioxin was detected at 4 locations (mean 6.4 ng/kg; range 0.9 - 13 ng/kg). 2,3,7,8-Tetrachlorodibenzofuran was detected at 12 locations (mean 8.3; range 1.8 - 15 ng/kg). (The 2 samples adjacent to the US Steel site had the highest TCDD-TEQs (2,3,7,8-TCDD and 2,3,7,8-TCDF only) with 8.9 and 14.3 ng/kg.) However, these data likely represent only a small fraction of the total TCDD-TEQ. As was seen in the 11 Lakes Dioxin Study (Cleverly et al. 1996; EPA 2000c), 2,3,7,8-TCDD accounted for between $1/20^{th}$ and $1/60^{th}$ of the TCDD-TEQs in sediment samples from three waterbodies in upstate New York.

The 1993 'Mudpuppy' Study 2,3,7,8-TCDD/F sample data are likely higher than either ambient 2,3,7,8-TCDD/F concentrations in the estuary or regional background 2,3,7,8-TCDD/F, because sediment samples were taken from areas with identified point sources. The 11 Lakes Study (data summarized in EPA 2000c) shows that total dioxins in sediment in the lakes and reservoirs studied contribute between 28 and 74% of the total TCDD-TEQ, with the single 2,3,7,8-TCDD congener contributing 1.4 to 28% of the total. Furans and PCBs account for the remainder of the TCDD-TEQ. In addition, the range of total TCDD-TEQs in the 11 Lakes Study varies over 2 orders of magnitude (0.19 to 18 ng/kg). Furthermore, the data suggest that background concentrations of CDD/CDF are different in different regions of the country. The eastern mean lake/reservoir TCDD-TEQ background concentration was 11.7 ng/kg (±5.7), whereas the western US mean TCDD-TEQ from the 11 Lakes Study was 0.93 ng/kg (±0.43). In the absence of regional data from Minnesota or the St. Louis River, a reasonable TCDD-TEQ background concentration in an area like the St. Louis River Estuary is likely to be within this range. However, it should be noted that the St. Louis River Estuary is not a lake, and background concentrations within a riverine system may be different.

4.2.1.2 Polychlorinated biphenyls

Polychlorinated biphenyls (PCBs) are a group of 209 chemicals with similar structure. Mixtures of these congeners were used as: dielectrics and thermostatic fluids; swelling agents in seals; additives or base for lubricants, oils and greases; and plasticizers (Verschueren 1977). Use of PCBs in the United States was banned in 1977.

Background data from the St. Louis River are sparse, and concentrations in locally collected samples may be elevated by industrial discharges and atmospheric deposition from local sources. Regional background sediment data are available from areas around Lake Superior, including

Siskiwit Lake on Isle Royale in Lake Superior (Swackhamer et al. 1988). The geometric mean total PCB (tPCB) concentration from the top 1 centimeter (cm) of four cores in Siskiwit Lake was 0.048 mg/kg. This concentration is higher than tPCB sediment concentrations in other areas around Lake Superior, and may be the result of deposition from sources in Thunder Bay, Ontario. Total solid deposition to the Siskiwit Lake sediments was shown to be about 0.19 cm per year, therefore; tPCB deposition is quite low (calculated to be 5.6 micrograms per square meter per year ($\mu g/(m^2 \cdot yr)$)).

The 1993 'Mudpuppy' Study showed a range of tPCB concentrations from 0.0043 - 0.439 mg/kg in the upper-most sections (0-31 cm) of cores from lower St. Louis River sediments (EPA and MPCA 1997a). In addition, 'Mudpuppy' data showed tPCB concentrations as high as 0.612 mg/kg in core segments from 36-66 cm. These tPCB data suggest, for a number of cores, either small sections of very high PCB contamination diluted by 30-31 cm of sediment, or a rate of deposition (flux) of tPCBs that was much greater than tPCB flux into Isle Royale sediments. Similarly to the CDD/CDDF sediment data reviewed above, these data suggest that total solids deposition rate, as well as sediment contaminant concentration is important in determining whether contaminants are a result of background levels, ambient contamination, or point sources. Most 'Mudpuppy' sample locations "were selected based on known proximity to current or former source discharges" (EPA and MPCA 1997a) and therefore it is assumed that these PCBs are the result of nearby pollution. tPCB concentrations in the 2 surficial core samples (0-31 cm depth) adjacent to US Steel were 0.190 and 0.116 mg/kg.

Total PCB (tPCB) concentration in sediment and water are used to calculate the non-cancer health hazard that may be associated with chronic exposure to PCBs. (Aroclor 1254 is used as conservative toxicity surrogate for tPCBs.) On the other hand, cancer risk from PCBs is presumed to be related to exposure to a subset of PCBs that are carcinogenic (cPCBs). These cPCB appear to have a mechanism of action similar to carcinogenic dioxins and dibenzofurans. Therefore the cancer risk of dioxin-like PCBs (cPCBs) is expressed in TCDD-TEQs. cPCB congeners are listed in Table 9 above; and a sediment screening value for cPCBs is not calculated separately, but is included in the calculation of a TCDD-TEQ SSV.

Different methods of chemical analysis of PCBs can give congener, mixture and/or homologue (based on the number of chlorines per congener) data. Congener analysis (EPA Method 1668a; not yet promulgated) results in the most useable data, because concentrations of all congeners can be added for tPCB evaluation, or the toxicity of individual dioxin-like PCBs can be weighted using a toxic equivalence (TEQ) method. On the other hand, mixture analysis (i.e. Aroclor analysis) limits evaluation to PCB mixtures and tPCBs. As a result, TEQs for dioxin-like PCBs cannot be calculated and cancer risk from dioxin-like compounds may be underestimated. Homologue analysis (based on the number of chlorines in each congener, i.e. mass weight) is not useful for human health risk characterization, because there are no data available with which to quantify risk from exposure to homologue concentrations or doses. Bioassay analyses, while potentially useful for evaluating TCDD-TEQs including the contribution of cPCBs, are not available for tPCB analyses. PCB congener analysis is therefore needed for tPCB and cPCB evaluation.

4.2.1.3 Other chlorinated organics (HCB, OCS)

Hexachlorobenzene (HCB) has been used in the manufacture of wood preservatives (e.g. pentachlorophenol), fungicides, tetrachloroethylene and aromatic fluorocarbons. Octachlorostyrene (OCS) is formed during smelting processes; fuel combustion and waste incineration processes; the production of ethylene dichloride/vinyl chloride; the manufacture of chlorinated phenols used in pesticides and wood preservation; and in the production of pulp and paper (EPA 1998b). In addition, chemicals such as HCB and OCS are industrial chemicals that may have been used or can be accidentally formed by industrial processes including coking and iron and steel production (EPA 1999; 1998b).

HCB and OCS are of potential concern in the lower St. Louis River because they are persistent and toxic chemicals that can accumulate in the food chain. HCB and OCS do not occur naturally. Concentrations in sediments may be elevated in areas of the St. Louis River. HCB ranges from non-detect (ND - varies with sample) to 2.0 µg/kg (ppb). The OCS range is ND (0.01 ppb) to 8.5 ppb (31 cm homogenized samples) (EPA and MPCA 1997a). HCB concentrations in sediment adjacent to the US Steel site were 0.11 and 0.99 µg/kg, and OCS measure concentrations were ND and 8.5 µg/kg. Mean surficial HCB concentrations in Lake Superior sediments (n=13) range from 0.2 to 0.7 µg/kg (1980 sample collection; 1 cm sample depth) (Oliver and Nicol 1982), and 0.09 to 1.80 in 8 arctic lakes (1979 - 1988 sample collection; 0.5 and 1.3 cm sample depths) (Muir et al. 1995). OCS sediment ambient and background data are not readily available.

4.2.2 Polycyclic aromatic hydrocarbons

PAHs are a group of hundreds of organic chemicals with similar structures. Generally, PAHs are products of fossil fuel or organic combustion (pyrogenic). They may also be found in non-combusted fossil fuels (petrogenic). PAHs are always found in the environment as complex mixtures. While the actual toxicity of individual PAHs to humans has been quantified for only a few of these compounds, PAHs are generally considered to affect the liver (Sipes and Gandolfi 1991). Additionally, PAH mixtures can cause acute dermal irritation due to photoactivation if they are exposed to light while on a person's skin (Johnson and Ferguson 1990).

A number of PAHs have been identified as probable human carcinogens (cPAHs) by the EPA (EPA 2003c), the International Agency for Research on Cancer (IARC 2005), the National Toxicology Program (NTP 2001), and the California EPA Office of Environmental Health Hazard Assessment (CA OEHHA 2002). Other PAHs have been shown to be carcinogenic to animals (e.g. napthalene; NTP 2000) or to be mutagens (e.g. 3-nitrobenzanthrone; Enya et al. 1997). Therefore, cancer slope factors for additional PAHs may be developed in the future as better human or animal data become available.

An EPA peer-consultation workshop (EPA 2002b) recommended the use of mixture surrogate (use of data from similar mixtures), mixture comparative potency (use of data from a group of similar mixtures and comparative assays) or individual compound potency equivalence (component evaluation) for evaluating the toxicity of PAH mixtures. The use of surrogate mixtures and comparative potency are preferable to using potency equivalents when evaluating sites. However at the screening stage, site-specific data are not available with which to conduct

whole mixture toxicity evaluations. Furthermore, analytical data are needed to evaluate the homogeneity of the sediments across the site prior to any whole mixture analysis. Therefore a potency equivalence approach as outlined in the MDH memo of May 2001 (MDH 2001a) for cPAHs screening assessment is appropriate.

MDH has a draft multimedia Health Risk Value of 0.001 µg/(kg·day) for benzo[a]pyrene (B[a]P) that is based on a slope factor of 7.3 $(mg/(kg \cdot day))^{-1}$ (MDH 2002b). This slope factor is the geometric mean of the B[a]P slope factor range used by the EPA (2003c).

MDH recommends that analyzed PAHs include cPAHs in Table 10 (MDH 2001a). The B[a]P-PEFs in Table 10 are based on individual cPAH cancer slope factors, or California Potency Equivalency Factors (PEF: CA OEHHA 2002). (Note: TCDD-TEQs are used to evaluate both cancer risk and chronic health hazard for dioxin-like compounds, as they are believed to be mediated by binding to a single receptor. B[a]P-PEF can only be used to evaluate cancer risk, as the mechanisms by which PAHs initiate cancer and chronic diseases may be different.) B[a]P, with a B[a]P-PEF of 1, is the index compound. Total B[a]P-PEQs can be calculated using an algorithm similar to *Equation 1* (above) for dioxin and dioxin-like TEQs. If the cancer risk for some individual cPAHs is a risk driver, further review of potency slopes may be needed. While some of the listed cPAHs may not be found in the St. Louis River, most have been found in sediments at other locations (e.g. Fernandez et al. 1992).

Sediment screening values for cPAHs have uncertainties similar to those described for TCDD-TEQ-based values. If PEFs are applied to sediment concentrations, some accuracy in calculated B[a]P-PEQs will be compromised. On the other hand, partitioning data are not available for all cPAHs and internal doses of all cPAHs cannot be calculated. For screening purposes, B[a]P-PEFs may be applied to sediment data. But uncertainties should be addressed for each individual site or application.

Table 10 - B[a]P-PEFs

cPAH	B[a]P-PEF	Log K_{ow}*
Benzo[a]pyrene**	1	6.13
Benz[a]anthracene	0.1	5.76
Benzo[b]fluoranthene	0.1	5.78
Benzo[j]fluoranthene	0.1	6.11
Benzo[k]fluoranthene	0.1	6.11
Dibenz[a,j]acridine	0.1	5.63
Dibenz[a,h]acridine	0.1	5.73
Dibenz[a,h]anthracene***	0.6	6.75
Dibenzo[a,e]pyrene	1	7.28
Dibenzo[a,h]pyrene	10	7.28
Dibenzo[a,i]pyrene	10	7.28
Dibenzo[a,l]pyrene	10	7.71
7H-Dibenzo[c,g]carbazole	1	5.8
7,12-dimethylbenzanthracene***	30	5.8
Indeno[1,2,3-c,d]pyrene	0.1	6.7
3-methylcholanthrene***	3	6.42
5-Methylchrysene	1	6.07
5-nitroacenaphthene***	0.02	3.85
1-Nitropyrene	0.1	5.06
4-Nitropyrene	0.1	
1,6-Dinitropyrene	10	
1,8-Dinitropyrene	1	
6-Nitrochrysene	10	
2-Nitrofluorene	0.01	3.37
Chrysene	0.01	5.81

* (Syracuse Research Corporation 2003)

** Index compound

*** Based on chemical's cancer slope factor and relative to B[a]P CSF (7.3 $(mg/(kg \cdot d))^{-1}$)

The primary health endpoints for non-carcinogenic PAHs (nPAHs) vary, but most have multiple toxicity endpoints that are similar. Therefore, given the general similarity between the non-cancer effects of PAHs, MDH has recommended that the hazard quotients for nPAHs be added in risk assessments for sites including the US Steel site (MDH 2001b).

5 Chemical specific data

Chemical-specific data necessary for exposure and toxicity analyses are shown in Appendix D, and include:

MW - molecular weight (g/mol)

K_H - Henry's Law constant (atm-m^3/mol)

K_{oc} - organic carbon partitioning constant (L/kg)
K_{ow} - octanol/water partitioning constant (unitless) - used only if K_{oc} is unavailable
BSAF - biota sediment accumulation factor (unitless)
ABS_{GI} - fraction of administered dose absorbed in primary study (unitless)
ABS_{Sed} - oral absorption adjustment - relative bioavailability (unitless)
ABS_{Derm} - fraction dermally absorbed from sediment (unitless)
K_p - permeability coefficient (dermal from water) (cm/hr)
FA - fraction dermally absorbed from water (unitless)
RfD - reference dose (mg/(kg·d))
RfC - reference concentration (mg/m^3)
SF - cancer slope factor – $(mg/(kg \cdot d))^{-1}$

5.1 Appropriate detection limits

MDH has developed a list of recommended detection limits for the contaminants of concern in sediments and in fish tissue. This table has been included in Appendix E. Detection limits are calculated directly from the Sediment Screening Values or reference concentrations (sediment detection limits), and fish ingestion assumptions (fish tissue detection limits). Detection limits used to characterize contaminant extent and magnitude or to address ecological concerns are low enough to address human health concerns for many contaminants of concern. A number of these chemicals are identified in Appendix E, Tables E-1a and E-1b.

5.2 Partitioning of chemicals

Chemicals in the environment are not confined to a single chemical phase, or a single medium, but can partition between various phases or media. In this way, chemicals that are in sediment can migrate into water, and even into air. The physical characteristics of each chemical will determine its fate in the environment: whether it tends to move into sediment, water, or air. Volatile chemicals tend to move to air, soluble chemicals into water, and hydrophobic chemicals into sediments or air.

Chemicals in the environment transfer (reversibly) from one phase into another according to thermodynamic principles. Thermodynamics describe equilibrium states, but in the environment the movement of chemicals from one phase to another can often be better described by looking at the kinetics of the transfers. If contaminants move from the sediments into water and then into air, they become diluted and dispersed in those media. This can decrease the potential exposure concentrations in water and air to levels that are of little concern for human health. However, if the water is relatively stagnant or shallow, the air is still, and the source is large enough, exposures to some contaminants could be significant. Describing the time-dependent (kinetic) concentrations in various media can be complicated and often depends on conditions that cannot easily be generalized (e.g. dilution from water mixing, or dilution in air from wind). Therefore, equilibrium partitioning is used to calculate relations between chemical concentrations in water and sediment, and air and water in this report.

The values in this report are suitable as screening values for use in the initial phases of site investigation. Information contained in this document is intended to help identify the types of exposure that may cause the greatest concern for different chemicals found in sediments. Values

developed from calculated water or air concentrations may be very conservative. But, comparing measured sediment concentrations to calculated sediment screening values provides important information about need for further analyses, exposures that may be of little importance, and the potential impact of a remedial action.

The relationship between chemical properties and equilibrium partitioning is particularly well understood for partitioning of non-polar organic compounds between sediment and surface water. All organic chemicals of concern in this report are non-polar organic compounds. For these chemicals:

C_{Sed} { mg/kg } = C_{SW} * K_{oc} * f_{oc} *Equation 2.*

Where:
C_{Sed} = concentration in sediment (mg/kg dry wt.)
C_{SW} = concentration in surface water (mg/L)
K_{oc} = organic carbon partitioning constant (L/kg; chemical specific)
f_{oc} = fraction organic carbon in sediment (unitless; site specific)

This equation describes the equilibrium that is achieved over time when sediment and water are mixed (Lyman 1995). For the purpose of developing screening concentrations, it is assumed that equilibrium is reached between the concentration of chemicals in water and sediment.

There is considerable information showing that non-polar organic compounds have different affinities for organic carbon of different origins. PAH partitioning into water has been shown to be decreased in areas with significant tar or pyrogenic materials (Maruya et al. 1996). In addition, research by Gustafsson et al. (1997) and others has shown that soot or black carbon in sediments does not allow PAHs to desorb as readily as natural or other organic carbon. Thus, PAH partitioning into water may be decreased if sediment is highly contaminated. As a result, chemical-specific K_{oc} and other measures that are affected by partitioning and bioavailability (e.g. BSAF) may be different from one site to another. Default values that characterize partitioning (used in Equation 2) are reasonable, peer-reviewed numbers that may be adjusted when reliable site-specific data are available.

K_{oc}s are not available for perylene and octachlorostyrene. For these chemicals K_{oc}s were calculated using the following empirically derived relationship (Di Toro et al. 1991):

log K_{oc} { L/kg } = 0.00028 + 0.983 (log K_{ow}) *Equation 3.*

Equilibrium partitioning between water and air can also be easily calculated (Schwarzenbach et al. 1993):

C_{SW} { mg/L } = C_A * (R * T) / (K_H * $CF_{L/m3}$ * $CF_{L/m3}$) *Equation 4.*

Where:
C_{SW} = concentration in surface water (mg/L)
C_A = concentration in air (mg/m^3)
R = ideal gas constant (L/(mol·ºK))

T = temperature (ºK) - 293.13ºK default
K_H = Henry's Law Constant (atm·m^3/mol; chemical specific)
$CF_{L/m3}$ = conversion factor (1,000 L/m^3)

Because of difficulties calculating sediment-surface water partitioning of metals, sediment screening values for metals do not include exposure by pathways requiring partitioning calculations (ingestion and dermal contact with surface water, and inhalation).

5.3 Contaminant accumulation in fish tissue

Aquatic organisms exposed to low concentrations of non-polar organic compounds in water will absorb some of these compounds. This partitioning is defined as bioconcentration. A bioconcentration factor (BCF; $mg/kg_{fish\ lipid}$ / mg/L_{sw} = $L/kg_{fish\ lipid}$) describes the non-active partitioning between water and aquatic organisms.

Because chemicals are also ingested and absorbed in the gut with food, accumulation of persistent chemicals in fish tissue is not solely the result of bioconcentration (from water). The accumulation of compounds by any and all routes-of-exposure, including ingestion, is called bioaccumulation and can be described by a bioaccumulation factor (BAF). Because the source of persistent chemical contaminants is generally sediments, it is often useful to describe the ratio of chemical in biota to the concentration of contaminant in sediments. Therefore, chemical-specific biota-sediment accumulation factors (BSAFs) are used to describe the accumulation of persistent organic chemicals in fish.

Sediment Screening Values are calculated using BSAFs and (for volatile organic compounds) BCFs to determine the accumulation potential of contaminants in fish tissue. BSAFs are calculated from field data, typically from edible fish tissue concentrations and sediments within their foraging range. There is likely a limit to the amount of contaminant that can accumulate in the fish tissue. This is either because fish (or their prey) will avoid the most contaminated areas, or because the health of the fish is adversely affected by absorption of contaminants. Useful indicators, or measures, of either contaminant avoidance or fish tissue concentrations when health impacts occur are not available. Therefore, route-specific Sediment Screening Values for the fish consumption route of exposure are calculated assuming that fish tissue will reach the concentrations projected by applying a BSAF to the contaminants concentration in sediment. BSAFs used in the SSV calculations are central tendency values of the range of published values. BSAFs and BCFs used in this document are listed in Table D-1a, Appendix D.

If fish consumption is not believed to be a significant route of exposure for a contaminant of concern, this route of exposure should be eliminated for the specific chemical. Further discussion and an illustrative example are provided in Section 7.3.

Because non-polar organics adsorb to organic carbon in sediments and are generally associated with lipids in aquatic organisms, BSAFs for non-polar organic compounds are normalized for organic carbon in sediment and lipid in fish ($mg/kg_{fish\ lipid}$ / $mg/kg_{sed\ oc}$). Lipid concentration in fish fillets is assumed to be 1.5% and the organic carbon concentration in sediments in the St. Louis River Estuary is assumed to be 2%. Further discussion of sediment total organic carbon

(TOC) is in Appendix F. Sometimes individual studies use the organic matter fraction in place of the organic carbon fraction. Organic carbon is typically ½ of the organic matter (Schwarzenbach et al. 1993).

BSAFs may be calculated for different species in a single waterbody, but in the literature they are typically not normalized for fish size. Consequently, mercury is the only chemical for which an index fish species and length was used to calculate a BSAF (described in Section 5.3.3 below).

5.3.1 BSAFs for non-polar organic chemicals

If intake is large relative to metabolism or excretion, a contaminant can accumulate in the food chain, so that higher trophic-level organisms will have higher concentrations of the contaminant than organisms further down the food chain. Older (and generally larger members of a species) will also have higher concentrations than younger members of a species. Many individual PCB, PCDD and PCDF congeners have long half-lives in aquatic organisms including fish.

PAHs are also readily accumulated by most aquatic organisms, but predicting PAH concentrations in fish is more problematic. Fish appear to metabolize PAHs once metabolizing enzymes have been induced (see Varanasi et al. 1989 for review). Metabolism of PAHs in fish results in (brief) exposure of the fish to toxic intermediate compounds which form adducts with DNA and proteins. While this limits human exposure to non-metabolized PAHs in fish tissue, it may also apply some genetic pressure in highly exposed fish subpopulations for less efficient metabolism of PAHs. Ownby et al. (2002) have demonstrated that PAH toxicity is heritably reduced in a wild mummichog population that inhabits a contaminated area of the Chesapeake Bay. This is presumably due to decreased activity of metabolic enzymes, which could in turn result in greater accumulation of PAHs in these fish.

It is expected that different species of fish will react differently to PAHs. Some fish metabolize and excrete some PAHs more rapidly than others. Niimi et al. have determined the half-lives of many PAHs in rainbow trout (Niimi and Palazzo 1986; Niimi and Dookhran 1989). The half-lives reported for acenaphthylene, phenanthrene and phenyl naphthalene were 1, 9 and 25 days, respectively. This wide range in half-lives in rainbow trout suggests that there are large differences in the accumulation of individual PAHs; from no accumulation to likely measurable accumulation. In addition, a review of PAH accumulation in marine organisms by Meador et al. (1995) states that

> A recurring theme in many studies indicates that organisms exposed to PAHs for a short time will completely eliminate their acquired burden when exposed to a clean environment, whereas species chronically exposed to these compounds tend to retain a portion of their acquired burden that is resistant to elimination by metabolism or passive diffusion. This is advantageous for animals exposed to PAHs in acute events (e.g., oil spills) but detrimental to those living in chronically contaminated environments.

Furthermore, when PAHs are analyzed and their toxicities are evaluated, heterocyclic PAHs, substituted PAHs and metabolic products are typically ignored. Therefore, predicting the concentration and toxicity of PAHs in fish tissue is complex.

PAHs have been found in whole fish from the St. Louis River (US FWS 2002). In addition, data from the Netherlands (Van Der Oost et al. 1994) and Massachusetts (ATSDR 1995) have shown PAHs in fish fillets. Currently there are no useable PAH fillet data from the St. Louis River, because detection limits have been too high.

Washington State (WA) Department of Health (DOH) developed BSAFs for PAHs based on a large database of fish tissue (fillet) and sediment concentrations from marine and freshwater environments (Washington State Department of Health 1995; 1996). Individual PAHs were classified according to their log K_{ow}s, and different BSAFs were applied to ranges of log K_{ow}s. Potential exposures to PAHs from fish consumption are calculated in this report using the WA DOH BSAFs. Further discussion of these PAH BSAFs is included in Appendix G.

5.3.2 BCFs for volatile/non-persistent organic chemicals

Benzene and other volatile organic chemicals are not generally persistent and degrade rapidly in the environment. Therefore, they are not likely to accumulate in aquatic organisms. However if there is a significant sediment source of benzene and other volatile organics, dissolved chemicals in interstitial and overlying waters may be significant. Lipid-normalized bioconcentration factors (BCFs) were calculated for volatile organics using the following equation (Veith and Kosian 1983):

$$BCF\ \{\ L/kg_{fish\ lipid}\ \} = (10^{(0.79*\log(Kow)-0.40)})/f_{lipid} \qquad \textit{Equation 5.}$$

Where:

BCF	= bioconcentration factor	($L/kg_{fish\ lipid}$)	
K_{ow}	= octanol water partitioning constant	(unitless)	
f_{lipid}	= fraction lipid in fish (7.6% for fathead minnow - test species)		($kg_{fish\ lipid}/kg_{fish}$)

The BCFs derived from the above equation are presented in Table D1a in Appendix D.

5.3.3 Mercury bioaccumulation

The primary human exposure pathway for mercury in sediments is fish consumption. Fish accumulate mercury through their diet. Mercury, generally, enters the food chain through transformation to methylmercury and uptake from sediment by benthic invertebrates. However, sediment mercury concentrations are not predictive of the amount of mercury found in fish across different waterbodies (Driscoll et al. 1995; Cabana et al. 1994; Wiener et al. 1990; Sorensen et al. 1990). This is usually attributed to effects of variable methylmercury production and differences between food chains in different waterbodies.

Chemical conditions and the local food-web can affect the amount of mercury accumulation in fish. Alkalinity, dissolved organic matter (DOM), and pH in surface waters affect mercury solubility and/or bioaccumulation. In addition, physical and chemical characteristics of sediments including oxygen depletion, sulfate availability, sulfide concentration, and groundwater flow may affect mercury solubility and/or bioaccumulation.

Bioaccumulation of mercury in a food-web often correlates with conditions that increase the methylation of inorganic mercury. Wetlands are a favorable environment for mercury

methylation (Rudd 1995; Saint Louis et al. 1996). Generally, factors that increase methylation include: increasing DOM, decreasing pH, increasing sulfate, decreasing sulfide, and increasing anoxia (Ullrich et al. 2001). In addition, groundwater discharge may increase methylation potential of mercury in sediment, as well as increase local flux into the river.

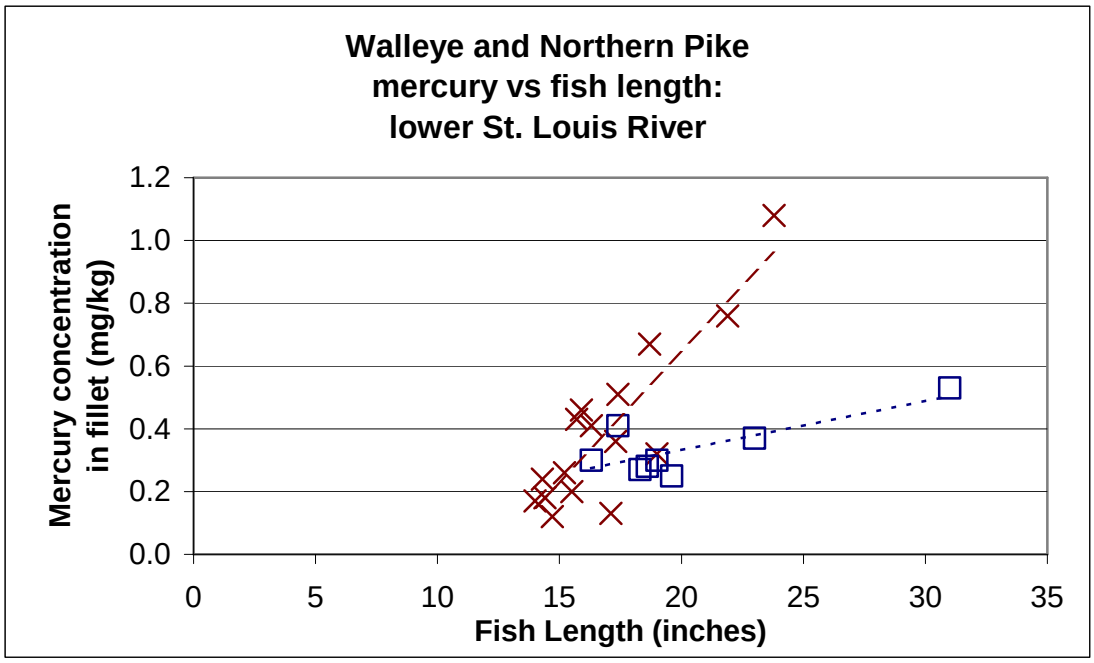
A biota sediment accumulation factor (BSAF; kg_{sed}/kg_{fish}) is the ratio of tissue concentration of a contaminant in an index fish, divided by the contaminant concentration in sediment where the fish lives. Mercury BSAFs are not generally consistent across different waterbodies. However while sediment mercury concentrations do not correlate well with fish concentrations of methylmercury across different waterbodies (among species and fish lengths), in a single waterbody there is a correlation between the mercury concentration in sediments and the methylmercury concentration in fish of similar species and length or age. Therefore, a locally-derived BSAF is used in this report to estimate the methylmercury concentration in an index fish. The EPA Mercury Study Report to Congress contains a review of BSAF derivations for a number of different waterbodies and aquatic species (see Volume VI, Section 2.3.1 of EPA 1997b).

5.3.3.1 Mercury in fish

It is believed that individual walleyes and northern pike range throughout the lower St. Louis River over time. Therefore, individual large sportfish consume smaller fish and other food throughout the lower St. Louis River. As a result of feeding on smaller fish throughout the estuary, their total intake is likely determined by the average concentration in smaller fish in all areas of the estuary.

The Minnesota Department of Natural Resources collected fish throughout the lower St. Louis River from June through September 2000. Data available for these fish include: total mercury in fish fillets (wet weight) and the length of fish. Length and fillet mercury concentration data, for sampled walleye and northern pike, are plotted in Figure 1 along with lines showing the linear regressions of the data for each species.

Figure 1 - Mercury in fish tissue vs fish length



X - Walleye data
□ - Northern pike data

For walleye, the dependence of mercury concentration in fish tissue on the length of the fish can be described by the equation:

Mercury in fillet { mg/kg } = 0.0826 * fish length - 1.0064 (R^2 = 0.7615) *Equation 6.*

For northern pike the dependence can be described by

Mercury in fillet { mg/kg } = 0.0157* fish length + 0.0194 (R^2 = 0.6163) *Equation 7.*

Using these data, a 20-inch walleye is expected to have about 0.65 mg mercury/kg fillet (wet weight) (5-95% confidence limit (CL)); 0.53 - 0.76 mg/kg), and a 30 inch northern pike may have about 0.49 mg methylmercury/kg fillet (wet weight) (5-95% CL; 0.38 - 0.60 mg/kg).

5.3.3.2 Mercury in sediment

In 1995, EPA collected sediment data from the lower St. Louis River for the Regional Environmental Monitoring and Assessment Program (REMAP; EPA 1995). Sample locations were chosen randomly, after eliminating known areas of contamination (see Attachment 1 for sample locations). Samples were analyzed for trace metals (including mercury) and some organic compounds. Some sampling locations were training sites and data from these locations were not used in this analysis. Mercury data from 87 sample locations in Class I & II habitat (shallow water and channel sediments) were used. The sediment mercury concentration are shown in Table 11.

5.3.3.3 Mercury BSAF calculation

BSAFs are calculated as the ratio of methylmercury in fish tissue to a representative sediment mercury concentration. Fish accumulate mercury primarily from their diet. It is expected that fish tissue mercury concentrations will be an integrated function of the mercury concentrations found in smaller, less mobile aquatic organisms. These organisms, in turn, are expected to have mercury concentrations that are a function of the local sediment concentration, local methylation rates, and the organism's trophic status. Highly contaminated areas typically are less biologically productive than non-contaminated areas. Therefore, using a mean mercury sediment concentration in BSAF calculations likely overestimates the contribution of organisms from contaminated areas to the diet of large fish (see Appendix C for further discussion). However, even if there is impairment of the food chain, some contaminated areas may still be biologically productive. Data suggest that a reasonable sediment concentration for calculating a BSAF for the St. Louis River estuary is the geometric mean of REMAP data (0.078 mg/kg mercury). This statistic discounts the highest mercury concentrations in sediments and may best reflect sediment mercury incorporation into the foodchain.

Table 11 shows calculated BSAFs for 20-inch walleye and 30-inch northern pike using different sediment and fish concentration data.

Table 11 - Calculated BSAFs (mercury)

REMAP Sediment Mercury Data statistic (mg/kg) used to calculate BSAF:	**Walleye - 20 in. 0.65 mg/kg mercury (Predicted from regression)**	**Northern Pike - 30 in. 0.49 mg/kg mercury (Predicted from regression)**
Mean (0.150)	4.3	3.3
Mean (5% CL) (0.121)	5.3	4.0
Mean (95% CL) (0.179)	3.6	2.7
Median (0.100)	6.5	4.9
Geometric mean (0.078)	**8.2**	**6.2**
GeoMean (5%CL) (0.063)	10.3	7.8
GeoMean (95% CL) (0.098)	6.6	5.0
	0.53 mg/kg mercury (10% C.L. of regression*)	**0.38 mg/kg mercury (10% C.L. of regression*)**
Mean (0.150)	3.5	2.5
Median (0.100)	5.3	3.8
Geometric mean (0.078)	6.7	4.8
	0.76 mg/kg mercury (90% C.L. of regression*)	**0.60 mg/kg mercury (90% C.L. of regression*)**
Mean (0.150)	5.1	4.0
Median (0.100)	7.7	6.0
Geometric mean (0.078)	9.7	7.6

* SAS 8.1

The geometric mean of the mercury REMAP data and the estimated mean methylmercury concentration for a 20-inch walleye were used to calculate the BSAF (8.2) used for screening in

this report. There are no published BSAFs for walleye. However, there are published northern pike BSAFs. Assuming the dry weight BSAF for northern pike is about 4 times the wet weight BSAF, the dry weight BSAF for 30-inch northern pike (fillets) in the St. Louis River is about 33 (unitless). This is within the range of northern pike (fillet) BSAFs reported in the EPA Mercury Study Report to Congress (EPA 1997b): 10.1 - 45.7 (unitless; dry fish tissue weight).

6 Risk Calculation Defaults

6.1 Health-based toxicity values used to calculate screening values

Published health-based toxicity values are used as safe levels of exposure for the general public in calculations of these human health-based sediment screening values (See Appendix D; Table D2). EPA Integrated Risk Information System (IRIS) (EPA 2003c) Reference Doses (RfDs) and Reference Concentrations (RfCs) are used to evaluate most chemicals of concern in sediments. MDH Health Risk Values (HRVs) (MDH 2002b); California Office of Environmental Health Hazard Assessment (CA OEHHA 2002; 2003); EPA Provisional Peer-Reviewed Toxicity Values (EPA 2002c), Health Effects Assessment Summary Tables(EPA 1997c) and Soil Screening Levels (EPA 1996); World Health Organization Provisional Tolerable Monthly Intake (dioxin, non-cancer) (FAO/WHO 2001), and; New York State Health Department acceptable daily intake rate (octachlorostyrene) (New York State DEC 1997) are used to evaluate the other chemicals of concern identified in this report.

It is reasonably anticipated that lead sediment criterion based on the MPCA Soil Reference Values (MPCA 1999) of 400 mg/kg will result in blood lead concentrations below 10 micrograms per deciliter (µg/dL) (MDH 2002a). However, note that a 10 µg/dL limit was determined by policy, as there is no known threshold for lead toxicity. The use of an ecological standard, such as the Ontario Ministry of the Environment lowest effects level (LEL) (1993) of 31 mg/kg, is more restrictive.

The values developed in this SSV Report are protective of sensitive sub-populations. However, they may not be protective of individuals, such as subsistence fishers or pica (soil eating) children, who may have higher intake of fish tissue or sediment.

6.2 Risk and hazard quotients

For screening, an acceptable hazard quotient for non-carcinogens is generally presumed to be 20% (i.e. it is assumed that an individual may have additional exposures to the same contaminant from other sources that add up to 80% of the EPA Reference Dose (RfD) or other health criteria). The hazard quotient for exposure to mercury, as methylmercury, as a result of fish consumption is 73% as suggested in the EPA Water Quality Criterion document on methylmercury (EPA 2001a).

Note that a hazard index should be calculated for simultaneous exposure to chemicals with similar endpoints.

MDH's health-based values for carcinogens are equal to a calculated incremental cancer risk of 1 case in 100,000 individuals exposed to site-related contaminants over their lifetime. Therefore, if exposure to all cancer-causing chemicals cause a calculated additional incremental risk of less

than or equal to 1 in 100,000, then contaminants are not considered to be a public health concern. Again, screening values do not directly quantify the total potential risk. These sediment screening values are to be used to identify individual chemicals (or groups of chemicals: i.e. B[a]P-PEQs, TCDD-TEQs, and tPCBs) needing further consideration, and likely overestimate actual risk.

No acute screening values are developed in this SSV Report. While the duration of individual exposure events are long enough to elicit an acute response, the availability of protective health criteria are limited.

6.3 Modifiers

Oral reference doses are ingested doses. However, toxicity is generally associated with the internal dose of a chemical, which is the ingested dose times the fraction of the dose that is absorbed (fraction absorbed = ABS_{GI}). EPA recommends that "in absence of a strong argument for making this adjustment ..., assume that the relative absorption efficiency between food or soil and water is 1.0 (EPA 1989)." Therefore, the availabilities of all chemicals from ingested water, and from fish consumption, are assumed to be the same as the availabilities in the primary toxicology study (ABS_{Sed}=1: Appendix Table D-1b). No adjustments were made to the reference doses for these routes-of-exposure.

However, because dermal exposure (direct sediment and water) calculations estimate an internal chemical dose, they need to be compared with modified reference doses that describe generally safe internal doses. Therefore, oral reference doses for chemicals of concern are adjusted to internal doses ($RfD*ABS_{GI}$) when evaluating dermal exposures.

Most inhalation criteria used in this report are published standards that have incorporated appropriate bioavailability adjustments. For screening purposes, route-to-route extrapolation of oral standards were included for some chemicals for which no reference concentrations are available (TCDD-TEQ cancer endpoint only, carbazole, dibenzofuran, hexachlorobenzene, octachlorostyrene, tPCBs), as noted in Appendix Table D-2. No toxicity adjustments were made to these provisional values based on different availability by the inhalation route (i.e. inhaled absorption assumed to be equal to ABS_{GI}).

7 Sediment Screening Values

Sediment screening values, calculated using the equations described in Appendix H (route-specific chronic noncancer endpoints), Appendix I (route-specific cancer endpoint) and Appendix J (all routes screening calculations), are listed in Table 12. In addition, the relative contributions of different routes-of-exposure are shown.

Table 12 - Sediment Screening Values

	Cas No.	Sediment Screening Value (mg/kg)	Route-of-exposure contribution (%)					
			Sediment Ingestion	Surfacewater Ingestion	Dermal Sediment	Dermal Surfacewater	Inhalation	Fish Consumption
Metals - Inorganics								
Arsenic	7440-38-2	48	65%	Not Evaluated	35%	Not Evaluated	Not Evaluated	Not Evaluated
Cancer endpoint		28	51%		49%			
Cadmium	7440-43-9	140	58%		42%			
Chromium III	16065-83-1	370000	100%		Not Evaluated			
Chromium VI	18540-29-9	730	100%					
Copper	7440-50-8	9000	100%					
Cyanide	57-12-5	4900	100%					
Lead	7439-92-1	400 *	*Not Evaluated*					
Mercury ** (inorganic in sediment; methylmercury in fish)	7439-97-6	0.021	0%					
Methyl Mercury	22967-92-6							100%
Nickel	various	4900	100%					Not Evaluated
Zinc	7440-66-6	73000	100%					
Volatile organic compounds (VOCs)								
Benzene	71-43-2	0.0049	0%	1%	Not Evaluated	1%	97%	1%
Cancer endpoint		0.0032	0%	0%		1%	98%	1%
Ethyl benzene	100-41-4	0.34	0%	1%		3%	91%	5%
Styrene	100-42-5	4.1	0%	1%		3%	91%	5%
Toluene	108-88-3	0.12	0%	1%		2%	94%	3%
Xylenes (mixed)	1330-20-7	0.078	0%	0%		0%	99%	0%
Polycyclic Aromatic Hydrocarbons (PAHs)								
Acenaphthene	83-32-9	22	0%	4%	0%	41%	23%	32%
Acenaphthylene (toxicity surrogate - acenaphthene)	208-96-8	20	0%	5%	0%	41%	23%	30%
Anthracene	120-12-7	690	1%	5%	2%	61%	12%	18%
Benzo(a)pyrene	50-32-8	*Not Evaluated*	*Not Evaluated*					
B[a]P-PEQs *Cancer endpoint*		0.071	1%	0%	3%	13%	0%	83%
Fluoranthene	206-44-0	82	1%	2%	2%	59%	1%	35%
Fluorene	86-73-7	49	0%	8%	1%	57%	18%	16%
Methylnaphthalene (toxicity surrogate - naphthalene)	1321-94-4	0.48	0%	0%	0%	1%	97%	2%
Naphthalene	91-20-3	0.11	0%	0%	0%	1%	99%	0%
Perylene (toxicity surrogate - pyrene)	198-55-0	54	1%	0%	2%	67%	0%	31%
Phenanthrene (toxicity surrogate - anthracene)	85-01-8	730	1%	4%	2%	56%	4%	33%
Pyrene	129-00-0	78	1%	2%	2%	50%	1%	44%
Dibenzo-p-dioxins/dibenzofurans								
2,3,7,8-TCDD (or TCDD-TEQs) **	1746-01-6	1.4E-06	0%	0%	0%	0%	0%	99%
TCDD-TEQs ** *Cancer endpoint*		4.7E-08	0%	0%	0%	0%	0%	100%
Other Organics								
Carbazole	86-74-8	*Not Evaluated*	*Not Evaluated*					
Cancer endpoint		2.8	0%	3%	0%	10%	0%	87%
Dibenzofuran (unsubstituted)	132-64-9	1.8	0%	3%	0%	23%	3%	71%
Hexachlorobenzene	118-74-1	0.88	0%	1%	1%	18%	62%	18%
Cancer endpoint		0.15	0%	0%	1%	19%	43%	36%
Octachlorostyrene	29082-74-4	0.019	0%	0%	0%	0%	0%	99%
Polychlorinated Biphenyls (tPCBs) **	1336-36-3	0.0046	0%	0%	0%	2%	1%	97%

* See Section 4.1.3

** (shaded chemicals) Sediment Screening Value may approach or be less than ambient or background concentration. See sections on individual chemicals (in Section 4, above), Section 7.1.1 (below) and Appendix C for information on ambient and background concentrations.

7.1 Discussion

If it was possible to calculate a protective sediment concentration limit for a chemical of concern by a potential route of exposure, a sediment screening value included that route. When the impact of a particular route of exposure is small, the contribution of that route of exposure to the overall sediment screening value is minimal, and the specific route of exposure can be eliminated from further consideration when evaluating the chemical at a site.

Note that the only chemicals for which sediment ingestion is a significant route of exposure are metals. This is, in part, because arsenic and cadmium are the only metals where other routes of exposure were considered. There are very little data available with which to quantitatively evaluate potential exposures to metals by other routes of exposure.

Note, from Table 12, that the largest exposure route for all VOCs is through inhalation as chemicals vaporize from surface water. Therefore if VOCs are present at a site above screening values concentrations, it may be necessary to analyze surface water concentrations during wading and swimming to determine if the model is too conservative for that specific site (i.e. if the partitioning model overestimates the water concentration).

Further note in Table 12, that even with an over-estimated fish tissue concentration of volatile organics as a result of using a (BCF) partitioning model, the fish consumption route of exposure for these chemicals is limited.

7.1.1 SSV's below ambient or background concentrations

The Sediment Screening Values for mercury, TCDD-TEQs, tPCBs, benzo[a]pyrene and naphthalene may be less than, or approaching background and/or ambient sediment concentrations. (Background sediment concentrations are calculated from samples that are collected in areas not impacted by local or regional point sources; in contrast, ambient concentrations may reflect local or regional conditions including nearby point or area contaminant sources.) Unfortunately, there are no data from the St. Louis River Estuary that can be used to accurately determine regional or waterbody background (or ambient) sediment concentrations for these contaminants. Background concentrations may be used as screening levels when background concentrations exceed the SSVs for individual chemicals.

7.1.1.1 Mercury

Background mercury concentrations in the upper St. Louis (0.02 mg/kg) are similar to the mercury SSV (Glass et al. 1999). Table 13 shows the range, geometric mean, arithmetic mean and median REMAP mercury concentrations for the lower St. Louis River. Note that 82% of the REMAP sample concentrations exceeded the calculated sediment screening value (SSV). REMAP data are also used in an analysis in Appendix C that suggests that background mercury concentrations in the St. Louis River Estuary are about 0.02 mg/kg, but that ambient concentrations in specific areas of the estuary may be above background due to local area or point sources. Therefore, it is reasonable to apply the calculated mercury sediment screening value (0.021 mg/kg) to evaluate sediments in identifiable discharge areas in the St. Louis River Estuary. An action level for mercury cleanup will need to consider ambient concentrations in the

river. Appendix C contains further discussion of the relationship between PAH and mercury contamination in the estuary.

7.1.1.2 PAHs (B[a]P-PEQs, Naphthalene)

Table 13 compares Sediment Screening Values (SSVs) for PAHs to available REMAP Class 1 and 2 data from the St. Louis River Estuary. These data show that concentrations at some REMAP sample locations in the St. Louis River Estuary exceeded naphthalene and B[a]P-PEQ Sediment Screening Values. REMAP data were intended to be representative of ambient data throughout the estuary. But samples were taken at some locations that were impacted by industrial and urban pollution. Background concentrations of chemicals cannot be determined from this database. However, the REMAP data do suggest that if PAH sampling data at a site exceeds Sediment Screening Values, local or site-related contamination is likely the source.

Note that in Table 13, when all Class 1 & 2 REMAP PAH data are compared with the SSVs, only naphthalene and B[a]P-PEQs (7 cPAHs only) exceeded the Sediment Screening Values. Naphthalene and B[a]P-PEQs exceeded SSVs at 22 and 58% of the sample locations, respectively. The REMAP median concentration for naphthalene is well below the SSVs. Therefore, it is likely that naphthalene concentrations in relatively unpolluted areas of the St. Louis River Estuary are below SSVs.

Background B[a]P-PEQs have not been established for Northern Minnesota or the St. Louis River Estuary. Ambient B[a]P-PEQ concentrations in some areas of the St. Louis River Estuary are greater than the B[a]P-PEQ SSV. Consequently, B[a]P-PEQ background concentrations may need to be evaluated and local or regional sources may need to be determined if B[a]P-PEQ screening data exceed the SSV at the US Steel site.

Table 13: Sediment Screening Values and REMAP data for PAHs and mercury

Chemical	Sediment Screening Value (mg/kg)	REMAP (Class 1,2 data)				% REMAP (Class 1,2) Above SSV
		Range (mg/kg)	Geometric mean (mg/kg)	Arithmetic mean (mg/kg)	Median (mg/kg)	
Acenaphthene	**22**	**0.000050 - 0.65**	0.0055	0.036	0.0051	**0%**
Acenaphthylene	**20**	**0.00050 - 0.47**	0.0086	0.041	0.0075	**0%**
Anthracene	**690**	**0.0014 - 2.6**	0.043	0.25	0.028	**0%**
Benzo(a)pyrene (or B[a]P-TEQs)	**0.071**	**0.0059 - 6.5**	0.17	0.83	0.15	**58%**
Fluoranthene	**82**	**0.0062 - 7.5**	0.21	0.99	0.14	**0%**
Fluorene	**49**	**0.00050 - 1.5**	0.021	0.12	0.018	**0%**
Methylnaphthalene	**0.48**					
Naphthalene	**0.11**	**0.00050 - 10**	0.033	0.51	0.036	**22%**
Perylene	**54**	**0.0035 - 1.6**	0.18	0.39	0.27	**0%**
Phenanthrene	**730**	**0.0037 - 6.1**	0.13	0.63	0.11	**0%**
Pyrene	**78**	**0.0068 - 5.4**	0.18	0.82	0.13	**0%**
Mercury	**0.021**	**0.0049 - 0.70**	0.078	0.15	0.100	**82%**

7.1.1.3 TCDD-TEQ

The TCDD-TEQ Sediment Screening Value (0.047 ng/kg) is much lower than national background concentrations (background concentrations from 11 lakes study; Cleverly et al. 1996). As a result, TCDD-TEQ background concentrations may be used to screen sediments for contamination. Data on the lower St. Louis River are not sufficient to determine a local background concentration for TCDD-TEQs. However, given available national data it is reasonable to assume that the background concentration is likely between 1 and 12 ng/kg (see discussion in Section 4.2.1.1).

7.1.1.4 tPCB

REMAP samples were not analyzed for PCBs, and tPCB background concentrations in the St. Louis River Estuary cannot be determined from available data. However, 1997 National Sediment Quality Survey data shows that tPCB Sediment Screening Value exceeds sediment concentrations at about 23% of the nationwide monitoring locations (EPA 1997d). Therefore background sampling in the region is needed to determine screening levels for PCBs in the St. Louis River.

7.2 Comparison of SSVs with SQTs

Human health-based Sediment Quality Targets (SQTs) from the MPCA are listed in Attachment 6 (EPA 2000a). SQTs were calculated by the New York State Department of Environmental Conservation as sediment criteria for human health (New York State DEC 1999). The SQTs were calculated directly from New York State water quality criteria (WQC) and partitioning (log K_{ow}). In contrast, the SSVs are based on reasonable assumptions about exposures to sediments and water, as well as current available toxicity criteria.

The SSVs, as noted in Section 5.3 and Appendix F, are based on an assumed organic carbon fraction of 2%. In Table 14, SQTs were adjusted to an assumed sediment organic carbon fraction of 2% ($SQT_{2\%OC}$). Significant differences between the 2 values are expected, because they are calculated using different assumptions. The SQTs are intended to account for bioaccumulation of chemicals from surface water. SSVs are calculated using BSAFs as a measure of bioaccumulation and, in addition, other routes of exposure are considered. The difference between SSVs and 2% organic carbon SQTs for PCBs and TCDD-TEQs are considerable; the difference is smaller for Benzo[a]pyrene-PEQs.

Table 14: Comparison of SSVs with SQTs

		SSV	$SQT_{2\%OC}$	SQT
		mg/kg	mg/kg	mg/kg_{OC}
Benzo(a)pyrene *2,3,7,8-TCDD TEQs (w/o cPCBs)* *Total PCBs*	*(calculated by NY DEC from NY WQC and K_{ow})*		*0.026* *0.0002* *0.000016*	*1.3* *0.01* *0.0008*
Benzo(a)pyrene PEQs 2,3,7,8-TCDD TEQs Total PCBs	(calculated in this document)	0.07 0.00001 * 0.0046		

* Recommended SSV to be applied to sites. Calculated SSV is well below background sediment concentration. Therefore, a reasonable background concentration is given instead of the calculated SSV.

7.3 Modifying Sediment Screening Values

If one or more potential routes-of-exposure are eliminated at a site, or if a route-of-exposure is considered unrealistic for a specific chemical, screening values can be recalculated without individual route-specific values using *Equations A-45 and A-46* (chronic and cancer endpoints, respectively) from Appendix J.

For example, it is likely that fish will avoid areas highly contaminated with PAHs. However, bioaccumulation of contaminants in this model assumes that there is no limit to the amount of chemical that can accumulate in fish tissue. Therefore in the extreme, the model may anticipate a fish tissue concentration that would kill a fish. For example: the Sediment Screening Value model anticipates 32% of exposure to acenaphthene in sediments occurs through the ingestion of contaminated fish tissue. If acenaphthene is present in sediments at the Sediment Screening Value, the model predicts an acenaphthene concentration in fish fillets of about 70 mg/kg. It is unlikely that fish could survive with this concentration of acenaphthene in their muscle tissue.

If reasonable fish tissue concentrations are exceeded (i.e. if there is an indication that the fish tissue concentration is toxic to fish), the sediment screening value for the chemical of interest should be recalculated without the fish consumption pathway. It is likely that the fish consumption pathway can be eliminated for most non-carcinogenic PAHs. However, without evidence to the contrary, the fish consumption pathway for cPAHs may still be important.

If the fish consumption route of exposure is eliminated (by removing $1/SSV_{fish}$ from *Appendix J -Equation A-45* and $1/SSV_{fish\text{-}c}$ from *Appendix J - Equation A-46*), the resulting site-specific screening values for PAHs are shown in Table 15. Similarly, the inhalation exposure pathway or any other potential route of exposure can be eliminated from the SSVs for application at a site.

Table 15 - PAH Sediment Screening Values - without fish consumption

	Cas No.	Partial Sediment Screening Value (No Fish Consumption)	Route-of-exposure contribution				
			Sediment Ingestion	Surfacewater Ingestion	Dermal Sediment	Dermal Surfacewater	Inhalation
Polycyclic Aromatic Hydrocarbons (PAHs)		**mg/kg**	**%**	**%**	**%**	**%**	**%**
Acenaphthene	83-32-9	**32**	0%	6%	1%	60%	34%
Acenaphthylene (toxicity surrogate - acenaphthene)	208-96-8	**29.3**	0%	8%	0%	59%	33%
Anthracene	120-12-7	**841**	1%	6%	3%	75%	15%
Benzo(a)pyrene PEQs *Cancer endpoint*	50-32-8	**0.427**	4%	0%	15%	80%	0%
Fluoranthene	206-44-0	**126**	1%	4%	3%	90%	2%
Fluorene	86-73-7	**58**	1%	9%	1%	68%	21%
Methylnaphthalene (toxicity surrogate - naphthalene)	1321-94-4	**0.489**	0%	0%	0%	1%	99%
Naphthalene	91-20-3	**0.106**	0%	0%	0%	1%	99%
Perylene (toxicity surrogate - pyrene)	198-55-0	**77.7**	1%	0%	2%	96%	0%
Phenanthrene (toxicity surrogate - anthracene)	85-01-8	**1080**	1%	6%	3%	84%	5%
Pyrene	129-00-0	**139**	2%	4%	4%	89%	1%

8 Children and other Special Populations

Exposure parameters developed for this report suggest that a child receptor may be exposed to contaminated sediments more than any other age group. As a result, the child receptor is used to calculate the chronic noncancer screening values. In addition, health-based toxicity values used in developing these SSVs are intended to be conservative and protective of sensitive individuals, including children. However, it is possible that these SSVs are not protective of groups that may have higher than projected exposures to sediments. These groups include pica children who ingest contaminated sediments, and subsistence fishers who consume fish that have accumulated contaminants. Protective criteria for these groups can be developed for application at specific sites.

As research continues on risk assessment and risk analysis, algorithms used in quantitative risk assessments will change. Recent research suggests that averaging exposures over a lifetime may underestimate the cancer risk from short-term exposures early in life (see Halmes et al. 2000; Ginsberg 2003 for reviews). Ginsberg (2003) proposes using an exposure rate (over a limited number of years) instead of a lifetime (70 year) average exposure for calculating exposure risk to children.

In addition, it has been demonstrated in laboratory studies that young animals may be more sensitive to carcinogens than adolescent and adult animals (EPA 2003a). There are not sufficient data to suggest the magnitude of the difference between adult and childhood sensitivity. However, given the use of conservative exposure assumptions, screening values for carcinogens are likely still protective. If site-specific or cleanup criteria are developed, above-noted uncertainties should be addressed.

9 Conclusions

9.1 General application of screening values

Only exposures to water-covered sediments were evaluated in this document. Exposures to upland, beach and intertidal sediments are likely to be different and should be evaluated separately.

Chemical concentrations in water-covered sediments at or below the human health-based Sediment Screening Values (SSVs) developed in this report are considered safe for the general public. Alternatively however, sediment concentrations greater than the screening values should not be considered unsafe, because the values were developed from conservative measures of exposure, bioavailability and toxicity. Local exceedance of these values suggests that site-specific conditions need to be evaluated prior to concluding that there is a reasonable chance that sediments may impact public health. In addition while this document evaluates a suite of persistent chemicals, it does not evaluate all chemicals that can be found in sediments and can impact public health.

The values developed in this report are appropriate for use when screening sediments throughout the lower St. Louis River. While exposures will vary from site to site, this document uses reasonable maximal exposures (RMEs) to describe exposures that may occur in the lower St. Louis River. RMEs are used, along with protective chemical-

specific health criteria, to calculate sediment concentrations of many different specific chemicals that should not impact the health of individuals who regularly use the St. Louis River for recreational activities. For other site-specific evaluations, potential exposures and sediment characteristics for specific sites should be compared with default values used to develop these Sediment Screening Values. Parameters that may affect the transport and availability of contaminants at specific sites may include: organic carbon, particle size, redox potential, mineral content, clay content and porosity.

In addition to their intended use in the lower St. Louis River, the screening values may be protective concentrations for contaminated sediments in other waterbodies.

9.2 Application to the US Steel St. Louis River Site

Criteria similar to these SSVs were given to US Steel in December 2002. The SSVs are intended to supercede those values. This document is intended to clarify the derivation of SSVs for US Steel and other interested parties.

US Steel is in the process of executing a workplan that is intended to characterize sediments adjacent to the US Steel Superfund site.

10 Recommendations

- Sediment Screening Values should not be used to screen or evaluate upland, intertidal or beach sediments.
- Analytical methods should be used that can achieve detection limits for chemicals of concern in sediments and fish tissue similar to detection limits in Tables E-1a and E-1b.
- If chemical concentrations in sediments adjacent to the US Steel Site exceed the Sediment Screening Values (or background levels for TCDD-TEQs), further evaluation should be undertaken to determine whether chemicals in the sediments may impact public health.
    - If chemical concentrations in sediments are below the values developed in this document, the screened chemicals in sediments will not adversely impact the health of the public.
    - Further evaluation may be needed to determine whether or not the health of special populations, such as subsistence fishers, are protected.
- Default RMEs should be reviewed prior to using these values to evaluate sediments in other waterbodies.

11 Public Health Action Plan

MDH will use the sediment screening values developed in this document to evaluate chemical data from the US Steel site and other sites in the lower St. Louis River with suspected sediment contamination. Site-specific protective recommendations may be developed in the future to address site-specific conditions and potential exposures. As data become available MDH will use the SSVs to evaluate potential human health concerns related to sediments at this site.

This report was prepared by:

Carl Herbrandson, Ph. D.
Toxicologist
Site Assessment and Consultation Unit
Environmental Surveillance and Consultation Section
Minnesota Department of Health

Helen Goeden, Ph. D.
Toxicologist
Health Risk Assessment Unit
Environmental Surveillance and Consultation Section
Minnesota Department of Health

Rita Messing, Ph. D.
Supervisor
Site Assessment and Consultation Unit
Environmental Surveillance and Consultation Section
Minnesota Department of Health

Mark Johnson, Ph. D.
Toxicologist
Region V
Agency for Toxic Substances and Disease Registry

References

Agency for Toxic Substances and Disease Registry (1995). Hocomonco Pond, Westborough, MA. Massachusetts Department of Public Health, Bureau of Environmental Health Assessment, Atlanta, GA. Public Health Assessment, January 10, 1995.

Bailey, R.A., H.M. Clarke, J.P. Ferris, S. Krause and R.L. Strong (1978). The Environmental Chemistry of Some Important Elements. Chemistry of the Environment. London, Academic Press: 361-407.

Becker, D.S. and G.N. Bigham (1995). Distribution of mercury in the aquatic food web of Onondaga Lake, New York. Water Air and Soil Pollution 80(1-4): 563-571.

Bloom, N.S. (1992). On the chemical form of mercury in edible fish and marine invertebrate tissue. Can J Fish Aquat Sci 49(5): 1010-1017.

Boerngen, J.G. and H.T. Shacklette (1981). Chemical analysis of soils and other surficial materials of the conterminous United States. U.S. Geological Survey Open-File Report 81-197: 143.

California Office of Environmental Health Hazard Assessment, (2003). Chronic Reference Levels. California Office of Environmental Health Hazard Assessment, Last updated 2/2005, http://www.oehha.org/air/chronic_rels/pdf/allchrels.pdf

California Office of Environmental Health Hazard Assessment, (2002). Cancer Potency Values. California Office of Environmental Health Hazard Assessment, Last updated 11/2002, http://www.oehha.org/air/hot_spots/pdf/TSDNov2002.pdf

Cabana, G., A. Tremblay, J. Kalff and J.B. Rasmussen (1994). Pelagic food chain structure on Ontario Lakes: A determinant of mercury levels in lake trout (Salvelinus namaycush). Canadian Journal Of Fisheries And Aquatic Sciences 51(2): 381-389.

Chou, C.L., L.A. Paon, J.D. Moffatt and B. Zwicker (2000). Copper contamination and cadmium, silver, and zinc concentrations in the digestive glands of American lobster (Homarus americanus) from the Inner Bay of Fundy, Atlantic Canada. Bull Environ Contam Toxicol 65(4): 470-7.

Cleverly, D. (2004). 11 Lakes Sediment Study. Washington, D.C. personal communication to Carl Herbrandson, February 24, 2004.

Cleverly, D., M. Monetti, L. Phillips, P. Cramer, M. Heit, S. McCarthy, K. O'Rourke, J. Stanley and D. Winters (1996). A time-trends study of the occurrences and levels of CDDs, CDFs, and dioxin-like PCBs in sediment cores from 11 geographically distributed lakes in the United States. Organohalogen Compounds 28: 77-82. http://oaspub.epa.gov/eims/eimscomm.getfile?p_download_id=36293

Di Toro, D.M., J.D. Mahony, D.J. Hansen, K.J. Scott, A.R. Carlson and G.T. Ankley (1992). Acid volatile sulfide predicts the acute toxicity of cadmium and nickel in sediments. Environ Sci Technol 26(1): 96-101.

Di Toro, D.M., C.S. Zarba, D.J. Hansen, W.J. Berry, R.C. Swartz, C.E. Cowan, S.P. Pavlou, H.E. Allen, N.A. Thomas and P.R. Paquin (1991). Technical Basis for Establishing Sediment Quality Criteria for Nonionic Organic Chemicals Using Equilibrium Partitioning. Environ Toxicol Chem 10(12): 1541-1584.

Driscoll, C.T., V. Blette, C. Yan, C.L. Schofield, R. Munson and J. Holsapple (1995). The role of dissolved organic carbon in the chemistry and bioavailability of mercury in remote Adirondack lakes. Water, Air, Soil Pollution 80(1-4): 499-508.

Enya, T., H. Suzuki and T. Watanabe (1997). 3-nitrobenzanthrone, a powerful bacterial mutagen and suspected human carcinogen found in diesel exhaust and airborne particulates. Environmental Science & Technology 31: 2772-6.

Environmental Protection Agency (2003a). Supplemental Guidance for Assessing Cancer Susceptibility from Early-Life Exposure to Carcinogens. Risk Assessment Forum Technical Panel, National Center for Environmental Assessment, Office of Research and Development, Washington, D.C. External Review Draft EPA/630/R-03/003, February 28, 2003.

Environmental Protection Agency (2003b). Five-Year Review Report: First Review For St. Louis River Superfund Site. Written by Army Corp of Engineers, Omaha, NE. September 2003.

Environmental Protection Agency, (2003c). Integrated Risk Information System (IRIS). Office of Health and Environmental Assessment, Environmental Criteria Office, Online, Last updated November 2003, http://www.epa.gov/iris/

Environmental Protection Agency (2002a). Child-Specific Exposure Factors Handbook. National Center for Environmental Assessment, Office of Research and Development, Washington, D.C. Interim Report EPA-600-P-00-002B, September 2002.

Environmental Protection Agency (2002b). Peer Consultation Workshop on Approaches to Polycyclic Aromatic Hydrocarbon (PAH) Health Assessment. National Center for Environmental Assessment, Office of Research and Development, Washington, DC. EPA/635/R-02/005, January 2002.

Environmental Protection Agency (2002c). Toxicological Review of Toluene. National Center for Environmental Assessment, Washington, D.C. *Draft* Report NCEA-S-1264, August 2002.

Environmental Protection Agency (2001a). Water Quality Criterion for the Protection of Human Health: Methylmercury. Office of Science and Technology, Office of Water, Washington, D.C. Final Report EPA-823-R-01-001, January 2001.

Environmental Protection Agency (2001b). Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual (Part E, Interm Supplemental Guidance for Dermal Risk Assessment). Office of Emergency and Remedial Response, Washington, D.C. Interim Report EPA/540/R/99/005, September 2001.

Environmental Protection Agency (2000a). Development of a Framework for Evaluating Numerical Sediment Quality Targets and Sediment Contamination in the St. Louis River Area of Concern. Minnesota Pollution Control Agency, Environmental Outcomes Division, St. Paul, MN. Report EPA 905-R-00-008, December 2000. http://www.pca.state.mn.us/water/sediments/sqt-slraoc.pdf, http://www.pca.state.mn.us/water/sediments/sqt-tables.pdf

Environmental Protection Agency (2000b). Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories: Volume 2, Risk Assessment and Fish Consumption Advice. Office of Water, Washington, D.C. 3rd Edition EPA 823-B-00-008, November 2000.

Environmental Protection Agency (2000c). DRAFT - Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo- p-Dioxin (TCDD)-and Related Compounds: Part I: Estimating Exposure to Dioxin-Like Compounds, Volume 3: Properties, Environmental Levels, and Background Exposures. Exposure

Assessment and Risk Characterization Group, National Center for Environmental Assessment - Washington Office, Office of Research and Development, Washington, D.C. EPA/600/P-00/001Bc, December 1997.

Environmental Protection Agency (1999). Great Lakes Binational Toxics Strategy Draft Report for Hexachlorobenzene (HCB): Sources and regulations. Great Lakes National Program Office, Chicago, IL. November 1, 1999. http://www.epa.gov/grtlakes/bns/baphcb/HCBSteps_web_1.PDF

Environmental Protection Agency (1998a). Multimedia Strategy For Priority Persistent, Bioaccumulative, and Toxic (PBT) Pollutants: Executive Summary. Washington, D.C. Draft, November 16, 1998. http://www.epa.gov/opptintr/pbt/execsumm.htm

Environmental Protection Agency (1998b). Great Lakes Binational Toxics Strategy Octachlorostyrene (OCS) Report: A review of potential sources - Draft. Great Lakes National Program Office, Chicago, IL, Written by Battelle Memorial Institute. December 22, 1998. http://www.epa.gov/grtlakes/bnsdocs/98summ/ocs.pdf

Environmental Protection Agency (1997a). Exposure Factors Handbook. Office of Research and Development, National Center for Environmental Assessment, Washington, D.C. Report EPA/600/P-95/002Fa, August, 1997.

Environmental Protection Agency (1997b). Mercury Study Report to Congress. Office of Air Quality Planning and Standards and Office of Research and Development, Washington, D.C., December 1997.

Environmental Protection Agency (1997c). Health Effects Assessment Summary Tables (HEAST): FY 1997 Update. Solid Waste and Emergency Response, Washington, D.C. EPA-540-R-97-036, July 1997.

Environmental Protection Agency (1997d). The Incidence and Severity of Sediment Contamination in Surface Waters for the United States: Volume 1, National Sediment Quality Survey. Office of Science and Technology, Washington, D.C. Report EPA 823-R-97-006, September 1997.

Environmental Protection Agency (1996). Soil Screening Guidance: Technical Background Document. Office of Emergency and Remedial Response, Washington, D.C. Report EPA/540/R-95/128, July 1996.

Environmental Protection Agency (1995). Regional Environmental Monitoring and Assessment Program (REMAP). Washington, D.C. Data, 1995.

Environmental Protection Agency (1989). Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual (Part A). Office of Emergency and Remedial Response, Washington, D.C. Report EPA/540/1-89/002, December 1989.

Environmental Protection Agency (1988). Superfund Exposure Assessment Manual. Office of Remedial Response, Washington, D.C. OSWER Directive 9285.5-1 EPA/540/1-881001, April 1988.

Environmental Protection Agency and Minnesota Pollution Control Agency (1997a). Survey of Sediment Quality in the Duluth/Superior Harbor: 1993 Sample Results. Great Lakes National Program Office, M. Schubauer-Berigan, J. L. Crane, Chicago, IL. EPA 905-R97-005, March 1997.

Environmental Protection Agency and Minnesota Pollution Control Agency (1997b). Sediment Assessment of Hotspot Areas in the Duluth/Superior Harbor. Great

Lakes National Program Office, M. Schubauer-Berigan, J.L. Crane, K. Schmude, Chicago, IL. EPA 905-R97-020, December 1997.

Food and Agriculture Organization of the United Nations and World Health Organization (2001). Summary of the fifty-seventh meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). Expert Committee on Food Additives, Rome, Italy. Meeting Summary Report, June 5-14, 2001.

Federal Water Pollution Control Administration (1967-8). Aerial Photographs of US Steel Facility and St. Louis River. US EPA, Courtesy G. Glass, Duluth, MN.

Fernandez, P., J. Albalges, M. Grifoll, A.M. Solanas and J.M. Bayona (1992). Bioassay-directed chemical analysis of genotoxic components in coastal sediments. Environmental Science & Technology 26(4): 817-829.

Ginsberg, G.L. (2003). Assessing cancer risks from short-term exposures in children. Risk Anal 23(1): 19-34.

Glass, G.E., J.A. Sorenson, G.R. Rapp Jr, M. Balcer and L. Schwarzkopf (1999). Mercury subsurface maxima in sediments: a diagnostic for anthropogenic origins. Mercury Contaminated Sites: Characterization, Risk Assessment and Remediation. R. Ebinghaus, R.R.T., L.D. de Lacerda, O. Vasiliev, W.Salomons. New York, Springer: 467-485.

Gustafsson, O., F. Haghseta, C. Chan, J. Macfarlane and P.M. Gschwend (1997). Quantification of the dilute sedimentary soot phase: Implications for PAH speciation and bioavailability. Environmental Science & Technology 31(1): 203-209.

Halmes, N.C., S.M. Roberts, J.K. Tolson and C.J. Portier (2000). Reevaluating cancer risk estimates for short-term exposure scenarios. Toxicol Sci 58(1): 32-42.

Hansen, D.J., W.J. Berry, J.D. Mahony, W.S. Boothman, D.M. Di Toro, D.L. Robson, G.T. Ankley, D. Ma, Q. Yan and C.E. Pesch (1996). Predicting the toxicity of metal-contaminated field sediments using interstitial concentration of metals and acid-volatile sulfide normalizations. Environmental Toxicology And Chemistry 15(12): 2080-2094.

International Agency for Research on Cancer (2005). Monographs. Lyon, France. Multiple volumes; list at URL http://monographs.iarc.fr/

International Technology Corporation (1997). Remedial Investigation Data Report Sediment Operable Unit St. Louis River/Interlake/Duluth Tar Site: Volume I, II. Duluth, Minnesota. RI, May, 1997.

International Technology Corporation (1996). Final Amendments to Draft Work Plan Sediment Operable Unit Supplemental Remedial Investigation and Feasibility Study for SLRIDT Site, Duluth, MN. H. Huls, St. Paul, Minnesota. Letter, July 31, 1996.

Johnson, B.E. and J. Ferguson (1990). Drug and chemical photosensitivity. Semin Dermatol. 9(1): 39-46. Review.

Kabata-Pendias, A. and H. Pendias (2001). Trace Elements in Soil and Plants. Third Edition. Boca Raton, CRC Press.

Krabbenhoft, D.P., J.G. Wiener, W.G. Brumbaugh, M.L. Olson, J.F. DeWild and T.J. Sabin (1999). A national pilot study of mercury contamination of aquatic ecosystems along multiple gradients. U.S. Geological Survey Toxic Substances Hydrology Program--Proceedings of the Technical Meeting, Volume 2.

Contamination of Hydrologic Systems and Related Ecosystems: U.S. Geological Survey Water-Resources Investigations Report 99-4018B. Morganwalp, D.W. and H.T. Buxton: 147-160.

Lancaster University, (2003). POPs Fate Modelling. Department of Environment, Food and Rural Affairs, http://www.es.lancs.ac.uk/ecerg/kcjgroup/modelling.html

Lasorsa, B. and S. Allen-Gil (1995). The Methylmercury to Total Mercury Ratio in Selected Marine Freshwater and Terrestrial Organisms. Water Air and Soil Pollution 80: 905-913.

Lyman, W.J. (1995). Transport and Transformation Processes. Fundamentals of Aquatic Toxicology. Rand, G.M. Washington, D.C., Taylor & Francis: 449-492.

Maruya, K.A., R.W. Risebrough and A.J. Horne (1996). Partitioning of polynuclear aromatic hydrocarbons between sediments from San Francisco Bay and their porewaters. Environmental Science & Technology 30(10): 2942-2947.

Massachusetts Department of Environmental Protection (2002). Weighted Skin-Soil Adherence Factors. Office of Research and Standards, Boston, MA. Technical Update, April, 2002.

Minnesota Department of Health, (2003a). Methods for Estimating the Carcinogenic Health Risks from Dioxin-Like Compounds. Environmental Assessment and Surveilance, Environmental Health, St. Paul, MN. http://www.health.state.mn.us/divs/eh/risk/chemicals/dioxinnm.html

Minnesota Department of Health, (2003b). Dioxins. Environmental Assessment and Surveilance, Environmental Health, St. Paul, MN. http://www.health.state.mn.us/divs/eh/risk/chemicals/dioxinpr.htm

Minnesota Department of Health (2002a). MDH guidance on lead contaminated soil. Messing, R., Environmental Assessment and Surveilance, Environmental Health, St. Paul, Minnesota. Letter To: Payne, G., November 26, 2002.

Minnesota Department of Health, (2002b). Health Risk Values (HRVs). Environmental Assessment and Surveilance, Environmental Health, St. Paul, MN. Minnesota Rule, Online, Last updated September 11, 2002, http://www.revisor.leg.state.mn.us/arule/4717/8000.html

Minnesota Department of Health, (2001a). Methods for Estimating the Carcinogenic Health Risks from Polycyclic Aromatic Hydrocarbons. Herbrandson, C. and C. Stroebel, Environmental Assessment and Surveilance, Environmental Health, St. Paul, MN. Memo To: Goeden, H., Minnesota Pollution Control Agency. May 29, 2001. http://www.health.state.mn.us/divs/eh/risk/chemicals/dioxinnm.html

Minnesota Department of Health (2001b). Interlake Site Remedial Action Alternatives. Memo from Herbrandson, C., Environmental Assessment and Surveilance, Environmental Health, St. Paul, MN to Mosel, J., Minnesota Pollution Control Agency. February 2, 2001.

Meador, J.P., J.E. Stein, W.L. Reichert and U. Varanasi (1995). Bioaccumulation of polycyclic aromatic hydrocarbons by marine organisms. Rev Environ Contam Toxicol 143: 79-165.

Minnesota Pollution Control Agency (2000). Mercury discussion. St. Paul. Conversation with: to Swain, E., September 5, 2000.

Minnesota Pollution Control Agency (1999). Risk-Based Guidance For The Soil - Human Health Pathway, Vol. 2. Policy and Planning Division, St. Paul, MN.

Minnesota Pollution Control Agency (1989). Record of Decision (US Steel Site). St. Paul, MN. Declaration, Administrative Record Index, Summary of remedial alternative selection, February, 22, 1989.

Muir, D.C.G., B.N. Billeck, G.J. Brunskill, N.P. Grift, W.L. Lockhart and P. Wilkinson (1995). Spatial trends and historical profiles of organochlorine pesticides in Arctic lake sediments. Science of the Total Environment 160-161: 447-457.

New York State Department of Environmental Conservation (1999). Technical Guidance for Screening Contaminated Sediments. Division of Fish, Wildlife and Marine Resources, Albany, NY. Report, January 25, 1999.

New York State Department of Environmental Conservation (1997). Ambient Water Quality Value for Protection of Sources of Potable Water: Octachlorostyrene. Division of Water, Albany, N.Y., January 29, 1997.

Niimi, A.J. and G.P. Dookhran (1989). Dietary Absorption Efficiencies and Elimination Rates of Polycyclic Aromatic Hydrocarbons in Rainbow Trout (*Salmo gairdneri*). Environmental Toxicology And Chemistry 8: 719-722.

Niimi, A.J. and V. Palazzo (1986). Biological Half-lives of Eight Polycyclic Aromatic Hydrocarbons in Rainbow Trout (*Salmo gairdneri*). Water Res 20: 503-507.

National Toxicology Program (2001). 9th Report on Carcinogens. National Institute of Environmental Health, U.S. Department of Health and Human Services, Public Health Service, Research Triangle Park, NC. http://ehis.niehs.nih.gov/roc/toc9.html

National Toxicology Program (2000). Toxicology and Carcinogenesis Studies of Naphthalene (CAS No. 91-20-3) in F344/N Rats (Inhalation Studies). National Institute Health, U.S. Department of Health and Human Services, Public Health Service, Research Triangle Park, NC. December 2000. http://ntp-server.niehs.nih.gov/htdocs/LT-studies/tr500.html

Oliver, B.G. and K.D. Nicol (1982). Chlorobenzenes in sediments, water, and selected fish from Lakes Superior, Huron, Erie, and Ontario. Environmental Science and Technology 16(8): 532-536.

Ontario Ministry of the Environment (1993). Guidelines for the protection and management of aquatic sediment quality in Ontario. Water Resources Branch, D. Persaud, R. Jaagumagi, A. Hayton, Toronto. Report ISBN 0-7729-9248-7, August 1993, 26 pg.

Ownby, D.R., M.C. Newman, M. Mulvey, W.K. Vogelbein, M.A. Unger and L.F. Arzayus (2002). Fish (Fundulus heteroclitus) populations with different exposure histories differ in tolerance of creosote-contaminated sediments. Environ Toxicol Chem 21(9): 1897-902.

Rudd, J.W. (1995). Sources of methyl mercury to freshwater ecosystems: A review. Water Air And Soil Pollution 80(1-4): 697-713.

Saint Louis, V.L., J.W. Rudd, C.A. Kelly, K.G. Beaty, R.J. Flett and N.T. Roulet (1996). Production and loss of methylmercury and loss of total mercury from boreal forest catchments containing different types of wetlands. Environmental Science & Technology 30(9): 2719-2729.

Schwarzenbach, R.P., P.M. Gschwend and D.M. Imboden (1993). Environmental Organic Chemistry. New York, John Wiley & Sons, Inc.

Sipes, I.G. and A.J. Gandolfi (1991). Biotransformation of toxicants. Casarett and Doull's Toxicology. Amdur, M.O., J. Doull and C.D. Klaassen. New York, Pergamon Press: 88-126.

Slemr, F., E.-G. Brunke, R. Ebinghaus, C. Temme, J. Munthe, I. Wängberg, W. Schroeder, A. Steffen and T. Berg (2003). Worldwide trend of atmospheric mercury since 1977. Geophys. Res. Lett., 30(10).

Sorensen, J.A., G.R. Rapp Jr, G.E. Glass, K.W. Schmidt and J.K. Huber (1990). Airborne mercury deposition and watershed characteristics in relation to mercury concentrations in water, sediments, plankton, and fish of eighty northern Minnesota lakes. Environmental Science & Technology 24(11): 1716-1727.

St. Louis River Community Action Committee (2000). Historic Reconstruction of Property Ownership and Land Uses along the Lower St. Louis River. D.K. Kellner, T. Kroska, K. Plass, Duluth, MN. slrcac@StLouisRiver.org, January 2000.

Stortz, K.R. and M. Sydor (1980). Transports in the Duluth-Superior Harbor. Journal of Great Lakes Research 6(3): 223-231.

Swackhamer, D.L., B.D. McVeety and R.A. Hites (1988). Deposition and Evaporation of Polychlorobiphenyl Congeners to and from Siskiwit Lake Isle Royale Lake Superior USA. Environ Sci Technol 22(6): 664-672.

Syracuse Research Corporation (SRC), (2003). Interactive PhysProp Database Demo. http://esc.syrres.com/interkow/physdemo.htm

Tremblay, A., M. Lucotte, M. Meili, L. Cloutier and P. Pichet (1996). Total mercury and methylmercury contents of insects from Boreal Lakes: Ecological, Spatial and temporal patterns. Water Quality Research Journal Of Canada 31(4): 851-873.

Ullrich, S.M., T.W. Tanton and S.A. Abdrashitova (2001). Mercury in the aquatic environment: a review of factors affecting methylation. Critical Reviews in Environmental Science and Technology 31(3): 241-93.

US Fish and Wildlife Service, (2002). Whole Fish PAH Data. A. Trowbridge, Data from A. Trowbridge,

Van den Berg, M., L. Birnbaum, A.T. Bosveld, B. Brunstrom, P. Cook, M. Feeley, J.P. Giesy, A. Hanberg, R. Hasegawa, S.W. Kennedy, T. Kubiak, J.C. Larsen, F.X. van Leeuwen, A.K. Liem, C. Nolt, R.E. Peterson, L. Poellinger, S. Safe, D. Schrenk, D. Tillitt, M. Tysklind, M. Younes, F. Waern and T. Zacharewski (1998). Toxic equivalency factors (TEFs) for PCBs, PCDDs, PCDFs for humans and wildlife. Environ Health Perspect 106(12): 775-92.

Van Der Oost, R., L. Van Gastel, D. Worst, M. Hanraads, K. Satumalay, F. Van Schooten, H. Hedia and N.P. Vermeulen (1994). Biochemical markers in feral roach (Rutilus rutilus) in relation to the bioaccumulation of organic trace pollutants. Chemosphere 29(4): 801-817.

Varanasi, U., J.E. Stein and M. Nishimoto (1989). Biotransformation and Disposition of Polycyclic Aromatic Hydrocarbons (PAH) in Fish. Metabolism of Polycyclic Aromatic Hydrocarbons in the Aquatic Environment. Varanasi, U. Boca Raton, FL, CRC Press, Inc.: 93-149.

Veith, G.D. and P. Kosian (1983). Estimating bioconcentration potential from octanol/water partition coefficients. Physical Behavior of PCBs in the Great

Lakes. Mackay, D., S. Patterson, S. Eisenreich and Simmons. Ann Arbor, MI, Ann Arbor Science: 269-282.

Verschueren, K. (1977). Handbook of Environmental Data on Organic Chemicals. New York, Van Nostrand Reinhold Company.

Washington State Department of Health (1996). Development of Sediment Quality Criteria for the Protection of Human Health. Office of Toxic Substances, Olympia, WA. Tier II Report, May 1996.

Washington State Department of Health (1995). Development of Sediment Quality Criteria for the Protection of Human Health. Office of Toxic Substances, Olympia, WA. Tier I Report, June 1995.

Wiener, J.G., R.E. Martini, T.B. Sheffy and G.E. Glass (1990). Factors influencing mercury concentrations in walleyes in northern Wisconsin (USA) lakes. Trans Am Fish Soc 119(5): 862-870.

Minnesota Department of Health

Appendices

Human Health Screening Values for ST. LOUIS RIVER Sediments: US Steel Site (SSV Report)

August 8, 2005

Appendices

Table of Contents

Appendix A - Glossary: Equation variables

ABS_{Derm} = dermally absorbed fraction (unitless)
ABS_{GI} = fraction of applied dose absorbed in primary (RfD) study (unitless)
ABS_{Sed}= oral absorption adjustment - relative bioavailability (unitless)
ABS_{SW} ($(mg/(cm^2 \cdot event))/(mg/cm^3)$) general term representing the dermally absorbed dose from a chemical concentration in water: dependent on event duration and chemical specific factors.
$ABS_{SW\text{-}met}$ = dermal absorption of metals from water $(mg/(cm^2 \cdot event))/(mg/cm^3)$ (*Equation #A-22*)
$ABS_{SW\text{-}org}$ = dermal absorption of organics from water $(mg/(cm^2 \cdot event))/(mg/cm^3)$ (*Equation #A-23*)
AC = Amount consumed (g/meal)
$AccptRsk_c$ = acceptable risk - cancer (unitless)
AF_{ED} = event duration-dependent adjustment factors (hr/event) (*Equation #A-30, #31*)
AF_{swm} = sediment adherence factor - swimming (mg/cm^2)
AF_{wad} = sediment adherence factor - wading (mg/cm^2)
$AT_{(c)}$ = cancer averaging period (70 yrs; EPA convention (EPA, 1989))
β = ratio of stratum corneum and epidermis permiabilities (unitless) (*Equation #A-25*)
BSAF = biota-sediment accumulation factor - non-polar organics - $(kg_{Sed\ oc}/kg_{fish\ lipid})$
- mercury - (kg_{Sed}/kg_{fish})
BCF = bioconcentration factor $(L_{SW}/kg_{fish\ lipid})$
BW = body weight (kg)
C_A = chemical concentration in air (mg/m^3)
C_{Sed} = chemical concentration in sediment (mg/kg)
C_{SW} = chemical concentration in surface water (mg/L)
$CF_{d/wk}$ = conversion factor (7 d/wk)
$CF_{d/y}$ = conversion factor (365 d/yr)
$CF_{g/kg}$ = conversion factor (1000 g/kg)
$CF_{hr/d}$ = conversion factor (24 hr/d)
$CF_{cm3/L}$= conversion factor $(1{,}000\ cm^3/L)$
$CF_{L/m3}$ = conversion factor $(1{,}000\ L/m^3)$
$CF_{\mu g/mg}$= conversion factor (1,000 µg/mg)
$CF_{mg/kg}$= conversion factor (1,000,000 mg/kg)
$CF_{mL/L}$ = conversion factor (1,000 mL/L)
ED = event duration (hr/event)
EF_{swm} = event frequency - swimming (event/yr)
$EF_{swm\text{-}d}$= event-day frequency (event-d/yr)
EF_{wad} = event frequency - wading (event/yr)
$EF_{wad\text{-}d}$ = event-day frequency (event-d/yr)
$EP_{(c)}$ = exposure period - cancer (33 years (EPA, 1997a))
FA = fraction absorbed from water (unitless)
$Fish_{Ing}$ = fish ingestion rate $(g_{fish}/(kg_{bw} \cdot d))$ (*Equation #A-12*)
$Fish_{Ing\text{-}c}$ = fish ingestion rate - lifetime average $(g_{fish}/(kg_{bw} \cdot d))$ (*Equation #A-13*)
f_{lipid} = fraction lipid in fish (g_{lipid}/g_{fish})
f_{oc} = fraction organic carbon in sediment (unitless)
HQ = hazard quotient non-cancer (chronic) endpoint (unitless)
i = from 1 – 33 (yrs)
$Ing_{Sed(swm)}$ = sediment ingested per hour swimming (mg/hr) (*Equation #A-1*)

$Ing_{Sed(wad)}$ = sediment ingested per hour wading (mg/hr) (*Equation #A-1*)
$Ing_{SW(swm)}$ = surface water ingested per hour swimming (L/hr)
$Ing_{SW(wad)}$ = surface water ingested per hour wading (L/hr)
Inh_{frac} = fraction of time onsite (unitless) (*Equation #A-10*)
Inh_{frac-c} = time onsite - lifetime average (unitless) (*Equation #A-11*)
InhRate = inhalation rate (m^3/d)
K_H = Henry's Law constant (atm-m^3/mol)
K_{oc} = organic carbon partitioning constant (L/kg)
K_{ow} = octanol water partitioning constant (unitless)
K_p = permeability coefficient (cm/hr)
MF = Fish meal frequency (meal/week)
MW = molecular weight of chemicals of interest (g/mol)
PEF = Potency equivalence factor (unitless)
R = ideal gas constant (@ 1 atm) (0.082057 L/ (mol · ºK))
RfC = reference concentration - safe chronic exposure concentration for general public (air) (mg/m^3)
RfD = reference dose - safe chronic exposure concentration for general public (ingestion) (mg/(kg·d))
SA_{ttl} = total surface area (cm^2)
$SA_{\%swm}$ = percent of body exposed swimming (%)
$SA_{\%wad}$ = percent of body exposed wading (%)
Sed_{Derm} = dermal sediment exposure ($mg_{sed}/(kg_{bw}·d)$) (*Equation #A-6*)
Sed_{Derm-c} = dermal sediment exposure - lifetime average ($mg_{sed}/(kg_{bw}·d)$) (*Equation #A-7*)
Sed_{Ing} = amount of sediment ingested ($mg_{sed}/(kg_{bw}·d)$) (*Equation #A-2*)
Sed_{Ing-c} = amount of sediment ingested - lifetime average ($mg_{sed}/(kg_{bw}·d)$) (*Equation #A-4*)
SF_c = oral cancer slope factor $((mg/(kg_{bw}·d))^{-1})$
SS_{SW} = suspended sediment concentration in surface water (mg/L)
$SSV_{\%x}$ = % contribution by individual routes of exposure to SSV_{ttl} (%) (*Equation #A-48*)
SSV_{Fish} = route-specific sediment value (chronic)
- fish consumption (mg/kg) (*Equations #A-33, #A-34, #A-35*)
SSV_{Fish-c} = route-specific sediment value (cancer)
- fish consumption (mg/kg) (*Equations #A-43, #A-44, #A-45*)
SSV_{Inh} = route-specific sediment value (chronic) - inhalation (mg/kg) (*Equation #A-32*)
SSV_{Inh-c} = route-specific sediment value (cancer) - inhalation (mg/kg) (*Equation #A-42*)
$SSV_{Sed(Derm)}$ = route-specific sediment value (chronic) - dermal sediment (mg/kg) (*Equation #A-19*)
$SSV_{Sed(Derm)-c}$ = route-specific sediment value (cancer) - dermal sediment (mg/kg) (*Equation #A-39*)
$SSV_{Sed(Ing)}$ = route-specific sediment value (chronic) - ingestion (mg/kg) (*Equation #A-16*)
$SSV_{Sed(Ing)-c}$ = route-specific sediment value (cancer) - ingestion (mg/kg) (*Equation #A-36*)
$SSV_{SW(Derm)}$ = route-specific sediment value (chronic) - dermal surface water (mg/kg) (*Equation #A-21*)
$SSV_{SW(Derm)-c}$ = route-specific sediment value (cancer) - dermal surface water (mg/kg) (*Equation #A-41*)
$SSV_{SW(Ing)}$ = route-specific sediment value (chronic) - water ingestion (mg/kg) (*Equation #A-18*)
$SSV_{SW(Ing)-c}$ = route-specific sediment value (cancer) - water ingestion (mg/kg) (*Equation #A-38*)
SSV_{ttl} = sediment screening value - chronic (mg/kg) (*Equation #A-46*)
SSV_{ttl-c} = sediment screening value - cancer (mg/kg) (*Equation #A-47*)
$SWC_{SW(Derm)}$ = partial water screening conc.(chronic)
- surface water dermal (mg/L) (*Equations #A-20, #A-20a*)
$SWC_{SW(Derm)-c}$ = partial water screening conc.(cancer)
- surface water dermal (mg/L) (*Equation #A-40*)

$SWC_{SW(Ing)}$ = partial water screening concentration (chronic)
- water ingestion (mg/L) (*Equation #A-17*)

$SWC_{SW(Ing)-c}$ = partial water screening concentration (cancer)
- water ingestion (mg/L) (*Equation #A-37*)

SW_{Derm} = surface area exposed to surface water (cm^2·event/(kg_{bw}·d)) (*Equation #A-8*)

SW_{Derm-c} = surface area exposed to surface water - lifetime average (cm^2·event/(kg_{bw}·d))(*Equation #A-9*)

SW_{Ing} = amount of water ingested (L/(kg_{bw}·d)) (*Equation #A-3*)

SW_{Ing-c} = amount of water ingested - lifetime average (L/(kg_{bw}·d)) (*Equation #A-5*)

τ = lag time per event (hr/event) (*Equation #A-24*)

T = temperature (293.13ºK default)

t^* = time to steady state (hr) (*Equation #A-26, #A-27*)

TCDD-TEQ = 2,3,7,8-Tetrachlorodibenzo-p-dioxin toxic equivalents

B[a]P-PEQ = Benzo(a)pyrene potency equivalents

TEF = toxic equivalence factor (unitless)

UR_c = unit risk - cancer ($(\mu g/m^3)^{-1}$)

Appendix B - Media Exposure/Contact Calculations

Variables are defined in Appendix A.

Ingestion of sediment and water

Annual daily average sediment and surface water ingestion rates were calculated using the following equations:

$Ing_{Sed(wad,swm)}$ {mg_{sed}/hr } = $Ing_{SW(wad,swm)}$ * SS_{SW} / $CF_{mL/L}$ *Equation #A-1.*

Child example: $Ing_{Sed(wad)}$ *= 25 mL/hr * 370 mg/L / 1000 mL/L*

= 9.25 mg/hr

$Ing_{Sed(swm)}$ *= 250 mL/hr * 370 mg/L / 1000 mL/L*

= 92.5 mg/hr

Sed_{Ing} { $mg_{sed}/(kg_{bw}\cdot d)$ } = ($Ing_{Sed(wad)}$*EF_{wad}*ED_{wad} + $Ing_{Sed(swm)}$*EF_{swm}*ED_{swm}) / (BW*$CF_{d/y}$) *Equation #A-2.*

Child example: Sed_{Ing} *=(9.25 mg/hr*17.2 events/yr*0.5 hr/event+92.5 mg/hr * 103events/yr*0.5hr/event) / (16 kg_{bw} * 365 d/yr)*

=0.829 mg/($kg_{bw}\cdot d$)

(difference between example and Table 3 due to rounding of intermediate calculations)

SW_{Ing} { $L/(kg_{bw}\cdot d)$ } = ($Ing_{SW(wad)}$*ED_{wad}*EF_{wad}+ $Ing_{SW(swm)}$*ED_{swm}*EF_{swm}) / (BW* $CF_{d/y}$)* $CF_{L/mg}$ *Equation #A-3.*

Calculation results are shown in Table B-1.

Lifetime average daily sediment and surface water intake rate calculations (cancer)

$Sed_{Ing\text{-}c}$ { $mg_{sed}/(kg_{bw}\cdot d)$ } = $(\sum_{i=1}^{33} Sed_{Ing(i)})/ AT_{(c)}$ *Equation #A-4.*

$SW_{Ing\text{-}c}$ { $L/(kg_{bw}\cdot d)$ } = $(\sum_{i=1}^{33} SW_{Ing(i)})/ AT_{(c)}$ *Equation #A-5.*

Where:

$Sed_{Ing(i)}$ = (Sed_{Ing} at age i) * 1 yr

Calculation results are shown in Table B-1.

Dermal exposure to sediment

As shown in SSV Report Table #5 (SSV Report Section 3.3.3), it is assumed that individuals wading in the St. Louis River expose 20% of their bodies to water and sediment. Swimming exposes 90% of the total surface area. Median surface areas for the identified age groups are in SSV Report Table #1 (SSV Report Section 3). Direct exposures to sediment continue after the

event until the sediment is washed off. Therefore, the event frequency for direct sediment exposure is measured in event-days per year (event-d/yr).

Total dermal exposure to sediment during wading and swimming (annual daily average) is:

$$Sed_{Derm} \{ mg_{sed}/(kg_{bw} \cdot d) \} = SA_{ttl} * (SA_{\%wad} * EF_{wad\text{-}d} * AF_{wad} + SA_{\%swm} * EF_{swm\text{-}d} * AF_{swm}) / (BW * CF_{d/y})$$ Equation #A-6.

Calculation results are shown in Table B-1 (SSV Report Table #5; Section 3.3.3).

Sediment Adherence Factor

Sediment adherence to skin is activity-dependent and has a large impact on dermal exposure. The 1992 EPA Interim Dermal Guidance (EPA, 1992) recommended using 0.2 to 1.0 mg/cm^2 as soil adherence factors for most exposures. Current interim guidance (EPA, 2001a) recommends using different values for children and for adults: with 0.07 mg/cm^2 as the central tendency for most adult activities, and 0.2 mg/cm^2 as the central tendency for most child activities. The Massachusetts Department of Environmental Protection uses a sediment adherence factor of 1.0 mg/cm^2 for exposures while swimming, playing, and wading (Massachusetts DEP, 2002).

The range of adherence (dermal loading) factors (geometric means of adherence factors for individuals in a limited number of studies) in the EPA draft Dermal Guidance is from a minimum of 0.01 mg/cm^2 [children indoors and groundskeeper] to a maximum of 21 mg/cm^2 [children playing in mud]. The 95th percentile of individual adherence factors found in these same studies were 0.06 mg/cm^2 and 231 mg/cm^2, respectively. There appears to be consensus in the scientific literature that soil adherence increases with greater moisture content; and that significant transfer of chemicals from soil (or sediment) on skin is likely limited to a monolayer, with additional layers of sediment having little or no effect on the amount of chemical transferred.

Chemical transfer from sediment through the skin (dermal absorption fractions; ABS_{Derm}) should be determined using a monolayer of sediment applied to the skin. Less than a monolayer can potentially transfer a higher proportion of the chemical from the sediment (sediment depletion; high ABS_{Derm}, but low transfer rate), and more than a monolayer results in exposure to a lesser fraction of the total sediment associated chemical (excess in sediment; lower ABS_{Derm}, but maximum transfer rate). Soil dermal absorption fractions (ABS_{Derm}; Table 3-4) from the EPA Interim Dermal Guidance (EPA, 2001a) were used as sediment absorption fractions in this document. Most of these chemical-specific absorption factors are based on studies by Wester et al. (1990; 1993b; 1993a). The Wester et al. studies were conducted using soils that were screened to a particle size between 185 and 330 µm. Forty mg/cm^2 of this soil was then applied to the dermal surface. Duff and Kissel (1996) calculate that this loading is equivalent to a monolayer of particles in the 185-330 µm range. On the other hand, Duff and Kissel used soil sieved to less than 150 µm in their own experiments and found that their soils created a monolayer when they were loaded to about 2 mg/cm^2.

The difference in exposure that results from monolayers of different size particles, or from sediments with different fractions of organic carbon, have not been adequately described.

Therefore, the effects of site-specific parameters need to be carefully evaluated if this dermal absorption model is used for purposes other than screening. (See Bunge and Parks, 1997; EPA, 2001a for additional information.)

The Massachusetts Department of Environmental Protection 1.0 mg/cm^2 sediment adherence factor for areas of the skin exposed to sediment during wading is reasonable and is used in this document. However, it is our judgment that lower values are appropriate for swimming. Therefore, 0.2 mg/cm^2 and 0.07 mg/cm^2 are used as adherence factors for swimming children (1-16) and adults (>16), respectively SSV Report Table #5 (SSV Report Section 3.3.3).

Lifetime average daily dermal sediment contact rate calculation (cancer)
The lifetime average surface area exposed to sediment during wading and swimming is:

$$Sed_{Derm\text{-}c}\ \{\ kg_{sed}/(kg_{bw}\cdot d)\ \} = (\sum_{i=1}^{33} Sed_{Derm(i)}) / AT_{(c)} \qquad \textit{Equation \#A-7.}$$

Calculation results are shown in Table B-1.

Dermal exposure to surface water
Since exposure to chemicals in water only occurs during an event, the event frequency for this route-of-exposure is in events per year (event/yr).

Annual daily average surface area exposed to surface water during wading and swimming is:

$$SW_{Derm}\ \{\ cm^2/(kg_{bw}\cdot d)\ \} = SA_{ttl} * (SA_{\%wad} * EF_{wad} + SA_{\%swm} * EF_{swm}) / (BW * CF_{d/y}) \qquad \textit{Equation \#A-8.}$$

Calculation results are shown in Table B-1.

Lifetime average daily dermal surface water contact rate calculation (cancer)
The lifetime average surface area exposed to surface water during wading and swimming is:

$$SW_{Derm\text{-}c}\ \{\ cm^2/(kg_{bw}\cdot d)\ \} = (\sum_{i=1}^{33} SW_{Derm(i)}) / AT_{(c)} \qquad \textit{Equation \#A-9.}$$

Calculation results are shown in Table B-1.

Inhalation fraction
Assuming, in a screening assessment, that inhalation during all types of activities is similar, the fraction of time (annual average) that air overlying contaminated sediments is breathed may be:

$$Inh_{frac}\ \{\ unitless\} = (EF_{wad} * ED_{wad} + EF_{swm} * ED_{swm}) / (CF_{d/y} * CF_{hr/d}) \qquad \textit{Equation \#A-10.}$$

Calculation results are shown in Table B-1.

Lifetime average activity-related inhalation fraction calculation (cancer)
The fraction of lifetime inhalation associated with wading and swimming on the site may be calculated from:

$$Inh_{frac\text{-}c}\ \{\ unitless\} = \left(\sum_{i=1}^{33} Inh_{frac(i)}\right) / AT_{(c)} \qquad \textit{Equation \#A-11.}$$

Calculation results are shown in Table B-1.

Fish consumption

As noted in the SSV Report (3.2.6), the default consumption of fish tissue of an exposed individual (70 kg adult: SSV Report Table 1) is assumed to be 210 grams of fish tissue per week. Consumption rate is assumed to be similar for different age groups (see Table B-1).

$$Fish_{Ing}\ \{\ g_{fish}/(kg_{bw}\cdot d)\ \} = MF_{18\text{-}33} * AC_{18\text{-}33} / (BW_{18\text{-}33} * CF_{d/wk}) \qquad \textit{Equation \#A-12.}$$

Calculation results are shown in Table B-1.

Lifetime average daily fish ingestion rate calculations (cancer)

Lifetime consumption of fish is:

$$Fish_{Ing\text{-}c}\ \{\ g_{fish}/(kg_{bw}\cdot d)\ \} = Fish_{Ing} * EP_{(c)} / AT_{(c)} \qquad \textit{Equation \#A-13.}$$

Calculation results are shown in Table B-1.

Table B-1 - - Calculated Media Exposure/Contact Rates

	Potential Exposure	Sed_{Ing}	SW_{Ing}	Sed_{Derm}	SW_{Derm}	Inh_{frac}	Ing_{Fish}
	Years	$mg_{sed}/(kg_{bw}\cdot d)$	$L/(kg_{bw}\cdot d)$	$mg_{sed}/(kg_{bw}\cdot d)$	$cm^2/(kg_{bw}\cdot d)$	(Unitless)	$g_{fish}/(kg_{bw}\cdot d)$
Chronic Exposures	**1 - 6**	0.818	2.21E-03	14.6	111	0.00687	0.431
	7 - 17	0.306	8.30E-04	10.9	82.5	0.00687	0.431
	18 - 33	0.00946	2.56E-05	3.62	19.0	0.00245	0.431
Lifetime Yearly Average Exposures		$Sed_{Ing\text{-}c}$	$SW_{Ing\text{-}c}$	$Sed_{Derm\text{-}c}$	$SW_{Derm\text{-}c}$	$Inh_{frac\text{-}c}$	$Ing_{Fish\text{-}c}$
	33 *	0.120	0.000326	3.79	26.8	0.00223	0.203

* (EPA, 1997a)

Appendix C - Additional information on ambient mercury concentration in St. Louis River sediments

In areas of sufficiently high contamination, the food chain is likely affected by the lack of benthic communities or suppression of phytoplankton and zooplankton communities. These effects may be seen in areas where sediment concentrations of contaminants exceed ecological Lowest Effect Levels (LELs). The Ontario Ministry of the Environment (OMOE) LEL for tPAHs is 4 mg/kg (1993). In areas with tPAH concentrations above 4 mg/kg, the aquatic food chain is likely affected. Therefore, mercury in sediments with greater than 4 mg/kg tPAH may not enter the food chain as readily as mercury in areas where the food chain is more robust. Table C-1 shows the mean and median mercury concentrations at all locations where tPAH concentrations were below 0.25, 0.5, 1, 2, 4, 8, 16, 32 and 64 mg/kg. tPAH is used as a surrogate for contamination effects on the food chain.

Table C-1: Relationship between REMAP* mercury and tPAH data

			Ontario (OMOE) LEL - tPAH = 4 mg/kg							all REMAP (class 1,2) locations
REMAP sites w/tPAH	<0.25 mg/kg	<0.5 mg/kg	<1 mg/kg	<2 mg/kg	<4 mg/kg	<8 mg/kg	<16 mg/kg	<32 mg/kg	All tPAH sites	All mercury sites
# of sites (n=)	8	12	19	23	27	29	37	40	42	87
tHg Geometric Mean (mg/kg)	0.012	0.013	0.017	0.020	0.026	0.027	0.047	0.051	0.054	0.078
tHg Arithmetic Mean (mg/kg)	0.013	0.015	0.022	0.027	0.047	0.050	0.131	0.135	0.138	0.150
tHg Median (mg/kg)	0.012	0.014	0.018	0.020	0.023	0.023	0.034	0.038	0.045	0.100

Note: Class 1 sites accounted for 61-75% of sites in each tPAH category.
* descriptions of REMAP data (Regional Environmental Monitoring and Assessment Program; EPA, 1995b) are in SSV Report Sections 1 and 5.3.3.2

Contamination may affect the productivity of benthic communities at tPAH concentrations above 4 mg/kg (shaded in Table C-1). As a result, the contribution of biomass to the food chain from areas with greater than 4 mg/kg tPAH may be reduced. Therefore, fish are likely to feed on prey from areas with less tPAH and subsequently, as shown in Table C-1, less mercury. The mean mercury concentration at all REMAP (PAH) sampling sites up to concentrations where the food chain may begin to be impaired by tPAHs (i.e. tPAH < 4 mg/kg) is 0.047 mg/kg (geometric mean = 0.026; median = 0.023 mg/kg). This analysis suggests that a reasonable estimate of the mean sediment concentration in areas where fish feed may be less than the overall REMAP geometric mean mercury concentration of 0.078 mg/kg, and possibly closer to 0.047 mg/kg.

The geometric mean mercury concentration of REMAP sediment data (0.078 mg/kg) was used in this document to calculate a waterbody BSAF for 20 inch walleyes (and the SSV). This resulted in a waterbody BSAF of 8.2. When similar methods are used to calculate a BSAF for 30 inch northern pike, the results (6.2 wet weight BSAF; ~ 25 estimated dry weight BSAF (SSV Report 5.3.3.3)) are similar to BSAFs found in the literature (10.1 - 45.7 calculated from dry fish tissue weight for northern pike of unpublished length; EPA, 1997c). Use of a lower estimated effective mercury concentration of 0.047 mg/kg (calculated above) suggests larger

BSAFs of 13.6 for 20 inch walleyes and 10.3 for 30 inch northern pike and a reduction of the calculated mercury SSV from 0.021 to 0.013 mg/kg. A BSAF of 10.3 (~ 41.2 adjusting for dry tissue; see SSV Report 5.3.3.3) is at the high end of the range of published BSAFs for northern pike.

Table C-1 shows that the arithmetic mean, geometric mean and median mercury concentrations (EPA, 1995b) in areas with minimal tPAH contamination (0.25 mg/kg) are 0.012-0.013 mg/kg. These data support the assumption in the SSV Report that background mercury concentrations in the St. Louis River Estuary are at or below 0.02 mg/kg.

Appendix D - Chemical-specific variable defaults

Table D-1a - Chemical-specific Values

		CAS No.	MW	K_H	K_{oc}	Log K_{ow}	BSAF *(BCF)* mg/kg$_{fish}$ / mg/kg$_{sed}$	
			g/mol	atm-m^3/mol	L/kg	Unitless	Value Used	Range
Metals - Inorganics								
Arsenic		7440-38-2						
Cadmium		7440-43-9						
Chromium III		16065-83-1						
Chromium VI		18540-29-9						
Copper		7440-50-8						
Cyanide		57-12-5						
Lead		7439-92-1						
Mercury	(inorganic in sediment; methylmercury in fish)	7439-97-6		0.0114 [a]				
Methyl Mercury		22967-92-6					8.2 [f]	3.0 - 12.2 [f]
Nickel		various						
Zinc		7440-66-6						
Volatile organic compounds (VOCs)							*mg/kg $_{fish\ lipid}$ / mg/L*	
Benzene		71-43-2	78.1	5.5E-03 [a]	66 [a]	2.13 [a]	*(320)* [g]	
Ethyl benzene		100-41-4	106	7.9E-03 [a]	210 [a]	3.14 [a]	*(1900)* [g]	
Styrene		100-42-5	104	2.8E-03 [a]	910 [a]	2.94 [a]	*(1300)* [g]	
Toluene		108-88-3	92.1	6.6E-03 [a]	150 [a]	2.75 [a]	*(950)* [g]	
Xylenes (mixed)		1330-20-7	106	7.3E-03 [b]	410 [b]	3.2 [c]	*(2000)* [g]	
Polycyclic Aromatic Hydrocarbons (PAHs)							mg/kg$_{fish\ lipid}$ / mg/kg$_{organic\ carbon}$	
Acenaphthene		83-32-9	154	1.6E-04 [a]	5000 [a]	3.9 [a]	0.55 [h]	0.083 - 2.3 [i]
Acenaphthylene (toxicity surrogate - acenaphthene)		208-96-8	152	1.1E-04 [c]	3500 [d]	3.9 [c]	0.55 [h]	0.05 - 0.6 [i]
Anthracene		120-12-7	178	6.5E-05 [a]	24000 [a]	4.6 [a]	0.05 [h]	0.05 - 0.6 [i]
Benzo(a)pyrene equivalents (B[a]P-PEQs)		50-32-8	252	1.1E-06 [a]	1200000 [a]	6.1 [a]	0.11 [h]	0.02 - 0.29 [i]
Fluoranthene		206-44-0	202	1.6E-05 [a]	49000 [a]	5.1 [a]	0.11 [h]	0.05 - 0.29 [i]
Fluorene		86-73-7	170	6.4E-05 [a]	8900 [a]	4.2 [a]	0.083 [h]	0.083 - 0.6 [i]
Methylnaphthalene (toxicity surrogate - naphthalene)		1321-94-4	142	5.8E-04 [c]	6800 [d]	3.7 [c]	0.55 [h]	0.29 - 2.3 [i]
Naphthalene		91-20-3	128	4.8E-04 [a]	1200 [a]	3.4 [a]	0.35 [h]	0.29 - 2.3 [i]
Perylene (toxicity surrogate - pyrene)		198-55-0	252	3.7E-06 [c]	390000 [e]	6.3 [c]	0.11 [h]	0.05 - 0.29 [i]
Phenanthrene (toxicity surrogate - anthracene)		85-01-8	178	2.6E-05 [c]	33000 [d]	4.5 [c]	0.083 [h]	0.1 - 0.6 [i]
Pyrene		129-00-0	202	1.1E-05 [a]	71000 [a]	5.1 [a]	0.11 [h]	0.05 - 0.29 [i]
Dibenzo-p-dioxins/dibenzofurans								
2,3,7,8-TCDD equivalents (2,3,7,8-TCDD-TEQs)		1746-01-6	322	9.2E-06 [d]	8300000 [d]	7 [d]	1 [j]	0.003 - 2.268 [i]
Other Organics								
Carbazole		86-74-8	167	8.7E-08 [c]	3400 [a]	3.6 [a]	1 [k]	Default
Dibenzofuran (unsubstituted)		132-64-9	168	1.3E-05 [b]	7800 [b]	4.1 [c]	1 [k]	Default
Hexachlorobenzene		118-74-1	285	1.3E-03 [a]	80000 [a]	5.9 [a]	0.1 [j]	0.057 - 0.105 [i]
Octachlorostyrene		29082-74-4	380	2.3E-04 [c]	14000000 [e]	7.5 [c]	0.98 [l]	
PCBs (Polychlorinated Biphenyls)		1336-36-3	268 [d]	4.2E-04 [d]	310000 [d]	5.6 [d]	2.6 [h]	1.392-4.134 [i]

K_H - Henry's Law Constant
K_{OC} - Organic carbon partitioning constant
K_{OW} - Octanol-water partitioning constant
BSAF - Biota-sediment accumulation factor
BCF - Biota-sediment concentration factor

References:

[a] (EPA, 1996a)
[b] (EPA, 2002a)
[c] (Syracuse Research Corporation, 2003)
[d] (Michigan DEQ, 2002)
[e] Calculated (SSV Report Section 5.2; Equation #3)
[f] MDH calculation (see SSV Report Section 5.3.3.3)
[g] BCF Calculated (SSV Report Section 5.3.2; Equation #5)
[h] (Washington State Department of Health, 1995, 1996)
[i] (EPA, 1997d; Washington State Department of Health, 1995; Washington State Department of Ecology, 1997)
[j] (Washington State Department of Ecology, 1997)
[k] Default - (EPA, 1997d)
[l] (EPA, 1995a)

Table D-1b - Chemical-specific Values

	ABS_{GI}	ABS_{Sed}	ABS_{Derm}	K_p	FA	ABS_{SW} (mg/(cm^2·event)) / (mg/cm^3) (calculated)	AF_{ED} hr/event (calculated)
	Unitless	Unitless	Unitless	cm/hr	Unitless		
Metals - Inorganics							
Arsenic	1	1	0.03	0.001		0.0005	
Cadmium	0.025	1	0.001	0.001		0.0005	
Cadmium (values for drinking water)	0.05						
Chromium III	0.013	1		0.001		0.0005	
Chromium VI	0.025	1		0.002		0.001	
Copper	0.6	1		0.001		0.0005	
Cyanide	1	1		0.001		0.0005	
Lead				1E-04		0.00005	
Mercury (inorganic in sediment/SW; methylmercury in fish)	0.07	1		0.001		0.0005	
Methyl Mercury	1	1					
Nickel	0.04	1		2E-04		0.0001	
Zinc	1	1		6E-04		0.0003	
Volatile organic compounds (VOCs)							
Benzene	1	1		0.015	1	0.016	1.05
Ethyl benzene	1	1		0.049	1	0.061	1.25
Styrene	1	1		0.037	1	0.046	1.24
Toluene	1	1		0.031	1	0.036	1.15
Xylenes (mixed)	1	1		0.053	1	0.066	1.25
Polycyclic Aromatic Hydrocarbons (PAHs)							
Acenaphthene	1	1	0.13	0.12	1	0.2	1.71
Acenaphthylene (toxicity surrogate - acenaphthene)	1	1	0.13	0.089	1	0.15	1.69
Anthracene	1	1	0.13	0.12	1	0.24	2
Benzo(a)pyrene equivalents (B[a]P-PEQs)	1	1	0.13	0.7	1	2.3	3.22
Fluoranthene	1	1	0.13	0.22	1	0.51	2.33
Fluorene	1	1	0.13	0.08	1	0.15	1.9
Methylnaphthalene (toxicity surrogate - naphthalene)	1	1	0.13	0.047	1	0.074	1.58
Naphthalene	1	1	0.13	0.047	1	0.068	1.45
Perylene (toxicity surrogate - pyrene)	1	1	0.13	1.6	1	5.2	3.22
Phenanthrene (toxicity surrogate - anthracene)	1	1	0.13	0.14	1	0.28	2
Pyrene	1	1	0.13	0.21	1	0.49	2.33
Dibenzo-p-dioxins/dibenzofurans							
2,3,7,8-TCDD equivalents (2,3,7,8-TCDD-TEQs)	1	1	0.03	0.81	0.5	2	2.52
Other Organics							
Carbazole	1	1	0.1	0.025	1	0.046	1.86
Dibenzofuran (unsubstituted)	1	1	0.1	0.079	1	0.15	1.87
Hexachlorobenzene	1	1	0.1	0.13	0.9	0.47	3.58
Octachlorostyrene	1	1	0.1	0.17	1	1.2	7.34
PCBs (Polychlorinated Biphenyls)	1	1	0.14	0.54	0.5	0.97	1.79

Reference: (EPA, 2001a)

ABS_{Derm} = dermally absorbed fraction (unitless)
ABS_{GI} = fraction of applied dose absorbed in primary (RfD) study (unitless)
ABS_{Sed} = oral absorption adjustment - relative bioavailability (unitless)
FA = fraction absorbed from water (unitless)
K_p = permeability coefficient (cm/hr)
AF_{ED} = event duration-dependent adjustment factors. Calculated in Appendix H with *Equations #A-30 or #A-31*) (hr/event)
ABS_{SW} general term representing the dermally absorbed dose from a chemical concentration in water: dependent on event duration and chemical specific factors. Calculated in Appendix H with *Equation #A-20* or *Equation #A-21.* ((mg/cm^2/event)/(mg/cm^3))

Table D-2 - Toxicity Criteria

	Reference Dose (RfD)	Reference Concentration (RfC)	Cancer Slope Factor (SF_c)	Cancer Unit Risk (UR_c)
Metals - Inorganics	mg/(kg·d)	mg/m^3	$(mg/(kg \cdot d))^{-1}$	$(\mu g/m^3)^{-1}$
Arsenic	0.0003 [a]	0.00003 [g]	1.5 [a]	0.004 [a]
Cadmium	0.001 [a]	0.00002 [g]		0.0018 [l]
Chromium III	1.5 [a]			
Chromium VI	0.003 [a]	0.0001 [a]		0.012 [l]
Copper	0.037 [b]			
Cyanide	0.02 [a]			
Lead				
Mercury (inorganic in sediment/SW; methylmercury in fish)	0.0003	0.0003 [a]		
Methyl Mercury	0.0001 [a]			
Nickel	0.02 [a]	0.00005 [g]		
Zinc	0.3 [a]			
Volatile organic compounds (VOCs)				
Benzene	0.004 [a]	0.03 [a]	0.055 [a]	0.0000078 [l]
Ethyl benzene	0.1 [a]	1 [a]		
Styrene	0.2 [a]	1 [h]		
Toluene	0.04 [c]	0.4 [h]		
Xylenes (mixed)	0.2 [a]	0.1 [a]		
Polycyclic Aromatic Hydrocarbons (PAHs)				
Acenaphthene	0.06 [a]	0.21 [i]		
Acenaphthylene (toxicity surrogate - acenaphthene)	0.06 [a]	0.21 [i]		
Anthracene	0.3 [a]	1.1 [i]		
Benzo(a)pyrene equivalents (B[a]P-PEQs)			7.3 [a]	0.0011 [g]
Fluoranthene	0.04 [a]	0.14 [i]		
Fluorene	0.04 [a]	0.14 [i]		
Methylnaphthalene (toxicity surrogate - naphthalene)	0.02 [a]	0.003 [a]		
Naphthalene	0.02 [a]	0.003 [a]		
Perylene (toxicity surrogate - pyrene)	0.03 [a]	0.11 [i]		
Phenanthrene (toxicity surrogate - anthracene)	0.3 [a]	1.1 [i]		
Pyrene	0.03 [a]	0.11 [i]		
Dibenzo-p-dioxins/dibenzofurans				
2,3,7,8-TCDD equivalents (2,3,7,8-TCDD-TEQs)	2.3E-09 [d]	0.00000004 [g]	1400000 [k]	332 [m]
Other Organics				
Carbazole			0.02 [b]	4.74E-06 [m]
Dibenzofuran (unsubstituted)	0.004 [e]	0.0083 [j]		
Hexachlorobenzene	0.0008 [a]	0.0017 [j]	1.6 [a]	0.000379 [m]
Octachlorostyrene	0.00003 [f]	0.000062 [j]		
Polychlorinated Biphenyls (toxicity surrogate - Aroclor 1254)	0.00002 [a]	0.000042 [j]		

References:

a (EPA, 2003)
b (EPA, 1997b)
c (EPA, 2002b)
d (FAO/WHO, 2001)
e (EPA, 1999)
f (New York State DEC, 1997)
g (CA OEHHA, 2003, 2002)
h (MDH, 2002)
i (EPA, 1996b)
j Route-to-route (chronic: see *Equation #A-14*, below)
k (MDH, 2003)
l (MDH, 2002; EPA, 2003)
m Route-to-route (cancer: see *Equation #A-15*, below)
n Calculated from (EPA, 2001b)

Route-to-route extrapolation of chronic inhalation criteria were determined from:

$RfC \{ mg/m^3 \} = RfD * ncBW / ncInhRate$ *Equation #A-14.*
$= RfD * 16.2 kg / 8.0 m^3/d$

Where:
ncBW from EPA Exposure Factors Handbook (EPA, 1997a) (SSV Report Table #1; Section 3)
ncInhRate from EPA Exposure Factors Handbook (EPA, 1997a).

Route-to-route extrapolation of cancer inhalation criteria were determined from:

$UR_c \{ (\mu g/m^3)^{-1} \} = SF_c / cBW * cInhRate$ *Equation #A-15.*
$= SF_c / 51.0 kg * 12.5 m^3/d$

Where:
cBW from EPA Exposure Factors Handbook (EPA, 1997a) (SSV Report Table #1; Section 3)
cInhRate from EPA Exposure Factors Handbook (EPA, 1997a).

Appendix E - Screening Detection Limits for PBTs in Sediment and Fish Tissue

Detection limits sufficient for screening sediment contamination for potential human health impacts are listed in Tables E-1a and E-1b. Sediment detection limits were calculated from the Sediment Screening Values and include 10X to 30X adjustments to allow for assessment of additivity of health impacts. Detection limits for analysis of fish tissue are calculated directly from health criteria and assumed ingestion rates (with adjustment for possible additivity). Use of analytical detection limits at and below recommended values at most sites will allow toxic endpoint additivity to be addressed. For some of the chemicals, lower detection limits may needed to characterize risks to aquatic organisms and the environment.

Table E-1a Recommended Sediment and Fish Tissue Detection Limits

Chemicals		Recommended (calculated) DLs			
		Sediment		Fish Tissue	
Metals - Inorganics (~ 10X below HI=1 or 10^{-6} cancer risk*)		mg/kg		mg/kg	
	Arsenic *	3		0.003	
	Cadmium	10	#	0.2	
	Chromium III	40000	#	300	#
	Chromium VI	70	#	0.7	
	Copper	900	#	9	#
	Cyanide	500	#	5	#
	Lead	40	#	0.8	
	Mercury	0.002		0.02	
	Nickel	500	#	5	#
	Zinc	7000	#	70	#
Volatile organic compounds (VOCs) (~ 10X below HI=1 or 10^{-6} cancer risk *)					
	Benzene *	0.0003		0.09	
	Ethyl benzene	0.03		20	#
	Styrene	0.4		50	#
	Toluene	0.01		9	#
	Xylenes (mixed)	0.008		50	#
Polycyclic Aromatic Hydrocarbons (PAHs) (~ 30X below HI=1)					
	Acenaphthene	0.7		5	#
	Acenaphthylene	0.7		5	#
	Anthracene	20	#	20	#
	Fluoranthene	3		3	
	Fluorene	2		3	
	Methylnaphthalene	0.02		2	
	Naphthalene	0.004		2	
	Perylene	2		2	
	Phenanthrene	20	#	20	#
	Pyrene	3		2	
Other Organics (~ 30X below HI=1 or 10^{-6} cancer risk *)					
	Carbazole *	0.09		0.08	
	Dibenzofuran	0.06		0.3	
	Hexachlorobenzene *	0.005		0.001	
	Octachlorostyrene	0.0006		0.002	
	tPCBs (Polychlorinated Biphenyls)	0.0002		0.002	

- No need for special detection limits / recommendations. Use detection limits recommended for ecological assessment.

Table E-1b Recommended Sediment and Fish Tissue Detection Limits

Chemicals				Recommended (calculated) DLs	
				Sediment	**Fish Tissue**
Benzo[a]pyrene Potency Equivalents (~ 3X below 10^{-6} cancer risk)			PEF	mg/kg	mg/kg
Benzo(a)pyrene	Dibenzo[a,e]pyrene	7H-Dibenzo[c,g]carbazole	1	2E-03	2E-04
	5-Methylchrysene	1,8-Dinitropyrene			
		7,12-dimethylbenzanthracene	30	8E-05	7E-06
Dibenzo[a,h]pyrene	Dibenzo[a,i]pyrene	Dibenzo[a,l]pyrene	10	2E-04	2E-05
	1,6-Dinitropyrene	6-Nitrochrysene			
		3-methylcholanthrene	3	8E-04	7E-05
		Dibenz[a,h]anthracene	0.6	4E-03	4E-04
Benz[a]anthracene	Benzo[b]fluoranthene	Benzo[j]fluoranthene	0.1	2E-02	2E-03
	Benzo[k]fluoranthene	Dibenz[a,j]acridine			
	Dibenz[a,h]acridine	Indeno[1,2,3-c,d]pyrene			
	1-Nitropyrene	4-Nitropyrene			
		5-nitroacenaphthene	0.02	1E-01	1E-02
	2-Nitrofluorene	Chrysene	0.01	2E-01	2E-02
2,3,7,8-TCDD-TEQs (~ 30X below risk calculated @ background)			TEF		
	2,3,7,8-TCDD	1,2,3,7,8-PentaCDD	1	3E-07	2E-07
		2,3,4,7,8-PentaCDF	0.5	7E-07	4E-07
1,2,3,4,7,8-HexaCDD/F	1,2,3,6,7,8-HexaCDD/F	1,2,3,7,8,9-HexaCDD/F	0.1	3E-06	2E-06
2,3,4,6,7,8-HexaCDF	2,3,7,8-TetraCDF	PCB 126			
		1,2,3,7,8-PentaCDF	0.05	7E-06	4E-06
	1,2,3,4,6,7,8-HeptaCDD/F	1,2,3,4,7,8,9-HeptaCDF	0.01	3E-05	2E-05
		PCB 169			
		Octa-CDDD/F	0.001	3E-04	2E-04
		PCBs 114,156,157	0.0005	7E-04	4E-04
		PCBs 77,81,105,118,123,189	0.0001	3E-03	2E-03
		PCB 167	0.00001	3E-02	2E-02

PEF - potency equivalency factor (MDH, 2001a)
TEF - toxic equivalency factor (MDH, 2003)

Appendix F - Additional information on default TOC

In first calculations without the benefit of site-specific data, total organic carbon (TOC) in littoral river sediment is often assumed to be about 2%. This value has been used as the default TOC throughout this document. Non-polar organic compounds sorb to organic carbon in sediment (OC). This affinity controls the partitioning of these compounds between sediment and water, as well as the availability of these compounds to aquatic organisms (bioavailability). REMAP data (Regional Environmental Monitoring and Assessment Program; EPA, 1995b) suggest that the average ambient TOC throughout the estuary is about 3.5 % OC. It is often suggested that some OC in highly contaminated areas is the organic carbon in the contaminants. This would require extremely high contamination to increase the TOC 1% (10,000 ppm = 1%). Regardless, in areas of sufficiently high contamination, the food chain is likely affected by the lack of benthic communities or suppression of phytoplankton and zooplankton communities. These effects may be seen in areas where sediment concentrations of contaminants exceed Lowest Effect Levels (LELs). The Ontario Ministry of the Environment LEL for tPAHs is 4 mg/kg (1993). Table F-1 shows tPAH concentrations as a surrogate for contamination effects on the food chain.

Table F-1: Relationship between REMAP TOC and tPAH data

			Ontario (OMOE) LEL - tPAH = 4 mg/kg							all REMAP (class 1,2) locations
REMAP sites w/tPAH	<0.25 mg/kg	<0.5 mg/kg	<1 mg/kg	<2 mg/kg	<4 mg/kg	<8 mg/kg	<16 mg/kg	<32 mg/kg	All tPAH sites	All TOC sites
# of sites (n=)	8	12	19	23	27	29	37	40	42	87
TOC Mean	0.3%	0.4%	0.9%	1.1%	2.1%	2.0%	3.0%	3.4%	3.4%	3.5%
Mean CL (5-95%)	± 0.12%	± 0.23%	± 0.62%	± 0.62%	± 1.45%	± 1.35%	± 1.47%	± 1.46%	± 1.39%	± 0.84%

Note: Class 1 sites accounted for 61-75% of sites in each tPAH category.

At tPAH concentrations in sediments below 4 mg/kg, food chain effects are unlikely. Above 4 mg/kg there may be changes in the local food chain, and some areas may become non-productive. Note that as the tPAH concentration in sediment increases, the mean TOC also increases. In addition, note that the mean TOC for samples below 8 mg/kg tPAH is below 2%, and that the median TOC for all samples analyzed for PAHs is 2%. These data suggest that 2% TOC is a reasonable default when considering partitioning and bioavailability of non-polar organic compounds in the St. Louis River Estuary.

Appendix G - PAH BSAFs and Washington State Department of Health PAH BSAFs

In order to model the effect of sediment contamination on the contaminant concentration in edible fish tissue a biota-sediment accumulation factor is needed. Availability of BSAF data is often limited. Even when data are available, fish BSAF data are often calculated for whole fish and not from fish fillet data. Therefore, the BSAF data may not be relevant. Also, a BSAF in one water body may not be equivalent to a similarly calculated BSAF for another water body. In addition there are often differences in site characteristics that can lead to changes in actual BSAFs. These may include: differences between the aquatic species measured and the species of concern; different places in the food chain are occupied by the same species at different sites; differences in the age or size of the species of interest; and, differences in lipid content in the species of interest. BSAF data for polycyclic aromatic hydrocarbons are further limited because most fish species can metabolize PAHs, and the metabolizing enzymes can often be induced leading to more rapid metabolism of PAHs in exposed fish.

Two different PAH BSAF schemes were reviewed for this document. EPA has recommended using a BSAF of 0.29 for all PAHs (EPA, 1997d). This BSAF was developed from aquatic organisms (primarily benthic organisms) including non-fish species that may not efficiently metabolize PAHs. Using this BSAF to estimate PAH concentrations in fish tissue is therefore likely to be over-protective.

The Washington State Department of Health (WA DOH) (Washington State Department of Health, 1995, 1996) has also developed BSAFs for PAHs. WA DOH BSAFs are based on fish fillet data from several fish species. Their model uses different BSAFs for PAHs with different K_{ow}s as shown in Table G-1 below. The WA DOH recommends using the 75% confidence limit (CL) of the mean BSAF as a conservative estimate of the BSAF.

Table G-1: Washington DOH Recommended PAH BSAFs

From WA Dept of Health review of fillet data in literature: forage and piscivorous fish					
Log K_{ow}	Mean	Median	**75% CL**	90% CL	n
< 3.0	0.136	0.075	**0.228**	0.443	10
3.0 - 3.49	0.522	0.011	**0.35**	1.019	33
3.5 - 3.99	0.716	0.1	**0.548**	4.394	13
4.0 - 4.49	0.119	0.023	**0.083**	0.495	18
4.5 - 4.99	0.033	0.033	**0.05**	0.084	17
> 4.99	0.59	0.018	**0.105**	0.66	29

To evaluate the applicability of these approaches to the St. Louis River Estuary, available fish and sediment data for the lower St. Louis River were reviewed. The MDH reviewed PAH fish fillet data collected from the estuary in 2000 (MDH, 2001b). Unfortunately, detection limits were too high and no PAHs were found above the detection limits. Appendix E recommends detection limits for the 7 cPAHs analyzed that are 2.5 (chrysene) to 250 times (benzo[a]pyrene) lower than the detection limits in this study.

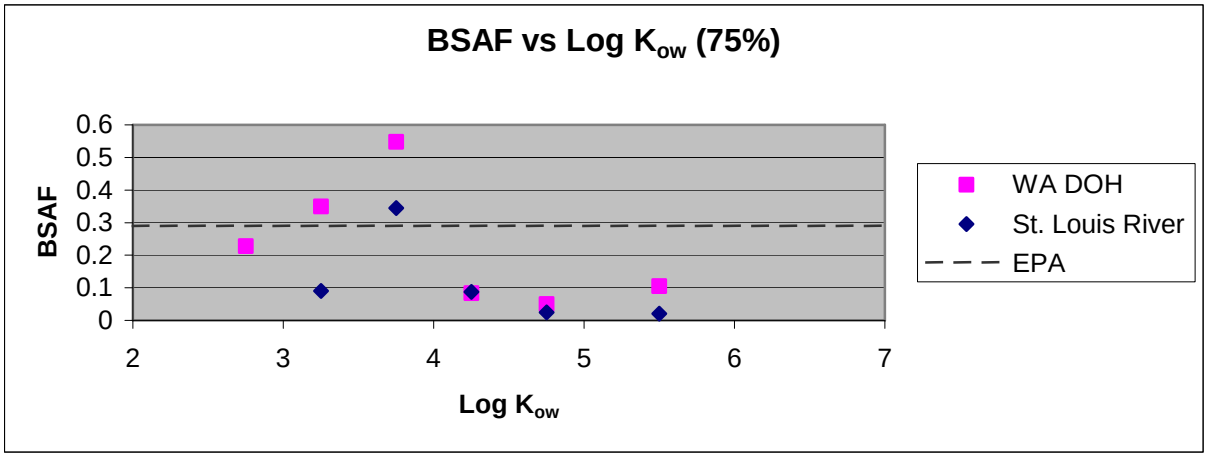
A limited amount of whole fish PAH data are available from the St. Louis River. The US Fish and Wildlife Service (US FWS, 2002) collected white suckers, northern pike and shorthead redhorse from 3 areas of the St. Louis River. Individual fish from all 3 species are assumed to range over the estuary. Therefore, they may ingest contaminants with prey in all biologically-productive areas of the river. Data describing PAH concentrations throughout the estuary were collected in the EPA REMAP study (Regional Environmental Monitoring and Assessment Program; EPA, 1995b). BSAFs calculated with USFWS (2002) fish data and REMAP sediment data from the St. Louis River are shown in Table G-2. (Fish with the highest whole-body concentration from the USFWS study, likely would have had fillet PAH concentrations (with about 1/3 the lipid found in whole fish) that were 1¼ (naphthalene) to 15 times (methylnaphthalene) less than detection limits in the fillet study cited above (11 PAHs only).)

Table G-2: Calculated St. Louis River PAH BSAFs (g_{oc}/g_{lipid})

From St. Louis River: FWS 2001 Whole Fish data / REMAP (class 1,2) mean sediment concentration					
Log K_{ow}	Mean	Median	**75%**	90%	n (chemicals)
< 3.0	-	-	**-**	-	0
3.0 - 3.49	0.059	0.041	**0.090**	0.111	1
3.5 - 3.99	0.275	0.243	**0.345**	0.395	2
4.0 - 4.49	0.065	0.037	**0.087**	0.106	2
4.5 - 4.99	0.025	0.024	**0.025**	0.028	1
> 4.99	0.020	0.015	**0.020**	0.029	3

BSAFs calculated using these data from whole fish do support the use of the Washington DOH method for calculating BSAFs. In Figure G-1, note that predicted BSAFs (WA DOH method) and St. Louis River BSAFs data in Table G-2 are in reasonable agreement.

Figure G-1: 75th percent Washington DOH and St. Louis River PAH BSAFs as a function of Log K_{ow}



Appendix H - Route-specific sediment value calculations - Non-cancer

Variables are defined in Appendix A. Default and chemical-specific values are listed in tables found in Appendix D. Calculated default exposures are shown in Table B-1. Route-specific sediment values derived in this appendix (non-cancer endpoint) are shown in Table H-1.

Calculating protective sediment concentrations for ingestion route exposure

Sediment ingestion

If an individual is exposed only because they ingest sediment, screening values for non-cancer endpoints would be:

$SSV_{Sed(Ing)}$ { mg/kg } = $HQ*RfD/(ABS_{Sed}*Sed_{Ing}) * CF_{mg/kg}$ *Equation #A-16.*

Calculation results are shown in Table H-1.

Water ingestion

A protective surface water concentration for a chemical can be calculated from:

$SWC_{SW(Ing)}$ { mg/L } = $HQ * RfD / SW_{Ing}$ *Equation #A-17.*

Section 5.2 of the SSV Report addressed the partitioning of chemicals between sediment and water. As noted, partitioning and water concentrations for screening were not calculated for metals. When calculating sediment screening values for organic compounds, it is assumed that the sediments are the source of all of the contaminant in the water column and that there is an equilibrium between surface water and sediments.

Sediment values dependent on equilibrium partitioning between sediment and water, and water ingestion alone are calculated by substituting *SSV Report Eq. #2 (5.2)* into *Eq. #A-16*:

$SSV_{SW(Ing)}$ { mg/kg } = $HQ * RfD * K_{oc} * f_{oc} / SW_{Ing}$ *Equation #A-18.*

Calculation results are shown in Table H-1.

Calculating protective sediment concentrations for dermal route exposure

Dermal exposure to sediment

Dermal uptake of contaminants directly from sediment is a function of contaminant concentration in the sediment, sediment loading or adherence to the skin, and the fraction of chemicals in contact with the skin that actually can traverse into the blood (absorption, diffusion). Values developed for non-cancer effects of sediment-dermal exposure alone will be:

$SSV_{Sed(Derm)}$ { mg/kg } = $HQ * RfD * ABS_{GI} / (ABS_{Derm}* Sed_{Derm}) * CF_{mg/kg}$ *Equation #A-19.*

Calculation results are shown in Table H-1.

Screening values for dermal exposure to sediment are not adjusted for chemical transfer time dependence because it is assumed that the exposure time is sufficient to allow the transfer of

the absorbed fraction to the skin. In addition, there is no adjustment for sediment particle size or the fraction organic carbon in sediment.

Dermal exposure to water

Dermal uptake of contaminants directly from water is a function of contaminant concentration in the sediment, equilibrium partitioning into water, the duration of any single activity, and the permiability of the skin to the chemical. Water values developed for non-cancer effects of water-dermal exposure alone will be:

$$SWC_{SW(Derm)} \{ mg/L \} = HQ * RfD * ABS_{GI} / (ABS_{SW} * SW_{Derm}) * CF_{cm3/L}$$ *Equation #A-20.*

Note that this is a simplified equation because the model assumes all event durations (swimming and wading) are the same. Site-specific application may require the use of different event durations. If event durations are different, ABS_{SW} (below) and SW_{Derm} should be calculated for each assumed exposure duration (ABS_{SW1}, ABS_{SW2}, ..., ABS_{swn}; SW_{Derm1}, SW_{Derm2}, ..., SW_{Dermn}) and *Equation #A-20* becomes:

$$SWC_{SW(Derm)} \{ mg/L \} = HQ * RfD * ABS_{GI} / (ABS_{SW1} * SW_{Derm1} + ABS_{SW2} * SW_{Derm2} +... + ABS_{swn} * SW_{Dermn}) * CF_{cm3/L}$$ *Equation #A-20a*

Sediment values dependent on equilibrium partitioning between sediment and water, and dermal-water exposure alone are calculated by substituting *Eq. #2 (SSV Report)* into *Eq. #A-20*:

$$SSV_{SW(Derm)} \{ mg/kg \} = HQ * RfD * ABS_{GI} * K_{oc} * f_{oc} / (ABS_{SW} * SW_{Derm}) * CF_{cm3/L}$$ *Equation #A-21.*

Unlike dermal absorption from sediment, which is a function of sediment adherence and a chemical-specific absorption fraction (ABS_{Derm}), dermal absorption from water is dependent on the event duration and individual chemical characteristics that effect chemical transfer and diffusion through the skin. Equations used to derive dermal exposure relationships are adapted from the EPA Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment) (EPA, 2001a).

The absorbed dose from exposure to dissolved metals is predicted by:

$$ABS_{SW\text{-}met} \{ mg/(cm^2 \cdot event))/(mg/cm^3 \} = K_p * ED$$ *Equation #A-22.*

For organics, dermal absorption from water is adjusted by additional factors to account for the time to equilibrium between chemical dissolved in water and in skin as well as chemical loss due to desquamation. An estimate of the dermally available dose from exposure to organics dissolved in water is calculated from the following equations:

$$ABS_{SW\text{-}org} \{ mg/(cm^2 \cdot event))/(mg/cm^3 \} = K_p * AF_{ED}$$ *Equation #A-23.*

Calculations of event duration-dependent factors are chemical specific:

$$\tau \{ hr/event \} = 0.105*10^{(0.0056 * MW)}$$ *Equation #A-24.*

β { unitless } = K_p * $\sqrt{MW}$ / 2.6 *Equation #A-25.*

If: $\beta \leq 0.6$

then: t^* { hr } = 2.4 * τ *Equation #A-26.*

If: $\beta > 0.6$

then: t^* { hr } = 6 * τ (b - $\sqrt{(b^2 - c^2)}$) *Equation #A-27.*

Where:

$b = 2 * (1 + \beta)^2 / \pi - c$ *Equation #A-28.*

$c = (1 + 3 * \beta + 3 * \beta^2) / (3 * (1 + \beta))$ *Equation #A-29.*

If: ED $\leq t^*$

then: AF_{ED} { hr/event } = 2 * FA * $\sqrt{(6 * \tau * ED / \pi)}$ *Equation #A-30.*

If: ED > t^*

then: AF_{ED} { hr/event } = FA* (ED / (1 + β) + 2 * τ * (1 + 3 * β + 3 * β^2) / $(1 + \beta)^2$)
Equation #A-31.

Calculation results are shown in Table H-1.

Calculating protective sediment concentrations for inhalation exposure

Estimates of potential air concentrations above contaminated sediments can be calculated using equations from SSV Report Section 5.2 on equilibrium partitioning. While these estimates may be realistic for air overlying a slurry of sediment and water, as the size of the disturbed area or amount of disturbance (suspension) decrease, and the depth of the water or air movement (wind) increase, any exposures will tend to decrease. Therefore, inhalation exposure calculations likely overestimate exposures at most sites, and should only be used for screening and for determining potentially important routes-of-exposure to individual chemicals.

SSV_{Inh} { mg/kg } = HQ * RfC * K_{oc} * f_{oc} * R * T / (Inh_{frac} * K_H * $CF_{L/m3}$ * $CF_{L/m3}$) *Equation #A-32.*

Calculation results are shown in Table H-1.

Calculating sediment screening values for fish ingestion route exposure

For organics (excluding volatiles):

SSV_{Fish} { mg/kg } = HQ * RfD * f_{oc} / (BSAF * $Fish_{Ing}$ * f_{lipid}) * $CF_{g/kg}$ *Equation #A-33.*

For volatile organics:

SSV_{Fish} { mg/kg } = HQ * RfD * K_{oc} * f_{oc} / (BCF * $Fish_{Ing}$ * f_{lipid}) * $CF_{g/kg}$ *Equation #A-34.*

For inorganics and mercury:

SSV_{Fish} { mg/kg } = HQ * RfD / (BSAF * $Fish_{Ing}$) * $CF_{g/kg}$ *Equation #A-35.*

Calculation results are shown in Table H-1.

Table H-1: Calculated route-specific Sediment Screening Values

Chronic (non-cancer) Endpoint								
	Cas No.	Sediment Ingestion	Surfacewater Ingestion	Dermal Sediment	Dermal Surfacewater	Inhalation	Fish Consumption
		$SSV_{Sed(Ing)}$	$SSV_{SW(Ing)}$	$SSV_{Sed(Derm)}$	$SSV_{SW(Derm)}$	SSV_{Inh}	SSV_{Fish}
Metals - Inorganics		mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg
Arsenic	7440-38-2	73	*Not Evaluated*	137	*Not Evaluated*	*Not Evaluated*	*Not Evaluated*
Cadmium	7440-43-9	240		342			
Chromium III	16065-83-1	370000		*Not Evaluated*			
Chromium VI	18540-29-9	730					
Copper	7440-50-8	9000					
Cyanide	57-12-5	4900					
Lead *	7439-92-1	*Not Evaluated*					
Mercury (inorganic in sediment;	7439-97-6	73					
Methyl Mercury methylmercury in fish)	22967-92-6						0.0206
Nickel	various	4900					*Not Evaluated*
Zinc	7440-66-6	73000					
Volatile organic compounds (VOCs)							
Benzene	71-43-2	980	0.477	*Not Evaluated*	0.608	0.00504	0.507
Ethyl benzene	100-41-4	24000	37.4		12.2	0.367	6.79
Styrene	100-42-5	49000	329		144	4.56	84.9
Toluene	108-88-3	9800	10.5		5.91	0.123	3.76
Xylenes (mixed)	1330-20-7	49000	148		44.6	0.0786	26
Polycyclic Aromatic Hydrocarbons (PAHs)							
Acenaphthene	83-32-9	15000	545	6320	53.6	95.4	67.7
Acenaphthylene (toxicity surrogate - acenaphthene)	208-96-8	15000	375	6320	50.1	89.2	67.7
Anthracene	120-12-7	73000	13200	31600	1120	5770	3710
Benzo(a)pyrene equivalents (B[a]P-PEQs)	50-32-8	*Not Evaluated*					
Fluoranthene	206-44-0	9800	3570	4210	140	6020	236
Fluorene	86-73-7	9800	643	4210	85.4	274	298
Methylnaphthalene (toxicity surrogate - naphthalene)	1321-94-4	4900	246	2110	66.4	0.494	22.6
Naphthalene	91-20-3	4900	44.5	2110	13.1	0.107	35.3
Perylene (toxicity surrogate - pyrene)	198-55-0	7300	21100	3160	80.9	164000	177
Phenanthrene (toxicity surrogate - anthracene)	85-01-8	73000	18000	31600	1290	20000	2240
Pyrene	129-00-0	7300	3840	3160	157	9910	177
Dibenzo-p-dioxins/dibenzofurans							
2,3,7,8-TCDD equivalents (2,3,7,8-TCDD-TEQs)	1746-01-6	5.60E-04	3.46E-02	1.05E-03	3.39E-04	0.507	1.42E-06
Other Organics							
Carbazole	86-74-8	*Not Evaluated*					
Dibenzofuran (unsubstituted)	132-64-9	980	56.4	548	7.61	69.8	2.47
Hexachlorobenzene	118-74-1	200	116	110	4.98	1.41	4.95
Octachlorostyrene	29082-74-4	7.3	759	4.11	12.4	53.1	0.0189
PCBs (Polychlorinated Biphenyls)	1336-36-3	4.9	11.1	1.96	0.229	0.424	0.00476

* 400 mg/kg = EPA soil lead number also used for screening soils in Minnesota.

Appendix I - Route-specific sediment value calculations - Cancer

Variables are defined in Appendix A. Default and chemical-specific values are listed in tables found in Appendix D. Calculated default exposures are shown in Table B-1. Route-specific sediment values derived in this appendix (cancer endpoint) are shown in Table I-1.

Calculating protective sediment concentrations for ingestion route exposure - Cancer

Sediment ingestion

$SSV_{Sed(Ing)-c}$ { mg/kg } = ($AccptRsk_c$ / SF_c) / (ABS_{sed}* Sed_{Ing-c}) * $CF_{mg/kg}$ *Equation #A-36.*

Calculation results are shown in Table I-1.

Water ingestion

A protective surface water concentration for cancer endpoints and the water ingestion pathway can be calculated from:

$SWC_{SW(Ing)-c}$ { mg/L } = ($AccptRsk_c$ / SF_c) / SW_{Ing-c} *Equation #A-37.*

A protective sediment concentration for cancer endpoints and the water ingestion pathway can be calculated from:

$SSV_{SW(Ing)-c}$ { mg/kg } = ($AccptRsk_c$ / SF_c) * K_{oc} * f_{oc} / SW_{Ing-c} *Equation #A-38.*

Calculation results are shown in Table I-1.

Calculating protective sediment concentrations for dermal exposure - Cancer

Dermal exposure to sediment

The screening values developed for the cancer effects of dermal-sediment exposure alone are:

$SSV_{Sed(Derm)-c}$ { mg/kg } = ($AccptRsk_c$ / SF_c) * ABS_{GI} / (ABS_{Derm} * Sed_{Derm-c}) * $CF_{mg/kg}$ *Equation #A-39.*

Calculation results are shown in Table I-1.

Dermal exposure to surface water

The screening values developed for the cancer effects of dermal-water exposure alone are:

$SWC_{SW(Derm)-c}$ { mg/L } = ($AccptRsk_c$ / SF_c) * ABS_{GI} / (ABS_{SW} * SW_{Derm-c}) * $CF_{cm3/L}$ *Equation #A-40.*

Note: if site-specific evaluations require multiple event durations, *Equation #A-40* (and *#A-41* below) should be adjusted as *Equation #A-20* above (Appendix H).

Sediment screening values dependent on equilibrium partitioning between sediment and water, and dermal-water exposure alone are calculated by substituting *SSV Report Eq. #2* (5.1) into *Eq. #A-39*:

$SSV_{SW(Derm)-c}$ { mg/kg } = ($AccptRsk_c$ / SF_c) * ABS_{GI} * K_{oc} * f_{oc} / (ABS_{SW} * SW_{Derm-c}) * $CF_{cm3/L}$ *Equation #A-41.*

Calculation results are shown in Table I-1.

Calculating protective sediment concentrations for inhalation exposure - Cancer
Estimates of potential air concentrations above contaminated sediments can be calculated using equations from the above section on equilibrium partitioning. While these estimates may be realistic for air overlying a slurry of sediment and water, as the size of the disturbed area or amount of disturbance (suspension) decrease, and the depth of the water or air movement (wind) increase, any exposures will tend to decrease. Therefore, inhalation exposure calculations likely overestimate exposures at most sites, and should only be used for screening and for determining potentially important routes-of-exposure to individual chemicals.

$$SSV_{Inh\text{-}c}\ \{\ mg/kg\ \} = (AccptRsk_c / UR_c) * K_{oc} * f_{oc} * R * T / (Inh_{frac\text{-}c} * K_H * CF_{L/m3} * CF_{L/m3} * CF_{\mu g/mg}) \qquad \textit{Equation \#A-42.}$$

Calculation results are shown in Table I-1.

Calculating sediment screening values for fish ingestion exposure - Cancer
For organics (excluding volatiles):

$$SSV_{Fish\text{-}c}\ \{\ mg/kg\ \} = (AccptRsk_c / SF_c) * f_{oc} / (BSAF * Fish_{Ing\text{-}c} * f_{lipid}) * CF_{g/kg} \qquad \textit{Equation \#A-43.}$$

For volatile organics:

$$SSV_{Fish\text{-}c}\ \{\ mg/kg\ \} = (AccptRsk_c / SF_c) * K_{oc} * f_{oc} / (BCF * Fish_{Ing\text{-}c} * f_{lipid}) * CF_{g/kg} \qquad \textit{Equation \#A-44.}$$

For inorganics:

$$SSV_{Fish\text{-}c}\ \{\ mg/kg\ \} = (AccptRsk_c / SF_c) / (BSAF * Fish_{Ing\text{-}c}) * CF_{g/kg} \qquad \textit{Equation \#A-45.}$$

Calculation results are shown in Table I-1.

Table I-1: Route-specific Sediment Screening Values

Cancer Endpoint							
	Cas No.	Sediment Ingestion	Surfacewater Ingestion	Dermal Sediment	Dermal Surfacewater	Inhalation	Fish Consumption
		$SSC_{sed(ing)-c}$	$SSC_{sw(ing)-c}$	$SSC_{sed(derm)-c}$	$SSC_{sed(derm)-c}$	SSC_{inh-c}	SSC_{fish-c}
Metals - Inorganics		mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg
Arsenic	7440-38-2	55.4	*Not Evaluated*	58.6	*Not Evaluated*		
Cadmium	7440-43-9	*Not Evaluated*					
Chromium III	16065-83-1						
Chromium VI	18540-29-9						
Copper	7440-50-8						
Cyanide	57-12-5						
Lead	7439-92-1						
Mercury (inorganic in sediment;	7439-97-6						
Methyl Mercury methylmercury in fish)	22967-92-6						
Nickel	various						
Zinc	7440-66-6						
Volatile organic compounds (VOCs)							
Benzene	71-43-2	1510	0.736	*Not Evaluated*	0.57	0.00332	0.244
Ethyl benzene	100-41-4	*Not Evaluated*					
Styrene	100-42-5						
Toluene	108-88-3						
Xylenes (mixed)	1330-20-7						
Polycyclic Aromatic Hydrocarbons (PAHs)							
Acenaphthene	83-32-9	*Not Evaluated*					
Acenaphthylene (toxicity surrogate - acenaphthene)	208-96-8						
Anthracene	120-12-7						
Benzo(a)pyrene equivalents (B[a]P-PEQs)	50-32-8	11.4	98	2.78	0.53	2030	0.0856
Fluoranthene	206-44-0	*Not Evaluated*					
Fluorene	86-73-7						
Methylnaphthalene (toxicity surrogate - naphthalene)	1321-94-4						
Naphthalene	91-20-3						
Perylene (toxicity surrogate - pyrene)	198-55-0						
Phenanthrene (toxicity surrogate - anthracene)	85-01-8						
Pyrene	129-00-0						
Dibenzo-p-dioxins/dibenzofurans							
2,3,7,8-TCDD equivalents (2,3,7,8-TCDD-TEQs)	1746-01-6	5.93E-05	3.65E-03	6.28E-05	2.17E-05	0.00589	4.69E-08
Other Organics							
Carbazole	86-74-8	4150	104	1320	27.3	17800	3.28
Dibenzofuran (unsubstituted)	132-64-9	*Not Evaluated*					
Hexachlorobenzene	118-74-1	51.9	30.7	16.5	0.802	0.345	0.41
Octachlorostyrene	29082-74-4	*Not Evaluated*					
PCBs (Polychlorinated Biphenyls)	1336-36-3						

Appendix J - Sediment Screening Value (SSV) Calculations

Variables are defined in Appendix A. Route-specific values are shown in Tables H-1 and I-1. Calculation results (sediment screening values - all routes, chronic and cancer endpoints) are shown in Table J-1.

Sediment Screening Values

Combined chronic sediment screening values for all routes-of-exposure analyzed in this SSV Report were determined using the following equation calculation.

$$SSV_{ttl}\ \{\ mg/kg\ \} = (1/SSV_{Sed(Ing)} + 1/SSV_{SW(Ing)} + 1/SSV_{Sed(Derm)} + 1/SSV_{SW(Derm)} + 1/SSV_{Inh} + 1/SSV_{Fish})^{-1}$$ *Equation #A-46.*

Calculation results are shown in Table J-1, as well as SSV Report Table #12 (Section 7).

Similarly, sediment screening values for cancer endpoints were calculated with:

$$SSV_{ttl\text{-}c}\ \{\ mg/kg\ \} = (1/SSV_{Sed(Ing)\text{-}c} + 1/SSV_{SW(Ing)\text{-}c} + 1/SSV_{Sed(Derm)\text{-}c} + 1/SSV_{SW(Derm)\text{-}c} + 1/SSV_{Inh\text{-}c} + 1/SSV_{Fish\text{-}c})^{-1}$$ *Equation #A-47.*

Calculation results are shown in Table J-1, as well as SSV Report Table #12 (Section 7).

Percent contribution by route-of-exposure

The percent an individual route-of-exposure contributes to the sediment screening value for each chemical for both chronic and cancer endpoints were determined by:

$$SSV_{\%x}\ \{\ \%\ \} = (1/SSV_x) / (1/SSV_{ttl}) * 100$$ *Equation #A-48.*

Calculation results are shown in Table J-1, as well as SSV Report Table #12 (Section 7).

Table J-1 - - Sediment Screening Values - Routes of Exposure

Chronic (non-cancer) Endpoint

	Cas No.	Chronic Sediment Screening Values (mg/kg)	Route-of-exposure contribution (%)					
			Sediment Ingestion	Surfacewater Ingestion	Dermal Sediment	Dermal Surfacewater	Inhalation	Fish Consumption
Metals - Inorganics								
Arsenic	7440-38-2	48	65%	*Not Evaluated*	35%	*Not Evaluated*	*Not Evaluated*	*Not Evaluated*
Cadmium	7440-43-9	140	58%		42%			
Chromium III	16065-83-1	370000	100%		*Not Evaluated*			
Chromium VI	18540-29-9	730	100%					
Copper	7440-50-8	9000	100%					
Cyanide	57-12-5	4900	100%					
Lead	7439-92-1	400 *	*Not Evaluated*					
Mercury (inorganic in sediment; methylmercury in fish)**	7439-97-6	0.021	0%					100%
Methyl Mercury	22967-92-6							
Nickel	various	4900	100%					*Not Evaluated*
Zinc	7440-66-6	73000	100%					
Volatile organic compounds (VOCs)								
Benzene	71-43-2	0.0049	0%	1%	*Not Evaluated*	1%	97%	1%
Ethyl benzene	100-41-4	0.34	0%	1%		3%	91%	5%
Styrene	100-42-5	4.1	0%	1%		3%	91%	5%
Toluene	108-88-3	0.12	0%	1%		2%	94%	3%
Xylenes (mixed)	1330-20-7	0.078	0%	0%		0%	99%	0%
Polycyclic Aromatic Hydrocarbons (PAHs)								
Acenaphthene	83-32-9	22	0%	4%	0%	41%	23%	32%
Acenaphthylene (toxicity surrogate - acenaphthene)	208-96-8	20	0%	5%	0%	41%	23%	30%
Anthracene	120-12-7	690	1%	5%	2%	61%	12%	18%
Benzo(a)pyrene (or B[a]P equivalents)	50-32-8	*Not Evaluated*	*Not Evaluated*					
Fluoranthene	206-44-0	82	1%	2%	2%	59%	1%	35%
Fluorene	86-73-7	49	0%	8%	1%	57%	18%	16%
Methylnaphthalene (toxicity surrogate - naphthalene)	1321-94-4	0.48	0%	0%	0%	1%	97%	2%
Naphthalene	91-20-3	0.11	0%	0%	0%	1%	99%	0%
Perylene (toxicity surrogate - pyrene)	198-55-0	54	1%	0%	2%	67%	0%	31%
Phenanthrene (toxicity surrogate - anthracene)	85-01-8	730	1%	4%	2%	56%	4%	33%
Pyrene	129-00-0	78	1%	2%	2%	50%	1%	44%
Dibenzo-p-dioxins/dibenzofurans								
2,3,7,8-TCDD (or 2,3,7,8-TCDD equivalents) **	1746-01-6	1.4E-06	0%	0%	0%	0%	0%	99%
Other Organics								
Carbazole	86-74-8	*Not Evaluated*	*Not Evaluated*					
Dibenzofuran (unsubstituted)	132-64-9	1.8	0%	3%	0%	23%	3%	71%
Hexachlorobenzene	118-74-1	0.88	0%	1%	1%	18%	62%	18%
Octachlorostyrene	29082-74-4	0.019	0%	0%	0%	0%	0%	99%
PCBs (Polychlorinated Biphenyls) **	1336-36-3	0.0046	0%	0%	0%	2%	1%	97%

Cancer Endpoint

Carcinogens		Cancer Sediment Screening Criteria	Sediment Ingestion	Surfacewater Ingestion	Dermal Sediment	Dermal Surfacewater	Inhalation	Fish Consumption
Arsenic	7440-38-2	28	51%	*Not Evaluated*	49%	*Not Evaluated*		
Benzene	71-43-2	0.0032	0%	0%	*Not Evaluated*	1%	98%	1%
Benzo(a)pyrene Equivalents	50-32-8	0.071	1%	0%	3%	13%	0%	83%
2,3,7,8-TCDD (or 2,3,7,8-TCDD equivalents) **	1746-01-6	4.7E-08	0%	0%	0%	0%	0%	100%
Carbazole	86-74-8	2.8	0%	3%	0%	10%	0%	87%
Hexachlorobenzene	118-74-1	0.15	0%	0%	1%	19%	43%	36%

* 400 mg/kg = EPA soil lead number also used for screening soils in Minnesota.

** (shaded chemicals) Sediment Screening Value may approach or be less than ambient or background concentration. See sections on individual chemicals (in SSV Report Section 4), SSV Report 7.1.1 and Appendix C for information on ambient and background concentrations.

Appendices References

Bunge, A.L. and J.M. Parks (1997). Predicting Dermal Absorption from Contact with Chemically Contaminated Soils. Environmental Toxicology and Risk Assessment: Modeling and Risk Assessment. Dwyer, F.J., T.R. Doane and M.L. Hinman, American Society for Testing and Materials: 227-244.

California Office of Environmental Health Hazard Assessment, (2003). Chronic Reference Levels. California Office of Environmental Health Hazard Assessment, Last updated 2/2005, http://www.oehha.org/air/chronic_rels/pdf/allchrels.pdf

California Office of Environmental Health Hazard Assessment, (2002). Cancer Potency Values. Last updated 11/2002, http://www.oehha.org/air/hot_spots/pdf/TSDNov2002.pdf

Duff, R.M. and J.C. Kissel (1996). Effect of soil loading on dermal absorption efficiency from contaminated soils. J Toxicol Environ Health 48(1): 93-106.

Environmental Protection Agency, (2003). Integrated Risk Information System (IRIS). Environmental Protection Agency, Office of Health and Environmental Assessment, Environmental Criteria Office, Online, Last updated November 2003, http://www.epa.gov/iris/

Environmental Protection Agency (2002a). PRG Physical/Chemical Data: InterCalc Tables. EPA Region 9, San Francisco, CA. October 1, 2002.

Environmental Protection Agency (2002b). Toxicological Review of Toluene. National Center for Environmental Assessment, Washington, D.C. *Draft* Report NCEA-S-1264, August 2002.

Environmental Protection Agency (2001a). Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual (Part E, Interm Supplemental Guidance for Dermal Risk Assessment). Office of Emergency and Remedial Response, Washington, D.C. Interim Report EPA/540/R/99/005, September 2001.

Environmental Protection Agency (2001b). Water Quality Criterion for the Protection of Human Health: Methylmercury. Office of Science and Technology, Office of Water, Washington, D.C. Final Report EPA-823-R-01-001, January 2001.

Environmental Protection Agency (1999). Risk Assessment Issue Paper for: Derivation of the Chronic Oral Reference Dose (RfD) - Dibenzofuran (CASRN. 132-64-9). Washington, D.C. 98-029/07-19-99, July 17,1999.

Environmental Protection Agency (1997a). Exposure Factors Handbook. Office of Research and Development, National Center for Environmental Assessment, Washington, D.C. Report EPA/600/P-95/002Fa, August, 1997.

Environmental Protection Agency (1997b). Health Effects Assessment Summary Tables (HEAST): FY 1997 Update. Solid Waste and Emergency Response, Washington, D.C. EPA-540-R-97-036, July 1997.

Environmental Protection Agency (1997c). Mercury Study Report to Congress. Office of Air Quality Planning and Standards and Office of Research and Development, Washington, D.C., December 1997.

Environmental Protection Agency (1997d). The Incidence and Severity of Sediment Contamination in Surface Waters for the United States: Volume 1, National Sediment Quality Survey. Office of Science and Technology, Washington, D.C. Report EPA 823-R-97-006, September 1997.

Environmental Protection Agency (1996a). Soil Screening Guidance: Technical Background Document. Part 5: Chemical-specific Parameters. Office of Emergency and Remedial Response, Washington, D.C. Report EPA/540/R-95/128, July 1996.

Environmental Protection Agency (1996b). Soil Screening Guidance: Technical Background Document. Office of Emergency and Remedial Response, Washington, D.C. Report EPA/540/R-95/128, July 1996.

Environmental Protection Agency (1995a). Great Lakes Water Quality Initiative Technical Support Document for the Procedure to Determine Bioaccumulation Factors. Washington, D.C. Report 822R94002, July 1994.

Environmental Protection Agency (1995b). Regional Environmental Monitoring and Assessment Program (REMAP). Washington, D.C. Data, 1995.

Environmental Protection Agency (1992). Dermal Exposure Assessment: Principles and Applications. Office of Health and Environmental Assessment, Washington, D.C. Interim Report EPA/600/8-91/01B.

Environmental Protection Agency (1989). Risk Assessment Guidance for Superfund: Volume I - Human Health Evaluation Manual (Part A). Office of Emergency and Remedial Response, Washington, D.C. Report EPA/540/1-89/002, December 1989.

Food and Agriculture Organization of the United Nations and World Health Organization (2001). Summary of the fifty-seventh meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). Expert Committee on Food Additives, Rome, Italy. Meeting Summary Report, June 5-14, 2001.

Massachusetts Department of Environmental Protection (2002). Weighted Skin-Soil Adherence Factors. Office of Research and Standards, Boston, MA. Technical Update, April, 2002.

Michigan Department of Environmental Quality (2002). Operational Memorandum #18: Part 201 Generic Cleanup Criteria - Rule 752 table. Remediation and Redevelopment Division, Lansing, MI. December 21, 2002. http://www.deq.state.mi.us/documents/deq-rrd-part201-rules-Rule752table.pdf

Minnesota Department of Health, (2003). Methods for Estimating the Carcinogenic Health Risks from Dioxin-Like Compounds. Environmental Assessment and Surveilance, Environmental Health, St. Paul, MN. http://www.health.state.mn.us/divs/eh/risk/chemicals/dioxinnm.html

Minnesota Department of Health, (2002). Health Risk Values (HRVs). Environmental Assessment and Surveilance, Environmental Health, St. Paul, MN. Minnesota Rule, Online, Last updated September 11, 2002, http://www.revisor.leg.state.mn.us/arule/4717/8000.html

Minnesota Department of Health, (2001a). Methods for Estimating the Carcinogenic Health Risks from Polycyclic Aromatic Hydrocarbons. Herbrandson, C. and C. Stroebel, Environmental Assessment and Surveilance, Environmental Health, St. Paul, MN. Memo To: Goeden, H., Minnesota Pollution Control Agency. May 29, 2001. http://www.health.state.mn.us/divs/eh/risk/chemicals/dioxinnm.html

Minnesota Department of Health (2001b). Fond du Lac/Grand Portage Fish Consumption Advisory Project. Environmental Assessment and Surveilance, Environmental Health, St. Paul, MN. Final Report, December 2001.

New York State Department of Environmental Conservation (1997). Ambient Water Quality Value for Protection of Sources of Potable Water: Octachlorostyrene. Division of Water, Albany, N.Y., January 29, 1997.

Ontario Ministry of the Environment (1993). Guidelines for the protection and management of aquatic sediment quality in Ontario. Water Resources Branch, D. Persaud, R. Jaagumagi, A. Hayton, Toronto. Report ISBN 0-7729-9248-7, August 1993, 26 pg.

Syracuse Research Corporation (SRC), (2003). Interactive PhysProp Database Demo. http://esc.syrres.com/interkow/physdemo.htm

US Fish and Wildlife Service, (2002). Whole Fish PAH Data. A. Trowbridge, Data from A. Trowbridge,

Washington State Department of Ecology (1997). Developing Health Based Sediment Quality Criteria for Cleanup Sites: A Case Study Report. Toxics Cleanup, Olympia, WA. Report 97-114S, December 1997.

Washington State Department of Health (1996). Development of Sediment Quality Criteria for the Protection of Human Health. Office of Toxic Substances, Olympia, WA. Tier II Report, May 1996.

Washington State Department of Health (1995). Development of Sediment Quality Criteria for the Protection of Human Health. Office of Toxic Substances, Olympia, WA. Tier I Report, June 1995.

Wester, R.C., H.I. Maibach, D.A. Bucks, L. Sedik, J. Melendres, C. Liao and S. DiZio (1990). Percutaneous absorption of [14C]DDT and [14C]benzo[a]pyrene from soil. Fundam Appl Toxicol 15(3): 510-6.

Wester, R.C., H.I. Maibach, L. Sedik, J. Melendres and M. Wade (1993a). In vivo and in vitro percutaneous absorption and skin decontamination of arsenic from water and soil. Fundam Appl Toxicol 20(3): 336-40.

Wester, R.C., H.I. Maibach, L. Sedik, J. Melendres and M. Wade (1993b). Percutaneous absorption of PCBs from soil: in vivo rhesus monkey, in vitro human skin, and binding to powdered human stratum corneum. J Toxicol Environ Health 39(3): 375-82.