Public Health Consultation

Updated Human Health Screening Values for

ST. LOUIS RIVER SEDIMENTS:

US STEEL SITE

DULUTH, ST. LOUIS COUNTY, MINNESOTA

EPA FACILITY ID: MND039045430

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Prepared by:
Minnesota Department of Health
Under a Cooperative Agreement with the
Agency for Toxic Substances and Disease Registry

Health Consultation: A disclaimer

The Minnesota Department of Health (MDH) Site Assessment and Consultation (SAC) unit collaborates with the Agency for Toxic Substances and Disease Registry (ATSDR), the lead federal public health agency, to prepare health consultation documents to determine if exposure to contaminants can harm people's health and to prevent and reduce exposures and illnesses. A health consultation is a written response to a specific request for information about health risks related to a specific site, a chemical release, or the presence of hazardous material, and considers the levels of hazardous substances at a site, whether people might be exposed to contaminants, by what pathways, and what potential harm the substances might cause to people. In order to prevent or mitigate exposures, a consultation may lead to specific actions and recommendations, such as restricting use of or replacing water supplies, intensifying environmental sampling, restricting site access, or removing the contaminated material. In addition, consultations may recommend additional public health actions, such as conducting health surveillance activities to evaluate exposure or trends in adverse health outcomes, conducting biological indicators or exposure studies to assess exposure, conducting health studies, characterizing demographics, and/or providing health education for health care providers and community members.

ATSDR provides technical assistance and funding to MDH to help identify and evaluate environmental health threats to communities using the best science, taking responsive public health actions, and providing trusted health information. While this health consultation was supported by funds from a cooperative agreement with ATSDR, it was not reviewed by ATSDR.

The conclusions and recommendations presented in this health consultation are based on an analysis of the environmental sampling data and information made available to MDH within a limited time frame. The availability of additional sampling data, new information and/or changes in site conditions could affect the conclusions and recommendations presented in this document. MDH will consider reviewing additional future data related to the site, if made available and deemed appropriate.

FOREWORD

This Public Health Consultation 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.

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Table Of Contents

Health Consultation: A disclaimer ---- **ii**

FOREWORD ---- **iii**

Table Of Contents ---- **iv**

List of Attachments ---- **vi**

List of Appendices ---- **vi**

Acronym and Abbreviation Glossary ---- **vii**

Executive Summary ---- **x**

Sediment Screening Values ---- **x**

Calculation of Sediment Screening Values ---- **xi**

Results and Discussion ---- **xii**

Conclusions and Recommendations ---- **xii**

Table - 2013 Sediment Screening Values (comparison with 2005 Sediment Screening Values) ---- xiii

Recommendations: ---- xiii

1 _Contamination of the Lower St. Louis River by US Steel and Other Industrial Operations ---- **1**

2 _Organization of the Public Health Consultation and Development of Sediment Screening Values for US Steel ---- **2**

3 _Exposure to Contaminants ---- **3**

Table 1 - Receptor Characteristics ---- 5

3.1 Type of sediments evaluated ---- **5**

3.2 Frequency of contact with sediments ---- **5**

Table 2 - Exposure Event Frequency ---- 6

3.3 Default exposure assumptions ---- **6**

3.3.1 Coordinating life-stage exposures and life-stage sensitivity to carcinogens ---- 7

3.3.2 Surface water ingestion ---- 7

Table 3 - Surface Water Ingestion ---- 8

3.3.3 Sediment ingestion ---- 8

Table 4 - Sediment Ingestion ---- 9

3.3.4 Dermal exposure to sediment ---- 9

Table 5 - Sediment Dermal Contact ---- 10

3.3.5 Dermal exposure to surface water ---- 10

Table 6 - Surface Water Dermal Contact ---- 10

3.3.6 Inhalation exposure to volatile chemicals ---- 11

Table 7 - Inhalation Fraction ---- 11

3.3.7 Fish consumption exposure ---- 11

Table 8 - Fish Ingestion ---- 11

4 _Potential Chemicals of Concern ---- **12**

4.1 Inorganic chemicals and metals ---- **13**

4.1.1 Mercury ---- 13

4.1.2 Cadmium ---- 14

4.1.3 Lead ---- 15

4.1.4 Arsenic ---- 16

4.2 Volatile organic compounds ---- **16**

4.3 Persistent organic compounds ---- **17**

4.3.1 Chlorinated organics ---- 17
Table 9 - TCDD-TEFs ... 19
4.3.2 Polycyclic aromatic hydrocarbons ---- 22
Table 10 - BaP-RPFs ... 24
5 _Chemical Specific Data ---- 26
5.1 Appropriate detection limits ... 26
5.2 Partitioning of chemicals ... 26
5.3 Contaminant accumulation in fish tissue ... 28
5.3.1 BSAFs for non-polar organic chemicals ---- 30
5.3.2 Mercury bioaccumulation ---- 31
Figure 1 - Mercury in Fish Tissue vs Fish Length ... 32
Table 11 - Calculated BSAFs (mercury) ... 33
6 _Screening Value Calculation Defaults ---- 34
6.1 Health-based toxicity values used to calculate screening values ... 34
6.2 Relative Source Contribution, Hazard Index, and Cancer Risk ... 35
6.3 Modifiers ... 35
7 _Sediment Screening Model Results ---- 36
Table 12 - Sediment Screening Model Results ... 37
7.1 Discussion of SSM results and SSVs ... 38
7.1.1 Comparison of SSM results with SQTs ---- 38
Table 13 - Comparison of SSM Results with MPCA SQTs ... 40
7.1.2 SSV's below ambient or background concentrations ---- 41
Table 14 - SSVs and REMAP Data for PAHs and Mercury ... 42
7.2 Sediment Screening Values ... 42
Table 15 - 2013 Sediment Screening Values – US Steel Site ... 43
7.3 Modifying Sediment Screening Values ... 43
Table 16 - PAH SSM Results - Without Fish Consumption ... 44
8 _Children and Other Special Populations ---- 44
9 _Conclusions ---- 44
9.1 General application of screening values ... 44
9.2 Application to the US Steel St. Louis River Site ... 45
10 Recommendations ---- 45
11 Public Health Action Plan ---- 46
12 References ---- 47

List of Attachments

Attachment 1 - Lower St. Louis River and REMAP sample locations
Attachment 2 - Aerial photo of the US Steel facility in 1951
Attachment 3 - Aerial photo of the St. Louis River and the US Steel facility in 1967
Attachment 4 - Aerial photo of the St. Louis River and the US Steel facility in 1967
Attachment 5 - Aerial photo of the St. Louis River and the US Steel facility in 1967

List of Appendices

Appendix A – Glossary of Equation Variables and Abbreviations **1**
Appendix B - Media Exposure/Contact Calculations **6**
Appendix C – Contaminated Sediments and Limitations on Mercury Bioaccumulation **10**
Appendix D – SSM Chemical-specific Variable Defaults **12**
Appendix E - Screening Detection Limits for Persistent Bioaccumulative Toxicants in Sediment and Fish Tissue **16**
Appendix F - Additional Information on Default Fraction Organic Carbon (foc) **18**
Appendix G - PAH BSAFs **19**
Appendix H - Route-Specific Sediment Screening Model Calculations - Non-Cancer **24**
Appendix I - Route-Specific Sediment Screening Model Calculations - Cancer **28**
Appendix J – Combining Routes of Exposure - Sediment Screening Model **31**
Appendices References **33**

Acronym and Abbreviation Glossary

(Appendix A Contains a Complete Glossary Including Equation Variables)

ADAF	-	age-dependent adjustment factor
atm	-	atmosphere(s)
ATSDR	-	US Agency for Toxic Substances and Disease Registry
AVS	-	acid volatile sulfide
BAF	-	biota accumulation factor
BaP	-	benzo[a]pyrene
BaP-PEQ	-	benzo[a]pyrene potency equivalent
BaP-RPF	-	benzo[a]pyrene relative potency factor
BCF	-	biota concentration factor
BNS	-	Binational Toxics Strategy
BSAF	-	biota-sediment accumulation factor
CAC	-	community action committee
CDC	-	Center for Disease Control and Prevention
CERCLIS	-	Comprehensive Environmental Response, Compensation, and Liability Information System
CL	-	confidence limit(s)
cm	-	centimeter(s)
COC	-	chemical of concern
cPAH	-	carcinogenic PAH (polycyclic aromatic hydrocarbon)
cPCB	-	carcinogenic PCB (polychlorinated biphenyl)
CSF	-	cancer slope factor
d	-	day(s)
DDT	-	dichlorodiphenyltrichloroethane
dl	-	deciliter(s)
DNA	-	deoxyribonucleic acid
EPA	-	US Environmental Protection Agency
g	-	gram(s)
HBV	-	health-based value
HCB	-	hexachlorobenzene
hr	-	hour(s)
HpCB	-	heptachlorinated biphenyl
HpCDD	-	heptachlorinated dibenzo-p-dioxin
HpCDF	-	heptachlorinated dibenzofuran
HxCB	-	hexachlorinated biphenyl
HxCDD	-	hexachlorinated dibenzo-p-dioxin
HxCDF	-	hexachlorinated dibenzofuran
IARC	-	International Agency for Research on Cancer
IRIS	-	Integrated Risk Information System (EPA)
kg	-	kilogram(s)
K_{oc}	-	organic carbon partioning constant
K_{ow}	-	octanol-water partioning constant
L	-	liter(s)
m	-	meter(s)

m^3	-	cubic meter(s)
MDH	-	Minnesota Department of Health
mg	-	milligram(s)
mL	-	milliliter(s)
MN	-	Minnesota
mol	-	mole(s)
MPCA	-	Minnesota Pollution Control Agency
MRL	-	minimum risk level
MW	-	molecular weight
n	-	number of samples
NAPL	-	non-aqueous phase liquid
ng	-	nanogram(s)
nPAH	-	non-carcinogenic PAH
NPL	-	National Priority List
NTP	-	National Toxicology Program
OC	-	organic carbon
OCDD	-	octachlorinated dibenzo-p-dioxin
OCDF	-	octachlorinated dibenzofuran
OCS	-	octachlorostyrene
OEHHA	-	Office of Environmental Health Hazard Assessment (California State)
PAH	-	polycyclic aromatic hydrocarbon
PBDD	-	polybrominated dibenzo-p-dioxin
PBDE	-	polybrominated diphenylether
PBDF	-	polybrominated dibenzofuran
PCB	-	polychlorinated biphenyl
PCDD	-	polychlorinated dibenzo-p-dioxin
PCDF	-	polychlorinated dibenzofuran
PeCB	-	pentachlorinated biphenyl
PeCDD	-	pentachlorinated dibenzo-p-dioxin
PeCDF	-	pentachlorinated dibenzofuran
PEF	-	potency equivalence factor
PEQ	-	potency equivalence
PHC	-	Public Health Consultation
PLP	-	Permanent List of Priorities (Minnesota)
REMAP	-	Regional Environmental Monitoring and Assessment Program
RfC	-	reference concentration
RfD	-	reference dose
RME	-	reasonable maximum exposure
RPF	-	relative potency factor
RSC	-	relative source contribution
RTK	-	Right-to-Know Act
SD	-	standard deviation
SLRIDT	-	St. Louis River Interlake-Duluth Tar Site
SQT	-	Sediment Quality Target
SRV	-	Soil Reference Value (MPCA)
SSM	-	Sediment Screening Model

SSV	-	Sediment Screening Value
STORET	-	EPA Storage and Retrieval database
TCB	-	tetrachlorinated biphenyl
TCDD	-	tetrachlorinated dibenzo-p-dioxin
TCDD-TEQ	-	2,3,7,8-tetrachlorinated dibenzo-p-dioxin toxic equivalents
TCDF	-	tetrachlorinated dibenzofuran
TEF	-	toxic equivalent factor
tPCB	-	total polychlorinated biphenyl
µg	-	microgram(s)
UR	-	unit risk
VOC	-	volatile organic compound
WHO	-	World Health Organization
wt.	-	weight
yr	-	year(s)

Executive Summary

The St. Louis River empties into Lake Superior through the ports of Duluth, Minnesota and Superior, Wisconsin. Historically, these twin ports have been industrial centers with considerable coke, iron and steel making. The US Steel Site on the St. Louis River was an active coking and steel-making facility from 1915 to 1979. Large discharges from this site to the River occurred over this time, leading to the contamination of river sediments. The River is currently used for recreation and people are exposed to site-related contaminants in the River. This Public Health Consultation (PHC) evaluates reasonable, maximal exposures to chemicals in St. Louis River sediments and calculates Sediment Screening Values (SSVs) that are protective of human health.

Sediment Screening Values

People are directly exposed to contaminants in sediments through contact with the skin and ingestion of suspended sediments in surface water. Chemical contaminants in sediments also partition into porewater and surface water. In addition, fish and other aquatic organisms accumulate some chemicals from food and sediment that they ingest, or through direct partitioning 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.

Six different exposure routes were quantitatively evaluated using a Sediment Screening Model (SSM): surface water ingestion, sediment ingestion, dermal surface water exposure, dermal sediment exposure, inhalation and fish consumption. Contributions of different routes to a total exposure are compared. The SSM evaluated chronic and lifetime exposure durations. Although 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, toxicity data are not available to determine a threshold exposure for effects of these short exposures. Due to the general lack of toxicity criteria for short exposures to chemicals, acute exposures are not addressed in this PHC.

The SSM also combined the six different routes of exposure into a multi-route model result, which is a reasonable chemical-specific human exposure sediment screening concentration. SSM results were reviewed for their relevance. If ecological sediment criteria were significantly more restrictive than the SSM results for a specific chemical, the SSM result for that chemical was not promoted to an SSV.

The PHC provides an overview of the default exposure values and health criteria used to develop the SSVs. It includes discussion of issues considered for selection of default values and appropriate use of the SSVs. The Appendices comprise a glossary of variables used in all equations, a description of equations used in the SSM and technical arguments for selection of some default values. The PHC and attached Appendices include all equations used in the SSM and needed to calculate SSVs. They also show how to calculate the percent contribution for each exposure route given the model's assumptions. It is expected that the default values and models used in this PHC may change as additional data are acquired and models become more refined.

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 human health consideration when evaluating the chemical in sediments at a site.

Only exposures to chemicals associated with water-covered sediments have been evaluated. Exposures to upland, beach and intertidal sediments are likely to be different and should be evaluated separately. While SSVs have been developed for a suite of persistent chemicals, they do not cover all chemicals found in sediments that can impact health.

The SSVs in this PHC are considered site-specific because reasonable maximal exposures (RMEs) for the lower St. Louis River were used to describe exposures in SSV calculations. In addition to their intended use in the lower St. Louis River, the SSVs may be protective for contaminated sediments in other waterbodies. However, MDH recommends reviewing site-specific RMEs before applying the SSVs to other sites. In particular, exposure parameters (Table 2) may vary between sites. Environmental parameters for which general default values have been used include: sediment organic carbon fraction, fish tissue lipid fraction and temperature. Fraction organic carbon in sediments is an important site-specific parameter for non-polar organic compounds because the models for sediment to water partitioning and biota-sediment fish tissue accumulation are dependent on contaminant concentrations relative to the fraction of organic carbon in sediments. Parameters that have not been evaluated in the SSVs that may impact the transport and availability of contaminants include: particle size, redox potential, mineral content, clay content and porosity of sediments.

Calculation of Sediment Screening Values

At the request of the MPCA, MDH developed SSVs for screening sediments at the US Steel site in the St. Louis River Estuary (MDH, 2002) in December 2002. US Steel used these 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 (MDH, 2005).

This 2013 SSV Public Health Consultation is an update of the 2005 report, incorporating the following changes:

- Body weight, dermal surface area and inhalation rates are updated using the latest EPA Exposures Factors Handbook (US EPA, 2011).
- A default age-dependent adjustment factor (ADAF) is used, as appropriate, to account for the higher sensitivity to carcinogens in early life.
- Age groups have been changed to facilitate integration of the ADAFs and exposure data.
- A new polycyclic aromatic hydrocarbon (PAH) biota-sediment accumulation factor (BSAF), based on Superfund data in largemouth bass and bluegills, is used.
- New guidance for determining the carcinogenic potency of mixtures of PAHs is recommended.

- New carcinogenic PAH (cPAH) Relative Potency Factors (RPFs) are recommended for use in calculating cPAH Potency Equivalents (PEQs).
- Newer toxicity reference values were used for many chemicals.
- A new oral cancer slope factor for chromium VI (CA OEHHA, 2011) was used to develop a cancer-based chromium VI SSV.
- Fourteen of the evaluated chemicals were not promoted to SSVs because ecological criteria are likely to be significantly more restrictive.
- SSVs were calculated for chromium VI, pentachlorophenol and quinolone. These chemicals were not evaluated in 2005.
- The pyrene reference dose was used in the SSM as a surrogate for a number of non-carcinogenic PAHs.
- Dilution factors have been added to the equilibrium partition model equations to account for dilution of chemicals as they move from sediment and water, mixing into water and air, respectively.

Results and Discussion

As expected, quantitative evaluation showed that largest exposure to chemicals with different chemical and physical characteristics will occur by different routes. For example: the largest exposure to volatile organic compounds in sediments is likely to occur by inhalation (e.g., during swimming), while the largest route for most metals is ingestion of sediments, and exposure to bioaccumulative compounds in sediments occurs mostly through the fish consumption pathway.

Conclusions and Recommendations

The Table, below, shows the 2013 and 2005 SSVs for comparison. The SSVs are a tool for screening contaminated sediments for potential impacts to human health. Chemical concentrations in water-covered sediments at or below the SSVs are considered safe for the general public. However, chemical concentrations in sediments exceeding the SSVs should not be considered unsafe because the SSVs were developed using conservative measures of exposure, bioavailability and toxicity. Rather, any exceedance of these values suggests that site-specific conditions need to be evaluated prior to concluding that contaminated sediments may impact health. Furthermore, the SSVs are not intended to be used as sediment cleanup values.

Table - 2013 Sediment Screening Values (comparison with 2005 Sediment Screening Values)

Chemical		CAS No.	Endpoint	2013 Sediment Screening Value	2005 Sediment Screening Value
				SSV	SSV
Metals - Inorganics				**mg/kg**	**mg/kg**
	Arsenic	7440-38-2	Non-cancer	**50**	**48**
			Cancer	**30**	**28**
	Cadmium	7440-43-9	Non-cancer	**10**	**140**
	Chromium III	16065-83-1	Non-cancer	**deferred**	**370000**
	Chromium VI	18540-29-9	Non-cancer	**300**	**730**
			Cancer	**40**	not evaluated
	Copper	7440-50-8	Non-cancer	**deferred**	**9000**
	Cyanide	57-12-5	Non-cancer	**2000**	**4900**
	Lead	7439-92-1	Non-cancer	**300 ***	**400 ***
	Mercury (SSV - tHg; exp - tHg)	7439-97-6	Non-cancer	**0.02 †**	**0.021 †**
	Mercury (SSV - tHg; exp - MeHg)		Non-cancer		
	Nickel	various	Non-cancer	**deferred**	**4900**
	Zinc	7440-66-6	Non-cancer	**deferred**	**73000**
Volatile organic compounds (VOCs)					
	Benzene	71-43-2	Non-cancer	**1**	**0.0049**
			Cancer	**0.8**	**0.0032**
	Ethyl benzene	100-41-4	Non-cancer	**70**	**0.34**
	Styrene	100-42-5	Non-cancer	**300**	**4.1**
	Toluene	108-88-3	Non-cancer	**50**	**0.12**
	Xylenes (mixed)	1330-20-7	Non-cancer	**40**	**0.078**
Polycyclic Aromatic Hydrocarbons (PAHs)			Non-cancer		
	Acenaphthene	83-32-9	Non-cancer	**deferred**	**22**
	Acenaphthylene (toxicity surrogate - pyrene)	208-96-8	Non-cancer	**deferred**	**20**
	Anthracene	120-12-7	Non-cancer	**deferred**	**690**
	Benzo(a)pyrene equivalents (BaP-PEQs)	50-32-8	Cancer	**0.2 †**	**0.071 †**
	Fluoranthene	206-44-0	Non-cancer	**deferred**	**82**
	Fluorene	86-73-7	Non-cancer	**deferred**	**49**
	Methylnaphthalene (toxicity surrogate - naphtha	1321-94-4	Non-cancer	**deferred**	**0.48**
	Naphthalene	91-20-3	Non-cancer	**deferred**	**0.11**
	Perylene (toxicity surrogate - pyrene)	198-55-0	Non-cancer	**deferred**	**54**
	Phenanthrene (toxicity surrogate - pyrene)	85-01-8	Non-cancer	**deferred**	**730**
	Pyrene	129-00-0	Non-cancer	**deferred**	**78**
Dibenzo-p-dioxins/dibenzofurans					
	2,3,7,8-TCDD equivalents (TCDD-TEQs)	1746-01-6	Non-cancer	**4E-07 †**	**1.4E-06 †**
			Cancer	**2E-08 †**	**4.7E-08 †**
Other Organics					
	Carbazole	86-74-8	Cancer	**80**	**2.8**
	Hexachlorobenzene	118-74-1	Non-cancer	**0.1**	**0.88**
			Cancer	**0.1**	**0.15**
	Octachlorostyrene	29082-74-4	Non-cancer	**0.02**	**0.019**
	Pentachlorophenol	87-86-5	Non-cancer	**6**	not evaluated
			Cancer	**0.7**	not evaluated
	Polychlorinated Biphenyls (PCBs)	1336-36-3	Non-cancer ‡	**0.005 †**	**0.0046 †**
			Cancer	**0.006 †**	not evaluated
	Quinoline	91-22-5	Cancer	**0.4**	not evaluated

Deferred – SSVs were not finalized because benthic criteria are likely to be more restrictive.
* MPCA Soil Reference Value - See Section 4.1.3
† (shaded chemicals) Sediment Screening Value may approach or be less than ambient or background concentration.

Recommendations:

- SSVs should not be used to screen or evaluate upland, intertidal or beach sediments.

- Laboratory 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.
- Additional evaluation may be required to determine whether chemicals in the sediments could impact public health, if chemical concentrations in sediments adjacent to the US Steel Site exceed the SSVs (or background levels for TCDD-TEQs).
    - If chemical concentrations in sediments are below the values developed in this PHC, the screened chemicals in sediments should not adversely impact the health of the public.
    - If special populations are likely to be exposed (e.g., subsistence fishers), further evaluation may be needed to determine whether or not they are protected.
- Default reasonable maximum exposures and site-specific data included in this PHC and attached appendices should be reviewed prior to using them for evaluating sediments in other water bodies.
- The SSM may be used to qualitatively evaluate the relative impact of different routes of exposure to chemicals in sediments.

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 outlet. The lower St. Louis River is often called a freshwater estuary, because seiches regularly reverse the river's flow 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, a 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 a repository (or sink) for such persistent chemicals, and high concentrations of contaminants can be found in some areas. Sediments can also serve as a reservoir (or source) of these chemicals in a dynamic environment to which aquatic organisms, wildlife and people may be exposed.

The St. Louis River Community Action Committee (CAC) published 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). In 1995, the U.S. Environmental Protection Agency (EPA) collected sediment data from the lower St. Louis River for the Regional Environmental Monitoring and Assessment Program (REMAP; US EPA, 1995). Sample locations were chosen randomly, after eliminating known areas of contamination. See Attachment 1 for an image of the lower St. Louis River and the REMAP sample locations. Samples were analyzed mercury, PAHs, organic carbon and a number of other chemicals. REMAP data are used in some portions of this Public Health Consultation (PHC). Two 1997 joint reports from the Minnesota Pollution Control Agency (MPCA) and the 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) (US EPA and MPCA, 1997b; 1997a). The MPCA has also conducted sediment sampling for metals and organic compounds in many areas of the lower St. Louis River. These data are available from the EPA and MPCA, respectively. In addition, more recent data from the St. Louis River are available at the MPCA St. Louis River Area of Concern (AOC) website: http://www.pca.state.mn.us/index.php/water/water-monitoring-and-reporting/contaminated-sediments/sediment-studies-st.-louis-river-area-of-concern.html?menuid=&redirect=1

The Minnesota Department of Health (MDH) has reviewed data from 2 large historic industrial sites in the Lower St. Louis River: 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 (US EPA, 2003). The US Steel site located in the Morgan Park area of Duluth, Minnesota began operation in 1915. The facilities onsite 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 on the site at different times. In addition, from about 1918 until 1929, benzene and toluene were produced on the 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; Superfund) by EPA under a single Comprehensive Environmental Response, Compensation, and Liability Information System (CERCLIS) 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 discharges from 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 Organization of the Public Health Consultation and Development of Sediment Screening Values for US Steel

The Public Health Consultation (PHC) is divided into 3 parts: the main document (referred to as the Public Health Consultation), the Appendices (A-J plus appendices references), and the Attachments (one figure and four aerial photographs of the site).

The PHC has a Glossary of Acronyms and Abbreviations (pages vi-viii), an Executive Summary (pages ix-xii), Conclusions (page 45), Recommendations (pages 45-46) and a Public Health Action Plan (page 46). The Table of Contents (pages iii-iv) is reasonably detailed and, along with the List of Appendices (page v), can guide the reader to sections of interest. Generally, the body of the PHC discusses potential exposures, potential chemicals of concern in St. Louis River sediments, data that were used in the Sediment Screening Model (SSM), how the Sediment Screening Values (SSVs) were determined, and how to modify SSM results (or the SSV) if a route of exposure for a specific chemical is eliminated. The Appendices contains a Glossary of

Acronyms, Abbreviations and Equation Variables, the equations used in the SSM, recommended sediment and fish tissue detection limits, and additional information on mercury bioaccumulation, organic carbon, and polycyclic aromatic hydrocarbon (PAH) biota-sediment accumulation factors (BSAFs). References cited in the PHC and the Appendices are included at the end of the PHC and Appendices, respectively.

A Sediment Screening Model (SSM) developed by MDH in 2002 was used to calculate human-exposure-related Sediment Screening Values (SSVs) in December 2002 at the request of MPCA and U.S. Steel. The SSVs were used at the US Steel Site in the St. Louis River (MDH, 2002). The 2002 SSVs were updated and published in a memo in 2005 (MDH, 2005). Differences between the values presented in this document and those developed in 2002 and 2005 reflect new health-based toxicity values and changes that resulted from corrections and improvements to the methods discussed below. For comparison, the 2005 SSVs are listed in the Table in the Executive Summary of this PHC.

Six different routes of exposure were evaluated in the SSM: surface water ingestion, sediment ingestion, dermal surface water exposure, dermal sediment exposure, inhalation and fish consumption. Along with developing screening values from the SSM, the relative contributions of various routes of exposure were calculated for each chemical. This PHC describes the methods and default parameters used in the SSM. Equations used in calculations are described in the Appendices. All variables are defined in Appendix A.

Note that this PHC evaluates the effect of sediment contaminants on *human* health alone and does not address their effects on aquatic plants and animals, or other wildlife. The effects of exposure to contaminants on aquatic organisms are often more severe than their impacts on people, and criteria developed for environmental protection may be more restrictive. As a result, it is necessary to compare sediment concentrations with ecological metrics or criteria in order to evaluate the impact of sediment contamination on the environment.

The MPCA developed Sediment Quality Targets (SQTs) for protection of sediment dwelling organisms (MPCA, 2007). SQTs are available for a number of inorganic and organic compounds. Prior to finalizing an SSV for a chemical, the multi-route SSM result was compared with the SQT for the chemical. If the SQTs was 10 times as restrictive or more than the proposed SSV for a specific chemical, an SSV was not incorporated. Additional discussion of the differences between the SQTs and the SSVs developed in this PHC are discussed in Section 7.1.1.

3 Exposure to Contaminants

The lower half of the St. Louis River estuary has been an industrial area for over 100 years. Currently 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 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.

Numerous activities can lead to exposure to chemicals in sediments. For screening purposes, it should be assumed that even small areas of contaminated sediments may be located in areas frequented for recreational purposes. By wading or swimming in contaminated areas, people may be exposed directly to contaminated sediments, to contaminated suspended sediments and to contaminated water. Chemicals in the sediments and water may be incidentally ingested or absorbed through the skin (dermal exposure). Some contaminants may be volatile, and people in or very near the water may inhale them. In addition, some of these chemicals (e.g., PCBs, methylmercury) are readily taken up by aquatic organisms, but only slowly metabolized and excreted. If people eat these organisms, they consume contaminants that have accumulated in the edible tissues.

Human-health-based sediment values are derived by assuming reasonable, maximal exposures (RMEs) for a hypothetical individual to chemicals in sediments. These exposures are calculated using the sum of the internal doses from both direct and indirect exposure to sediments. Direct exposures include ingestion, dermal contact, and 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 SSM calculates screening sediment concentrations for the 6 potential routes of exposure and then combines these route-specific screening values into a total exposure screening value. The percent contribution of individual routes of exposure for each chemical is calculated as well.

Direct exposure to contaminated sediments usually occurs while swimming and wading in water during the summer. The MPCA and MDH developed RMEs for wading and swimming in the St. Louis River; discussed in detail below. MDH also developed an indirect RME for consumption of contaminated fish tissue that accounts for individuals who are not subsistence fishers, or who do not eat large amounts of river-caught fish. Wild rice may be harvested in parts of the St. Louis River, but MDH is unaware of any harvesting in the lower St. Louis River. Further, MDH is not aware of any other aquatic organisms that are consumed from the St. Louis River. Crayfish may be a small portion of some individuals' diet, but there are no available data on consumption or contamination of crayfish. In the SSM, exposure to contaminants from consumption of non-fish organisms is accounted for in a relative source contribution (RSC) adjustment, and indirect exposures are limited to fish ingestion.

Modeled exposures were grouped into 4 age ranges: birth through 1 year-old; 2 through 5 years; 6 through 15 years; and adults (assumed to be 16 through 32 years). The sum of these potential exposures is 33 years; which is the 90^{th} percentile estimate for living in one's current home (US EPA, 1997a). The SSM assumes exposed individuals have mean body weights and skin surface areas for the age groups represented (see Table 1).

Table 1 - Receptor Characteristics

	Potential Exposure	Averaging time	Body Weight *	Body surface area *
	Years	Years	kg	cm^2
Chronic Exposures	0 - 1	2	9.62	4,650
	2 - 5	4	17.4	7,230
	6 - 15	10	44.3	13,400
	16 - 32	17	77.5	20,000

* Mean for age range - (US EPA, 2011)

3.1 Type of sediments evaluated

Sediments are materials that sink to the bottom of waterbodies. Upland sediments include materials that are exposed to air, but were at one time covered with water (e.g., those within flood plains and areas above normal tides). Materials present in intertidal zones, or shallow areas and beaches that are under water at different times (such as parts of days or years) are also sediments. The only type of sediments evaluated using the SSM are those typically covered by water.

Exposures to each of these sediment types are likely to be different. For example, if a child is playing on the shore of a lake or river, their exposure to upland sediments are likely to be similar to exposures to soil in a sandbox or at a playground. Similarly, because dried sediments above flood elevations will not affect the concentration of sediment-associated contaminants in fish tissue, upland sediments should be evaluated as if they are soils.

The highest exposures to soils are likely to include exposure to intertidal sediments. Where the water meets the shore, children often play in a medium that more resembles mud than water or dry soil. As children play in increasingly 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.

A described above, this PHC only calculates SSVs for sediments that are typically covered by water. Although exposures to intertidal and water-covered sediments may be similar at times, it is appropriate in most cases to evaluate intertidal sediments differently from either sediment or soils. However, if the SSM is used to evaluate intertidal sediments, MDH recommends: increasing the sediment ingestion by a factor of 4 to adjust ingestion to approximate soil ingestion levels (US EPA, 1997a); adjusting the dermal-sediment adherence and contact area to reflect potential exposure to mud, and; adjusting the dermal-water exposure to reflect exposures of a child playing in the intertidal zone. In addition, the frequency of exposure may also need to be adjusted.

3.2 Frequency of contact with sediments

Direct contact with chemicals from sediments (i.e., 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, which are based on discussions with residents. During the summer, children may swim twice per day, but most exposures are assumed to occur once per day and only 2-4 days per week. The duration of each event, wading or swimming, is assumed to be ½ hour. Expected exposures at other sites in Minnesota may be considerably different.

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 (US EPA, 2002b). This level of activity is more appropriate for climates warmer than Minnesota. RME scenarios used in this PHC are shown in Table 2.

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
0 - 1	1	1	0	0
2 - 5	1	2	0	0
6 - 15	1	2	0	0
16 - 32	1	2	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
0 - 1	0	0	2	2
2 - 5	0	0	2	4
6 - 15	0	0	2	4
16 - 32	0	0	1	2

3.3 Default exposure assumptions

General default exposure 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 effects at higher levels of exposure (e.g., benzene has an acute MDH Health Risk Value of 1,000 micrograms per cubic meter of air based on a developmental endpoint). While acute workplace and first-responder exposure limits are available for many chemicals, very few acute toxicity criteria exist that are protective of public health. Furthermore, because calculating acute SSVs would require an additional layer of complexity, acute SSVs are not calculated in this PHC. They may need to be addressed in a future document.

Exposures that can lead to chronic health impacts typically occur over periods of months to years. Chronic values developed in the SSM are for yearly average exposures. For non-cancer endpoints, children (ages 2-5) have the potential to be the most highly exposed group (Tables 3 -

8; see Appendix B for calculations). Therefore, data related to exposure of 2-5 years old are used to calculate 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 (US EPA, 1997a). For the purpose of evaluating cancer risk, it is assumed that 33-year exposures (all age groups in Table 1) are the total lifetime exposure. By convention, cancer risk is determined by averaging lifetime exposures over a 70 year lifetime (US EPA, 1989). Therefore, lifetime average exposures for the purpose of estimating cancer risk assume a total of 33 years of exposure over a 70 year lifetime (Tables 3-8).

3.3.1 Coordinating life-stage exposures and life-stage sensitivity to carcinogens

Exposures to chemicals in the environment are different for children than they are for adults. In addition, it has been shown that laboratory animals are more sensitive to exposure to many carcinogens when they are young (US EPA, 2005). MDH recommends applying a default early-life sensitivity adjustment to carcinogens for which there are not specific early-life sensitivity data available (MDH, 2010). The recommended default early-life sensitivity adjustments are 10 times and 3 times the adult sensitivity for children up to 2 and 16 years of life, respectively. Age-dependent exposures and sensitivities to carcinogens are coordinated in this PHC by incorporating age-dependent adjustment factors (ADAFs) into the lifetime exposure calculations. This ADAF-adjusted lifetime exposure replaces the actual lifetime exposure calculation in appropriate carcinogenic SSM calculations. Among the chemicals evaluated in this PHC, arsenic is the only carcinogen where early-life sensitivity was included in the calculation of potency and, therefore, an ADAF was not applied.

3.3.2 Surface water ingestion

Swimming and wading typically result in incidental ingestion of some water. The Superfund Exposure Assessment Manual (US EPA, 1988) states that 50 milliliters per hour (mL/hr) swimming is a reasonable estimate for incidental water ingestion by an adult. Because children playing in water ingest considerably more water than adults, the SSM assumes that children and adolescents ingest five times the adult ingestion. In addition, there are no published data on water ingestion during wading. The SSM assumes that a reasonable adult ingestion during wading 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 0 - 15 year-olds is $1/10^{th}$ of their swimming ingestion (see Table 3). Using these data, assumed body weight (Table 1) and event frequency (Table 2), average daily surface water intake rates were calculated with *Equation A-1*, Appendix B. Results are shown in Table 3. Average daily surface water intake rates for evaluating carcinogenic chemicals were calculated using *Equations A-4*, Appendix B.

Table 3 - 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	0 - 1	25	8.60	250	51.6	0.5	0.00187	
	2 - 5	25	17.2	250	103		0.00206	
	6 - 15	25	17.2	250	103		0.00081	
	16 - 32	0.50	17.2	50 *	25.8		2.3E-05	
Lifetime Yearly Average Exposures	0 - 69						0.000292	SW_{Ing-c}
							0.00124	$SW_{Ing-c-ADAF}$

SW_{Ing} = water ingested (Appendix *Equation A-1*) {L/(kg·d)}
SW_{Ing-c} = water ingested - lifetime average (Appendix *Equation A-4*) {L/(kg·d)}
$SW_{Ing-c-ADAF}$ = water ingested - ADAF-adjusted - lifetime average (Appendix *Equation A-4*) {L/(kg·d)}
* (US EPA, 1988)

3.3.3 Sediment ingestion

EPA *Risk Assessment Guidance for Superfund* (US EPA, 1989) recommends using soil ingestion rates as sediment ingestion rates in risk assessments. Therefore, on an event day a child may ingest about 200 mg of intertidal or upland sediments(US EPA, 2002a). Arguably, 200 mg/event-day may be a high estimate of the amount of submerged sediments ingested by a child playing in water. Because this PHC is calculating screening values for sediments under water only, the calculated amount of sediment ingested per event-day is limited to the amount of water that may be ingested multiplied by the concentration of suspended sediment in the water (*Equation A-2*, 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 3.

The responsible party at the SLRIDT 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 upper 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 3) are used in *Equation A-2*, Appendix B, to calculate the modeled sediment ingestion rate ($Ing-Sed_{(wad,swm)}$ $\{mg_{sed}/hr\}$). The modeled sediment ingestion for 2-5 year-olds is 92.5 mg/hr and 9.25 mg/hr (see example; *Equation A-2*, Appendix B). Considering a maximum exposure in the SSM is 2 - ½ hour events, the average daily sediment ingestion is considerably lower than the EPA estimated daily ingestion rate of 200 mg/day for a young child playing outdoors (US EPA, 2002a).

The daily sediment ingestion rate for swimming and wading, body weight (Table 1), event frequency (Table 2) and event duration (Table 4) are used in *Equation A-3*, Appendix B, to calculate sediment ingestion rates. Average daily sediment ingestion rates (Sed_{Ing}) calculated in the SSM are listed in Table 4. Average daily sediment intake rate for evaluating carcinogenic chemicals (Sed_{Ing-c}, $Sed_{Ing-c-ADAF}$) were calculated using *Equation A-5*, Appendix B.

Table 4 - Sediment Ingestion

	Potential Exposure	Wading		Swimming		Suspended Sediment Concentration	Event Duration	Sed_{Ing}	
	Years	mL / hr	events / yr	mL / hr	events / yr	mg/L	hr/event	$mg_{sed}/(kg_{bw} \cdot d)$	
Chronic Exposures	**0 - 1**	25	8.60	250	51.6	370 *	0.5	0.691	
	2 - 5	25	17.2	250	103			0.763	
	6 - 15	25	17.2	250	103			0.3	
	16 - 32	0.50	17.2	50	25.8			8.5E-03	
Lifetime Average Exposures	**0 - 69**							0.108	Sed_{Ing-c}
								0.459	$Sed_{Ing-c-ADAF}$

Sed_{Ing} = sediment ingested (Appendix *Equation A-3*) $\{mg_{sed}/(kg_{bw} \cdot d)\}$
Sed_{Ing-c} = sediment ingested - lifetime average (Appendix *Equation A-5*) $\{mg_{sed}/(kg_{bw} \cdot d)\}$
$Sed_{Ing-c-ADAF}$ = sediment ingested – ADAF-adjusted lifetime average (Appendix *Equation A-5*) $\{mg_{sed}/(kg_{bw} \cdot d)\}$
* 75% C.L. mean of SLRIDT Site data (IT Corporation, 1997)

3.3.4 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. Guidance published by EPA (US 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 be expected to adhere to the skin and may not be removed without active washing.

Dermal exposure to a chemical in sediment occurs by the non-active transfer into the body of a fraction of chemical from sediment that adheres to skin. A sediment film covering the skin will typically stay on the skin until it is washed away with soap. This PHC assumes as a default 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) (US 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 number of exposures during one year equals the number of event-days).

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

The EPA Exposure Factors Handbook (US EPA, 1997a) was used as a reference for the 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	0 - 1	20%	1 *	8.60	90% **	0.2	25.8	8.42	
	2 - 5			17.2			51.6	14.5	
	6 - 15			17.2			51.6	10.5	
	16 - 32			17.2		0.07	25.8	3.57	
Lifetime Yearly	0 - 69							3.44	Sed_{Derm-c}
								10.3	$Sed_{Derm-c-ADAF}$

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

3.3.5 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 non-actively transferred through the skin and into the body. Exposure only occurs while the event is occurring. Therefore, more than one event during a single day results in more than one exposure. Uncertainties 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	0 - 1	20%	8.60	90% *	51.6	63.7	
	2 - 5		17.2		103	109	
	6 - 15		17.2		103	79.4	
	16 - 32		17.2		25.8	18.8	
Lifetime Yearly Average Exposures	0 - 69					24	SW_{Derm-c}
						75.5	$SW_{Derm-c-ADAF}$

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

3.3.6 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 multiplied by 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	0 - 1	8.60	51.6	0.5	0.00344	
	2 - 5	17.2	103		0.00686	
	6 - 15	17.2	103		0.00686	
	16 - 32	17.2	25.8		0.00245	
Lifetime Yearly Average Exposures	0 - 69				0.00207	Inh_{frac-c}
					0.00569	$Inh_{frac-c-ADAF}$

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

3.3.7 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 (US EPA, 2000). 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). For this PHC, a fish consumption rate for an adult of 30 g/d is assumed. This intake rate is based on the presumed ingestion of a single 210 g meal per week by a 150 pound 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	$Fish_{Ing}$	
	Years	meals / wk	grams / meal	$g_{fish}/(kg_{bw} \cdot d)$	
Chronic Exposures	0 - 1			0.22	
	2 - 5			0.441	
	6 - 15			0.441	
	16 - 32	1	210	0.441	
Lifetime Yearly Average Exposures	0 - 69			0.202	$Fish_{Ing-c}$
				0.435	$Fish_{Ing-c-ADAF}$

$Fish_{Ing}$ = fish ingestion rate (Appendix *Equation A-12*) $\{g_{fish}/(kg_{bw} \cdot d)\}$
$Fish_{Ing-c}$ = fish ingestion rate - lifetime average (Appendix *Equation A-13*) $\{g_{fish}/(kg_{bw} \cdot d)\}$
$Fish_{Ing-c-ADAF}$ = fish ingestion rate - ADAF-adjusted - lifetime average (Appendix *Equation A-13*) $\{g_{fish}/(kg_{bw} \cdot d)\}$

4 Potential Chemicals of Concern

Potential chemicals of concern can be categorized as metallic compounds, inorganic and organic compounds. Most 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 (activity of hydrogen ion) 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 or 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 has been 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 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 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 that are persistent in the environment 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 (US EPA, 1998b). These substances are priority contaminants to be addressed during environmental sampling and remedial action in the Great Lakes Region.

This PHC addresses contaminants that may have been used or produced, intentionally or as a byproduct at the US Steel facility in Morgan Park. From the list of 12 BNS chemicals, these include: benzo[a]pyrene, hexachlorobenzene, mercury and compounds, octachlorostyrene, PCBs, and dioxins and furans. Persistent and/or bioaccumulative chemicals that are not addressed in this PHC 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 chlorinated naphthalenes), polybrominated dibenzo-p-dioxins (PBDDs) and polybrominated dibenzofurans (PBDFs). SSVs could be developed for a number of these chemicals if they are site-specific contaminants of concern.

4.1 Inorganic chemicals and metals

Calculating exposure to metals from all six routes-of-exposure addressed in this PHC 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 phase) and total metal in sediment (solid phase) 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, thereby shifting equilibrium and possibly increasing or decreasing the availability of the metal. The magnitude 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. 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 (US 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 PHC are data from Class 1 and Class 2 cores. Note that REMAP data are not background concentrations (background in this PHC, unless otherwise noted, is not meant to describe pre-anthropogenic background, but signifies chemical concentrations without known local or regional sources), but the data set may be a good representation of the ambient concentrations in areas that are not adjacent to local potential sources of contaminants. In dynamic systems such as rivers and waterbodies affected by seiches, contaminants can accumulate far from the sources of contamination. REMAP mercury data (see Appendix C) appear to agree with upstream data from Glass et al. (1999), suggesting that the background concentration of mercury in the St. Louis River is 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 RETrieval database) reviewed by MPCA in 2005). 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 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 PHC.

4.1.2 Cadmium

A till survey (1-2 meter depth) of 250 sites throughout MN (MNGS, 2007) showed a mean cadmium concentration of 0.28 mg.kg (standard deviation [SD] = 0.20). The mean concentration of cadmium in soils worldwide is about 0.5 mg/kg (Kabata-Pendias and Pendias, 2001). Cadmium concentrations may 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 have been 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. People 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 PHC 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 MPCA ecotoxicity cadmium SQTs of 0.99 and 5 mg/kg (Level 1 and Level 2 SQTs, respectively) shown in Table 14 will be more restrictive than human health-based values.

A joint EPA, MPCA study (1993 Mudpuppy Study; US EPA and MPCA, 1997b) found cadmium in St. Louis River sediment between 0.52 and 7.4 mg/kg (mean = 2.3, SD = 1.4, 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).

4.1.3 Lead

A Minnesota Geological Till Survey (MNGS, 2007) showed a mean lead concentration for samples (1-2 m depth) from 250 sites throughout MN to be 14.7 mg/kg (SD = 6.2). A study by Boerngen and Shacklette (Boerngen and Shacklette, 1981) showed mean subsurface soil concentrations of lead in rural areas of Minnesota of 13.5 mg/kg (SD = 3.4; 37 samples, approximately 8 inch depth). 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. As noted in 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 PHC. Because there is no known threshold exposure below which lead has no effect, lead exposure should be minimized when reasonably possible. In the absence of more specific criteria, it is reasonable to apply soil criteria to sediment as recommended by EPA (US EPA, 1989). The current Minnesota Soil Reference Value (SRV) for lead is 300 mg/kg (MPCA, 2009). This SRV has been incorporated as an SSV.

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.

Recently the Center for Disease Control and Prevention (CDC) recommended lowering the childhood blood lead level at which public health actions are initiated from 10 µg/dl to 5 µg/dl (CDC, 2012 and http://www.cdc.gov/nceh/lead/ACCLPP/blood_lead_levels.htm). At 300 mg/kg soil concentrations and standard default exposure parameters, the EPA Integrated Exposure Uptake and Biokinetic Model (IEUBK; Win 1.1) calculates that about 25% and 1.5% of infants will have blood leads above 5 µg/dl and 10 µg/dl, respectively. At 100 mg/kg soil concentrations

and standard default exposure parameters, about 1.5% and 0.013% of infants may be anticipated to have blood leads above 5 µg/dl and 10 µg/dl, respectively. It is likely that a protective ecological sediment value (e.g., MPCA Level 1 and 2 SQTs of 36 and 130 mg/kg, respectively) will be more restrictive than a value based on public health policy.

The EPA, MPCA 1993 'Mudpuppy' Study (US EPA and MPCA, 1997b) examined lead concentrations near potential sources and found a range of lead concentrations in sediment between 1.5 and 548 mg/kg (mean = 58.3, SD = 105, 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.

4.1.4 Arsenic

The Minnesota Geological Till Survey (MNGS, 2007) showed a mean arsenic concentration in samples from 250 sites (1-2 m depth) in MN to be 8.5 (SD = 4.2). Mean concentration of arsenic at 8 inch depth soil throughout Minnesota is 5.5 mg/kg (Boerngen and Shacklette, 1981; SD = 4.6 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 wood treated with chromated copper arsenate (CCA) can have very high concentrations of arsenic, chrome and copper. Arsenic may also be found in high concentrations in some 'natural' soil supplements.

The 1993 'Mudpuppy' Study (US EPA and MPCA, 1997b) studied 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, SD = 8.3, 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 somewhat 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 Volatile organic compounds

A considerable amount of volatile organic compounds (VOCs) were produced at the US Steel site. Contribution from 5 routes of exposure to benzene, ethyl benzene, styrene, toluene and xylenes (mixed) were calculated in the SSM. Exposure through fish consumption was not addressed because estimating VOC concentrations in fish tissue in the wild is highly uncertain. Human exposure to VOCs by fish consumption is likely to be a very limited route of exposure.

Dermal absorption fractions (ABS_{derm}) from soil for VOCs are not available. The dermal absorption fraction was set to 0.5 for all VOCs as a default to allow the SSM to calculate a dermal sediment SSV ($SSV_{Sed(derm)}$). The result is likely to be very conservative and is included to suggest a maximum route-specific contribution for VOCs. The contribution of direct dermal sediment exposure to the total VOC exposure from sediments appears to be minimal.

Inhalation of VOCs from contaminated, submerged sediments may be an important route of exposure for many VOCs at contaminated sediments sites. However, exposure estimates are very uncertain. If VOC SSVs are exceeded, surface water concentrations should be measured and compared with water concentration limits calculated with *Equations A-17* (non-cancer, ingestion), *A-37* (cancer, ingestion), *A-20* (non-cancer, dermal), *A-40* (cancer, dermal), *A-33* (non-cancer, inhalation), and *A-43* (cancer, inhalation).

4.3 Persistent organic compounds

Historic human activity in the St. Louis River area has deposited persistent organic compounds in sediments. Canada and the United States developed a Binational Toxics Strategy (BNS) to address many of these toxicants. Not all of the BNS targeted substances have been evaluated; however, the methods used in the SSM can be used to calculate similar values for other persistent organics.

Modeled concentrations of organic contaminants in fish tissue can be inaccurate because models rely on empirical data from specific sites for biota-sediment accumulation factors (BSAFs). Site topography, food chain structure and species-specific metabolism of different organic chemicals at sites may differentially impact accumulation in fish.

Organic pollutants evaluated in the SSM for all 6 routes of exposure are: polychlorinated dibenzo-p-dioxins and polychlorinated dibenzofurans, polychlorinated biphenyls, hexachlorobenzene, octachlorostyrene, polycyclic aromatic hydrocarbons. Carbazole, pentachlorophenol, and quinolone were not evaluated for fish consumption, although they were evaluated for other exposure routes.

4.3.1 Chlorinated organics

4.3.1.1 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., 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 like dioxins and have been identified by the World Health Organization's (WHO) as toxicologically "dioxin-like" (Van den Berg et al., 2006).

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 mixtures of PCBs, PCDDs and PCDFs to which people are exposed as probable or likely human carcinogens. Individual congeners and 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 use of the WHO 2005 toxic equivalency factor (TEF_{WHO05}) scheme (Van den Berg et al., 2006) 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 individual chemical in this group. Total 2,3,7,8-TCDD Toxic Equivalency (TCDD-TEQ) exposure concentration is equal to:

$$\text{TCDD-TEQ } \{ mg_{sed}/(kg_{bw} \cdot d) \} = \sum Exp_i * TEF_i \quad \text{for i = each chemical with a } TEF_{WHO05} \qquad \textit{Equation 1.}$$

Where:

Exp_i = the exposure concentration for each dioxin or dioxin-like compound {mg/(kg·d)}
TEF_i = the TEF_{WHO05} 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/chemical characteristics of dioxin-like chemicals can differ, partitioning between sediment and water can vary for different congeners. For example, the octanol-water coefficient (K_{ow}) of 2,3,7,8-TCDD is about 10 times less than for 1,2,3,4,7,8-HxCDD (see Table 9 below). Therefore, the ratio between sediment organic carbon (OC) and water for 2,3,7,8-TCDD is expected to be about 10 times greater than for HxCDD, resulting in a 10-fold difference in estimates of the concentrations 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 site-specific exposure. 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 PHC. (For additional information on the toxicity of dioxin and dioxin-like compounds please see MDH, 2003b; 2003a).

The TEF_{WHO05} values and partition information (Log K_{ow}) are listed in Table 9 below.

Table 9 - TCDD-TEFs

Compound	TCDD-TEF$_{WHO2005}$*	Log K_{ow}**
Polychlorinated Dibenzo-p-dioxins (PCDDs)		
2,3,7,8-TCDD	1	6.8
Other TCDD	0	
1,2,3,7,8-PeCDD	1	6.64
Other PeCDD	0	
1,2,3,4,7,8-HxCDD	0.1	7.8
1,2,3,6,7,8-HxCDD	0.1	8.21
1,2,3,7,8,9-HxCDD	0.1	8.21
Other HxCDD	0	
1,2,3,4,6,7,8-HpCDD	0.01	8.2
Other HpCDD	0	
1,2,3,4,6,7,8,9-OCDD	0.0003	8.2
Polychlorinated Dibenzofurans (PCDFs)		
2,3,7,8-TCDF	0.1	6.53
Other TCDF	0	
1,2,3,7,8-PeCDF	0.03	6.92
2,3,4,7,8-PeCDF	0.3	6.92
Other PeCDF	0	
1,2,3,4,7,8-HxCDF	0.1	7.92
1,2,3,6,7,8-HxCDF	0.1	7.92
2,3,4,6,7,8-HxCDF	0.1	7.92
1,2,3,7,8,9-HxCDF	0.1	7.58
Other HxCDF	0	
1,2,3,4,6,7,8-HpCDF	0.01	7.92
1,2,3,4,7,8,9-HpCDF	0.01	7.92
Other HpCDF	0	
1,2,3,4,6,7,8,9-OCDF	0.0003	8.6
Polychlorinated Biphenyls (cPCBs)		
Structure (IUPAC#)		
3,3',4,4'-TCB (77)	0.0001	6.63
3,4,4',5-TCB (81)	0.0003	6.34
2,3,3',4,4'-PeCB (105)	0.00003	6.5
2,3,4,4',5-PeCB (114)	0.00003	6.98
2,3',4,4',5-PeCB (118)	0.00003	7.12
2',3,4,4',5-PeCB (123)	0.00003	6.98
3,3',4,4',5-PeCB (126)	0.1	6.98
2,3,3',4,4',5-HxCB (156)	0.00003	7.6
2,3,3',4,4',5'-HxCB (157)	0.00003	7.6
2,3',4,4',5,5'-HxCB (167)	0.00003	7.5
3,3',4,4',5,5'-HxCB (169)	0.03	7.41
2,3,3',4,4',5,5'-HpCB (189)	0.00003	8.27

* (Van den Berg et al., 2006)

** (US EPA, 2012b) (unshaded are experimental data; shaded are estimated)

Background concentrations of dioxin-like compounds in sediments are also characterized as TCDD-TEQs. The EPA Dioxin Reassessment (US EPA, 2003) states that background TCDD-TEQs for US lakes (11 lakes and reservoirs) are between 0.12 and 15.4 ng/kg, with a mean of 4.7 ng/kg (total PCDDs / PCDFs from 9.1 to 2,916 ng/kg; total cPCBs from 83 to 2159 ng/kg)

(Cleverly, David, 2004). Note that TCDD-TEQs calculated prior to 2005 used different TEFs that are defined in Van den Berg (1998). The sediment concentrations found in the 11 lakes are well above the SSVs calculated in this PHC. Therefore, local or regional background concentrations may be more reasonable site-specific SSVs.

The 1993 'Mudpuppy' Study (40 sample locations specifically targeting potential source areas) evaluated 2,3,7,8-TCDD and 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF) (US EPA and MPCA, 1997b). 2,3,7,8-TCDD was detected at 4 locations (mean 6.4 ng/kg; range 0.9 - 13 ng/kg). 2,3,7,8-TCDF 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, D., 1996; US EPA, 2003), 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 backgroun, because sediment samples were taken from areas with identified point sources. The 11 Lakes Study shows that total dioxins in sediment in the lakes and reservoirs studied contribute between 28% and 74% of the total TCDD-TEQ (WHO_{1998} TEFs), 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 (0.12 to 15.4 ng/kg). Furthermore, the data suggest that background concentrations of PCDD/PCDF 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, sediment deposition rates may also impact chemical concentrations. It is important to note that the estuary is not a lake and background concentrations within a riverine system and a system subject to seiches or tides may be different. Additional sediment data are becoming available, from which it may be possible to further evaluate background dioxin levels and sources.

4.3.1.2 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 PCB 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. An alternative explanation is that total solid deposition to the Siskiwit Lake sediments was shown to be about 0.19 cm per year. Because the source of PCBs to the lake is likely aerial deposition, low solid sedimentation rates may result in relatively high PCB concentration in sediments.

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 (US EPA and MPCA, 1997b). In addition, these 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 PCDD/PCDF 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" (US EPA and MPCA, 1997b) 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.

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 PCB analysis can give congener, mixture and/or homologue data. Congener analysis results in the most useable data, because concentrations of all congeners can be summed for tPCB evaluation, or the toxicity of individual dioxin-like PCBs can be weighted using a toxic equivalence (TEQ) method. 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 data which quantify risk from exposure to homologue concentrations or doses does not exist. Bioassay analyses, while potentially useful for evaluating TCDD-TEQs (including the contribution of cPCBs), are not available for tPCB analyses. Therefore, tPCB and cPCB evaluation require congener analysis.

4.3.1.3 Chlorinated organics: hexachlorobenzene, octachlorostyrene and pentachlorophenol
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 pulp and paper production (US EPA, 1998a). In addition, chemicals such as HCB and OCS may have been used, or can be accidentally formed by industrial processes including coking and iron and steel production (US EPA, 1998a; 1999).

HCB and OCS are of potential concern in the lower St. Louis River because they 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) (US EPA and MPCA, 1997b). 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). Ambient and background OCS sediment concentration data are not readily available.

Pentachlorophenol is a fungicide and insecticide that has been commonly used as a wood preservative. Other uses have been as a pre-emergence herbicide, as a preharvest defoliant in cotton and as an industrial water biocide. Pentachlorophenol has historically been contaminated with dioxins. Pentachlorophenol is considered to be moderately persistent in soil and water. MDH has not reviewed sediment or water data for pentachlorophenol in the St. Louis River Estuary.

4.3.2 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 present in non-combusted fossil fuels (petrogenic). PAHs exist in the environment as complex mixtures. While the toxicity to humans has been quantified for only a few individual PAHs, PAHs, in general, are considered to affect the liver (Sipes and Gandolfi, 1991). They have also been shown in laboratory animals to cause decreased fertility, developmental neurological effects and renal toxicity at relatively high exposures. Additionally, PAH 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 individual PAHs have been identified as likely or probable human carcinogens by the EPA (US EPA, 2010; 2012a), the International Agency for Research on Cancer (IARC, 2010), the National Toxicology Program (NTP, 2011), and the California EPA Office of Environmental Health Hazard Assessment (CA OEHHA, 2002). Cancer slope factors (CSFs) or

relative potency factors (RPFs) have been developed for many PAHs including the 34 carcinogenic PAHs (cPAHs) listed in Table 10.

In order of preference, MDH recommends using site-specific mixture potency, surrogate mixture potency (use of data from similar mixtures), or individual compound potency equivalence (component evaluation) for evaluating the cancer potency of PAH mixtures. However, site-specific mixture potency is not typically known due to the expense of conducting appropriate toxicity studies for a site. Furthermore, while surrogate mixtures may be used for characterizing the potency of a PAH mixture, there can be rather large differences in mixture composition among samples from similar sources. Therefore, in practice it may be difficult to determine an appropriate surrogate mixture. A single potency for industrial pyrogenic PAH mixtures is outlined below as a policy alternative. However, in general, a potency equivalence approach may be the most useful alternative for evaluating the cancer potency of PAH mixtures.

Given the industrial processes at US Steel, it is very likely that a large portion of the PAH mixture on the site is coal tar or a similar mixture. MDH has recommended that the cancer potency of PAHs in coal tar be considered as 10 times the potency that is suggested by the concentration of benzo[a]pyrene (BaP) alone in the mixture (MDH, 2011). However, coal tar potency was determined from 2 coal tar mixtures (Culp et al., 1998) with somewhat different cPAH composition from the coal tar found in sediments associated with the US Steel site (MDH, 2006). Furnaces and coal-burning conditions at various facilities are likely to be different, and weathering of emissions in the environment may have considerable effect on the ratios of PAHs. Similarity between mixtures is likely to be relatively subjective. However, an analysis of the carcinogenic potency of industrial PAH mixtures by Schneider et al. (2002) suggests a potency of 10 times BaP may be reasonable as a generally applied potency for industrial PAH mixtures in contaminated soils. Using a cPAH potency of 10 times the BaP concentration in US Steel associated PAH mixtures seems to be reasonable.

If whole mixture evaluation (site-specific or surrogate) is not feasible, component evaluation of cPAHs in the sediments should be conducted. Chemical analysis of cPAHs in complex mixtures is difficult. Note in Table 10 that 7 cPAHs have molecular weights (MWs) of 252 and 6 cPAHs have MWs of 302. In addition, there are numerous non-carcinogenic PAHs (nPAHs) with identical MWs. MDH recommends analyzing for a limited number of the more potent cPAHs as a way of minimizing the analytical requirements while maximizing the amount of information obtained about the mixture potency. Since 2001 MDH has been recommending analyzing for the California Office of Environmental Health Hazard Assessment (OEHHA) list shown in Table 10 (MDH, 2001a). However, MDH is currently in the process of changing that recommendation. The 18 cPAHs listed as "Priority" cPAHs in Table 10 are MDH Draft priority analytes. "Secondary" cPAHs in Table 10 are additional PAHs for which relative potency factors have been published. However, they are excluded from the draft MDH list of analytes at this time because laboratory standards are not easily available; impact on the BaP Potency Equivalents (BaP-PEQ – described below) is likely to be minimal; and/or data on potency are limited. MDH anticipates finalizing cPAH draft recommendations in 2013.

Table 10 - BaP-RPFs

Polycyclic Aromatic Hydrocarbons	CAS #	Molecular Weight	cPAH Lists: * - EPA-15	ǂ - EPA-RTK,TRI	¥ - OEHHA CSF	§ - OEHHA 2009 (MDH 2001)	¶ - EC "15+1"	£ - EPA Draft RPF	Relative Potency Factors (RPF)	Analyte Priority	Log K_{ow} (EPA EPI Suite)
Total # PAHs = 34			9	22	5	24	16	23		**Priority | 18** Secondary | 19	Est
Anthanthrene	191-26-4	276.34						£	0.4	**Priority**	7.04
Benz(a)anthracene	56-55-3	228.29	*	ǂ		§	¶	£	0.2	**Priority**	5.76
Benzo(a)pyrene	50-32-8	252.31	*	ǂ	¥		¶		1	**Priority**	6.13
Benzo(b)fluoranthene	205-99-2	252.32	*	ǂ		§	¶	£	0.8	**Priority**	5.78
Benzo(c)fluorene	205-12-9	216.28					¶	£	20	**Priority**	5.19
Benzo(g,h,i)perylene	191-24-2	276.34	*	ǂ			¶	£	0.009	**Priority**	6.63
Benzo(j)fluoranthene	205-82-3	252.32		ǂ		§	¶	£	0.3	**Priority**	6.11
Benzo(k)fluoranthene	207-08-9	252.32	*	ǂ		§	¶	£	0.03	**Priority**	6.11
Chrysene	218-01-9	228.29	*	ǂ		§	¶	£	0.1	**Priority**	5.81
Cyclopenta(c,d)pyrene	27208-37-3	226.28					¶	£	0.4	**Priority**	5.7
Dibenz(a,h)anthracene	53-70-3	278.35	*	ǂ	¥	§	¶	£	10	**Priority**	6.75
Dibenzo(a,l)pyrene	191-30-0	302.38		ǂ		§	¶	£	30	**Priority**	7.71
7,12-Dimethylbenz(a)anthracene	57-97-6	256.34		ǂ	¥	§			150	**Priority**	5.8
Fluoranthene	206-44-0	202.26	*	ǂ				£	0.08	**Priority**	5.16
Indeno(1,2,3-cd)pyrene	193-39-5	276.34	*	ǂ		§	¶	£	0.07	**Priority**	6.7
3-Methylcholanthrene	56-49-5	268.35		ǂ	¥	§			13	**Priority**	6.42
5-Methylchrysene	3697-24-3	242.31		ǂ		§	¶		1	**Priority**	6.07
6-Nitrochrysene	7496-02-8	273.29				§			10	**Priority**	5.34
11H-Benz(b,c)aceanthrylene	202-94-8	240.3						£	0.05	Secondary	
Benz(e)aceanthrylene	199-54-2	252.32						£	0.8	Secondary	
Benz(j)aceanthrylene	202-33-5	252.32						£	60	Secondary	
Benz(l)aceanthrylene	211-91-6	252.32						£	5	Secondary	
4H-Cyclopenta(d,e,f)chrysene	202-98-2	240.3						£	0.3	Secondary	5.78
Dibenz(a,h)acridine	226-36-8	279.33		ǂ		§			0.1	Secondary	5.67
Dibenz(a,j)acridine	224-42-0	279.33		ǂ		§			0.1	Secondary	5.67
Dibenzo(a,e)fluoranthene	5385-75-1	302.38		ǂ				£	0.9	Secondary	7.28
Dibenzo(a,e)pyrene	192-65-4	302.38		ǂ		§	¶	£	0.4	Secondary	7.71
Dibenzo(a,h)pyrene	189-64-0	302.38		ǂ		§	¶	£	0.9	Secondary	7.28
Dibenzo(a,i)pyrene	189-55-9	302.38		ǂ		§	¶	£	0.6	Secondary	7.28
7H-Dibenzo(c,g)carbazole	194-59-2	267.32		ǂ		§			1	Secondary	6.4
1,6-Dinitropyrene	42397-64-8	292.25				§			10	Secondary	4.57
1,8-Dinitropyrene	42397-65-9	292.25				§			1	Secondary	4.57
Naphtho(2,3-e)pyrene	193-09-9	302.38						£	0.3	Secondary	7.28
5-Nitroacenaphthene	602-87-9	199.21			¥	§			0.02	Secondary	3.85
2-Nitrofluorene	607-57-8	211.22				§			0.01	Secondary	3.37
1-Nitropyrene	5522-43-0	247.25		ǂ		§			0.1	Secondary	5.06
4-Nitropyrene	57835-92-4	247.25				§			0.1	Secondary	4.75

- RPF based on chemical's cancer slope factor (CSF), relative to BaP CSF

***** - EPA-15 - US EPA - PAHs on the Clean Water Act List of Priority Pollutants - http://water.epa.gov/scitech/methods/cwa/pollutants.cfm

ǂ - EPA RTK – Right-to-Know Act (US EPA, 2001a)

¥ - OEHHA CSF – Cancer Slope Factors available (CA OEHHA, 2002)

§ - OEHHA PEF - Potency Equivalence Factors available (CA OEHHA, 2011)

¶ - EC "15+1" - (EC Commission Regulation, 2006)

£ - EPA Draft RPF - Relative Potency Factors available (potency =0 not included) - (US EPA, 2010)

EPA EPI Suite - (US EPA, 2012b)

RPFs in Table 10 are based on individual cPAH cancer slope factors (CSFs), EPA Draft RPFs or California Potency Equivalence Factors. Note that the EPA anticipates modifying their 2010 Draft RPFs and MDH will likely update RPFs in Table 10 when new information becomes available. BaP, with a RPF of 1, is the index compound. Total BaP-PEQs are similar to TCDD-TEQs and can be calculated using an algorithm similar to *Equation 1* (Section 4.3.1) for dioxin and dioxin-like TEQs. However, RPFs and PEFs should only be used to evaluate cancer risk, as the mechanisms by which PAHs are carcinogenic may differ from the mechanisms by which they impact chronic diseases.

Detection limits used for cPAH analyses are very important. Individual Priority cPAHs have been found in environmental samples. Non-detection of these cPAHs in many environmental studies may be the result of analytical detection limits being too high relative to the concentrations which are necessary to evaluate human health risks. Recommended detection limits for screening PAHs in sediments and fish tissue are shown in Appendix E. If the detection limit for an individual cPAH cannot be achieved, cPAH potency uncertainty should be addressed.

At sites with reasonably homogeneous cPAH mixtures, it is possible to infer the cancer potency of individual samples from an accurate site-specific cPAH fingerprint and concentration data for an index PAH analyzed in each sample (typically BaP). If the cancer risk for a specific cPAH is a risk driver, further review of analytical uncertainties and cancer potency may be needed.

Application of SSVs to data from a component evaluation of cPAHs will have uncertainties similar to those described for TCDD-TEQ-based values. RPFs applied to sediment concentrations (as opposed to concentrations in the human body) do not account for differences in exposure and partitioning that may be expected for different cPAHs with different physical/chemical characteristics. For screening purposes, RPFs may be applied to sediment data, but uncertainties should be reviewed or addressed for each individual site or application.

The primary health endpoints for nPAHs vary, but most have multiple toxicity endpoints that are similar. Given the general similarity between the non-cancer effects of PAHs, MDH recommends that hazard quotients for nPAHs be added in risk assessments for PAH sites (MDH, 2001b).

Carbazole and quinolone may be loosely defined as PAHs; however, they are generally evaluated as individual semi-volatile organic compounds. Both carbazole and quinolone are found in coal tar in sufficient quantities that coal tar has been a major source of these chemicals. Both compounds are used in the production of dyes. MDH is not aware of data on their concentration in St. Louis River estuary sediments.

5 Chemical Specific Data

Chemical-specific data necessary for exposure and toxicity analyses (shown in Appendix D), 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 (used only if K_{oc} is unavailable)	{unitless}
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}
CSF - cancer slope factor	{mg/(kg·d)}$^{-1}$
UR – unit risk	{(µg/m^3)$^{-1}$}

5.1 Appropriate detection limits

MDH developed recommended detection limits for the contaminants of concern in sediments and in fish tissue (see Appendix E). Detection limits are calculated directly from the SSVs (sediment detection limits), or fish ingestion assumptions and toxicity criteria (fish tissue detection limits). Detection limits commonly 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. If recommended detection limits are not achieved, an uncertainty analysis may be appropriate to evaluate the potential impact on site characterization.

5.2 Partitioning of chemicals

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

Chemicals in the environment move between phases according to thermodynamic principles. Whereas thermodynamics describe equilibrium states, the movement of chemicals from one phase to another in the environment can often be better described by looking at the kinetics of the transfers. As contaminants move from the sediments into water and then into air, they become diluted and dispersed. 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 concentrations in various

media can be complicated and often depends on conditions that cannot easily be generalized (i.e., dilution from water mixing, or dilution in air from wind).

Equilibrium partitioning can be used to approximate the concentration of individual chemicals in static systems. As systems become dynamic, chemicals are moved away from a boundary layer and disperse. Chemical concentrations in water are diluted away from the sediment/water boundary. A default 10-fold dilution from equilibrium water concentration was incorporated into the SSM to approximate dilution at the location where exposures occur. Similarly, an additional 50-fold dilution of air was assumed for vapor-phase exposures in the SSM. This results in a 10-fold decrease in surface water ingestion and dermal-water exposures and a 500-fold decrease in inhalation exposures when compared to the 2005 sediment screening model. Results from the current SSM should present a more accurate model of exposures, as well as a better indication of the relative importance of different routes of exposure.

The values in this PHC are suitable for use as screening values in the initial phases of site investigation. The SSM is intended to help identify the types of exposure that may cause the greatest concern for chemicals in sediments. Values developed from calculated water or air concentrations may be very conservative. Comparing measured sediment concentrations to the route-specific SSM results provides important information about need for further analyses, exposures that may be of little importance, and the potential impact of a remedial action. If the contribution by routes requiring exposure to water or air is minimal, the impact of a site-specific dilution factor on the SSM results will be inconsequential for the specific chemical being evaluated. Alternatively, if the model suggests that exposure to dissolved chemical or vapor may be important, water concentration should be measured for more accurate assessment of potential chemical exposure.

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

$$C_{SW}\ \{\ mg/L\ \} = C_{Sed} / (K_{oc} * f_{oc} * DF_{sed\text{-}sw}) \qquad \textit{Equation 2.}$$

Where:

C_{SW}	= concentration in surface water	{mg/L}
C_{Sed}	= concentration in sediment	{mg/kg dry wt.}
K_{oc}	= organic carbon partitioning constant	{L/kg; chemical specific}
f_{oc}	= fraction organic carbon in sediment	{unitless; site specific}
$DF_{sed\text{-}sw}$	= dilution factor for chemical in water	{unitless; 10(default)}

Equation 2 describes the equilibrium achieved over time when sediment and water are mixed (Lyman, 1995); a static system. In addition, a surface water dilution factor has been added to represent mixing in an open system. Note that the fraction organic carbon (f_{oc}) is inversely proportional to the concentration of the chemical in surfacewater (C_{SW}). As a result, if site-specific data suggest that f_{oc} is 1% instead of the 2% default value, for example, the anticipated

concentration of non-polar organic compounds in surface water will be twice as high as the concentration used in the default SSM.

In addition, there is considerable information showing that non-polar organic compounds have different affinities for organic carbon of varying origins. For example, PAH partitioning into water has been shown to be lower 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 less 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 differ 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.

Partitioning between water and air, with incorporation of a dilution factor (described above), are calculated using the following equation (adapted from Schwarzenbach et al., 1993):

$$C_A \{ mg/m^3 \} = C_{SW} * K_H / (R * T * DF_{sw\text{-}air}) * (CF_{L/m3} * CF_{L/m3}) \qquad \textit{Equation 3.}$$

Where:

C_A	= concentration in air	{mg/m^3}
C_{SW}	= concentration in surface water	{mg/L}
K_H	= Henry's Law Constant	{$atm \cdot m^3/mol$; chemical specific}
R	= ideal gas constant	{$L/(mol \cdot ºK)$}
T	= temperature	{ºK; 293.13ºK (default)}
$DF_{sw\text{-}air}$	= dilution factor for chemical in air	{unitless; 50 (default)}
$CF_{L/m3}$	= conversion factor	{1,000 L/m^3}

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

5.3 Contaminant accumulation in fish tissue

Aquatic organisms exposed to low concentrations of non-polar organic compounds in water absorb some of these compounds. This is defined as bioconcentration. A bioconcentration factor describes the non-active partitioning between water and aquatic organisms.

Bioconcentration can be calculated from equilibrium partitioning between water and fish tissue. 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). Furthermore, fish are constantly metabolizing and excreting chemicals. Therefore, significant accumulation in aquatic organisms typically requires that a chemical have a relatively long half-life in an organism – i.e., a slow clearance time. Bioconcentration Factors (BCFs) were not used to calculate fish tissue concentrations in the SSM.

Accumulation of compounds in aquatic organisms by any and all routes-of-exposure, including ingestion, is called bioaccumulation which can be described by a bioaccumulation factor (BAF). Bioaccumulation is an empirical measure of uptake, metabolism and excretion. It cannot easily be, and is not typically, modeled. 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. Chemical-specific biota-sediment accumulation factors (BSAFs) are often used to describe the accumulation of persistent organic chemicals in fish.

BSAFs are calculated from field data; from fish tissue concentrations and sediment concentrations within their foraging range. While a BSAF implies that sediment and fish tissue concentrations for a specific chemical are directly and linearly related, there is a limit to the amount of contaminant that can accumulate in the fish tissue. This occurs either because fish (or their prey) avoid the most contaminated areas or 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. As a result, route-specific SSM results 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 SSM are central tendency values of the range of published values. BSAFs are listed in SSM results shown in Table D-1a, Appendix D. When BSAFs were not available, SSVs did not include the fish consumption pathway.

If fish consumption is not believed to be a significant route of exposure for a contaminant of concern, it should be eliminated for that chemical. Further discussion and an illustrative example are provided in Section 7.2.

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 fraction organic carbon (f_{oc}) is in Appendix F. Some 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 water body, but in the literature they typically are not normalized for fish size. 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 exceeds metabolism and excretion, a contaminant can accumulate in the food chain, so that higher trophic-level organisms have higher concentrations of the contaminant than those at lower levels. Older (and generally larger) members of a species will also have higher concentrations than younger members. 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 problematic. Fish appear to metabolize PAHs once metabolizing enzymes have been induced (see Varanasi et al., 1989 for review). It is expected that different fish species 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:

> "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."

Metabolism of PAHs in fish results in (brief) exposure 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. Research over the past years has demonstrated that PAH toxicity can be heritably reduced in wild fish populations that inhabit some contaminated areas on the east coast (for example Meyer et al., 2002; Ownby et al., 2002). Some of this decreased sensitivity is presumably due to decreased activity of metabolic enzymes, which could result in greater accumulation of PAHs in these fish.

Furthermore, when PAHs in fish are measured and their toxicities are evaluated, heterocyclic PAHs, substituted PAHs and metabolic products are typically ignored. Therefore, predicting the concentration and toxicity of PAH mixtures 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.

The US EPA has compiled a database of BSAFs from Superfund sites around the country (US EPA, 2008). The database contains PAH BSAF data on largemouth bass and bluegills from 2 Superfund sites. Both of these fish species are found in Minnesota and the St. Louis River. MDH evaluated these data to determine PAH BSAFs used as defaults in this PHC. Individual

PAHs were classified according to the number of 6-carbon rings in their structure. Because PAHs with more rings are typically more lipophilic and are less easily metabolized, BSAFs were calculated for 2,3,4,5 and 6-carbon ring PAHs and used as surrogates for similar PAHs. Three to six-ring data (n=73) were used to estimate a composite cPAH BSAF that was used for calculating the BaP-PEQ SSV. Further discussion of the PAH BSAFs is included in Appendix G.

5.3.2 Mercury bioaccumulation

The primary human exposure pathway for mercury in sediments is fish consumption. Fish accumulate methylmercury 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 (or methylmercury) found in fish across different water bodies (Sorensen et al., 1990; Wiener et al., 1990; Cabana et al., 1994; Driscoll et al., 1995). This is usually attributed to variable methylmercury production and differences between food chains in different water bodies.

Chemical conditions and the local foodweb 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 foodweb often correlates with conditions that increase the methylation of inorganic mercury. For example, 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 a waterbody.

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 methylmercury concentrations across different water bodies (among species and fish lengths), the mercury concentration in sediments and the methylmercury concentration in fish of similar species and length or age are often correlated within a single water body.

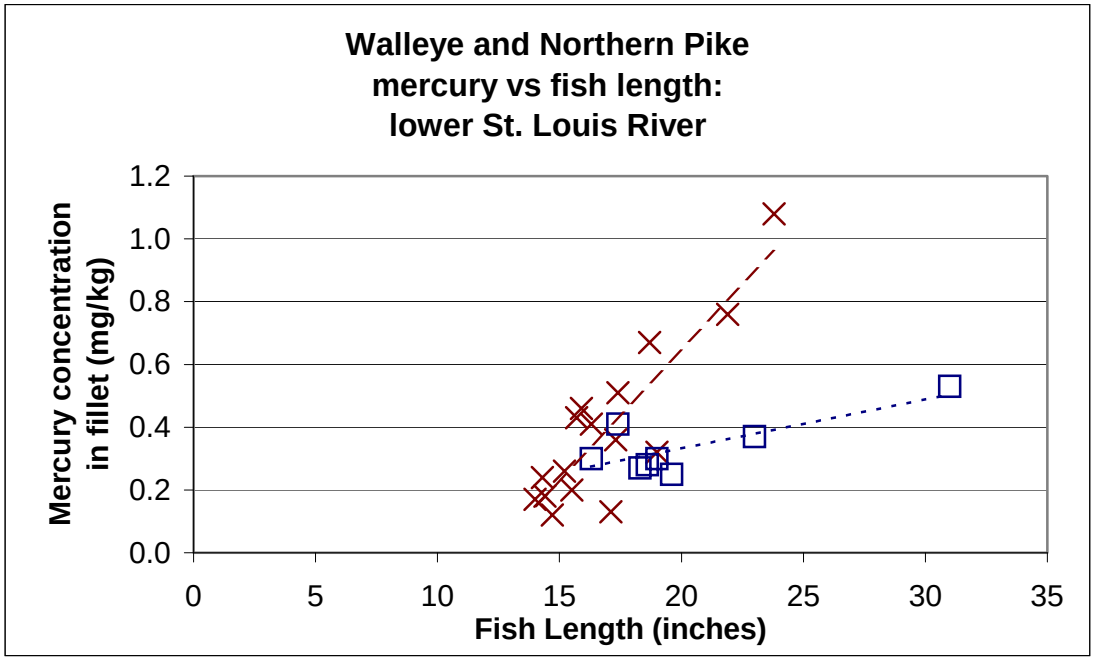
A locally-derived BSAF is used in this PHC to estimate the methylmercury concentration in an index fish. The EPA Mercury Study Report to Congress contains a review of BSAF derivations for a number of different water bodies and aquatic species (see Volume VI, Section 2.3.1 of US EPA, 1997b).

5.3.2.1 Mercury in fish

Individual walleyes and northern pike are believed to range throughout the lower St. Louis River over time. As a result of feeding on smaller fish throughout the lower St. Louis River, the total methylmercury intake for large sportfish is likely determined by a range of different mercury concentrations found 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 4.*

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 5.*

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).

5.3.2.2 Mercury in sediment

In 1995, EPA collected sediment data from the lower St. Louis River for the Regional Environmental Monitoring and Assessment Program (REMAP; US EPA, 1995). Sample locations were chosen randomly, after eliminating known areas of contamination (see Attachment 1 for sample locations). Samples were analyzed for mercury, PAHs, organic carbon and a number of other chemicals. Some sampling locations were training sites and data from these locations were not used in the mercury BSAF calculation. Mercury data from 87 sample locations in Class I & II habitat (shallow water and channel sediments, respectively) were used. Statistics from the sediment mercury concentrations are shown in Table 11.

5.3.2.3 Mercury BSAF calculation

BSAFs are calculated as the ratio of methylmercury in fish tissue to a representative sediment mercury concentration. Since fish accumulate mercury primarily from their diet, fish tissue mercury concentrations are expected to 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 uncontaminated 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. A reasonable central tendency for calculating a BSAF for the St. Louis River estuary is the geometric mean of REMAP mercury sediment concentration data (0.078 mg/kg mercury). This statistic discounts the highest mercury concentrations in sediments and may best reflect sediment mercury incorporation into the food chain.

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

Table 11 - Calculated BSAFs (mercury)

REMAP Sediment Mercury Data Statistic (10% - 90% CL) mg_{Hg}/kg_{sed_dry}		**Mercury concentration in St. Louis River (Predicted from regression: *Equations 5 and 6*)** Mean (10% - 90% CL) mg_{Hg}/kg_{tissue_wet}		**Calculated Biota-Sediment Accumulation Factor (BSAF)** kg_{sed_dry} / kg_{tissue_wet}	
		Walleye - 20"	Northern Pike - 30"	**Walleye - 20"**	**Northern Pike - 30"**
Mean	0.150 (0.121 - 0.179)	0.65 (0.53-0.76)	0.49 (0.38 - 0.60)	4.3	3.3
Median	0.100			6.5	4.9
Geometric Mean	0.078 (0.063 - 0.098)			**8.2**	**6.2**

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 PHC. MDH is not aware of published BSAFs for walleye. However, there are published BSAFs for northern pike summarized in the EPA Mercury Study Report to Congress (US EPA, 1997b). Published northern pike BSAFs range from 10.1 - 45.7 (unitless; dry fish tissue weight). The proposed St. Louis River northern pike BSAF is 6.2, however, it is based on fish tissue wet weight mercury concentration. Assuming the dry weight BSAF for northern pike is about 4 times the wet weight BSAF (US EPA, 1997b), the dry weight BSAF for 30-inch northern pike (fillets) in the St. Louis River is about 25 (unitless). This is in the middle of the range of northern pike BSAFs reported.

6 Screening Value 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 the SSM (See Appendix D; Table D2). EPA Integrated Risk Information System (IRIS) (US EPA, 2012a) reference doses (RfDs), reference concentrations (RfCs), cancer slope factors (CSFs) and unit risk (UR) values are used to evaluate most chemicals of concern in sediments. In addition, ATSDR Minimum Risk Levels (MRLs; ATSDR, 2012 - http://www.atsdr.cdc.gov/mrls/index.asp) were used in the SSM for a number of chemicals with non-cancer endpoints. Additional RfCs, RfDs, CSFs and UFs from MDH, the California Office of Environmental Health Hazard Assessment, EPA Region 9 2004 Provisional Risk Guidance, EPA 1996 Soil Screening Levels, and New York State Health Department are used to evaluate the other chemicals of interest identified in this PHC. Citations accompany the data in the Appendices. These data sources are generally used by the MPCA in regulatory programs for site remediation.

The SSM results are protective of sensitive sub-populations. However, the results 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.

Of note, the non-cancer toxicity criteria used in the SSM were higher than published MRLs in the following cases:

- MDH uses the EPA elemental mercury inhalation RfC instead of the MRL because the RfC evaluation used a more realistic inhalation metric for human exposure studies.
- MDH has developed ingestion and inhalation values for toluene that are newer than the 2000 MRLs.
- The copper MRL was not used because it is below the Recommended Daily Allowance (RDA) for copper (IOM, 2001).
- MDH has a newer inhalation health-based value for naphthalene than the MRL.

Note that copper and naphthalene were evaluated in the SSM. However, an SSV was not finalized for these chemicals because ecological criteria are likely to drive any sediment contamination decisions.

As noted in Section 4.1.3, the lead SSV is not a SSM result, but has been set equivalent to the MPCA lead Soil Reference Value.

6.2 Relative Source Contribution, Hazard Index, and Cancer Risk

The default relative source contribution (RSC) for non-carcinogens in the SSM is 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 RfD or other health criteria). The RSC for exposure to methylmercury as a result of fish consumption is 73% as recommended in the EPA Water Quality Criterion document on methylmercury (US EPA, 2001c).

Note that a hazard index for simultaneous exposure to chemicals with similar endpoints is not calculated in the SSVs, but should be applied when evaluating site-specific data.

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. The SSVs are to be used to identify individual chemicals (or groups of chemicals: i.e., BaP-PEQs, TCDD-TEQs, and tPCBs) needing further consideration, and likely overestimate actual risk.

No acute screening values are developed in this PHC. 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 (US EPA, 1989)." Therefore, the availabilities of all chemicals from ingested water and 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.

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 PHC 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 (carbazole, hexachlorobenzene, octachlorostyrene, pentachlorophenol, tPCBs and quinoline), 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 Model Results

The SSM consists of equations described in Appendix H (route-specific chronic non-cancer endpoints), Appendix I (route-specific cancer endpoint) and Appendix J (all routes screening calculations). The SSM results are shown in Table 12, which includes the relative contributions of different exposure routes.

Table 12 - Sediment Screening Model Results

Chemicals Evaluated with SSM	CAS No.	Endpoint (Non-cancer / Cancer)	Multi-route Sediment Screening Model Result	Contributions From Different Exposure Routes (model attribution)					
				Sediment Ingestion	Surfacewater Ingestion	Dermal from Sediment	Dermal from Surfacewater	Inhalation	Fish Consumption
Metals - Inorganics			mg/kg	% Sed(Ing)	% SW(Ing)	% Sed(Derm)	% SW(Derm)	% Inh	% Fish
Arsenic	7440-38-2	Non-cancer	50	**64%**	*Not Evaluated*	36%	*Not Evaluated*	*Not Evaluated*	*Not Evaluated*
		Cancer	**30**	**51%**		49%			
Cadmium	7440-43-9	Non-cancer	**10**	**57%**		43%			
Chromium III	16065-83-1	Non-cancer	**400000**	**100%**		*Not Evaluated*			
Chromium VI	18540-29-9	Non-cancer	300	**100%**					
		Cancer	**40**	**100%**					
Copper	7440-50-8	Non-cancer	**10000**	**100%**					
Cyanide	57-12-5	Non-cancer	**2000**	**100%**					
Lead	7439-92-1	Non-cancer	**300** *	*Not Evaluated*					
Mercury (SSM - tHg; exp - tHg)	7439-97-6	Non-cancer	0.02 †	**0.00023%**					
Mercury (SSM - tHg; exp - MeHg)									**100%**
Nickel	various	Non-cancer	**5000**	**100%**					*Not Evaluated*
Zinc	7440-66-6	Non-cancer	**80000**	**100%**					
Volatile organic compounds (VOCs)									
Benzene	71-43-2	Non-cancer	1	0.13%	12%	1.3%	10%	**76%**	*Not Evaluated*
		Cancer	**0.8**	0.21%	20%	2.4%	19%	**59%**	
Ethyl benzene	100-41-4	Non-cancer	**70**	0.28%	8.4%	2.6%	28%	**61%**	
Styrene	100-42-5	Non-cancer	**300**	0.62%	19%	5.9%	**46%**	29%	
Toluene	108-88-3	Non-cancer	**50**	0.26%	15%	2.4%	28%	**55%**	
Xylenes (mixed)	1330-20-7	Non-cancer	**40**	0.085%	3%	0.8%	11%	**86%**	
Polycyclic Aromatic Hydrocarbons (PAHs)		Non-cancer							
Acenaphthene	83-32-9	Non-cancer	**50**	0.33%	0.88%	0.8%	6.7%	0.13%	**91%**
Acenaphthylene (toxicity surrogate - pyrene)	208-96-8	Non-cancer	**30**	0.33%	0.87%	0.8%	7%	0.075%	**91%**
Anthracene	120-12-7	Non-cancer	**800**	0.97%	0.8%	2.4%	12%	0.034%	**84%**
Benzo(a)pyrene equivalents (BaP-PEQs)	50-32-8	Cancer	**0.2** †	1.5%	0.034%	4.3%	4.7%	0.000038%	**90%**
Fluoranthene	206-44-0	Non-cancer	**100**	1%	0.25%	2.5%	6.8%	0.0018%	**89%**
Fluorene	86-73-7	Non-cancer	**30**	0.33%	0.49%	0.82%	5%	0.037%	**93%**
Methylnaphthalene (toxicity surrogate - naphthalene)	1321-94-4	Non-cancer	**10**	0.29%	1.6%	0.72%	12%	3.9%	**81%**
Naphthalene	91-20-3	Non-cancer	**10**	0.29%	2.5%	0.72%	9.2%	5.5%	**81%**
Perylene (toxicity surrogate - pyrene)	198-55-0	Non-cancer	**200**	2.8%	0.63%	6.9%	**89%**	0.0017%	0.97%
Phenanthrene (toxicity surrogate - pyrene)	85-01-8	Non-cancer	**80**	0.97%	0.79%	2.4%	12%	0.025%	**84%**
Pyrene	129-00-0	Non-cancer	**500**	6.1%	1.5%	15%	37%	0.014%	**41%**
Dibenzo-p-dioxins/dibenzofurans									
2,3,7,8-TCDD equivalents (TCDD-TEQs)	1746-01-6	Non-cancer	4E-07	0.23%	0.012%	0.13%	1.3%	0.00003%	**98%**
		Cancer	**2E-08**	0.14%	0.0076%	0.094%	0.94%	0.000039%	**99%**
Other Organics									
Carbazole	86-74-8	Cancer	**80**	7.4%	11%	17%	**65%**	0.001%	*Not Evaluated*
Hexachlorobenzene	118-74-1	Non-cancer	0.1	0.93%	2%	1.8%	**50%**	5.2%	40%
		Cancer	0.1	0.71%	1.5%	1.6%	44%	2.1%	**50%**
Octachlorostyrene	29082-74-4	Non-cancer	**0.02**	0.19%	0.047%	0.36%	18%	0.016%	**82%**
Pentachlorophenol	87-86-5	Non-cancer	6	0.48%	1.3%	2.3%	**96%**	0.000049%	*Not Evaluated*
		Cancer	**0.7**	0.31%	0.86%	2.5%	**96%**	0.000025%	
Polychlorinated Biphenyls (PCBs)	1336-36-3	Non-cancer	**0.005** †	0.086%	0.015%	0.23%	2.9%	0.0093%	**97%**
		Cancer	0.006 †	0.053%	0.0091%	0.17%	2.1%	0.00072%	**98%**
Quinoline	91-22-5	Cancer	**0.4**	5.8%	**51%**	13%	30%	0.067%	*Not Evaluated*

* MPCA Soil Reference Value - See Section 4.1.3

Shaded Routes of Exposure – Boundary layer exposure dilution factor(s) applied. See Section 5.2

† (shaded chemicals) Sediment Screening Model result may approach or be less than ambient or background concentration. See sections on individual chemicals (in Section 4, above), Section 7.1.2 (below) and Appendix C for information on ambient and background concentrations.

7.1 Discussion of SSM results and SSVs

Results from the SSM should not be applied directly as SSVs without being evaluated for their relevance and usefulness. If the model results for a specific chemical suggest human health concern only at a relatively high sediment concentration it is possible that the chemical may be toxic to aquatic organisms at a lower concentration. Alternatively, if the results suggest an extremely low sediment concentration (e.g, TCDD-TEQ) could be of concern to people, model results should be compared with background concentrations in sediments. Further discussion of these issues is in the sections below.

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

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 even considered. There are very little data available with which to quantitatively evaluate potential exposures to metals by other routes of exposure.

Note also that the largest exposure for many 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 or air concentration near the water surface).

7.1.1 Comparison of SSM results with SQTs

The relevance of a human health based SSV is questionable when the model calculates a concentration of concern that is above (less restrictive than) levels that will severely impact aquatic organisms. The MPCA has developed Sediment Quality Targets (SQTs) for protection of sediment-dwelling organisms (MPCA, 2007). SQTs are derived from empirical data and were not normalized to organic carbon. Significant differences between SSM results and SQTs are expected, primarily because the SQTs are derived from studies using aquatic organisms exposed to contamination from sediments throughout their life. In contrast, human exposure to chemicals in sediments occurs infrequently. Level I SQTs are set to provide a high level of protection for benthic invertebrates (i.e., set at a concentration, below which harmful effects on benthic organisms are unlikely). Level II SQTs are expected to provide a moderate level of protection for benthic invertebrates(i.e., set at a concentration, above which harmful effects on benthic organisms are likely).

Table 13 compares available MPCA SQTs with SSM results. Note that SQTs are less restrictive than the SSM results for most bioaccumulative chemicals (methyl mercury, dioxin and PCBs). The SSM results also appear to be more restrictive for carcinogens. On the other hand, the SSM results for many chemicals are extremely high when compared with the SQTs. These chemicals include copper, nickel, zinc and the

individual nPAHs. For some of these chemicals, at sediment concentrations similar to the SSM results, it is quite likely that benthic organisms will not be able to survive. Because SSVs for these chemicals at concentrations suggested by the SSM are not likely to be useful, MDH has chosen to not develop SSVs for chemicals when the SSM result is 10 times or greater than the MPCA Level II SQT. Information from the SSM such as the contribution of different routes of exposure to the result may be useful, but the quantitative SSM results for chemicals identified by gray shading in Table 13 can otherwise be ignored for the reasons described above.

Table 13 - Comparison of SSM Results with MPCA SQTs

Chemical	MDH SSM Results	MPCA SQT_{2007}	
		Level 1	Level 2
	mg/kg	mg/kg	mg/kg
Arsenic	32	9.8	33
Cadmium	15	0.99	5
Chromium III	390000		
Chromium VI	44		
Chromium		43	110
Copper	10000	32	150
Cyanide	1600		
Lead	300	36	130
Mercury	0.02	0.18	1.1
Nickel	5200	23	49
Zinc	79000	120	460
Benzene	0.85		
Ethyl benzene	76		
Styrene	340		
Toluene	55		
Xylenes (mixed)	45		
Acenaphthene	51	0.0067	0.089
Acenaphthylene	25	0.0059	0.13
Anthracene	760	0.057	0.85
BaP-PEQs	0.19		
Benzo[a]pyrene		0.15	1.5
Fluoranthene	110	0.42	2.2
Fluorene	35	0.077	0.54
Methylnaphthalene	12		
2-Methylnaphthalene		0.02	0.2
Naphthalene	12	0.18	0.56
Perylene	220		
Phenanthrene	77	0.2	1.2
Pyrene	480	0.2	1.5
Total PAHs		1.6	23
2,3,7,8-TCDD-TEQs	2.2E-08 §	8.50E-07 ¥	2.15E-05 ¥
Carbazole	79		
Hexachlorobenzene	0.096		
Octachlorostyrene	0.015		
Pentachlorophenol	0.72		
Polychlorinated Biphenyls (PCBs)	0.0045	0.06	0.68
Quinoline	0.42		

Bolded Chemicals - - SQTs likely to be more restrictive than SSVs

SSM Model results - - not finalized SSVs due to likelihood of effects on aquatic organisms (SSM result>10 times Level 2 SQT)

§ - use Van den Berg et al. (2006) human TEFs

¥ - use Van den Berg et al. (1998) fish TEFs

Likely to approach local ambient or background levels

Likely below national background and national ambient levels

7.1.2 SSV's below ambient or background concentrations

The SSM results for mercury, TCDD-TEQs, tPCBs, and carcinogenic PAHs including benzo[a]pyrene may be less than, or approaching background and/or ambient sediment concentrations (see Table 13). The SSM results for these chemicals are recommended as SSVs by MDH. Background sediment concentrations are calculated from samples 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. Background concentrations may be used as screening levels when they exceed the SSVs for individual chemicals. Unfortunately, background levels for many contaminants in the St. Louis River estuary are not known.

7.1.2.1 Mercury

Background mercury concentrations in the upper St. Louis (about 0.02 mg/kg; Glass et al., 1999) are equal to the mercury SSV. Table 14 shows the range, geometric mean, arithmetic mean and median REMAP mercury concentrations for the lower St. Louis River. Note that 84% of the REMAP sample concentrations exceeded the mercury SSV. REMAP data are also used in an analysis of background mercury concentrations in Appendix C. The analysis suggests that background mercury concentrations in the St. Louis River estuary are about 0.02 mg/kg, but that ambient concentrations in some areas of the estuary may be above background due to local area or point sources. Therefore, it is reasonable to use the SSV (0.02 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. Note from Table 14 that 32% of the REMAP mercury sample concentrations also exceed the Level I SQT.

7.1.2.2 PAHs (BaP-PEQs)

Table 14 compares SSM results and SQTs for PAHs to available REMAP data from the St. Louis River Estuary. Concentrations of individual PAHs at many REMAP locations in the St. Louis River Estuary exceeded PAH SQTs (21% - 54% of the samples for individual PAHs). However, when REMAP PAH data are compared with the SSM results, only BaP-PEQs exceeded the model results. Note that REMAP cPAH analysis was limited to only 7 cPAHs and therefore, the BaP-PEQ is likely underestimated in all samples.

BaP-PEQs exceeded model results at about 40% of the sample locations, whereas individual nPAH sediment concentrations were well below SSM results. These data suggest that chronic human exposure to nPAHs does not appear to impact a sensitive human endpoint, and provides additional support for not developing nPAH SSVs (Section 7.1). Table 14 shows that the SQTs are more restrictive than the SSM results for nPAHs.

REMAP data were intended to be representative of ambient data throughout the estuary. It is likely that samples were impacted by discharges from industrial and urban sources, and that historic deposition from air pollution impacted most samples. Background BaP-PEQs have not been established for northern Minnesota or the St. Louis River estuary.

As shown in Table 14, ambient BaP-PEQ concentrations in some areas of the St. Louis River estuary are greater than the BaP-PEQ SSV. While REMAP data seems to suggest that background BaP-PEQs for northern Minnesota and the lower St. Louis River are below than the BaP-PEQ SSV, additional data may be needed.

Table 14 - SSVs and REMAP Data for PAHs and Mercury

Chemical	REMAP (data below Fond du Lac Dam)				Sediment Criteria (mg/kg)		% REMAP Above SSM results	*% REMAP Above SQT - Level 1*
	Range (mg/kg)	Geometric mean (mg/kg)	Arithmetic mean (mg/kg)	Median (mg/kg)	Sediment Screening Model results	*Sediment Quality Targets - Level 1*		
Acenaphthene	**0.000050 - 0.65**	0.0055	0.036	0.0051	**50**	***0.0067***	**0%**	***44%***
Acenaphthylene	**0.00050 - 0.47**	0.0086	0.041	0.0075	**30**	***0.0059***	**0%**	***54%***
Anthracene	**0.0014 - 2.6**	0.043	0.25	0.028	**800**	***0.057***	**0%**	***42%***
BaP-PEQs (7 cPAHs only)	**0.0059 - 6.5**	0.17	0.83	0.15	**0.2**		**41% ***	
Benzo(a)pyrene	**0.0040 - 4.7**	0.11	0.58	0.093		***0.15***		***41% *****
Fluoranthene	**0.0062 - 7.5**	0.21	0.99	0.14	**100**	***0.42***	**0%**	***36%***
Fluorene	**0.00050 - 1.5**	0.021	0.12	0.018	**30**	***0.077***	**0%**	***28%***
Methylnaphthalene					**10**	***0.02***		
Naphthalene	**0.00050 - 10**	0.033	0.51	0.036	**10**	***0.18***	**0%**	***21%***
Perylene	**0.0035 - 1.6**	0.18	0.39	0.27	**200**		**0%**	
Phenanthrene	**0.0037 - 6.1**	0.13	0.63	0.11	**80**	***0.2***	**0%**	***43%***
Pyrene	**0.0068 - 5.4**	0.18	0.82	0.13	**500**	***0.2***	**0%**	***46%***
Mercury	**0.0049 - 0.70**	0.078	0.15	0.100	**0.02**	***0.18***	**84%**	***32%***

* BaP-PEQ SSV compared with BaP-PEQ for 7cPAHs in REMAP data

** Benzo(a)pyrene SQT compared with benzo(a)pyrene REMAP data

Shaded chemicals: SSVs set equal to SSM results – no SSVs for other chemicals in Table

7.1.2.3 TCDD-TEQ

The TCDD-TEQ SSM result (TCDD-TEQ SSV) of 0.02 ng/kg is much lower than national background concentrations (background concentrations from 11 lakes study; Cleverly, David et al., 1996). Therefore, TCDD-TEQ background concentrations may be used to screen sediments for contamination. Newer data from the lower St. Louis River have not been analyzed yet to determine a local background concentration for TCDD-TEQs. However in the absence of local 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).

7.1.2.4 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, comparison to 1997 National Sediment Quality Survey data shows that the tPCB SSV exceeds sediment concentrations at about 20% of the nationwide monitoring locations (US EPA, 1997c). Therefore background sampling in the region is needed to determine screening levels for PCBs in the St. Louis River.

7.2 Sediment Screening Values

Table 15 shows the final 2013 MDH SSVs for the US Steel Site. In addition, route-specific SSVs are listed. These route-specific values can be used if only a single route of exposure is anticipated or to calculate site-specific screening values if a limited number of routes of exposure are possible.

Table 15 - 2013 Sediment Screening Values – US Steel Site

Final SSVs		CAS No.	Endpoint	Sediment Screening Value	Sediment Ingestion	Surfacewater Ingestion	Dermal Sediment	Dermal Surfacewater	Inhalation	Fish Consumption
				SSV	$SSV_{Sed(Ing)}$	$SSV_{SW(Ing)}$	$SSV_{Sed(Derm)}$	$SSV_{SW(Derm)}$	SSV_{Inh}	SSV_{Fish}
Metals - Inorganics				**mg/kg**	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg
	Arsenic	7440-38-2	Non-cancer	**50**	**78.7**		138			
			Cancer	**30**	**61.6**		64.6			
	Cadmium	7440-43-9	Non-cancer	**10**	**26.2**		34.5			
	Chromium VI	18540-29-9	Non-cancer	**300**	**262**					
			Cancer	**40**	**43.6**					
	Cyanide	57-12-5	Non-cancer	**2000**	**1570**					
	Lead	7439-92-1	Non-cancer	**300 ***						
	Mercury (SSV - tHg; exp - tHg)	7439-97-6	Non-cancer	**0.02 †**	**79**					
	Mercury (SSV - tHg; exp - MeHg)		Non-cancer							**0.0202**
Volatile organic compounds (VOCs)										
	Benzene	71-43-2	Non-cancer	**1**	1050	11.3	111	13.6	**1.84**	
			Cancer	**0.8**	396	4.28	35.4	4.46	**1.42**	
	Ethyl benzene	100-41-4	Non-cancer	**70**	26200	866	2760	265	**119**	
	Styrene	100-42-5	Non-cancer	**300**	52500	1730	5530	**712**	1140	
	Toluene	108-88-3	Non-cancer	**50**	21000	363	2210	192	**98.8**	
	Xylenes (mixed)	1330-20-7	Non-cancer	**40**	52500	1490	5530	421	**51.8**	
Polycyclic Aromatic Hydrocarbons (PAHs)			Non-cancer							
	Benzo(a)pyrene equivalents (BaP-PEQs)	50-32-8	Cancer	**0.2 †**	12.8	557	4.41	4.06	494000	**0.212**
Dibenzo-p-dioxins/dibenzofurans										
	2,3,7,8-TCDD equivalents (TCDD-TEQs)	1746-01-6	Non-cancer	**4E-07 †**	0.0165	0.304	0.00131	1.8E-04	3.91	**4.2E-07**
			Cancer	**2E-08 †**	1.6E-05	2.9E-04	2.3E-05	2.3E-06	0.0554	**2.2E-08**
Other Organics										
	Carbazole	86-74-8	Cancer	**80**	1090	739	487	**124**	8080000	
	Hexachlorobenzene	118-74-1	Non-cancer	**0.1**	33.4	15.3	9.52	0.335	2.34	**0.302**
			Cancer	**0.1**	13.6	6.25	6.09	0.22	4.66	**0.192**
	Octachlorostyrene	29082-74-4	Non-cancer	**0.02**	707	2880	16.8	0.484	258	**0.0185**
	Pentachlorophenol	87-86-5	Non-cancer	**6**	1310	481	276	**6.61**	13000000	
			Cancer	**0.7**	231	84.8	29.1	**0.753**	2850000	
	Polychlorinated Biphenyls (PCBs)	1336-36-3	Non-cancer ‡	**0.005 †**	5.25	30.3	1.97	0.154	48.3	**0.00465**
			Cancer	**0.006 †**	10.9	63	3.48	0.279	795	**0.0059**
	Quinoline	91-22-5	Cancer	**0.4**	7.27	**0.83**	3.25	1.43	631	

* MPCA Soil Reference Value - 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 (Section 4), Section 7.1.2 and Appendix C for information on ambient and background concentrations.

Most important route of exposure for each chemical (SSM-determined) is bolded

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 chemical, screening values can be recalculated with route-specific values in Table 15, using *Equations A-46 and A-47* (chronic and cancer endpoints, respectively) from Appendix J.

For example, PAHs are metabolized by vertebrates including fish. As a result, it has been assumed that bioaccumulation of PAHs in fish tissue is minimal and fish consumption is a minor route of exposure to PAHs. Data on PAH concentrations in fish fillets are quite sparse and often PAH detection limits in fish studies have been higher than needed to determine risk. However, PAH BSAFs used in the SSM are based on BSAFs calculated from fish tissue and sediment data from Superfund sites (US EPA, 2008) and provide empirical evidence of PAH accumulation in fish. The fish consumption pathway has been included in calculating a SSV for BaP-PEQs. Note from Table 12 that the contribution of the fish consumption pathway to the overall SSM result is quite high for most PAHs. However, if data from future studies demonstrate that the fish consumption

route of exposure should not be evaluated, the SSV for the specific PAH should be recalculated without the fish consumption pathway.

If the fish consumption route of exposure is eliminated (by removing $1/SSV_{fish}$ from *Appendix J -Equation A-46* and $1/SSV_{fish\text{-}c}$ from *Appendix J - Equation A-47*) for PAHs, the resulting site-specific screening model results for PAHs are shown in Table 16. Similarly, the inhalation exposure pathway or any other potential route of exposure can be eliminated from the SSVs for any individual chemical at a site.

Table 16 - PAH SSM Results - Without Fish Consumption

	Cas No.	Partial Sediment Screening Model result	Route-of-exposure contribution				
		(No Fish Consumption)	Sediment Ingestion	Surfacewater Ingestion	Dermal Sediment	Dermal Surfacewater	Inhalation
Polycyclic Aromatic Hydrocarbons (PAHs)		**mg/kg**	**%**	**%**	**%**	**%**	**%**
Acenaphthene	83-32-9	**580**	4%	10%	9%	75%	1%
Acenaphthylene (toxicity surrogate - acenaphthene)	208-96-8	**280**	4%	10%	9%	76%	1%
Anthracene	120-12-7	**4800**	6%	5%	15%	74%	0%
Benzo(a)pyrene (or BaP-PEQ) *Cancer endpoint*	50-32-8	**1.8**	14%	0%	41%	44%	0%
Fluoranthene	206-44-0	**1000**	10%	2%	23%	63%	0%
Fluorene	86-73-7	**520**	5%	7%	12%	74%	1%
Methylnaphthalene (toxicity surrogate - naphthalene)	1321-94-4	**65**	2%	8%	4%	65%	21%
Naphthalene	91-20-3	**66**	2%	14%	4%	51%	30%
Perylene (toxicity surrogate - pyrene)	198-55-0	**220**	3%	1%	7%	88%	0%
Phenanthrene (toxicity surrogate - anthracene)	85-01-8	**490**	6%	5%	15%	74%	0%
Pyrene	129-00-0	**810**	10%	3%	25%	62%	0%

8 Children and Other Special Populations

Exposure parameters developed for this PHC 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 non-cancer 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 people that may have higher-than-projected exposures to sediments. These include children who ingest contaminated sediments (pica behavior), and subsistence fishers who eat fish that have accumulated contaminants. Protective criteria for such people can be developed for application at specific sites.

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). This has led to incorporation of a default age-dependent adjustment factor (ADAF) into cancer risk evaluations including the SSVs (US EPA, 2005; MDH, 2010).

9 Conclusions

9.1 General application of screening values

Only exposures to water-covered sediments were evaluated in this PHC. 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 SSVs developed in this PHC 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. Furthermore, while this PHC evaluates a suite of persistent chemicals, it does not evaluate all chemicals that can be found in sediments and impact public health.

The values developed in this PHC are appropriate for screening sediments throughout the lower St. Louis River. While exposures will vary from site to site, this PHC 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 for many chemicals that should be protective of the health of individuals who regularly use the St. Louis River for recreational activities.

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. For other site-specific evaluations, potential exposures and sediment characteristics for a specific site should be compared with default values used to develop these SSVs. Parameters that may affect the transport and availability of contaminants at specific sites may include: organic carbon, temperature, particle size, redox potential, mineral content, clay content and porosity.

9.2 Application to the US Steel St. Louis River Site

This PHC is intended to describe the derivation of SSVs for the US Steel Site. The 2013 SSVs supersede sediment screening values developed previously.

10 Recommendations

- SSVs should not be used to screen or evaluate upland, intertidal or beach sediments.
- Laboratory analytical methods 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 should be used.
- If chemical concentrations in sediments adjacent to the US Steel Site exceed the SSVs (or background levels for TCDD-TEQs) further evaluation may be necessary to determine whether chemicals in the sediments could impact public health.
- If chemical concentrations in sediments are below the values developed in this PHC, the screened chemicals in sediments should 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 reasonable maximum exposures and site-specific data included in this PHC and attached appendices should be reviewed prior to using them for evaluating sediments in other water bodies.
- The SSM may be used to evaluate the relative human health impacts of different exposure routes for chemicals in sediments.

11 Public Health Action Plan

MDH will use the SSM described in this PHC 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.

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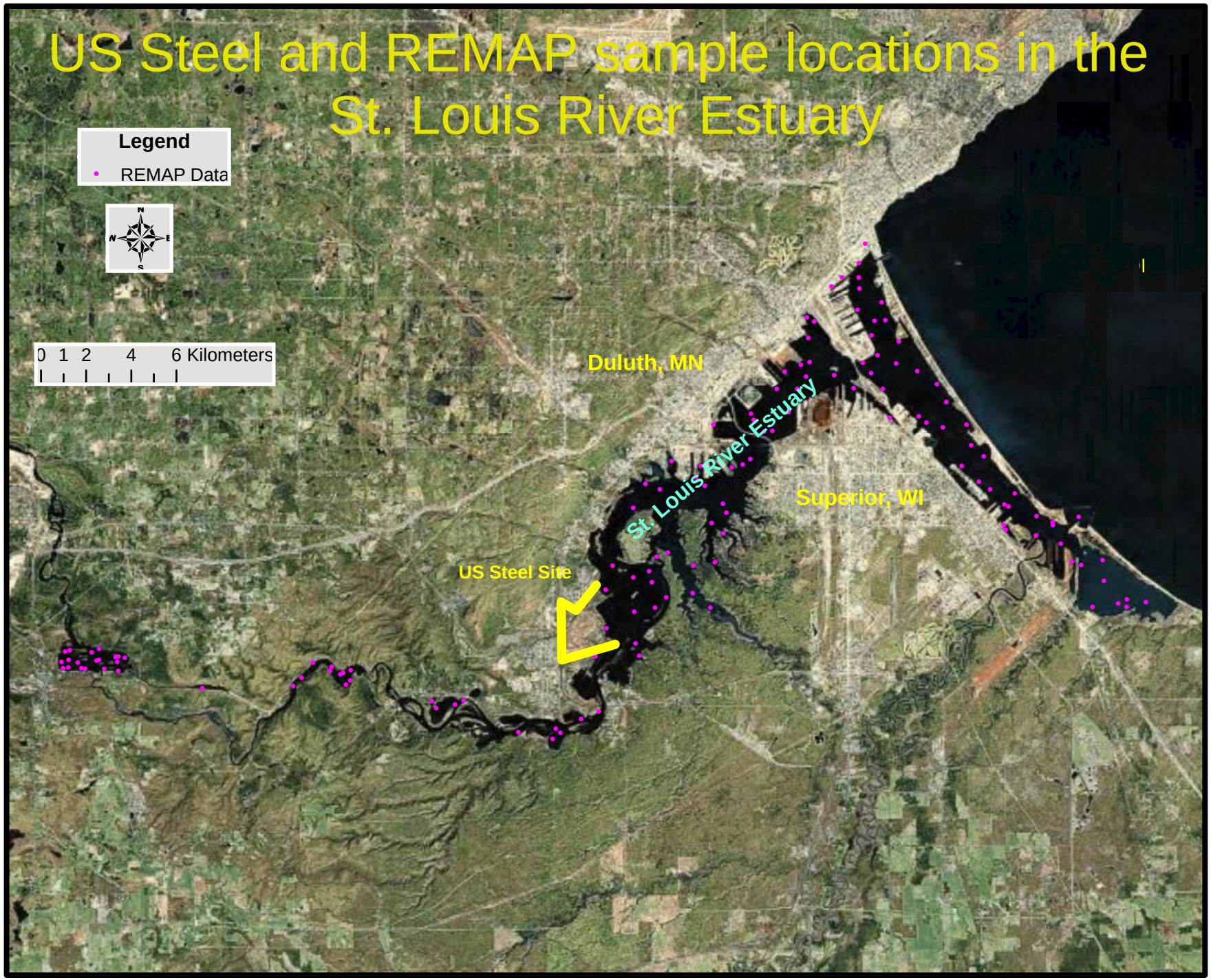
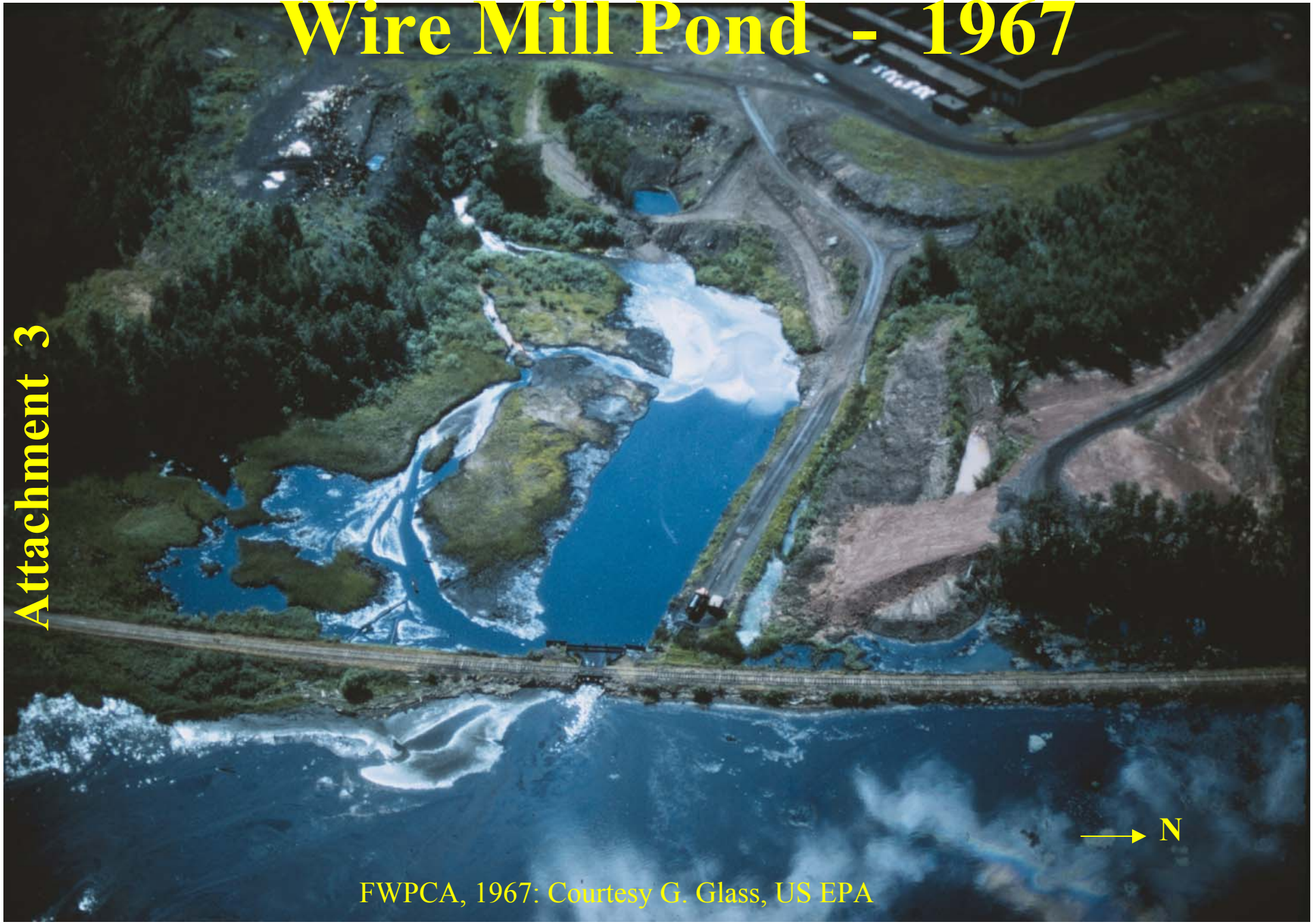
Attachment #1




Attachment 2
N
November 1951
Tweed Museum Exhibition, 1992

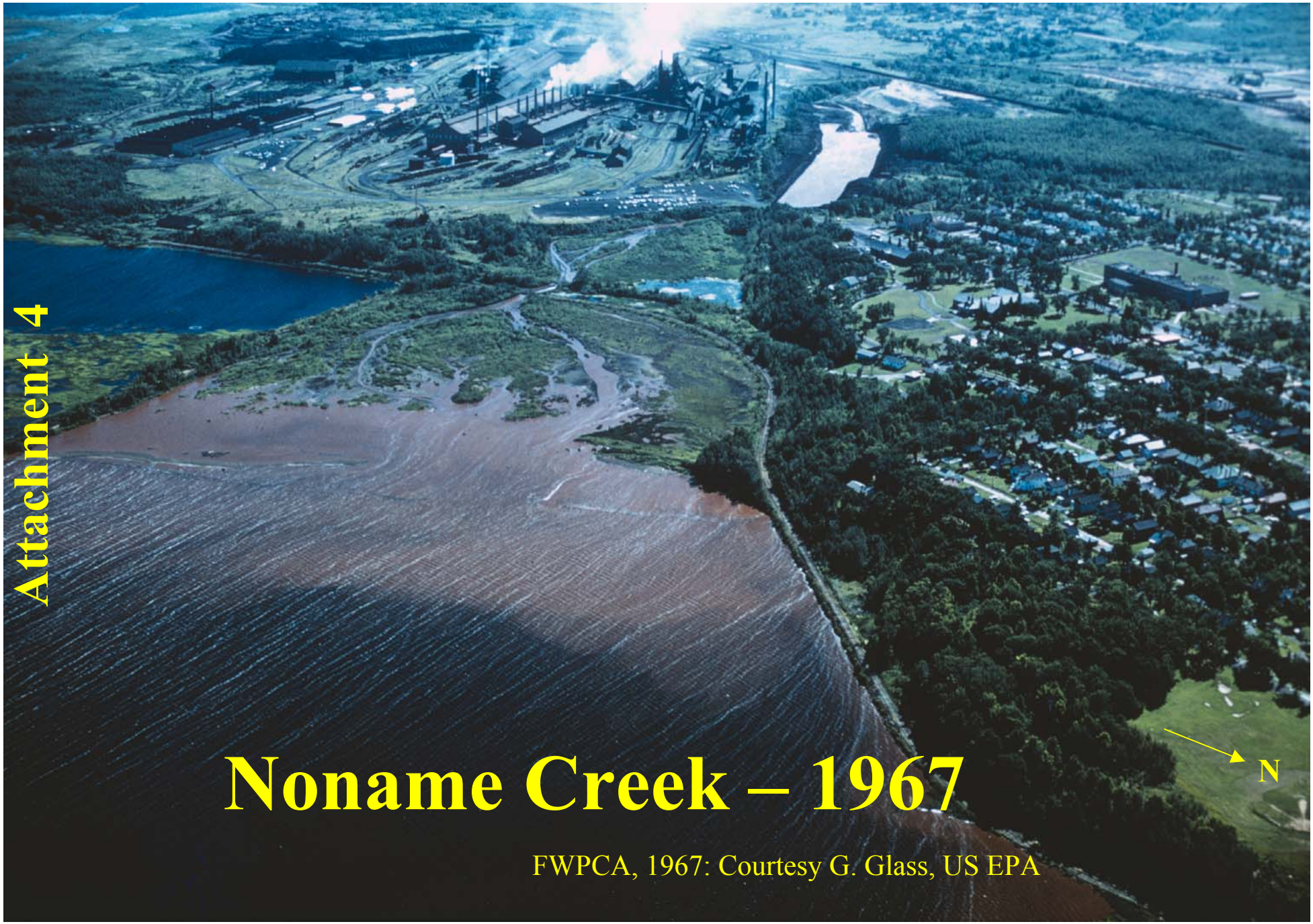
Wire Mill Pond - 1967

Attachment 3



FWPCA, 1967: Courtesy G. Glass, US EPA

Attachment 4



Noname Creek – 1967

FWPCA, 1967: Courtesy G. Glass, US EPA

USX Settling Basin 1967

FWPCA, 1967: Courtesy G. Glass, US EPA

Appendices

Appendices

Table of Contents

Appendix A – Glossary of Equation Variables and Abbreviations ---------- 1

Appendix B - Media Exposure/Contact Calculations ---------- 6

Ingestion of sediment and water.... 6

Lifetime average daily sediment and surface water age-adjusted potency equivalent intake rate calculations (cancer).... 6

Dermal exposure to sediment.... 7

Sediment Adherence Factor.... 7

Lifetime average daily dermal sediment contact rate calculation (cancer).... 8

Dermal exposure to surface water.... 8

Lifetime average daily dermal surface water contact rate calculation (cancer).... 8

Inhalation fraction.... 8

Lifetime average activity-related inhalation fraction calculation (cancer).... 9

Fish consumption.... 9

Lifetime average daily fish ingestion rate calculations (cancer).... 9

Table B-1 - Calculated Media Exposure/Contact Rates.... 9

Appendix C – Contaminated Sediments and Limitations on Mercury Bioaccumulation 10

Table C-1 - Relationship Between REMAP* Mercury and tPAH Data.... 10

Appendix D – SSM Chemical-specific Variable Defaults ---------- 12

Table D-1a - Chemical-specific Values.... 12

Table D-1b - Chemical-specific Values.... 13

Table D-2 - Toxicity Criteria.... 14

Appendix E - Screening Detection Limits for Persistent Bioaccumulative Toxicants in Sediment and Fish Tissue ---------- 16

Table E-1a - Recommended Sediment and Fish Tissue Detection Limits.... 16

Table E-1b - Recommended Sediment and Fish Tissue Detection Limits.... 17

Appendix F - Additional Information on Default Fraction Organic Carbon (f_{oc}) ------- 18

Table F-1 - Relationship between REMAP foc and tPAH data.... 18

Appendix G - PAH BSAFs ---------- 19

Developing a cPAH BSAF.... 19

Figure G-1 - Biota (Largemouth Bass, Bluegill) **and Associated Sediment PAH Concentrations** (lipid and organic carbon adjusted).... 20

Table G-1 - PAH BSAF Summary Data (Largemouth Bass, Bluegills).... 21

Figure G-2 - PAH BSAFs by Fish Tissue Analyzed.... 22

Figure G-3 - PAH BSAFs - # of 6-C Rings.... 22

Table G-2 - Sediment Screening Model Biota-Sediment Accumulation Factors(BSAFs): 2005, 2013.... 23

Appendix H - Route-Specific Sediment Screening Model Calculations - Non-Cancer -- 24

Calculating protective sediment concentrations for ingestion route exposure.... 24

Sediment ingestion.... 24

Water ingestion.... 24

Calculating protective sediment concentrations for dermal route exposure.... 24

Dermal exposure to sediment.... 24

Dermal exposure to water.... 25

Calculating protective sediment concentrations for inhalation exposure.... 26

Calculating route-specific sediment screening values for fish ingestion route exposure 26

Table H-1 - Calculated Route-Specific Sediment Screening Model Results 27
Appendix I - Route-Specific Sediment Screening Model Calculations - Cancer --------- 28
Calculating protective sediment concentrations for ingestion route exposure - Cancer .28
Sediment ingestion 28
Water ingestion 28
Calculating protective sediment concentrations for dermal exposure - Cancer 28
Dermal exposure to sediment.....28
Dermal exposure to surface water.....28
Calculating protective sediment concentrations for inhalation exposure - Cancer.....29
Calculating sediment screening values for fish ingestion exposure - Cancer.....29
Table I-1 - Route-specific Sediment Screening Model Results 30
Appendix J – Combining Routes of Exposure - Sediment Screening Model---------- 31
Sediment Screening Model Results.....31
Percent contribution by route-of-exposure.....31
Table J-1 - - Sediment Screening Model Results – Contribution from Different Routes of Exposure 32
Appendices References ---------- 33

Appendix A – Glossary of Equation Variables and Abbreviations

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} = general term representing the dermally absorbed dose from a chemical concentration in water: dependent on event duration and chemical specific factors. {$(mg/(cm^2 \cdot event))/(mg/cm^3)$}

$ABS_{SW\text{-}met}$ = 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}
ADAF = age-dependent adjustment factor {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 (US EPA, 1989)}
atm - atmosphere(s)
ATSDR - US Agency for Toxic Substances and Disease Registry
AVS - acid volatile sulfide
β = ratio of stratum corneum and epidermis permiabilities {unitless} (*Equation A-25*)
BAF - biota accumulation factor
BaP - benzo[a]pyrene
BaP-PEQ = benzo(a)pyrene potency equivalent {appropriate units of concentration or exposure}
BaP-RPF - benzo[a]pyrene relative potency factor
BCF - biota concentration factor
BNS - Binational Toxics Strategy
BSAF = biota-sediment accumulation factor - non-polar organics - {$kg_{Sed\ oc}/kg_{fish\ lipid}$}
- mercury - {kg_{Sed}/kg_{fish}}
BW = body weight {kg}
C_A = chemical concentration in air {mg/m^3} (*PHC Equation 3)*
CAC - community action committee
CDC - Center for Disease Control and Prevention
CERCLIS - Comprehensive Environmental Response, Compensation, and Liability Information System
$CF_{\mu g/mg}$ = conversion factor {1,000 µg/mg}
$CF_{cm3/L}$ = conversion factor {1,000 cm^3/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}
$CF_{L/m3}$ = conversion factor {1,000 L/m^3}
$CF_{mg/kg}$ = conversion factor {1,000,000 mg/kg}
$CF_{mL/L}$ = conversion factor {1,000 mL/L}
CL - confidence limit(s)
cm - centimeter(s)
COC - chemical of concern

cPAH	- carcinogenic PAH (polycyclic aromatic hydrocarbon)	
cPCB	- carcinogenic PCB (polychlorinated biphenyl)	
C_{Sed}	= chemical concentration in sediment	{mg/kg}
CSF	= oral cancer slope factor	$\{(mg/(kg_{bw}\cdot d))^{-1}\}$
C_{SW}	= chemical concentration in surface water	{mg/L} (*PHC Equation 2)*
d	- day(s)	
DDT	- dichlorodiphenyltrichloroethane	
DF_{sed-sw}	= default sediment-to-surfacewater dilution factor	{10, unitless}
DF_{sw-air}	= default surfacewater-to-air dilution factor	{50, unitless}
dl	- deciliter(s)	
DNA	- deoxyribonucleic acid	
ED	= event duration	{hr/event}
EF_{swm}	= event frequency - swimming	{event/yr}
EF_{swm-d}	= event-day frequency	{event-d/yr}
EF_{wad}	= event frequency - wading	{event/yr}
EF_{wad-d}	= event-day frequency	{event-d/yr}
EPA	- US Environmental Protection Agency	
FA	= fraction absorbed from water	{unitless}
$Fish_{Ing}$	= fish ingestion rate	$\{g_{fish}/(kg_{bw}\cdot d)\}$(*Equation A-12*)
$Fish_{Ing-c}$	= 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}
g	- gram(s)	
HBV	= health-based value	{*typ* $mg/(kg_{bw}\cdot d)$}
HCB	- hexachlorobenzene	
HpCB	- heptachlorinated biphenyl	
HpCDD	- heptachlorinated dibenzo-p-dioxin	
HpCDF	- heptachlorinated dibenzofuran	
hr	- hour(s)	
HxCB	- hexachlorinated biphenyl	
HxCDD	- hexachlorinated dibenzo-p-dioxin	
HxCDF	- hexachlorinated dibenzofuran	
IARC	- International Agency for Research on Cancer	
$Ing_{Sed(swm)}$	= sediment ingested per hour swimming	{mg/hr} (*Equation A-2*)
$Ing_{Sed(wad)}$	= sediment ingested per hour wading	{mg/hr} (*Equation A-2*)
$Ing_{SW(swm)}$	= 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	$\{m^3/d\}$
IRIS	- Integrated Risk Information System (EPA)	
kg	- kilogram(s)	
K_H	= Henry's Law constant	$\{atm\text{-}m^3/mol\}$
K_{oc}	= organic carbon partitioning constant	{L/kg}
K_{ow}	= octanol-water partitioning constant	{unitless}
K_p	= permeability coefficient	{cm/hr}

L	- liter(s)	
m	- meter(s)	
m^3	- cubic meter(s)	
MDH	- Minnesota Department of Health	
MF	= fish meal frequency	{meal/week}
mg	- milligram(s)	
mL	- milliliter(s)	
MN	- Minnesota	
mol	- mole(s)	
MPCA	- Minnesota Pollution Control Agency	
MRL	- minimum risk level	
MW	= molecular weight of chemicals of interest	{g/mol}
MW	- molecular weight	
n	- number of samples	
NAPL	- non-aqueous phase liquid	
ng	- nanogram(s)	
nPAH	- non-carcinogenic PAH	
NPL	- National Priority List	
NTP	- National Toxicology Program	
OC	- organic carbon	{unitless}
OCDD	- octachlorinated dibenzo-p-dioxin	
OCDF	- octachlorinated dibenzofuran	
OCS	- octachlorostyrene	
OEHHA	- Office of Environmental Health Hazard Assessment (California State)	
PAH	- polycyclic aromatic hydrocarbon	
PBDD	- polybrominated dibenzo-p-dioxin	
PBDE	- polybrominated diphenylether	
PBDF	- polybrominated dibenzofuran	
PCB	- polychlorinated biphenyl	
PCDD	- polychlorinated dibenzo-p-dioxin	
PCDF	- polychlorinated dibenzofuran	
PeCB	- pentachlorinated biphenyl	
PeCDD	- pentachlorinated dibenzo-p-dioxin	
PeCDF	- pentachlorinated dibenzofuran	
PEF	= potency equivalence factor	{unitless}
PEQ	- potency equivalence	
PHC	- Public Health Consultation	
PLP	- Permanent List of Priorities (Minnesota)	
R	= ideal gas constant (@ 1 atm)	{0.082057 L/ (mol · ºK)}
REMAP	- Regional Environmental Monitoring and Assessment Program	
RfC	= reference concentration (air)	{mg/m^3}
RfD	= reference dose (ingestion)	{mg/(kg·d)}
RME	- reasonable maximum exposure	
RPF	= relative potency factor	{unitless}
RSC	= relative source contribution	{unitless}
RTK	- Right-to-Know Act	

$SA_{frac\text{-}swm}$ = percent of body exposed swimming {unitless}
$SA_{frac\text{-}wad}$ = percent of body exposed wading {unitless}
SA_{ttl} = total surface area {cm^2}
SD - standard deviation
Sed_{Derm} = dermal sediment exposure {$mg_{sed}/(kg_{bw}\cdot d)$}(*Equation A-6*)
$Sed_{Derm\text{-}c}$ = dermal sediment exposure - lifetime average {$mg_{sed}/(kg_{bw}\cdot d)$}(*Equation A-7*)
Sed_{Ing} = amount of sediment ingested {$mg_{sed}/(kg_{bw}\cdot d)$} (*Equation A-3*)
$Sed_{Ing\text{-}c}$ = amount of sediment ingested - lifetime average {$mg_{sed}/(kg_{bw}\cdot d)$}(*Equation A-5*)
SLRIDT - St. Louis River Interlake-Duluth Tar Site
SQT = MPCA Sediment Quality Target {mg/kg}
SRV - Soil Reference Value (MPCA)
SSM - Sediment Screening Model
SS_{SW} = suspended sediment concentration in surface water {mg/L}
SSV = Sediment Screening Value {mg/kg}
$SSV_{\%x}$ = % 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-34, A-35*)
$SSV_{Fish\text{-}c}$ = route-specific sediment value (cancer) - fish consumption {mg/kg}(*Equations A-44, A-45*)
SSV_{Inh} = route-specific sediment value (chronic) - inhalation {mg/kg} (*Equation A-32*)
$SSV_{Inh\text{-}c}$ = 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*)
$SSV_{Sed(Derm)\text{-}c}$ = 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*)
$SSV_{Sed(Ing)\text{-}c}$ = 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*)
$SSV_{SW(Derm)\text{-}c}$ = 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*)
$SSV_{SW(Ing)\text{-}c}$ = route-specific sediment value (cancer) - water ingestion {mg/kg} (*Equation A-38*)
SSV_{ttl} = sediment screening value - chronic {mg/kg} (*Equation A-46*)
$SSV_{ttl\text{-}c}$ = sediment screening value - cancer {mg/kg} (*Equation A-47*)
STORET - EPA Storage and Retrieval database
SWC_{Inh} = partial water screening concentration (chronic)
- surface water inhalation {mg/L} (*Equation A-33*)
$SWC_{Inh\text{-}c}$ = partial water screening concentration (cancer)
- surface water inhalation {mg/L} (*Equations A-43*)
$SWC_{SW(Derm)}$ = partial water screening concentration (chronic)
- surface water dermal {mg/L}(*Equations A-20, A-20a*)
$SWC_{SW(Derm)\text{-}c}$ = partial water screening concentration (cancer)
- surface water dermal {mg/L} (*Equation A-40*)
$SWC_{SW(Ing)}$ = partial water screening concentration (chronic)
- water ingestion {mg/L} (*Equation A-17*)
$SWC_{SW(Ing)\text{-}c}$ = partial water screening concentration (cancer)
- water ingestion {mg/L} (*Equation A-37*)
SW_{Derm} = surface area exposed to surface water {$cm^2\cdot$event/$(kg_{bw}\cdot d)$}(*Equation A-8*)
$SW_{Derm\text{-}c}$ = surface area exposed to surface water - lifetime average {$cm^2\cdot$event/$(kg_{bw}\cdot d)$}(*Equation A-9*)
SW_{Ing} = amount of water ingested {$L/(kg_{bw}\cdot d)$} (*Equation A-1*)
$SW_{Ing\text{-}c}$ = amount of water ingested - lifetime average {$L/(kg_{bw}\cdot d)$} (*Equation A-4*)

T	= temperature	{293.13ºK default}
τ	= lag time per event	{hr/event} (*Equation A-24*)
t^*	= time to steady state	{hr} (*Equation A-26, A-27*)
TCB	- tetrachlorinated biphenyl	
TCDD	- tetrachlorinated dibenzo-p-dioxin	
TCDD-TEQ	= 2,3,7,8-tetrachlorodibenzo-p-dioxin toxic equivalents	{mg/(kg_{bw}·d)} (*PHC Equation 1)*
TCDF	- tetrachlorinated dibenzofuran	
TEF	= toxic equivalence factor	{unitless}
tPCB	- total polychlorinated biphenyl	
µg	- microgram(s)	
UR_c	= unit risk - cancer	{(µg/m^3)$^{-1}$}
VOC	- volatile organic compound	
WHO	- World Health Organization	
wt.	- weight	
yr	- year(s)	

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:

SW_{Ing} { $L/(kg_{bw} \cdot d)$ } = $(Ing_{SW(wad)} * ED_{wad} * EF_{wad} + Ing_{SW(swm)} * ED_{swm} * EF_{swm}) / (BW * CF_{d/y}) * CF_{L/mL}$ *Equation A-1.*

*$Child_{2-5}$ example: SW_{Ing} =(25 mL/hr * 17.2 events/yr*0.5 hr/event+250 mL/hr * 103events/yr*0.5hr/event) / (17.4 kg_{bw} * 365 d/yr) * 0.001 L/mL*

=0.206 mg/($kg_{bw} \cdot d$)

$Ing_{Sed(wad,swm)}$ {mg_{sed}/hr } = $Ing_{SW(wad,swm)} * SS_{SW} / CF_{mL/L}$ *Equation A-2.*

*$Child_{2-5}$ 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-3.*

*$Child_{2-5}$ example: Sed_{Ing} =(9.25 mg/hr*17.2 events/yr*0.5 hr/event+92.5 mg/hr * 103events/yr*0.5hr/event) / (17.4 kg_{bw} * 365 d/yr)*

=0.762 mg/($kg_{bw} \cdot d$)

Calculation results for all ages are shown in Table B-1.

<u>Lifetime average daily sediment and surface water age-adjusted potency equivalent intake rate calculations (cancer)</u>

$SW_{Ing\text{-}c}$ { $L/(kg_{bw} \cdot d)$ } = $(\sum_{i=0}^{32} (SW_{Ing(i)} * ADAF_{(i)})) / AT_{(c)}$ *Equation A-4.*

$Sed_{Ing\text{-}c}$ { $mg_{sed}/(kg_{bw} \cdot d)$ } = $(\sum_{i=0}^{32} (Sed_{Ing(i)} * ADAF_{(i)})) / AT_{(c)}$ *Equation A-5.*

Where:

$SW_{Ing(i)}$ = (SW_{Ing} at age i) * 1 yr
$Sed_{Ing(i)}$ = (Sed_{Ing} at age i) * 1 yr
$ADAF_{(i)}$ = 10 for i = 0, 1 years of age
$ADAF_{(i)}$ = 3 for i = 2 - 15 years of age
$ADAF_{(i)}$ = 1 for i > 15 years of age

For individual carginogens with potency data based on whole life human epidemiology studies:

$ADAF_{(i)}$ = 1 for all i

Calculation results are shown in Table B-1.

Dermal exposure to sediment

As shown in Table 5 (PHC Section 3.3.4), 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 PHC Table 1 (PHC 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:

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

Calculation results are shown in Table B-1 (Table 5; PHC Section 3.3.4).

Sediment Adherence Factor

Sediment adherence to skin is activity dependent and has a large impact on dermal exposure. The 1992 EPA Interim Dermal Guidance (US EPA, 1992) recommended using 0.2 to 1.0 mg/cm^2 as soil adherence factors for most exposures. Current interim guidance (US EPA, 2001) 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 95^{th} 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 D-1b) from the EPA Interim Dermal Guidance (US EPA, 2001) were used as sediment absorption fractions in this PHC. Most of these chemical-specific absorption factors are based on studies by Wester et al. (1990; 1993a; 1993b). 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; US EPA, 2001 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 PHC. 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 (0-15) and adults (>15), respectively PHC Table 5 (PHC Section 3.3.4).

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

$$Sed_{Derm\text{-}c}\ \{\ mg_{sed}/(kg_{bw}\cdot d)\ \} = (\sum_{i=0}^{32}(Sed_{Derm(i)} * ADAF_{(i)})) / AT_{(c)} \qquad \textit{Equation A-7.}$$

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:

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

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:

$$SW_{Derm\text{-}c}\ \{\ cm^2\cdot event/(kg_{bw}\cdot d)\ \} = (\sum_{i=0}^{32}(SW_{Derm(i)} * ADAF_{(i)})) / AT_{(c)} \qquad \textit{Equation A-9.}$$

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} = $(EF_{wad} * ED_{wad} + EF_{swm} * ED_{swm}) / (CF_{d/y} * CF_{hr/d})$ *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:

$$Inh_{frac\text{-}c} \{ \text{unitless} \} = \left(\sum_{i=0}^{32} (Inh_{frac(i)} * ADAF_{(i)})\right) / AT_{(c)}$$ *Equation A-11.*

Calculation results are shown in Table B-1.

Fish consumption
In Minnesota, a water body is considered impaired if the recommended fish consumption rate for any member of the population is less than one meal per week (Minnesota Rule 7050.0150). The default consumption of fish for an exposed individual (150 pound adult) is assumed to be 210 grams of fish fillet per week. In the SSM, consumption rate (g fish / kg body weight) is assumed to be similar for different age groups, with the exception of 0 to 1 year of age, when fish consumption is assumed to be ½ of the 2 to 32 year-old consumption (see Table B-1).

$Fish_{Ing}$ { $g_{fish}/(kg_{bw} \cdot d)$ }= $MF * AC / (BW * CF_{d/wk})$ *Equation A-12.*

Calculation results are shown in Table B-1.

Lifetime average daily fish ingestion rate calculations (cancer)
Lifetime consumption of fish is:

$$Fish_{Ing\text{-}c} \{ g_{fish}/(kg_{bw} \cdot d) \} = \left(\sum_{i=0}^{32} (Fish_{Ing} * ADAF_{(i)})\right) / AT_{(c)}$$ *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	**0 - 1**	0.691	0.00187	8.42	63.7	0.00344	0.220
	2 - 5	0.763	0.00206	14.5	109	0.00686	0.441
	6 - 15	0.300	0.000810	10.5	79.4	0.00686	0.441
	16 - 32	0.00849	0.0000229	3.57	18.8	0.00245	0.441
Cancer lifetime Annual Average Exposures		$Sed_{Ing\text{-}c}$	$SW_{Ing\text{-}c}$	$Sed_{Derm\text{-}c}$	$SW_{Derm\text{-}c}$	$Inh_{frac\text{-}c}$	$Ing_{Fish\text{-}c}$
	0 - 69	0.108	0.000292	3.44	24.0	0.00207	0.202
	0 - 69 (ADAF)	3	0.00124	10.3	75.5	0.00569	0.435

Appendix C – Contaminated Sediments and Limitations on Mercury Bioaccumulation

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 Sediment Quality Targets (MPCA, 2007; discussed in PHC Section 7.1). In areas with tPAH concentrations above Level I criteria (1.6 mg/kg), the aquatic food chain is likely affected. In areas above Level II criteria (23 mg/kg) it is unlikely that a population of benthic invertebrates is surviving. Therefore, mercury in sediments with greater than 1.6 mg/kg tPAH may not enter the food chain as readily as mercury in areas where the food chain is more robust, and at levels greater than 23 mg/kg it is unlikely that there is a complete food chain for bioaccumulation of mercury. Table C-1 shows the mean and median mercury concentrations at all locations where tPAH concentrations were below 0.25, 0.5, 1, **1.6**, 2, 4, 8, 16, **23**, 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

	REMAP sites w/tPAH Concentrations											all REMAP estuary locations
	(tPAH < MPCA SQTs)				(tPAH > MPCA Level I SQT)					(tPAH >MPCA Level II SQT)		
	<0.25 mg/kg	<0.5 mg/kg	<1 mg/kg	<1.6 mg/kg	<2 mg/kg	<4 mg/kg	<8 mg/kg	<16 mg/kg	<23 mg/kg	<32 mg/kg	All tPAH sites	mercury sites
# of sites (n=)	8	12	19	21	23	27	29	37	40	40	42	87
tHg Geometric Mean (mg/kg)	0.012	0.013	0.017	0.019	0.020	0.026	0.027	0.047	0.051	0.051	0.054	0.078
tHg Arithmetic Mean (mg/kg)	0.013	0.015	0.022	0.024	0.027	0.047	0.050	0.131	0.135	0.135	0.138	0.150
tHg Median (mg/kg)	0.012	0.014	0.018	0.020	0.020	0.023	0.023	0.034	0.038	0.038	0.045	0.100

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

* a description of REMAP data (Regional Environmental Monitoring and Assessment Program; US EPA, 1995a) is in PHC Section 1

Contamination may affect the productivity of benthic communities at tPAH concentrations above 1.6 mg/kg (shaded in Table C-1). As a result, the contribution of biomass to the food chain from areas with greater than 1.6 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 = 1.6 mg/kg) is greater than 0.024 mg/kg (arithmetic mean) (geometric mean = 0.019 mg/kg; median = 0.020 mg/kg). And the level at which the impairment of the food chain by tPAH (i.e., tPAH > 23 mg/kg) is likely to be severe is at mercury concentrations of 0.135 mg/kg (arithmetic mean) (geometric mean 0.051 mg/kg; median 0.038 mg/kg) and greater. 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 below 0.05 mg/kg.

Data in Table C-1 suggest that using the arithmetic mean of mercury data from all REMAP sites is not likely to provide a reasonable representation for mercury accumulation in the St. Louis River food chain, because the food chain in areas that contain higher mercury

concentrations is likely to be significantly impaired by PAHs in the sediment. Therefore, the geometric mean mercury concentration (0.78 mg/kg) was used in site-specific BSAF calculations because it provides less weight to the modeled accumulation in more highly contaminated areas. This resulted in a waterbody BSAF of 8.2 for 20 inch walleyes. 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 (PHC 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; US EPA, 1997). Use of a lower estimated effective mercury concentration of 0.047 mg/kg (for sites with REMAP tPAH concentrations less than 4 mg/kg) 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 0.02 to 0.01 mg/kg. A BSAF of 10.3 for northern pike (~ 41.2 adjusting for dry tissue; see PHC 5.3.3.3) is at the high end of the range of published BSAFs.

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

Appendix D – SSM Chemical-specific Variable Defaults

Table D-1a - Chemical-specific Values

	CAS No.	MW		K_H		K_{oc}		Log K_{ow}		BSAF	
		g/mol		atm-m^3/mol		L/kg		Unitless		Chem type dependent	
Metals - Inorganics										mg/kg_{fish} / mg/kg_{sed}	
Arsenic	7440-38-2	74.9									
Cadmium	7440-43-9	112									
Chromium III	16065-83-1	52									
Chromium VI	18540-29-9	52									
Copper	7440-50-8	63.5									
Cyanide	57-12-5	26									
Lead	7439-92-1	207									
Mercury (SSV - tHg; exp - tHg)	7439-97-6	201		2.5E-02	a						
Methyl Mercury (SSV - tHg; exp - MeHg)	22967-92-6	215								8.2	b
Nickel	various	58.7									
Zinc	7440-66-6	65.4									
Volatile organic compounds (VOCs)											
Benzene	71-43-2	78.1		5.6E-03	a	150	a	2.13	a		
Ethyl benzene	100-41-4	106		7.9E-03	a	450	a	3.15	a		
Styrene	100-42-5	104		2.8E-03	a	450	a	2.95	a		
Toluene	108-88-3	92.1		6.6E-03	a	230	a	2.73	a		
Xylenes (mixed)	1330-20-7	106		5.2E-03	a	380	a	3.12	a		
Polycyclic Aromatic Hydrocarbons (PAHs)										$mg/kg_{fish\ lipid}$ / $mg/kg_{organic\ carbon}$	
Acenaphthene	83-32-9	154		1.8E-04	a	5000	a	3.92	a	0.65	c
Acenaphthylene (toxicity surrogate - pyrene)	208-96-8	152		1.1E-04	a	5000	a	3.94	a	0.65	c
Anthracene	120-12-7	178		5.6E-05	a	16000	a	4.45	a	0.2	c
Benzo(a)pyrene equivalents (BaP-PEQs)	50-32-8	252		4.6E-07	a	590000	a	6.13	a	0.085	c
Fluoranthene	206-44-0	202		8.9E-06	a	55000	a	5.16	a	0.2	c
Fluorene	86-73-7	170		9.6E-05	a	9200	a	4.18	a	0.65	c
Methylnaphthalene (toxicity surrogate - naphthalene)	1321-94-4	142		5.1E-04	a	2500	a	3.87	a	0.65	c
Naphthalene	91-20-3	128		4.4E-04	a	1500	a	3.3	a	0.65	c
Perylene (toxicity surrogate - pyrene)	198-55-0	252		3.7E-06	a	60000	a	6.25	a	0.0008	c
Phenanthrene (toxicity surrogate - pyrene)	85-01-8	178		4.2E-05	a	17000	a	4.46	a	0.2	c
Pyrene	129-00-0	202		1.2E-05	a	54000	a	4.88	a	0.016	c
Dibenzo-p-dioxins/dibenzofurans											
2,3,7,8-TCDD equivalents (TCDD-TEQs)	1746-01-6	322		5.0E-05	a	250000	a	6.8	a	1	d
Other Organics											
Carbazole	86-74-8	167		1.2E-07	a	9200	a	3.72	a		
Hexachlorobenzene	118-74-1	285		1.7E-03	a	6200	a	5.73	a	0.1	d
Octachlorostyrene	29082-74-4	380		2.3E-04	a	55000	a	7.46	a	0.98	e
Pentachlorophenol	87-86-5	266		2.5E-08	a	5000	a	5.12	a		
Polychlorinated Biphenyls (PCBs)	1336-36-3	292	a	4.2E-04	a	78000	a	7.1	a	2.6	f
Quinoline	91-22-5	129		1.7E-06	a	1500	a	2.03	a		

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

References:

[a] EPA EPI Database (US EPA, 2012a)
[b] MDH calculation (see PHC Section 5.3.2.3)
[c] MDH calculation (see Appendix G)
[d] (Washington State Department of Ecology, 1997)
[e] (US EPA, 1995b)
[f] (Washington State Department of Health, 1995; 1996)

Table D-1b - Chemical-specific Values

	CAS No.	ABS_{GI}	ABS_{Sed}	ABS_{Derm}	K_p	FA	ABS_{SW}	AF_{ED}
		Unitless	Unitless	Unitless	cm/hr	Unitless	(mg/(cm^2 event)) / (mg/cm^3) (calculated)	hr/event (calculated)
Metals - Inorganics								
Arsenic	7440-38-2	1	1	0.03	0.001		0.0005	
Cadmium	7440-43-9	0.025	1	0.001	0.001		0.0005	
		0.05						
Chromium III	16065-83-1	0.013	1		0.001		0.0005	
Chromium VI	18540-29-9	0.025	1		0.002		0.001	
Copper	7440-50-8	1	1		0.001		0.0005	
Cyanide	57-12-5	1	1		0.001		0.0005	
Lead	7439-92-1				0.0001		0.00005	
Mercury (SSV - tHg; exp - tHg)	7439-97-6	0.07	1		0.001		0.0005	
Methyl Mercury (SSV - tHg; exp - MeHg)	22967-92-6	1	1					
Nickel	various	0.04	1		0.0002		0.0001	
Zinc	7440-66-6	1	1		0.0006		0.0003	
Volatile organic compounds (VOCs)								
Benzene	71-43-2	1	1	0.5	0.015	1	0.016	1.05
Ethyl benzene	100-41-4	1	1	0.5	0.049	1	0.061	1.25
Styrene	100-42-5	1	1	0.5	0.037	1	0.046	1.24
Toluene	108-88-3	1	1	0.5	0.031	1	0.036	1.15
Xylenes (mixed)	1330-20-7	1	1	0.5	0.053	1	0.066	1.25
Polycyclic Aromatic Hydrocarbons (PAHs)								
Acenaphthene	83-32-9	1	1	0.13	0.084	1	0.14	1.71
Acenaphthylene (toxicity surrogate - pyrene)	208-96-8	1	1	0.13	0.089	1	0.15	1.69
Anthracene	120-12-7	1	1	0.13	0.14	1	0.28	2
Benzo(a)pyrene equivalents (BaP-PEQs)	50-32-8	1	1	0.13	0.7	1	2.3	3.22
Fluoranthene	206-44-0	1	1	0.13	0.22	1	0.51	2.33
Fluorene	86-73-7	1	1	0.13	0.1	1	0.19	1.9
Methylnaphthalene (toxicity surrogate - naphthalene)	1321-94-4	1	1	0.13	0.091	1	0.14	1.58
Naphthalene	91-20-3	1	1	0.13	0.047	1	0.068	1.45
Perylene (toxicity surrogate - pyrene)	198-55-0	1	1	0.13	0.82	1	2.6	3.22
Phenanthrene (toxicity surrogate - pyrene)	85-01-8	1	1	0.13	0.14	1	0.28	2
Pyrene	129-00-0	1	1	0.13	0.19	1	0.45	2.33
Dibenzo-p-dioxins/dibenzofurans								
2,3,7,8-TCDD equivalents (TCDD-TEQs)	1746-01-6	1	1	0.03	0.81	0.5	2	2.52
Other Organics								
Carbazole	86-74-8	1	1	0.1	0.052	1	0.098	1.86
Hexachlorobenzene	118-74-1	1	1	0.1	0.13	0.9	0.47	3.58
Octachlorostyrene	29082-74-4	1	1	0.1	0.99	1	7.3	7.34
Pentachlorophenol	87-86-5	1	1	0.25	0.39	1	1.4	3.52
Polychlorinated Biphenyls (PCBs)	1336-36-3	1	1	0.14	1.8	0.5	3.7	2.08
Quinoline	91-22-5	1	1	0.1	0.0066 *	1	0.0095	1.46

Reference: (US EPA, 2001)

ABS_{GI} = fraction of applied dose absorbed in primary (RfD) study (unitless)
ABS_{Sed} = oral absorption adjustment - relative bioavailability (unitless)
ABS_{Derm} = dermally absorbed fraction (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-22* or *Equation A-23.* ((mg/cm^2/event)/(mg/cm^3))

Table D-2 - Toxicity Criteria

	CAS No.	Reference Dose (RfD) mg/(kg·d)		Reference Concentration mg/m^3		Cancer Slope Factor (CSF) $(mg/(kg·d))^{-1}$		Cancer Unit Risk (UR_c) $(\mu g/m^3)^{-1}$	
Metals - Inorganics									
Arsenic	7440-38-2	0.0003	a,c			1.5	a	0.0033	b
Cadmium	7440-43-9	0.0001	c					0.0018	a
Chromium III	16065-83-1	1.5	a						
Chromium VI	18540-29-9	0.001	c			0.5	b	0.012	a
Copper	7440-50-8	0.04	d						
Cyanide	57-12-5	0.006	a						
Lead *	7439-92-1								
Mercury (SSV - tHg; exp - tHg)	7439-97-6	0.0003	a						
Methyl Mercury (SSV - tHg; exp - MeHg)	22967-92-6	0.0001	a						
Nickel	various	0.02	a						
Zinc	7440-66-6	0.3	a,c						
Volatile organic compounds (VOCs)									
Benzene	71-43-2	0.004	c	0.01	c	0.055	a	0.0000078	a
Ethyl benzene	100-41-4	0.1	a	0.3	c				
Styrene	100-42-5	0.2	c	1	a				
Toluene	108-88-3	0.08	e	0.4	e				
Xylenes (mixed)	1330-20-7	0.2	a,c	0.1	a				
Polycyclic Aromatic Hydrocarbons (PAHs)									
Acenaphthene	83-32-9	0.06	a	0.21	f				
Acenaphthylene (toxicity surrogate - pyrene)	208-96-8	0.03		0.11					
Anthracene	120-12-7	0.3	a	1.1	f				
Benzo(a)pyrene equivalents (BaP-PEQs)	50-32-8					1.7	j	0.0011	b
Fluoranthene	206-44-0	0.04	a	0.14	f				
Fluorene	86-73-7	0.04	a	0.14	f				
Methylnaphthalene (toxicity surrogate - naphthalene)	1321-94-4	0.016		0.009					
Naphthalene	91-20-3	0.016	e	0.009	e				
Perylene (toxicity surrogate - pyrene)	198-55-0	0.03		0.11					
Phenanthrene (toxicity surrogate - pyrene)	85-01-8	0.03		0.11					
Pyrene	129-00-0	0.03	a	0.11	f				
Dibenzo-p-dioxins/dibenzofurans									
2,3,7,8-TCDD equivalents (TCDD-TEQs)	1746-01-6	7E-10	a	0.00000004	b	1400000	e	38	b
Other Organics									
Carbazole	86-74-8					0.02	g	0.0000041	h
Hexachlorobenzene	118-74-1	0.00005	c	0.000092	h	1.6	a	0.00033	h
Octachlorostyrene	29082-74-4	0.00003	i	0.000055	h				
Pentachlorophenol	87-86-5	0.005	a	0.0092	h	0.4	a	0.000083	h
Polychlorinated Biphenyls (PCBs)	1336-36-3	2E-05 *	a,c	0.000037	h	2	a	0.0001	a
Quinoline	91-22-5					3	a	0.00062	h

* No toxicity criteria for lead (SSV based on MPCA lead Soil Reference Value)

References:

a (US EPA, 2012b)
b (CA OEHHA, 2002; 2003)
c (ATSDR, 2012)
d (CA OEHHA, 2008; 2011)
e (MDH, 2002)
f (US EPA, 1996)
g (US EPA, 2004)
h Route-to-route (chronic: see *Equation A-14*, below)
Route-to-route (cancer: see *Equation A-15*, below)
i (New York State DEC, 1997)
j (MDH, 2012)

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

$$RfC\ \{\ mg/m^3\ \} = RfD * BW_{2\text{-}5} / InhRate_{2\text{-}5} = RfD * 17.4\ kg / 9.5\ m^3/d$$ *Equation A-14.*

Where:
$BW_{2\text{-}5}$ from PHC Table 1; Section 3
$InhRate_{2\text{-}5}$ from US EPA (2011)

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

$$UR_c\ \{\ (\mu g/m^3)^{-1}\ \} = CSF / BW_{adult} * InhRate_{adult} = CSF / 77.5\ kg * 16\ m^3/d$$ *Equation A-15.*

Where:
BW_{adult} from US EPA (2011)
$InhRate_{adult}$ from US EPA (2011)

Appendix E - Screening Detection Limits for Persistent Bioaccumulative Toxicants 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 are 20 times below the SSM results for chemicals (PAHs and dioxins) that may be evaluated using a potency equivalence scheme; and 10 times below other SSM results. Detection limits for analysis of fish tissue are calculated directly from health criteria and assumed ingestion rates, and include similar margins to allow for possible health endpoint additivity.

While the fish consumption exposure pathway was not included in SSM evaluation of inorganic chemicals and VOCs, appropriate fish tissue detection limits are included for all chemicals with SSV. Detection of contaminants in fish tissue above the recommended detection limits suggests that the pathway should be evaluated further. 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 be needed to characterize risks to aquatic organisms and the environment.

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

Chemicals		Recommended	
		Sediment	Fish
Metals - Inorganics		mg/kg	mg/kg
	Arsenic	3	0.003
	Chromium VI	4	0.005
	Cyanide	200	0.3
	Lead *	30	
	Mercury	0.002	0.02
Volatile organic compounds (VOCs)			
	Benzene	0.08	0.04
	Ethyl benzene	7	5
	Styrene	30	9
	Toluene	5	4
	Xylenes (mixed)	4	9
Other Organics			
	Carbazole	8	0.1
	Hexachlorobenzene	0.01	0.001
	Octachlorostyrene	0.002	0.001
	Pentachlorophenol	0.07	0.006
	Polychlorinated Biphenyls (PCBs)as Arochlor 1254	0.0005	0.0009
	Quinoline	0.04	0.0008

* Based on MPCA lead SRV of 300 mg/kg in soil. See lead discussion in PHC Section 4.1.3

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

Chemicals				Recommended (calculated) DLs		
					Sediment	Fish Tissue
Benzo(a)pyrene Potency Equivalents				**RPFs**	**mg/kg**	**mg/kg**
	Benz(a)anthracene	Benzo(j)fluoranthene	Cyclopenta(c,d)pyrene	0.2	0.05	3E-03
	Benzo(a)pyrene	7H-Dibenzo(c,g)carbazole	5-Methylchrysene	1	0.01	7E-04
	Benzo(b)fluoranthene			0.8	0.01	8E-04
	Benzo(c)fluorene			20	5E-04	3E-05
	Benzo(g,h,i)perylene			0.009	1	0.08
	Benzo(j)fluoranthene			0.3	0.03	2E-03
	Benzo(k)fluoranthene			0.03	0.3	0.02
	Chrysene	Fluoranthene	Indeno(1,2,3-cd)pyrene	0.1	0.1	7E-03
	Dibenz(a,h)anthracene	6-Nitrochrysene		10	1E-03	7E-05
	Dibenzo(a,l)pyrene			30	3E-04	2E-05
	7,12-Dimethylbenz(a)anthracene			150	7E-05	5E-06
	3-Methylcholanthrene			13	8E-04	5E-05
Dioxins and dibenzofurans - analyze to levels below expected in background				**All TEFs**	5E-07	5E-07

RPF – relative potency factor
TEF - toxic equivalency factor

Appendix F - Additional Information on Default Fraction Organic Carbon (f_{oc})

In first calculations without the benefit of site-specific data, fraction organic carbon (f_{oc}) in littoral river sediment is often assumed to be about 2%. This value has been used as the default f_{oc} throughout this PHC. 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; US EPA, 1995a) suggest that the average ambient f_{oc} 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. Extremely high contamination would be required to increase the f_{oc} 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 Sediment Quality Targets (MPCA, 2007: discussed in PHC 7.1). In areas with tPAH concentrations above Level I criteria (1.6 mg/kg), the aquatic food chain is likely affected. In areas above Level II criteria (23 mg/kg), it is unlikely that a population of benthic invertebrates is surviving. Table F-1 shows tPAH concentrations as a surrogate for contamination effects on the food chain.

Table F-1 - Relationship between REMAP foc and tPAH data

	REMAP sites w/tPAH Concentrations									all REMAP estuary locations
	(tPAH < MPCA SQTs)			(tPAH > MPCA Level I SQT)				(tPAH >MPCA Level II SQT)		
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 f_{oc} sites
# of sites (n=)	8	12	19	23	27	29	37	40	42	87
f_{oc} 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 (shallow areas) accounted for 61-75% of sites in each tPAH category.

These data suggest that 2% f_{oc} is a reasonable default when considering partitioning and bioavailability of non-polar organic compounds in the St. Louis River Estuary.

Appendix G - PAH BSAFs

PAH BSAFs for fish are difficult to predict because:

- there are hundreds of PAHs, alkylated PAHs, C-substituted PAHs, halogenated PAHs that may be of interest for evaluation;
- fish are generally believed to metabolize most or all PAHs by a mechanism that is inducible;
- fish fillet cPAH data are typically not available at levels of interest due to elevated analytical detection limits and a limited list of analytes;
- PAH concentrations are likely to vary between fish of the same species depending on the size of the fish;
- contaminants in sediments are generally most available for accumulation when the sediments are biologically active sediments. As a result, BSAFs may be lower in more highly contaminated areas, making BSAF modeling uncertain.
- there is a general lack of data on the accuracy of different models for predicting relationship between physical/chemical characteristics of individual PAHs and their availability and food chain accumulation from sediments.

Due to uncertainties in available BSAF databases and inherent difficulties in deriving BSAFs for PAHs that may be applied across many sites, MDH recommends that fish that feed on or near PAH-contaminated sites be collected and analyzed using appropriate detection limits (Appendix E – Table E-1b). Site-specific PAH BSAFs can then be derived. In the absence of sufficient data, a BSAF of 0.085 may be used to approximate the exposure to BaP-PEQs in fish tissue. This BSAF is highly uncertain, but it is recommended to provide some level of protection from exposure to cPAHs by fish consumption. This BSAF is used for calculating BaP-PEQ fish consumption exposure in the 2013 SSVs.

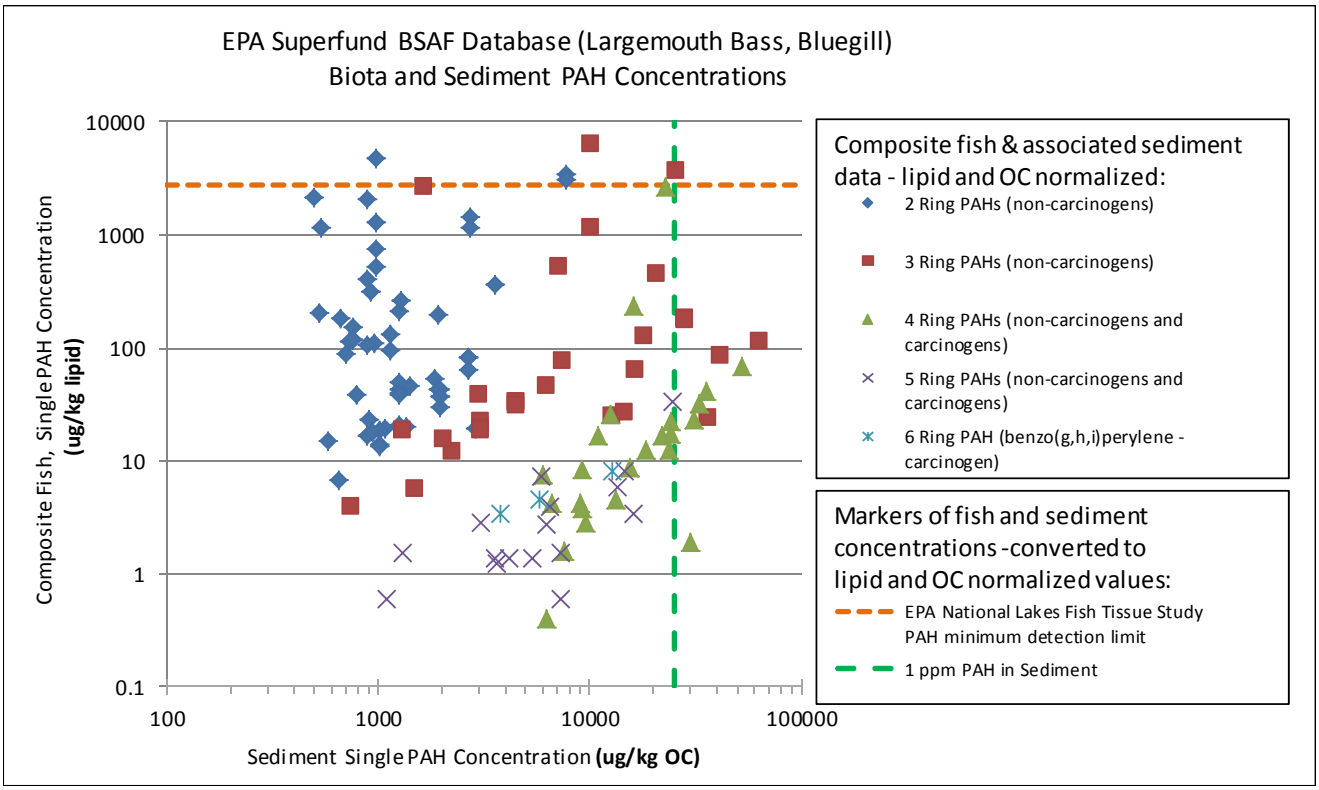
Developing a cPAH BSAF

There are only limited PAH fish tissue data available. In part, this is because detection limits for PAHs in fish tissues have generally been well above levels of concern. For instance, the EPA National Lake Fish Tissue Study PAH analytical method detection limit (MDL) was 111 µg/kg, with a method quantitation limit of 333 µg/kg. With BaP at the MDL, the calculated cancer risk from BaP is $8.2*10^{-5}$ for eating 1 fish fillet per week for a lifetime. This is the risk for a single cPAH at the method detection limit. For other MDH Priority cPAHs, the calculated cancer risk at 111 µg/kg in fish tissue ranges from $0.07*10^{-5}$ to $1.2*10^{-2}$ (benzo(g,h,i)perylene and 7,12-dimethylbenzanthracene, respectively). Data from the US EPA Superfund BSAF Database (US EPA, 2008; http://www.epa.gov/med/Prods_Pubs/bsaf.htm) demonstrates that nPAHs have been found in some fish tissue (including fillets) at this level (see Table G-1, below). While cPAH levels in sediments and in fish generally below nPAH levels, these data suggest that cPAH concentrations as BaP-PEQs may reach levels of concern in some fish.

Included in the US EPA Superfund BSAF Database are 128 PAH BSAFs calculated for largemouth bass and bluegills; both species are fished in Minnesota waters. Figure G-1 shows the sediment and fish tissue data used to calculate these BSAFs. The graph of log-transformed data suggests that there is a relationship between the concentration of PAHs in sediments and the concentration of those same PAHs in resident fish over a wide range of concentrations at

contaminated sites. The relationships between sediment and fish tissue concentrations appear to be confined to PAHs with three to six 6-carbon rings, with no apparent relationship for PAHs with two 6-carbon rings.

Figure G-1 - Biota (Largemouth Bass, Bluegill) and Associated Sediment PAH Concentrations (lipid and organic carbon adjusted)



The EPA database does not include data for most cPAHs. Generally, fish tissue databases do not contain useful cPAH data because cPAH analyte lists are limited and cPAH detection limits are too high. However, the EPA data are suggestive of some general trends in BSAFs based on similarities between the physical/chemical properties of the PAHs analyzed and the physical/chemical properties of cPAHs of interest.

Table G-1 shows the PAHs from the Superfunds BSAF Database for which BSAFs were calculated: the number of samples (BSAFs), PAH atomic weights, octanol-water constants, the number of 6-C rings in the PAH, median, mean and standard deviation for individual PAHs.

Table G-1 - PAH BSAF Summary Data (Largemouth Bass, Bluegills)

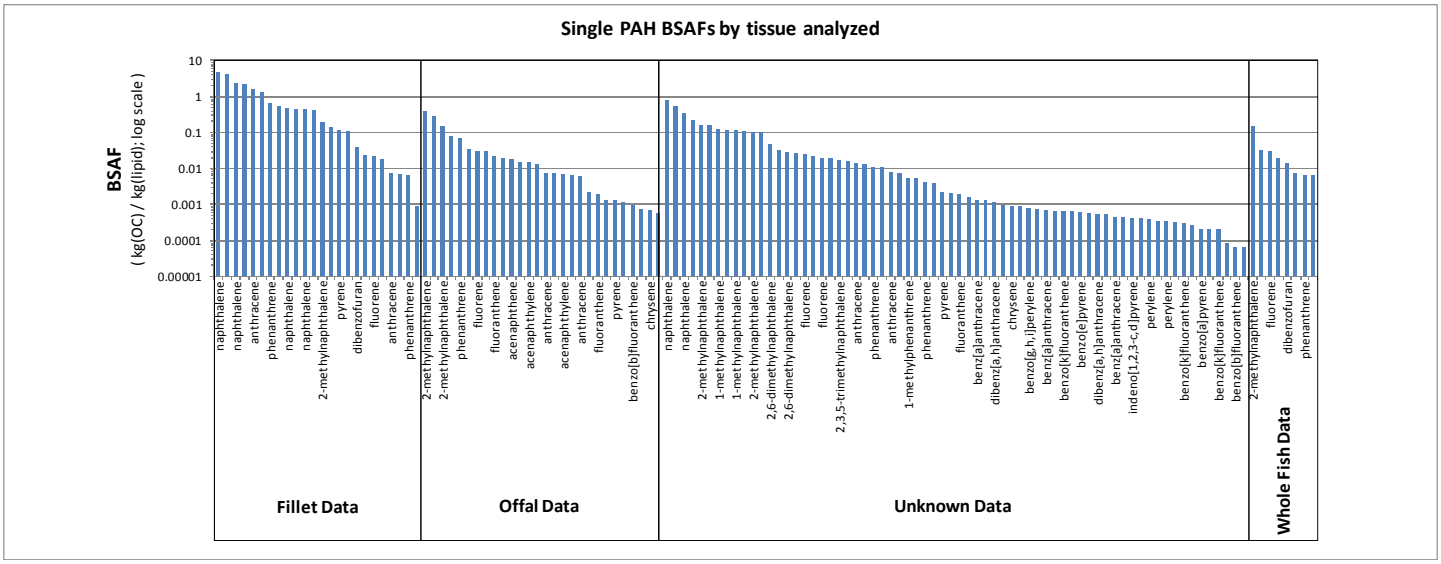
Polycyclic Aromatic Hydrocarbon	Molecular Weight	Log Kow	# of 6-C Rings	sample n	Median BSAF	Mean BSAF	Standard Deviation	95% UCL (Mean)
1-methylnaphthalene	142	3.87	2	3	0.13	0.13	0.023	0.16
2,3,5-trimethylnaphthalene	170	4.81	2	1		0.016		
2,6-dimethylnaphthalene	156	4.31	2	3	0.033	0.037	0.011	0.049
2-methylnaphthalene	142	3.86	2	7	0.16	0.18	0.096	0.25
acenaphthene	154	3.92	2	5	0.027	0.87	1.9	2.5
acenaphthylene	152	3.94	2	5	0.014	0.45	0.97	1.3
dibenzofuran	168	4.12	2	6	0.024	0.025	0.011	0.033
fluorene	170	4.18	2	9	0.022	0.024	0.0046	0.027
naphthalene	128	3.3	2	16	0.43	0.82	1.2	1.4
1-methylphenanthrene	192	5.08	3	3	0.0053	0.0048	0.00093	0.0059
anthracene	178	4.45	3	10	0.0073	0.17	0.5	0.48
fluoranthene	202	5.16	3	7	0.002	0.025	0.053	0.064
phenanthrene	178	4.46	3	10	0.0072	0.085	0.19	0.2
benz(a)anthracene	228	5.76	4	5	0.00068	0.00075	0.00032	0.001
benzo(b)fluoranthene	252	5.78	4	4	0.00021	0.00036	0.00043	0.00078
benzo(k)fluoranthene	252	6.11	4	3	0.0003	0.00038	0.00023	0.00064
chrysene	228	5.81	4	6	0.00082	0.00078	0.00029	0.001
pyrene	202	4.88	4	6	0.0018	0.023	0.046	0.059
benzo(a)pyrene	252	6.13	5	3	0.00021	0.00017	0.000074	0.00025
benzo(e)pyrene	252	6.44	5	3	0.00056	0.00048	0.00019	0.00069
dibenz(a,h)anthracene	278	6.75	5	3	0.00095	0.00089	0.00032	0.0012
indeno(1,2,3-c,d)pyrene	276	6.7	5	4	0.00044	0.00064	0.00047	0.0011
perylene	252	6.25	5	3	0.00039	0.00065	0.00049	0.0012
benzo(g,h,i)perylene	276	6.63	6	3	0.00079	0.00078	0.00013	0.00092
Summary all PAHs (by number of 6-C rings)								
			# Rings	**n=**	**Median**	**Mean**	**StDev**	**95% UCL**
			2	55	0.083	0.4	0.94	0.65
			3	30	0.007	0.09	0.31	0.2
			4	24	0.00074	0.0061	0.023	0.015
			5	16	0.00044	0.00057	0.00039	0.00076
			6	3	0.00079	0.00078	0.00013	0.00092
			2,3,4,5,6	128		0.19	0.65	0.31
			3,4,5,6	73		0.039	0.2	0.085
			4,5,6	43		0.0037	0.018	0.0089

Bolded PAHs are carcinogenic

Note that PAHs with 2 rings generally have the highest BSAFs.

The EPA database includes fillet data, offal data, data from unreported tissue, and whole fish data. Figure G-2 shows individual BSAF data separated into types of tissues analyzed.

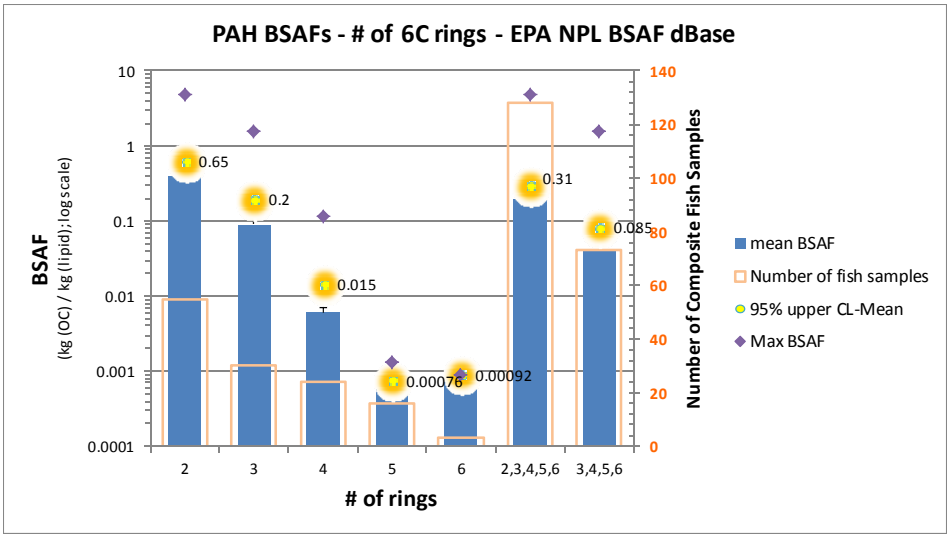
Figure G-2 - PAH BSAFs by Fish Tissue Analyzed



Note that the offal, unknown and whole fish BSAFs were generally below BSAFs for PAHs in fillets. This result is counter to what is generally believed. However, data are not sufficient for statistical comparison, and there are fewer PAHs measured in fillets than in offal and unknown samples, so the data may be misleading.

Figure G-3 shows mean BSAFs, 95% upper confidence limits means, and maximum reported BSAF for PAHs with two to six C-6 rings in largemouth bass and bluegills.

Figure G-3 - PAH BSAFs - # of 6-C Rings



Composite BSAF data are also shown for all PAHs (2,3,4,5,and 6 rings) and for PAHs with more than 3 or more rings. Again, note that PAHs with 2 and 3 rings have higher BSAFs than 4, 5, and 6 ring PAHs. This is somewhat counterintuitive: larger, more lipophilic PAHs are

generally thought to accumulate up the food chain more easily than small PAHs. It seems likely that these data demonstrate soluble PAHs evading surficial sediments. Fish may be directly exposed to dissolved PAHs in the water column. For lighter PAHs, this route of fish exposure may be much greater than through accumulation and transfer or magnification up the aquatic food chain.

PAH data from the EPA Superfund BSAF Database suggests that largemouth bass and bluegill fish tissue PAH concentrations are related to sediment PAH concentrations at Superfund sites, for PAHs with 3-6 C-6 rings. The MDH list of Priority cPAHs is predominantly made up of 4, 5 and 6 ring PAHs. However, benzo(c)fluorene is an important 3-ring cPAH that is found in environmental mixtures. Due to uncertainties in the data, the 95% upper confidence limit of the mean (95% UCL) PAH BSAF for 3-6 C-6 ring PAHs for largemouth bass and bluegills in the EPA Superfund BSAF Database was used to estimate fish cPAH bioaccumulation and fish tissue concentration in the SSM. PAH BSAFs used in the SSM, as well BSAFs used in the 2005 SSVs are listed in Table G-2.

Table G-2 - Sediment Screening Model Biota-Sediment Accumulation Factors(BSAFs): 2005, 2013

Polycyclic Aromatic Hydrocarbons (PAHs)	Molecular Weight	# of 6-C rings	Log K_{ow}	2005 SSV	2013 SSM
				WaDOH Log K_{ow} Analysis	EPA Data 6-C Ring Analysis
Benzo(a)pyrene equivalents (BaP-PEQs)	**202 - 302**	**3-6**	**5.16 - 7.71**	**0.11**	**0.085**
Acenaphthene	154	2	3.92	0.55	0.65
Acenaphthylene	152	2	3.94	0.55	0.65
Anthracene	178	3	4.45	0.05	0.2
Benzo(a)pyrene	252	5	6.13	0.11	0.00076
Fluoranthene	202	3	5.16	0.11	0.2
Fluorene	170	2	4.18	0.083	0.65
Methylnaphthalene	142	2	3.87	0.55	0.65
Naphthalene	128	2	3.3	0.35	0.65
Perylene	252	5	6.25	0.11	0.0008
Phenanthrene	178	3	4.46	0.083	0.2
Pyrene	202	4	4.88	0.11	0.016

It is important to note that there is a strong linear log-log relationship in available data between the octanol-water constant (K_{ow}) and individual PAH BSAFs (largemouth bass and bluegills) (Log BSAF = -1.03 x Log K_{ow} + 3.2; R^2 = 0.86). If the relationship holds when data are available for additional cPAHs at reasonable detection limits, it may be appropriate to apply modeled BSAFs to individual cPAHs in proportion to their potency and concentration at contaminated sites.

Appendix H - Route-Specific Sediment Screening Model 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 } = RSC * RfD/(ABS_{Sed}*Sed_{Ing}) * $CF_{mg/kg}$ *Equation A-16.*

Calculation results are shown in Table H-1.

Water ingestion

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

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

Section 5.2 of the Public Health Consultation (PHC) 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, that there is a small boundary layer at the sediment water interface where the chemical phases are at equilibrium and that there is a 10-fold default dilution in surface water.

Back-calculated route-specific sediment limits for organic compounds are dependent on surface water dilution, equilibrium partitioning between sediment and water, and water ingestion rate. The water ingestion route-specific SSM results ($SSV_{SW(Ing)}$) are calculated by substituting *PHC Eq. 2 (5.2)* into *Eq. A-17*:

$SSV_{SW(Ing)}$ { mg/kg } = RSC * RfD * K_{oc} * f_{oc} * $DF_{sed\text{-}sw}$ / 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). Model results for non-cancer effects of sediment-dermal exposure alone are:

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

Calculation results are shown in Table H-1.

Screening model results 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. A protective dissolved surface water concentration for non-cancer effects from water-dermal exposure alone will be:

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

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:

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

Back-calculating sediment values are dependent on surface water dilution and equilibrium partitioning between sediment and water. The dermal-water route-specific SSM results ($SSV_{SW(Derm)}$) are calculated by substituting *Eq. 2 (PHC)* into *Eq. A-20*:

$$SSV_{SW(Derm)} \{ mg/kg \} = RSC * RfD * ABS_{GI} * K_{oc} * f_{oc} * DF_{sed-sw} / (ABS_{SW} * SW_{Derm}) * CF_{cm3/L} \quad \textit{Equation A-21.}$$

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) (US EPA, 2001).

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

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

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:

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

Calculations of event duration-dependent factors are chemical specific:

τ { 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 } = $2.4 * \tau$ *Equation A-26.*

If: $\beta > 0.6$

then: t^* { hr } = $6 * \tau (b - \sqrt{(b^2 - c^2)})$ *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 } = $FA* (ED / (1 + \beta) + 2 * \tau * (1 + 3 * \beta + 3 * \beta^2) / (1 + \beta)^2)$ *Equation A-31.*

Calculation results are shown in Table H-1.

Calculating protective sediment concentrations for inhalation exposure

Back-calculation of sediment concentrations necessary to achieve inhalation exposure limits for non-polar organic compounds is performed using inhalation exposure limits, site-specific exposure calculations, and equations incorporating water dilution, air dilution and equilibrium partitioning from PHC Section 5.2.

SSV_{Inh} { mg/kg } = $RSC * RfC / Inh_{frac} * K_{oc} * f_{oc} * DF_{sed\text{-}sw} * R * T * DF_{sw\text{-}air} / (K_H * CF_{L/m3} * CF_{L/m3})$ *Equation A-32.*

Calculation results are shown in Table H-1.

A protective dissolved surface water concentration for the inhalation pathway can be calculated from:

SWC_{Inh} { mg/L } = $RSC * RfC / Inh_{frac} * R * T * DF_{sw\text{-}air} / (K_H * CF_{L/m3} * CF_{L/m3})$ *Equation A-33.*

Calculating route-specific sediment screening values for fish ingestion route exposure

For organics (excluding volatiles):

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

For inorganics and mercury:

SSV_{Fish} { mg/kg } = RSC * 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 Model Results

Route-Specific SSM Results: Non-Cancer	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	**78.7**		138			
Cadmium	7440-43-9	**26.2**		34.5			
Chromium III	16065-83-1	**393000**					
Chromium VI	18540-29-9	**262**					
Copper	7440-50-8	**10500**					
Cyanide	57-12-5	**1570**					
Lead *	7439-92-1	**300**					
Mercury (SSV - tHg; exp - tHg)	7439-97-6	**78.7**					
Methyl Mercury (SSV - tHg; exp - MeHg)	22967-92-6	95.7					**0.0202**
Nickel	various	**5250**					
Zinc	7440-66-6	**78700**					
Volatile organic compounds (VOCs)							
Benzene	71-43-2	1050	11.3	111	13.6	**1.84**	
Ethyl benzene	100-41-4	26200	866	2760	265	**119**	
Styrene	100-42-5	52500	1730	5530	**712**	1140	
Toluene	108-88-3	21000	363	2210	192	**98.8**	
Xylenes (mixed)	1330-20-7	52500	1490	5530	421	**51.8**	
Polycyclic Aromatic Hydrocarbons (PAHs)							
Acenaphthene	83-32-9	15700	5850	6380	767	40200	**56.2**
Acenaphthylene (toxicity surrogate - pyrene)	208-96-8	7870	2930	3190	367	34000	**28.1**
Anthracene	120-12-7	78700	95300	31900	6510	2270000	**907**
Benzo(a)pyrene equivalents (BaP-PEQs)	50-32-8						
Fluoranthene	206-44-0	10500	43000	4250	1580	6140000	**121**
Fluorene	86-73-7	10500	7110	4250	696	93500	**37.4**
Methylnaphthalene (toxicity surrogate - naphthalene)	1321-94-4	4200	785	1700	103	310	**15**
Naphthalene	91-20-3	4200	479	1700	133	221	**15**
Perylene (toxicity surrogate - pyrene)	198-55-0	7870	34900	3190	**249**	12700000	22700
Phenanthrene (toxicity surrogate - pyrene)	85-01-8	7870	9720	3190	656	304000	**90.7**
Pyrene	129-00-0	7870	31600	3190	1310	3520000	**1170**
Dibenzo-p-dioxins/dibenzofurans							
2,3,7,8-TCDD equivalents (TCDD-TEQs)	1746-01-6	1.84E-04	0.00338	3.22E-04	3.12E-05	1.4	**4.23E-07**
Other Organics							
Carbazole	86-74-8						
Hexachlorobenzene	118-74-1	13.1	6.01	6.91	**0.243**	2.34	0.302
Octachlorostyrene	29082-74-4	7.87	32.1	4.14	0.0833	92.4	**0.0185**
Pentachlorophenol	87-86-5	1310	481	276	**6.61**	13000000	
Polychlorinated Biphenyls (PCBs)	1336-36-3	5.25	30.3	1.97	0.154	48.3	**0.00465**
Quinoline	91-22-5						

* MPCA Soil Reference Value (MPCA, 2009)

Shaded Routes of exposure incorporate boundary layer-to-exposure dilution factors described in PHC Section 5.2

Bolded route-specific SSM results identify the most important route-of-exposure for each chemical

Table H-1 contains SSM results to 3 significant digits because these data are intermediate calculations. SSVs are screening values and application should be limited to 1 significant digit.

Appendix I - Route-Specific Sediment Screening Model 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. Age-dependent potency adjustment factors (ADAFs) have been incorporated into the exposure factors previously (Appendix B) to match age-specific exposures and sensitivity. Use of ADAFs for individual carcinogens are noted in Table D-2.

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

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

Calculation results are shown in Table I-1.

Water ingestion

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

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

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

$SSV_{SW(Ing)\text{-}c}$ { mg/kg } = ($AccptRsk_c$ / CSF) / $SW_{Ing\text{-}c}$ * K_{oc} * f_{oc} * $DF_{sed\text{-}sw}$ *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:

$SSV_{Sed(Derm)\text{-}c}$ { mg/kg } = ($AccptRsk_c$ / CSF) * ABS_{GI} / (ABS_{Derm} * $Sed_{Derm\text{-}c}$) * $CF_{mg/kg}$ *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:

$SWC_{SW(Derm)\text{-}c}$ { mg/L } = ($AccptRsk_c$ / CSF) * ABS_{GI} / (ABS_{SW} * $SW_{Derm\text{-}c}$) * $CF_{cm3/L}$ *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 *PHC Eq. 2* (5.1) into *Eq. A-40*:

$$SSV_{SW(Derm)\text{-}c}\ \{\ mg/kg\ \} = (AccptRsk_c / CSF) * ABS_{GI} / (ABS_{SW} * SW_{Derm\text{-}c}) * K_{oc} * f_{oc} * DF_{sed\text{-}wat} * CF_{cm3/L} \qquad \textit{Equation A-41.}$$

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.

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

Calculation results are shown in Table I-1.

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

$$SWC_{Inh\text{-}c}\ \{\ mg/L\ \} = (AccptRsk_c / UR_c) / Inh_{frac\text{-}c} * R * T * DF_{sw\text{-}air} / (K_H * CF_{L/m3} * CF_{L/m3} * CF_{\mu g/mg}) \qquad \textit{Equation A-43.}$$

If measured water concentrations exceed $SWC_{Inh\text{-}c}$, additional characterization of the inhalation route of exposure is recommended.

Calculating sediment screening values for fish ingestion exposure - Cancer

For organics (excluding volatiles):

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

For inorganics:

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

Calculation results are shown in Table I-1.

Table I-1 - Route-specific Sediment Screening Model Results

Route-Specific SSVs: Cancer	Cas No.	Sediment Ingestion	Surfacewater Ingestion	Dermal Sediment	Dermal Surfacewater	Inhalation	Fish Consumption
		$SSV_{sed(ing)-c}$	$SSV_{sw(ing)-c}$	$SSV_{sed(derm)-c}$	$SSV_{sed(derm)-c}$	SSV_{inh-c}	SSV_{fish-c}
		mg/kg	mg/kg	mg/kg	mg/kg	mg/kg	mg/kg
Arsenic	7440-38-2	**61.6**		64.6			
Chromium VI	18540-29-9	**43.6**					
Benzene	71-43-2	396	4.28	35.4	4.46	**1.42**	
Benzo(a)pyrene equivalents (BaP-PEQs)	50-32-8	12.8	557	4.41	4.06	494000	**0.212**
2,3,7,8-TCDD equivalents (TCDD-TEQs)	1746-01-6	1.56E-05	0.000287	2.32E-05	2.30E-06	0.0554	**2.19E-08**
Carbazole	86-74-8	1090	739	487	**124**	8080000	
Hexachlorobenzene	118-74-1	13.6	6.25	6.09	0.22	4.66	**0.192**
Pentachlorophenol	87-86-5	231	84.8	29.1	**0.753**	2850000	
PCBs (Polychlorinated Biphenyls)	1336-36-3	10.9	63	3.48	0.279	795	**0.0059**
Quinoline	91-22-5	7.27	**0.83**	3.25	1.43	631	

Shaded Routes of exposure incorporate boundary layer-to-exposure dilution factors described in PHC Section 5.2

Bolded carcinogens have default ADAF applied to SSM

Bolded SSM results identify the most important route-of-exposure for each chemical

Table I-1 contains data to 3 significant digits because these data are intermediate calculations. SSVs are screening values and application should be limited to 1 significant digit.

Appendix J – Combining Routes of Exposure - Sediment Screening Model

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

Sediment Screening Model Results

Combined chronic sediment screening values for all routes-of-exposure analyzed in this PHC 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 PHC 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 PHC 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 PHC Table 12 (Section 7).

Table J-1 - - Sediment Screening Model Results – Contribution from Different Routes of Exposure

Chemicals Evaluated with SSM	CAS No.	Endpoint (Non-cancer / Cancer)	Multi-route Sediment Screening Model Result	Contributions From Different Routes-of-Exposure (model attribution)					
				Sediment Ingestion	Surfacewater Ingestion	Dermal from Sediment	Dermal from Surfacewater	Inhalation	Fish Consumption
Metals - Inorganics			mg/kg	% Sed(Ing)	% SW(Ing)	% Sed(Derm)	% SW(Derm)	% Inh	% Fish
Arsenic	7440-38-2	Non-cancer	50	**64%**	*Not Evaluated*	36%	*Not Evaluated*	*Not Evaluated*	*Not Evaluated*
		Cancer	**30**	**51%**		49%			
Cadmium	7440-43-9	Non-cancer	**10**	**57%**		43%			
Chromium III	16065-83-1	Non-cancer	**400000**	**100%**		*Not Evaluated*			
Chromium VI	18540-29-9	Non-cancer	300	**100%**					
		Cancer	**40**	**100%**					
Copper	7440-50-8	Non-cancer	**10000**	**100%**					
Cyanide	57-12-5	Non-cancer	**2000**	**100%**					
Lead	7439-92-1	Non-cancer	**300 ***	*Not Evaluated*					
Mercury (SSM - tHg; exp - tHg)	7439-97-6	Non-cancer	0.02 †	**0.00023%**					**100%**
Mercury (SSM - tHg; exp - MeHg)									
Nickel	various	Non-cancer	**5000**	**100%**					*Not Evaluated*
Zinc	7440-66-6	Non-cancer	**80000**	**100%**					
Volatile organic compounds (VOCs)									
Benzene	71-43-2	Non-cancer	1	0.13%	12%	1.3%	10%	**76%**	*Not Evaluated*
		Cancer	**0.8**	0.21%	20%	2.4%	19%	**59%**	
Ethyl benzene	100-41-4	Non-cancer	**70**	0.28%	8.4%	2.6%	28%	**61%**	
Styrene	100-42-5	Non-cancer	**300**	0.62%	19%	5.9%	**46%**	29%	
Toluene	108-88-3	Non-cancer	**50**	0.26%	15%	2.4%	28%	**55%**	
Xylenes (mixed)	1330-20-7	Non-cancer	**40**	0.085%	3%	0.8%	11%	**86%**	
Polycyclic Aromatic Hydrocarbons (PAHs)		Non-cancer							
Acenaphthene	83-32-9	Non-cancer	**50**	0.33%	0.88%	0.8%	6.7%	0.13%	**91%**
Acenaphthylene (toxicity surrogate - pyrene)	208-96-8	Non-cancer	**30**	0.33%	0.87%	0.8%	7%	0.075%	**91%**
Anthracene	120-12-7	Non-cancer	**800**	0.97%	0.8%	2.4%	12%	0.034%	**84%**
Benzo(a)pyrene equivalents (BaP-PEQs)	50-32-8	Cancer	**0.2** †	1.5%	0.034%	4.3%	4.7%	0.000038%	**90%**
Fluoranthene	206-44-0	Non-cancer	**100**	1%	0.25%	2.5%	6.8%	0.0018%	**89%**
Fluorene	86-73-7	Non-cancer	**30**	0.33%	0.49%	0.82%	5%	0.037%	**93%**
Methylnaphthalene (toxicity surrogate - naphthalene)	1321-94-4	Non-cancer	**10**	0.29%	1.6%	0.72%	12%	3.9%	**81%**
Naphthalene	91-20-3	Non-cancer	**10**	0.29%	2.5%	0.72%	9.2%	5.5%	**81%**
Perylene (toxicity surrogate - pyrene)	198-55-0	Non-cancer	**200**	2.8%	0.63%	6.9%	**89%**	0.0017%	0.97%
Phenanthrene (toxicity surrogate - pyrene)	85-01-8	Non-cancer	**80**	0.97%	0.79%	2.4%	12%	0.025%	**84%**
Pyrene	129-00-0	Non-cancer	**500**	6.1%	1.5%	15%	37%	0.014%	**41%**
Dibenzo-p-dioxins/dibenzofurans									
2,3,7,8-TCDD equivalents (TCDD-TEQs)	1746-01-6	Non-cancer	4.0E-07	0.23%	0.012%	0.13%	1.3%	0.00003%	**98%**
		Cancer	**0.00000002** †	0.14%	0.0076%	0.094%	0.94%	0.000039%	**99%**
Other Organics									
Carbazole	86-74-8	Cancer	**80**	7.4%	11%	17%	**65%**	0.001%	*Not Evaluated*
Hexachlorobenzene	118-74-1	Non-cancer	0.1	0.93%	2%	1.8%	**50%**	5.2%	40%
		Cancer	0.1	0.71%	1.5%	1.6%	44%	2.1%	**50%**
Octachlorostyrene	29082-74-4	Non-cancer	**0.02**	0.19%	0.047%	0.36%	18%	0.016%	**82%**
Pentachlorophenol	87-86-5	Non-cancer	6	0.48%	1.3%	2.3%	**96%**	0.000049%	*Not Evaluated*
		Cancer	**0.7**	0.31%	0.86%	2.5%	**96%**	0.000025%	
Polychlorinated Biphenyls (PCBs)	1336-36-3	Non-cancer ‡	**0.005** †	0.086%	0.015%	0.23%	2.9%	0.0093%	**97%**
		Cancer	0.006 †	0.053%	0.0091%	0.17%	2.1%	0.00072%	**98%**
Quinoline	91-22-5	Cancer	**0.4**	5.8%	**51%**	13%	30%	0.067%	*Not Evaluated*

Shaded Routes of Exposure – Boundary layer exposure dilution factor(s) applied. See PHC Section 5.2

* MPCA Soil Reference Value

† (shaded chemicals) SSM results may approach or be less than ambient or background concentration. See sections on individual chemicals in PHC Section 4, PHC Section 7.1.2 and Appendix C for information on ambient and background concentrations.

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